THE HEALTH OF CHILDREN AND YOUNG PEOPLE
WITH CHRONIC CONDITIONS AND DISABILITIES

IN THE NORTHERN DISTRICT HEALTH BOARDS
The Health of Children and Young People with Chronic Conditions and Disabilities in the Northern District Health Boards

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BACKGROUND AND AIMS

Background
The 2006 Disability Survey [1] estimated that 10% of New Zealand children aged 0–14 years had a disability, with the most common disability cited (5% of all children) being the requirement for special education. A further 4% of children had chronic health conditions such as severe asthma, cerebral palsy, or diabetes, while 2% had a psychiatric or psychological disability. Around half (52%) of disabled children in the Survey had a disability arising from a condition that had existed since birth, while 26% had disabilities that were caused by a disease or illness, and 3% by an injury [1].

More recently, the 2011/12 NZ Health Survey [2] estimated that 20.7% of New Zealand children aged 2–14 years were overweight and that 10.3% were obese. Further, it was found that the proportion of children who were obese had increased significantly since the 2006/07 NZ Health Survey. Such increases are of concern, as in addition to being associated with conditions like type 2 diabetes and slipped upper femoral epiphysis in adolescence [3,4], childhood obesity increases the risk of high blood pressure, coronary heart disease, and stroke in later life [3,5].

Aims of this Report
While such surveys provide very broad prevalence estimates, their lack of clinical precision means it is very difficult to obtain a detailed understanding of the nature and causes of disabilities and chronic conditions (including obesity) in New Zealand children and young people. This paucity of information in turn, makes it difficult for those working in the health sector to plan services to meet future demand, or to develop evidence-based strategies for prevention. Despite this, children and young people with disabilities and chronic conditions require a range of health and disability support services to reach their full potential, and it is undesirable that a paucity of data should preclude them featuring prominently in prioritisation, planning and resource allocation decisions.

With these issues in mind, this report collates a range of routinely collected data sources with a view to:

1. Estimating the prevalence of conditions arising in the perinatal period (e.g. preterm births, congenital and chromosomal anomalies) which may lead to greater health and disability support service demand during childhood and adolescence
2. Identifying the numbers of children and young people with specific chronic conditions and disabilities, who are accessing secondary healthcare services
3. Reviewing the distribution of overweight and obesity and its determinants (nutrition, physical activity) in children and young people

In-Depth Topics
In addition, two issues were selected for more in-depth review by participating DHBs at the beginning of the year, with one of these issues, the treatment of obesity in children and adolescents, being split onto two parts due to the large volume of literature in this area. This year’s in depth topics are thus:

1. **The Determinants and Consequences of Overweight and Obesity**: This in-depth topic begins by providing some background information on the distribution of obesity, including its prevalence in different population groups, before reviewing the range of definitions for overweight and obesity currently used in the literature. The natural history of obesity over the lifespan is then briefly described, before the determinants of obesity are reviewed and the short and long term consequences discussed.

2. **The Treatment of Obesity in Children and Adolescents**: This in depth topic provides information on evidence-based interventions for the treatment of obesity in children
and adolescents. It begins by discussing some of the difficulties associated with identifying and engaging children (and their parents) who are candidates for weight management interventions, before considering the findings of a 2009 Cochrane review of obesity interventions. Insights from other relevant reviews are then discussed, before current New Zealand interventions are summarised. The evidence for the effectiveness of brief primary care interventions is then considered, and a number of individual primary care programmes are presented. The in-depth topic concludes with a brief summary of the key findings from the literature in this area.

3. **Children of Parents with Mental Illness and Alcohol and Other Addictions (COPMIA):** This in-depth topic considers issues experienced by the children of parents with mental health issues and alcohol and other addictions and identifies evidence-based programmes that could be implemented to reduce risk and enhance resilience in these children. It begins by reviewing the New Zealand prevalence and health and support needs of children of parents with mental illness and addiction issues, as well as the impacts on their health, development and psycho-social wellbeing. Optimal service delivery models are then reviewed from an international perspective, and an example of a best practice systems model is presented. New Zealand strategies and plans are then briefly summarised, along with examples of current COPMIA services in this country. The review concludes with a series of recommendations as to how services for COPMIA might be improved locally.

**REPORT STRUCTURE AND CONTENT**

This report is the third of a three-part series on the health of children and young people in the Northern DHBs and fits into the reporting cycle as follows:

- **Year 1** The Health Status of Children and Young People
- **Year 2** The Determinants of Health for Children and Young People
- **Year 3** Children and Young People with Chronic Conditions and Disabilities

As previously, this report is based on an *Indicator Framework* [6] developed by the NZ Child and Youth Epidemiology Service, with all of the indicators in the *Chronic Conditions and Disabilities* stream being updated in this year’s edition. These indicators have been grouped into four sections, as outlined below, with an in-depth topic on the children of parents with mental health issues and alcohol and other addictions (COPMIA) forming the fifth and final section.

**Section 1: Conditions Arising in the Perinatal Period**

This section is divided into two parts, with the first reviewing two key perinatal outcomes: fetal deaths and preterm births. The second part begins with a brief overview of antenatal and newborn screening, before using hospital birth data to review the prevalence of congenital anomalies in newborn babies. This review is spread across four chapters, with the first exploring the range of anomalies (from minor to severe) identified in hospital born babies. Subsequent chapters provide additional detail on three anomalies which are usually identifiable at birth, and which may lead to significant health and/or disability support service utilisation. These are cardiovascular anomalies, chromosomal anomalies including Down syndrome, and spina bifida and other neural tube defects.

**Section 2: Other Disabilities**

This section begins with a review of children and young people with permanent hearing loss using NZ Deafness Notification Database and Newborn Hearing Screening data. Then, as a result of a paucity of other routinely collected data sources, it uses hospital admission data to explore access to secondary health services in children and young people aged 0–24 years with any mention of cerebral palsy or autism spectrum disorder in any of their first 15 diagnoses. For each condition, the main reasons for hospital admission are explored, along with their distribution by age, ethnicity and gender.
Section 3: Chronic Medical Conditions
This section reviews hospital admissions for children aged 0–14 years with eczema and dermatitis, as well as hospital admissions and mortality for children and young people aged 0–24 years with inflammatory bowel disease, cystic fibrosis, type 1 diabetes, and epilepsy. Again the main reasons for hospital admission are described, along with their distribution by age, ethnicity and gender. Cancer incidence and mortality in children and young people aged 0–24 years is then explored using data from the NZ Cancer Registry and the National Mortality Collection.

Section 4: Obesity, Nutrition and Physical Activity
This section is divided into two parts, with the first reviewing overweight and obesity in children and young people and the second reviewing breastfeeding, nutrition and physical activity. Part 1 begins with an in-depth topic which explores the determinants and consequences of obesity in children and young people, before reviewing the distribution of overweight and obesity and its complications using 2011/12 NZ Health Survey, Youth’12 Survey and hospital admission data. Part 1 concludes with a second in-depth topic, which explores the treatment of obesity in children and adolescents. Part 2 then begins with a review of breastfeeding and the early introduction of solids using Plunket and 2011/12 NZ Health Survey data, before exploring a range of nutrition and physical activity indicators using 2011/12 NZ Health Survey and Youth’12 Survey data.

Section 5: Children of Parents with Mental Illness and Alcohol and Other Addictions (COPMIA)
This in-depth topic considers the current issues experienced by the children of parents with mental health issues and alcohol and other addictions in New Zealand and identifies evidence-based effective programmes that could be implemented to reduce risk and enhance resilience in these children.

Reviews of Evidence-Based Interventions
Each of the chapters in this report concludes with a brief overview of local policy documents and evidence-based reviews which consider population level approaches to prevention or management. Appendix 1 provides an overview of the methodology used to develop these reviews. As previously, the quality and depth of evidence available varied from indicator to indicator (e.g. a large number of reviews were available on the medical management of those with cystic fibrosis, but few (with the exception of folate for neural tube defects) were available on the primary prevention of congenital anomalies).

Notes on Data Quality and the Signalling of Statistical Significance
One of the main purposes of this report is to inform health needs assessment. Thus, as previously, where high quality data was not available, yet an issue was deemed to be of public health importance, “bookmark” indicators have been included (e.g. hospital admissions for those with autism spectrum disorders) so that the needs of these children and young people do not fall below the public health radar. In such cases, the reader is urged to read the cautions on interpretation which accompany these indicators, in order to gain a better understanding of the strengths and weaknesses of the data used.

Further, Appendix 2 outlines the rationale for the use of statistical significance testing in this report and Appendix 3–Appendix 5 contain information on the data sources used to develop each indicator. Readers are urged to be aware of the contents of these Appendices when interpreting the information in this report.

In particular (as outlined in Appendix 2), in order to assist the reader to determine whether tests of statistical significance have been used in a particular section, the statistical significance of the associations presented has been signalled in the text with the words significant, or not significant in italics. Where the words significant or not significant do not appear in the text, then the associations described do not imply statistical significance or non-significance.
OVERVIEW OF REPORT’S KEY FINDINGS

Previous reports in this series have focused on infectious and respiratory diseases, where hospital admissions and mortality have tended to track in a manner consistent with the region’s demographic profile (e.g. with rates being much higher for DHBs with a high proportion of children living in the most deprived (NZDep deciles 9–10) areas). However, for chronic conditions and disabilities the picture is more complex, as the prevalence of many of these conditions is not influenced by socioeconomic factors. Instead factors such as genetics (e.g. for cystic fibrosis) and maternal age (e.g. for some congenital anomalies) play much greater roles. For many chronic conditions, such as autism spectrum disorder and cancer, the underlying cause is unknown in the vast majority of cases.

Further adding to the complexity is the role that local health services play, as the majority of care for children and young people with chronic conditions and disabilities is delivered in primary care or the outpatients setting. In this context, local service delivery configurations (e.g. which children are admitted vs. which are managed in outpatients for various procedures or conditions) may heavily influence hospital admission rates.

Thus when considering Table 1, which provides an overview of the indicators in this year’s report, and the key findings as they relate to the DHBs in the Northern region, it is difficult to make any overall generalisations about the way any individual DHB’s health outcomes are tracking with respect to national rates. Rather what is presented is a mixed picture, with rates in some of the Northern DHBs being higher than the New Zealand rate for some conditions, and lower for others, but with no consistent pattern emerging across the region. Thus for this year’s report, it will be necessary to review Table 1 on a condition by condition basis, in order to obtain an picture of what is occurring within the region.

Concluding Comments

This report reviews the prevalence of conditions arising in the perinatal period that may increase the demand for health and disability support services, as well as the secondary health service utilisation patterns of children and young people with chronic conditions and disabilities. In addition, it aims to provide some insights into the consequences and management of overweight and obesity in children and young people, as well as the needs of children with parents with mental health issues and alcohol and other addictions.

While the data presented are at times imperfect, and at best only provide a glimpse of the health needs of these diverse groups of children and young people, the current paucity of data should not preclude DHBs reviewing the health and disability support services available locally (including those with a health promotion focus), with a view to considering whether further improvements are required within the region.

Further, while high quality evidence (e.g. from randomised controlled trials) is often lacking, there is nevertheless sufficient information to direct future initiatives in many areas. These include the development of integrated services for the children of parents with mental health issues and alcohol and other addictions, and interventions for the prevention and management of overweight and obesity in children and young people.
Table 1. Overview of the Health of Children and Young People with Chronic Conditions and Disabilities in the Northern DHBs

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<tr>
<th>Stream</th>
<th>Indicator</th>
<th>New Zealand Distribution and Trends</th>
<th>Northern DHBs Distribution and Trends</th>
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|        | Fetal Deaths | • During 2006–2010, unspecified cause was the most frequent fetal cause of intermediate fetal deaths (IFD, 20 to 27 weeks gestation), followed by prematurity/LBW and congenital and chromosomal anomalies.  
  • Unspecified cause was the most frequent fetal cause of late fetal deaths (LFD, 28+ weeks gestation) followed by malnutrition/slow fetal growth. Congenital anomalies still made a significant contribution.  
  • Fetal deaths exhibited a J-shaped distribution with gestational age, with a peak at <25 weeks, and rates increasing again after 37 weeks. Fetal deaths from congenital anomalies and prematurity/LBW were highest in babies <25 weeks, while unspecified fetal deaths increased rapidly after 37 weeks.  
  • During 2006–2010, there were no significant gender, ethnic, or NZDep06 differences in IFD rates. Mortality was significantly higher for the babies of younger (<25 years) and older (35+ years) women, than for those 30–34 years.  
  • During 2006–2010, LFDs were significantly higher for Pacific > Māori > European/Other babies, and babies from average to deprived (NZDep deciles 5–10) areas. Rates were significantly higher for babies of teenage women, than those 30–34 years. | • In all four Northern DHBs during 2006–2010, congenital and chromosomal anomalies collectively were the most frequently listed main fetal causes of IFD, followed by unspecified causes and prematurity/low birth weight.  
  • Unspecified cause was the most frequently listed fetal cause of LFD in all four DHBs, although malnutrition/slow fetal growth, intrauterine hypoxia and congenital and chromosomal anomalies also made a contribution.  
  • In all four Northern DHBs, IFD rates were not significantly different from the NZ rate. While LFD rates in the Northland, Waitemata and Auckland DHBs were also not significantly different from the NZ, in Counties Manukau rates were significantly higher (RR 1.36 95% CI 1.17–1.58). |
| Perinatal Conditions | Preterm Birth in Singleton | • During 2000–2012, singleton preterm birth rates at 20–27, 28–31, and 32–36 weeks were static. The actual number of preterm babies born increased however, as the result of a rising birth rate. The largest increases were in those born at 32–36 weeks.  
  • During 2008–2012, preterm birth rates (20–36 weeks) were significantly higher for males and for Māori babies. Rates were also significantly higher for those in more deprived (NZDep deciles 7–10) areas, and for the babies of younger (<25 years) and older (35+ years) mothers, than for those aged 25–29 years.  
  • When broken down by gestational age, the excess risk of preterm birth seen for Māori, Pacific and Asian/Indian babies, the babies of teenage mothers, and those from the more deprived areas, was greatest in births at lower gestations. | • During 2008–2012, 126.0 Northland, 436.2 Waitemata, 350.4 Auckland and 531.6 Counties Manukau babies per year were born <37 weeks gestation, with the majority being in 32–36 weeks.  
  • During 2000–2012, preterm births in Northland and Auckland were lower than the NZ rate. In Waitemata, a gradual decline saw rates become lower than the NZ rate from 2008–09. In Counties Manukau there was considerable variability.  
  • In the Northland, Auckland and Counties Manukau DHBs, preterm birth rates were higher for Māori than for European/Other babies. In the Waitemata DHB there were no consistent ethnic differences in rates. |
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| Perinatal Conditions         | Preterm Birth in Multiple Pregnancies          | • During 2008–2012, preterm birth rates were 6.0% for singletons, 55.1% for twins and 98.7% for triplets. The risk of preterm birth was 9.13 (95% CI 8.92–9.35) times higher for twins and 16.34 (95% CI 16.02–16.68) times higher for triplets, than for singletons.  
• During 2000–2012, preterm birth rates were static in singletons and triplets. Rates in twins increased, with the majority of this increase occurring after 2008.  
• During 2008–2012, there was no significant gender, NZDep06, or maternal age differences in preterm birth rates in twins. Rates for Pacific twins however, were significantly (albeit marginally) lower than for European/Other babies. | In the Northern DHBs during 2008–2012, a large number of congenital anomalies were identified at birth, with these ranging in severity from minor (e.g. tongue tie) through to serious (e.g. malformations of the great arteries).  
• On average during 2008–2012, 111 Northland, 496 Waitemata, 366 Auckland and 399 Counties Manukau babies per year (range 4.6–6.5% of all births) had one or more congenital anomalies identified at birth. Rates in Counties Manukau were similar to the NZ rate, while rates in the Northland, Waitemata, and Auckland DHBs were significantly higher.  
• It is unclear whether DHB vs. NZ differences in congenital anomaly rates reflect real differences in the underlying prevalence of congenital anomalies in the community, or differences in the thoroughness with which minor congenital anomalies are recorded in the clinical notes, or the National Minimum Dataset. |
| Congenital Anomalies Evident at Birth | Congenital Anomalies Evident at Birth | • During 2008–2012, a large number of congenital anomalies were identified at birth, with these ranging in severity from minor (e.g. skin tags, non-neoplastic nevus) to anomalies which were incompatible with life (e.g. anencephaly).  
• While the largest absolute numbers of babies with congenital anomalies were born to women aged 30–34 years, congenital anomaly rates rose with increasing maternal age, with the highest rates being seen in the babies of mothers aged 40+ years. The babies of mothers aged 40+ years had congenital anomaly rates that were 1.32 (95% CI 1.20–1.44) times higher than the babies of teenage mothers.  
• The proportion of babies with one or more congenital anomalies identified at birth was significantly higher for males, Asian/Indian and Pacific > European/Other > Māori babies and those from less deprived (NZDep06 deciles 1–2 vs. 5–10) areas. |
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<td>Congenital Heart Disease</td>
<td>During 2008–2012, patent ductus arteriosus (PDA) was the most frequent cardiovascular (CVS) anomaly identified at birth, with 64.2% of PDAs being in preterm babies with no other CVS anomalies. Atrial septal defects (ASDs) and ventricular septal defects (VSDs) were the next most frequent causes.</td>
<td>There were no significant ethnic differences in the proportion of babies born with CVS anomalies. Rates were significantly higher however, for babies from the least deprived (NZDep06 deciles 1–2 vs. deciles 5–10) areas, for males, and for those with older (40+ years vs. &lt;20 years) mothers.</td>
<td>In all four Northern DHBs during 2008–2012, PDA was the most frequent CVS anomaly identified at birth, although over half (range 57.6%–68.9%) were in preterm babies with no other CVS anomalies. ASDs and VSDs were the next most frequent anomalies identified.</td>
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<td>Congenital Anomalies Evident at Birth</td>
<td>During 2000–2012, on average 53 babies per year were identified as having Down syndrome at birth. During 2008–2012, 45.9% of babies with Down syndrome had one or more cardiovascular anomalies, with the most frequent being patent ductus arteriosus and atrial septal defects. There were no significant NZDep06, ethnic or gender differences in the proportion of babies identified with Down syndrome at birth. Rates for the babies of mothers aged 40+ years however were 25.7 (95% CI 9.36–70.45) times higher than for teenage mothers.</td>
<td>In the Northern region during 2008–2012, 10 Northland, 32 Waitemata, 29 Auckland and 44 Counties Manukau babies were identified as having Down syndrome at the time of birth, with a small number of babies also having other chromosomal anomalies. The proportion of babies identified with Down syndrome at birth in all four Northern DHBs was not significantly different from the NZ rate.</td>
<td>In the Northern DHBs (combined) during 2008–2012, a total of 5 cases anencephaly, 9 of encephalocele and 22 of spina bifida were identified at the time of birth, with these accounting for 17.9% of all nervous system anomalies during this period. In the Waitemata, Auckland and Counties Manukau DHBs, rates were not significantly different from the NZ rate, while in Northland, numbers were too small to make any meaningful comparisons.</td>
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<td>Neural Tube Defects (NTDs)</td>
<td>During 2000–2012, on average 13.5 babies per year had one or more NTDs identified at birth. Large year to year variations, possibly due to small numbers made trends difficult to interpret. During 2008–2012, 79 NTDs were identified at birth (spina bifida (n=53), anencephaly (n=13), encephalocele (n=13)), with on average 16 NTDs identified per year. NTDs accounted for 17.8% of all nervous system anomalies during this period. There were no significant ethnic, NZDep06 or gender differences in the proportion of babies born with NTDs. The highest rates however, were seen in Pacific babies, the babies of teenage mothers, and those born into the most deprived (NZDep06 deciles 9–10) areas.</td>
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| Other Disabilities | Permanent Hearing Loss: Deafness Notification Database (DND) | - During 2012, 3% of notifications to the DND were for profound hearing losses, 1% for severe losses, 42% for moderate losses and 54% for mild losses.  
- During 2012, when unilateral, acquired, mild, and overseas born cases were excluded, the average age at confirmation of a hearing loss was 50 months, although the average age of suspicion was much earlier (42 months).  
- During 2010–2012, the largest numbers of notifications to the DND were for babies <1 year. Numbers then dropped away during the preschool years. A second peak was evident at five years of age, likely as a result of the B4 School Check. The peak in notifications in babies <1 year increased during this period (2010 n=23; 2011 n=34; 2012 n=38) possibly as a result of the progressive roll out of newborn hearing screening. | - In Northland during 2012, 15 children were notified to the Deafness Notification Database, while in Waitemata 6 children were notified, in Auckland DHB 7 children were notified and in Counties Manukau 26 children were notified. |
| Other Disabilities | Permanent Hearing Loss: Newborn Hearing Screening | - During 1st October 2011–31st March 2012, the caregivers of 88.6% of eligible babies consented to newborn hearing screening. Of those completing screening, 92.8% did so at <1 month of age, with 1.5% receiving an audiology referral, and 5.1% having risk factors for delayed onset/progressive hearing loss (e.g. family history, craniofacial anomalies, and intrauterine infections) which warranted follow up over time.  
- During the same period, 254 babies commenced an audiology assessment, with 85.9% completing their assessment by 3 months of age. During this period, 30 babies were identified as having a permanent congenital hearing loss, while 73 had a conductive hearing loss. | - In the Northern region during 1st October 2011–31st March 2012, the proportion of caregivers of eligible babies who consented to newborn hearing screening ranged from 71.5%–90.1%. Of those completing screening, the majority (range 67.8%–94.8%) did so <1 month. During this period 4.5% of Northland, 1.1% of Waitemata, 1.7% of Auckland and 1.8% of Counties Manukau babies received an audiology referral, with a further 9.3% of Northland, 4.2% of Waitemata and Auckland and 6.1% of Counties Manukau babies having risk factors for delayed onset/progressive hearing loss.  
- During this period, one Auckland DHB baby was identified as having a permanent congenital hearing loss, while 7 Northland and 12 Auckland DHB babies were identified as having a conductive hearing loss. No information was available however, for the Waitemata DHB. |
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|        | Cerebral Palsy | • During 2008–2012, only 10.4% of acute and arranged hospitalisations in children and young people with cerebral palsy (CP) had CP listed as their primary reason for admission. Instead 17.5% of hospitalisations were for epilepsy or convulsions and 22.3% for respiratory diseases. Acute and arranged admissions (vs. waiting list) made up 48.6% of all admissions in those with CP.  
• 51.4% of admissions in those with CP were from the waiting list, with injections into ligaments, tendons, or soft tissue accounting for 42.2% of waiting list admissions. Orthopaedic procedures collectively were the leading reasons for waiting list admissions, followed by dental procedures.  
• CP admissions increased during infancy, reached a peak at three years, and then declined. In contrast, mortality was more evenly distributed across the age range. During 2006–2010, 78 children and young people had CP listed as their main underlying cause of death, or as a contributory cause.  
• CP admissions were significantly higher for males and for Pacific > European/Other > Māori > Asian/Indian children and young people. | • In the Northern region during 2008–2012, a total of 57 Northland, 231 Waitemata, 156 Auckland and 257 Counties Manukau children and young people were hospitalised with a diagnosis of cerebral palsy. Admission rates per 100,000 in the Waitemata and Counties Manukau DHBs were significantly higher than the NZ rate, while in Northland rates were significantly lower. In Auckland DHB, rates were similar to the NZ rate. |
|        | Autism Spectrum Disorder | • During 2008–2012, autism and other pervasive developmental disorders (APDD) were listed as the primary diagnosis in only 14.3% of hospitalisations for children and young people with APDD in any of the first 15 diagnoses. Of those with APDD listed as the primary diagnosis, 64.0% had childhood autism, 22.8% had Asperger syndrome and 13.3% had other pervasive developmental disorders. Overall, 23.8% of admissions in those with APDD were for dental caries/oral health conditions, and 8.5% were for epilepsy or convulsions  
• APDD admissions increased during the preschool years, reached a peak at eight years, and then declined. During 2006–2010, 5 children or young people had APDD listed as the main underlying cause of death, or as a contributory cause, with all deaths being in those aged >10 years.  
• APDD admissions were significantly higher for males. Admissions were also significantly higher for European/Other > Māori and Asian/Indian > Pacific children and young people. | • In the Northern region during 2008–2012, 38 Northland, 246 Waitemata, 164 Auckland and 187 Counties Manukau children and young people were hospitalised with a diagnosis of autism or other pervasive developmental disorders.  
• Admission rates per 100,000 were significantly lower than the NZ rate in Northland and Counties Manukau but significantly higher in the Waitemata DHB. Rates in Auckland DHB however, were not significantly different from the NZ rate. |
### Chronic Medical Conditions

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| **Eczema and Dermatitis** | • During 2008–2012, only 28.7% of hospitalisations in children with eczema or dermatitis listed their first 15 diagnoses, had eczema or dermatitis listed as the primary reason for admission. Atopic and other dermatitis (12.6%) and infective dermatitis (10.8%) were the most frequent primary diagnoses assigned to those with eczema or dermatitis, while bronchiolitis and asthma and wheeze were the most frequent non-eczema related reasons for admission.  
  • Admissions for infective dermatitis and other forms of eczema and dermatitis were highest in infants <1 year, with rates then tapering off during the preschool years. Admissions were lowest amongst children over five years of age.  
  • Admissions for those with a primary diagnosis of infective eczema, or other eczema and dermatitis were both significantly higher in males than females. Rates were also significantly higher for Māori and Pacific > Asian/Indian > European/Other children. Admissions for both outcomes increased for all ethnic groups during 2000–2012.  | • In the Northern DHBs during 2008–2012, less than a quarter (range 20.4%–24.2%) of children hospitalised with eczema or dermatitis, had eczema or dermatitis listed as the primary reason for their admission. Atopic and other dermatitis was the most frequent primary diagnoses assigned in those with eczema or dermatitis in all four DHBs, followed by infective dermatitis.  
  • During this period, 116 Northern, 242 Waitemata, 223 Auckland and 379 Counties Manukau children were admitted with a primary diagnosis of eczema or dermatitis. Admission rates per 100,000 in the Waitemata and Auckland DHBs were significantly lower than the NZ rate, while in Northland and Counties Manukau, rates were not significantly different from the NZ rate. |
| **Inflammatory Bowel Disease** | • During 2008–2012, 82.0% of acute/arranged hospitalisations in children and young people with Crohn’s disease in any of their first 15 diagnoses, had Crohn’s listed as the primary reason for admission. The remaining 18.0% were for a range of conditions, including anal/rectal abscesses and fistulae and intestinal obstructions. For waiting list admissions, injections or infusions of therapeutic substances (38.4%) and fibreoptic colonoscopies (27.6%) were the most frequent primary procedures listed.  
  • 85.0% of acute and arranged hospitalisations in children and young people with ulcerative colitis (UC) in any of their first 15 diagnoses, had UC listed as their primary reason for admission. Of those admitted from the waiting list, fibreoptic colonoscopies (60.9%) and injections or infusions of therapeutic substances (15.8%) were the most frequent primary procedures listed.  
  • Admissions for Crohn’s were significantly higher for males, although no significant gender differences were evident for UC. Admissions for Crohn’s were also significantly higher for European/Other > Asian/Indian > Māori > Pacific children and young people, while rates for UC were significantly higher for European/Other > Asian/Indian > Māori and Pacific children and young people.  | • In the Northern region during 2008–2012, 24 Northland, 140 Waitemata, 84 Auckland and 98 Counties Manukau children and young people were admitted with a diagnosis of Crohn’s disease. Hospital admissions per 100,000 in the Waitemata DHB were significantly higher than the NZ rate, while in Counties Manukau rates were significantly lower. In the Northland and Auckland DHBs rates were not significantly different from the NZ rate.  
  • Similarly during 2008–2012, 16 Northland, 81 Waitemata, 29 Auckland and 40 Counties Manukau children and young people were admitted with a diagnosis of ulcerative colitis. Hospital admissions per 100,000 in the Waitemata DHB were significantly higher than the NZ rate, while in Counties Manukau rates were significantly lower. In the Northland and Auckland DHBs rates were not significantly different from the NZ rate. |
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<td>Chronic Medical Conditions</td>
<td>Cystic Fibrosis</td>
<td>During 2008–2012, 83.7% of admissions in children and young people with cystic fibrosis (CF) listed in any of their first 15 diagnoses, had CF listed as the primary reason for admission. The remainder were for a variety of infectious and respiratory diseases, digestive system problems and other issues. Of those with CF listed as the primary diagnosis, the majority (92.6%) also had a secondary diagnosis. Of these, unspecified acute lower respiratory infections were the most frequent, followed by a range of infectious (e.g. <em>S. aureus</em>, aspergillosis), respiratory (e.g. bronchiectasis) and other conditions. CF admissions were relatively evenly distributed across the age range, although a small peak was evident during adolescence. Mortality was more common in the late teens and early twenties, with 20 young people having CF listed as the main underlying or a contributory cause of death in 2006–2010. CF admissions were <strong>significantly</strong> higher for females and for European/Other &gt; Māori &gt; Pacific and Asian/Indian children and young people.</td>
<td>In the Northern region during 2008–2012, 8 Northland, 34 Waitemata, 39 Auckland and 27 Counties Manukau children and young people were hospitalised with a diagnosis of cystic fibrosis. Admission rates per 100,000 in the Auckland, Waitemata and Counties Manukau DHBs were <strong>significantly</strong> lower than the NZ rate while in Northland rates were not <strong>significantly</strong> different from the New Zealand rate.</td>
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<td>Type 1 Diabetes</td>
<td>During 2008–2012, 70.0% of hospitalisations for children and young people with type 1 diabetes had a diabetes-related primary diagnosis, with ketoacidosis/lactic acidosis +/- coma accounting for 33.3% and type 1 diabetes without complications for 17.7% of admissions. A further 30.0% were for diagnoses other than diabetes, including gastroenteritis, injuries and poisoning, and pregnancy and childbirth. Admissions increased during childhood, reached a peak at 14 years, and then fluctuated. Mortality was highest amongst those in their late teens and early twenties, with 15 young people having type 1 diabetes listed as the main underlying cause of death, or as a contributory cause, during 2006–2010. Admissions were <strong>significantly</strong> higher for females and for European/Other &gt; Māori &gt; Pacific &gt; Asian/Indian children and young people.</td>
<td>In the Northern region during 2008–2012, around two thirds (range 65.1%–66.7%) of hospital admissions in children and young people with type 1 diabetes listed in any of their first 15 diagnoses, had a diabetes-related primary diagnosis. Ketoacidosis/lactic acidosis +/- coma was the most frequent diabetes-related primary diagnosis in all four DHBs. During this period, 95 Northland, 383 Waitemata, 252 Auckland and 319 Counties Manukau children and young people were hospitalised with a diagnosis of type 1 diabetes. Admission rates per 100,000 in the Northland, Auckland and Counties Manukau DHBs were <strong>significantly</strong> lower than the NZ rate, while rates in the Waitemata DHB were not <strong>significantly</strong> different from the NZ rate.</td>
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<td>Stream</td>
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<td>Chronic Medical Conditions</td>
<td>Epilepsy</td>
<td>During 2008–2012, 75.3% of hospitalisations in children and young people with epilepsy or status epilepticus had an epilepsy-related primary diagnosis. Generalised idiopathic epilepsy (24.4%) and unspecified epilepsy (20.1%) were the most frequent epilepsy-related diagnoses. A further 24.7% were for unrelated conditions, with respiratory conditions and injury and poisoning being the most frequent diagnoses. The secondary diagnoses assigned to those with epilepsy or status epilepticus as a primary diagnosis, fell into two categories: conditions which may have increased the risk of epilepsy (e.g. cerebral palsy, congenital anomalies); and acute concurrent illnesses such as respiratory and viral infections. Admissions were highest during the first four years, with rates declining during childhood, to reach their lowest point at 14 years. Rates then increased slightly, to reach a plateau in those in their late teens–early twenties. Mortality during 2006–2010 occurred across the age range, although rates were generally higher in the early 20s, than in late childhood. Admissions were significantly higher for males and for Māori and Pacific children and young people, than for European/Other and Asian/Indian children and young people.</td>
<td>In the Northern DHBs during 2008–2012, around ¾ (range 66.0%–79.6%) of all hospital admissions in children and young people with epilepsy or status epilepticus had an epilepsy-related diagnosis listed as the primary reason for admission. Unspecified epilepsy and generalised idiopathic epilepsy were the most frequent diagnoses in all four DHBs. During this period, 182 Northland, 523 Waitemata, 330 Auckland and 644 Counties Manukau children and young people were hospitalised with a diagnosis of epilepsy or status epilepticus. Admission rates per 100,000 were significantly higher than the NZ rate in Northland and Counties Manukau and significantly lower in Auckland DHB. In the Waitemata DHB, admission rates were not significantly different from the NZ rate.</td>
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<td>Cancer</td>
<td>During 2002–2011, acute lymphoblastic leukaemia (ALL) was the most frequent malignant neoplasm notified to the NZ Cancer Registry in children and young people aged 0–24 years, followed by malignant melanoma of the skin. Carcinoma in situ of the cervix however, was the most frequent reason for a NZ Cancer Registry notification, accounting for 61.3% of all notifications during this period. During 2001–2010, cancers of the brain were the leading cause of cancer mortality in children and young people aged 0–24 years, followed by acute lymphoblastic leukaemia.</td>
<td>In all four Northern DHBs during 2002–2011, ALL was the most frequent malignant neoplasm notified to the NZ Cancer Registry in children and young people. Other frequently notified malignant neoplasms included non-Hodgkin lymphomas, malignant melanomas, and malignant neoplasms of the testis and brain. Carcinoma in situ of the cervix however, was the most frequent reason for a NZ Cancer Registry notification in all four DHBs. In the Northern DHBs during 2001–2010, cancers of the brain and bone and cartilage and ALL were the leading causes of cancer-related mortality in children and young people.</td>
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<td><strong>Obesity, Nutrition and Physical Activity</strong></td>
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<td>• The proportion of NZ children aged 2–14 years who were obese increased <em>significantly</em> between NZ Health Surveys, with rates rising from 8.4% (95% CI 7.5–9.4) in 2006/07, to 10.3% (95% CI 8.9–11.9) in 2011/12.</td>
<td>• When broken down by region, the proportion of children aged 2–14 years in the Northern region who were obese did not increase <em>significantly</em> between NZ Health Surveys, with rates being 9.5% (95% CI 8.1–11.0) in 2006/07 and 11.2% (95% CI 9.0–13.7) in 2011/12.</td>
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<td>• In the children's component of the 2011/12 NZHS, there were no <em>significant</em> differences in obesity by age. Rates were 9.2% (95% CI 6.5–12.5) in those 2–4 years, 10.6% (8.6–12.9) in those 5–9 years and 10.8% (95% CI 8.7–13.2) in those 10–14 years. In the adult survey, obesity rates were <em>significantly</em> higher for those 18–24 years (22.9% (95% CI 19.6–25.6%)) than for those 15–17 years (12.0% (95% CI 8.0–17.2%)).</td>
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<td>• In the 2011/12 NZHS, there were no <em>significant</em> gender differences in the proportion of children age 2–14 years who were obese, once rates were adjusted for age.</td>
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<td>• Māori children however were 2.10 (95% CI 1.64–2.68) times more likely to be obese than non-Māori children, while Pacific children were 3.08 (95% CI 2.41–3.93) times more likely to be obese than non-Pacific children, once rates were adjusted for age and gender. There were no <em>significant</em> differences, in obesity rates between Asian and non-Asian children.</td>
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<td>Overweight and Obesity</td>
<td>Distribution of Overweight and Obesity: NZ Health Surveys</td>
<td>• Children living in the most deprived (NZDep06 deciles 9–10) areas were 2.33 (95% CI 1.37–3.93) times more likely to be obese than children in the least deprived (NZDep06 deciles 1–2) areas, once adjusted for age, sex an ethnic group.</td>
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<td>• In the Youth’12 survey, 24.1% (95% CI 22.8–25.4) of students were overweight, and 12.6% (95% CI 10.1–15.1) obese, as compared to the Youth’07 survey where 24.2% (95% CI 22.7–25.6) were overweight and 10.4% (95% CI 8.8–11.9) obese.</td>
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<td>• In Youth’12 there were no <em>significant</em> gender, age (by single year of age), or rural/urban differences in the proportion of students that were overweight or obese.</td>
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<td>• However, the proportion of students who were overweight or obese was <em>significantly</em> higher for those from the most deprived (NZDep deciles 8–10) areas, than for those from the least deprived (NZDep06 deciles 1–3) areas.</td>
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### Consequences of Obesity: Type 2 Diabetes

- During 2008–2012, 21.2% of hospitalisations for children and young people with type 2 diabetes had diabetes listed as the primary diagnosis. The remaining 78.8% were for other conditions including pregnancy and childbirth, skin infections and respiratory diseases.
- Hospitalisations were infrequent during childhood, but increased thereafter, with the highest rates being seen in those in their early 20s.
- Rates were also significantly higher for females and for Pacific > Māori > Asian/Indian and European/Other children and young people. Similar ethnic differences were seen during 2000–2012, with rates increasing for Pacific and Māori children and young people during this period.

### Consequences of Obesity: Slipped Upper Femoral Epiphysis (SUFE)

- During 2008–2012, 96.3% of admissions in children and young people 0–24 years with SUFE in any of their first 15 diagnoses had SUFE listed as the primary reason for admission.
- Of the 748 SUFE admissions during 2008–2012, 705 (94.3%) were acute or arranged (<7 days of referral) admissions, while 43 (5.7%) were from the waiting list.
- 95.9% of SUFE admissions also had a primary procedure recorded, with closed reductions of a SUFE (47.6%) and epiphysiodesis of the femur (21.5%) being the most frequently listed primary procedures.
- SUFE admissions were infrequent during early childhood, but increased rapidly after eight years of age. Admissions reached a peak at 11 years in females and 12 years in males, before declining again during the early-mid teens.
- There were no significant gender differences in SUFE rates, although rates were significantly higher for Pacific and Māori > European/Other > Asian/Indian children and young people.

### Northern DHBs Distribution and Trends

- In the Northern region during 2008–2012, 25 Northland, 43 Waitemata, 64 Auckland and 114 Counties Manukau children and young people were hospitalised with a diagnosis of type 2 diabetes.
- Admission rates per 100,000 in the Northland, Auckland and Counties Manukau DHBs were significantly higher than the NZ rate, while rates in the Waitemata DHB were not significantly different from the NZ rate.

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<td>- Rates were also significantly higher for females and for Pacific &gt; Māori &gt; Asian/Indian and</td>
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<td>during the early-mid teens.</td>
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<td>higher for Pacific and Māori &gt; European/Other &gt; Asian/Indian children and young people.</td>
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### Overweight and Obesity

**Consequences of Obesity: Bariatric Surgery**

- During 2008–2012, obesity was the most frequent primary diagnosis in young people aged 15–24 years admitted for bariatric surgery, accounting for 65.9% of admissions. Type 2 diabetes and mechanical complications of gastrointestinal prosthetic devices made a smaller contribution.
- Laparoscopic gastric reductions (41.5%) were the most frequent primary procedure listed in young people admitted for bariatric surgery, followed by gastric bypasses (29.3%).
- Admissions in young people increased from 0.5 admissions per year in 2000–01, to 10 per year during 2010–2012.
- Admissions were infrequent during the early teens, but increased thereafter, with the highest rates being in those in their early twenties.
- While admissions were higher for Pacific > European/Other > Māori young people, these differences did not reach statistical significance. Admission rates however, were significantly higher for females than for males.

- In the Northern DHBs during 2008–2012, there were 23 admissions for bariatric surgery in young people aged 15–24 years (<3 in Northland, 11 in Waitemata, <3 in Auckland DHB and 8 in Counties Manukau).

### Nutrition and Physical Activity

**Breastfeeding and Solids: Breastfeeding in Plunket Babies**

- During the years ending June 2006–2012, the proportion of Plunket babies who were exclusively or fully breastfed remained relatively static. Exclusive/full breastfeeding rates in the year ending June 2012 were 66.1% at <6 weeks, 54.6% at 3 months and 24.9% at 6 months of age.
- Exclusive/full breastfeeding rates at <6 weeks were higher for European babies than for other ethnic groups. At 3 and 6 months, rates were generally higher European > Asian > Māori and Pacific babies, with differences between Asian and European babies decreasing as the period progressed.
- In the year ending June 2012, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were lower for babies from the most deprived (NZDep decile 10) areas, than for babies from average or less deprived areas.
- In Northland and Waitemata during June 2006–2012, exclusive/full breastfeeding rates at <6 weeks and 3 months were higher than the NZ rate, while rates at 6 months were similar. In Counties Manukau breastfeeding rates were much lower than the NZ rate at all three ages. In Auckland DHB, rates at 3 months were higher than the NZ rate, although at <6 weeks rates were more similar.
- In Northland breastfeeding rates at all three ages were higher for European than for Māori babies. In the Waitemata, Auckland and Counties Manukau DHBs, rates at <6 weeks were much higher for European babies than for babies of other ethnic groups. In the Waitemata and Auckland DHBs at 3 and 6 months however, rates were generally higher for European > Asian > Māori and Pacific babies.
- In all four Northern DHBs during the year ending June 2012, breastfeeding rates at all three ages were lower for babies living in the most deprived (NZDep decile 10) areas, than for babies living in the least deprived (NZDep decile 1) areas.
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| Breastfeeding and Solids: Babies Given Solids at <4 Months of Age | • The proportion of children 4 months–4 years given solid food at <4 months of age decreased significantly (p=0.00) between NZ Health Surveys, with rates falling from 15.8% (95% CI 13.7–18.1) in 2006/07, to 9.5% (95% CI 7.9–11.4) in 2011/12.  
• In the 2011/12 NZHS, Māori children were 2.23 (95% CI 1.56–3.19) times more likely to be given solid food <4 months than non-Māori children, while Pacific children were 1.67 (95% CI 1.09–2.56) times more likely to be given solid food <4 months than non-Pacific children, once rates were adjusted for age and gender. In contrast, Asian children were significantly less likely to be given solid food <4 months (aRR 0.28 (95% CI 0.11–0.68)) than non-Asian children. | • When broken down by region, the proportion of children aged 4 months to 4 years in the Northern region given solid food before four months of age decreased significantly between NZ Health Surveys, with rates falling from 14.1% (95% CI 10.9–17.8) in 2006/07, to 7.5% (95% CI 4.9–10.9) in 2011/12. |
| Nutrition and Physical Activity             | • Eating Breakfast at Home: The % of children 2–14 years who ate breakfast at home every day in the last week did not change significantly between NZ Health Surveys. Rates were 87.9% (95% CI 86.6–89.0) in 2006/07 and 87.3% (95% CI 85.7–88.7) in 2011/12.  
• Māori children were significantly less likely than non-Māori children to eat breakfast at home. Rates were also significantly lower for Pacific than non-Pacific children and for children from the most deprived (NZDep06 deciles 9–10 vs. deciles 1–2) areas.  
• Fast Food: The % of children 2–14 years who ate fast food 3+ times in the past week did not change significantly between NZ Health Surveys, with rates being 7.2% (95% CI 6.3–8.2) in 2006/07 and 6.5% (95% CI 5.3–7.9) in 2011/12.  
• Māori children were significantly more likely than non-Māori children to have eaten fast food. Rates were also significantly higher for Pacific than for non-Pacific children. Children from the most deprived areas were also significantly more likely have eaten fast food than children from the least deprived areas.  
• Fizzy Drinks: The % of children 2–14 years who had consumed fizzy drinks 3+ times in the past week did not change significantly between NZ Health Surveys, with rates being 19.6% (95% CI 18.0–21.2) in 2006/07 and 19.6% (95% CI 17.9–21.4) in 2011/12.  
• Māori children were significantly more likely than non-Māori children to have consumed fizzy drinks. Rates were also significantly higher for Pacific than for non-Pacific children, for Asian than for non-Asian children and for children from the most deprived areas. | • Eating Breakfast at Home: When broken down by region, there were no significant changes in the proportion of Northern children who ate breakfast at home every day in the last week, between the 2006/07 and 2011/12 NZ Health Surveys. In the 2011/12 NZHS, 87.7% (95% CI 84.9–90.2) of Northern children ate breakfast at home every day in the last week. |
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<td><strong>Nutrition and Physical Activity</strong></td>
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<td>Other Nutritional Indicators: Youth’12 Survey</td>
<td>• <strong>Breakfast</strong>: In the Youth’12 Survey, 16.7% (95% CI 15.1–18.2) of secondary students said they hardly ever ate breakfast, with the % of females (20.8% (95% CI 18.4–23.3)) being significantly higher than for males (11.7% (95% CI 10.6–12.8)).&lt;br&gt;While there were no age differences, a significantly higher proportion of students from the most deprived (NZDep deciles 8–10) areas said they hardly ever ate breakfast, than students from the least deprived (NZDep06 deciles 1–3) areas.&lt;br&gt;There were no significant urban vs. rural differences in the proportion of students who said they hardly ever ate breakfast.&lt;br&gt;<strong>Fruit and Vegetables</strong>: In Youth’12, 30.0% (95% CI 28.4–31.6) of students said that they ate 2+ fruit and 3+ vegetables per day.&lt;br&gt;There were no significant gender or age differences in the % of students who ate 2+ fruit and 3+ vegetables per day. Rates were also not significantly different between those in the most and least deprived NZDep06 areas, or urban and rural areas.</td>
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<td>Physical Activity: Youth’12 Survey</td>
<td>• <strong>Participation in Physical Activity</strong>: In Youth’12, while 61.9% (95% CI 59.9–64.0) of students had participated in &gt;20 minutes vigorous physical activity on 3+ occasions in the past seven days, only 9.6% (95% CI 8.7–10.5) reported achieving the recommended 60+ minutes of physical activity daily.&lt;br&gt;The proportion of males undertaking &gt;20 minutes vigorous physical activity was significantly higher than for females. Rates were also significantly higher for younger students (≤15 years vs. 16+ years), and for students from less deprived (NZDep06 decile1–3 vs. deciles 8–10) or rural areas.&lt;br&gt;<strong>Travel to School by Active Means</strong>: In Youth’12, 32.7% (95% CI 29.5–35.9) of students usually travelled to school by active means (walk, bike or skate) 6+ times in the past seven days.&lt;br&gt;While there were no significant gender differences in the proportion who usually travelled to school by active means, rates were significantly higher for younger students (≤15 years vs. 17+ years), for students from more deprived (NZDep06 deciles 8–10 vs. decile1–3) or urban areas.&lt;br&gt;<strong>Sedentary Leisure</strong>: In Youth’12, 28.2% (95% CI 25.9–30.4) of students spent 3+ hours each day watching TV, while 19.5% (95% CI 17.4–21.7) spent 3+ hours playing computer games, and 34.8% (95% CI 33.2–36.4) spent 3+ hours on the internet.</td>
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Nutrition and Physical Activity

- **Travel to School by Active Means:** The % of children aged 5–14 years who usually travelled to school by active means did not change significantly (p=0.51) between NZ Health Surveys, with rates being 46.1% (95% CI 43.3–48.8) in 2006/07 and 47.5% (95% CI 44.2–50.7) in 2011/12.

- In the 2011/12 NZHS, once adjusted for age and gender, Māori children were significantly more likely than non-Māori children to travel to school by active means, with rates also being significantly higher for Pacific than for non-Pacific children.

- Once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly more likely to travel to school by active means than children in the least deprived (NZDep06 deciles 1–2) areas.

- **Watching 2+ Hours TV per Day:** In the 2011/12 NZHS, children aged 5–9 years (49.1% (95% CI 45.2–53.1)) were significantly less likely to watch 2+ hours of TV per day than children aged 10–14 years (57.8% (95% CI 54.5–60.9)).

- Once adjusted for age and gender, Māori children were significantly more likely than non-Māori children to watch 2+ hours of TV per day, with rates also being significantly higher for Pacific children than for non-Pacific children.

- Once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly more likely to watch 2+ hours of TV per day than children in the least deprived (NZDep06 deciles 1–2) areas.
PERINATAL CONDITIONS
**Introduction**

The Perinatal and Maternal Mortality Review Committee defines a fetal death as “the death of a baby born at 20 weeks gestation or beyond, or weighing at least 400g if gestation is unknown. Fetal death includes stillbirth and termination of pregnancy” [7].

Internationally, fetal death rates in high-income countries have shown little or no improvement over the past two decades [8]. Maternal overweight and obesity has been identified as one of the leading modifiable risk factors. Other risk factors include advanced maternal age (35+ years), smoking and primiparity [8]. A range of pregnancy-related disorders also increase the risk of a fetal death including fetal growth restriction, placental abruption, and maternal diabetes and hypertension [8].

In New Zealand, research suggests that the risk of fetal death is higher for older women (35+ years), and those in their first or fourth or higher pregnancies. A range of lifestyle and social factors are also associated with an increased risk, including smoking, being overweight or obese, not being married or in paid work, and living in a more deprived (NZDep deciles 9–10) area [9,10,11]. Indian and Pacific women have higher fetal death rates than European women [9,10], although in one recent study, the excess risk for Pacific women disappeared once a number of other risk factors were taken into account [11]. In this same study, unexplained antepartum deaths (39.4%) and fetal growth restriction (18.7%) accounted for almost 60% of fetal deaths, with the post mortem rate being 47% (73 of 155 cases) [11].

The following section reviews the distribution of fetal deaths in the Northern DHBs using information from the National Mortality Collection and the Birth Registration Dataset. The section concludes with a brief review of policy documents and evidence-based reviews which consider how fetal deaths might be prevented at the population level.

### Data Sources and Methods

**Indicator 1. Intermediate Fetal Deaths**

**Numerator:** National Mortality Collection: Fetal deaths occurring between 20 and 27 weeks gestation.

**Denominator:** Birth Registration Dataset: All births 20+ weeks gestation.

**Indicator 2. Late Fetal Deaths**

**Numerator:** National Mortality Collection: Fetal deaths occurring at 28+ weeks gestation.

**Denominator:** Birth Registration Dataset: All births 28+ weeks gestation.

In the National Mortality Collection, all fetal deaths are assigned a main underlying fetal cause of death. In addition other fetal and maternal causes contributing to the death are also listed. In this section, the main underlying fetal cause of death was assigned using the following ICD-10-AM codes: Malnutrition/Slow Fetal Growth (P05), Prematurity/Low Birth Weight (P07), Intrauterine Hypoxia (P20), Congenital Pneumonia (P23), Infections Specific to Perinatal Period (P35–P39), Hydrops Fetalis not due to Haemolytic Disease (P83.2), Aspiration of Meconium/Amniotic Fluid/Mucus (P24.0, P24.1), Polycythaemia Neonatorum (P61.1), Fetal Blood Loss (P50), Unspecified Cause (P95), Congenital Anomalies: Central Nervous System (Q00–Q07), Congenital Anomalies: Cardiovascular System (Q20–Q28), Chromosomal Anomalies (Q90–Q99), Congenital Anomalies: Other (remainder Q08–Q89), Other Causes (remainder ICD-10-AM).

For gestational age specific rates, the denominator was those remaining in utero at the specified gestational age (e.g. the 22 week denominator excludes births occurring at 20 and 21 weeks).

**Notes on Interpretation**

Note 1: Death Registration data do not differentiate between spontaneous fetal deaths and late terminations of pregnancy, as all fetal deaths 20+ weeks gestation require a death registration. The admixture of spontaneous and induced fetal deaths is likely to be most prominent at earlier gestations (e.g. the high number of deaths attributed to congenital anomalies prior to 25 weeks gestation) and this must be taken into account when interpreting the data in this section.
New Zealand Distribution and Trends

New Zealand Trends

In New Zealand, intermediate fetal deaths increased during the early 2000s, but became relatively static after 2004–05, while late fetal deaths were relatively static throughout 2000–2010 (Figure 1).

Figure 1. Intermediate and Late Fetal Deaths, New Zealand 2000–2010

Distribution by Cause

Intermediate Fetal Deaths: In New Zealand during 2006–2010, unspecified cause was the most frequently listed main fetal cause of death for babies dying in utero between 20 and 27 weeks of gestation, followed by prematurity/low birth weight and congenital and chromosomal anomalies (Table 2).

Late Fetal Deaths: During 2006–2010, unspecified cause was also the most frequently listed main fetal cause of death for babies dying in utero at 28+ weeks gestation, followed by malnutrition/slow fetal growth and intrauterine hypoxia. Congenital anomalies as a group, however, still made a significant contribution (Table 2).

Distribution by Gestational Age and Cause

In New Zealand during 2006–2010, fetal deaths exhibited a J-shaped distribution with gestational age, with a peak occurring prior to 25 weeks, and then rates increasing rapidly again after 37 weeks. When broken down by cause, fetal deaths arising from congenital anomalies and prematurity/low birth weight were highest in babies less than 25 weeks gestation, while unspecified fetal deaths increased rapidly after 37 weeks. Note: These rates were calculated by dividing the number of fetal deaths at each gestational age by the number of babies remaining in utero. Thus, while the absolute number of babies dying in utero did not rise exponentially towards term, the risk for those remaining in utero did. Further, it was not always possible to distinguish between spontaneous fetal deaths and late terminations of pregnancy and thus the high mortality rates (e.g. from congenital anomalies) prior to 25 weeks must be interpreted with this in mind (Figure 2).
Table 2. Intermediate and Late Fetal Deaths by Main Fetal Cause of Death, New Zealand 2006–2010

<table>
<thead>
<tr>
<th>Main Fetal Cause of Death</th>
<th>No. of Deaths: Total 2006–2010</th>
<th>No. of Deaths: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>% of Fetal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intermediate Fetal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unspecified Cause</td>
<td>318</td>
<td>63.6</td>
<td>99.17</td>
<td>24.5</td>
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<tr>
<td>Prematurity/Low Birth Weight</td>
<td>188</td>
<td>37.6</td>
<td>58.63</td>
<td>14.5</td>
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<tr>
<td>Chromosomal Anomalies</td>
<td>165</td>
<td>33.0</td>
<td>51.46</td>
<td>12.7</td>
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<tr>
<td>Congenital Anomalies: CNS</td>
<td>123</td>
<td>24.6</td>
<td>38.36</td>
<td>9.5</td>
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<tr>
<td>Congenital Anomalies: CVS</td>
<td>91</td>
<td>18.2</td>
<td>28.38</td>
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<td>135</td>
<td>27.0</td>
<td>42.10</td>
<td>10.4</td>
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<tr>
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<td>95</td>
<td>19.0</td>
<td>29.63</td>
<td>7.3</td>
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<td>6.8</td>
<td>10.60</td>
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<tr>
<td>Infections Specific to Perinatal Period</td>
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<tr>
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<tr>
<td>Hydrops Fetalis (not Haemolytic Disease)</td>
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<td>5.93</td>
<td>1.5</td>
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<tr>
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<td></td>
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<td>32.07</td>
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<td>9.6</td>
<td>15.09</td>
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<tr>
<td>Fetal Blood Loss</td>
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<td>11.64</td>
<td>3.5</td>
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<tr>
<td>Infections Specific to Perinatal Period</td>
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<td>5.4</td>
<td>8.49</td>
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<td>Hydrops Fetalis (not Haemolytic Disease)</td>
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<td>1.89</td>
<td>0.6</td>
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<td>Polycythaemia Neonatorum</td>
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<td>0.94</td>
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<td>335.83</td>
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Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset
Figure 2. Fetal Deaths by Gestational Age and Main Fetal Cause of Death, New Zealand 2006–2010

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset

Figure 3. Intermediate and Late Fetal Deaths by Ethnicity, New Zealand 2000–2010

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset; Note: Ethnicity is Level 1 Prioritised
Distribution by Ethnicity, NZDep Decile, Maternal Age and Gender

Intermediate Fetal Deaths: In New Zealand during 2006–2010, there were no significant gender, ethnic or socioeconomic (as measured by NZDep06) differences in intermediate fetal death rates. Mortality however, was significantly higher for the babies of younger (<25 years) and older (35+ years) women, than for the babies of women aged 30–34 years (Table 3). During 2000–2010, intermediate fetal death rates were consistently higher for Asian/Indian babies than for European/Other babies, although rates for Māori and Pacific babies were more variable (Figure 3).

Late Fetal Deaths: In New Zealand during 2006–2010, late fetal deaths were significantly higher for Pacific > Māori > European/Other babies, and for babies from average to more deprived (NZDep deciles 5–10) areas. Rates were also significantly higher for the babies of teenage women, than for the babies of women aged 30–34 years (Table 3). During 2000–2010, late fetal death rates were consistently higher for Pacific babies than for babies from other ethnic groups (Figure 3).

Table 3. Intermediate and Late Fetal Deaths by Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand 2006–2010

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
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<td></td>
<td><strong>Intermediate Fetal Deaths</strong></td>
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<td>Prioritised Ethnicity</td>
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<td></td>
<td></td>
<td>Gender</td>
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</tr>
<tr>
<td>Asian/Indian</td>
<td>448.2</td>
<td>1.11</td>
<td>0.93–1.32</td>
<td>Female</td>
<td>394.6</td>
<td>1.00</td>
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<tr>
<td>European/Other</td>
<td>405.1</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>407.7</td>
<td>1.03</td>
<td>0.93–1.15</td>
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<tr>
<td>Māori</td>
<td>374.3</td>
<td>0.92</td>
<td>0.81–1.05</td>
<td>Maternal Age</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Pacific</td>
<td>425.2</td>
<td>1.05</td>
<td>0.88–1.25</td>
<td>&lt;20 Years</td>
<td>442.9</td>
<td>1.25</td>
<td>1.00–1.55</td>
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<td>NZ Deprivation Index</td>
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<td></td>
<td></td>
<td>20–24 Years</td>
<td>422.3</td>
<td>1.19</td>
<td>1.01–1.41</td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>373.2</td>
<td>1.00</td>
<td></td>
<td>25–29 Years</td>
<td>380.9</td>
<td>1.07</td>
<td>0.92–1.26</td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>447.0</td>
<td>1.20</td>
<td>0.99–1.46</td>
<td>30–34 Years</td>
<td>354.8</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>427.3</td>
<td>1.14</td>
<td>0.95–1.39</td>
<td>35+ Years</td>
<td>464.2</td>
<td>1.31</td>
<td>1.12–1.53</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>402.9</td>
<td>1.08</td>
<td>0.90–1.30</td>
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</tr>
<tr>
<td>Deciles 9–10</td>
<td>384.0</td>
<td>1.03</td>
<td>0.86–1.23</td>
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<tr>
<td><strong>Late Fetal Deaths</strong></td>
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<td></td>
<td></td>
<td><strong>Late Fetal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>326.0</td>
<td>1.14</td>
<td>0.92–1.41</td>
<td>Female</td>
<td>347.3</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
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<td>1.00</td>
<td></td>
<td>Male</td>
<td>323.7</td>
<td>0.93</td>
<td>0.83–1.05</td>
</tr>
<tr>
<td>Māori</td>
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<td>1.25</td>
<td>1.09–1.44</td>
<td>Maternal Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>503.4</td>
<td>1.76</td>
<td>1.48–2.10</td>
<td>&lt;20 Years</td>
<td>422.5</td>
<td>1.36</td>
<td>1.09–1.71</td>
</tr>
<tr>
<td>NZ Deprivation Index</td>
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<td></td>
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<td>20–24 Years</td>
<td>349.3</td>
<td>1.13</td>
<td>0.94–1.35</td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>224.7</td>
<td>1.00</td>
<td></td>
<td>25–29 Years</td>
<td>305.0</td>
<td>0.99</td>
<td>0.83–1.17</td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>263.8</td>
<td>1.17</td>
<td>0.91–1.51</td>
<td>30–34 Years</td>
<td>309.6</td>
<td>1.00</td>
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</tr>
<tr>
<td>Deciles 5–6</td>
<td>334.8</td>
<td>1.49</td>
<td>1.18–1.88</td>
<td>35+ Years</td>
<td>362.0</td>
<td>1.17</td>
<td>0.99–1.39</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>327.8</td>
<td>1.46</td>
<td>1.16–1.83</td>
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</tr>
<tr>
<td>Deciles 9–10</td>
<td>448.0</td>
<td>1.99</td>
<td>1.61–2.47</td>
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<td></td>
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</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset; Note: Baby’s ethnicity is Level 1 Prioritised; Decile is NZDep2006
Northern DHBs Distribution and Trends

**Northern DHBs Distribution by Cause**

*Intermediate Fetal Deaths:* In all four Northern DHBs during 2006–2010, congenital and chromosomal anomalies collectively were the most frequently listed main fetal causes of death in babies dying in utero between 20 and 27 weeks of gestation, followed by unspecified causes and prematurity/low birth weight (Table 4, Table 5).

Table 4. Intermediate Fetal Deaths by Main Fetal Cause of Death, Northland and Waitemata DHBs 2006–2010

<table>
<thead>
<tr>
<th>Main Fetal Cause of Death</th>
<th>No. of Deaths: Total 2006–2010</th>
<th>No. of Deaths: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>% of Intermediate Fetal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intermediate Fetal Deaths</strong> Northland</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>10</td>
<td>2.0</td>
<td>84.82</td>
<td>23.8</td>
</tr>
<tr>
<td>Prematurity/Low Birth Weight</td>
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<td>2.2</td>
<td>93.30</td>
<td>26.2</td>
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<tr>
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<tr>
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<td>Congenital Anomalies: CVS/Other</td>
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<td>59.37</td>
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<td>356.23</td>
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<td><strong>Waitemata</strong></td>
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Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset
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<th>% of Intermediate Fetal Deaths</th>
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<td>3.6</td>
<td>54.13</td>
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<td>27.06</td>
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<td>1.6</td>
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<td>3.0</td>
<td>45.10</td>
<td>10.6</td>
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<tr>
<td>Malnutrition/Slow Fetal Growth</td>
<td>14</td>
<td>2.8</td>
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</tr>
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<td>2.1</td>
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<tr>
<td>Hydrops fetalis not due to Haemolytic Disease</td>
<td>5</td>
<td>1.0</td>
<td>15.03</td>
<td>3.5</td>
</tr>
<tr>
<td>Other Causes</td>
<td>13</td>
<td>2.6</td>
<td>39.09</td>
<td>9.2</td>
</tr>
<tr>
<td>Auckland DHB Total</td>
<td>142</td>
<td>28.4</td>
<td>426.99</td>
<td>100.0</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unspecified Cause</td>
<td>43</td>
<td>8.6</td>
<td>97.57</td>
<td>23.9</td>
</tr>
<tr>
<td>Prematurity/Low Birth Weight</td>
<td>42</td>
<td>8.4</td>
<td>95.31</td>
<td>23.3</td>
</tr>
<tr>
<td>Chromosomal Anomalies</td>
<td>15</td>
<td>3.0</td>
<td>34.04</td>
<td>8.3</td>
</tr>
<tr>
<td>Congenital Anomalies: CNS</td>
<td>15</td>
<td>3.0</td>
<td>34.04</td>
<td>8.3</td>
</tr>
<tr>
<td>Congenital Anomalies: CVS</td>
<td>11</td>
<td>2.2</td>
<td>24.96</td>
<td>6.1</td>
</tr>
<tr>
<td>Congenital Anomalies: Other</td>
<td>15</td>
<td>3.0</td>
<td>34.04</td>
<td>8.3</td>
</tr>
<tr>
<td>Malnutrition/Slow Fetal Growth</td>
<td>18</td>
<td>3.6</td>
<td>40.85</td>
<td>10.0</td>
</tr>
<tr>
<td>Congenital Pneumonia</td>
<td>6</td>
<td>1.2</td>
<td>13.62</td>
<td>3.3</td>
</tr>
<tr>
<td>Hydrops fetalis not due to Haemolytic Disease</td>
<td>5</td>
<td>1.0</td>
<td>11.35</td>
<td>2.8</td>
</tr>
<tr>
<td>Infections Specific to Perinatal Period</td>
<td>4</td>
<td>0.8</td>
<td>9.08</td>
<td>2.2</td>
</tr>
<tr>
<td>Other Causes</td>
<td>6</td>
<td>1.2</td>
<td>13.62</td>
<td>3.3</td>
</tr>
<tr>
<td>Counties Manukau Total</td>
<td>180</td>
<td>36.0</td>
<td>408.48</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset

Late Fetal Deaths: In all four Northern DHBs during 2006–2010, unspecified cause was the most frequently listed fetal cause of death in babies dying at 28+ weeks gestation, although malnutrition/slow fetal growth, intrauterine hypoxia and congenital and chromosomal anomalies also made a contribution (Table 6).
Table 6. Late Fetal Deaths by Main Fetal Cause of Death, Northern DHBs 2006–2010

<table>
<thead>
<tr>
<th>Main Fetal Cause of Death</th>
<th>No. of Deaths: Total 2006–2010</th>
<th>No. of Deaths: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>% of Late Fetal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Late Fetal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unspecified Cause</td>
<td>29</td>
<td>5.8</td>
<td>247.82</td>
<td>60.4</td>
</tr>
<tr>
<td>Congenital/Chromosomal Anomalies</td>
<td>7</td>
<td>1.4</td>
<td>59.82</td>
<td>14.6</td>
</tr>
<tr>
<td>Infections Specific to Perinatal Period</td>
<td>3</td>
<td>0.6</td>
<td>25.64</td>
<td>6.3</td>
</tr>
<tr>
<td>Other Causes</td>
<td>9</td>
<td>1.8</td>
<td>76.91</td>
<td>18.8</td>
</tr>
<tr>
<td>Northland Total</td>
<td>48</td>
<td>9.6</td>
<td>410.19</td>
<td>100.0</td>
</tr>
<tr>
<td>Waitemata</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unspecified Cause</td>
<td>71</td>
<td>14.2</td>
<td>182.72</td>
<td>55.5</td>
</tr>
<tr>
<td>Malnutrition/Slow Fetal Growth</td>
<td>14</td>
<td>2.8</td>
<td>36.03</td>
<td>10.9</td>
</tr>
<tr>
<td>Intrauterine Hypoxia</td>
<td>8</td>
<td>1.6</td>
<td>20.59</td>
<td>6.3</td>
</tr>
<tr>
<td>Chromosomal Anomalies</td>
<td>5</td>
<td>1.0</td>
<td>12.87</td>
<td>3.9</td>
</tr>
<tr>
<td>Congenital Anomalies: CNS</td>
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<td>2.0</td>
<td>25.74</td>
<td>7.8</td>
</tr>
<tr>
<td>Congenital Anomalies: CVS/Other</td>
<td>6</td>
<td>1.2</td>
<td>15.44</td>
<td>4.7</td>
</tr>
<tr>
<td>Aspiration Meconium/Amniotic Fluid/Mucus</td>
<td>3</td>
<td>0.6</td>
<td>7.72</td>
<td>2.3</td>
</tr>
<tr>
<td>Fetal Blood Loss</td>
<td>3</td>
<td>0.6</td>
<td>7.72</td>
<td>2.3</td>
</tr>
<tr>
<td>Infections Specific to Perinatal Period</td>
<td>3</td>
<td>0.6</td>
<td>7.72</td>
<td>2.3</td>
</tr>
<tr>
<td>Other Causes</td>
<td>5</td>
<td>1.0</td>
<td>12.87</td>
<td>3.9</td>
</tr>
<tr>
<td>Waitemata Total</td>
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<td>25.6</td>
<td>329.42</td>
<td>100.0</td>
</tr>
<tr>
<td>Auckland DHB</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Unspecified Cause</td>
<td>35</td>
<td>7.0</td>
<td>106.19</td>
<td>34.7</td>
</tr>
<tr>
<td>Malnutrition/Slow Fetal Growth</td>
<td>16</td>
<td>3.2</td>
<td>48.54</td>
<td>15.8</td>
</tr>
<tr>
<td>Intrauterine Hypoxia</td>
<td>8</td>
<td>1.6</td>
<td>24.27</td>
<td>7.9</td>
</tr>
<tr>
<td>Aspiration Meconium/Amniotic Fluid/Mucus</td>
<td>6</td>
<td>1.2</td>
<td>18.20</td>
<td>5.9</td>
</tr>
<tr>
<td>Chromosomal Anomalies</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Congenital Anomalies: CNS</td>
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<td>0.8</td>
<td>12.14</td>
<td>4.0</td>
</tr>
<tr>
<td>Congenital Anomalies: CVS/Other</td>
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<td>2.4</td>
<td>36.41</td>
<td>11.9</td>
</tr>
<tr>
<td>Fetal Blood Loss</td>
<td>4</td>
<td>0.8</td>
<td>12.14</td>
<td>4.0</td>
</tr>
<tr>
<td>Infections Specific to Perinatal Period</td>
<td>6</td>
<td>1.2</td>
<td>18.20</td>
<td>5.9</td>
</tr>
<tr>
<td>Other Causes</td>
<td>8</td>
<td>1.6</td>
<td>24.27</td>
<td>7.9</td>
</tr>
<tr>
<td>Auckland DHB Total</td>
<td>101</td>
<td>20.2</td>
<td>306.43</td>
<td>100.0</td>
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<tr>
<td>Counties Manukau</td>
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<tr>
<td>Unspecified Cause</td>
<td>108</td>
<td>21.6</td>
<td>247.29</td>
<td>54.3</td>
</tr>
<tr>
<td>Malnutrition/Slow Fetal Growth</td>
<td>18</td>
<td>3.6</td>
<td>41.22</td>
<td>9.0</td>
</tr>
<tr>
<td>Intrauterine Hypoxia</td>
<td>22</td>
<td>4.4</td>
<td>50.37</td>
<td>11.1</td>
</tr>
<tr>
<td>Fetal Blood Loss</td>
<td>6</td>
<td>1.2</td>
<td>13.74</td>
<td>3.0</td>
</tr>
<tr>
<td>Infections Specific to Perinatal Period</td>
<td>6</td>
<td>1.2</td>
<td>13.74</td>
<td>3.0</td>
</tr>
<tr>
<td>Chromosomal Anomalies</td>
<td>5</td>
<td>1.0</td>
<td>11.45</td>
<td>2.5</td>
</tr>
<tr>
<td>Congenital Anomalies: CNS/CVS</td>
<td>4</td>
<td>0.8</td>
<td>9.16</td>
<td>2.0</td>
</tr>
<tr>
<td>Congenital Anomalies: Other</td>
<td>5</td>
<td>1.0</td>
<td>11.45</td>
<td>2.5</td>
</tr>
<tr>
<td>Aspiration Meconium/Amniotic Fluid/Mucus</td>
<td>3</td>
<td>0.6</td>
<td>6.87</td>
<td>1.5</td>
</tr>
<tr>
<td>Other Causes</td>
<td>22</td>
<td>4.4</td>
<td>50.37</td>
<td>11.1</td>
</tr>
<tr>
<td>Counties Manukau Total</td>
<td>199</td>
<td>39.8</td>
<td>455.66</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset; Note: s: suppressed due to small numbers
Northern DHBs vs. New Zealand

In all four Northern DHBs during 2006–2010, intermediate fetal death rates were not significantly different from the New Zealand rate. While late fetal death rates in the Northland, Waitemata and Auckland DHBs were also not significantly different from the New Zealand rate, in Counties Manukau rates were significantly higher (RR 1.36 95% CI 1.17–1.58) (Table 7).

Table 7. Intermediate and Late Fetal Deaths, Northern DHBs vs. New Zealand 2006–2010

<table>
<thead>
<tr>
<th>DHB</th>
<th>Number of Deaths: Total 2006–2010</th>
<th>Number of Deaths: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermediate Fetal Deaths</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>42</td>
<td>8.4</td>
<td>356.2</td>
<td>0.88</td>
<td>0.65–1.20</td>
</tr>
<tr>
<td>Waitemata</td>
<td>183</td>
<td>36.6</td>
<td>467.1</td>
<td>1.16</td>
<td>0.99–1.35</td>
</tr>
<tr>
<td>Auckland</td>
<td>142</td>
<td>28.4</td>
<td>427.0</td>
<td>1.06</td>
<td>0.89–1.26</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>180</td>
<td>36.0</td>
<td>408.5</td>
<td>1.01</td>
<td>0.87–1.18</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1,296</td>
<td>259.2</td>
<td>404.2</td>
<td>1.00</td>
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</tr>
<tr>
<td>Late Fetal Deaths</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>48</td>
<td>9.6</td>
<td>410.2</td>
<td>1.22</td>
<td>0.92–1.63</td>
</tr>
<tr>
<td>Waitemata</td>
<td>128</td>
<td>25.6</td>
<td>329.4</td>
<td>0.98</td>
<td>0.82–1.18</td>
</tr>
<tr>
<td>Auckland</td>
<td>101</td>
<td>20.2</td>
<td>306.4</td>
<td>0.91</td>
<td>0.74–1.12</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>199</td>
<td>39.8</td>
<td>455.7</td>
<td>1.36</td>
<td>1.17–1.58</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1,068</td>
<td>213.6</td>
<td>335.8</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset

Northern DHBs Trends

In all four Northern DHBs during 2000–2010, large year-to-year variations, possibly as the result of small numbers, made trends in intermediate and late fetal deaths difficult to interpret (Figure 4).
Figure 4. Intermediate and Late Fetal Deaths, Northern DHBs vs. New Zealand 2000–2010

Source: Numerator: National Mortality Collection; Denominator: Birth Registration Dataset
Local Policy Documents and Evidence-Based Reviews Relevant to Fetal Deaths

In New Zealand at present, there is no single strategy which focuses on the prevention of fetal deaths. Thus any local strategies developed will need to incorporate evidence from a variety of sources. Table 8 provides an overview of a range of New Zealand policy documents and overseas evidence-based reviews which may be useful in this context.

Table 8. Local Policy Documents and Evidence-Based Reviews Relevant to Fetal Deaths

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>These standards provide guidance for the provision of safe, equitable and high quality maternity services throughout New Zealand. They consist of three high level strategic statements to guide the funding, planning, provision and monitoring of maternity services by the Ministry of Health, DHBs, service providers and health practitioners. The standards underpin the DHB maternity service specifications, the Primary Maternity Services Notice 2007, the Maternal Referral Guidelines, and other high-level guidelines and requirements.</td>
</tr>
</tbody>
</table>

| These guidelines are intended for lead maternity carers and outline criteria and processes for referral to primary care, referral for specialist consultation, and referral for the transfer of clinical responsibility for care, transfer of clinical responsibility for care in an emergency and emergency transport. |

<table>
<thead>
<tr>
<th>International and Australasian Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>The purpose of this guideline is to provide advice to clinicians, based on the best available evidence, on the management of women presenting with reduced fetal movements in pregnancy (excluding those with multiple pregnancy). The guidelines are structured as a series of clinical questions. The authors note that the available evidence is limited and that this is reflected in the low grading of some of the recommendations. Appendix 1 provides a care algorithm (flowchart) and Appendix 2 explains the grading scheme used for the evidence and recommendations. There is a comprehensive list of references.</td>
</tr>
</tbody>
</table>

| These guidelines are intended for health professionals in Queensland maternity services and they are consistent with the Perinatal Society of Australia and New Zealand (PSANZ) Clinical Practice guideline for Perinatal Mortality. They cover clinical standards, diagnosis and birth, investigations, autopsy and subsequent pregnancy care. They are concise and well referenced but do not discuss the research evidence. |

| The purpose of this guideline is to assist clinicians provide evidence-based best-practice management for women with singleton pregnancies who report, or are concerned about, decreased fetal movements (DFM) in the third trimester of pregnancy. It does not deal with the management of specific pregnancy conditions such as fetal growth restriction, hypertension or diabetes which may be identified in the course of care. Mothers are often concerned about DFM and there is good evidence that maternal perception of DFM is associated with many adverse outcomes. Fetal growth restriction appears to be a major contributor to these. While women should be made aware of the importance of fetal movement and provided with information, routine fetal movement counting is not recommended. The guidelines discuss the evidence and recommendations are each accompanied by an indication of the evidence level and the strength of the recommendation although the authors note that there is an absence of robust research in this area and more high quality research is needed on both screening tools and management. |
The purpose of this guideline, which is primarily for obstetricians and midwives, is to identify evidence-based options for women (and their families) who have a late intra-uterine death (after 24 weeks) and to provide guidance on general care before, during and after birth, and care in subsequent pregnancies. The levels of evidence and the grades of recommendations in this guideline follow the system used by the Scottish Intercollegiate Guidelines Network (SIGN). They cover diagnosis, investigations, labour and birth, the puerperium, psychological and social aspects of care, follow-up, pregnancy following unexplained stillbirth, clinical governance and recommendations for further research.


The purpose of this guideline is to assist clinicians in the audit of perinatal deaths, to enable a systematic approach to perinatal audit in Australia and New Zealand, and also to provide guidance on dealing with the psychological and social aspects of perinatal bereavement, perinatal post-mortem examination, investigation of stillbirths and neonatal deaths and the use of perinatal mortality classifications.


This very comprehensive 300+ page guideline, which is complementary to the NICE guideline ‘Antenatal care: routine care for the healthy pregnant woman’ (NICE clinical guideline 62), applies to pregnant women with complex social factors, in particular:
- women who misuse substances (alcohol and/or drugs)
- women who are recent migrants, asylum seekers or refugees, or who have difficulty speaking English,
- young women aged under 20
- women who experience domestic abuse

It is intended for health professionals caring for pregnant women, those responsible for commissioning and planning health services and it may be of relevance to those working in social services and education. It is based on, and reports on, systematic reviews of the literature aiming to determine which interventions lead to improved pregnancy outcomes.

Also available through national Institute for Health and Clinical Excellence is a “NICE Pathway” that provides an overview for pregnancy and complex social factors: http://pathways.nice.org.uk/pathways/pregnancy-and-complex-social-factors.


Obese women who become pregnant are at increased risk of complications during pregnancy and childbirth and babies born to obese women face higher risks of a number of adverse outcomes: fetal death, stillbirth, congenital abnormality, shoulder dystocia, macrosomia (large body size) and subsequent obesity. Pregnant women are not encouraged to diet but they can be encouraged to take regular exercise and not to “eat for two”. This guideline on dietary and physical activity interventions for weight management before, during and after pregnancy are intended for NHS and other commissioners, health service managers and health professionals. The evidence reviews on which the guideline was based, and some other relevant background publications can be found at: http://guidance.nice.org.uk/PH27


The purpose of this guideline is to assist clinicians in identifying pregnant women who smoke and assisting them to quit. Smoking cessation interventions for pregnant women can reduce smoking rates and reduce pre term births and low birth weights. Smoking rates are particularly high among teenage and indigenous Australians. The guideline is based on the “5As” approach to smoking cessation (Ask, Advise, Assess, Assist, Arrange Support). For women not ready to quit, motivation interventions using the 5R’s framework (relevance, risk, rewards, roadblocks and repetition) may be used to improve motivation to quit. Recommendations in the guidelines are accompanied by a grade indicating the level of evidence and by references.

Smoking during pregnancy increases the risk of the mother having complications during pregnancy and the baby being born preterm (before 37 weeks). This updated systematic review identifies that psychosocial interventions to support women to stop smoking in pregnancy can increase the proportion of women who stop smoking in late pregnancy and reduce low birthweight and preterm births. Eighty-six trials were included in the review with 79 of them involving 29,000 women providing data on smoking abstinence in late pregnancy. Most were conducted in high income countries. Detailed results are provided in the review, with the key findings being that interventions that provided an incentive to stop smoking appeared to support the most women to quit (one study; RR 3.64, 95% CI 1.84–7.23) and an alternative intervention (one study; RR 4.05, 95% CI 1.48–11.11). In studies comparing counselling and usual care (the largest comparison), it was unclear whether interventions prevented smoking relapse among women who had stopped smoking spontaneously in early pregnancy (eight studies; average RR 1.06, 95% CI 0.93–1.21). However, a clear effect was seen in smoking abstinence at zero to five months postpartum (10 studies; average RR 1.76, 95% CI 1.05–2.95), a borderline effect at six to 11 months (six studies; average RR 1.33, 95% CI 1.00 to1.77), and a significant effect at 12 to 17 months (two studies, average RR 2.20, 95% CI 1.23–3.96), but not in the longer term. Feedback interventions had a significant effect only when compared with usual care and provided in conjunction with other strategies, such as counselling (two studies; average RR 4.39, 95% CI 1.89–10.21), but the effect was unclear when compared with a less intensive intervention (two studies; average RR 1.19, 95% CI 0.45–3.12). Peer provided social support appeared effective (five studies; average RR 1.49, 95% CI 1.01–2.19), but the effect of partner support was not clear (one study).


Cardiotocography (CTG, electronic fetal monitoring) records changes in the fetal heart rate in relation to uterine contractions. It is used to identify babies who may be hypoxic so that additional methods of assessment of fetal wellbeing (e.g. blood sampling) can be used or delivery expedited by instrumental methods (with vacuum extraction or forceps) or caesarean section. This review included 13 RCTs or quasi-RCTs (37,000+ women in total), only two of which were of high quality. The authors concluded that continuous CTG during labour is associated with a reduction in neonatal seizures but no significant differences in cerebral palsy, infant mortality, or other standard measures of infant wellbeing. Continuous CTG monitoring was, however, associated with increases in caesarean section (RR 1.63, 95% CI 1.29 to 2.07, 11 trials, n=18,861) and instrumental vaginal births (RR 1.15, 95% CI 1.01 to 1.33, 10 trials n=18,615).


In the developed world it is widely accepted that a perinatal death is devastating for the parents and family. This review assessed the effects of the provision of counselling or any form of medical, nursing, social or psychological support, or both, for mothers, fathers and families after perinatal death. There were no RCTs identified and the review authors state that more research is needed to determine what kinds of support and counselling are most helpful. However, some well-designed descriptive studies have shown that, under the right circumstances and guided by compassionate, sensitive, experienced staff, parents’ experiences of seeing and holding their deceased baby is often very positive. The sensitive nature of this topic and small sample sizes make it difficult to develop rigorous clinical trials. Hence, other research designs may further inform practice in this area. Where justified, methodologically rigorous trials are needed. However, methodologically rigorous trials should be considered comparing different approaches to support.


Women who have a multiple pregnancy are at greater risk of a number of adverse outcomes, including prematurity (the greatest risk), hypertension, gestational diabetes, and stillbirth. This review assessed the benefits and harms of “specialised” antenatal clinics compared to standard antenatal care, for women with multiple pregnancy. Only one study provided data, and this was on perinatal mortality with no statistically significant differences identified between specialised antenatal care and standard care (RR 1.02; 95% CI 0.26 to 4.03). Women receiving specialised care were significantly more likely to have a caesarean section (RR 1.38; 95% CI 1.06 to 1.81).

---

**Systematic and Other Reviews from the International Literature**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Title</th>
<th>Publication Details</th>
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<tbody>
<tr>
<td>Alfirevic Z, Devane D, Gyte GML. 2013.</td>
<td><em>Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour</em></td>
<td>Cochrane Database of Systematic Reviews Issue 5. Art. No.: CD006066. DOI:10.1002/14651858.CD006066.pub2</td>
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</tbody>
</table>

Pharmacotherapies (nicotine replacement therapy (NRT), bupropion and varenicline) are effective treatments for smoking cessation among the non-pregnant population, but the efficacy and safety of these therapies are not known for smokers who are pregnant. This review included six trials of NRT (n = 1745 pregnant smokers). No statistically significant difference was seen for smoking cessation in later pregnancy after using NRT as compared to control (RR 1.33 95% CI 0.93 to 1.91, six studies, 1745 women). Subgroup analysis comparing placebo-RCTs with those which did not use placebos found that efficacy estimates for cessation varied with trial design (placebo RCTs, RR 1.20, 95% CI 0.93 to 1.56, four studies, 1524 women; non-placebo RCTs, RR 7.81, 95% CI 1.51 to 40.35, two studies, 221 women). There were no statistically significant differences in rates of miscarriage, stillbirth, premature birth, birthweight, low birthweight, admissions to neonatal intensive care or neonatal death between NRT or control groups. However, further research evidence is required for determining efficacy and safety as there was insufficient evidence to determine whether NRT was effective or safe for promoting smoking cessation, or what effect NRT had on birth outcomes.


There are wide variations in the policies and protocols for fetal surveillance in pregnancies where fetal growth impairment is suspected and there are many different techniques used for assessment of fetal growth and wellbeing. This review reports on one RCT done in New Zealand (167 women and babies) which compared a twice-weekly surveillance regimen (biophysical profile, non-stress tests, umbilical artery and middle cerebral artery Doppler and uterine artery Doppler) with the same regimen applied fortnightly (both groups had fetal growth assessed fortnightly). There was no difference between the groups in the primary maternal outcome (emergency caesarean for fetal distress) but women in the twice-weekly surveillance group were more likely to have induction of labour than those in the fortnightly surveillance group (Risk ratio 1.25, 95% CI 1.04-1.50) and overall their babies were born four day earlier. There was insufficient data to assess perinatal mortality or serious morbidity. No fetal deaths occurred in either group.


This paper, which is one of six in the Lancet's 2011 Stillbirth Series, notes that in developed countries, disparities in stillbirth rates between different population groups indicate that there is scope for further reductions in stillbirth rates. Overweight, obesity and smoking are important modifiable risk factors. Advanced maternal age is also a risk factor. A substantial proportion of stillbirths are linked to placental pathologies and infection associated with preterm birth. National perinatal mortality audit programmes aimed at improving the quality of care could reduce stillbirth rates and an international consensus on definitions and classifications related to stillbirth is necessary. All parents should be offered thorough investigation including a high-quality autopsy and placental histopathology. Future research should focus on screening and interventions to reduce antepartum stillbirth as a result of placental dysfunction.

The other papers in the Lancet stillbirth series, which provide a global perspective on the issue of stillbirth, are:


This systematic review included 96 population-based studies. The highest ranking modifiable risk factor for stillbirth was found to be maternal obesity with a population attributable risk (PAR) calculated to be 8.18% across five countries (Australia, Canada, Netherlands, UK, and USA). Advanced maternal age (> 35 years) had a PAR of 7.11% and maternal smoking had a PAR of 4.7%. In disadvantaged populations the PAR for smoking could be as high as 20%. Primiparity contributes to about 15% of stillbirths. Placental pathology has an important role in stillbirth, as indicated by the PARs for small-for-gestational-age (23%) and placental abruption (15%). Pre-existing maternal diabetes and hypertension still contribute to stillbirth in high income countries. Priority areas for stillbirth prevention are raising awareness and implementing interventions to address obesity, maternal age and smoking.

The Cochrane Collection contains a large number of other reviews relating to tests which may be used to assess fetal wellbeing. Some of the interventions which have been the subject of Cochrane reviews are: fetal movement counting, fetal and umbilical Doppler ultrasound, amniotic fluid index vs. single deepest vertical pocket as a screening test, biochemical tests of placental function, biophysical profiles, routine ultrasound at 24 weeks, symphysis-fundal height measurement, fetal fibronectin testing, and near infrared spectroscopy.
The Perinatal and Maternal Mortality Review Committee (PMMRC) reviews all perinatal and maternal deaths in New Zealand with the aim of identifying ways for improvement in maternal and newborn care. This report is based on the data collected by the Mortality Review Data Group. A perinatal death is defined as one occurring after 20 weeks gestation (or of a baby weighing at least 400g if gestation is unknown) and up to and including the 28th day of life. Besides reporting statistics, the report also makes recommendations for future work by the PMMRC, the Ministry of Health, lead maternity cares, DHBs and others. This report notes a significant increase in perinatal-related mortality among babies born in multiple pregnancies from 2007 to 2011. Of all perinatal-related deaths, 19% were identified as potentially avoidable in 2011. The most common contributory factors were barriers to access or engagement with care followed by personnel factors. Māori and Pacific mothers were significantly more likely to have potentially avoidable perinatal-related deaths than New Zealand European mothers, and there was a significant increase in potentially avoidable perinatal-related death with increasing socioeconomic deprivation. The report also noted that in an audit of babies who had died in 2010 with potentially identifiable congenital abnormalities, one in four women who had sought care with a primary health care provider before 14 weeks was not offered first or second trimester antenatal screening and that folate prophylaxis was infrequent and poorly documented.


This paper examines the role of antenatal care plays in the prevention of stillbirth in high income countries. The 151 cases in this study were women with a singleton, late stillbirth without congenital abnormality, with controls being 310 ongoing pregnancies randomly selected at the same gestation at which the stillbirth occurred. Cases and controls constituted 72% of each of their respective potential cohorts. Findings indicate that a two fold increase in late stillbirth was associated with accessing less than half the recommended antenatal visits (adjusted odds ratio, aOR, 2.68; 95% CI, 1.04-6.90) when compared with accessing the recommended number of visits. Compared to babies identified as being small-for-gestational-age (SGA) during the antenatal period, babies who were SGA or had not been identified as SGA prior to birth, were more at risk of being stillborn (aOR, 9.46; 95% CI, 1.98-45.13). The authors concluded that the study reiterated the importance of regular antenatal care attendance.


This paper reports on the Auckland Stillbirth study, a case-control study conducted from July 2006 to June 2009. Women who had a late stillbirth (≥ 28 weeks) were matched with two controls of the same gestation as each case. In the univariate analysis of results, Pacific ethnicity, overweight and obesity, grand multiparity, not being married, not being in paid work, social deprivation, exposure to tobacco smoke and use of recreational drugs were associated with an increased risk of late stillbirth. In the multivariate analysis Maternal overweight and obesity, nulliparity, grand multiparity, not being married and not being in paid work were independently associated with late stillbirth but Pacific ethnicity was no longer significant (adjusted Odds Ratio 0.99; 0.51-1.91). The disparity in stillbirth rates between Pacific and European women can be explained by confounding factors such as high parity and maternal obesity.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
**Preterm Birth**

**Introduction**

Preterm birth is defined as the birth of a baby prior to 37 weeks completed gestation. It can be further subdivided into moderate/late preterm (32–36 completed weeks), very preterm (28–31 completed weeks) and extremely preterm (less than 28 weeks gestation) birth [12].

Preterm birth is not a single entity, but has a variety of causes (e.g. infections, stress, multiple pregnancy, cervical insufficiency), and pathways (e.g. inflammation, hormone activation, uterine over-distension) [12]. It is traditionally subdivided into three categories: births arising from 1) preterm labour with intact fetal membranes; 2) preterm rupture of the fetal membranes; and 3) iatrogenic preterm birth, where delivery is induced for maternal or fetal reasons [12]. In developed countries, it has been estimated that around 40–45% of preterm births follow preterm labour, 25–40% follow preterm premature rupture of the fetal membranes, and 30–35% are indicated deliveries [12].

Infants born prematurely may experience a range of adverse outcomes. When compared to term infants, preterm infants have higher rates of temperature instability, respiratory distress, infections, apnoea, low blood sugar, jaundice, and feeding difficulties. Preterm birth is also the leading cause of early neonatal death, with the risk increasing with decreasing gestational age [12]. Those born very or extremely prematurely (<32 weeks gestation) are also at an increased risk of developmental disabilities such as cerebral palsy, and intellectual disabilities [13].

Internationally, there have been large increases in preterm birth rates during the past two decades, largely confined to the late preterm (34–36 weeks) category. These increases have been attributed to increasing obstetric intervention, higher rates of twins as a result of assisted reproductive techniques, and delayed childbearing [13]. New Zealand also experienced an increase in preterm birth rates during the 1980s and 1990s, with the most rapid increases occurring amongst those living in the most affluent (NZDep deciles 1–2) areas, and in European/Other women [14,15].

The following section reviews preterm birth rates in the Northern DHBs using information from the Birth Registration Dataset. The section concludes with a brief overview of policy documents and evidence-based reviews which consider how preterm birth might be addressed at the population level.

---

### Data Sources and Methods

**Indicator**

1. Preterm birth rates per 100 live births in singleton live born babies by gestational age
2. Preterm birth rates per 100 live births in live born babies by plurality (singletons, twins, triplets)

**Data Sources**

   Denominator: Birth Registration Dataset: All singleton live born babies 20+ weeks gestation.
   Denominator: Birth Registration Dataset: All live born babies 20+ weeks gestation by plurality.

**Notes on Interpretation**

Note 1: Year is year of registration, rather than year of birth.
Note 2: See Appendix 4 for an overview of the Birth Registration Dataset
Note 3: In this analysis, stillborn babies have been excluded due to advice from the Ministry of Health that the Birth Registration dataset provides less reliable information on stillborn babies than the National Mortality Collection.
New Zealand Distribution and Trends

Preterm Births in Singleton Pregnancies

New Zealand Trends

Preterm Birth Rates: In New Zealand during 2000–2012, preterm birth rates per 100 live births were relatively static. Rates for those born at 20–27 weeks gestation were 0.38 in 2000 and 0.35 in 2012, while rates for those born at 28–31 weeks were 0.60 in 2000 and 0.62 in 2012, and rates for those born at 32–36 weeks were 5.0 in 2000 and 5.1 in 2012 (Figure 5).

Figure 5. Preterm Birth Rates in Singleton Live Born Babies by Gestational Age, New Zealand 2000–2012

[Graph showing preterm birth rates by gestation age from 2000 to 2012]

Source: Birth Registration Dataset: Numerator: all singleton live born babies 20–36 weeks gestation; Denominator: all singleton live born babies 20+ weeks gestation

Number of Preterm Births: During the same period however, the actual number of preterm babies born increased, as the result of a rising birth rate, with the majority of this increase occurring between 2003 and 2008. The largest increases were seen in those born at 32–36 weeks, with numbers in this category rising from 2,744 in 2000 to 3,082 in 2012. While the number of babies born at 28–31 weeks gestation also rose, from 331 in 2000 to 374 in 2012, the number of babies born at 20–27 weeks gestation did not change (211 in both 2000 and 2012) (Figure 6).
Figure 6. Number of Preterm Births in Singleton Live Born Babies by Gestational Age, New Zealand 2000–2012

Source: Birth Registration Dataset, All singleton live born babies 20–36 weeks gestation

Figure 7. Preterm Birth Rates in Singleton Live Born Babies by Baby’s Ethnicity, New Zealand 2000–2012

Source: Birth Registration Dataset: Numerator: All singleton live born babies 20–36 weeks gestation; Denominator: All singleton live born babies 20+ weeks gestation; Note: Ethnicity is Level 1 Prioritised
**Distribution by Ethnicity, NZDep Decile, Gender and Maternal Age**

*All Preterm Births (20–36 Weeks):* In New Zealand during 2008–2012, preterm birth rates were significantly higher for males and for Māori babies than for Asian/Indian, European/Other and Pacific babies. Rates were also significantly higher for those living in more deprived (NZDep deciles 7–10) areas, and for the babies of younger (<25 years) and older (35+ years) mothers, than for the babies of mothers aged 25–29 years (Table 9). Similarly, during 2000–2012, preterm birth rates for Māori babies were consistently higher than for Asian/Indian, European/Other and Pacific babies (Figure 7).

Table 9. Preterm Birth Rates in Singleton Live Born Babies by Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Preterm Singletons (20–36 Weeks)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ Deprivation Index Decile</td>
<td></td>
<td></td>
<td></td>
<td>Maternal Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>5.58</td>
<td>1.00</td>
<td></td>
<td>&lt;20 Years</td>
<td>7.51</td>
<td>1.34</td>
<td>1.26–1.41</td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>5.65</td>
<td>1.01</td>
<td>0.96–1.06</td>
<td>20–24 Years</td>
<td>6.14</td>
<td>1.09</td>
<td>1.05–1.14</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>5.77</td>
<td>1.03</td>
<td>0.98–1.08</td>
<td>25–29 Years</td>
<td>5.62</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>6.15</td>
<td>1.10</td>
<td>1.05–1.16</td>
<td>30–34 Years</td>
<td>5.65</td>
<td>1.01</td>
<td>0.97–1.05</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>6.61</td>
<td>1.19</td>
<td>1.13–1.24</td>
<td>35+ Years</td>
<td>6.46</td>
<td>1.15</td>
<td>1.10–1.20</td>
</tr>
<tr>
<td>Baby’s Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>5.89</td>
<td>1.02</td>
<td>0.98–1.07</td>
<td>Female</td>
<td>5.53</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>5.76</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>6.52</td>
<td>1.18</td>
<td>1.15–1.21</td>
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<tr>
<td>Māori</td>
<td>6.64</td>
<td>1.15</td>
<td>1.12–1.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>5.83</td>
<td>1.01</td>
<td>0.97–1.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Birth Registration Dataset; Numerator: All singleton live born babies 20–36 weeks gestation; Denominator: All singleton live born babies 20+ weeks gestation; Note: Rates are per 100 live births; Baby’s Ethnicity is Level 1 prioritised; Decile is NZDep2006

When broken down by gestational age, a common theme emerged, with the magnitude of the excess risk of preterm birth seen for Māori, Pacific and Asian/Indian babies, the babies of teenage mothers, and those from the more deprived areas, being most marked amongst births at lower gestations (Table 10). Specifically:

**20–27 Weeks:** During 2008–2012, preterm birth rates at 20–27 weeks gestation were significantly higher for males, and for those living in more deprived (NZDep deciles 7–10) areas. Rates were also significantly higher for Māori, Pacific and Asian/Indian babies than for European/Other babies, and for the babies of teenage mothers, followed by those aged 20–24 years (Table 10).

**28–31 Weeks:** During 2008–2012, preterm birth rates at 28–31 weeks gestation were also significantly higher for males and for those living in average and more deprived (NZDep deciles 3–4 and 7–10) areas. Rates were also significantly higher for Pacific and Māori babies than for European/Other babies, and for the babies of teenage mothers, followed by those aged 20–24 years (Table 10).

**32–36 Weeks:** During 2008–2012, preterm birth rates at 32–36 weeks gestation were significantly higher for males, for Māori babies than for European/Other, Asian/Indian and Pacific babies, and for those living in the most deprived (NZDep deciles 9–10) areas. Preterm birth rates were also significantly higher for the babies of younger (<25 years) and older (35+ years) mothers than for the babies of mothers aged 25–29 years (Table 10).

Preterm Birth - 61
Table 10. Preterm Birth Rates in Singleton Live Born Babies by Ethnicity, NZ Deprivation Index Decile, Gender, Maternal Age and Gestational Age, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preterm Singletons 20–27 Weeks</strong></td>
<td></td>
<td></td>
<td></td>
<td><strong>Maternal Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>0.26</td>
<td>1.00</td>
<td></td>
<td>&lt;20 Years</td>
<td>0.69</td>
<td>2.14</td>
<td>1.75–2.62</td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>0.29</td>
<td>1.11</td>
<td>0.87–1.42</td>
<td>20–24 Years</td>
<td>0.43</td>
<td>1.34</td>
<td>1.12–1.60</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>0.29</td>
<td>1.13</td>
<td>0.89–1.43</td>
<td>25–29 Years</td>
<td>0.32</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>0.36</td>
<td>1.37</td>
<td>1.10–1.70</td>
<td>30–34 Years</td>
<td>0.28</td>
<td>0.87</td>
<td>0.73–1.04</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>0.50</td>
<td>1.92</td>
<td>1.57–2.36</td>
<td>35+ Years</td>
<td>0.33</td>
<td>1.02</td>
<td>0.85–1.22</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Baby's Ethnicity</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Asian/Indian</td>
<td>0.39</td>
<td>1.42</td>
<td>1.17–1.72</td>
</tr>
<tr>
<td>European/Other</td>
<td>0.27</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>0.46</td>
<td>1.66</td>
<td>1.45–1.91</td>
</tr>
<tr>
<td>Pacific</td>
<td>0.44</td>
<td>1.59</td>
<td>1.32–1.91</td>
</tr>
</tbody>
</table>

| **Preterm Singletons 28–31 Weeks** |      |            |        | **Maternal Age**          |      |            |        |
| Deciles 1–2               | 0.45 | 1.00       |        | <20 Years                 | 0.78 | 1.36       | 1.14–1.62 |
| Deciles 3–4               | 0.56 | 1.23       | 1.03–1.47 | 20–24 Years               | 0.69 | 1.19       | 1.04–1.37 |
| Deciles 5–6               | 0.54 | 1.18       | 0.99–1.41 | 25–29 Years               | 0.58 | 1.00       |        |
| Deciles 7–8               | 0.64 | 1.41       | 1.20–1.67 | 30–34 Years               | 0.54 | 0.93       | 0.81–1.06 |
| Deciles 9–10              | 0.77 | 1.70       | 1.45–1.99 | 35+ Years                 | 0.66 | 1.14       | 1.00–1.30 |

<table>
<thead>
<tr>
<th>Baby's Ethnicity</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian/Indian</td>
<td>0.60</td>
<td>1.11</td>
<td>0.96–1.29</td>
</tr>
<tr>
<td>European/Other</td>
<td>0.54</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>0.71</td>
<td>1.32</td>
<td>1.19–1.46</td>
</tr>
<tr>
<td>Pacific</td>
<td>0.72</td>
<td>1.33</td>
<td>1.16–1.54</td>
</tr>
</tbody>
</table>

| **Preterm Singletons 32–36 Weeks** |      |            |        | **Maternal Age**          |      |            |        |
| Deciles 1–2               | 4.87 | 1.00       |        | <20 Years                 | 6.03 | 1.28       | 1.20–1.36 |
| Deciles 3–4               | 4.78 | 0.98       | 0.93–1.04 | 20–24 Years               | 5.02 | 1.06       | 1.01–1.12 |
| Deciles 5–6               | 4.92 | 1.01       | 0.96–1.07 | 25–29 Years               | 4.72 | 1.00       |        |
| Deciles 7–8               | 5.15 | 1.06       | 1.00–1.11 | 30–34 Years               | 4.84 | 1.02       | 0.98–1.07 |
| Deciles 9–10              | 5.34 | 1.10       | 1.05–1.15 | 35+ Years                 | 5.47 | 1.16       | 1.11–1.21 |

<table>
<thead>
<tr>
<th>Baby's Ethnicity</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian/Indian</td>
<td>4.90</td>
<td>0.99</td>
<td>0.94–1.04</td>
</tr>
<tr>
<td>European/Other</td>
<td>4.94</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>5.48</td>
<td>1.11</td>
<td>1.07–1.15</td>
</tr>
<tr>
<td>Pacific</td>
<td>4.67</td>
<td>0.94</td>
<td>0.90–1.00</td>
</tr>
</tbody>
</table>

Source: Birth Registration Dataset: Numerator: All singleton live born babies 20–36 weeks gestation; Denominator: All singleton live born babies 20+ weeks gestation; Note: Rates are per 100 live births; Baby’s Ethnicity is Level 1 prioritised; Decile is NZDep2006
Preterm Births in Multiple Pregnancies

Figure 8. Gestational Age at Delivery by Plurality, New Zealand Live Births 2008–2012

Source: Birth Registration Dataset: All live born babies 20–36 weeks gestation
Distribution by Gestational Age
In New Zealand during 2008–2012, 94.0% of singleton babies were born after 36 weeks gestation, as compared to only 44.9% of twins and 1.3% of triplets, with the gestational age curve shifting increasingly towards the left (i.e. towards younger gestational ages) as the number of babies increased. During this period, the most frequent gestational age for the delivery of a singleton baby was 40 weeks, as compared to 37 weeks for twins and 34 weeks for triplets (Figure 8).

Risk of Preterm Birth by Plurality
In New Zealand during 2008–2012, preterm birth rates per 100 live births were 6.0 for singletons, 55.1 for twins and 98.7 for triplets, with the risk of preterm birth being 9.13 (95% CI 8.92–9.35) times higher for twins and 16.34 (95% CI 16.02–16.68) times higher for triplets, than for singleton babies (Table 11).

Table 11. Preterm Birth Rates by Plurality, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Plurality</th>
<th>Number Preterm Births: Total 2008–2012</th>
<th>Number Preterm Births: Annual Average</th>
<th>Number Live Births: Annual Average</th>
<th>Preterm Birth Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Singleton</td>
<td>18,625</td>
<td>3,725</td>
<td>61,664</td>
<td>6.04</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Triplet</td>
<td>232</td>
<td>46</td>
<td>47</td>
<td>98.72</td>
<td>16.34</td>
<td>16.02–16.68</td>
</tr>
</tbody>
</table>

Source: Birth Registration Dataset; Numerator: All live born babies 20–36 weeks gestation; Denominator: All live born babies 20+ weeks gestation; Note: Preterm birth rates are per 100 live births

New Zealand Trends
Preterm Birth Rates: In New Zealand during 2000–2012, preterm birth rates per 100 live births were relatively static in singletons and triplets, with rates for singletons being 6.0 in 2000 and 6.1 in 2012. Similarly rates for triplets were 96.3 in 2000 and 100.0% in 2012. In contrast, preterm birth rates in twins increased, from 49.9 in 2000 to 59.3 in 2000, with the majority of this increase occurring after 2008 (Figure 9).

Number of Preterm Births: During the same period, the actual number of singleton preterm babies born increased (from 3,286 in 2000 to 3,667 in 2012), as the result of a rising birth rate, with the majority of this increase being between 2003 and 2008. Similarly, the number of twin preterm births increased, from 866 in 2000 to 979 in 2012. The number of triplet births however was more static (Figure 10).
Figure 9. Preterm Birth Rates in Live Born Babies by Plurality, New Zealand 2000–2012

Source: Birth Registration Dataset: Numerator: All live born babies 20–36 weeks gestation; Denominator: All live born babies 20+ weeks gestation

Figure 10. Number of Preterm Live Births by Plurality, New Zealand 2000–2012

Source: Birth Registration Dataset; All live born babies 20–36 weeks gestation
Distribution by Ethnicity, NZDep Decile, Gender and Maternal Age

In New Zealand during 2008–2012, there were no significant gender, socioeconomic (as measured by NZDep06), or maternal age-related differences in preterm birth rates amongst twins. Preterm birth rates for Pacific twins however, were significantly (albeit only marginally) lower than for European/Other babies (Table 12).

Table 12. Preterm Birth Rates in Live Born Twins by Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm Twins 20–36 Weeks</td>
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<td></td>
<td></td>
<td>Maternal Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ Deprivation Index Decile</td>
<td></td>
<td></td>
<td></td>
<td>&lt;20 Years</td>
<td>59.72</td>
<td>1.08</td>
<td>0.98–1.18</td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>57.07</td>
<td>1.00</td>
<td></td>
<td>20–24 Years</td>
<td>55.65</td>
<td>1.00</td>
<td>0.94–1.07</td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>54.90</td>
<td>0.96</td>
<td>0.90–1.03</td>
<td>25–29 Years</td>
<td>55.53</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>54.78</td>
<td>0.96</td>
<td>0.90–1.02</td>
<td>30–34 Years</td>
<td>52.94</td>
<td>0.95</td>
<td>0.90–1.01</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>55.49</td>
<td>0.97</td>
<td>0.92–1.03</td>
<td>35+ Years</td>
<td>56.17</td>
<td>1.01</td>
<td>0.96–1.07</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>53.98</td>
<td>0.95</td>
<td>0.89–1.00</td>
<td>Baby’s Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>59.32</td>
<td>1.06</td>
<td>1.00–1.14</td>
<td>Female</td>
<td>55.87</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>55.73</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>54.41</td>
<td>0.97</td>
<td>0.94–1.01</td>
</tr>
<tr>
<td>Māori</td>
<td>54.56</td>
<td>0.98</td>
<td>0.94–1.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>50.91</td>
<td>0.91</td>
<td>0.85–0.98</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Birth Registration Dataset; Numerator: All live born babies 20–36 weeks gestation; Denominator: All live born babies 20+ weeks gestation; Note: Rates are per 100 live births; Baby’s Ethnicity is Level 1 prioritised; Decile is NZDep2006

Northern DHBs Distribution and Trends

Northern DHBs Distribution

In the Northern region during 2008–2012, on average 126.0 Northland, 436.2 Waitemata, 350.4 Auckland and 531.6 Counties Manukau babies per year were born prior to 37 weeks gestation, with the majority of births being in the 32–36 weeks category (Table 13).

In the Northland and Auckland DHBs preterm birth rates at 20–27 and 28–31 weeks were not significantly different from New Zealand rates, while rates at 32–36 weeks were significantly lower (Table 13).

In the Waitemata DHB, preterm birth rates at 20–27 and 32–36 weeks were not significantly different from New Zealand rates, while rates at 28–31 weeks were significantly lower (Table 13).

In Counties Manukau, preterm birth rates at 20–27 weeks and 28–31 weeks were significantly higher than New Zealand rates, while rates at 32–36 weeks were similar (Table 13).

Northern DHBs Trends

In the Northern region during 2000–2012, preterm birth rates in the Northland and Auckland DHBs were consistently lower than the New Zealand rate. In the Waitemata DHB, a gradual decline in preterm births saw rates become lower than the New Zealand rate from 2008–09 onwards, while in Counties Manukau there was considerable year to year variability. Preterm birth rates in Counties Manukau however, were higher than the New Zealand rate during 2010–2012 (Figure 11).
### Table 13. Preterm Birth Rates in Singleton Live Born Babies by Gestational Age, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Preterm Birth Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preterm Births</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Northland</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–27 weeks</td>
<td>47</td>
<td>9.4</td>
<td>0.41</td>
<td>1.15</td>
<td>0.86–1.54</td>
</tr>
<tr>
<td>28–31 weeks</td>
<td>71</td>
<td>14.2</td>
<td>0.62</td>
<td>1.01</td>
<td>0.80–1.28</td>
</tr>
<tr>
<td>32–36 weeks</td>
<td>512</td>
<td>102.4</td>
<td>4.49</td>
<td>0.89</td>
<td>0.81–0.97</td>
</tr>
<tr>
<td>37+ weeks</td>
<td>10,774</td>
<td>2,154.8</td>
<td>94.48</td>
<td>1.15</td>
<td>0.86–1.54</td>
</tr>
<tr>
<td>Northland Total</td>
<td>11,404</td>
<td>2,280.8</td>
<td>100.00</td>
<td>1.15</td>
<td>0.86–1.54</td>
</tr>
<tr>
<td><strong>Waitemata</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–27 weeks</td>
<td>115</td>
<td>23.0</td>
<td>0.30</td>
<td>0.83</td>
<td>0.69–1.01</td>
</tr>
<tr>
<td>28–31 weeks</td>
<td>173</td>
<td>34.6</td>
<td>0.45</td>
<td>0.73</td>
<td>0.62–0.85</td>
</tr>
<tr>
<td>32–36 weeks</td>
<td>1,893</td>
<td>378.6</td>
<td>4.92</td>
<td>0.97</td>
<td>0.93–1.02</td>
</tr>
<tr>
<td>37+ weeks</td>
<td>36,267</td>
<td>7,253.4</td>
<td>94.33</td>
<td>1.15</td>
<td>0.86–1.54</td>
</tr>
<tr>
<td>Waitemata Total</td>
<td>38,448</td>
<td>7,689.6</td>
<td>100.00</td>
<td>1.15</td>
<td>0.86–1.54</td>
</tr>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–27 weeks</td>
<td>130</td>
<td>26.0</td>
<td>0.40</td>
<td>1.13</td>
<td>0.94–1.35</td>
</tr>
<tr>
<td>28–31 weeks</td>
<td>172</td>
<td>34.4</td>
<td>0.53</td>
<td>0.86</td>
<td>0.74–1.01</td>
</tr>
<tr>
<td>32–36 weeks</td>
<td>1,450</td>
<td>290.0</td>
<td>4.51</td>
<td>0.89</td>
<td>0.84–0.94</td>
</tr>
<tr>
<td>37+ weeks</td>
<td>30,414</td>
<td>6,082.8</td>
<td>94.55</td>
<td>1.15</td>
<td>0.94–1.35</td>
</tr>
<tr>
<td>Auckland DHB Total</td>
<td>32,166</td>
<td>6,433.2</td>
<td>100.00</td>
<td>1.15</td>
<td>0.94–1.35</td>
</tr>
<tr>
<td><strong>Counties Manukau</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–27 weeks</td>
<td>200</td>
<td>40.0</td>
<td>0.47</td>
<td>1.31</td>
<td>1.12–1.52</td>
</tr>
<tr>
<td>28–31 weeks</td>
<td>316</td>
<td>63.2</td>
<td>0.74</td>
<td>1.20</td>
<td>1.06–1.35</td>
</tr>
<tr>
<td>32–36 weeks</td>
<td>2,142</td>
<td>428.4</td>
<td>5.02</td>
<td>0.99</td>
<td>0.95–1.04</td>
</tr>
<tr>
<td>37+ weeks</td>
<td>40,024</td>
<td>8,004.8</td>
<td>93.77</td>
<td>1.15</td>
<td>0.94–1.35</td>
</tr>
<tr>
<td>Counties Manukau Total</td>
<td>42,682</td>
<td>8,536.4</td>
<td>100.00</td>
<td>1.15</td>
<td>0.94–1.35</td>
</tr>
<tr>
<td><strong>New Zealand</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–27 weeks</td>
<td>1,106</td>
<td>221.2</td>
<td>0.36</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>28–31 weeks</td>
<td>1,906</td>
<td>381.2</td>
<td>0.62</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>32–36 weeks</td>
<td>15,613</td>
<td>3,122.6</td>
<td>5.06</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>37+ weeks</td>
<td>289,694</td>
<td>57,938.8</td>
<td>93.96</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>New Zealand Total</td>
<td>308,319</td>
<td>61,663.8</td>
<td>100.00</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: Birth Registration Dataset: Numerator: All singleton live born babies 20–36 weeks gestation; Denominator: All singleton live born babies 20+ weeks gestation; Note: Preterm birth rates are per 100 live births

**Northern DHBs Distribution by Ethnicity**

In the Northland, Auckland and Counties Manukau DHBs during 2000–2012, preterm birth rates were higher for Māori than for European/Other babies. In the Waitemata DHB however, there were no consistent differences in preterm birth rates between Māori and European/Other babies (Figure 12).
Figure 11. Preterm Birth Rates in Singleton Live Born Babies, Northern DHBs vs. New Zealand 2000–2012

Source: Birth Registration Dataset: Numerator: All singleton live born babies 20–36 weeks gestation; Denominator: All singleton live born babies 20+ weeks gestation
Figure 12. Preterm Birth Rates in Singleton Live Born Babies by Baby’s Ethnicity, Northern DHBs vs. New Zealand 2000–2012

Source: Birth Registration Dataset: Numerator: All singleton live born babies 20–36 weeks gestation; Denominator: All singleton live born babies 20+ weeks gestation; Note: Ethnicity is Level 1 Prioritised
Local Policy Documents and Evidence-Based Reviews Relevant to Preterm Birth

In New Zealand at present, there is no single strategy which focuses on the prevention of preterm birth. Thus any local strategies developed will need to incorporate evidence from a variety of sources. Table 14 provides an overview of a range of New Zealand policy documents and overseas evidence-based reviews which may be useful in this context.

Table 14. Local Policy Documents and Evidence-Based Reviews Relevant to Preterm Birth

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>In New Zealand a number of Government policy documents identify and are beginning to report on clinical indicators related to the quality and delivery of maternity services including preterm births.</td>
</tr>
</tbody>
</table>

| A set of twelve maternity clinical indicators have been established for the New Zealand setting that can be drawn from available data collections. The intent of these clinical indicators is to assist DHBs and maternity stakeholders to use national benchmarked data in local maternity quality and safety programmes. Indicator 12 is premature birth (32–36 weeks gestation). |

| This is the report for the first year of operation by the New Zealand National Maternity Monitoring Group (NMMG). The NMMG was established to oversee the implementation of the NZ Maternity Standards. It contains a chapter that reports on the monitoring conducted on late preterm birth (34-36 weeks gestation) and a further chapter on smoking amongst pregnant women. Each chapter includes a set of expectations for DHBs with respect to the indicators to be collected in the coming year and how they will meet the Ministry of Health’s targets. Preterm births varied by DHB, and the report identifies changes the NMMG expects to see: specifically that each DHB audit preterm births in their region, particularly those from 34 to 36 weeks. The proportion of mothers smoking at both delivery and 2 weeks post-delivery differed significantly across DHBs. The report notes that the NMMG expects changes from the Ministry, proposing it expand its data collection from primary care providers. Changes are expected from DHBs too. It is proposed that they work with all aspects of maternity services to meet the Ministry’s target on advice and support for pregnant women to quit smoking. |

| Women with twin and triplet pregnancies have a higher risk of preterm birth. This guideline is complementary to the NICE guideline ‘Antenatal care: routine care for the healthy pregnant woman’ (NICE clinical guideline 62) and it specifies the additional or different care that women with twin or triplet pregnancies should receive. Chapter 8 deals specifically with preterm birth. Following discussion of the research evidence, the following recommendations are made regarding the prevention of preterm birth and its associated risks: |
| • Be aware that women who have had a previous premature singleton birth are at increased risk. |
| • Do not use fibronectin testing alone, home uterine activity monitoring, or routine cervical length measuring (with or without fetal fibronectin) to predict the risk of spontaneous preterm birth in twin and triplet pregnancies. |
| • Do not use the following interventions (either alone or in combination) routinely to prevent spontaneous preterm birth in twin and triplet pregnancies: bed rest (either at home or in hospital), intramuscular or vaginal progesterone, cervical cerclage or oral tocolytics. |
| • Inform women with twin and triplet pregnancies their risk of preterm birth and about the benefits of targeted (i.e. when birth is imminent) corticosteroids. |
| • Do not use single or multiple untargeted (routine) courses of corticosteroids and inform women that there is no benefit from using untargeted corticosteroids. |

The guideline appendices, which include the details of the evidence review (including the evidence tables) can be found at [http://guidance.nice.org.uk/CG129/Guidance/Appendices](http://guidance.nice.org.uk/CG129/Guidance/Appendices) |

In 2013, priority statements to drive measurable quality improvements were issued: Multiple Pregnancy Quality Standards (QS46). These are available at [http://publications.nice.org.uk/multiple-pregnancy-qs46](http://publications.nice.org.uk/multiple-pregnancy-qs46) |

A NICE Pathways is available on multiple pregnancy. It provides a very accessible and rapid reference to information for patients and the public, information and guidance for professionals, and quality standards and practice resources for professionals and health systems. It is available at [http://pathways.nice.org.uk/pathways/multiple-pregnancy](http://pathways.nice.org.uk/pathways/multiple-pregnancy)

This systematic review of randomised controlled trials and cohort studies on the effectiveness of cervical pessaries to prevent preterm birth concluded that while there is some potential effectiveness in the intervention, more RCTs are required before pessaries could be used in practice. Data from six cohort studies and four RCTs were examined in this review. One RCT (n=380) indicated a lower delivery rate prior to 34 weeks (RR 0.24; 95% CI, 0.13–0.43) while another smaller trial (n=108) did not show any positive effect at this stage (RR 1.73; 95% CI 0.43–6.88).

Psychosocial interventions for supporting women to stop smoking in pregnancy. Cochrane Database of Systematic Reviews, Issue 10. Art. No.: CD001055. DOI:10.1002/14651858.CD001055.pub4

Smoking during pregnancy increases the risk of the mother having complications during pregnancy and the baby being born preterm (before 37 weeks). This updated systematic review identifies that psychosocial interventions to support women to stop smoking in pregnancy can increase the proportion of women who stop smoking in late pregnancy and reduce low birthweight and preterm births. Eighty six trials were included in the review with 79 of them involving 29,000 women providing data on smoking abstinence in late pregnancy. Most were conducted in high income countries. Detailed results are provided in the review, with the key findings being that interventions that provided an incentive to stop smoking appeared to support the most women to quit (one study: RR 3.64; 95% CI 1.84–7.23) and an alternative intervention (one study: RR 4.05; 95% CI 1.48–11.11). In studies comparing counselling and usual care (the largest comparison), it was unclear whether interventions prevented smoking relapse among women who had stopped smoking spontaneously in early pregnancy (eight studies; average RR 1.06; 95% CI 0.93–1.21). However, a clear effect was seen in smoking abstinence at zero to five months postpartum (10 studies; average RR 1.76; 95% CI 1.05–2.95), a borderline effect at six to 11 months (six studies; average RR 1.33, 95% CI 1.00–1.77), and a significant effect at 12 to 17 months (two studies, average RR 2.20, 95% CI 1.23–3.96), but not in the longer term. Feedback interventions had a significant effect only when compared with usual care and provided in conjunction with other strategies, such as counselling (two studies; average RR 4.39, 95% CI 1.89–10.21), but the effect was unclear when compared with a less intensive intervention (two studies; average RR 1.19, 95% CI 0.45–3.12). Peer provided social support appeared effective (five studies; average RR 1.49, 95% CI 1.01–2.19), but the effect of partner support was not clear (one study).


Bacterial vaginosis is an overgrowth of anaerobic bacteria and a lack of normal lactobacillary vaginal flora. It has been associated with preterm birth and other poor perinatal outcomes. This updated review aimed to assess the effects of antibiotic treatment of bacterial vaginosis in pregnancy. Twenty one good quality RCTs (7847 women) were included. Antibiotic therapy was effective at eradicating bacterial vaginosis during pregnancy (average RR 0.42; 95% CI 0.31–0.56) and reduced the risk of late miscarriage (RR 0.20; 95% CI 0.05–0.76). Treatment did not reduce the risk of preterm birth before 37 weeks (average RR 0.89; 95% CI 0.71–1.09), or the risk of preterm pre-labour rupture of membranes (RR 0.74; 95% CI 0.30–1.84). It did increase the risk of side-effects sufficient to stop or change treatment (RR 1.66; 95% CI 1.02–2.68). Treatment before 20 weeks' gestation did not reduce the risk of preterm birth (average RR 0.85; 95% CI 0.62–1.17). In women with a previous preterm birth, treatment did not affect the risk of subsequent preterm birth (average RR 0.78; 95% CI 0.42–1.48). In women with abnormal vaginal flora (intermediate flora or bacterial vaginosis), treatment may reduce the risk of preterm birth (RR 0.53; 95% CI 0.34–0.84). One small trial of 156 women compared metronidazole and clindamycin, both oral and vaginal, with no significant differences seen for any of the pre-specified primary outcomes. Statistically significant differences were seen for the outcomes of prolongation of gestational age (days) (mean difference (MD) 1.00; 95% CI 0.26–1.74) and birthweight (grams) (MD 75.18; 95% CI 25.37–124.99) however these represent relatively small differences in the clinical setting. The authors concluded that antibiotic treatment can eradicate bacterial vaginosis in pregnancy; however, the overall risk to preterm birth was not significantly reduced. There was little evidence that screening and treating all pregnant women with bacterial vaginosis will prevent preterm birth and its consequences.

Cervical stitch (cerclage) for preventing preterm birth in singleton pregnancy. Cochrane Database of Systematic Reviews. Issue 4:CD0008991. DOI: 10.1002/14651858.CD0008991.pub2

The use of cervical cerclage, in which a stitch is positioned around the neck of the cervix intended to reduce the risk of preterm birth, is still considered controversial for safety and effectiveness reasons. This review is a further assessment of this procedure, specifically focusing on whether the use of such a stitch in singleton pregnancy at high risk of loss based on a woman’s history and/or ultrasound finding of a short cervix or physical examination improves subsequent obstetric care and fetal outcome. Twelve trials were included (n = 3328 women). Compared to no treatment, cervical cerclage made no significant difference to perinatal death (8.4% versus 10.7%) (RR 0.78; 95% CI 0.61–1.00) and neonatal morbidity (9.6% versus 10.2%) (RR 0.95; 95% CI 0.63–1.43), but did show significant reduction in preterm births (average RR 0.80; 95% CI 0.69–0.95). Cervical cerclage was associated with significantly higher rates of caesarean sections (RR 1.19; 95% CI 1.01–1.40) and higher rates of maternal side effects (vaginal discharge and bleeding, pyrexia) (average RR 2.25; 95% CI 0.89 to 5.69). The review concluded that cerclage reduces the incidence of preterm birth in women at risk of recurrent preterm birth, although there was no significant reduction in perinatal mortality or neonatal morbidity and it was unclear about the effect long term on the baby. Caesarean section is more likely where cervical cerclage has been utilised. The authors’ advice is that decisions should be ‘personalised’, based on the woman’s informed choice, the clinical circumstances and the expertise of the clinical team.

This review article explains that, largely because of the limited understanding of the basic biology underlying preterm delivery, there are few opportunities for prevention. Two strategies which could have a very small effect in reducing rates of preterm birth are decreasing higher-order multiple births resulting from the use of assisted reproductive technology and improving estimates of gestational age in early pregnancy in order to reduce the number of infants inadvertently delivered preterm because of inaccurate dates. Public health approaches to prematurity include ensuring that premature infants are delivered in a suitable facility able to deal with neonatal complications, minimising variations in quality of care between institutions, early developmental support for such infants and support for families.


There have been many scoring systems designed to facilitate prediction of preterm birth so that appropriate interventions might reduce the incidence of preterm and very preterm birth and the associated adverse outcomes. Extensive searching by the authors of this review failed to reveal any RCTs evaluating such scoring systems. The value of scoring systems is thus unknown. Prospective studies are needed, followed by RCTs of promising systems.


Heavy vaginal colonisation with ureaplasma is suspected of playing a role in preterm rupture of membranes and preterm birth but the benefits of treating it with antibiotics are unclear. Based on a review of one RCT of 3 types antibiotic treatment vs. placebo in 1105 pregnant women (between 22 and 33 weeks gestation), which did not report on rates of preterm birth, the authors concluded that there was insufficient evidence to either support or refute the use of antibiotics for ureaplasma infection to prevent preterm birth.


Previous preterm delivery is strong predictor of preterm delivery and for this reason specialised care for women with a previous history of preterm delivery is common practice. This review considered three RCTs conducted in the 1980s comparing specialised care with standard care in women with a singleton pregnancy who were considered to be at high risk of preterm labour (3400 women all in the U.S.). The authors reported that overall there was very little difference in outcomes between specialised and standard care groups, but due to differences in study designs most outcomes were only reported by one study which limited statistical power to detect significant differences. All three studies reported on preterm birth before 37 weeks and a pooled analysis of the results suggested that there may have been fewer preterm births in the specialised care mothers but the difference was not statistically significant (RR 0.87, 95% CI 0.69–1.08).


This series of seven reviews provides a global perspective on preterm birth. The third review in the series is:


This systematic review discusses the evidence for the effectiveness of interventions to prevent preterm birth and to improve survival among preterm newborns particularly those applicable to low-to-middle income countries. Recommendations are rated in four categories (from strong in favour to strong against) based on the quality of evidence, how the evidence may be translated to practice in a specific setting such as low-to-middle income countries, the level of baseline risk, and on potential trade-offs between expected benefits, harms and costs. The two interventions strongly recommended for preventing preterm births are smoking cessation and the use of progesterone. The authors note that since specialised clinics are now an accepted part of antenatal services in many countries it is unlikely that further RCTs will be carried out. They suggest that further research should focus on service development.


Numerous studies have consistently shown a relationship between social disadvantage and low birthweight (<2500g). Many countries have programmes to assist women who are thought to be at risk of having a low birthweight baby and these may include advice and counselling, practical assistance (e.g. transport to clinic appointments or help with household responsibilities and care of other children), and emotional support. This review included 17 RCTs (12,264 women) of additional support, provided by either a professional (social worker, midwife or nurse) or a trained lay person, compared to routine care. Programmes of extra support made no difference to rates of either low birthweight or preterm births but they were associated with a reduced likelihood of antenatal hospital admission (3 trials, 737 women, RR 0.79, 95% CI 0.68–0.92) and of caesarean birth (9 trials, 4522 women, RR 0.87, 95% CI 0.78–0.97).

This is the report for a very sizeable project which aimed to identify combinations of tests and treatments to predict and prevent preterm labour. It includes both two systematic reviews and a decision analysis (health economic evaluation). One systematic review aimed to determine the accuracy of 22 different tests for the prediction of preterm birth in asymptomatic women in early pregnancy and in women symptomatic with threatened preterm labour in later pregnancy and the other review assessed the effectiveness of interventions with potential to reduce spontaneous preterm births in asymptomatic women in early pregnancy and to reduce spontaneous preterm births or improve neonatal outcomes in women with a viable pregnancy and symptoms of threatened preterm labour. The economic evaluation incorporated the combined effects of test and treatments and costs in a model-based analysis.

### Cervical assessment by ultrasound for preventing preterm delivery

**Cochrane Database Systematic Review. Issue 1:CD007235. doi: 10.1002/14651858.CD007235.pub3.**

While measurement of cervical length by trans-vaginal ultrasound (TVU) can be used to predict preterm birth (the shorter the cervical length, the higher the risk) it is uncertain if it is useful as a screening test for the prevention of preterm birth. This review aimed to assess the effect of knowledge of cervical length on the effectiveness of antenatal management in preventing preterm birth. This review included five RCTs (507 women) of knowledge of cervical length (obtained by TVU) vs. no knowledge of cervical length. In the three trials (290 women) involving singleton gestations with preterm labour, knowledge of cervical length was associated with a non-significant decrease in preterm birth at < 37 weeks (22.3% versus 34.7%, respectively; risk ratio 0.59, 95% CI 0.26–1.32) and delivery occurred on average 0.64 weeks later (95% CI 0.03–1.25 weeks). There were no differences in other outcomes measured. One trial in singleton gestations with premature rupture of membranes (92 women) evaluated the safety of TVU in this situation and found no difference in maternal or neonatal infection rates between the group that had TVU and the group that did not. In the one trial in twin gestations (125 women, with or without preterm labour) there was no difference between the TVU and no TVU groups in preterm birth at 36, 34 or 30 weeks, or in gestational age at delivery or other perinatal and maternal outcomes. Life table analysis showed significantly less (p = 0.02) preterm birth at <35 weeks in the TVU group compared to the no TVU group. The authors of this review concluded that there is insufficient evidence to recommend routine screening of either symptomatic or asymptomatic pregnant women with cervical length measurement via TVU.

### Fetal fibronectin testing for reducing the risk of preterm birth

**Cochrane Database Systematic Reviews. Issue 4. Art. No.: CD006843. DOI: 10.1002/14651858.CD006843.pub2.**

Fetal fibronectin (FFN) is a protein which is localised at the maternal-fetal interface of the amniotic membranes and is normally found only at very low levels in cervico-vaginal secretions. Levels greater ≥50 ng/l at or after 22 weeks have been associated with an increased risk of preterm birth and high FFN levels have been found to be one of the best predictors of preterm birth in all populations studied to date. The aim of this review was to assess the effectiveness of management based on knowledge of FFN levels, compared to management without such knowledge, for the prevention of preterm birth. This review included five RCTs (474 women) of knowledge vs. no knowledge of FFN. There was a significant decrease in preterm birth at <37 weeks in the knowledge group compared to the no-knowledge group (15.6% vs. 28.6%, RR 0.54, 95% CI 0.34–0.87). All other outcomes measured were similar in both groups (preterm birth at <34, 32, or 28 weeks; gestational age at delivery; birthweight < 2500 grams; perinatal death; maternal hospitalization; tocolytics; steroids for fetal lung maturity; and time to evaluate i.e. time between hospital arrival and a management decision being made). The authors of this review concluded that, although FFN measurements are commonly used in labour and delivery units, there is currently little evidence to recommend such measurements. Given the association found in this review between knowledge of FFN results and a lower incidence of preterm birth before 37 weeks, further research is worthwhile and should be encouraged.

Genital tract infection is a cause of preterm birth and infection screening has been used as a means of preventing preterm birth. There are some adverse effects from treating such infections including cost and increased antibiotic resistance. The authors of this review identified one high quality RCT (4155 women). In the intervention group the results of screening for bacterial vaginosis, trichomonas vaginalis and candidiasis were reported and women received treatment if tests were positive, and in the control group results of tests were not reported. There were significantly fewer preterm births in the intervention group (3% vs. 5%, relative risk 0.55, 95% CI 0.41–0.75) and also fewer preterm very low birthweight (<1500g) infants (RR 0.34, 95% CI 0.15–0.75) and preterm low birthweight (<2500g) infants (RR 0.48, 95% CI 0.34–0.66). The review authors concluded that infection screening and treatment programmes in pregnant women before 20 weeks gestation reduce both preterm births and preterm low birth weights.


Despite high level evidence showing that antenatal smoking cessation programmes are effective in reducing the number of women who smoke during pregnancy and the number of low birthweight and preterm births, few Australian hospitals have adopted a systematic approach to assist pregnant women to stop smoking. This study aimed to assess the effectiveness of a smoking cessation guideline, developed specifically for clinicians providing antenatal care in public maternity hospitals, combined with an implementation programme on the uptake of evidence-based practice. A clinical practice guideline was developed and an implementation strategy was tested, using a prospective before-and-after study design. Women were surveyed in late pregnancy, pre- and post-implementation. The primary outcome measures were women's report of appropriate smoking cessation support received, specifically, information brochures and referral to Quitline. Secondary outcome measures included women's report of smoking status in late pregnancy and relapse rates. Post-implementation, more women reported receiving written materials on smoking cessation (76% vs. 35%; relative risk (RR) 3.4; 95% CI 2.7–4.2) and referral to Quitline (67% vs. 14%; RR 4.9; 95% CI 3.0–8.0). While not statistically significant, fewer women post-implementation reported smoking in late pregnancy (19.5% vs. 16.7%) and fewer reported smoking >10 cigarettes per day (38% vs. 25%). The authors concluded that clinical practice guidelines specifically designed for a public maternity care setting combined with an implementation programme resulted in an increase in evidence-based practice with some indication of improved smoking behaviour for women.


Asymptomatic bacteriuria is relatively common in pregnancy (2–10% of women) and, if this is untreated, about 30% of those affected will develop acute pyelonephritis. Asymptomatic bacteriuria has been associated with both low birthweight and preterm birth. This review aimed to assess the effect of antibiotic treatment on bacteriuria detected by screening in asymptomatic pregnant women. It included 14 RCTs of generally poor quality comparing antibiotics to placebo. Compared to placebo, antibiotics were effective at clearing symptomatic bacteriuria (risk ratio (RR) 0.25, 95% CI 0.14 to 0.48) and reducing the incidence of pyelonephritis (RR 0.23, 95% CI 0.13–0.41). Antibiotic treatment was also associated with a reduction in the proportion of low birthweight babies (RR 0.66, 95% CI 0.49–0.89) but not in the proportion of deliveries that were preterm. The review authors concluded that antibiotics were effective in reducing the risk of pyelonephritis in pregnancy and that, although the observed effect on reducing low birthweight is consistent with accepted theories about the role of infection in adverse pregnancy outcomes, this association should be viewed cautiously in view of the poor qualities of the included studies.


Progesterone has a role in maintaining pregnancy and the mechanism is thought to be inhibition of uterine smooth muscle contraction. It may be given either by intramuscular injection or as a vaginal pessary. There is little long term safety data and little information about the optimal dose, route of administration, gestation to begin therapy, or duration of therapy. This review included 36 RCTs (8523 women and 12,515 infants) involving comparison of progesterone vs. placebo in a variety of situations. In women with past history of spontaneous preterm birth progesterone was associated with a statistically significant reduction in the risk of preterm birth less than 34 weeks (average RR 0.31, 95% CI 0.14–0.69), preterm birth less than 37 weeks (average RR 0.55, 95% CI 0.42 to 0.74) and infant birthweight less than 2500 g (RR 0.58, 95% CI 0.42–0.79). Also noted was a statistically significant increase in pregnancy prolongation in weeks (mean difference (MD) 4.47, 95% CI 2.15–6.79). For women with a short cervix identified on ultrasound, progesterone was associated with a statistically significant reduction in the risk of preterm birth < 34 weeks (RR 0.64, 95% CI 0.45–0.90) and preterm birth at less than 28 weeks' gestation (RR 0.59, 95% CI 0.37–0.93). An increased risk of urticaria in women when compared with placebo was noted (RR 5.03, 95% CI 1.11–22.78). In women with a threatened labour, progesterone was associated with significant reduction in the risk of infant birthweight < 2500 grams (RR 0.52, 95% CI 0.28–0.98); in women with “other” risk factors for preterm birth progesterone was associated with a statistically significant reduction in the risk of infant birthweight less than 2500 g (RR 0.48, 95% CI 0.25–0.91). No statistically significant differences were noted in the reported outcomes for women with a multiple pregnancy. The authors state that further research is required to determine the optimal timing, mode of administration and dose of progesterone.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
CONGENITAL ANOMALIES
EVIDENT AT BIRTH
Antenatal and Newborn Screening

Overseas research suggests that up to 25% of babies with severe life-threatening forms of congenital heart disease are discharged from hospital undiagnosed [16]. In New Zealand, as in other countries, a small number of babies each year are born with inborn errors of metabolism (e.g. galactosaemia), which, if left untreated, may lead to permanent end organ damage within a relatively short period of time [17]. Even for non-life threatening conditions, delayed diagnosis may lead to the loss of opportunities for early intervention (e.g. congenital hearing loss: identified in the first three months with newborn screening vs. at an average age of 35.1 months if screening is based on the presence of risk factors [18]).

The early detection of these conditions thus confers significant advantages, and antenatal diagnosis also provides the opportunity to exclude additional congenital or chromosomal abnormalities, to discuss pregnancy options with parents, and to plan for delivery in a tertiary centre if additional services will be required [19]. For a number of conditions, however, (e.g. congenital deafness, inborn errors of metabolism where the placenta clears metabolites in-utero) antenatal diagnosis is not possible, and, in these cases, early detection in the neonatal period becomes of critical importance.

In New Zealand, a number of screening programmes have been established to detect congenital anomalies and inborn errors of metabolism in the antenatal period, or as soon as possible after birth. The following sections briefly review each of these in turn.

Screening During the Antenatal Period

Antenatal Screening for Down Syndrome and Other Conditions

Antenatal screening for Down syndrome and other conditions has been available to pregnant women since 1968 [20]. However, concerns during the mid-2000s that the existing screening processes were ad-hoc [21], led the National Screening Unit to release a set of guidelines for maternity providers in 2009 [20]. These guidelines recommended that all pregnant women be offered antenatal screening for Down syndrome and other conditions in either the first or second trimester of pregnancy as follows:

1. **For Women Presenting in their First Trimester**: A blood test that measures two maternal serum markers (pregnancy-associated plasma protein A (PAPP-A) and Beta-human chorionic gonadotrophin (βhCG)) should be combined with the results of an ultrasound which assesses nuchal translucency (a measurement of the fluid filled space in the tissue at the back of a fetus' neck which is a marker for chromosomal and other anomalies) and other parameters (e.g. crown-rump length). The optimal time for screening using maternal serum markers is 10–12 weeks, while the optimal time for an ultrasound to assess nuchal translucency is 11.5–13.5 weeks [20].

2. **For Women Presenting in their Second Trimester**: A blood test that measures four maternal serum markers (βhCG, alpha-fetoprotein, unconjugated oestriol and inhibin A), best done at 14–18 weeks [20].

In its updated 2012 Guidelines for Health Practitioners [22], the National Screening Unit outlines the expectation that health practitioners will provide accurate and non-directive information to women considering antenatal screening, including informing them of their right to decline screening or further investigations. After undergoing screening, all women who are deemed to be at a high risk of having a baby with Down syndrome or other conditions should be offered an obstetric referral to discuss diagnostic testing options including: chorionic villus sampling (usually performed at 10–13 weeks); and amniocentesis (usually performed at 15–20 weeks). Maternity providers should also advise women with an increased risk of the availability of genetic counselling services [22].

In addition, while they are not part of a formal screening programme, ultrasound examinations are frequently performed between 18–20 weeks of gestation to screen for
obvious structural anomalies, although such scans are thought not to be as effective for detecting Down Syndrome as the screening modalities listed above [20].

### Screening During the Neonatal Period

#### Newborn Examination
The Well Child/Tamariki Ora Schedule recommends that a detailed clinical examination be undertaken within 48 hours of birth (the initial examination is usually undertaken at birth), with a further clinical examination being undertaken within 7 days, and another at 4–6 weeks (at the time of discharge from maternity services) [23]. At the initial (newborn) examination the Schedule recommends that clinicians undertake a thorough assessment which includes: the child’s overall health and wellbeing, weight, length and head circumference, and a more detailed examination of their hips, cardiovascular system (heart, umbilicus, and femoral pulses), eyes (red reflex), colour, respiration, tone, Moro reflex, grasp reflex, movements, skin, head, fontanelles, ears, mouth, lungs, abdomen, umbilicus, genitalia, anus, spine, and limbs [24].

#### Newborn Metabolic Screening Programme
When New Zealand first commenced newborn metabolic screening in 1969, screening was undertaken only for phenylketonuria (PKU) [17]. The current Newborn Metabolic Screening Programme (NMSP), however, screens for 28 metabolic disorders [17]. These conditions are outlined in **Table 15**.

**Table 15. Conditions Included in New Zealand’s Newborn Metabolic Screening Programme**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital Hypothyroidism</td>
<td>1 in 4,000 babies (=15 babies a year)</td>
</tr>
<tr>
<td>Cystic Fibrosis</td>
<td>1 in 7,000 babies (= 8 babies a year)</td>
</tr>
<tr>
<td>Amino Acid Disorders (14 disorders including e.g. Phenylketonuria (PKU))</td>
<td>1 in 12,000 babies (= 5 babies a year)</td>
</tr>
<tr>
<td>Fatty Acid Oxidation Disorders (9 disorders including e.g. Medium Chain acyl-CoA Dehydrogenase Deficiency)</td>
<td>1 in 12,000 babies (= 5 babies a year)</td>
</tr>
<tr>
<td>Congenital Adrenal Hyperplasia</td>
<td>1 in 20,000 babies (= 3 babies a year)</td>
</tr>
<tr>
<td>Galactosemia</td>
<td>1 in 100,000 (= 1 baby every 2 years)</td>
</tr>
<tr>
<td>Biotinidase Deficiency</td>
<td>1 in 150,000 (= 1 baby every 3 years)</td>
</tr>
</tbody>
</table>


Lead Maternity Carers (LMCs) are responsible for undertaking newborn metabolic screening, with their tasks including giving information and advice, offering screening, ensuring informed consent, taking the sample and following up on the results. The National Screening Unit recommends that LMCs take samples when the baby is 48 hours old, or as soon as possible thereafter. Timing is important, as samples taken earlier (e.g. at the time of birth) may be negative due to the placenta having eliminated abnormal markers, while samples taken later may result in a lost window for early intervention, as severe forms of some metabolic disorders can be fatal within 7–10 days, although the baby may not show any signs or symptoms until irreversible damage has occurred [17]. Blood samples are usually taken by heel prick. Blood is collected onto a blood spot card, which has two main parts: a smaller portion with specimen collection paper for the sample itself, and a larger portion for demographic and other information [17]. When the sample is taken, parents are asked whether they wish the card to be stored for possible future use, or returned to them, after analysis.

#### National Newborn Hearing Screening Programme
In New Zealand each year, it is estimated that 135–170 babies are born with mild to profound permanent congenital hearing loss, representing an incidence of 3 per 1,000 births [25]. In response to concerns regarding the late age of diagnosis of congenital hearing losses, which occurred at average age of 35.1 months when screening was based on the presence of risk factors [18], the Government, in its 2006 Budget, announced a
funding package to establish a National Newborn Hearing Screening Programme ($16 million over four years). The Programme was rolled out progressively across the country during 2007–2010, and screening is now underway in all 20 DHBs [26]. For further detail see the Newborn Hearing Screening Section commencing on Page 147.

Conditions Detectable by Antenatal and Newborn Screening

This report reviews a number of conditions which are potentially detectable by antenatal or newborn screening. These include:

- Congenital Anomalies Evident at Birth (Page 85)
- Cardiovascular Anomalies Evident at Birth (Page 107)
- Down Syndrome (Page 117)
- Neural Tube Defects (Page 129)
- Newborn Hearing Screening (Page 145)
- Cystic Fibrosis (Page 208)

While local policy documents and evidence-based reviews relevant to these conditions are reviewed at the end of each chapter, Table 16 provides an overview of publications which consider antenatal and newborn screening more generally.

Local Policy Documents and Evidence-Based Reviews Relevant to Antenatal and Newborn Screening

In New Zealand there are a number of policy documents which provide guidance on antenatal and newborn screening. These are summarised in Table 16, along with a range of evidence-based reviews which consider these issues in the overseas context. Note: Publications which considered antenatal screening for maternal infectious disease status were seen as being outside of the scope of this review including HIV, syphilis, Hepatitis B, and rubella.

In addition, Table 29 on Page 102 considers publications relevant to congenital anomalies collectively, while Table 35 on Page 115 considers cardiovascular anomalies, Table 41 on Page 125 considers Down syndrome and other chromosomal anomalies, and Table 46 on Page 134 considers neural tube defects. Finally Table 53 on Page 150 provides a brief overview of publications relevant to newborn screening for congenital hearing loss.

Table 16. Local Policy Documents and Evidence-Based Reviews Relevant to Antenatal and Newborn Screening

<table>
<thead>
<tr>
<th>New Zealand Policy Documents and Publications</th>
</tr>
</thead>
<tbody>
<tr>
<td>These guidelines are intended for all practitioners who have involvement in any part of the antenatal screening process for Down syndrome and other conditions. Further detail on this publication is provided in the Down syndrome section.</td>
</tr>
<tr>
<td>The policy framework for the Newborn Metabolic Screening programme is set out in this document for the guidance for all programme providers. The nine sections in the framework cover background information, programme policy, the responsibilities of programme providers, lead maternity carers and laboratories, the return, storage and uses of residual blood spot samples, new technologies and changes to the disorder panel.</td>
</tr>
<tr>
<td>There are nine national indicators used to monitor the Newborn Metabolic Screening Programme. The Monitoring Framework sets out how these are used to assess the performance of the programme. There are tables for each indicator which include a description of the indicator, the rationale for the indicator, the relevant outcome, the standard against which the indicator is to be compared, and the methodology for calculating the indicator. There is also a summary table giving the reporting frequency and details for each of the indicators.</td>
</tr>
</tbody>
</table>
Lead Maternity Carers are contractually obliged to provide services within screening programmes endorsed by the Ministry of Health, including the Newborn Metabolic Screening Programme, under the Primary Maternity Services Notice 2007. These best practice guidelines are for all practitioners involved in the Newborn Metabolic Screening Programme including Lead Maternity Carers, hospital midwives, nurses and phlebotomists.

International Guidelines and Systematic and Other Reviews


In order to make an informed choice about whether to undergo screening, patients need information about the risk of having the condition being screened for, the nature of the condition, the advantages and disadvantages of screening and the accuracy of the screening tests. Personalised risk communication is the provision of risk information that is tailored for a specific person, based on characteristics such as age, family history, and cultural and/or educational background.

This review compared personalised vs. general risk communications for promoting informed decision making about screening participation. The review included 41 RCTs, only one of which related to antenatal screening (most related to breast or colorectal cancer). The authors concluded that there was strong evidence, from three trials, that inclusion of personalised risk estimates in screening programme information enhanced informed choice. They stated that the evidence that personalised information increased uptake of screening was weak and that it was unclear if increased uptake was associated with informed choice. Because of the diversity of the screening programmes, the authors were unable to draw any conclusions about the best ways to provide personalised risk communications.


Homocystinuria is a rare genetic disorder in which a deficiency of the enzyme cystathione beta synthetase causes raised levels of the amino acids homocysteine and methionine in the blood and tissues. Children with this disorder appear normal initially but later develop a number of severe health problems including learning difficulties, bone and eye problems and a high risk of blood clots. If started very early in life, dietary intervention can prevent the development of these complications. This review considered whether newborn screening for homocystinuria leads to clinical benefits compared to later diagnosis based on symptoms. The authors found no RCTs addressing this issue and therefore stated that they could not draw any conclusions based on controlled studies, but they stated that they did know of uncontrolled case series which supported the efficacy of newborn screening and early treatment for homocystinuria.


The use of a heel lance is the usual way blood samples are collected from neonates for screening purposes. There are easy to use automated heel piercing devices. Venepuncture is a procedure that requires some training and skill. This review considered whether venepuncture is a more effective and less painful method than heel lancing for obtaining blood samples from neonates. It included 6 studies, of variable quality, which were either RCTs or quasi-RCTs (478 babies in total) comparing pain responses (assessed by validated behavioural pain measures) in infants who had blood samples collected by venepuncture, with those in infants who had heel lancing. In some studies both groups of infants received a sweet tasting solution before the procedure. Meta-analysis of the data for 288 infants who did not receive a sweet tasting solution indicated a significant pain reduction in the venepuncture group vs. the heel lance group (Standard mean difference -0.76, 95% CI -1.00 to -0.54, I² = 0%). For the infants who did receive a sweet tasting solution, the difference was less but still significant: SMD - 0.38, 95% CI -0.69 to -0.07. Data from four studies (n= 254) yielded the risk difference for requiring more than one skin puncture for venepuncture vs. heel lance: (RD = -0.34 95% CI -0.43 to -0.25; I² = 97%) and gave a number needed to treat with venepuncture to avoid one repeat skin puncture of 3 (95% CI 2 to 4). The authors concluded that venepuncture performed by a skilled phlebotomist was the method of choice for collecting blood samples from term neonates and that sweet tasting solutions should be provided to reduce pain before both venepuncture and heel lancing.


This technical update from the Society of Obstetricians and Gynaecology Canada provides concise information on the benefits of fetal or perinatal autopsy, the consent process and the alternatives when the family decline a full autopsy in situations where there has been a prenatal diagnosis of non-chromosomal malformations followed by fetal loss, stillbirth or neonatal death. Autopsy is important for obtaining an accurate diagnosis of the cause of death, which is necessary for genetic counselling and may permit prenatal diagnosis for future pregnancies. The guidelines are based on a review of the published literature (restricted to systematic reviews, RCTs/controlled clinical trials and observational studies) and grey (unpublished) literature sourced from clinical practice guideline collections, national and international medical specialty societies, clinical trial registries and websites of health technology assessment and health technology assessment-related agencies. The quality of the evidence is graded and the strength of the recommendations are classified according to criteria adapted from the Canadian Taskforce on Preventive Healthcare.
This review considered whether routine ultrasound in early pregnancy has an effect on diagnosis of multiple pregnancies, fetal malformations, intervention rates, or the incidence of adverse fetal outcomes, compared to selective use of early pregnancy ultrasound (for particular indications). It found that there was good evidence that routine ultrasound improved the detection of multiple pregnancies before 24 weeks’ gestation and was associated with a reduction in inductions for “post term” pregnancy. Only two of the 11 RCTs addressed the diagnosis of fetal malformations (the RADIUS study from the U.S. and the Helsinki ultrasound trial). In the 17,158 pregnancies in these two studies, there were 387 congenital abnormalities reported, with most of these (346, 89%) not detected at 24 weeks. Those who received ultrasound screening were more likely than control groups to have fetal abnormalities detected by 24 weeks (unweighted percentages 16% vs. 4%, risk ratio 3.46, 94% CI 1.67 to 7.14). The Helsinki trial showed better fetal abnormality detections rates than RADIUS, leading to increased terminations and reductions in perinatal mortality. The authors noted that considerable expertise is needed to detect fetal malformations using ultrasound and that since these two studies were done (in the 1990s) there have been advances in equipment and expertise which mean that the results of these trials are probably not relevant to the current situation.


This review compared high and low feedback provided to women receiving prenatal ultrasound scans. During high feedback scans, women can see images of their fetus on a screen and they receive detailed explanations of the images, while during low feedback scans women do not view the screen and are told the results of the scan at the end of the procedure. The review included four RCTs (365 women). The authors concluded that there was insufficient evidence to determine whether high or low feedback during scans is better for reducing maternal anxiety and promoting maternal health behaviour such as stopping smoking or drinking.


Women tend to experience anxiety while waiting for prenatal test results. This review considered whether providing amniocentesis or chorionic villus sampling (CVS) results on a fixed date as opposed to “when available” altered maternal anxiety. It also considered whether providing early results from a rapid molecular test altered maternal anxiety and whether the method of communication (e.g. phone, fax, email, in person) made a difference to parents’ satisfaction and anxiety levels. The authors identified two randomised trials (286 women in total) which compared the impact of receiving early results from rapid testing with waiting, on average for 18 days, for definitive karyotype results. One study reported a significant difference and the other did not. The authors concluded that there was no conclusive evidence that issuing early results from rapid testing while awaiting karyotype results reduced maternal anxiety nor was there any evidence to support the view that it is better to issue amniocentesis results as soon as they are available rather than on a pre-specified date. They stated that studies on the different methods of communicating test results are needed.


In the U.K. there is newborn screening for both cystic fibrosis (CF) and sickle cell anaemia. New Zealand does not screen for sickle cell anaemia as relatively few people belong to ethnic groups with high rates of the condition (mostly those of African descent). Screening for CF is a 2-stage process. All blood spot samples are tested for levels of immunoreactive trypsin and then those with levels above the 99th percentile undergo DNA testing for the most common CF mutations. A child with two CF mutations is likely to have CF, while a child with one is probably a carrier. Although the testing process is not designed to test for carrier status and aims to identify a minimum number of carriers, nevertheless the end result of the testing process is that some babies are identified as carriers of CF (indicating that at least one parent is almost certainly a carrier). This information has implications for the child (in adulthood) and the parents regarding their risk of having a child with CF. This study explored the practice, methods and experiences related to communicating carrier status following newborn screening in the U.K. It found that there was considerable variation from place to place and that parents’ needs for timely and appropriate information were not always met. It recommended that professionals involved in testing receive guidance in communication and that notification of carrier status should be done in person by a well-trained professional.
The use of tandem mass spectrometry (TMS) has made it possible to test for multiple conditions using the same blood spot and so the number of diseases that are potentially detectable by newborn screening has increased considerably. These guidelines from the National Academy of Clinical Biochemistry (NACB) in the U.S. cover the evidence–based rationale for expanded newborn screening (a summary table gives details on 47 inborn errors of amino acid, fatty acid, or organic acid metabolism; testing for most of these is recommended by the NACB), technical and analytical issues relating to follow-up testing of newborns with positive screens, disease-specific follow-up testing recommendations, patient outcomes from early diagnosis by expanded newborn screening, and future directions in the field. It is acknowledged that in some conditions, such as maple syrup urine disease, infants may become severely ill before screening results are available and in others there may be no clinical evidence of disease despite the identified metabolic abnormality. The NACB has ranked diseases that can be detected by TMS according to the strength of evidence for improved patient outcomes in neonatally-detected patients (in descending order): medium chain acyl-CoA dehydrogenase deficiency (MCAD: A-I: 16–18), maple syrup urine disease (MSUD: A-I: 19), glutaric acidemia type 1 (GA-1: A-I: 20–23), and the so-called “classical” organic acidemias, propionic acidemia, methylmalonic acidemia, and isovaleric acidemia (A-II: 11). Within the guidelines the NACB has graded the quality of the overall evidence on a three point scale and determined the strength of the recommendations using criteria modified from those in the US Preventive Services Task Force Recommendations for Preventive Services.

Screening for Congenital Hypothyroidism: A Literature Update for the US Preventive Services Task Force


The literature review which forms the basis for the latest USPSTF recommendations is: Meyers D, Haering S. March 2008. Screening for Congenital Hypothyroidism: A Literature Update for the US Preventive Services Task Force. The Annals of Family Medicine, 6(2), 166. http://www.annfammed.org/content/suppl/2008/03/14/6.2.166-a.DC1/TorchiaHypothyroid1Online_RecStat-rev.pdf

Congenital hypothyroidism affects one in every 3000–4000 newborns. Without treatment, it can result in mental retardation, growth failure and neuropsychological complications including motor abnormalities, learning disabilities and speech disorders. The US Preventive Services Task Force (USPSTF) has previously published (in 1996) recommendations for screening for congenital hypothyroidism which had a strong evidence base. This reaffirmation recommendation statement from the USPSTF is the result of a targeted review of the medical literature from January 1, 1995, to September 14, 2006, which aimed to find new, high-quality evidence about the benefits and potential harms of screening for congenital hypothyroidism. The USPSTF gives newborn screening for congenital hypothyroidism as an “A” grade recommendation. A clinical summary of the USPSTF recommendations, intended for primary care clinicians, can be found here: http://www.uspreventiveservicestaskforce.org/uspstf08/conhyp/conhypsum.htm


This review notes that since the benefits of early treatment of congenital hypothyroidism are well-established, there have been no new RCTs on screening for this disorder. Recent studies have focused on identifying the optimal timing and dosage of thyroid replacement therapy. The authors identified three studies reporting on harms associated with screening for hypothyroidism: two on false positive rates and one on the effects of a false positive test result on family dynamics. There are wide variations in estimates of false positive rates and different programmes use different cut-off points for defining a positive test. Receiving a false positive test result may be stressful for parents. Parental anxiety may be reduced with education and good communication from health professionals.

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These British guidelines state that a basic physical examination of the newborn should be done routinely immediately after birth and that it is accepted as good practice (and recommended by the National Screening Committee) that a more thorough physical examination is performed within 72 hours after delivery in order to either reassure parents that their baby is normal if no abnormality is detected, or to act promptly if any abnormalities are detected. The guidelines include a concise outline of what should be included in the newborn physical examination and the specific recommendations of the National Screening Committee. It is stated that the examination at 6 to 8 weeks (the time of the first immunisations) should repeat the assessments made at the postnatal examination and also include an assessment of the baby’s social smile and visual fixation and following. The guidelines state that “there is no high level evidence base for the conduct and content of the physical examination of the newborn” and that therefore the guideline’s examination recommendations are based on expert opinion and good practice.

Diagnostic ultrasound is used in late pregnancy where there are specific clinical indications such as poor fetal growth or antepartum haemorrhage. There is debate about whether ultrasound screening for all women in late pregnancy is of value. For such screening to be useful it needs to be able to detect conditions that place the mother or the fetus at high risk of an adverse outcome and that could not be detected by other means and for which there are effective treatments or management strategies that improve perinatal outcomes. This review includes eight trials (RCTs and quasi-RCTs, involving a total of 27,024 women and of satisfactory quality overall) of routine ultrasound in late pregnancy to assess some or all of the following: fetal size, presentation or anatomy, placental site or grading and amniotic fluid volume. No differences were found between the intervention and control groups in antenatal, obstetric or neonatal interventions or in morbidity. Caesarean section rates were a little higher in the screened group but the difference was not statistically significant. Overall perinatal mortality was no better in the screened group. One trial assessed placental grading as an adjunct to third trimester ultrasound examination and found that it was associated with a significant reduction in the stillbirth rate. The authors found that there was limited information on long term outcomes such as neurodevelopment and no data on maternal psychological effects. They concluded that, based on existing evidence, routine ultrasound in late pregnancy for unselected or low-risk populations does not have benefits for mother or baby.

This literature survey was commissioned by the Fetal Anomalies steering group on behalf of the UK National Screening Committee. It relates to routine mid-trimester ultrasound screening and its main purpose was to populate tables relating to the detection rates, false positives and frequencies of a specified list of anomalies, organised under the following headings: Central Nervous System (CNS), Cardio Vascular System (CVS), Chest, Abdomen, Renal, Limbs and Face. A list of more recent papers from a November 2010 literature search requested by the NHS fetal anomaly screening programme can be found here: http://fetalanomaly.screening.nhs.uk/getdata.php?id=11283.

This review assessed the effectiveness of preimplantation screening (PGS) in women undergoing in vitro fertilisation (IVF) or intracytoplasmatic sperm injection (ICSI) because of infertility. Such screening ensures that only embryos with a normal number of chromosomes (for the chromosomes tested) are implanted, in the hope that this will improve pregnancy rates. Nine RCTs were included in the review. IVF/ICSI with PGS was associated with significantly lower live birth rates than IVF/ICSI without PGS in women of advanced maternal age (5 studies, OR 0.59; 95% CI 0.44 to 0.81) and in women with repeated IVF failure (1 study, OR 0.41, 95% CI 0.20 to 0.88). In good prognosis patients (3 studies) the same trend was apparent but it was not significant (OR 0.50, 95% CI 0.20 to 1.26, random effects model). The authors noted that new techniques for PGS are being developed but they state that until these new developments have been properly evaluated PGS should not be offered in any form as part of routine patient care.

This review considered 106 publications relating to psychosocial aspects of screening in pregnancy for genetic disorders (plus a few non-genetic disorders particularly neural tube defects and congenital hypothyroidism). It aimed to address questions in the following areas: knowledge, anxiety, factors associated with participation (or not) in screening programmes, and the long term sequelae of false positive, true positive (in newborns only) and true negative results. The authors considered that the most important issues which antenatal and neonatal screening programmes needed to address were in order of priority: the inadequacy of current ways of achieving informed consent, the cost of providing a satisfactory service, the unmet needs of patients who receive false positive screening results and the unmet needs of women’s partners particularly in carrier screening.
## Other Relevant Publications and Websites

<table>
<thead>
<tr>
<th>Publication/Website</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>NHS non-invasive prenatal testing and diagnostic services for pregnancy. 2013. How does NIPT work? <a href="http://www.rapid.nhs.uk/">http://www.rapid.nhs.uk/</a> accessed April 2013.</td>
<td>In the U.K., the National Institute for Health Research has funded a 5-year national research programme, the RAPID programme (Reliable Accurate Prenatal non-Invasive Diagnosis). This research programme aims to improve the quality of NHS prenatal diagnostic services by evaluating early non-invasive prenatal diagnosis (NIPD) based on cell free fetal (cff) DNA and RNA in maternal plasma. Currently in the U.K., NIPD testing is available for sex determination where there are clinical indications and for some skeletal dysplasias e.g. achondroplasia and thanatophoric dysplasia. The RAPID website has a range of resources and publications relating to non-invasive prenatal diagnosis.</td>
</tr>
<tr>
<td>UK National Screening Committee. 2013. UK NSC Policy Database. <a href="http://www.screening.nhs.uk/policydb.php">http://www.screening.nhs.uk/policydb.php</a> accessed April 2013.</td>
<td>This website provides links to all the policies of the UK National Screening Committee (UK NSC). The policies are reviewed regularly on a three year cycle. The policy pages for each condition include expert reviews, the evidence base for the current policy and links to relevant publications. The UK NSC currently recommends screening in the antenatal period for the following conditions affecting babies: Down syndrome, fetal anomalies, maternal hepatitis B, syphilis, HIV, sickle cell and thalassaemia, neural tube defect and maternal rubella susceptibility. Screening is not recommended in the antenatal period for cystic fibrosis, cytomegalovirus, familial dysautonomia, fragile X, maternal HTLV, maternal hepatitis C, Tay Sachs disease, thrombophilia, toxoplasmosis and maternal varicella (chickenpox) susceptibility. For newborns, screening is recommended for congenital cataracts, heart disease and hypothyroidism, cryptorchidism (undescended testes), cystic fibrosis, developmental dislocation of the hip, hearing loss, Medium Chain Acyl CoA Dehydrogenase Deficiency, PKU and sickle cell disease. It is not recommended for amino acid metabolism disorders, biliary atresia, biotinidase deficiency, Canavan’s disease, congenital adrenal hyperplasia, Duchenne muscular dystrophy, fatty acid oxidation disorders, galactosaemia, Gaucher’s disease, kernicterus, neuroblastoma, organic acid metabolism disorders and thrombophilia.</td>
</tr>
<tr>
<td>Wilson C, Kerruish NJ, Wilcken B, et al. 2012. Diagnosis of disorders of intermediary metabolism in New Zealand before and after expanded newborn screening: 2004-2009. New Zealand Medical Journal, 125(1348), 42–50.</td>
<td>This paper reports on a study comparing diagnosis rates for inborn errors of intermediary metabolism (IEMS) for two three year periods, before and after in commencement of expanded newborn screening (ENBS) using tandem mass spectrometry in December 2006. In the three years prior to December 2006 there were 15 patients diagnosed. In the three years from December 2006 42 cases were diagnosed, 30 of these by EBNS.</td>
</tr>
<tr>
<td>Kerruish NJ, Webster D, Dickson N. 2008. Information and consent for newborn screening: practices and attitudes of service providers. Journal of Medical Ethics, 34(9), 648-52.</td>
<td>This New Zealand study, undertaken in 2002 involved sending a questionnaire concerning provision of information and parental consent for newborn screening (NBS) to all lead maternity carers (LMCs). 93% of LMCs reported giving mothers information about NBS, mostly after delivery (73%) and in the third trimester (60%). Most (85%) LMCs get either verbal or written consent from parents for NBS and 94% consider this to be the ideal approach although 23% of LMCs thought NBS should be mandatory. Of those LMCs who believed NBS should be mandatory, most still believed parental consent should be obtained (89%) and of those who believed NBS should not be mandatory only 10% would accept parental refusal without question. The study authors stated that the survey results indicated a consensus that parents should be provided with good quality information about NBS but that there was less agreement on how much parents should be involved in the decision to allow babies to undergo NBS. They stated that a policy that strongly recommends NBS but also permits parental choice seems to be most consistent with the views of the surveyed LMCs.</td>
</tr>
</tbody>
</table>

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
**Introduction**

Congenital anomalies (sometimes referred to as birth defects or congenital malformations), range in severity from minor conditions that are of no functional or cosmetic importance, to conditions that are incompatible with life. There are a large number of rare syndromes which are characterised by multiple congenital anomalies [27]. Congenital anomalies are one of the leading causes of fetal and infant deaths in New Zealand [28].

In New Zealand, the NZ Birth Defects Registry (NZBDR) has collected data on all babies with a diagnosed birth defect born or treated in a public hospital since 1977 [29]. The NZBDR contributes New Zealand data on 39 categories of birth defects to the International Clearinghouse for Birth Defects Surveillance Research (ICBDSR) based in Rome [30]. New Zealand data published in the latest (2010) ICBDSR report indicated an overall rate of congenital anomalies for 2004–2008 of around two anomalies per 100 births [30]. The Plunket National Child Health Study of 4286 children born in 1990–1 reported an overall prevalence of birth defects at six weeks of age of 4.3% [31].

Other developed countries have reported similar figures [32,33,34]. The 2008 report from the Victorian Perinatal Data Collection Unit states that combined data from 2005–2006 showed that there was a birth defect in 4.2% of all births in Victoria [33]. EUROCAT (European Surveillance of Congenital Anomalies) collates data from 38 registries in 21 European countries. It collects information on major congenital anomalies that require surgical treatment, have serious effects on health or development, or have significant cosmetic impact. In 2010 the live birth prevalence of all EUROCAT anomalies was 176 per 10,000 (1.76%) [35].

The following section uses the National Minimum Dataset to review the number of congenital anomalies evident at birth, as well as the number of babies born with one or more congenital anomalies. Subsequent chapters consider cardiovascular anomalies, Down syndrome and neural tube defects in more detail. Note: In reviewing this data, it is important to remember that the analysis includes all congenital anomalies in the ICD-10-AM Q00–Q99 range (structural and chromosomal anomalies but not metabolic disorders), irrespective of whether they were minor (e.g. skin tags) or major (e.g. spina bifida). For this reason the overall prevalence estimates presented here may be higher than comparable overseas estimates (which may have included only major anomalies).

---

**Data Source and Methods**

**Definition**

1. *Number of congenital anomalies identified at birth (by anomaly type)*
2. *Number of babies with one or more congenital anomalies identified at birth*

**Data Source**

1. National Minimum Dataset

   **Numerator:** Hospital admissions with event type = birth and a congenital anomaly (Q00–Q99) listed in any of the first 15 diagnoses.

   For this indicator, the unit of analysis is the number of congenital anomalies rather than the number of babies, with many babies having more than one anomaly.

   For a list of the ICD-10-AM codes used to assign anomaly type see Appendix 8.

2. National Minimum Dataset

   **Numerator:** Hospital admissions with event type = birth and a congenital anomaly (Q00–Q99) listed in any of the first 15 diagnoses.

   For this indicator, the unit of analysis is the number of babies with one or more congenital anomalies.

   **Denominator:** All hospital admissions with and event type = birth
Notes on Interpretation

Note 1: This analysis includes all admissions recorded in the National Minimum Dataset (NMDS) where the Event Type was listed as a Birth. In the NMDS only one birth event is allowed per NHI number, with babies born prior to hospital admission, or readmitted shortly after discharge, being listed as a routine inpatient event. Thus the analysis excludes babies born prior to hospital admission, babies born at home, or babies whose congenital anomaly was overlooked at the time of initial discharge, but who re-presented shortly thereafter.

Note 2: This analysis is likely to significantly undercount those conditions where the congenital or chromosomal anomaly usually only becomes evident at a later age, when the child fails to achieve their normal developmental milestones (e.g. many chromosomal or CNS anomalies), or where the condition may be difficult to detect on routine newborn examination.

Note 3: Because of the large number of ICD-10-AM diagnoses in the Q00–Q99 range, and the lack of additional supporting information, no attempt has been made to grade the severity of the congenital anomalies identified. The reader must thus bear in mind that in this analysis, minor anomalies such as skin tags, and anomalies which may (in some cases) be considered part of normal physiological development (e.g. isolated patent ductus arteriosus in preterm babies), have been counted equally alongside more serious anomalies such as spina bifida and Tetralogy of Fallot. Thus when considering the overall impact of congenital anomalies on children’s subsequent developmental trajectories, or on future health service demand, it is necessary to consider the data presented on an anomaly by anomaly basis.

Note 4: In the New Zealand level analyses, large reductions in congenital anomaly rates are seen between 2007 and 2009, with rates then reverting to their pre-existing baseline by around 2012. It remains unclear however, whether these changes reflect real changes in the number of babies born with congenital anomalies, changes in the comprehensiveness of the recording of minor congenital anomalies by clinicians, or changes in the way in which congenital anomalies were coded in the hospital admission dataset.

New Zealand Distribution and Trends

New Zealand Distribution

In New Zealand during 2008–2012, a large number of congenital anomalies were identified at the time of birth, with these ranging in severity from minor skin conditions (e.g. skin tags, non-neoplastic nevus), through to anomalies which were incompatible with life (e.g. anencephaly). When interpreting the information in Table 17 and Table 18, it must be remembered that the figures presented relate to the number of anomalies identified, rather than the number of babies, and that many babies have more than one anomaly.

New Zealand Trends

In New Zealand, the number of babies with one or more congenital anomalies identified at birth increased gradually during the early 2000s, reached a peak in 2007 and then declined, with the most rapid declines occurring between 2007 and 2009. Rates then gradually increased again, reaching 5.5% of births by 2012 (Figure 13). It is unclear, however, whether the large declines seen between 2007 and 2009, and their subsequent rebound, reflect real changes in the number of babies born with congenital anomalies, changes in the comprehensiveness of the recording of minor congenital anomalies by clinicians, or changes in the way in which congenital anomalies were coded in the hospital admission dataset.

Distribution by Maternal Age

In New Zealand during 2008–2012, while the largest absolute numbers of babies with congenital anomalies were born to women aged 30–34 years, congenital anomaly rates rose steadily with increasing maternal age, with the highest rates being seen in the babies of mothers aged 40+ years. The babies of mothers aged 40+ years had congenital anomaly rates that were 1.32 (95% CI 1.20–1.44) times higher than the babies of teenage mothers (Figure 14, Table 19).
### Table 17. Congenital Anomalies Evident at Birth, New Zealand 2008–2012 (Table 1 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anencephaly</td>
<td>13</td>
<td>2.6</td>
<td>4.3</td>
</tr>
<tr>
<td>Encephalocele</td>
<td>13</td>
<td>2.6</td>
<td>4.3</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>61</td>
<td>12.2</td>
<td>20.2</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>77</td>
<td>15.4</td>
<td>25.5</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>190</td>
<td>38.0</td>
<td>62.9</td>
</tr>
<tr>
<td>Spina Bifida</td>
<td>53</td>
<td>10.6</td>
<td>17.5</td>
</tr>
<tr>
<td>Other Spinal Cord Malformations</td>
<td>14</td>
<td>2.8</td>
<td>4.6</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>24</td>
<td>4.8</td>
<td>7.9</td>
</tr>
<tr>
<td><strong>Total Malformations of the Nervous System</strong></td>
<td><strong>445</strong></td>
<td><strong>89.0</strong></td>
<td><strong>147.2</strong></td>
</tr>
<tr>
<td>Eyelid/Lacrimal/Eye/Orbit Malformations</td>
<td>105</td>
<td>21.0</td>
<td>34.7</td>
</tr>
<tr>
<td>Ear Malformations Impairing Hearing</td>
<td>19</td>
<td>3.8</td>
<td>6.3</td>
</tr>
<tr>
<td>Accessory Auricle</td>
<td>255</td>
<td>51.0</td>
<td>84.4</td>
</tr>
<tr>
<td>Other Ear Malformations</td>
<td>181</td>
<td>36.2</td>
<td>59.9</td>
</tr>
<tr>
<td>Other Face/Neck Malformations</td>
<td>62</td>
<td>12.4</td>
<td>20.5</td>
</tr>
<tr>
<td><strong>Total Malformations of Eye, Ear, Face and Neck</strong></td>
<td><strong>622</strong></td>
<td><strong>124.4</strong></td>
<td><strong>205.7</strong></td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>190</td>
<td>38.0</td>
<td>62.9</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>533</td>
<td>106.6</td>
<td>176.3</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>588</td>
<td>117.6</td>
<td>194.5</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>42</td>
<td>8.4</td>
<td>13.9</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>92</td>
<td>18.4</td>
<td>30.4</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>185</td>
<td>37.0</td>
<td>61.2</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>111</td>
<td>22.2</td>
<td>36.7</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>246</td>
<td>49.2</td>
<td>81.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>1,490</td>
<td>298.0</td>
<td>492.9</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>313</td>
<td>62.6</td>
<td>103.5</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>52</td>
<td>10.4</td>
<td>17.2</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>165</td>
<td>33.0</td>
<td>54.6</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>15</td>
<td>3.0</td>
<td>5.0</td>
</tr>
<tr>
<td><strong>Total Malformations of the Circulatory System</strong></td>
<td><strong>4,022</strong></td>
<td><strong>804.4</strong></td>
<td><strong>1,330.4</strong></td>
</tr>
<tr>
<td>Nose Malformations</td>
<td>44</td>
<td>8.8</td>
<td>14.6</td>
</tr>
<tr>
<td>Larynx Malformations</td>
<td>105</td>
<td>21.0</td>
<td>34.7</td>
</tr>
<tr>
<td>Trachea/Bronchus Malformations</td>
<td>32</td>
<td>6.4</td>
<td>10.6</td>
</tr>
<tr>
<td>Lung Malformations</td>
<td>118</td>
<td>23.6</td>
<td>39.0</td>
</tr>
<tr>
<td>Other Respiratory Malformations</td>
<td>3</td>
<td>0.6</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>Total Malformations of the Respiratory System</strong></td>
<td><strong>302</strong></td>
<td><strong>60.4</strong></td>
<td><strong>99.9</strong></td>
</tr>
<tr>
<td>Ankyloglossia (Tongue Tie)</td>
<td>3,699</td>
<td>739.8</td>
<td>1,223.6</td>
</tr>
<tr>
<td>Tongue/Mouth/Pharynx Malformations</td>
<td>79</td>
<td>15.8</td>
<td>26.1</td>
</tr>
<tr>
<td>Oesophagus/Upper Alimentary Malformations</td>
<td>80</td>
<td>16.0</td>
<td>25.6</td>
</tr>
<tr>
<td>Intestinal Malformations</td>
<td>270</td>
<td>54.0</td>
<td>89.3</td>
</tr>
<tr>
<td>Other Digestive Malformations</td>
<td>42</td>
<td>8.4</td>
<td>13.9</td>
</tr>
<tr>
<td><strong>Total Malformations of the Digestive System</strong></td>
<td><strong>4,170</strong></td>
<td><strong>834.0</strong></td>
<td><strong>1,379.3</strong></td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly
### Table 18. Congenital Anomalies Evident at Birth, New Zealand 2008–2012 (Table 2 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleft Palate</td>
<td>186</td>
<td>37.2</td>
<td>61.5</td>
</tr>
<tr>
<td>Cleft Lip</td>
<td>68</td>
<td>13.6</td>
<td>22.5</td>
</tr>
<tr>
<td>Cleft Palate and Lip</td>
<td>116</td>
<td>23.2</td>
<td>38.4</td>
</tr>
<tr>
<td><strong>Total Cleft Lip and Palate</strong></td>
<td><strong>370</strong></td>
<td><strong>74.0</strong></td>
<td><strong>122.4</strong></td>
</tr>
<tr>
<td>Female Genital Malformations</td>
<td>64</td>
<td>12.8</td>
<td>21.2</td>
</tr>
<tr>
<td>Undescended Testicle</td>
<td>735</td>
<td>147.0</td>
<td>243.1</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>576</td>
<td>115.2</td>
<td>190.5</td>
</tr>
<tr>
<td>Other Male Genital Malformations</td>
<td>157</td>
<td>31.4</td>
<td>51.9</td>
</tr>
<tr>
<td>Indeterminate Sex/Pseudohermaphrodism</td>
<td>24</td>
<td>4.8</td>
<td>7.9</td>
</tr>
<tr>
<td><strong>Total Malformations of the Genital Organs</strong></td>
<td><strong>1,556</strong></td>
<td><strong>311.2</strong></td>
<td><strong>514.7</strong></td>
</tr>
<tr>
<td>Renal Agenesis/Reduction Defects</td>
<td>94</td>
<td>18.8</td>
<td>31.1</td>
</tr>
<tr>
<td>Cystic Kidney Disease</td>
<td>144</td>
<td>28.8</td>
<td>47.6</td>
</tr>
<tr>
<td>Renal Pelvis Obstruction/Ureter Malformations</td>
<td>403</td>
<td>80.6</td>
<td>133.3</td>
</tr>
<tr>
<td>Other Kidney/Urinary Malformations</td>
<td>376</td>
<td>75.2</td>
<td>124.4</td>
</tr>
<tr>
<td><strong>Total Malformations of the Urinary System</strong></td>
<td><strong>1,017</strong></td>
<td><strong>203.4</strong></td>
<td><strong>336.4</strong></td>
</tr>
<tr>
<td>Congenital Dislocation/Subluxation Hip</td>
<td>60</td>
<td>12.0</td>
<td>19.9</td>
</tr>
<tr>
<td>Other Deformities Hip</td>
<td>461</td>
<td>92.2</td>
<td>152.5</td>
</tr>
<tr>
<td>Foot Deformities</td>
<td>1,516</td>
<td>303.2</td>
<td>501.5</td>
</tr>
<tr>
<td>Polydactyly</td>
<td>279</td>
<td>55.8</td>
<td>92.3</td>
</tr>
<tr>
<td>Syndactyly</td>
<td>106</td>
<td>21.2</td>
<td>35.1</td>
</tr>
<tr>
<td>Reduction Defects/Other Limb Malformations</td>
<td>177</td>
<td>35.4</td>
<td>58.6</td>
</tr>
<tr>
<td>Skull/Facial Bones/Spine/Thorax Malformations</td>
<td>214</td>
<td>42.8</td>
<td>70.8</td>
</tr>
<tr>
<td>Other Musculoskeletal Malformations</td>
<td>532</td>
<td>106.4</td>
<td>176.0</td>
</tr>
<tr>
<td>Osteochondrodysplasia</td>
<td>35</td>
<td>7.0</td>
<td>11.6</td>
</tr>
<tr>
<td><strong>Total Malformations of the Musculoskeletal System</strong></td>
<td><strong>3,380</strong></td>
<td><strong>676.0</strong></td>
<td><strong>1,118.0</strong></td>
</tr>
<tr>
<td>Ichthyosis</td>
<td>8</td>
<td>1.6</td>
<td>2.7</td>
</tr>
<tr>
<td>Non-Neoplastic Naevus</td>
<td>451</td>
<td>90.2</td>
<td>149.2</td>
</tr>
<tr>
<td>Epidermolysis Bullosa</td>
<td>11</td>
<td>2.2</td>
<td>3.6</td>
</tr>
<tr>
<td>Other Skin Malformations</td>
<td>450</td>
<td>90.0</td>
<td>148.9</td>
</tr>
<tr>
<td>Breast Malformations</td>
<td>15</td>
<td>3.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Other Integument Malformations</td>
<td>434</td>
<td>86.8</td>
<td>143.6</td>
</tr>
<tr>
<td>Other Malformations</td>
<td>396</td>
<td>79.2</td>
<td>131.0</td>
</tr>
<tr>
<td><strong>Total Other Congenital Malformations</strong></td>
<td><strong>1,765</strong></td>
<td><strong>353.0</strong></td>
<td><strong>583.8</strong></td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>257</td>
<td>51.4</td>
<td>85.0</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>40</td>
<td>8.0</td>
<td>13.2</td>
</tr>
<tr>
<td>Other Autosomal Trisomies</td>
<td>15</td>
<td>3.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Turners Syndrome</td>
<td>10</td>
<td>2.0</td>
<td>3.3</td>
</tr>
<tr>
<td>Sex Chromosome Anomalies Male Phenotype</td>
<td>12</td>
<td>2.4</td>
<td>4.0</td>
</tr>
<tr>
<td>Monosomies/Autosomal Deletions/Rearrangements</td>
<td>25</td>
<td>5.0</td>
<td>8.3</td>
</tr>
<tr>
<td>Other Chromosome Anomalies</td>
<td>33</td>
<td>6.6</td>
<td>10.9</td>
</tr>
<tr>
<td><strong>Total Chromosomal Anomalies</strong></td>
<td><strong>392</strong></td>
<td><strong>78.4</strong></td>
<td><strong>129.7</strong></td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly.
Figure 13. Babies with Congenital Anomalies Evident at Birth, New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a congenital anomaly listed in any of first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more anomalies, rather than number of anomalies

Figure 14. Babies with Congenital Anomalies Evident at Birth by Maternal Age, New Zealand Hospital Births 2008–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a congenital anomaly listed in any of first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more anomalies, rather than number of anomalies
Distribution by Prioritised Ethnicity, NZDep Index Decile and Gender

In New Zealand during 2008–2012, the proportion of babies with one or more congenital anomalies identified at birth was significantly higher for males, Asian/Indian and Pacific > European/Other > Māori babies, and those from the least deprived (NZDep06 deciles 1–2 vs. 5–10) areas (Table 19). Similarly, congenital anomaly rates were generally higher for Asian/Indian and Pacific babies, than for European/Other and Māori babies during 2000–2012 (Figure 15).

Table 19. Babies with Congenital Anomalies Evident at Birth by Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of Babies: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Babies with Congenital Anomalies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Prioritised Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>559</td>
<td>4,190</td>
<td>0.92</td>
<td>0.88–0.96</td>
</tr>
<tr>
<td>Pacific</td>
<td>339</td>
<td>5,003</td>
<td>1.09</td>
<td>1.04–1.15</td>
</tr>
<tr>
<td>European/Other</td>
<td>1,516</td>
<td>4,573</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>378</td>
<td>5,528</td>
<td>1.21</td>
<td>1.15–1.27</td>
</tr>
<tr>
<td><strong>NZ Deprivation Index</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>427</td>
<td>5,035</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>432</td>
<td>4,790</td>
<td>0.95</td>
<td>0.90–1.01</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>516</td>
<td>4,584</td>
<td>0.91</td>
<td>0.86–0.96</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>651</td>
<td>4,618</td>
<td>0.92</td>
<td>0.87–0.97</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>774</td>
<td>4,420</td>
<td>0.88</td>
<td>0.83–0.92</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1,116</td>
<td>3,797</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1,690</td>
<td>5,441</td>
<td>1.43</td>
<td>1.39–1.48</td>
</tr>
<tr>
<td><strong>Maternal Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 Years</td>
<td>195</td>
<td>4,662</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>20–24 Years</td>
<td>471</td>
<td>4,216</td>
<td>0.90</td>
<td>0.84–0.97</td>
</tr>
<tr>
<td>25–29 Years</td>
<td>646</td>
<td>4,354</td>
<td>0.93</td>
<td>0.87–1.00</td>
</tr>
<tr>
<td>30–34 Years</td>
<td>775</td>
<td>4,640</td>
<td>1.00</td>
<td>0.93–1.07</td>
</tr>
<tr>
<td>35–39 Years</td>
<td>555</td>
<td>5,098</td>
<td>1.09</td>
<td>1.02–1.17</td>
</tr>
<tr>
<td>40+ Years</td>
<td>165</td>
<td>6,137</td>
<td>1.32</td>
<td>1.20–1.44</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a congenital anomaly listed in any of first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more anomalies, rather than number of anomalies; Rate Ratios are unadjusted.
Northern DHBs Distribution and Trends

Northern DHBs Distribution

In the Northern DHBs during 2008–2012, a large number of congenital anomalies were identified at birth, with these ranging in severity from minor (e.g. tongue tie) through to serious (e.g. malformations of the great arteries) (Table 20 to Table 27).

When the number of babies with one or more congenital anomalies, rather than the number of anomalies was considered, on average during 2008–2012, 111 Northland, 496 Waitemata, 366 Auckland and 399 Counties Manukau babies per year (range 4.6–6.5% of all births) had one or more congenital anomalies identified at birth. Rates in Counties Manukau were similar to the New Zealand rate, while rates in the Northland, Waitemata, and Auckland DHBs were significantly higher (Table 28).

Note: It remains unclear whether DHB vs. NZ differences in congenital anomaly rates reflect real differences in the underlying prevalence of congenital anomalies in the community, or differences in the thoroughness with which minor congenital anomalies are recorded in the clinical notes, or coded in the National Minimum Dataset.
<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Northland</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microcephaly</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>11</td>
<td>2.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>22</td>
<td>4.4</td>
<td>205.9</td>
</tr>
<tr>
<td>Eyelid/Lacrimal/Eye/Orbit Malformations</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Accessory Auricle</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Other Ear Malformations</td>
<td>7</td>
<td>1.4</td>
<td>65.5</td>
</tr>
<tr>
<td>Other Face/Neck Malformations</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Total Malformations of Eye, Ear, Face and Neck</td>
<td>25</td>
<td>5.0</td>
<td>234.0</td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>19</td>
<td>3.8</td>
<td>177.9</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>30</td>
<td>6.0</td>
<td>280.8</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>52</td>
<td>10.4</td>
<td>486.8</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>11</td>
<td>2.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>5</td>
<td>1.0</td>
<td>46.8</td>
</tr>
<tr>
<td>Other Heart/Circulatory Malformations</td>
<td>11</td>
<td>2.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>152</td>
<td>30.4</td>
<td>1,422.8</td>
</tr>
<tr>
<td>Nose Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Lung Malformations</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Larynx Malformations</td>
<td>&lt;3</td>
<td></td>
<td>s</td>
</tr>
<tr>
<td>Total Malformations of the Respiratory System</td>
<td>11</td>
<td>2.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Ankyloglossia (Tongue Tie)</td>
<td>89</td>
<td>17.8</td>
<td>833.1</td>
</tr>
<tr>
<td>Tongue/Mouth/Pharynx Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Intestinal Malformations</td>
<td>8</td>
<td>1.6</td>
<td>74.9</td>
</tr>
<tr>
<td>Other Digestive Malformations</td>
<td>8</td>
<td>1.6</td>
<td>74.9</td>
</tr>
<tr>
<td>Total Malformations of the Digestive System</td>
<td>109</td>
<td>21.8</td>
<td>1,020.3</td>
</tr>
<tr>
<td>Female Genital Malformations</td>
<td>&lt;3</td>
<td></td>
<td>s</td>
</tr>
<tr>
<td>Undescended Testicle</td>
<td>49</td>
<td>9.8</td>
<td>458.7</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>19</td>
<td>3.8</td>
<td>177.9</td>
</tr>
<tr>
<td>Other Male Genital Malformations</td>
<td>7</td>
<td>1.4</td>
<td>65.5</td>
</tr>
<tr>
<td>Total Malformations of the Genital Organs</td>
<td>76</td>
<td>15.2</td>
<td>711.4</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly; s: suppressed due to small numbers
Table 21. Congenital Anomalies Evident at Birth, Northland 2008–2012 (Table 2 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleft Palate</td>
<td>7</td>
<td>1.4</td>
<td>65.5</td>
</tr>
<tr>
<td>Cleft Lip</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Cleft Palate and Lip</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Total Cleft Lip and Palate</td>
<td>15</td>
<td>3.0</td>
<td>140.4</td>
</tr>
<tr>
<td>Renal Agenesis/Reduction Defects</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Cystic Kidney Disease</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Renal Pelvis Obstruction/Ureter Malformations</td>
<td>15</td>
<td>3.0</td>
<td>140.4</td>
</tr>
<tr>
<td>Other Kidney/Urinary Malformations</td>
<td>17</td>
<td>3.4</td>
<td>159.1</td>
</tr>
<tr>
<td>Total Malformations of the Urinary System</td>
<td>39</td>
<td>7.8</td>
<td>365.1</td>
</tr>
<tr>
<td>Congenital Dislocation/Subluxation Hip</td>
<td>5</td>
<td>1.0</td>
<td>46.8</td>
</tr>
<tr>
<td>Other Deformities Hip</td>
<td>19</td>
<td>3.8</td>
<td>177.9</td>
</tr>
<tr>
<td>Foot Deformities</td>
<td>91</td>
<td>18.2</td>
<td>851.8</td>
</tr>
<tr>
<td>Other Musculoskeletal Malformations</td>
<td>29</td>
<td>5.8</td>
<td>271.5</td>
</tr>
<tr>
<td>Polysactyly</td>
<td>7</td>
<td>1.4</td>
<td>65.5</td>
</tr>
<tr>
<td>Syndactyly</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Reduction Defects/Other Limb Malformations</td>
<td>9</td>
<td>1.8</td>
<td>84.3</td>
</tr>
<tr>
<td>Skull/Facial Bones/Spine/Thorax Malformations</td>
<td>16</td>
<td>3.2</td>
<td>149.8</td>
</tr>
<tr>
<td>Total Malformations of the Musculoskeletal System</td>
<td>179</td>
<td>35.8</td>
<td>1,675.6</td>
</tr>
<tr>
<td>Non-Neoplastic Naevus</td>
<td>33</td>
<td>6.6</td>
<td>308.9</td>
</tr>
<tr>
<td>Other Skin Malformations</td>
<td>29</td>
<td>5.8</td>
<td>271.5</td>
</tr>
<tr>
<td>Other Integument Malformations</td>
<td>22</td>
<td>4.4</td>
<td>205.9</td>
</tr>
<tr>
<td>Other Malformations</td>
<td>21</td>
<td>4.2</td>
<td>196.6</td>
</tr>
<tr>
<td>Total Other Congenital Malformations</td>
<td>105</td>
<td>21.0</td>
<td>982.9</td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>10</td>
<td>2.0</td>
<td>93.6</td>
</tr>
<tr>
<td>Other Chromosome Anomalies</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>16</td>
<td>3.2</td>
<td>149.8</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset; Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly; s: suppressed due to small numbers
### Table 22. Congenital Anomalies Evident at Birth, Waitemata 2008–2012 (Table 1 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Waitemata</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly/Encephalocele</td>
<td>4</td>
<td>0.8</td>
<td>10.4</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>11</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>6</td>
<td>1.2</td>
<td>15.6</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>21</td>
<td>4.2</td>
<td>54.7</td>
</tr>
<tr>
<td>Spina Bifida</td>
<td>7</td>
<td>1.4</td>
<td>18.3</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>3</td>
<td>0.6</td>
<td>7.8</td>
</tr>
<tr>
<td><strong>Total Malformations of the Nervous System</strong></td>
<td>52</td>
<td>10.4</td>
<td>135.5</td>
</tr>
<tr>
<td>Eyelid/Lacrimal/Eye/Orbit Malformations</td>
<td>14</td>
<td>2.8</td>
<td>36.5</td>
</tr>
<tr>
<td>Ear Malformations Impairing Hearing</td>
<td>3</td>
<td>0.6</td>
<td>7.8</td>
</tr>
<tr>
<td>Accessory Auricle</td>
<td>69</td>
<td>13.8</td>
<td>179.9</td>
</tr>
<tr>
<td>Other Ear Malformations</td>
<td>46</td>
<td>9.2</td>
<td>119.9</td>
</tr>
<tr>
<td>Other Face/Neck Malformations</td>
<td>6</td>
<td>1.2</td>
<td>15.6</td>
</tr>
<tr>
<td><strong>Total Malformations of Eye, Ear, Face and Neck</strong></td>
<td>138</td>
<td>27.6</td>
<td>359.7</td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>19</td>
<td>3.8</td>
<td>49.5</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>45</td>
<td>9.0</td>
<td>117.3</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>46</td>
<td>9.2</td>
<td>119.9</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>3</td>
<td>0.6</td>
<td>7.8</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>13</td>
<td>2.6</td>
<td>33.9</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>30</td>
<td>6.0</td>
<td>78.2</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>11</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>32</td>
<td>6.4</td>
<td>83.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>142</td>
<td>28.4</td>
<td>370.2</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>44</td>
<td>8.8</td>
<td>114.7</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>5</td>
<td>1.0</td>
<td>13.0</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>55</td>
<td>11.0</td>
<td>143.4</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Total Malformations of the Circulatory System</strong></td>
<td>447</td>
<td>89.4</td>
<td>1,165.2</td>
</tr>
<tr>
<td>Nose Malformations</td>
<td>8</td>
<td>1.6</td>
<td>20.9</td>
</tr>
<tr>
<td>Larynx Malformations</td>
<td>11</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td>Lung Malformations</td>
<td>15</td>
<td>3.0</td>
<td>39.1</td>
</tr>
<tr>
<td>Other Respiratory Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Total Malformations of the Respiratory System</strong></td>
<td>36</td>
<td>7.2</td>
<td>93.8</td>
</tr>
<tr>
<td>Cleft Palate</td>
<td>27</td>
<td>5.4</td>
<td>70.4</td>
</tr>
<tr>
<td>Cleft Lip</td>
<td>11</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td>Cleft Palate and Lip</td>
<td>13</td>
<td>2.6</td>
<td>33.9</td>
</tr>
<tr>
<td><strong>Total Cleft Lip and Palate</strong></td>
<td>51</td>
<td>10.2</td>
<td>132.9</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly; s: suppressed due to small numbers
Table 23. Congenital Anomalies Evident at Birth, Waitemata 2008–2012 (Table 2 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Waitemata</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankyloglossia (Tongue Tie)</td>
<td>770</td>
<td>154.0</td>
<td>2,007.1</td>
</tr>
<tr>
<td>Tongue/Mouth/Pharynx Malformations</td>
<td>19</td>
<td>3.8</td>
<td>49.5</td>
</tr>
<tr>
<td>Oesophagus/Upper Alimentary Malformations</td>
<td>12</td>
<td>2.4</td>
<td>31.3</td>
</tr>
<tr>
<td>Intestinal Malformations</td>
<td>30</td>
<td>6.0</td>
<td>78.2</td>
</tr>
<tr>
<td>Other Digestive Malformations</td>
<td>7</td>
<td>1.4</td>
<td>18.3</td>
</tr>
<tr>
<td><strong>Total Malformations of the Digestive System</strong></td>
<td>838</td>
<td>167.6</td>
<td>2,184.4</td>
</tr>
<tr>
<td>Female Genital Malformations</td>
<td>17</td>
<td>3.4</td>
<td>44.3</td>
</tr>
<tr>
<td>Undescended Testicle</td>
<td>150</td>
<td>30.0</td>
<td>391.0</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>87</td>
<td>17.4</td>
<td>226.8</td>
</tr>
<tr>
<td>Other Male Genital Malformations</td>
<td>44</td>
<td>8.8</td>
<td>114.7</td>
</tr>
<tr>
<td>Indeterminate Sex/Pseudohermaphrodisism</td>
<td>4</td>
<td>0.8</td>
<td>10.4</td>
</tr>
<tr>
<td><strong>Total Malformations of the Genital Organs</strong></td>
<td>302</td>
<td>60.4</td>
<td>787.2</td>
</tr>
<tr>
<td>Renal Agenesis/Reduction Defects</td>
<td>11</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td>Cystic Kidney Disease</td>
<td>23</td>
<td>4.6</td>
<td>60.0</td>
</tr>
<tr>
<td>Renal Pelvis Obstruction/Ureter Malformations</td>
<td>39</td>
<td>7.8</td>
<td>101.7</td>
</tr>
<tr>
<td>Other Kidney/Ureter Malformations</td>
<td>102</td>
<td>20.4</td>
<td>265.9</td>
</tr>
<tr>
<td><strong>Total Malformations of the Urinary System</strong></td>
<td>175</td>
<td>35.0</td>
<td>456.2</td>
</tr>
<tr>
<td>Congenital Dislocation Hip</td>
<td>7</td>
<td>1.4</td>
<td>18.3</td>
</tr>
<tr>
<td>Other Deformities Hip</td>
<td>69</td>
<td>13.8</td>
<td>179.9</td>
</tr>
<tr>
<td>Foot Deformities</td>
<td>272</td>
<td>54.4</td>
<td>709.0</td>
</tr>
<tr>
<td>Polydactyly</td>
<td>47</td>
<td>9.4</td>
<td>122.5</td>
</tr>
<tr>
<td>Syndactyly</td>
<td>26</td>
<td>5.2</td>
<td>67.8</td>
</tr>
<tr>
<td>Reduction Defects/Other Limb Malformations</td>
<td>35</td>
<td>7.0</td>
<td>91.2</td>
</tr>
<tr>
<td>Other Musculoskeletal Malformations</td>
<td>88</td>
<td>17.6</td>
<td>229.4</td>
</tr>
<tr>
<td>Skull/Facial Bones/Spine/Thorax Malformations</td>
<td>46</td>
<td>9.2</td>
<td>119.9</td>
</tr>
<tr>
<td><strong>Total Malformations of the Musculoskeletal System</strong></td>
<td>590</td>
<td>118.0</td>
<td>1,537.9</td>
</tr>
<tr>
<td>Non-Neoplastic Naevus</td>
<td>112</td>
<td>22.4</td>
<td>292.0</td>
</tr>
<tr>
<td>Other Skin Malformations</td>
<td>115</td>
<td>23.0</td>
<td>299.8</td>
</tr>
<tr>
<td>Other Integument Malformations</td>
<td>97</td>
<td>19.4</td>
<td>252.9</td>
</tr>
<tr>
<td>Other Malformations</td>
<td>62</td>
<td>12.4</td>
<td>161.6</td>
</tr>
<tr>
<td><strong>Total Other Congenital Malformations</strong></td>
<td>386</td>
<td>77.2</td>
<td>1,006.2</td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>32</td>
<td>6.4</td>
<td>83.4</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>6</td>
<td>1.2</td>
<td>15.6</td>
</tr>
<tr>
<td>Other Chromosome Anomalies</td>
<td>6</td>
<td>1.2</td>
<td>15.6</td>
</tr>
<tr>
<td><strong>Total Chromosomal Anomalies</strong></td>
<td>44</td>
<td>8.8</td>
<td>114.7</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly.
<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auckland DHB</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly/Encephalocele</td>
<td>4</td>
<td>0.8</td>
<td>12.2</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>6</td>
<td>1.2</td>
<td>18.2</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>12</td>
<td>2.4</td>
<td>36.5</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>33</td>
<td>6.6</td>
<td>100.3</td>
</tr>
<tr>
<td>Spina Bifida</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Other Spinal Cord Malformations</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>63</td>
<td>12.6</td>
<td>191.5</td>
</tr>
<tr>
<td>Eyelid/Lacrimal/Eye/Orbit Malformations</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Ear Malformations Impairing Hearing</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Accessory Auricle</td>
<td>37</td>
<td>7.4</td>
<td>112.5</td>
</tr>
<tr>
<td>Other Ear Malformations</td>
<td>20</td>
<td>4.0</td>
<td>60.8</td>
</tr>
<tr>
<td>Other Face/Neck Malformations</td>
<td>5</td>
<td>1.0</td>
<td>15.2</td>
</tr>
<tr>
<td>Total Malformations of Eye, Ear, Face and Neck</td>
<td>76</td>
<td>15.2</td>
<td>231.1</td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>15</td>
<td>3.0</td>
<td>45.6</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>69</td>
<td>13.8</td>
<td>209.8</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>72</td>
<td>14.4</td>
<td>218.9</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>5</td>
<td>1.0</td>
<td>15.2</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>27</td>
<td>5.4</td>
<td>82.1</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>32</td>
<td>6.4</td>
<td>97.3</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>184</td>
<td>36.8</td>
<td>559.4</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>52</td>
<td>10.4</td>
<td>158.1</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>24</td>
<td>4.8</td>
<td>73.0</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>514</td>
<td>102.8</td>
<td>1,562.8</td>
</tr>
<tr>
<td>Trachea/Bronchus Malformations</td>
<td>12</td>
<td>2.4</td>
<td>36.5</td>
</tr>
<tr>
<td>Lung Malformations</td>
<td>14</td>
<td>2.8</td>
<td>42.6</td>
</tr>
<tr>
<td>Larynx Malformations</td>
<td>21</td>
<td>4.2</td>
<td>63.9</td>
</tr>
<tr>
<td>Other Respiratory Malformations</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Total Malformations of the Respiratory System</td>
<td>50</td>
<td>10.0</td>
<td>152.0</td>
</tr>
<tr>
<td>Cleft Palate</td>
<td>14</td>
<td>2.8</td>
<td>42.6</td>
</tr>
<tr>
<td>Cleft Lip</td>
<td>10</td>
<td>2.0</td>
<td>30.4</td>
</tr>
<tr>
<td>Cleft Palate and Lip</td>
<td>16</td>
<td>3.2</td>
<td>48.7</td>
</tr>
<tr>
<td>Total Cleft Lip and Palate</td>
<td>40</td>
<td>8.0</td>
<td>121.6</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly; s: suppressed due to small numbers
Table 25. Congenital Anomalies Evident at Birth, Auckland DHB 2008–2012 (Table 2 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankyloglossia (Tongue Tie)</td>
<td>456</td>
<td>91.2</td>
<td>1,386.4</td>
</tr>
<tr>
<td>Tongue/Mouth/Pharynx Malformations</td>
<td>16</td>
<td>3.2</td>
<td>48.7</td>
</tr>
<tr>
<td>Oesophagus/Upper Alimentary Malformations</td>
<td>5</td>
<td>1.0</td>
<td>15.2</td>
</tr>
<tr>
<td>Intestinal Malformations</td>
<td>32</td>
<td>6.4</td>
<td>97.3</td>
</tr>
<tr>
<td>Other Digestive Malformations</td>
<td>7</td>
<td>1.4</td>
<td>21.3</td>
</tr>
<tr>
<td><strong>Total Malformations of the Digestive System</strong></td>
<td>516</td>
<td>103.2</td>
<td>1,568.9</td>
</tr>
<tr>
<td>Female Genital Malformations</td>
<td>13</td>
<td>2.6</td>
<td>39.5</td>
</tr>
<tr>
<td>Undescended Testicle</td>
<td>121</td>
<td>24.2</td>
<td>367.9</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>61</td>
<td>12.2</td>
<td>185.5</td>
</tr>
<tr>
<td>Other Male Genital Malformations</td>
<td>27</td>
<td>5.4</td>
<td>82.1</td>
</tr>
<tr>
<td>Indeterminate Sex/Pseudohermaphrodisism</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Total Malformations of the Genital Organs</strong></td>
<td>224</td>
<td>44.8</td>
<td>681.1</td>
</tr>
<tr>
<td>Renal Agenesis/Reduction Defects</td>
<td>10</td>
<td>2.0</td>
<td>30.4</td>
</tr>
<tr>
<td>Cystic Kidney Disease</td>
<td>15</td>
<td>3.0</td>
<td>45.6</td>
</tr>
<tr>
<td>Renal Pelvis Obstruction/Ureter Malformations</td>
<td>30</td>
<td>6.0</td>
<td>91.2</td>
</tr>
<tr>
<td>Other Kidney/Urinary Malformations</td>
<td>49</td>
<td>9.8</td>
<td>149.0</td>
</tr>
<tr>
<td><strong>Total Malformations of the Urinary System</strong></td>
<td>104</td>
<td>20.8</td>
<td>316.2</td>
</tr>
<tr>
<td>Congenital Dislocation Hip</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Other Deformities Hip</td>
<td>42</td>
<td>8.4</td>
<td>127.7</td>
</tr>
<tr>
<td>Foot Deformities</td>
<td>175</td>
<td>35.0</td>
<td>532.1</td>
</tr>
<tr>
<td>Polydactyly</td>
<td>51</td>
<td>10.2</td>
<td>155.1</td>
</tr>
<tr>
<td>Syndactyly</td>
<td>15</td>
<td>3.0</td>
<td>45.6</td>
</tr>
<tr>
<td>Reduction Defects/Other Limb Malformations</td>
<td>28</td>
<td>5.6</td>
<td>85.1</td>
</tr>
<tr>
<td>Osteochondrodysplasia</td>
<td>5</td>
<td>1.0</td>
<td>15.2</td>
</tr>
<tr>
<td>Skull/Facial Bones/Spine/Thorax Malformations</td>
<td>28</td>
<td>5.6</td>
<td>85.1</td>
</tr>
<tr>
<td>Other Musculoskeletal Malformations</td>
<td>45</td>
<td>9.0</td>
<td>136.8</td>
</tr>
<tr>
<td><strong>Total Malformations of the Musculoskeletal System</strong></td>
<td>392</td>
<td>78.4</td>
<td>1,191.9</td>
</tr>
<tr>
<td>Non-Neoplastic Naevus</td>
<td>135</td>
<td>27.0</td>
<td>410.5</td>
</tr>
<tr>
<td>Other Skin Malformations</td>
<td>67</td>
<td>13.4</td>
<td>203.7</td>
</tr>
<tr>
<td>Other Integument Malformations</td>
<td>80</td>
<td>16.0</td>
<td>243.2</td>
</tr>
<tr>
<td>Other Malformations</td>
<td>45</td>
<td>9.0</td>
<td>136.8</td>
</tr>
<tr>
<td><strong>Total Other Congenital Malformations</strong></td>
<td>327</td>
<td>65.4</td>
<td>994.2</td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>29</td>
<td>5.8</td>
<td>88.2</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Sex Chromosome Anomalies</td>
<td>4</td>
<td>0.8</td>
<td>12.2</td>
</tr>
<tr>
<td>Monosomies/Autosomal Deletions/Rearrangements</td>
<td>4</td>
<td>0.8</td>
<td>12.2</td>
</tr>
<tr>
<td>Other Chromosome Anomalies</td>
<td>3</td>
<td>0.6</td>
<td>9.1</td>
</tr>
<tr>
<td><strong>Total Chromosomal Anomalies</strong></td>
<td>43</td>
<td>8.6</td>
<td>130.7</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly; s: suppressed due to small numbers
Table 26. Congenital Anomalies Evident at Birth, Counties Manukau 2008–2012 (Table 1 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Counties Manukau</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly/Encephalocele</td>
<td>6</td>
<td>1.2</td>
<td>14.0</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>7</td>
<td>1.4</td>
<td>16.3</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>11</td>
<td>2.2</td>
<td>25.6</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>23</td>
<td>4.6</td>
<td>53.5</td>
</tr>
<tr>
<td>Spina Bifida</td>
<td>10</td>
<td>2.0</td>
<td>23.3</td>
</tr>
<tr>
<td>Other Spinal Cord Malformations</td>
<td>3</td>
<td>0.6</td>
<td>7.0</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>4</td>
<td>0.8</td>
<td>9.3</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>64</td>
<td>12.8</td>
<td>148.8</td>
</tr>
<tr>
<td>Eyelid/Lacrimal/Eye/Orbit Malformations</td>
<td>14</td>
<td>2.8</td>
<td>32.5</td>
</tr>
<tr>
<td>Ear Malformations Impairing Hearing</td>
<td>4</td>
<td>0.8</td>
<td>9.3</td>
</tr>
<tr>
<td>Accessory Auricle</td>
<td>70</td>
<td>14.0</td>
<td>162.7</td>
</tr>
<tr>
<td>Other Ear Malformations</td>
<td>26</td>
<td>5.2</td>
<td>60.4</td>
</tr>
<tr>
<td>Other Face/Neck Malformations</td>
<td>14</td>
<td>2.8</td>
<td>32.5</td>
</tr>
<tr>
<td>Total Malformations of Eye, Ear, Face and Neck</td>
<td>128</td>
<td>25.6</td>
<td>297.5</td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>29</td>
<td>5.8</td>
<td>67.4</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>29</td>
<td>5.8</td>
<td>67.4</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>45</td>
<td>9.0</td>
<td>104.6</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>4</td>
<td>0.8</td>
<td>9.3</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>5</td>
<td>1.0</td>
<td>11.6</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>23</td>
<td>4.6</td>
<td>53.5</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>12</td>
<td>2.4</td>
<td>27.9</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>32</td>
<td>6.4</td>
<td>74.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>183</td>
<td>36.6</td>
<td>425.4</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>21</td>
<td>4.2</td>
<td>48.8</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>26</td>
<td>5.2</td>
<td>60.4</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>3</td>
<td>0.6</td>
<td>7.0</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>414</td>
<td>82.8</td>
<td>962.3</td>
</tr>
<tr>
<td>Nose Malformations</td>
<td>7</td>
<td>1.4</td>
<td>16.3</td>
</tr>
<tr>
<td>Larynx Malformations</td>
<td>7</td>
<td>1.4</td>
<td>16.3</td>
</tr>
<tr>
<td>Lung Malformations</td>
<td>15</td>
<td>3.0</td>
<td>34.9</td>
</tr>
<tr>
<td>Other Respiratory Malformations</td>
<td>3</td>
<td>0.6</td>
<td>7.0</td>
</tr>
<tr>
<td>Total Malformations of the Respiratory System</td>
<td>32</td>
<td>6.4</td>
<td>74.4</td>
</tr>
<tr>
<td>Cleft Palate</td>
<td>29</td>
<td>5.8</td>
<td>67.4</td>
</tr>
<tr>
<td>Cleft Lip</td>
<td>4</td>
<td>0.8</td>
<td>9.3</td>
</tr>
<tr>
<td>Cleft Palate and Lip</td>
<td>16</td>
<td>3.2</td>
<td>37.2</td>
</tr>
<tr>
<td>Total Cleft Lip and Palate</td>
<td>49</td>
<td>9.8</td>
<td>113.9</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly; s: suppressed due to small numbers
Table 27. Congenital Anomalies Evident at Birth, Counties Manukau 2008–2012 (Table 2 of 2)

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Counties Manukau</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankyloglossia (Tongue Tie)</td>
<td>419</td>
<td>83.8</td>
<td>974.0</td>
</tr>
<tr>
<td>Tongue/Mouth/Pharynx Malformations</td>
<td>8</td>
<td>1.6</td>
<td>18.6</td>
</tr>
<tr>
<td>Oesophagus/Upper Alimentary Malformations</td>
<td>6</td>
<td>1.2</td>
<td>14.0</td>
</tr>
<tr>
<td>Intestinal Malformations</td>
<td>25</td>
<td>5.0</td>
<td>58.1</td>
</tr>
<tr>
<td>Other Digestive Malformations</td>
<td>4</td>
<td>0.8</td>
<td>9.3</td>
</tr>
<tr>
<td>Total Malformations of the Digestive System</td>
<td>462</td>
<td>92.4</td>
<td>1,073.9</td>
</tr>
<tr>
<td>Female Genital Malformations</td>
<td>8</td>
<td>1.6</td>
<td>18.6</td>
</tr>
<tr>
<td>Undescended Testicle</td>
<td>166</td>
<td>33.2</td>
<td>385.9</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>93</td>
<td>18.6</td>
<td>216.2</td>
</tr>
<tr>
<td>Other Male Genital Malformations</td>
<td>16</td>
<td>3.2</td>
<td>37.2</td>
</tr>
<tr>
<td>Indeterminate Sex/Pseudohermaphrodisim</td>
<td>5</td>
<td>1.0</td>
<td>11.6</td>
</tr>
<tr>
<td>Total Malformations of the Genital Organs</td>
<td>288</td>
<td>57.6</td>
<td>669.5</td>
</tr>
<tr>
<td>Renal Agenesis/Reduction Defects</td>
<td>9</td>
<td>1.8</td>
<td>20.9</td>
</tr>
<tr>
<td>Cystic Kidney Disease</td>
<td>29</td>
<td>5.8</td>
<td>67.4</td>
</tr>
<tr>
<td>Renal Pelvis Obstruction/Ureter Malformations</td>
<td>22</td>
<td>4.4</td>
<td>51.1</td>
</tr>
<tr>
<td>Other Kidney/Urinary Malformations</td>
<td>52</td>
<td>10.4</td>
<td>120.9</td>
</tr>
<tr>
<td>Total Malformations of the Urinary System</td>
<td>112</td>
<td>22.4</td>
<td>260.3</td>
</tr>
<tr>
<td>Skull/Facial Bones/Spine/Thorax Malformations</td>
<td>28</td>
<td>5.6</td>
<td>65.1</td>
</tr>
<tr>
<td>Foot Deformities</td>
<td>318</td>
<td>63.6</td>
<td>739.2</td>
</tr>
<tr>
<td>Polydactyly</td>
<td>38</td>
<td>7.6</td>
<td>88.3</td>
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<tr>
<td>Syndactyly</td>
<td>8</td>
<td>1.6</td>
<td>18.6</td>
</tr>
<tr>
<td>Congenital Dislocation Hip</td>
<td>6</td>
<td>1.2</td>
<td>14.0</td>
</tr>
<tr>
<td>Other Deformities Hip</td>
<td>68</td>
<td>13.6</td>
<td>158.1</td>
</tr>
<tr>
<td>Reduction Defects/Other Limb Malformations</td>
<td>19</td>
<td>3.8</td>
<td>44.2</td>
</tr>
<tr>
<td>Other Musculoskeletal Malformations</td>
<td>69</td>
<td>13.8</td>
<td>160.4</td>
</tr>
<tr>
<td>Total Malformations of the Musculoskeletal System</td>
<td>554</td>
<td>110.8</td>
<td>1,287.8</td>
</tr>
<tr>
<td>Non-Neoplastic Naevus</td>
<td>54</td>
<td>10.8</td>
<td>125.5</td>
</tr>
<tr>
<td>Epidermolysis Bullosa</td>
<td>3</td>
<td>0.6</td>
<td>7.0</td>
</tr>
<tr>
<td>Other Skin Malformations</td>
<td>95</td>
<td>19.0</td>
<td>220.8</td>
</tr>
<tr>
<td>Breast Malformations</td>
<td>7</td>
<td>1.4</td>
<td>16.3</td>
</tr>
<tr>
<td>Other Integument Malformations</td>
<td>86</td>
<td>17.2</td>
<td>199.9</td>
</tr>
<tr>
<td>Other Malformations</td>
<td>53</td>
<td>10.6</td>
<td>123.2</td>
</tr>
<tr>
<td>Total Other Congenital Malformations</td>
<td>298</td>
<td>59.6</td>
<td>692.7</td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>44</td>
<td>8.8</td>
<td>102.3</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>5</td>
<td>1.0</td>
<td>11.6</td>
</tr>
<tr>
<td>Other Chromosome Anomalies</td>
<td>8</td>
<td>1.6</td>
<td>18.6</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>57</td>
<td>11.4</td>
<td>132.5</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly
Table 28. Babies with Congenital Anomalies Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Rate per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>557</td>
<td>111</td>
<td>5,214</td>
<td>1.12</td>
<td>1.03–1.22</td>
</tr>
<tr>
<td>Waitemata</td>
<td>2,480</td>
<td>496</td>
<td>6,465</td>
<td>1.39</td>
<td>1.34–1.45</td>
</tr>
<tr>
<td>Auckland</td>
<td>1,828</td>
<td>366</td>
<td>5,558</td>
<td>1.20</td>
<td>1.14–1.26</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>1,995</td>
<td>399</td>
<td>4,637</td>
<td>1.00</td>
<td>0.95–1.05</td>
</tr>
<tr>
<td>New Zealand</td>
<td>14,033</td>
<td>2,807</td>
<td>4,642</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a congenital anomaly listed in any of first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more anomalies, rather than number of anomalies; Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics

**Northern DHBs Trends**

In the Northern region during 2000–2012, while individual DHB trends varied, rates in the Northland, Waitemata and Auckland DHBs remained higher than the New Zealand rate. In the case of Northland and Auckland, however, the magnitude of this difference reduced as the period progressed. Rates in Counties Manukau were also higher than the New Zealand rate during the early 2000s, but became similar from 2008–2009 onwards (Figure 16). Again it is unclear whether the trends presented reflect any real change in the prevalence of congenital anomalies, or just changes in the way in which minor congenital anomalies were recorded in the hospital admission dataset over time.
Figure 16. Babies with Congenital Anomalies Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a congenital anomaly listed in any of first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more anomalies, rather than number of anomalies; Note: *Numbers are per 2 year period, except for 2012 which is for a single year only
Local Policy Documents and Evidence-Based Reviews Relevant to Congenital Anomalies

In New Zealand there is a paucity of publications relevant to the prevention or management of congenital anomalies as a group. The publications that are available are summarised in Table 29, along with a range of reviews which consider these issues in the overseas context.

In addition, Table 16 on Page 79 considers publications relevant to antenatal and newborn screening, while Table 35 on Page 115 considers cardiovascular anomalies, Table 41 on Page 125 considers Down syndrome and other chromosomal anomalies, and Table 46 on Page 134 considers neural tube defects. Finally Table 53 on Page 150 provides a brief overview of publications relevant to newborn screening for congenital hearing loss.

Table 29. Local Policy Documents and Evidence-Based Reviews Relevant to Congenital Anomalies

<table>
<thead>
<tr>
<th>Ministry of Health Documents</th>
</tr>
</thead>
</table>

Chapter 12 of the Immunisation handbook deals with rubella. It states that maternal infection with rubella in the first eight weeks of pregnancy leads to fetal damage in 85% of infants and that multiple defects are common. The current immunisation schedule contains two doses of measles, mumps and rubella vaccine, offered at ages 15 months and four years. It is recommended that all women of childbearing age, particularly immigrants from less developed countries, be offered screening to check their rubella immunity in their early reproductive years, when they are planning a pregnancy and when they are pregnant. Non-pregnant non-immune women should be offered vaccination immediately, and pregnant women should be offered vaccination after delivery. Women should not be vaccinated if they are pregnant and they should avoid pregnancy for four weeks after immunisation.

<table>
<thead>
<tr>
<th>International Guidelines and Systematic and Other Reviews</th>
</tr>
</thead>
</table>

Toxoplasmosis is caused by an animal parasite, Toxoplasma gondii. Infection occurs by ingesting oocysts excreted by cats, contaminated soil or water, or by eating the undercooked meat of infected animals. Infection in pregnancy can result in transmission of infection to the fetus. Congenital toxoplasmosis can lead to intrauterine death or stillbirth, mental retardation, malformations and deafness and blindness of the infant. Both prenatal screening of women and postnatal screening of babies are possible but such screening has a number of limitations and may not be effective for improving fetal outcomes. Prenatal education of pregnant women encourages them to adopt preventive measures including not eating insufficiently cooked meat, washing hands after gardening or handling raw meat, and avoiding contact with cats’ faeces, either directly or indirectly through soil or contaminated fruit or vegetables. This review assessed the effects of prenatal education for preventing congenital toxoplasmosis. It included two RCTs, both of low methodological quality, one conducted in Canada (432 women) and one in France (5023 women). Both trials had high losses to follow up. The authors of the Canadian trial reported only p values (< 0.05 for all outcomes), not measures of association. They concluded that prenatal education can change women’s behaviour as it increased personal, food and pet hygiene. The authors of the French trial concluded that prenatal education significantly improved women’s knowledge but had no effect on behaviour. The French trial measured sero-conversion rates and found no difference between the intervention group (13/2591) and the control group (4/1358). The authors concluded that although evidence from observational studies suggests that prenatal education is effective in reducing rates of congenital toxoplasmosis there is little evidence from RCTs to support the practice.


This open-access systematic review reports on a meta-analysis of observational studies published in 1959–2010 (172 publications in total). There were significant positive associations found between maternal smoking and the following birth defects: cardiovascular/heart defects (Odds Ratio 1.09); musculoskeletal defects (OR 1.16); limb reduction defects (OR 1.26); missing/extra digits (OR 1.18); clubfoot (OR 1.28); craniosynostosis (OR 1.33); facial defects (OR 1.19); eye defects (OR 1.25); oro facial clefts (OR 1.28); gastrointestinal defects (OR 1.27); gastrochisis (OR 1.50); anal atresia (OR 1.20); hemia (OR 1.40); and undescended testes (OR 1.13). There was a reduced risk for hypospadias (OR 0.90) and skin defects (OR 0.82). For all defects combined the OR was 1.01 (0.96–1.07). This was non-significant due to including defects with a reduced risk and those with no association (including chromosomal defects). The authors stated that information on the specific defects that are associated with smoking in pregnancy should be included in public health educational information to encourage more women to stop smoking before or early on in pregnancy and that such information should be targeted at the groups who have the highest prevalence of smoking: younger women and those from lower socio-economic groups.
Congenital Anomalies Evident at Birth - 103

These concise evidence-based Canadian guidelines are intended for genetic counsellors, midwives, nurses, and physicians who may be involved in care for a pregnant woman whose fetus has been prenatally diagnosed with isolated or multiple structural congenital anomalies. (Around 1% of prenatal ultrasound examinations reveal a fetal structural anomaly.) The evidence is graded, and the recommendations are classified, using criteria adapted from those of The Canadian Task Force on Preventive Health Care.


There are a number of potentially modifiable risk factors for poor pregnancy outcomes (including congenital anomalies). These include poor nutrition, smoking and drinking excess alcohol. This review assessed the effectiveness of routine pre-pregnancy health promotion (compared to usual care or no pre-pregnancy care) for improving pregnancy outcomes. It included four trials (2300 women) but only one of them involved follow up of pregnancy outcomes. In this study there was no strong evidence of a difference between the intervention and control groups for preterm birth, congenital anomalies or weight for gestational age. There was a statistically significant difference in mean birthweight: mean difference (intervention group – control group) -97.00g, 95% confidence interval -168.05g to -25.95g but this finding was based on outcome data for only half of the randomised women. There was some evidence that health promotion interventions were associated with positive behaviour change including reduced binge drinking (risk ratio 1.24, 95% CI 1.06 to 1.44). The authors concluded that, overall there was little evidence on the effects of pre-pregnancy health promotion on pregnancy outcomes and that more research in this area is needed.


These concise and well-referenced guidelines include guidance on the management of congenital urological anomalies. The authors stated that, where possible statements in the guidelines have been classified in terms of level of evidence and grade of recommendation but that because there have been a limited number of RCTs in this field and a considerable number of the treatment options involve surgical interventions on a wide spectrum of different congenital problems, most of the recommendations are based on consensus.


A pregnant woman who is infected with syphilis can pass the infection to her fetus. When a woman acquires the infection close to the time she becomes pregnant or during pregnancy the fetus is often aborted or stillborn. Pregnancies in an untreated woman later in the course of her disease may lead to a baby born with congenital syphilis. These babies may be born prematurely, small for gestational age and with low birthweight. They may have visible signs of the disease but most are asymptomatic at birth, developing signs of infection after a few weeks or months. Without treatment they may develop blindness, deafness, and facial, dental, skeletal and neurological abnormalities later in life. In developed countries pregnant women are screened for syphilis infection and congenital syphilis is rare. The standard treatment for syphilis in adults is long-acting penicillin by injection but there have been concerns raised that the standard treatment regimen may not be optimal, particularly in women who also have HIV. The authors of this review assessed RCTs and quasi RCTs evaluating treatment regimens (in terms of dose, length of treatment course, and administration) for pregnant women with syphilis, with and without concomitant HIV infection. They were unable to identify any studies that met their criteria and they stated that more research is needed on this issue.


Congenital infection with toxoplasmosis may lead to an infant being with mental retardation and blindness. The authors of this review assessed whether treating toxoplasmosis infection in pregnancy reduces the risk of congenital toxoplasmosis infection. They did not identify any RCTs addressing treatment for pregnant women who had serological evidence of recent toxoplasma infection so they concluded that it is unknown whether such treatment is effective. They noted that some countries (France and Austria) have introduced toxoplasmosis screening for pregnant women. They stated that since screening is expensive, the benefits of treatment are uncertain and there may be adverse effects from treatment drugs, screening should not be introduced in other countries other than as part of a carefully controlled trial.

International guidelines and systematic reviews concerning the management of congenital anomalies

Guidelines from the National Institute of Health and Care Excellence:


Glutamine supplementation for young infants with severe gastrointestinal disease. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD005947.pub3

Interventions for congenital talipes equinovarus (clubfoot). Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD006545.pub2

Interventions for infantile haemangiomas (strawberry birthmarks) of the skin. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD006545.pub2

Interventions for congenital choanal atresia. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD009547.pub3


Electropalatography for articulation disorders associated with cleft palate. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD007037.pub2

Surgical interventions for bilateral congenital cataract. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD003171.pub2

Surgical treatment for the thumb-in-palm deformity in patients with cerebral palsy. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD004093.pub2

Ward reduction without general anaesthesia versus reduction and repair under general anaesthesia for gastrochisis in newborn infants. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD003671

Late versus early surgical correction for congenital diaphragmatic hernia in newborn infants. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD001695


Cochrane Reviews:


### Other Relevant Publications and Websites

<table>
<thead>
<tr>
<th>Topic</th>
<th>Publication/Website</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticonvulsants and congenital malformations</td>
<td><a href="http://medsafe.govt.nz/profs/puarticles/anticonvulsants-feb09.htm">http://medsafe.govt.nz/profs/puarticles/anticonvulsants-feb09.htm</a></td>
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<td>SSRI Use in Pregnancy - Collaborative Decision-Making is Key</td>
<td><a href="http://medsafe.govt.nz/profs/puarticles/ssnipreg.htm">http://medsafe.govt.nz/profs/puarticles/ssnipreg.htm</a></td>
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<td>Avoiding Teratogenicity with Isotretinoin</td>
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</tr>
<tr>
<td>ACE Inhibitors in Early Pregnancy</td>
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<tr>
<td>National Collaborating Centre for Women’s and Children’s Health</td>
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<td><a href="http://medsafe.govt.nz/profs/puarticles/anticonvulsants-feb09.htm">http://medsafe.govt.nz/profs/puarticles/anticonvulsants-feb09.htm</a></td>
</tr>
<tr>
<td>There are a number of commonly prescribed drugs that have been</td>
<td></td>
</tr>
<tr>
<td>associated with increased risks of congenital anomalies. The</td>
<td></td>
</tr>
<tr>
<td>following webpages on the Medsafe website provide information on</td>
<td></td>
</tr>
<tr>
<td>this topic:</td>
<td></td>
</tr>
<tr>
<td>diagnosis in a tertiary fetal medicine unit</td>
<td></td>
</tr>
<tr>
<td>This study reports on the accuracy of prenatal diagnosis in the</td>
<td></td>
</tr>
<tr>
<td>fetal medicine unit in Christchurch. It involved a review</td>
<td></td>
</tr>
<tr>
<td>681 cases seen over an 18 month period from 1 June 2004 to 30</td>
<td></td>
</tr>
<tr>
<td>November 2005 in which prenatal diagnoses made</td>
<td></td>
</tr>
<tr>
<td>via ultrasound were compared those made postnatally (or at post-</td>
<td></td>
</tr>
<tr>
<td>mortem in cases where there was a termination or an intrauterine</td>
<td></td>
</tr>
<tr>
<td>or postnatal death. For the live born babies 93.6% had their</td>
<td></td>
</tr>
<tr>
<td>prenatal diagnosis confirmed, 5.1% had an issue which had resolved</td>
<td></td>
</tr>
<tr>
<td>by the time of birth, and 1.3% had an additional major abnormality</td>
<td></td>
</tr>
<tr>
<td>that had a significant clinical effect. Fifty two percent of the</td>
<td></td>
</tr>
<tr>
<td>fetal or neonatal deaths with a normal karyotype were followed by</td>
<td></td>
</tr>
<tr>
<td>a post-mortem examination and there was only one new finding that</td>
<td></td>
</tr>
<tr>
<td>changed the prenatal diagnosis significantly. The authors stated</td>
<td></td>
</tr>
<tr>
<td>that parents and staff need to be informed that, although not all</td>
<td></td>
</tr>
<tr>
<td>abnormalities will be detected prenatally, inaccurate prenatal</td>
<td></td>
</tr>
<tr>
<td>diagnosis is rare.</td>
<td></td>
</tr>
<tr>
<td>International Centre on Birth Defects - ICBDSR Centre.</td>
<td></td>
</tr>
<tr>
<td>This publication reports on two collaborative research projects</td>
<td></td>
</tr>
<tr>
<td>involving data from several countries: one on multiple congenital</td>
<td></td>
</tr>
<tr>
<td>anomalies and one on prenatal diagnosis and Down syndrome. It</td>
<td></td>
</tr>
<tr>
<td>provides surveillance data on 39 birth defects that are monitored</td>
<td></td>
</tr>
<tr>
<td>by 44 member programmes around the world. It includes data from the</td>
<td></td>
</tr>
<tr>
<td>New Zealand Birth Defects Registry.</td>
<td></td>
</tr>
</tbody>
</table>

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
**Congenital Heart Disease**

**Introduction**

Congenital heart disease (CHD) is the most common congenital disorder in newborns [36]. Definitional issues have led to large variations in prevalence estimates. In 2002, Hoffman et al. [37] estimated the birth prevalence of severe CHD requiring expert cardiological care (e.g. transposition of the great arteries, Tetralogy of Fallot) to be 2.5–3.0 per 1,000 live births, with moderately severe forms of CHD (e.g. large atrial septal defects, complex forms of ventricular septal defects) accounting for another 3 per 1,000 live births. The overall prevalence increased to 75 per 1,000 if minor anomalies (e.g. small ventricular septal defects, atrial septal defects, or patent ductus arteriosus) were included. Subsequent reviews, however, have yielded much lower prevalence estimates, ranging from 3–6 per 1,000 live births [36].

The aetiology of CHD remains largely unknown, with only around 15% of cases being traced to a known cause. These causes include chromosomal anomalies, such as Down syndrome and trisomies 13 and 18, which account for around 8–10% of cases, and single gene defects, which account for a further 3–5%. The causes of non-syndromal CHD are less clear, with only around 2% of cases being attributed to known environmental factors such as maternal diabetes, obesity, alcohol use, rubella, febrile illnesses and use of certain drugs [36]. Genetic factors may also play a role, as the risk of recurrence is 1–6% if one sibling is affected, and up to 10% if two siblings are affected [36].

While prevention may need to await a better understanding of causal pathways, for effective treatment early detection and timely management are crucial. In this context, research has suggested that up to 25% of babies with severe forms of congenital heart disease may be discharged from hospital undiagnosed. Recent studies have suggested that pulse oximetry, if used in conjunction with a clinical examination prior to discharge, may improve the detection rate of some forms of CHD [16]. However, it is still likely that a number of babies with serious cardiovascular anomalies will be missed in the neonatal period and so antenatal screening has become established practice in many centres [16].

In tertiary centres which specialise in the diagnosis and management of congenital cardiac anomalies a high degree of diagnostic accuracy is possible, and most (but not all) major forms of CHD can be detected antenatally [16]. Most of cases of congenital heart disease, however, occur in low risk groups and will only be detected antenatally if detailed examination of the fetal heart is included as part of routine obstetric ultrasound screening (by obtaining a four chamber view of the fetal heart). In these cases detection rates are likely to depend on the level of sonographer training and experience, the adequacy of the equipment available, and the time allowed for sonographers to undertake routine examinations [16]. Investing in these resources is important, as early detection provides an opportunity to exclude associated extra-cardiac and chromosomal abnormalities, discuss pregnancy options, prepare parents, and plan for delivery in a tertiary centre [16].

The following section uses data from the National Minimum Dataset to review the number of babies with cardiovascular anomalies evident at the time of birth.

<table>
<thead>
<tr>
<th>Data Source and Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
</tr>
<tr>
<td>1. Number of cardiovascular anomalies evident at birth (by anomaly type)</td>
</tr>
<tr>
<td>2. Number of babies with one or more cardiovascular anomalies evident at birth (by anomaly type)</td>
</tr>
<tr>
<td><strong>Data Source</strong></td>
</tr>
<tr>
<td>1. National Minimum Dataset</td>
</tr>
<tr>
<td>Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly (ICD-10 Q20-Q28) listed in any of the first 15 diagnoses.</td>
</tr>
<tr>
<td>Denominator: All hospital admissions with event type = birth</td>
</tr>
</tbody>
</table>
Notes on Interpretation

Note 1: The analysis includes all admissions in the National Minimum Dataset where the Event Type was listed as a Birth (Note: Only one birth event is allowed per NHI number, with babies being born prior to hospital admission, or being readmitted shortly after discharge being listed as a routine inpatient event). Thus the analysis does not include babies born prior to hospital admission, babies born at home, or babies whose cardiovascular anomaly was overlooked at the time of initial hospital discharge, but who re-presented shortly thereafter. Further, the methodology used may significantly undercount those cardiovascular anomalies which are difficult to detect on routine newborn examination.

Note 2: In the analysis which follows, 64.2% of patent ductus arteriosus (PDA) identified during 2008–2012 were in preterm babies (<37 weeks gestation) who had no other cardiovascular anomalies. Prematurity is known to increase the risk of PDA as a result of increased exposure to hypoxia and underdeveloped heart and lungs. During 2008–12, 23.8% of all cardiovascular anomalies were isolated PDAs in preterm infants, many of whom would not have had a PDA had they been born at term. As the possibility that any analysis of risk factors for cardiovascular anomalies may be inadvertently distorted by the risk factor profile of those babies being born prematurely, preterm (<37 weeks) babies with isolated PDAs (i.e. a PDA with no other cardiovascular anomaly) have been excluded from rate calculations after the initial overview table.

For a list of the ICD-10-AM codes used to assign cardiovascular anomaly types see in Appendix 8.

New Zealand Distribution and Trends

Cardiovascular Anomalies Evident at Birth

In New Zealand during 2008–2012, patent ductus arteriosus (PDA) was the most frequent cardiovascular anomaly identified at birth, with 64.2% of PDAs being in preterm babies (<37 weeks gestation) who had no other cardiovascular anomalies. Prematurity is known to increase the risk of PDA as a result of increased exposure to hypoxia and underdeveloped heart and lungs. During 2008–12, 23.8% of all cardiovascular anomalies were isolated PDAs in preterm infants, many of whom would not have had a PDA had they been born at term. As the possibility that any analysis of risk factors for cardiovascular anomalies may be inadvertently distorted by the risk factor profile of those babies being born prematurely, preterm (<37 weeks) babies with isolated PDAs (i.e. a PDA with no other cardiovascular anomaly) have been excluded from rate calculations after the initial overview table.

New Zealand Trends

In New Zealand during the mid-2000s the number of babies born with one or more cardiovascular anomalies was relatively stable. Rates declined between 2007 and 2009 and then remained static until 2012, when a small upswing was evident (Figure 17). It remains unclear however, whether the decline in rates during 2007–2009, and the subsequent rebound, reflects real changes in the number of babies born with cardiovascular anomalies, changes in the thoroughness of the recording of cardiovascular anomalies by clinicians, or changes in the way these anomalies were coded in the hospital admission dataset.

Distribution by Maternal Age

While in numerical terms, the largest number of babies born with cardiovascular anomalies during 2008–2012 had mothers who were aged 30–34 years, the risk of cardiovascular anomalies rose progressively with increasing maternal age. Thus babies whose mothers were aged 40+ years had cardiovascular anomaly rates which were 1.81 (95% CI 1.42–2.32) times higher than those whose mothers gave birth in their teens (Figure 18).

Distribution by Prioritised Ethnicity, NZDep Index Decile and Gender

In New Zealand during 2008–2012, there were no significant ethnic differences in the proportion of babies born with cardiovascular anomalies. Rates were significantly higher however, for babies from the least deprived (NZDep06 deciles 1–2 vs. deciles 5–10) areas, for males, and for those with older (40+ years vs. <20 years) mothers (Table 31). While cardiovascular anomaly rates were higher for European babies than for Māori babies during the early 2000s, rates became similar from 2010–11 onwards. Differences between European, Pacific and Asian/Indian babies however, were less consistent during 2000–2012 (Figure 19).
Table 30. Cardiovascular Anomalies Evident at Birth, New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Cardiovascular Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>190</td>
<td>38.0</td>
<td>62.9</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>533</td>
<td>106.6</td>
<td>176.3</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>588</td>
<td>117.6</td>
<td>194.5</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>42</td>
<td>8.4</td>
<td>13.9</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>92</td>
<td>18.4</td>
<td>30.4</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>185</td>
<td>37.0</td>
<td>61.2</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>111</td>
<td>22.2</td>
<td>36.7</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>246</td>
<td>49.2</td>
<td>81.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)*</td>
<td>1,490</td>
<td>298.0</td>
<td>492.9</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>313</td>
<td>62.6</td>
<td>103.5</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>52</td>
<td>10.4</td>
<td>17.2</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>165</td>
<td>33.0</td>
<td>54.6</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>15</td>
<td>3.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>4,022</td>
<td>804.4</td>
<td>1,330.4</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: *Anomalies per 100,000 births refers to total number of anomalies rather than number of babies, as some babies have more than one anomaly; *Patent Ductus Arteriosus includes 957 cases of Isolated PDA in preterm infants (<37 weeks), which have been excluded in subsequent analyses

Figure 17. Babies with Cardiovascular Anomalies Evident at Birth, New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more CVS anomalies, rather than the number of anomalies; Preterm (<37 weeks) babies with Isolated Patent Ductus Arteriosus have been excluded
Figure 18. Babies with Cardiovascular Anomalies Evident at Birth by Maternal Age, New Zealand Hospital Births 2008–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a Cardiovascular Anomaly listed in any of first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more CVS anomalies, rather than the number of anomalies; Preterm (<37 weeks) babies with Isolated Patent Ductus Arteriosus excluded.

Figure 19. Babies with Cardiovascular Anomalies Evident at Birth by Prioritised Ethnicity, New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more CVS anomalies, rather than the number of anomalies; Preterm (<37 weeks) babies with Isolated Patent Ductus Arteriosus excluded.
Table 31. Distribution of Babies with Cardiovascular Anomalies Evident at Birth by Prioritised Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of Babies: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Any Cardiovascular Anomaly</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Prioritised Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>71.8</td>
<td>538.2</td>
<td>0.95</td>
<td>0.84–1.07</td>
</tr>
<tr>
<td>Pacific</td>
<td>36.2</td>
<td>533.9</td>
<td>0.94</td>
<td>0.80–1.10</td>
</tr>
<tr>
<td>European/Other</td>
<td>187.8</td>
<td>566.4</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>35.8</td>
<td>523.2</td>
<td>0.92</td>
<td>0.79–1.08</td>
</tr>
<tr>
<td><strong>NZ Deprivation Index</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>55.6</td>
<td>655.3</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>53.4</td>
<td>592.1</td>
<td>0.90</td>
<td>0.76–1.07</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>60.4</td>
<td>536.2</td>
<td>0.82</td>
<td>0.70–0.96</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>75.8</td>
<td>537.4</td>
<td>0.82</td>
<td>0.70–0.96</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>88.4</td>
<td>504.8</td>
<td>0.77</td>
<td>0.66–0.89</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>151.6</td>
<td>515.7</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>182.2</td>
<td>586.5</td>
<td>1.14</td>
<td>1.03–1.25</td>
</tr>
<tr>
<td><strong>Maternal Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 Years</td>
<td>23.0</td>
<td>551.0</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>20–24 Years</td>
<td>52.2</td>
<td>467.4</td>
<td>0.85</td>
<td>0.68–1.06</td>
</tr>
<tr>
<td>25–29 Years</td>
<td>74.0</td>
<td>498.6</td>
<td>0.91</td>
<td>0.73–1.12</td>
</tr>
<tr>
<td>30–34 Years</td>
<td>89.4</td>
<td>535.1</td>
<td>0.97</td>
<td>0.79–1.19</td>
</tr>
<tr>
<td>35–39 Years</td>
<td>68.4</td>
<td>628.0</td>
<td>1.14</td>
<td>0.92–1.41</td>
</tr>
<tr>
<td>40+ Years</td>
<td>26.8</td>
<td>999.3</td>
<td>1.81</td>
<td>1.42–2.32</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more CVS anomalies, rather than the number of anomalies; Preterm (<37 weeks) babies with Isolated Patent Ductus Arteriosus excluded; Decile is NZDep06

**Northern DHBs Distribution and Trends**

Table 32. Number of Babies with One or More Cardiovascular Anomalies Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Rate per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiovascular Anomalies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>73</td>
<td>14.6</td>
<td>683.3</td>
<td>1.24</td>
<td>0.98–1.56</td>
</tr>
<tr>
<td>Waitemata</td>
<td>194</td>
<td>38.8</td>
<td>505.7</td>
<td>0.92</td>
<td>0.79–1.06</td>
</tr>
<tr>
<td>Auckland</td>
<td>213</td>
<td>42.6</td>
<td>647.6</td>
<td>1.17</td>
<td>1.02–1.35</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>148</td>
<td>29.6</td>
<td>344.0</td>
<td>0.62</td>
<td>0.53–0.74</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1,669</td>
<td>333.8</td>
<td>552.1</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more CVS anomalies, rather than the number of anomalies; Preterm (<37 weeks) babies with Isolated Patent Ductus Arteriosus excluded; Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics
Northern DHBs vs. New Zealand

When the number of babies with one or more CVS anomalies (rather than the number of CVS anomalies) was considered, on average 14.6 Northland, 38.8 Waitemata, 42.6 Auckland and 29.6 Counties Manukau babies each year (excluding isolated preterm PDAs) were born with one or more CVS anomalies. While rates in the Northland and Waitemata DHBs were not significantly different from the New Zealand rate, rates in the Auckland DHB were significantly higher, while rates in Counties Manukau were significantly lower (Table 32) (Figure 20).

Table 33. Cardiovascular Anomalies Evident at Birth, Northland and Waitemata Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Cardiovascular Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>6</td>
<td>1.2</td>
<td>56.2</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>19</td>
<td>3.8</td>
<td>177.9</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>30</td>
<td>6.0</td>
<td>280.8</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)*</td>
<td>52</td>
<td>10.4</td>
<td>486.8</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>11</td>
<td>2.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>3</td>
<td>0.6</td>
<td>28.1</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>5</td>
<td>1.0</td>
<td>46.8</td>
</tr>
<tr>
<td>Other Heart/Circulatory Malformations</td>
<td>11</td>
<td>2.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>152</td>
<td>30.4</td>
<td>1,422.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiovascular Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>19</td>
<td>3.8</td>
<td>49.5</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>45</td>
<td>9.0</td>
<td>117.3</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>46</td>
<td>9.2</td>
<td>119.9</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>3</td>
<td>0.6</td>
<td>7.8</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>13</td>
<td>2.6</td>
<td>33.9</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>30</td>
<td>6.0</td>
<td>78.2</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>11</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>32</td>
<td>6.4</td>
<td>83.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)*</td>
<td>142</td>
<td>28.4</td>
<td>370.2</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>44</td>
<td>8.8</td>
<td>114.7</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>5</td>
<td>1.0</td>
<td>13.0</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>55</td>
<td>11.0</td>
<td>143.4</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>447</td>
<td>89.4</td>
<td>1,165.2</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: *Anomalies per 100,000 refers to total number of anomalies rather than number of babies, as some babies have more than one anomaly; +Patent Ductus Arteriosus includes 31 Northland and 83 Waitemata cases of isolated PDA in preterm (<37 weeks) infants, which have been excluded from subsequent analyses; s: suppressed due to small numbers
Northern DHBs Distribution by Cause

In all four Northern DHBs during 2008–2012, patent ductus arteriosus was the most frequent cardiovascular anomaly identified at birth, although over half (range 57.6%–68.9%) were in preterm babies with no other cardiovascular anomalies. Atrial septal and ventricular septal defects were the next most frequent anomalies identified (Table 33, Table 34).

Table 34. Cardiovascular Anomalies Evident at Birth, Auckland and Counties Manukau DHB Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Cardiovascular Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>15</td>
<td>3.0</td>
<td>45.6</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>69</td>
<td>13.8</td>
<td>209.8</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>72</td>
<td>14.4</td>
<td>218.9</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>5</td>
<td>1.0</td>
<td>15.2</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>27</td>
<td>5.4</td>
<td>82.1</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>32</td>
<td>6.4</td>
<td>97.3</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)*</td>
<td>184</td>
<td>36.8</td>
<td>559.4</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>52</td>
<td>10.4</td>
<td>158.1</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>11</td>
<td>2.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>24</td>
<td>4.8</td>
<td>73.0</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>514</td>
<td>102.8</td>
<td>1,562.8</td>
</tr>
<tr>
<td><strong>Counties Manukau</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malformations Cardiac Chambers/Connections</td>
<td>29</td>
<td>5.8</td>
<td>67.4</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>29</td>
<td>5.8</td>
<td>67.4</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>45</td>
<td>9.0</td>
<td>104.6</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>4</td>
<td>0.8</td>
<td>9.3</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>5</td>
<td>1.0</td>
<td>11.6</td>
</tr>
<tr>
<td>Pulmonary/Tricuspid Valve Malformations</td>
<td>23</td>
<td>4.6</td>
<td>53.5</td>
</tr>
<tr>
<td>Aortic/Mitral Valve Malformations</td>
<td>12</td>
<td>2.4</td>
<td>27.9</td>
</tr>
<tr>
<td>Other Heart Malformations</td>
<td>32</td>
<td>6.4</td>
<td>74.4</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)*</td>
<td>183</td>
<td>36.6</td>
<td>425.4</td>
</tr>
<tr>
<td>Malformations Great Arteries (Excluding PDA)</td>
<td>21</td>
<td>4.2</td>
<td>48.8</td>
</tr>
<tr>
<td>Malformations Great Veins</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Other Peripheral Vascular Malformations</td>
<td>26</td>
<td>5.2</td>
<td>60.4</td>
</tr>
<tr>
<td>Other Circulatory Malformations</td>
<td>3</td>
<td>0.6</td>
<td>7.0</td>
</tr>
<tr>
<td>Total Malformations of the Circulatory System</td>
<td>414</td>
<td>82.8</td>
<td>962.3</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset; Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: *Anomalies per 100,000 refers to total number of anomalies rather than number of babies, as some babies have more than one anomaly; "Patent Ductus Arteriosus includes 106 Auckland and 126 Counties Manukau cases of isolated PDA in preterm (<37 weeks) infants, which have been excluded from subsequent analyses; s: suppressed due to small numbers
Figure 20. Babies with Cardiovascular Anomalies Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospital admissions with event type = birth and a cardiovascular anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more CVS anomalies, rather than the number of anomalies; *Numbers are per 2 year period, except for 2012 which is for a single year only; Preterm (<37 weeks) babies with Isolated Patent Ductus Arteriosus excluded.
Local Policy Documents and Evidence-Based Reviews Relevant to Congenital Heart Disease

In New Zealand there is a paucity of publications relevant to the prevention or management of congenital heart disease. Table 35 summarises a number of reviews which consider these issues in the overseas context. In addition, Table 16 on Page 79 considers publications relevant to antenatal and newborn screening, while Table 29 on Page 102 considers congenital anomalies collectively and Table 41 on Page 125 considers Down syndrome (which is of relevance as a high proportion of babies with Down syndrome also have congenital heart disease).

Table 35. Policy Documents and Evidence-Based Reviews Relevant to Cardiovascular Anomalies

<table>
<thead>
<tr>
<th>International Guidelines and Systematic and Other Reviews</th>
</tr>
</thead>
<tbody>
<tr>
<td>In 2007, the National Institute of Health Research Health Technology Assessment Programme in the U.K. commissioned a project to evaluate pulse oximetry as a screening procedure to detect major congenital heart defects (CHDs) in newborn infants. (Major CHDs are defined as those resulting in death or requiring invasive intervention in infancy and they are subdivided into critical defects which usually present in the first few days or weeks of life and serious CHDs which tend to present after 28 days of life.) The project comprised three distinct pieces of work which assessed the accuracy, acceptability (to parents and healthcare staff) and cost-effectiveness of pulse oximetry testing compared to existing strategies for detecting CHDs (antenatal ultrasound and newborn clinical examination). The test accuracy study included 20,055 newborns. Sensitivity of pulse oximetry was found to be 75.0% for critical cases and 49.1% for critical plus serious cases combined. The false positive rate was 0.84 % giving a specificity of 99.16%. Of those babies with a false positive test result, 3.5% had a significant but non-major heart defect and 24% had a respiratory or infective illness requiring hospital treatment. Overall, 37% of babies who had a positive test result had a condition requiring medical intervention. Parents and staff were predominantly satisfied with pulse oximetry screening and satisfaction was predicted by greater understanding of heart disease and the possibilities for treatment, and lower anxiety, stress and depression. The average time taken for pulse oximetry testing was 6.9 minutes (median 5 minutes, range 1 to 30 minutes) and each test was estimated to cost £6.24. Pulse oximetry plus clinical examination was twice as costly as clinical examination alone. The project concluded that pulse oximetry is a feasible, safe, simple, non-invasive and reasonably accurate test that is more sensitive than antenatal ultrasound screening and clinical examination and is likely to identify cases of critical CHD that would otherwise have been missed at an estimated cost of £24,000 per additional case. Pulse oximetry, like the other screening methods, misses some cases. Most of these are associated with obstruction of the aortic arch which may not produce hypoxaemia.</td>
</tr>
</tbody>
</table>

| This systematic review included data from 13 studies (229,421 babies) assessing the accuracy of pulse oximetry for the detection of critical congenital heart defects (CHDs) in newborn babies. The reviewers calculated sensitivity and specificity (with associated 95% CIs) for the individual studies and used a hierarchical summary receiver operating characteristics model to obtain summary estimates of sensitivity and specificity and also to assess whether the variability in test accuracy between studies was related to timing of test (< 24 hours vs. ≥ 24 hours after birth), method of testing (right hand and foot vs. foot only), oxygen saturation (functional vs. fractional) or antenatal screening (previously identified cases excluded vs. included). Overall sensitivity for the detection of critical CHDs was found to be 76.5% (95% CI 67.7–83.5). The specificity was 99.9% (95% CI 99.7–99.9) and the false positive rate 0.14% (95% CI 0.06%–0.33%). It was especially noteworthy that the false positive rate was significantly lower when pulse oximetry was done more than 24 hours after birth than less than 24 hours (0.05% [95% CI 0.02–0.12] vs. 0.50 [95% CI 0.29–0.86]; p=0.0017). The reviewers concluded that pulse oximetry meets criteria for universal screening by being highly specific and moderately sensitive. This review was reviewed by the Centre for Reviews and Dissemination (CRD) which stated that it was generally a well-conducted review and that the authors’ conclusions were likely to be reliable. The CRD review is available at: http://www.crd.york.ac.uk/CRDWeb/ShowRecord.asp?id=12012020804 |

| Abnormal flow in the ductus venosus (DV) can be detected via Doppler ultrasound in the first trimester of pregnancy, when nuchal translucency (NT) is measured, and it has been associated with adverse perinatal outcomes, chromosomal abnormalities and congenital heart disease (CHD). This review assessed the diagnostic performance of DV waveform examination as a screening technique for CHD in chromosomally normal fetuses with normal and also abnormal NT status. The review included seven studies assessing DV performance regardless of NT status (50,354 fetuses), nine where there was increased NT (n= 2908) and seven (n= 47,610) where NT was normal. Overall the summary sensitivity of DV for CHD detection was 50% (0.50, 95% CI 0.27–0.73) and the specificity 93% (95% CI 0.88–0.96). In participants with increased NT the sensitivity and specificity were 83% and 80% respectively and in those with normal NT they were 19% and 96%, respectively. |

This study from the U.K. was by far the largest of those included in the above meta-analysis. The aim of the study was to determine whether assessment of ductus venosus (DV) flow at 11–13 weeks could be used to improve the rate of detection of cardiac defects beyond that achieved by nuchal translucency (NT) measurements. The study population of chromosomally normal fetuses included 40,905 fetuses without heart defects and 85 with major heart defects. Of the fetuses with major heart defects, 35.3% had a NT above the 95th percentile and 21.2% above the 99th. By comparison, of the fetuses without major heart defects, 4.8% had a NT above the 95th percentile and 0.7% above the 99th. Reversed A wave on DV Doppler was seen in 28.2% of the fetuses with cardiac defects and 2.1% of those without. The authors state that providing specialist echocardiography to those with NT above the 99th percentile and those with reversed A wave on DV Doppler, regardless of NT value, would detect 38.8% of major cardiac defects at an overall false positive rate of 2.7%. They concluded that assessment of DV flow improves the performance of NT screening for cardiac defects by about 10% and they suggest that, ideally, fetal echocardiography should be offered to all those with reversed A wave and those with NT above the 95th percentile. Where there are insufficient resources to achieve this, specialist examination could be limited to those with NT above the 99th percentile and those with abnormal DV Doppler.


Section 9.1 of this publication covers screening for structural anomalies and reviews studies relating to the use of ultrasound and serum screening (alpha-fetoprotein, AFP) for this purpose. One systematic review (of 5 studies) and two other studies looked at foetal echocardiography. There was a wide range of reported values for sensitivity by centre and condition but the reported specificity was generally high. There was some evidence from 2 uncontrolled observational studies that babies with transposition of the great arteries (and possibly hypoplastic left heart syndrome) diagnosed prenatally had reduced mortality compared with those diagnosed postnatally. Nuchal translucency measurement seemed to have poor diagnostic value for detecting cardiac anomalies.

A short version of this guideline can be found at: http://www.nice.org.uk/nicemedia/live/11947/40115/40115.pdf

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Introduction

Down syndrome is the most common chromosomal anomaly in newborn babies and it results from extra genetic material from chromosome 21. In around 95% of cases there is an extra copy of chromosome 21 (Trisomy 21) and in about 4% there are 46 chromosomes as usual, but there is a translocation between chromosome 21q and another chromosome (usually chromosome 14 or 22). The risk of having a child with Trisomy 21, but not translocation Down syndrome, rises steeply with maternal age. There is a relatively high recurrence risk for translocation Down syndrome when a parent, especially the mother, is a carrier of the translocation [38].

In New Zealand, between 50 and 80 babies with Down syndrome are born each year [39]. Worldwide the incidence of Down syndrome is around 1 per 1,000 live births, with variations from country to country depending largely on average maternal age and attitudes to prenatal testing and termination [40].

In New Zealand, the Ministry of Health requires that all pregnant women be informed about antenatal screening for Down syndrome and that practitioners support and respect women’s screening choices [22]. The antenatal screening guidelines published by the National Screening Unit recommend that women presenting in their first trimester be offered a nuchal translucency scan, plus a blood test measuring two maternal serum markers, while women who present later, be offered a blood test measuring four serum markers [22]. Women whose test results indicate an increased risk (> 1 in 300) are offered referral for more definitive testing [22].

Children born with Down syndrome usually have a distinctive facial appearance, low muscle tone and delayed development. They are at risk of a number of medical problems including hearing loss (75%), vision problems (severe refractive errors in 50% and cataracts in 15%), obstructive sleep apnoea (50–75%), congenital heart defects (50%), gastrointestinal atresias (12%), thyroid disease (4–18%) and seizures (1–13%) [41]. There are a number of guidelines on the clinical care of children with Down syndrome [40,41,42,43], including one from the Ministry of Health [44]. They suggest that in addition to continuing surveillance for medical, dental, developmental and behavioural problems, children with Down syndrome and their families also require a range of special education and disability support services.

The following section uses the National Minimum Dataset to review the number of babies born with Down syndrome. The section concludes with a brief overview of local policy documents and evidence-based reviews which are of relevance in this context.

### Data Source and Methods

**Definition**

1. Number of chromosomal anomalies identified at birth (by anomaly type)
2. Number of babies with Down syndrome identified at birth

**Data Source**

1. National Minimum Dataset

Numeratior: Hospital admissions with event type = birth and a chromosomal anomaly (ICD-10 Q90–Q99) listed in any of the first 15 diagnoses.

For this indicator, the unit of analysis is the number of chromosomal anomalies rather than the number of babies. Specific anomalies include: Down Syndrome (Q90), Edwards and Patau Syndromes (Q91), Other Autosomal Trisomies (Q92), Monosomies and Autosomal Deletions/Other Rearrangements (Q93, Q95), Turner's Syndrome (Q96), Other Sex Chromosome Anomalies Female Phenotype (Q97), Sex Chromosome Anomalies Male Phenotype (Q98), Other Chromosome Anomalies (Q99)
New Zealand Distribution and Trends

Chromosomal Anomalies Evident at Birth

In New Zealand during 2008–2012, Down syndrome was the most frequent chromosomal anomaly identified at birth, accounting for 65.6% of the chromosomal anomalies identified during this period. These figures however, may significantly underestimate the prevalence of chromosomal anomalies, as in the absence of karyotyping, many anomalies (e.g. sex chromosome anomalies) may be undetectable by routine newborn examination (Table 36).

Table 36. Chromosomal Anomalies Evident at Birth, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Chromosomal Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Down Syndrome</td>
<td>257</td>
<td>51.4</td>
<td>85.01</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>40</td>
<td>8.0</td>
<td>13.23</td>
</tr>
<tr>
<td>Other Autosomal Trisomies</td>
<td>15</td>
<td>3.0</td>
<td>4.96</td>
</tr>
<tr>
<td>Turners Syndrome</td>
<td>10</td>
<td>2.0</td>
<td>3.31</td>
</tr>
<tr>
<td>Sex Chromosome Anomalies Male Phenotype</td>
<td>12</td>
<td>2.4</td>
<td>3.97</td>
</tr>
<tr>
<td>Monosomies/Autosomal Deletions/Other Rearrangements</td>
<td>25</td>
<td>5.0</td>
<td>8.27</td>
</tr>
<tr>
<td>Other Chromosome Anomalies</td>
<td>33</td>
<td>6.6</td>
<td>10.91</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>392</td>
<td>78.4</td>
<td>129.66</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a chromosomal anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly

Babies with Down Syndrome and Other Congenital Anomalies

In New Zealand during 2008–2012, 45.9% of babies with Down syndrome identified at the time of birth had one or more co-existing cardiovascular anomalies, most frequently patent ductus arteriosus and atrial septal defects. A smaller proportion had anomalies of other organ systems (Table 37).

New Zealand Trends

In New Zealand during 2000–2012, on average 53 babies per year were identified as having Down syndrome at the time of birth, with numbers fluctuating during this period (Figure 21).

Distribution by Maternal Age

In New Zealand during 2008–2012, while the largest absolute number of babies with Down syndrome were born to women aged 35–39 years, Down syndrome rates rose exponentially with increasing maternal age, so that the highest rates were seen in the babies of mothers aged 40+ years (Figure 22).
Table 37. Babies with Down Syndrome who also had Other Congenital Anomalies Evident at Birth, New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Congenital Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>% of Babies with Down Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Babies with Down Syndrome (n=257)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>One or more Malformations of the Nervous System</td>
<td>4</td>
<td>1.6</td>
</tr>
<tr>
<td>One or more Malformations of Eye, Ear, Face and Neck</td>
<td>9</td>
<td>3.5</td>
</tr>
<tr>
<td>One or more Malformations of the Circulatory System*</td>
<td>118</td>
<td>45.9</td>
</tr>
<tr>
<td>One or more Malformations of the Respiratory System</td>
<td>5</td>
<td>1.9</td>
</tr>
<tr>
<td>One or more Other Malformations of the Digestive System</td>
<td>18</td>
<td>7.0</td>
</tr>
<tr>
<td>One or more Malformations of the Genital Organs</td>
<td>8</td>
<td>3.1</td>
</tr>
<tr>
<td>One or more Malformations of the Urinary System</td>
<td>3</td>
<td>1.2</td>
</tr>
<tr>
<td>One or more Malformations of the Musculoskeletal System</td>
<td>13</td>
<td>5.1</td>
</tr>
<tr>
<td>One or more Other Congenital Malformations</td>
<td>5</td>
<td>1.9</td>
</tr>
</tbody>
</table>

*Babies with Down Syndrome and a Circulatory System Anomaly (n=118)

<table>
<thead>
<tr>
<th>Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>% of Babies with Down Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular Septal Defect</td>
<td>19</td>
<td>7.4</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>55</td>
<td>21.4</td>
</tr>
<tr>
<td>Atrioventricular Septal Defect</td>
<td>24</td>
<td>9.3</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>5</td>
<td>1.9</td>
</tr>
<tr>
<td>One or more Pulmonary/Tricuspid Valve Malformations</td>
<td>5</td>
<td>1.9</td>
</tr>
<tr>
<td>One or more Aortic/Mitral Valve Malformations</td>
<td>3</td>
<td>1.2</td>
</tr>
<tr>
<td>One or more Other Heart Malformations</td>
<td>7</td>
<td>2.7</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>56</td>
<td>21.8</td>
</tr>
<tr>
<td>One or more Malformations Great Arteries (Excluding PDA)</td>
<td>8</td>
<td>3.1</td>
</tr>
<tr>
<td>One or more Other Circulatory Malformations</td>
<td>&lt;3</td>
<td>s</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of the first 15 diagnoses, plus another congenital anomaly listed in first 15 diagnoses; Denominator: All hospitalisations with event type = birth and Down syndrome listed in any of the first 15 diagnoses; Note: Numbers and percentages do not sum to 100%, as some babies have more than one anomaly for each of the systems listed; s: suppressed due to small numbers
Figure 21. Babies with Down Syndrome Evident at Birth, New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth

Figure 22. Babies with Down Syndrome Evident at Birth by Maternal Age, New Zealand Hospital Births 2008–2012

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth
## Distribution by Prioritised Ethnicity, NZDep Index Decile and Gender

In New Zealand during 2008–2012, there were no statistically significant socioeconomic, (as measured by NZDep06 quintile), ethnic or gender differences in the proportion of babies identified with Down syndrome at the time of birth. Rates however, were **significantly** higher for the babies of older women, with rates for the babies of mothers aged 40+ years being 25.68 (95% CI 9.36–70.45) times higher than for the babies of teenage mothers (Table 38). During 2000–2012, large variations in rates (possibly as the result of small numbers) made ethnic specific trends difficult to interpret, although rates were generally lower for Māori, than for European babies, for the majority of this period (Figure 23).

Table 38. Babies with Down Syndrome Evident at Birth by Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of Babies: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Down Syndrome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>9.4</td>
<td>70.45</td>
<td>0.76</td>
<td>0.55–1.05</td>
</tr>
<tr>
<td>Pacific</td>
<td>6.2</td>
<td>91.44</td>
<td>0.98</td>
<td>0.67–1.45</td>
</tr>
<tr>
<td>European/Other</td>
<td>30.8</td>
<td>92.89</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>4.8</td>
<td>70.16</td>
<td>0.76</td>
<td>0.49–1.16</td>
</tr>
<tr>
<td><strong>NZ Deprivation Index</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>8.0</td>
<td>94.29</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>8.2</td>
<td>90.92</td>
<td>0.96</td>
<td>0.62–1.49</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>8.2</td>
<td>72.79</td>
<td>0.77</td>
<td>0.50–1.19</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>11.6</td>
<td>82.23</td>
<td>0.87</td>
<td>0.58–1.30</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>15.2</td>
<td>86.79</td>
<td>0.92</td>
<td>0.63–1.35</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>22.8</td>
<td>77.57</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28.6</td>
<td>92.06</td>
<td>1.19</td>
<td>0.93–1.52</td>
</tr>
<tr>
<td><strong>Maternal Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 Years</td>
<td>0.8</td>
<td>19.16</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>20–24 Years</td>
<td>5.0</td>
<td>44.78</td>
<td>2.34</td>
<td>0.81–6.71</td>
</tr>
<tr>
<td>25–29 Years</td>
<td>5.8</td>
<td>39.08</td>
<td>2.04</td>
<td>0.72–5.80</td>
</tr>
<tr>
<td>30–34 Years</td>
<td>9.2</td>
<td>55.06</td>
<td>2.87</td>
<td>1.03–7.98</td>
</tr>
<tr>
<td>35–39 Years</td>
<td>17.4</td>
<td>159.76</td>
<td>8.34</td>
<td>3.06–22.71</td>
</tr>
<tr>
<td>40+ Years</td>
<td>13.2</td>
<td>492.17</td>
<td>25.68</td>
<td>9.36–70.45</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Rate Ratios are unadjusted
Figure 23. Babies with Down Syndrome Evident at Birth by Ethnicity, New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset; Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Ethnicity is Level 1 Prioritised

**Northern DHBs Distribution**

**Northern DHBs Distribution**

In the Northern region during 2008–2012, 10 Northland, 32 Waitemata, 29 Auckland and 44 Counties Manukau babies were identified as having Down syndrome at the time of birth, with a small number of babies also having other chromosomal anomalies (Table 39). The proportion of babies identified with Down syndrome at the time of birth in all four Northern DHBs was not significantly different from the New Zealand rate (Table 40).

**Northern DHBs Trends**

In the Northern DHBs during 2000–2012, large year to year variations (likely as the result of small numbers) made trends in Down syndrome rates difficult to interpret (Figure 24).
Table 39. Chromosomal Anomalies Evident at Birth, Northern DHBs 2008–2012

<table>
<thead>
<tr>
<th>Chromosomal Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Northland</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>10</td>
<td>2.0</td>
<td>93.61</td>
</tr>
<tr>
<td>Other Chromosomal Anomalies</td>
<td>6</td>
<td>1.2</td>
<td>56.16</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>16</td>
<td>3.2</td>
<td>149.77</td>
</tr>
<tr>
<td><strong>Waitemata</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>32</td>
<td>6.4</td>
<td>83.41</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>6</td>
<td>1.2</td>
<td>15.64</td>
</tr>
<tr>
<td>Other Chromosomal Anomalies</td>
<td>6</td>
<td>1.2</td>
<td>15.64</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>44</td>
<td>8.8</td>
<td>114.69</td>
</tr>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>29</td>
<td>5.8</td>
<td>88.17</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>3</td>
<td>0.6</td>
<td>9.12</td>
</tr>
<tr>
<td>Sex Chromosome Anomalies</td>
<td>4</td>
<td>0.8</td>
<td>12.16</td>
</tr>
<tr>
<td>Monosomies/Autosomal Deletions/Rearrangements</td>
<td>4</td>
<td>0.8</td>
<td>12.16</td>
</tr>
<tr>
<td>Other Chromosomal Anomalies</td>
<td>3</td>
<td>0.6</td>
<td>9.12</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>43</td>
<td>8.6</td>
<td>130.73</td>
</tr>
<tr>
<td><strong>Counties Manukau</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Down Syndrome</td>
<td>44</td>
<td>8.8</td>
<td>102.28</td>
</tr>
<tr>
<td>Edwards and Patau Syndromes</td>
<td>5</td>
<td>1.0</td>
<td>11.62</td>
</tr>
<tr>
<td>Other Chromosomal Anomalies</td>
<td>8</td>
<td>1.6</td>
<td>18.58</td>
</tr>
<tr>
<td>Total Chromosomal Anomalies</td>
<td>57</td>
<td>11.4</td>
<td>132.48</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a chromosomal anomaly in any of the first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have >1 anomaly

Table 40. Babies with Down Syndrome Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Down Syndrome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>10</td>
<td>2.0</td>
<td>93.61</td>
<td>1.10</td>
<td>0.59–2.07</td>
</tr>
<tr>
<td>Waitemata</td>
<td>32</td>
<td>6.4</td>
<td>83.41</td>
<td>0.98</td>
<td>0.68–1.42</td>
</tr>
<tr>
<td>Auckland</td>
<td>29</td>
<td>5.8</td>
<td>88.17</td>
<td>1.04</td>
<td>0.71–1.52</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>44</td>
<td>8.8</td>
<td>102.28</td>
<td>1.20</td>
<td>0.87–1.66</td>
</tr>
<tr>
<td>New Zealand</td>
<td>257</td>
<td>51.4</td>
<td>85.01</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics
Figure 24. Babies with Down Syndrome Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and Down syndrome listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: *Numbers are per 2 year period, except for 2012 which is for a single year only
Local Policy Documents and Evidence-Based Reviews Relevant to Down Syndrome and Other Chromosomal Anomalies

In New Zealand there are a number of policy documents relevant to the diagnosis and management of those with Down syndrome. These are summarized in Table 41, along with a range of reviews which consider these issues in the overseas context. In addition, Table 16 on Page 79 considers publications relevant to antenatal and newborn screening, while Table 29 on Page 102 considers congenital anomalies collectively, and Table 35 on Page 115 considers cardiovascular anomalies, which are known to be much higher in those with Down syndrome.

Table 41. Local Policy Documents and Evidence-Based Reviews Relevant to Down Syndrome and Other Chromosomal Anomalies

<table>
<thead>
<tr>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>These guidelines are intended for all practitioners who have involvement in any part of the antenatal screening process for Down syndrome and other conditions. Under the Primary Maternity Services Notice 2007, issued pursuant to section 88 of the New Zealand Public Health and Disability Act 2000, all health practitioners advising women about maternity care are obliged to provide advice about the available screening services that are endorsed by the Ministry of Health, including antenatal screening for Down syndrome and other conditions. The advice must include up to date information about the risks, benefits and harms of screening so that a woman can make an informed choice about whether to participate in screening or not and a woman's choice must be respected. Specific guidelines for first trimester ultrasound reports can be found here: <a href="http://www.nsu.govt.nz/health-professionals/3814.aspx">http://www.nsu.govt.nz/health-professionals/3814.aspx</a>.</td>
</tr>
<tr>
<td>This report reviewed the main issues relating to antenatal screening for Down syndrome and considered possible screening options. The members of the advisory group agreed that the opportunistic screening practice current in 2007 was unsafe, inequitable, not in accord with international best practice and resulted in women unnecessarily being referred for invasive diagnostic tests (chorionic villus sampling and amniocentesis) which have a risk of miscarriage. The members of the group were not able to agree on the best way to proceed with screening. A large majority wished to continue to offer screening and to improve the quality and safety of screening tests by means of a nationally organised screening programme. They offered suggestions on the best practice screening methods in particular situations. A minority of group members did not support the introduction of a national screening programme as they felt it would imply an intention to reduce the incidence of Down syndrome. They considered that the best option was to recommend additional funding for disability support services.</td>
</tr>
<tr>
<td>This literature review was performed at the request of the Ministry of Health to assist with assessing options for antenatal Down syndrome screening and determining whether or not New Zealand should have a national antenatal screening programme. The review appraised the international evidence on screening technologies and strategies. Key results of the literature review are presented under the following headings: Accuracy of screening methods, Difficulties in implementing any screening strategies, Uptake of invasive testing following receipt of screening results, Changes in the rate of invasive testing with the introduction of a screening programme and Rates of fetal loss associated with invasive testing procedures.</td>
</tr>
<tr>
<td>This document provides information and guidance on the medical management of Down syndrome throughout the lifespan as well as on therapy, education, vocational and social support for people with Down syndrome and their families. Part 1 provides a general overview of Down syndrome including the major clinical features, incidence, genetics and a brief Maori perspective. Part 2 provides recommendations for care at the various stages of life.</td>
</tr>
</tbody>
</table>
There is a higher risk of chromosomal abnormalities in twin pregnancies, mostly because twin pregnancies are more common in older mothers. Screening for Down syndrome is more complicated in twin pregnancies. Twin pregnancies may be either monozygotic (a single fertilized oocyte splits in two producing genetically identical twins) or dizygotic (when two separate oocytes are fertilized). Dizygotic twins are always dichorionic (a separate placenta for each twin) but monozygotic twins may be either dichorionic (about 33% of cases) or monochorionic (both twins share one placenta, about 66% of cases). Chorionicity has a major impact on the screening process and so needs to be determined by ultrasound in the first trimester. These Canadian guidelines provide evidence-based recommendations on prenatal screening for and diagnosis of fetal aneuploidy (e.g. Down syndrome and trisomy 18) in twin pregnancies. The evidence is graded and the recommendations are classified using criteria adapted from those provided in the Canadian Task Force on Preventive Health Care.


There is a higher risk of chromosomal abnormalities in twin pregnancies, mostly because twin pregnancies are more common in older mothers. Screening for Down syndrome is more complicated in twin pregnancies. Twin pregnancies may be either monozygotic (a single fertilized oocyte splits in two producing genetically identical twins) or dizygotic (when two separate oocytes are fertilized). Dizygotic twins are always dichorionic (a separate placenta for each twin) but monozygotic twins may be either dichorionic (about 33% of cases) or monochorionic (both twins share one placenta, about 66% of cases). Chorionicity has a major impact on the screening process and so needs to be determined by ultrasound in the first trimester. These Canadian guidelines provide evidence-based recommendations on prenatal screening for and diagnosis of fetal aneuploidy (e.g. Down syndrome and trisomy 18) in twin pregnancies. The evidence is graded and the recommendations are classified using criteria adapted from those provided in the Canadian Task Force on Preventive Health Care.


The guidelines from the American Academy of Pediatrics are intended for paediatricians who care for children with Down syndrome or may be involved in counselling a pregnant woman who has received a prenatal diagnosis of Down syndrome. They provide recommendations for health supervision at different stages: prenatally, newborn, infancy, early childhood, late childhood, and adolescence to early adulthood. There is a comprehensive list of references and links to resources for parents.


This literature survey was commissioned by the Fetal Anomaly Screening Programme Steering Group (FASPSG) to inform the policy review of Down Syndrome screening in the U.K. taking place in 2010. It covers screening strategies, specific ultrasound features and serum markers, screening between 8 and 10 weeks gestation, and screening for trisomy 13 and trisomy 18 both prior to 14 weeks of gestation and at 18 to 21 weeks (for comparison purposes).


These concise document prepared by committees of the Society of Obstetricians and Gynaecologists of Canada and the Canadian College of Medical Geneticists provides evidence-based recommendations on screening for Fragile X in women receiving obstetric or gynaecological healthcare. The evidence and the recommendations are graded according to the criteria of the Canadian Taskforce on Preventive Health Care.


These guidelines are the result of a meeting of the Turners Syndrome Consensus Study Group (multidisciplinary panel of experts with relevant clinical and research experience) that met in Maryland in April 2006. The group used peer reviewed published information as the basis for its recommendations and expert opinion where evidence was lacking. The guidelines cover genetic, cardiological, growth-related, psychological, gynaecological, and general medical concerns.
Fetal cells for genetic testing can be obtained from amniotic fluid or the placenta (chorionic villus tissue). Amniocentesis involves inserting a needle through the abdominal wall to withdraw a sample of amniotic fluid (usually under ultrasound guidance). It is usually done at around 16 weeks’ gestation but can be done earlier (at 9–14 weeks). Chorionic villus sampling (CVS) involves aspiration of placential tissue via either a transabdominal or a transcervical approach. It is usually done late in the first trimester. This review assessed the comparative safety and accuracy of second trimester amniocentesis, early amniocentesis, transabdominal and transcervical CVS. Sixteen RCTs were included. One study in a low-risk population (4606 women aged between 25 and 34) found that amniocentesis (compared to routine antenatal care) was associated with an increase of 1% in total pregnancy loss (3.2% vs. 2.2%), which was not statistically significant (1.41; 95% CI 0.99 to 2.00), and an increase in spontaneous miscarriages of 0.8% (2.1% vs. 1.3%) which was statistically significant (RR 1.60; 95% CI 1.02 to 2.52). Compared to second trimester amniocentesis, early amniocentesis is associated with greater total pregnancy loss (RR 1.29; 95% CI 1.03 to 1.61) and a higher number of babies with talipes equinovarus (clubfoot). 1.3% vs. 0.09%. Compared to second trimester amniocentesis, transcervical CVS increased the risk of total pregnancy loss, mainly due to spontaneous miscarriages although there was heterogeneity between studies. The one study that compared second trimester amniocentesis with transabdominal CVS found no significant difference in pregnancy loss rates between the two procedures. Compared to transabdominal CVS, transcervical CVS is more technically demanding and more likely to cause vaginal bleeding immediately after the procedure. The authors were unable to assess the diagnostic accuracies of the various techniques because of incomplete karyotype data in most studies. They concluded that amniocentesis the preferred procedure for second trimester testing and transabdominal CVS is the first choice procedure for testing before 15 weeks gestation.

Other Relevant Publications and Websites

<table>
<thead>
<tr>
<th>Title</th>
<th>Year</th>
</tr>
</thead>
</table>

This is a brief publication intended for pregnant women with information about antenatal screening and testing for Down Syndrome and other conditions.


Maternal serum contains cell free DNA and a small proportion of this is fetal cell free DNA, thought to be derived primarily from the placenta. There is new technology known as massively parallel genomic sequencing which can detect trisomy 21 (Down syndrome), trisomy 13 and trisomy 18 as early as the 10th week of pregnancy via a maternal blood test. This has the advantages of reducing the need for amniocentesis and allowing termination early in pregnancy (if desired). This committee opinion from the American College of Obstetricians and Gynecologists (ACOG) notes the great potential of this technology as a screening tool. It recommends that such testing should not be part of routine antenatal care for low risk women or women with multiple pregnancies because it has not been sufficiently well evaluated in these groups, but offered to women at increased risk of aneuploidy. In these women several large studies have shown detection rates for trisomy 13, trisomy 18, and trisomy 21 of greater than 98% with very low false-positive rates (less than 0.5%). Women at increased risk of aneuploidy include women aged over 35 years, women with suspicious ultrasound findings, women with a previous affected child, and cases where a parent carries a balanced Robertsonian translocation with increased risk of trisomy 13 or trisomy 21. The ACOG states that the test can also be used as a follow up test for patients who have abnormal results on current first or second trimester screening tests. It recommends that women with positive cell free DNA test should be referred for genetic counselling and offered invasive testing to confirm the test results (amniocentesis or chorionic villus sampling). This publication includes references to the relevant studies.

The test referred to above is commercially available as MateriNT21 PLUS™. Information about the test from Sequenom, the company that developed the test, can be found here: [http://www.sequenomcmm.com/Home/Health-Care-Professionals/Trisomy-21/About-the-Test](http://www.sequenomcmm.com/Home/Health-Care-Professionals/Trisomy-21/About-the-Test).


The Down's Syndrome Medical Interest Group (DSMIG) is a network of doctors from the UK and the Republic of Ireland who have a specialist interest in Down's syndrome. The DSMIG has produced evidence-based surveillance guidelines for cardiac disease, thyroid dysfunction, hearing and vision disorders, growth and cervical spine instability as well as number of other useful publications. Also on their website is a library of relevant publications from the medical literature.


Useful publications on this website include:

- EDSA Essentials 2 Health Care Guidelines for People with Down Syndrome
- EDSA Essentials 3 The Person with Down Syndrome: Orientations for Families

Down Syndrome Education International is a UK-based charity that works to improve education for young people with Down syndrome through scientific research and global information and advice services. It publishes the journal Down Syndrome Research and Practice.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Neural Tube Defects

Introduction

Neural tube defects (NTDs) have their origin very early in pregnancy. A localised thickening of embryonic cells known as the neural plate is observable approximately 18 days after conception. This elongates and develops a central groove and then the edges of the groove fold to produce the neural tube. The neural tube closes at around day 25 at the cranial end and at around day 27 at the sacral end [45]. The walls of the neural tube thicken to produce the brain and spinal cord. The closed neural tube stimulates the development of the bony structures of the vertebral column and skull. Neural tube defects are the malformations that occur when the neural tube fails to close and the bone fails to form above an unclosed region of the neural tube [46]. Defects may occur anywhere along the neural axis. Cranial defects include anencephaly (a lethal defect) and the rarer conditions encephalocele. Defects elsewhere in the neural tube result in spina bifida.

Spina bifida occulta is a common and often asymptomatic anomaly in which there is a midline defect in the vertebral bodies without protrusion of the spinal cord or meninges. When the meninges herniate through a defect in the posterior vertebral arches or the anterior sacrum a meningomyelocele is formed. These defects are mostly well covered with skin. They require surgical correction but the spinal cord is usually normal and the prognosis is good [47]. The most severe form of neural tube defect is myelomeningocele where there is protrusion of central nervous system tissue through the defect in the vertebral column. This condition most commonly occurs in the lumbosacral region (75% of cases) where it is associated motor and sensory disabilities in the lower limbs and bowel and bladder dysfunction [48]. Surgery in the newborn period allows most children with myelomeningocele to survive and most survivors have normal intelligence (>70%). Many can walk with assistive devices when they are young but ambulation becomes more difficult with age and increasing body mass. The most life-threatening aspect of the condition long term is renal dysfunction as a result of a neurogenic bladder [47].

Since the publication of the results of the MRC international multicentre randomised controlled trial of periconceptional folic acid supplementation in 1991 [49] it has been accepted that folic acid supplementation can prevent up to two thirds of NTDs [45]. In many countries, including the U.S., Canada and Australia, there is mandatory fortification of staple foods (flours) with folic acid [50]. In New Zealand voluntary fortification of certain foods (cereal products, bread and fruit juice) with folic acid is permitted and folic acid supplementation for women planning a pregnancy is recommended. The Ministry of Health recommends that women at low risk of having a child with an NTD take 800µg of folic acid daily for at least four weeks prior to conception and for 12 weeks after. High risk women, including those who have previously had a pregnancy affected by a NTD, those who have a family history of NTD in their own or their partner's family, those with insulin dependent diabetes and those taking medications known to affect folate metabolism (including the anti-epileptic drugs valproate and carbamazepine) are recommended to take 5mg of folic acid daily over the same period. Subsidised 800 µg and 5mg folic acid tablets are available over the counter from pharmacies [51,52].

The following section uses the National Minimum Dataset to review the number of babies born with neural tube defects. The section concludes with a brief review of policy documents and evidence-based reviews which are relevant to their prevention.
Neural Tube Defects

Data Source and Methods

Definition

1. Number of central nervous system anomalies identified at birth (by anomaly type)
2. Number of babies with neural tube defects (NTD) identified at birth

Data Source

1. National Minimum Dataset

Numerator: Hospital admissions with event type = birth and a nervous system anomaly (ICD-10 Q00–07) listed in any of the first 15 diagnoses.

For this indicator, the unit of analysis is the number of nervous system anomalies rather than the numbers of babies, as some babies have more than one anomaly. Specific anomalies include: Anencephaly (Q00), Encephalocele (Q01), Microcephaly (Q02), Congenital Hydrocephalus (Q03), Other Brain Malformations (Q04), Spina Bifida (Q05), Other Spinal Cord Malformations (Q06), Other CNS Malformations (Q07).

2. National Minimum Dataset

Numerator: Hospital admissions with event type = birth and a neural tube defect listed in any of the first 15 diagnoses.

For this indicator, the unit of analysis is the number of babies with one or more NTDs rather than the numbers of NTDs. Specific NTDs include: Anencephaly (Q00), Encephalocele (Q01), and Spina Bifida (Q05).

Denominator: All Hospital Admissions with event type = birth.

Notes on Interpretation

Note: This analysis includes all admissions recorded in the National Minimum Dataset (NMDS) where the Event Type was listed as Birth. In the NMDS only one birth event is allowed per NHI number, with admissions for babies born prior to hospital admission, or readmitted shortly after discharge being listed as a routine inpatient event. Thus the analysis does not include babies born prior to hospital admission, babies born at home, or babies whose NTD was overlooked at the time of discharge, but who re-presented shortly thereafter.

New Zealand Distribution and Trends

New Zealand Distribution

In New Zealand during 2008–2012, 79 neural tube defects (NTDs) were identified at the time of birth (anencephaly (n=13), encephalocele (n=13), spina bifida (n=53)), with on average 16 NTDs being identified per year. NTDs accounted for 17.8% of all nervous system anomalies identified during this period. Note: The unit of analysis is the number of NTDs, rather than the number of babies with one or more NTD (Table 42).

Table 42. Nervous System Anomalies Evident at Birth, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Nervous System Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Anomalies per 100,000 Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly (NTD)</td>
<td>13</td>
<td>2.6</td>
<td>4.30</td>
</tr>
<tr>
<td>Encephalocele (NTD)</td>
<td>13</td>
<td>2.6</td>
<td>4.30</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>61</td>
<td>12.2</td>
<td>20.18</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>77</td>
<td>15.4</td>
<td>25.47</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>190</td>
<td>38.0</td>
<td>62.85</td>
</tr>
<tr>
<td>Spina Bifida (NTD)</td>
<td>53</td>
<td>10.6</td>
<td>17.53</td>
</tr>
<tr>
<td>Other Spinal Cord Malformations</td>
<td>14</td>
<td>2.8</td>
<td>4.63</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>24</td>
<td>4.8</td>
<td>7.94</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>445</td>
<td>89.0</td>
<td>147.20</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset; Numerator: Hospitalisations with event type = birth and a nervous system anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have more than one anomaly; NTD denotes neural tube defect
Figure 25. Babies with Neural Tube Defects Evident at Birth, New Zealand Hospital Births 2000–2012

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a neural tube defect listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more neural tube defects

Figure 26. Babies with Neural Tube Defects Evident at Birth by Maternal Age, New Zealand Hospital Births 2008–2012

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a neural tube defect listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more neural tube defects
New Zealand Trends
In New Zealand during 2000–2012, on average 13.5 babies per year had one or more neural tube defects identified at the time of birth. Large year to year variations (possibly as a result of small numbers) made trends in NTD rates difficult to interpret (Figure 25).

Distribution by Maternal Age
In New Zealand during 2008–2012, the babies of teenage mothers had the highest neural tube defect rates, although in most cases differences between the babies of teenage and older mothers did not reach statistical significance. In contrast, mothers aged 30–34 years had the largest actual number of babies born with a NTD (as a result of the higher number of overall births in this age group (Figure 26, Table 43)).

Distribution by Prioritised Ethnicity, NZDep Index Decile and Gender
In New Zealand during 2008–2012, there were no significant ethnic, socioeconomic (as measured by NZDep06 quintile) or gender differences in the proportion of babies born with a neural tube defect. The highest rates however, were seen amongst Pacific babies, the babies of teenage mothers, and those born into the most deprived (NZDep06 deciles 9–10) areas (Table 43).

Table 43. Babies with Neural Tube Defects Evident at Birth by Ethnicity, NZ Deprivation Index Decile, Gender and Maternal Age, New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of Babies: Annual Average</th>
<th>Rate per 100,000 Births</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neural Tube Defects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>4.2</td>
<td>31.48</td>
<td>1.45</td>
<td>0.85–2.48</td>
</tr>
<tr>
<td>Pacific</td>
<td>2.4</td>
<td>35.40</td>
<td>1.63</td>
<td>0.85–3.13</td>
</tr>
<tr>
<td>European/Other</td>
<td>7.2</td>
<td>21.71</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>1.6</td>
<td>23.39</td>
<td>1.08</td>
<td>0.50–2.32</td>
</tr>
<tr>
<td>NZ Deprivation Index</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>2.0</td>
<td>23.57</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>2.0</td>
<td>22.18</td>
<td>0.94</td>
<td>0.39–2.26</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>3.2</td>
<td>28.41</td>
<td>1.21</td>
<td>0.55–2.66</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>2.8</td>
<td>19.85</td>
<td>0.84</td>
<td>0.37–1.90</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>5.4</td>
<td>30.83</td>
<td>1.31</td>
<td>0.63–2.70</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>7.0</td>
<td>23.81</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>8.6</td>
<td>27.68</td>
<td>1.16</td>
<td>0.74–1.82</td>
</tr>
<tr>
<td>Maternal Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 Years</td>
<td>2.0</td>
<td>47.91</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>20–24 Years</td>
<td>2.2</td>
<td>19.70</td>
<td>0.41</td>
<td>0.17–0.97</td>
</tr>
<tr>
<td>25–29 Years</td>
<td>3.4</td>
<td>22.91</td>
<td>0.48</td>
<td>0.22–1.04</td>
</tr>
<tr>
<td>30–34 Years</td>
<td>3.8</td>
<td>22.74</td>
<td>0.47</td>
<td>0.22–1.02</td>
</tr>
<tr>
<td>35–39 Years</td>
<td>3.6</td>
<td>33.05</td>
<td>0.69</td>
<td>0.32–1.49</td>
</tr>
<tr>
<td>40+ Years</td>
<td>0.6</td>
<td>22.37</td>
<td>0.47</td>
<td>0.13–1.70</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a neural tube defect listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Rate Ratios are unadjusted; Rate per 100,000 refers to number of babies with one or more neural tube defects.
Northern DHBs Distribution

In the Northern DHBs (combined) during 2008–2012, a total of 5 cases anencephaly, 9 of encephalocele and 22 of spina bifida were identified at the time of birth, with these accounting for 17.9% of all nervous system anomalies during this period (Table 44). In the Waitemata, Auckland and Counties Manukau DHBs, rates were not significantly different from the New Zealand rate, while in Northland, numbers were too small to make any meaningful comparisons (Table 45).

Table 44. Nervous System Anomalies Evident at Birth, Northern DHBs 2008–2012

<table>
<thead>
<tr>
<th>Nervous System Anomaly</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Number of Anomalies per 100,000Births*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Northland</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microcephaly</td>
<td>3</td>
<td>0.6</td>
<td>28.08</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>4</td>
<td>0.8</td>
<td>37.44</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>11</td>
<td>2.2</td>
<td>102.97</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>4</td>
<td>0.8</td>
<td>37.44</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>22</td>
<td>4.4</td>
<td>205.93</td>
</tr>
<tr>
<td><strong>Waitemata</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly (NTD)/Encephalocele (NTD)</td>
<td>4</td>
<td>0.8</td>
<td>10.42</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>11</td>
<td>2.2</td>
<td>28.67</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>6</td>
<td>1.2</td>
<td>15.64</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>21</td>
<td>4.2</td>
<td>54.74</td>
</tr>
<tr>
<td>Spina Bifida (NTD)</td>
<td>7</td>
<td>1.4</td>
<td>18.25</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>3</td>
<td>0.6</td>
<td>7.82</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>52</td>
<td>10.4</td>
<td>135.54</td>
</tr>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly (NTD)/Encephalocele (NTD)</td>
<td>4</td>
<td>0.8</td>
<td>12.16</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>6</td>
<td>1.2</td>
<td>18.24</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>12</td>
<td>2.4</td>
<td>36.49</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>33</td>
<td>6.6</td>
<td>100.33</td>
</tr>
<tr>
<td>Spina Bifida (NTD)</td>
<td>3</td>
<td>0.6</td>
<td>9.12</td>
</tr>
<tr>
<td>Other Spinal Cord Malformations</td>
<td>3</td>
<td>0.6</td>
<td>9.12</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>63</td>
<td>12.6</td>
<td>191.54</td>
</tr>
<tr>
<td><strong>Counties Manukau</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly (NTD)/Encephalocele (NTD)</td>
<td>6</td>
<td>1.2</td>
<td>13.95</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>7</td>
<td>1.4</td>
<td>16.27</td>
</tr>
<tr>
<td>Congenital Hydrocephalus</td>
<td>11</td>
<td>2.2</td>
<td>25.57</td>
</tr>
<tr>
<td>Other Brain Malformations</td>
<td>23</td>
<td>4.6</td>
<td>53.46</td>
</tr>
<tr>
<td>Spina Bifida (NTD)</td>
<td>10</td>
<td>2.0</td>
<td>23.25</td>
</tr>
<tr>
<td>Other Spinal Cord Malformations</td>
<td>3</td>
<td>0.6</td>
<td>6.97</td>
</tr>
<tr>
<td>Other CNS Malformations</td>
<td>4</td>
<td>0.8</td>
<td>9.30</td>
</tr>
<tr>
<td>Total Malformations of the Nervous System</td>
<td>64</td>
<td>12.8</td>
<td>148.77</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset: Numerator: Hospitalisations with event type = birth and a nervous system anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have more than one anomaly; s: suppressed due to small numbers
Table 45. Babies with Neural Tube Defects Evident at Birth, Northern DHBs vs. New Zealand Hospital Births 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Rate per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Waitemata</td>
<td>11</td>
<td>2.2</td>
<td>28.67</td>
<td>1.11</td>
<td>0.59–2.09</td>
</tr>
<tr>
<td>Auckland</td>
<td>7</td>
<td>1.4</td>
<td>21.28</td>
<td>0.82</td>
<td>0.38–1.79</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>16</td>
<td>3.2</td>
<td>37.19</td>
<td>1.44</td>
<td>0.84–2.47</td>
</tr>
<tr>
<td>New Zealand</td>
<td>78</td>
<td>15.6</td>
<td>25.80</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset; Numerator: Hospitalisations with event type = birth and a neural tube defect listed in any of first 15 diagnoses; Denominator: All hospitalisations with event type = birth; Note: Rate per 100,000 refers to number of babies with one or more neural tube defects; Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics; s: suppressed due to small numbers.

Local Policy Documents and Evidence-Based Reviews Relevant to Neural Tube Defects

In New Zealand there are a number of policy documents relevant to the prevention or diagnosis of neural tube defects. These are summarized in Table 46, along with a range of reviews which consider these issues in the overseas context. In addition, Table 16 on Page 79 considers publications relevant to antenatal and newborn screening, while Table 29 on Page 102 considers congenital anomalies collectively.

Table 46. Local Policy Documents and Evidence-Based Reviews Relevant to Neural Tube Defects

<table>
<thead>
<tr>
<th>New Zealand Policy Documents, Publications and Websites</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ministry of Health, 2010. Folate/folic acid. <a href="http://www.health.govt.nz/our-work/preventative-health-wellness/nutrition/folate-folic-acid">http://www.health.govt.nz/our-work/preventative-health-wellness/nutrition/folate-folic-acid</a> accessed May 2013. This web page provides information on folate and New Zealand’s policy on folic acid supplementation for reducing neural tube defects. The Ministry of Health recommends that women planning to become pregnant take 800 µg of folic acid daily for at least four weeks before conception and for 12 weeks after. Women at high risk of having a baby with a NTD, including those with a previous affected pregnancy or a family history of NTD and those with some medical conditions including insulin-dependent diabetes, are recommended to take 5000 µg (5g) of folic acid for the same period. Subsidised 800 µg and 5mg folic acid tablets can be purchased over the counter from pharmacies.</td>
</tr>
<tr>
<td>Ministry of Health. 2010. Folic Acid and Spina Bifida/Iodine and Iodine Deficiency. Wellington: Ministry of Health. <a href="https://www.healthed.govt.nz/system/files/resource-files/HE4147_0.pdf">https://www.healthed.govt.nz/system/files/resource-files/HE4147_0.pdf</a> This is a brief pamphlet for pregnant women or women planning to become pregnant about the benefits of folic acid and iodine supplement tablets.</td>
</tr>
</tbody>
</table>
This publication provides background information on the benefits of folic acid and reviews the current policy situation (in 2003) regarding folic acid in New Zealand, Australia, Canada, the U.K. and the U.S. It considers four policy options for improving folate status in women of childbearing age:

- Increasing dietary folate intake
- Consumption of folic acid supplements (status quo)
- Voluntary fortification of staple food products with folic acid (status quo)
- Mandatory fortification of staple food products with folic acid.

The key recommendations were:

- An education campaign to make women aware of the benefits of increased folate consumption
- Considering mandatory fortification of either bread or flour
- Continuing to recommend folic acid supplements to women planning pregnancy whether fortification remains voluntary or becomes mandatory
- Making 400mg folic acid tablets available to women planning pregnancy as a registered medicine
- Continuing monitoring of neural tube defects, improved reporting of terminations of pregnancy to include the type of neural tube defect involved and monitoring of the folate status and folic acid intake of New Zealanders

International Guidelines and Systematic and Other Reviews


The benefits of folic acid supplementation for preventing neural tube defects (NTDs) are well established and the World Health Organization recommends that women take 400μg of folic acid from the time they start trying to conceive until 12 weeks of pregnancy. This review, which updates an earlier Cochrane review, assessed whether folic acid supplementation reduces NTDs and also other birth defects, including cleft palate, without adverse effects for mothers or babies. It included five RCTs, all published before 2001, (6105 women, of whom 1949 had a history of a pregnancy affected by a NTD and 4156 had not) in which women received either folic acid (alone or in combination with other vitamins and minerals) or placebo (or vitamin and mineral combinations without folic acid). Overall the results of these five trials indicated a protective effect for daily periconceptional folic acid supplementation, compared to no intervention, placebo or alternative vitamins/minerals, in reducing the risk of having a baby with an NTD (Risk ratio 0.28, 95% CI 0.15 to 0.52). In women with a previous NTD-affected pregnancy (four trials) folic acid had a significant protective effect for recurrence: (RR 0.32, 95% CI 0.17 to 0.60). In the one trial involving women with no previous history of NTDs (4156 women) there were no affected pregnancies in the supplementation group but the difference between the intervention and control groups was not a statistically significant (RR 0.08, 95% CI 0.00 to 1.33). Folic acid had no statistically significant effect on rates of cleft palate, cleft lip, congenital cardiovascular defects, miscarriages or any other birth defects. The authors concluded that folic acid does prevent NTDs but no other birth defects.


Section 9.1 of this publication covers screening for structural anomalies and reviews studies relating to the use of ultrasound and serum screening (alpha-fetoprotein, AFP) for this purpose. There were two studies identified that investigated maternal serum alpha-fetoprotein (AFP) as a screening test for neural tube defects. One found that maternal AFP had good diagnostic value in both predicting and ruling out anomalies while the other found it to have less diagnostic value than a routine ultrasound. There was no evidence on the value and effectiveness of a combination of routine ultrasound and maternal AFP screening.

A short version of this guideline can be found at: http://www.nice.org.uk/nicemedia/live/11947/40115/40115.pdf

Other Relevant Publications and Websites


This review searched for new evidence, published since 1996, on the benefits and harms of folic acid supplementation for women of childbearing age for the prevention of neural tube defects (NTDS), to inform the updated recommendations of the U.S. Preventive Services Taskforce. The authors identified four observational studies which reported a reduced risk of NTDs associated with supplements containing folic acid. The studies were too different in type and methodology to permit the calculation of a summary risk reduction. The authors stated that this new observational evidence supported previous evidence from RCTs and that the reported association of folic acid use with twin pregnancies may be confounded by an association with in vitro fertilisation.

This report is in three parts. The first part provides an overview of neural tube defects in seven chapters: Introduction, Background, The Public Health Response to Evidence Concerning the Protective Effect of Folic Acid, NTD Prevalence Rates in Europe 1980-2002, The Case for Fortification of Staple Foods in Europe, Conclusions, and References. Parts IIA and IIB have a chapter for each of the countries in EUROCAT giving information on local policy and statistics.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
OTHER DISABILITIES
Introduction

In New Zealand each year, it is estimated that 135–170 babies are born with mild to profound permanent congenital hearing loss, representing an incidence of 3 per 1,000 births [25]. The symptoms likely to be experienced by children with such hearing losses [18] are briefly outlined below.

<table>
<thead>
<tr>
<th>Type of Loss</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild Losses (26–40 dBHL)</td>
<td>This may result in some difficulties in hearing soft speech and conversations (people sound as if they are mumbling) but children can often manage in quiet situations with clear voices. Speech and language usually develop normally if the child is fitted with hearing aids early.</td>
</tr>
<tr>
<td>Moderate Losses (41–65 dBHL)</td>
<td>This may result in difficulty understanding conversational speech, particularly in the presence of background noise. The volume of the TV and radio will need to be turned up to be heard. Speech and language will generally be affected if a hearing aid is not provided early. A hearing aid will assist most hearing difficulties if speech discrimination is good and the listening environment is not too noisy.</td>
</tr>
<tr>
<td>Severe Losses (66–95 dBHL)</td>
<td>This will result in normal conversational speech being inaudible and only raised voices at close distance being understood. Speech and language will not develop spontaneously in children with severe hearing loss. Hearing aids will amplify many speech sounds and will greatly assist children in developing speech, although speech quality is likely to be affected. Some children may benefit from a cochlear implant.</td>
</tr>
<tr>
<td>Profound Losses (96+ dBHL)</td>
<td>Learning to speak without significant support is very difficult, although there is individual variation. There is greater inconsistency in benefit derived from hearing aids: some children can understand clear speech in quiet conditions when wearing a hearing aid, while others derive little benefit. Children with losses in this range should be considered for cochlear implants, as there is evidence that these are likely to be of benefit, especially if they are implanted at a young age.</td>
</tr>
</tbody>
</table>

The Identification of Hearing Loss in New Zealand

In response to concerns regarding the late age of diagnosis of congenital hearing losses in New Zealand, which occurred at an average age 35.1 months when screening was based on the presence of risk factors [53], in 2006 the Government announced a funding package to establish a Universal Newborn Hearing Screening and Early Intervention Programme (UNHSEIP). The UNHSEIP was rolled out during 2007–2010, and all DHBs now offer hearing screening to the families of newborn babies [26].

In tandem with this roll out, the Ministry of Health commissioned a series of UNHSEIP monitoring reports, which review the offer and uptake of newborn hearing screening at the DHB level, as well as the proportion of babies referred for audiology assessment, and the outcome of these investigations. Information on the number of babies diagnosed with permanent congenital hearing losses as a result of the UNHSEIP, as well as the average age at diagnosis, is also available from these reports [26].

Prior to the NZHSEIP, the only source of information on children and young people with permanent hearing loss in New Zealand was the Deafness Notification Database, which collected information on children with hearing losses which met specific criteria [54]. Information from this Database is available as far back as 1998, although there was a gap in coverage during 2006–2009.

The following section begins by presenting a range of historical and contemporary data from the Deafness Notification Database on permanent hearing loss in children and young people, before reviewing information from the most recent NZHSEIP monitoring reports on the number of babies diagnosed with permanent congenital hearing loss in recent years, and the ages at which these diagnoses occurred.
The Deafness Notification Database

Background
The aim of the Deafness Notification Database (DND) is to collect and report on new cases of permanent hearing loss diagnosed in New Zealand children and young people. The DND was funded by the Ministry of Health between 1982 and 2005, but was not operational during 2006–2009. In 2010 it was re-launched by the NZ Audiological Society, and Ministry of Health funding resumed from 2012. Although a number of changes have been made to the way in which the data are collected and reported (see notification criteria below), as much continuity in reporting has been maintained as possible between the two periods [54].

Data Sources and Methods
 Indicator
 Notifications to the New Zealand Deafness Notification Database

Data Source
NZ Deafness Notification Database
All of the data in this section were derived from the National Audiology Centre’s Annual Deafness Notification Database Reports 1998–2004 [53], or from the 2010–2012 Deafness Notification Reports produced by Digby et al. [54]. These reports are downloadable at http://www.audiology.org.nz/deafness-notification-database.aspx

Changes to the DND Notification Criteria
During 1982–2005, when the DND was managed by the National Audiology Centre, children needed to meet the following criteria [53]:

- Be less than 18 years of age, and have a congenital hearing loss or any hearing loss not remediable by medical or surgical means which required hearing aids and/or surgical intervention.
- Have an average bilateral hearing loss (over 4 audiometric frequencies 500–4000 Hz) of >26 dBHL in the better ear.
- Children were excluded if their hearing loss was <26 dBHL, unilateral, acquired, or they were born overseas.

In 2010 the DND was re-launched by the NZ Audiological Society, with audiologists being encouraged to notify newly diagnosed cases via a new online form. Following consultation, the database was extended to include:

- Children with an average hearing loss (over 4 audiometric frequencies 500–4000 Hz) of >26 dBHL in ONE ear (i.e. unilateral losses).
- Children who were born outside of New Zealand.

Additional audiologic guidance also suggested that while hearing losses arising from atresia, congenital ossicular fixation, meningitis and other acquired hearing losses should be included, hearing losses which could be fixed by the use of grommets (e.g. hearing losses associated with otitis media) should be excluded [54].

Additional Notes on Interpretation
DND data are reported by year of notification, rather than year of identification, with the degree of hearing loss assessed using the dBHL ranges outlined in the grey box above. As notification is not mandatory, these statistics may undercount the number of children with permanent hearing loss. In addition, the DND’s notification criteria changed during the reporting period (as outlined above) and this must be taken into account when interpreting the data in this section.

New Zealand Distribution by Severity of Loss
In New Zealand during 2012, only 3% of notifications to the DND were for children with profound hearing losses. A further 1% of notifications were for children with severe hearing losses, while 42% were for children with moderate losses and 54% were for children with mild losses (Table 47). Note that the data in this table differs from that reported previously, due to a change in the way the authors of the 2012 DND report assessed the severity of hearing loss (in order to more closely align it with the way severity was calculated in 2005). This resulted in a lower proportion of children being reported as having severe or profound losses in the 2012 report, than in the 2010 report [54].
Table 47. Notifications to the Deafness Notification Database by Degree of Hearing Loss, Using the Old Notification Criteria, New Zealand 2001–2004 and 2010–2012

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>47</td>
<td>47</td>
<td>56</td>
<td>43</td>
<td>59</td>
<td>60</td>
<td>54</td>
</tr>
<tr>
<td>Moderate</td>
<td>35</td>
<td>39</td>
<td>33</td>
<td>34</td>
<td>33</td>
<td>28</td>
<td>42</td>
</tr>
<tr>
<td>Severe</td>
<td>10</td>
<td>9</td>
<td>6</td>
<td>15</td>
<td>4</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Profound</td>
<td>8</td>
<td>5</td>
<td>5</td>
<td>7</td>
<td>5</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

Source: Deafness Notification Database via Digby et al. [54]; Note: Those with unilateral losses, who were born overseas, or who had acquired losses were removed in order to maintain consistency with earlier criteria

New Zealand Distribution by Ethnicity

In New Zealand during 2012, 103 children notified to the DND identified as European, 73 as Māori, 23 as Pacific, 12 as Asian/Indian and <3 as Middle Eastern/Latin American/African. As total response ethnicity was used, it was not possible to provide an overall breakdown of the proportions of children notified from each ethnic group.

Average Age at Suspicion and Confirmation of Hearing Loss

In New Zealand during 2012, when unilateral, acquired, mild, and overseas born cases were excluded (in order to ensure comparability with previous years) the average age at confirmation of a hearing loss was 50 months, although the average age of suspicion was much earlier (42 months) (Figure 27).

Figure 27. Average Age of Suspicion and Confirmation of Hearing Loss, New Zealand Deafness Notification Database 2001–2005 and 2010–2012

Source: Deafness Notification Database via Digby et al. [54]; Note: In order to ensure comparability with previous DND criteria, mild, acquired, unilateral, and overseas cases have been excluded
Number of Notifications by Age

During 2010–2012, the largest numbers of notifications to the DND were for babies under one year of age, largely as a result of newborn hearing screening. Numbers then dropped away during the preschool years. A second peak was evident at five years of age, most probably as a result of the B4 School Check, with numbers then falling away during mid-childhood. Note that this figure includes those with mild losses meeting DND criteria, those with acquired losses and those born overseas, all factors which may be associated with a later age of diagnosis of permanent hearing loss (Figure 28). Further, in the 2012 DND Report [54] the peak in notifications in babies under one year increased during this period (2010 n=23; 2011 n=34; 2012 n=38) probably as a result of the progressive roll out of newborn hearing screening [54].

Figure 28. Number of Notifications to the Deafness Notification Database by Age, New Zealand 2010–2012

Source: Deafness Notification Database via Digby et al. [54]
### Distribution by Region

Table 48 reviews the number of notifications received by the Deafness Notification Database by region using its old criteria during 1998–2004, while Table 49 reviews the number of notifications received by DHB using the new criteria during 2010–2012.

**Table 48. Number of Notifications Meeting the Old Criteria for Inclusion in the Deafness Notification Database by Region of Residence, New Zealand 1998–2004**

<table>
<thead>
<tr>
<th>Region of Residence</th>
<th>Notification Year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1998</td>
</tr>
<tr>
<td>Northland</td>
<td>10</td>
</tr>
<tr>
<td>Auckland Region</td>
<td>21</td>
</tr>
<tr>
<td>Waikato</td>
<td>7</td>
</tr>
<tr>
<td>Lakeland</td>
<td>3</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>10</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>3</td>
</tr>
<tr>
<td>Taranaki</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Hawke’s Bay</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Manawatu</td>
<td>3</td>
</tr>
<tr>
<td>Wellington</td>
<td>3</td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>&lt;3</td>
</tr>
<tr>
<td>West Coast</td>
<td>0</td>
</tr>
<tr>
<td>Canterbury</td>
<td>0</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>0</td>
</tr>
<tr>
<td>Otago</td>
<td>0</td>
</tr>
<tr>
<td>Southland</td>
<td>&lt;3</td>
</tr>
<tr>
<td>New Zealand Total</td>
<td>65</td>
</tr>
</tbody>
</table>

Source: National Audiology Centre [53]; Note: *2001 figures include 44 retrospective notifications; *During 2004 an additional 157 retrospective cases were added to the database, but are not included in this total; s: suppressed due to small numbers
Northern DHBs Distribution

In Northland during 2012, 15 children were notified to the Deafness Notification Database, while in Waitemata 6 children were notified, in Auckland DHB 7 children were notified and in Counties Manukau 26 children were notified (Table 49).

Table 49. Number of Notifications Meeting New Criteria for Deafness Notification Database by District Health Board, New Zealand 2010–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>12</td>
<td>5</td>
<td>15</td>
</tr>
<tr>
<td>Waitemata</td>
<td>4</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Auckland</td>
<td>10</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>25</td>
<td>20</td>
<td>26</td>
</tr>
<tr>
<td>Waikato</td>
<td>15</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>13</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Lakes</td>
<td>&lt;3</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>&lt;3</td>
<td>&lt;3</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Taranaki</td>
<td>6</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Hawke’s Bay</td>
<td>9</td>
<td>11</td>
<td>13</td>
</tr>
<tr>
<td>MidCentral</td>
<td>4</td>
<td>&lt;3</td>
<td>6</td>
</tr>
<tr>
<td>Whanganui</td>
<td>0</td>
<td>&lt;3</td>
<td>3</td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>5</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Capital &amp; Coast</td>
<td>24</td>
<td>24</td>
<td>8</td>
</tr>
<tr>
<td>Wairarapa</td>
<td>0</td>
<td>5</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>&lt;3</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>&lt;3</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Canterbury</td>
<td>44</td>
<td>27</td>
<td>23</td>
</tr>
<tr>
<td>West Coast</td>
<td>&lt;3</td>
<td>&lt;3</td>
<td>0</td>
</tr>
<tr>
<td>Southern</td>
<td>&lt;3</td>
<td>15</td>
<td>23</td>
</tr>
<tr>
<td>New Zealand</td>
<td>180</td>
<td>187</td>
<td>191</td>
</tr>
</tbody>
</table>

Source: Deafness Notification Database via Digby et al. [54]; Note: s: suppressed due to small numbers
Newborn Hearing Screening

Background
In response to concerns regarding the late age of diagnosis of congenital hearing losses, in 2006 the Government announced funding for the development of a Universal Newborn Hearing Screening and Early Intervention Programme (UNHSEIP). The UNHSEIP was implemented over the three year period 2007–2010, with a view to ensuring that [26]:

- Babies are screened for hearing loss by one month of age
- Audiology assessments are completed by three months of age
- Initiation of appropriate medical, audiological and early intervention education services occurs by six months of age.

The UNHSEIP is jointly overseen by the Ministry of Health, which is responsible for screening, audiological diagnosis and medical interventions; and the Ministry of Education, which is responsible for early intervention services. DHBs in turn, are the main providers of newborn hearing screening, follow-up audiology services and medical interventions. DHBs must offer newborn hearing screening to the family/whānau of all eligible babies in their region, whether they are born at hospital or home, using a framework of nationally consistent policies, standards and guidelines [26].

Screening is usually undertaken while the baby is asleep or quietly resting. Two types of screening are available:

- **Automated Otoacoustic Emissions (AOAE):** Sensory cells in the cochlea of the inner ear oscillate in response to an external sound. These oscillations generate an ‘echo’, which passes from the inner ear to the ear canal, and which can be detected as sound. These sounds, known as otoacoustic emissions (OAEs), are a sign that the ear is functioning normally and the measurement of OAEs can thus be used to test normal cochlear function in the newborn. Testing involves placing a small earphone and microphone in the ear, playing a sound and recording the response from the ear. If a baby has a normal functioning inner ear, an OAE is produced and this can be picked up by the microphone in the ear-canal [18].

- **Automated Auditory Brainstem Response (AABR):** The AABR is a series of electrical waves that can be recorded from electrodes on the scalp in response to brief sounds played into the ear. The presence of these waves with changing sound intensity is highly correlated with different hearing thresholds. The AABR can be used to assess the integrity of the ear and auditory nerve pathways to the brainstem in newborn babies [18].

The following section reviews data from the UNHSEIP’s monitoring reports [26]. The most recent report includes data for the 6-month period from 1st October 2011–31st March 2012.
Data Sources and Methods

Indicators

1. Proportion of eligible newborns whose parents/guardians consent to newborn hearing screening
   **Numerator:** Number of eligible newborns whose parents/guardians consent to newborn hearing screening
   **Denominator:** Number of eligible live births

2. The proportion of eligible newborns that complete the UNHS screening protocol by one month of age
   **Numerator:** Number of eligible newborns who complete newborn hearing screening by one month of age
   **Denominator:** Number of eligible newborns who complete newborn hearing screening

3. Proportion of newborns who do not pass hearing screening and are referred to audiology
   **Numerator:** Number of eligible newborns who complete screening with a referral for audiology assessment
   **Denominator:** Number of eligible newborns who complete screening

4. Proportion of newborns that pass screening but have risk factors for developing late onset or progressive hearing loss
   **Numerator:** Number of newborns that pass screening but have risk factors for developing late onset or progressive hearing loss (e.g. family history, craniofacial anomalies, jaundice, NICU >5 days, intrauterine infections, meningitis)
   **Denominator:** Number of eligible newborns who passed screening.

5. Proportion of newborn babies who completed audiology by 3 months of age
   **Numerator:** Number of newborns who completed audiology by 3 months of age
   **Denominator:** Number of newborns who completed audiology

6. Number of newborns with a permanent congenital hearing loss
   **Numerator:** Number of newborns who had a permanent hearing loss confirmed by audiology AND where the aetiology was an auditory neuropathy, mixed or sensorineural in at least one ear AND where the baby was referred through the UNHSEIP

7. Number of newborns with a conductive hearing loss
   **Numerator:** Number of newborns who had hearing loss confirmed by audiology, where the aetiology was NOT an auditory neuropathy, mixed or sensorineural AND where the baby was referred through the UNHSEIP. The majority of babies were identified as having a temporary conductive hearing loss.

**Data Source**


**Notes on Interpretation**

Note 1: The majority of data in this section were derived from the UNHSEIP’s monitoring report covering the six month period 1 October 2011 to 31st March 2012. However, trend data on the number of babies with permanent congenital hearing losses is drawn from UNHSEIP monitoring reports going as far back as 1st April 2010–30st Sept 2010. While all but one DHB (Southern) had implemented newborn hearing screening by the beginning of this earlier period, the outcome of audiology referrals may have been less complete for some DHBs due to the time taken for babies to progress through the referral pathways.

Note 2: The denominators for earlier UNHSEIP reports were derived from the Birth Registration Dataset and included live births for the relevant period. For the past 18 months however, birth data has been sourced from the National Maternity Database (which combines live birth registrations from Births Deaths and Marriages with hospital discharge data and Lead Maternity Carer claims). It is thought that this provides a much more complete data set due to the often long lag time for reporting birth registration data.

Note 3: In the newborn hearing screening table DHB refers to DHB of birth, whereas for the audiology tables, DHB refers to the DHB where the testing took place. Further audiology information is incomplete, as some DHBs did not submit information, or the information submitted was incomplete. In the 1st October 2011 to 31st March 2012 report, audiology information was available for only 254 of the 408 babies referred to audiology.
Newborn Hearing Screening

New Zealand Distribution
In New Zealand during 1st October 2011–31st March 2012, the caregivers of 88.6% of eligible babies consented to newborn hearing screening. Of those completing screening, 92.8% did so within one month, with 1.5% of those completing screening receiving an audiology referral. Of those babies who passed screening, a further 5.1% were deemed to have risk factors for delayed onset/progressive hearing loss (e.g. family history, craniofacial anomalies, and intrauterine infections) which warranted follow up over time (Table 50).

Northern DHBs Distribution
In the Northern region during 1st October 2011–31st March 2012, the proportion of caregivers of eligible babies who consented to newborn hearing screening ranged from 71.5% to 90.1%. Of those completing screening, the majority (range 67.8% to 94.8%) did so within one month. During this period 4.5% of Northland, 1.1% of Waitemata, 1.7% of Auckland and 1.8% of Counties Manukau babies who completed screening received an audiology referral. Of those who passed screening, a further 9.3% of Northland, 4.2% of Waitemata and Auckland and 6.1% of Counties Manukau babies were deemed to have risk factors for delayed onset/progressive hearing loss which warranted follow up (Table 50).

Table 50. Newborn Hearing Screening Indicators by District Health Board, New Zealand 1st October 2011–31st March 2012

<table>
<thead>
<tr>
<th>District Health Board</th>
<th>Number of Births in Period</th>
<th>Consenting to Screening (%)</th>
<th>Completed Screening ≤1 Month* (%)</th>
<th>Referrals to Audiology* (%)</th>
<th>Targeted for Follow Up* (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn Hearing Screening</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>1,172</td>
<td>77.1</td>
<td>67.8</td>
<td>4.5</td>
<td>9.3</td>
</tr>
<tr>
<td>Waitemata</td>
<td>3,987</td>
<td>84.5</td>
<td>90.0</td>
<td>1.1</td>
<td>4.2</td>
</tr>
<tr>
<td>Auckland</td>
<td>3,331</td>
<td>90.1</td>
<td>94.8</td>
<td>1.7</td>
<td>4.2</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>4,327</td>
<td>71.5</td>
<td>88.5</td>
<td>1.8</td>
<td>6.1</td>
</tr>
<tr>
<td>Waikato</td>
<td>2,708</td>
<td>94.0</td>
<td>94.8</td>
<td>1.3</td>
<td>5.7</td>
</tr>
<tr>
<td>Lakes</td>
<td>779</td>
<td>96.8</td>
<td>97.9</td>
<td>0.9</td>
<td>3.4</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>1,417</td>
<td>89.6</td>
<td>92.6</td>
<td>1.7</td>
<td>2.1</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>368</td>
<td>88.6</td>
<td>97.8</td>
<td>1.2</td>
<td>8.2</td>
</tr>
<tr>
<td>Taranaki</td>
<td>792</td>
<td>92.8</td>
<td>99.3</td>
<td>2.4</td>
<td>10.3</td>
</tr>
<tr>
<td>Hawke's Bay</td>
<td>1,110</td>
<td>95.2</td>
<td>98.8</td>
<td>0.9</td>
<td>5.7</td>
</tr>
<tr>
<td>Whanganui</td>
<td>429</td>
<td>87.4</td>
<td>97.8</td>
<td>0.3</td>
<td>7.3</td>
</tr>
<tr>
<td>MidCentral</td>
<td>1,112</td>
<td>73.8</td>
<td>70.9</td>
<td>1.1</td>
<td>7.6</td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>1,054</td>
<td>99.5</td>
<td>99.5</td>
<td>0.6</td>
<td>3.4</td>
</tr>
<tr>
<td>Capital &amp; Coast</td>
<td>1,943</td>
<td>99.3</td>
<td>97.9</td>
<td>1.6</td>
<td>4.6</td>
</tr>
<tr>
<td>Wairarapa</td>
<td>280</td>
<td>96.1</td>
<td>90.3</td>
<td>1.5</td>
<td>7.5</td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>759</td>
<td>99.6</td>
<td>95.1</td>
<td>1.6</td>
<td>5.4</td>
</tr>
<tr>
<td>West Coast</td>
<td>211</td>
<td>71.6</td>
<td>96.6</td>
<td>0.7</td>
<td>3.4</td>
</tr>
<tr>
<td>Canterbury</td>
<td>2,980</td>
<td>97.6</td>
<td>95.8</td>
<td>1.4</td>
<td>3.8</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>289</td>
<td>95.8</td>
<td>99.3</td>
<td>0.7</td>
<td>2.6</td>
</tr>
<tr>
<td>Southern</td>
<td>1,781</td>
<td>97.1</td>
<td>95.0</td>
<td>1.2</td>
<td>5.2</td>
</tr>
<tr>
<td>New Zealand</td>
<td>30,829</td>
<td>88.6</td>
<td>92.8</td>
<td>1.5</td>
<td>5.1</td>
</tr>
</tbody>
</table>

Source: National Screening Unit 2012 [26]; Note: *See Methods for Indicator Definitions
Audiology Referrals and Outcomes

New Zealand Distribution

In New Zealand during 1\textsuperscript{st} October 2011–31\textsuperscript{st} March 2012, 254 babies commenced an audiology assessment, with 85.9\% of those who completed their audiology assessment doing so by 3 months of age. During this period, 30 babies were identified as having a permanent congenital hearing loss, while 73 were identified as having a conductive hearing loss (Table 51).

Northern DHBs Distribution

In the Northern region during 1\textsuperscript{st} October 2011–31\textsuperscript{st} March 2012, 38 Northland, 40 Auckland and 17 Counties Manukau babies commenced an audiology assessment, with the majority (range 71.1\% to 100.0\%) of those who completed their audiology assessment doing so by 3 months of age. During this period, one Auckland DHB baby was identified as having a permanent congenital hearing loss, while 7 Northland and 12 Auckland DHB babies were identified as having a conductive hearing loss (Table 51). No information was available however, for the Waitemata DHB.

Table 51. Newborn Audiology Indicators by District Health Board, New Zealand 1\textsuperscript{st} October 2011–31\textsuperscript{st} March 2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Commenced Audiology (Number)</th>
<th>Completed Audiology ≤3 months (%)</th>
<th>Permanent Congenital Hearing Loss (Number)</th>
<th>Conductive Hearing Loss (Number)</th>
<th>Permanent Congenital Hearing Loss (% of Completed)</th>
<th>Conductive Hearing Loss (% of Completed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>38</td>
<td>71.1</td>
<td>0</td>
<td>7</td>
<td>0</td>
<td>18.4</td>
</tr>
<tr>
<td>Waitemata</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland</td>
<td>40</td>
<td>100.0</td>
<td>1</td>
<td>12</td>
<td>2.6</td>
<td>30.8</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>17</td>
<td>83.3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Waikato</td>
<td>30</td>
<td>76.7</td>
<td>9</td>
<td>13</td>
<td>30.0</td>
<td>43.3</td>
</tr>
<tr>
<td>Lakes</td>
<td>5</td>
<td>100.0</td>
<td>2</td>
<td>2</td>
<td>40.0</td>
<td>40.0</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>13</td>
<td>92.3</td>
<td>3</td>
<td>0</td>
<td>23.1</td>
<td>0</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taranaki</td>
<td>17</td>
<td>88.2</td>
<td>2</td>
<td>8</td>
<td>11.8</td>
<td>47.1</td>
</tr>
<tr>
<td>Hawke's Bay</td>
<td>8</td>
<td>87.5</td>
<td>2</td>
<td>1</td>
<td>25.0</td>
<td>12.5</td>
</tr>
<tr>
<td>Whanganui</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>MidCentral</td>
<td>10</td>
<td>100.0</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>30.0</td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>12</td>
<td>100.0</td>
<td>2</td>
<td>8</td>
<td>16.7</td>
<td>66.7</td>
</tr>
<tr>
<td>Capital &amp; Coast</td>
<td>14</td>
<td>92.9</td>
<td>3</td>
<td>3</td>
<td>21.4</td>
<td>21.4</td>
</tr>
<tr>
<td>Wairarapa</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>11</td>
<td>90.9</td>
<td>2</td>
<td>3</td>
<td>18.2</td>
<td>27.3</td>
</tr>
<tr>
<td>West Coast</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canterbury</td>
<td>21</td>
<td>76.2</td>
<td>2</td>
<td>7</td>
<td>9.5</td>
<td>33.3</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>2</td>
<td>100.0</td>
<td>1</td>
<td>0</td>
<td>50.0</td>
<td>0</td>
</tr>
<tr>
<td>Southern</td>
<td>15</td>
<td>80.0</td>
<td>1</td>
<td>6</td>
<td>6.7</td>
<td>40.0</td>
</tr>
<tr>
<td>New Zealand</td>
<td>254</td>
<td>85.9</td>
<td>30</td>
<td>73</td>
<td>12.1</td>
<td>29.4</td>
</tr>
</tbody>
</table>

Source: National Screening Unit 2012 [26]; Note: See Methods for Indicator Definitions
Trends in the Identification of Permanent Congenital Hearing Losses

New Zealand Distribution
In New Zealand, a total of 81 babies were identified as having permanent congenital hearing losses in the UNHSEIP’s six-monthly monitoring reports spanning the period 1st April 2010–31st March 2012 (Table 52).

Northern DHBs Distribution
In the Northern DHBs, two Northland, seven Auckland DHB and one Counties Manukau babies were identified as having permanent congenital hearing losses in the UNHSEIP’s six-monthly monitoring reports spanning the period 1st April 2010–31st March 2012 (Table 52). No information was available however, for the Waitemata DHB.

Table 52. Number of Babies Identified by Newborn Hearing Screening as Having Permanent Congenital Hearing Losses by District Health Board and Monitoring Period, New Zealand 1st April 2010–31st March 2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>6 Month Monitoring Period</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Permanent Congenital Hearing Losses Identified by Newborn Screening</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Waitemata</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Waikato</td>
<td>2</td>
<td>4</td>
<td>4</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Lakes</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
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<tr>
<td>Bay of Plenty</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taranaki</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Hawke's Bay</td>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Whanganui</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>MidCentral</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Capital &amp; Coast</td>
<td>1</td>
<td>0</td>
<td>7</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Wairarapa</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>West Coast</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canterbury</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>South Canterbury</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Southern*</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td>11</td>
<td>16</td>
<td>24</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>

Source: National Screening Unit [26]; Note: *Data for Southern DHB is only from August 2010 onwards
Local Policy Documents and Evidence-Based Reviews Relevant to the Identification and Management of Congenital Hearing Loss

In New Zealand there are a range of policy documents which consider newborn hearing screening. These are briefly summarised in **Table 53**, along with a number of other publications which consider congenital hearing losses in the overseas context.

**Table 53. Local Policy Documents and Evidence-Based Reviews Relevant to the Early Detection and Management of Permanent Hearing Loss in Children**

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>These National Policy and Quality Standards form part of the contract between the Ministry of Health and District Health Boards (DHBs) for the provision of services for the Universal Newborn Hearing Screening and Early Intervention Programme (UNHSEIP). The standards are intended to increase knowledge about the programme, outline requirements of services, and assist DHBs to achieve high standards of practice.</td>
</tr>
<tr>
<td>This Technical Brief produced by New Zealand Health Technology Assessment was commissioned by the New Zealand Ministry of Health. It compared the effectiveness of cochlear implantation at earlier and later ages. No eligible systematic reviews were found so 15 studies that were cross-sectional, case control or cohort studies were appraised. Implantation at less than 24 months of age was found to be more effective in terms of audiological performance, communication outcomes, educational achievement and quality of life than implantation at more than 24 months but it was unclear whether implantation at less than 12 months was more effective than implantation at more than 12 months.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>International Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muse C, et al. 2013. <strong>Supplement to the JCIH 2007 position statement: principles and guidelines for early intervention after confirmation that a child is deaf or hard of hearing.</strong> Pediatrics, 131(4), e1324-49.  <a href="http://pediatrics.aappublications.org/content/131/4/e1324.full.pdf+html">http://pediatrics.aappublications.org/content/131/4/e1324.full.pdf+html</a></td>
</tr>
<tr>
<td>This best practice statement from the US, advocates for the implementation of coordinated state wide systems with the expertise to provide individualized, high-fidelity early intervention programmes for children who are deaf or hard of hearing and their families. It outlines comprehensive guidelines for early hearing detection and intervention programmes and establishing strong early intervention systems with appropriate expertise to meet the needs of children who are deaf or hard of hearing. Consistent monitoring of child and family outcomes is an essential step toward ensuring optimal outcomes. The establishment of practice standards, implementation of developmentally appropriate protocols for monitoring of outcomes, and commitment to research collaborations are critical steps toward this goal.</td>
</tr>
<tr>
<td>This is a consensus statement on paediatric cochlear implantation by the European Bilateral Pediatric Cochlear Implant Forum, as determined by a review of the current scientific literature. &quot;Currently we feel that the infant or child with unambiguous cochlear implant candidacy should receive bilateral cochlear implants simultaneously as soon as possible after definitive diagnosis of deafness to permit optimal auditory development; an atraumatic surgical technique designed to preserve cochlear function, minimize cochlear damage, and allow easy, possibly repeated re-implantation is recommended.&quot;</td>
</tr>
</tbody>
</table>

These NICE guidelines evaluate the clinical effectiveness and cost-effectiveness of cochlear implants in England and Wales for children (12 months to 18 years) with severe to profound sensorineural hearing loss and for adults with severe to profound deafness. Thirty-three papers were included to evaluate clinical effectiveness, and 17 published studies were evaluated to assess cost-effectiveness. The major recommendations were:

- Unilateral cochlear implantation is recommended as an option for people with severe to profound deafness who do not receive adequate benefit from acoustic hearing aids.
- If different cochlear implant systems are considered to be equally appropriate, the least costly should be used.
- Assessment of cost should take into account acquisition costs, long-term reliability and the support package offered.
- Simultaneous bilateral cochlear implantation is recommended as an option for the following groups of people with severe to profound deafness who do not receive adequate benefit from acoustic hearing aids:
  - Children
  - Adults who are blind or who have other disabilities that increase their reliance on auditory stimuli as a primary sensory mechanism for spatial awareness
- Acquisition of cochlear implant systems for bilateral implantation should be at the lowest cost and include currently available discounts on list prices equivalent to 40% or more for the second implant.

Sequential bilateral cochlear implantation is not recommended as an option for people with severe to profound deafness.


This is the Australian national protocol for amplification for hearing impaired children. It gives guidelines for selecting candidates for hearing aid fitting or referral to cochlear implant programmes and also covers management of children who have auditory neuropathy spectrum disorder and children who have mild and unilateral hearing loss. It describes the protocol for selection of hearing aids, hearing aid fitting and verification procedure and hearing-aid evaluation and also the criteria for supplying personal frequency modulated (FM) systems.


The position statement of the Joint Committee on Infant Hearing endorses screening of all newborns in order to ensure that infants with hearing loss can receive the earliest possible intervention with a view to maximising their opportunities to develop linguistic, literary, cognitive and social-emotional competence, and so that their educational and vocational attainment in adulthood can be as good as that of their hearing peers. It provides guidelines on screening protocols, evaluation of hearing impaired children detected by screening programmes, early intervention programmes, continued surveillance of infants and toddlers, protection of infant and family rights, information infrastructure, benchmarks and quality indicators. It also reports on current challenges, opportunities, and future directions in the field.

Systematic and Other Reviews From the International Literature


This systematic review considered the cost-effectiveness of universal newborn screening for bilateral permanent congenital hearing impairment. Twenty-two observational or modelled evaluations were identified of which only 2 clearly compared universal newborn hearing screening to risk factor screening for bilateral permanent congenital hearing impairment. Of these, the single evaluation that examined long-term costs and outcomes found that universal newborn hearing screening could be cost-saving if early intervention led to a substantial reduction in future treatment costs and productivity losses. There is a clear need for further research on long-term costs and outcomes to establish the cost-effectiveness of universal newborn hearing screening in relation to other approaches to screening, and to establish whether it is a good long term investment.


This systematic review evaluated the outcome of bilateral versus unilateral bone-anchored hearing aids (BAHA) for individuals with bilateral permanent conductive hearing loss in children and adults. Eleven observational studies were included. Bilateral BAHA provided audiologic benefit and patient’s subjective benefit compared to unilateral BAHA. The studies had small sample sizes and were limited in number.
The causes of Permanent Childhood Hearing Impairment (PCHI) are often quoted as being hereditary in 50%, acquired in 25%, and unknown in 25% of cases. This population based study and systematic review investigated the evidence for the reported distribution of causes. In the study-population (n = 185) a hereditary cause was found in 38.9%, acquired cause in 29.7%, miscellaneous cause in 7.1%, and the cause was unknown in 24.3%. The systematic review (n = 9 articles) resulted in a weighted mean of 30.4% hereditary, 19.2% acquired, and 48.3% unknown causes of PCHI.


The objective of this evidence-based statement was to evaluate, among school age children with single sided deafness, if amplification bone conduction hearing aids versus no amplification improve quality of life. The authors concluded that amplification should be offered, noting that selected educational and family outcomes are important to monitor when amplification is used or if a decision is made not to provide amplification and that adequate information to families and the child are necessary for informed decision making regarding interventions.


The objective of this statement was to evaluate whether amplification (i.e. digital hearing aid (HA), frequency modulation (FM) system, contra lateral routing of signal CROS) link aid etc.) compared to no amplification improves educational or functional performance in school-age children with severe to profound unilateral sensorineural hearing loss (USNHL) or mild to moderately severe USNHL.

In children with severe to profound USNHL it is recommended that school-aged children be fitted with an FM system as the first line of amplification technology, selecting an FM system with the most open fit to decrease occlusion in the good ear. It is recommended that provision of a HA in children with severe-profound UHL be on a case-by-case basis.

For children with mild to moderate sensorineural UHL it is recommended that children be fitted with a hearing aid (FM ready) as the first line intervention, and that provision of an FM system with or without a HA be discussed with the family. Note that the quality of evidence of UHL is moderate due to small numbers of studies and small sample sizes.


This review was an update for the U.S. Preventive Services Task Force on universal newborn hearing screening (UNHS) to detect moderate to severe permanent, bilateral congenital hearing loss. Children with hearing loss who had UNHS had better language outcomes at school age than those not screened. Infants identified with hearing loss through universal screening had significantly earlier referral, diagnosis and treatment than those identified other ways.

**Systematic and Other Reviews on Cochlear Implants**


This systematic review compared the outcome of cochlear implantation in children with developmental disability with children without developmental disability. Thirteen studies were included where expressive and/or receptive language outcomes were compared with children with cochlear implants and normal development. Seven studies demonstrated a worse outcome for children with developmental disability. Six articles showed no difference in the outcome between the 2 groups. Children with developmental disability may not benefit from cochlear implantation based on traditional assessment tools but appear to improve their environmental awareness and quality of life. More work is needed to define the term benefit when used in this context. Autistic children consistently had a negative outcome.


This systematic review evaluated the range of acoustic hearing in the implanted ear that can be effectively preserved for an electric-acoustic fitting; what benefits are provided by combining acoustic stimulation with electric stimulation; and what clinical fitting practices have been developed for devices that combine electric and acoustic stimulation. Twenty-seven articles were identified. The effectiveness of combined electric and acoustic stimulation as compared with electric stimulation alone was consistently demonstrated, highlighting the potential value of preservation and utilization of low frequency hearing in the implanted ear. However, clinical procedures for best fitting of electric-acoustic devices were varied. There is a need for further investigation of fitting procedures aimed at maximizing outcomes for recipients of electric-acoustic devices.

In this economic evaluation of cochlear implant (CI) in children, nine studies were included. Cost analysis, cost-effectiveness analysis and an analysis of educational costs associated with CI were performed. The direct cost ranged between € 39,507 and € 68,235 (2011 values). The studies related to cost-effectiveness analysis were not easily comparable: one study reported a cost per QALY ranging between $ 5197 and $ 9209; another referred a cost of $2154 for QALY if benefits were not discounted, and $16,546 if discounted. Educational costs are significant, and increase with the level of hearing loss and type of school attended. This review shows that the healthcare costs are high, but savings in terms of indirect and quality of life costs are also significant. Cochlear implantation in children is cost-effective.


This systematic review summarized the results of scientific publications on the clinical effectiveness of the cochlear implant (CI) in children. Studies suggest that children implanted within the first year of life present hearing and communicative outcomes that are better than those of children implanted after 12 months of age. For children implanted after the first year of life, all studies confirm an advantage and many document an advantage in children who received cochlear implants under 18 months of age compared to those implanted at a later stage. Studies demonstrate that bilateral CI offers advantages in terms of hearing in noise, sound localization and during hearing in a silent environment compared to unilateral CI. There is, however, a wide range of variability. The studies also document the advantages after sequential bilateral CI. The studies also indicate that CI is also suitable for children with disabilities associated with deafness.


This review considers the current knowledge on cochlear implantation in children aged less than 12 months, regarding diagnostic, surgical and anaesthetic challenges. The studies reviewed included 3 meta-analyses, 4 prospective controlled studies, 25 prospective studies, 21 retrospective studies, 1 guideline, 8 review articles and 4 books. Based on a meta analysis of 125 infants the authors conclude that there is not an increased anaesthetic or surgical risk associated with infancy. Detection of other developmental issues which may affect the likelihood of developing normal speech and language is challenging but there are appropriate evaluation techniques for reliable assessment of the prelexical domains of infant development.


This review evaluated the published research on bilateral cochlear implants. Because of the relative newness of this topic the authors searched for Randomized controlled trials (RCTs), prospective cohort studies, retrospective cohort studies, case studies and series, reviews, and qualitative studies. Of the 29 studies that met the inclusion criteria there were no RCTs, 4 reviews, 1 national survey of Cochlear Implant centres in the US, 15 cohort studies and 2 case control studies and 7 case series or case studies. All of the studies had relatively small numbers of participants (less than 50). It found that sound localisation and speech recognition in noise seem to be improved with bilateral cochlear implants compared to a unilateral implant and that the greatest benefits occur when the second implant is done early. It recommended further research into cost-effectiveness, quality of life, speech, language and psycho-educational measures.


This review investigated whether it is clinically effective and cost-effective to provide a unilateral cochlear implant for severely to profoundly deaf people (who do or do not use hearing aids), and whether it is clinically effective and cost-effective to provide bilateral cochlear implants for severely to profoundly deaf people who have a single cochlear implant (who do or do not use a hearing aid as well). This study reported on a systematic review of the literature which found 33 suitable papers of which only 2 were randomised controlled trials. All of the studies reviewed found that for children there were gains on all outcome measures when comparing one cochlear implant with non-technological support or an acoustic hearing aid. Earlier implantation in children produced the greatest benefits. From the Markov model base-case analysis the authors estimated that, for prelingually profoundly deaf children, the incremental cost-effectiveness ratio (ICER) for unilateral implantation compared with no implantation was £13,413 per quality-adjusted life-year (QALY). The best evidence for the benefits of bilateral cochlear implants was in understanding speech in noisy conditions. The authors conclude that unilateral cochlear implantation for children and adults is cost effective but state “decisions on the cost-effectiveness of bilateral cochlear implants should take into account the high degree of uncertainty within the model regarding the probable utility gain.”


This systematic review evaluated the strength of evidence when comparing the effectiveness of unilateral cochlear implants with non-technological support or acoustic hearing aids in children with permanent bilateral hearing loss (PBHL) in the UK. Fifteen studies were identified. They were of moderate to poor quality and heterogeneity in design and outcomes precluded meta-analysis. However, all studies reported that unilateral cochlear implants improved scores on all outcome measures. Additionally five economic evaluations found unilateral cochlear implants to be cost-effective for profoundly deaf children at UK implant centres.
This review looks at the literature on bilateral cochlear implantation in children and recommends simultaneous bilateral implantation when possible and if not then the shortest possible interval between implantation of the first and second ears. It recommends further research to determine the interval after which bilateral cochlear implantation provides so little benefit that it is not cost-effective.

### Other Relevant Publications on Cochlear Implants

This paper reports on a review of 75 paediatric referrals the Southern Cochlear Implant Programme from March 2003-March 2008 (before the introduction of the newborn hearing screening programme). The mean age at referral was 17 months with a range of 1 to 203 months. The authors state that the age of referral has been unacceptably high and that children with known risk factors for significant sensorineural hearing loss have not been receiving early diagnosis.

This article provides a local perspective on the issue and points out that there is an increasing body of evidence on the benefits of bilateral implants in children. (Currently only one implant per child is normally publicly funded.)

This study reports on cochlear implant failure in 27 European centres and notes that while overall cochlear implant systems are satisfactory there is considerable variation in the reliability of different systems. A common industry independent failure database using uniform reporting protocols would be beneficial to users and clinicians.

### Documents Relating to the Universal Newborn Hearing Screening Programme in New Zealand

**Ministry of Health. 2012.** *Quality improvement review of a screening event in the Universal Newborn Hearing Screening and Early Intervention Programme.* Wellington: Ministry of Health.  
This review was undertaken by the National Screening Unit (NSU) with the input of an Incident Review Group of the Universal Newborn Hearing Screening and Early Intervention Programme following issues identified between July and November 2012 where babies were not screened correctly for permanent congenital hearing loss. The review outlines the findings and recommendations to improve DHB service provision and strengthen the leadership and surveillance of the programme by the NSU.

**Universal Newborn Hearing Screening and Early Intervention Programme**  
Newborn Hearing Screening and Audiology Workforce Strategy and Action Plan June 2008  
This workforce development strategy and action plan was to address the development of the newborn hearing screening and audiology workforce required for implementing the UNHSEIP. The strategy outlines initiatives that would need to be implemented by the National Screening Unit, DHBs, professional bodies and educational institutions in a collaborative manner.

This report contains the findings and recommendations of the Universal Newborn Hearing Screening Advisory Group to the National Screening Unit regarding high-level policy and implementation issues for a (then) future universal newborn hearing screening programme for New Zealand. It contains background information on congenital hearing loss, New Zealand statistics and summarises the benefits of lowering the average age of detection of hearing loss. It also addresses issues relevant to intervention services and the design and operation of screening services.

This very comprehensive report (with 435 references) includes information on hearing loss in general, the effects of permanent congenital hearing loss, New Zealand data, and issues relating to universal newborn hearing screening and early intervention programmes and international experience with them. The authors state “This proposal is well supported within the sector, with both professional and consumer groups unified around its value, across health and education, deaf and hearing-impaired, Māori and non- Māori.”
### Other Relevant Websites

<table>
<thead>
<tr>
<th>Website</th>
<th>URL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ministry of Health webpage related to hearing loss with information on symptoms, hearing checks for children and hearing services available in New Zealand. The webpage includes a section on cochlear implants</td>
<td><a href="https://www.health.govt.nz/your-health/conditions-and-treatments/disabilities/hearing-loss">https://www.health.govt.nz/your-health/conditions-and-treatments/disabilities/hearing-loss</a></td>
</tr>
<tr>
<td>The National Foundation for the Deaf Incorporated is a foundation with nine member organisations set up to promote the interests, advancement, independence and wellbeing of deaf and hearing impaired New Zealanders. The foundation also raises awareness of the health, social, educational, economic, environmental and cultural barriers encountered by deaf and hearing impaired people and their families. They facilitate communication and coordination among professional and community organisations working with deaf and hearing impaired people.</td>
<td></td>
</tr>
<tr>
<td>The Deafness Notification Database (DND) was in operation from 1982-2005 to collect and report on the number and nature of new cases of hearing loss diagnosed among children and young people born in New Zealand. It was funded by the Ministry of Health. From 2006-2009 the database was not in operation but it was re-launched in 2010 funded by the New Zealand Audiological Society. Eligibility now includes those children born overseas and those with unilateral hearing losses. Reports are now available for 2010, 2011 and</td>
<td></td>
</tr>
<tr>
<td>The New Zealand Audiological Society</td>
<td><a href="http://www.audiology.org.nz">http://www.audiology.org.nz</a></td>
</tr>
<tr>
<td>The New Zealand Audiological Society is a self-governing body representing more than 300 audiologists that was incorporated in 1976. Their aim is to assist the hearing impaired community of all ages to participate as fully as possible in all aspects of life. The website has information for the public on hearing loss, screening, and hearing aids. There is also information for audiologists including standards of practice, events and other links.</td>
<td></td>
</tr>
<tr>
<td>Hearing Association New Zealand Te Kāhui Rongo o Aotearoa</td>
<td><a href="http://www.hearing.org.nz">http://www.hearing.org.nz</a></td>
</tr>
<tr>
<td>The Hearing Association New Zealand sets out to advance the interests and general welfare of all persons with a hearing loss, their family, whānau and all who support them, to improve their quality of life. This national body supports 32 separate associations which are dedicated to raising the profile of hearing issues, services and information. There are branches throughout New Zealand. Information on hearing screening, hearing aids and other products, various classes and other information sheets are available on the website.</td>
<td></td>
</tr>
</tbody>
</table>

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Cerebral Palsy

Introduction

The term cerebral palsy refers to a group of disorders of movement or posture that arise from a non-progressive insult to the central nervous system during early development. The insult may occur prior to, during, or shortly after birth and, while the insult itself non-progressive, its physical consequences can evolve over time [55].

Cerebral palsy is the commonest cause of physical disability in early childhood. It occurs in 2–3 per 1,000 live births, but rates may be as high as 40–100 per 1,000 in very low birth weight or preterm babies [56]. Despite this, around 55% of children with cerebral palsy are born at term, while 20% are born at 32–36 weeks and 25% at <32 weeks [56].

As the typical neurological signs of cerebral palsy take time to develop, it is generally accepted that a child should be four years of age before a diagnosis is established, although earlier diagnoses are not precluded in individual cases. Around 80% of children with cerebral palsy have spastic cerebral palsy (characterised by weakness, increased muscle tone, overactive reflexes and a tendency to contractures), while 7% have dyskinetic cerebral palsy (characterised by involuntary movements that disappear during sleep) and 4% have ataxic cerebral palsy (characterised by problems with coordination, gait and rapid movements of the distal extremities) [56,57]. While the term “cerebral palsy” refers to the motor impairment, additional features such as seizures, intellectual impairment and learning disabilities are common [57].

Children and young people with cerebral palsy require a variety of personal health care and disability support services to achieve their highest possible functioning within their family and community environments. Physical and occupational therapy are beneficial for the management of motor impairments, as proper positioning and handling is necessary to minimise difficulties with posture, trunk control, and feeding. Passive and active exercises to stretch tight tendons may be used to maintain normal alignment of bone, joint and soft tissue and to prevent contractures. Medical and surgical procedures may be necessary to correct contractures that do not respond to physiotherapy, and to re-establish motor balance between opposing muscle groups. In addition, a variety of equipment, such as walkers and standing frames, motorised wheel chairs, feeding tubes, and computers to augment communication, and other supports, for example speech therapy, medications, ophthalmology, tailored educational programmes, respite care, may be required [58].

While plans for a New Zealand Cerebral Palsy Register are under way, at present there is no reliable information on the prevalence of cerebral palsy in New Zealand children and young people. In the absence of such information, the following section reviews hospital admissions for children and young people with any mention of cerebral palsy in any of their first 15 diagnoses, as well as mortality for children and young people with cerebral palsy listed as the main underlying cause of death, or as a contributory cause.
**Data Source and Methods**

**Definition**
1. Hospital admissions for children and young people aged 0–24 years with cerebral palsy listed in any of the first 15 diagnoses
2. Mortality in children and young people aged 0–24 years with cerebral palsy listed as the main underlying cause of death or as a contributory cause

**Data Source**
1. National Minimum Dataset
   - **Numerator**: Hospital admissions for children and young people aged 0–24 years with cerebral palsy (ICD-10-AM G80) listed in any of the first 15 diagnoses.
2. National Mortality Collection
   - **Numerator**: Mortality in children and young people aged 0–24 years with cerebral palsy (ICD-10-AM G80) listed as the main underlying cause of death, or as a contributory cause.
   - **Denominator**: Statistics New Zealand Estimated Resident Population (projected from 2007).

**Notes on Interpretation**
Note 1: This analysis focuses on hospital admissions and mortality for children and young people with cerebral palsy listed in any of the first 15 diagnoses, or as the main underlying or a contributory cause of death. The rationale for looking beyond the primary diagnosis was the need to highlight the full spectrum of health issues experienced by those with cerebral palsy, and their consequent requirement for health services. For example, during 2008–2012, focusing on the primary diagnosis would have identified only 10% of acute and arranged hospitalisations in those with cerebral palsy, with the majority being admitted for other reasons (e.g. epilepsy/convulsions, pneumonitis). Similarly 51% of admissions were from the waiting list, with a large proportion being for injections into ligaments, tendons or soft tissue, or for other orthopaedic procedures. The presence of a small number of admissions which were unrelated to cerebral palsy (e.g. acute upper respiratory infections) however, may slightly overestimate the impact cerebral palsy has on acute service demand.

Note 2: As the majority of those with cerebral palsy are managed in the outpatient or primary care setting, it is likely that this analysis significantly underestimates the number of children and young people with cerebral palsy, particularly those at the milder end of the spectrum. The rationale for the methodology used however, was the absence of other more reliable sources of information on the number of children and young people with cerebral palsy in the community.

Note 3: If no mention of cerebral palsy was made in any of the first 15 diagnoses, these cases were not included, even if the patient had been assigned a cerebral palsy-related code on a previous admission.

---

**New Zealand Distribution and Trends**

**Distribution by Primary Diagnosis and Procedure**

**Acute and Arranged Admissions by Primary Diagnosis**: In New Zealand during 2008–2012, only 10.4% of acute and arranged hospitalisations in children and young people with cerebral palsy listed in their first 15 diagnoses, had cerebral palsy listed as their primary reason for admission. Instead 17.5% of acute and arranged admissions were for epilepsy or convulsions and 22.3% for respiratory infections and diseases. Acute and arranged admissions collectively made up 48.6% of all admissions for children and young people with cerebral palsy during this period (Table 54).

**Waiting List Admissions by Procedure**: During the same period, 51.4% of admissions in children and young people with cerebral palsy were from the waiting list, with injections into ligaments, tendons, or soft tissue accounting for 42.2% of all waiting list admissions. Orthopaedic procedures collectively were the leading reasons for waiting list admissions in children and young people with cerebral palsy, followed by dental procedures (Table 55).
Table 54. Acute and Arranged Hospital Admissions for Children and Young People Aged 0–24 Years with Cerebral Palsy by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Acute and Arranged Admissions</th>
<th>% of All Admissions in those with Cerebral Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy, Status Epilepticus, Convulsions</td>
<td>434</td>
<td>86.8</td>
<td>5.68</td>
<td>17.5</td>
<td>8.5</td>
</tr>
<tr>
<td>Cerebral Palsy</td>
<td>257</td>
<td>51.4</td>
<td>3.37</td>
<td>10.4</td>
<td>5.0</td>
</tr>
<tr>
<td>Influenza and Pneumonia</td>
<td>178</td>
<td>35.6</td>
<td>2.33</td>
<td>7.2</td>
<td>3.5</td>
</tr>
<tr>
<td>Unspecified Acute Lower Respiratory Infection</td>
<td>159</td>
<td>31.8</td>
<td>2.08</td>
<td>6.4</td>
<td>3.1</td>
</tr>
<tr>
<td>Pneumonitis due to Food and Vomit</td>
<td>128</td>
<td>25.6</td>
<td>1.68</td>
<td>5.2</td>
<td>2.5</td>
</tr>
<tr>
<td>Acute Upper Respiratory Infections</td>
<td>89</td>
<td>17.8</td>
<td>1.17</td>
<td>3.6</td>
<td>1.7</td>
</tr>
<tr>
<td>Other Respiratory Infections and Diseases</td>
<td>76</td>
<td>15.2</td>
<td>1.00</td>
<td>3.1</td>
<td>1.5</td>
</tr>
<tr>
<td>Constipation</td>
<td>56</td>
<td>11.2</td>
<td>0.73</td>
<td>2.3</td>
<td>1.1</td>
</tr>
<tr>
<td>Other Diseases Digestive System</td>
<td>152</td>
<td>30.4</td>
<td>1.99</td>
<td>6.1</td>
<td>3.0</td>
</tr>
<tr>
<td>Complications of Surgical and Medical Care</td>
<td>107</td>
<td>21.4</td>
<td>1.40</td>
<td>4.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Infectious and Parasitic Diseases</td>
<td>103</td>
<td>20.6</td>
<td>1.35</td>
<td>4.1</td>
<td>2.0</td>
</tr>
<tr>
<td>Respite Care</td>
<td>32</td>
<td>6.4</td>
<td>0.42</td>
<td>1.3</td>
<td>0.6</td>
</tr>
<tr>
<td>Other Factors Influencing Health Service Contact</td>
<td>67</td>
<td>13.4</td>
<td>0.88</td>
<td>2.7</td>
<td>1.3</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>645</td>
<td>129.0</td>
<td>8.45</td>
<td>26.0</td>
<td>12.6</td>
</tr>
<tr>
<td>Total Acute and Arranged Admissions</td>
<td>2,483</td>
<td>496.6</td>
<td>32.52</td>
<td>100.0</td>
<td>48.6</td>
</tr>
<tr>
<td>Total Waiting List Admissions</td>
<td>2,625</td>
<td>525.0</td>
<td>34.38</td>
<td>51.4</td>
<td></td>
</tr>
<tr>
<td>Total Admissions in those with Cerebral Palsy</td>
<td>5,108</td>
<td>1,021.6</td>
<td>66.89</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, Acute and arranged admissions by primary diagnosis for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
### Table 55. Waiting List Hospital Admissions in Children and Young People Aged 0–24 Years with Cerebral Palsy by Primary Procedure, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Procedure</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Waiting List Admissions</th>
<th>% of All Admissions in those with Cerebral Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injection into Ligament, Tendon or Soft Tissue</td>
<td>1,107</td>
<td>221.4</td>
<td>14.50</td>
<td>42.2</td>
<td>21.7</td>
</tr>
<tr>
<td>Dental Procedures</td>
<td>233</td>
<td>46.6</td>
<td>3.05</td>
<td>8.9</td>
<td>4.6</td>
</tr>
<tr>
<td>Osteotomy of Proximal Femur</td>
<td>107</td>
<td>21.4</td>
<td>1.40</td>
<td>4.1</td>
<td>2.1</td>
</tr>
<tr>
<td>Release of Hip Contracture</td>
<td>85</td>
<td>17.0</td>
<td>1.11</td>
<td>3.2</td>
<td>1.7</td>
</tr>
<tr>
<td>Forage of Neck and/or Head of Femur</td>
<td>65</td>
<td>13.0</td>
<td>0.85</td>
<td>2.5</td>
<td>1.3</td>
</tr>
<tr>
<td>Lengthening/Repair of Achilles Tendon</td>
<td>60</td>
<td>12.0</td>
<td>0.79</td>
<td>2.3</td>
<td>1.2</td>
</tr>
<tr>
<td>Other Orthopaedic Procedures: Lower Limbs</td>
<td>147</td>
<td>29.4</td>
<td>1.93</td>
<td>5.6</td>
<td>2.9</td>
</tr>
<tr>
<td>Lengthening of Tendon, Unspecified</td>
<td>90</td>
<td>18.0</td>
<td>1.18</td>
<td>3.4</td>
<td>1.8</td>
</tr>
<tr>
<td>Spinal Fusion</td>
<td>48</td>
<td>9.6</td>
<td>0.63</td>
<td>1.8</td>
<td>0.9</td>
</tr>
<tr>
<td>Orthopaedic Procedures: Upper Limbs</td>
<td>31</td>
<td>6.2</td>
<td>0.41</td>
<td>1.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Other Orthopaedic Procedures</td>
<td>160</td>
<td>32.0</td>
<td>2.10</td>
<td>6.1</td>
<td>3.1</td>
</tr>
<tr>
<td>Injection/Infusion of Other Therapeutic/Prophylactic Substance</td>
<td>57</td>
<td>11.4</td>
<td>0.75</td>
<td>2.2</td>
<td>1.1</td>
</tr>
<tr>
<td>Insertion of Percutaneous Endoscopic Gastrostomy (PEG) Tube</td>
<td>34</td>
<td>6.8</td>
<td>0.45</td>
<td>1.3</td>
<td>0.7</td>
</tr>
<tr>
<td>Insertion of Percutaneous Non-endoscopic Gastrostomy Button</td>
<td>19</td>
<td>3.8</td>
<td>0.25</td>
<td>0.7</td>
<td>0.4</td>
</tr>
<tr>
<td>Fundoplasty</td>
<td>34</td>
<td>6.8</td>
<td>0.45</td>
<td>1.3</td>
<td>0.7</td>
</tr>
<tr>
<td>Insertion/Replacement of Intrauterine Contraceptive Device</td>
<td>27</td>
<td>5.4</td>
<td>0.35</td>
<td>1.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Tonsillectomy and/or Adenoidectomy</td>
<td>24</td>
<td>4.8</td>
<td>0.31</td>
<td>0.9</td>
<td>0.5</td>
</tr>
<tr>
<td>MRI of Brain</td>
<td>20</td>
<td>4.0</td>
<td>0.26</td>
<td>0.8</td>
<td>0.4</td>
</tr>
<tr>
<td>Other Procedures</td>
<td>208</td>
<td>41.6</td>
<td>2.72</td>
<td>7.9</td>
<td>4.1</td>
</tr>
<tr>
<td>No Procedure Listed</td>
<td>69</td>
<td>13.8</td>
<td>0.90</td>
<td>2.6</td>
<td>1.4</td>
</tr>
<tr>
<td>Total Waiting List Admissions</td>
<td>2,625</td>
<td>525.0</td>
<td>34.38</td>
<td>100.0</td>
<td>51.4</td>
</tr>
<tr>
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<td>48.6</td>
<td>48.6</td>
</tr>
<tr>
<td>Total Admissions in those with Cerebral Palsy</td>
<td>5,108</td>
<td>1,021.6</td>
<td>66.89</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, Waiting list admissions by primary procedure for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Distribution by Age

In New Zealand during 2008–2012, hospital admissions for children and young people with cerebral palsy increased during infancy, reached a peak at three years of age, and then gradually declined. In contrast, mortality was more evenly distributed across the age range. During 2006–2010, 78 children and young people 0–24 years had cerebral palsy listed as their main underlying cause of death, or as a contributory cause (Figure 29).

Figure 29. Hospital Admissions (2008–2012) and Mortality (2006–2010) for New Zealand Children and Young People with Cerebral Palsy by Age

![Graph showing hospital admissions and mortality by age for children and young people with cerebral palsy in New Zealand.](image)

Source: Numerator Admissions: National Minimum Dataset, hospital admissions for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Numerator Mortality: National Mortality Collection, Deaths with cerebral palsy listed as the main underlying or a contributory cause of death; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Distribution by Ethnicity and Gender

In New Zealand during 2008–2012, hospital admissions for children and young people with cerebral palsy were significantly higher for males and for Pacific > European/Other > Māori > Asian/Indian children and young people (Table 56).

Table 56. Hospital Admissions for Children and Young People Aged 0–24 Years with Cerebral Palsy by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral Palsy Prioritised Ethnicity</td>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>36.86</td>
<td>0.53</td>
<td>0.47–0.59</td>
<td>Female</td>
<td>57.56</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>70.01</td>
<td>1.00</td>
<td>Male</td>
<td>75.76</td>
<td>1.32</td>
<td>1.25–1.39</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>63.10</td>
<td>0.90</td>
<td>0.84–0.97</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>92.23</td>
<td>1.32</td>
<td>1.21–1.43</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population
**Trends by Ethnicity**

While admission rates for Pacific, Māori and European/Other children and young people with cerebral palsy were similar during the early-mid 2000s, diverging trends saw rates for Pacific children and young people become higher than for European/Other and Māori children and young people from 2008–09 onwards. Admission rates for Asian/Indian young people however, were lower than for Pacific, Māori and European/Other children and young people throughout 2000–2012 (Figure 30).

Figure 30. Hospital Admissions for Children and Young People Aged 0–24 Years with Cerebral Palsy by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population; Note: Ethnicity is Level 1 Prioritised

**Northern DHBs Distribution and Trends**

**Northern DHBs Distribution**

In the Northern region during 2008–2012, a total of 57 Northland, 231 Waitemata, 156 Auckland and 257 Counties Manukau children and young people were hospitalised with a diagnosis of cerebral palsy. Admission rates per 100,000 population in the Waitemata and Counties Manukau DHBs were significantly higher than the New Zealand rate, while in Northland rates were significantly lower. In Auckland DHB, rates were similar to the New Zealand rate (Table 57).
Table 57. Hospital Admissions for Children and Young People Aged 0–24 Years with Cerebral Palsy, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A*</td>
<td>B*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>52</td>
<td>57</td>
<td>144</td>
<td>0.51</td>
<td>53.29</td>
<td>0.80</td>
</tr>
<tr>
<td>Waitemata</td>
<td>218</td>
<td>231</td>
<td>728</td>
<td>0.63</td>
<td>77.12</td>
<td>1.15</td>
</tr>
<tr>
<td>Auckland</td>
<td>144</td>
<td>156</td>
<td>455</td>
<td>0.58</td>
<td>60.75</td>
<td>0.91</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>250</td>
<td>257</td>
<td>750</td>
<td>0.58</td>
<td>75.91</td>
<td>1.13</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1,657</td>
<td>5,108</td>
<td>62</td>
<td>0.62</td>
<td>66.89</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (i.e. DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (i.e. the sum of DHB totals exceeds New Zealand total); Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics.

**Northern DHBs Trends**

In Counties Manukau during 2000–2012, hospital admissions for children and young people with cerebral palsy were consistently higher than the New Zealand rate. In Northland, admissions exhibited a downward trend, with rates during the mid-late 2000s being generally lower than the New Zealand rate. Large year to year variations in the Waitemata and Auckland DHBs made trends for these DHBs difficult to interpret (Figure 31).
Figure 31. Hospital Admissions for Children and Young People Aged 0–24 Years with Cerebral Palsy, Northern DHBs vs. New Zealand 2000–2012

Admissions per 100,000 (0–24 Years)

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Policy Documents and Evidence-Based Reviews Relevant to the Diagnosis or Management of Cerebral Palsy

In New Zealand there is a paucity of policy documents relevant to children and young people with cerebral palsy. Table 58 however summarises a range of overseas publications which may be relevant in this context.

Table 58. Policy Documents and Evidence-Based Reviews Relevant to Cerebral Palsy

<table>
<thead>
<tr>
<th>International Guidelines and Useful Websites</th>
</tr>
</thead>
</table>

Children with cerebral palsy (CP) often have feeding and swallowing problems which may lead to growth failure and repeated aspiration of food resulting in chronic pulmonary disease. If a child cannot take sufficient food orally to maintain growth, there is frequent aspiration, or the level of work required by the child or their caregiver to maintain an adequate oral intake is excessive then feeding via a surgically implanted gastrostomy or jejunostomy tube may be recommended. This review assessed the effects of interventions for feeding and nutrition problems in individuals with CP using evidence from research studies. The authors explored the evidence relating to specified “key questions”. They identified one systematic review on behavioural interventions and 12 unique primary studies, most of which were surgical case series. They stated that, overall, there was little data to guide care, that the study populations were almost entirely children with severe CP, and that most studies were short term and did not consistently assess harms of interventions. They concluded that the evidence for behavioural interventions ranged from insufficient to moderate and noted that some studies suggested that sensorimotor interventions such as oral appliances (moderate strength of evidence) and positioning (low strength of evidence) may be beneficial. They also concluded that the evidence for surgical interventions ranged from insufficient to low but that all studies to date had demonstrated significant weight gain with gastrostomy. They stated that there is considerable uncertainty over the harms of feeding interventions both in the short and the long term. They stated that harms associated with gastrostomy can be common and include overfeeding, site infection, stomach ulcer, and reflux.

**Childhood Integrated Neuroscience Discovery Network (CP-NET) [http://cpnet.canchild.ca/en/](http://cpnet.canchild.ca/en/)**

This site is part of the website of the CanChild Centre for Childhood Disability Research located at McMaster University in Hamilton, Ontario, Canada. A collection of research articles on CP can be found here: [http://cpnet.canchild.ca/en/Research_Articles_on_CP.asp](http://cpnet.canchild.ca/en/Research_Articles_on_CP.asp)


This clinical guideline covers the management of spasticity and co-existing motor disorders and their early musculoskeletal complications in children and young people aged 0–18 years with non-progressive brain disorders, the most common of which is cerebral palsy. It does not cover the management of other aspects of cerebral palsy. This guideline is an abbreviated version of the full guideline:


These comprehensive evidence-based guidelines are intended for healthcare professionals, service commissioners and planners, social service and education professionals, and families, carers and children and young people. They cover physical therapy (physiotherapy and/or occupational therapy), orthoses, oral drugs, botulinum toxin, intrathecal baclofen, orthopaedic surgery, and selective dorsal rhizotomy. There is also a chapter dealing with the health economics of these therapies. For each review question addressed in the guidelines there is an “evidence profile”, a table listing the relevant studies, and, for each study, numbers of participants, size of effect (with 95% confidence interval) and study quality.
This brief document provides guidance on selective dorsal rhizotomy for spasticity in cerebral palsy. The procedure involves surgery to cut some of the sensory nerve rootlets leaving the lumbar region of the spinal cord with the aim of reducing sensory input to the sensory–motor reflex arcs responsible for increased muscle tone. Three non-randomised comparative studies (142, 108 and 142 patients) have reported positive results from the procedure. This publication states that there is a risk of serious but well-recognised complications and that the evidence for efficacy is adequate. More details of the studies on which the guidance document is based are contained in the following publication:


**Guidelines from the James M. Anderson Center for Health Systems Excellence at Cincinnati Children's Hospital** [http://www.cincinnatichildrens.org/service/j/anderson-center/default/](http://www.cincinnatichildrens.org/service/j/anderson-center/default/)

The Anderson Center at Cincinnati Children’s Hospital has published a number of “Best Evidence Statements” related to care issues for children with cerebral palsy. They contain details of the studies relevant to the clinical questions addressed and care recommendations. Guidance on the following CP-related topics (with year of publication) can be found at these links:

- Lower Extremity Orthoses for Children with Hemiplegic Cerebral Palsy (2010)
- Aquatic Therapy for Children with Hemiplegic Cerebral Palsy (2010)
- Strengthening (progressive resistive exercise) for individuals with Cerebral Palsy age 4-20 years who demonstrate muscle weakness (2010)
- Biofeedback Intervention for Children with Hemiplegic Cerebral Palsy (2010)
- Pediatric Constraint Induced Movement Therapy (CIMT) (2009)

**American Academy for Cerebral Palsy and Developmental Medicine** [http://www.aacpdm.org](http://www.aacpdm.org)

This organisation describes itself as “A global leader in the multidisciplinary scientific education of health professionals and researchers dedicated to the well-being of people with childhood-onset disabilities.” It provides scientific information for health professionals and promotes excellence in research and services. It publishes the journal *Developmental Medicine & Child Neurology*. The following evidence reports can be found on this website: [http://www.aacpdm.org/publications/outcome](http://www.aacpdm.org/publications/outcome). Titles are followed by publication date. Where there is no publication date on a report, the date given is the latest year of the literature search on which each report is based.

- Effects of Conductive Education for Cerebral Palsy (2003)
- Effects of Intrathecal Baclofen for Spastic and Dystonic Cerebral Palsy (2000)
- Effects of Neurodevelopmental Treatment (NDT) for Cerebral Palsy (2001)
- Effects of Gastrostomy Feeding in Children with Cerebral Palsy (2002)

**Systematic and Other Reviews from the International Literature**


This thorough and concise review, describing itself as a systematic review of systematic reviews, aimed to describe the best available evidence on interventions for children with cerebral palsy (CP) using the GRADE system and to assist clinicians with translating evidence to practice and deciding what to do. This evidence is complemented with the Evidence Alert Traffic Light System. The authors found 166 articles meeting the review’s inclusion criteria, 74 of which were systematic reviews. These articles addressed 64 discrete interventions and reported on 131 outcomes. The ‘green’ interventions (those with the highest quality favourable evidence and therefore strong recommendations) included anticonvulsants, bimanual training, botulinum toxin, bisphosphonates, casting, constraint-induced movement therapy, context-focused therapy, diazepam, fitness training, goal-directed training, hip surveillance, home programmes, occupational therapy after botulinum toxin, pressure care, and selective dorsal rhizotomy. Most interventions (76%) had lower level evidence and the authors noted that many of these interventions are in common use in standard care which is a problem for people with CP, healthcare providers, funders and purchasers.
The development or worsening of gastro-oesophageal reflux (GOR) is a widely reported complication of gastrostomy tube placement. To reduce the possibility of this occurring surgical anti-reflux treatment in the form of fundoplication or another anti-reflux procedure is frequently performed at the same time as gastrostomy surgery. Fundoplication involves wrapping the fundus of the stomach around the oesophagus at the gastro-oesophageal junction to strengthen the barrier to acid reflux. The authors did not identify any RCTs involving children aged up to 18 years with neurological impairments and GOR who were undergoing gastrostomy tube insertion and therefore they concluded that there is considerable uncertainty about the optimal treatment when faced with the choice of fundoplication vs. anti-reflux medications for children with GOR and neurological impairment who are undergoing gastrostomy insertion.


The authors of this review aimed to evaluate the effects of nutritional supplementation given via gastrostomy or jejunostomy to children with feeding difficulties due to CP. They did not identify any RCTs relating to this issue and so they concluded that there remains considerable uncertainty about the effects of gastrostomy for children with CP.


Magnesium sulphate is widely used in obstetrics for the prevention and treatment of eclampsia and it has been shown to be effective for neuroprotection of the fetus when given to women at risk of very preterm birth. Since more than half of all cases of cerebral palsy occur in children born at term it is important to determine whether antenatal administration of magnesium sulphate to women at term would also protect the fetus from brain injury and associated disabilities, including CP. This was the aim of this review. The authors identified one RCT involving 135 women with mild pre-eclampsia at term. This trial did not report on any of the review’s pre-specified primary outcomes and so the authors concluded that there is currently insufficient evidence to assess the efficacy and safety of magnesium sulphate when given to women at term for neuroprotection of the fetus.


Severe brain damage as the result of hypoxic ischaemia may follow a cardiopulmonary arrest and resuscitation and lead to the development of cerebral palsy, blindness, seizures and hypothalamic and pituitary insufficiency. Very severe brain damage can be fatal or lead to a persistent vegetative state. Therapeutic hypothermia (lowering the core body temperature to between 32°C and 34°C) may reduce brain injury in the period after circulation has been restored. This therapy has been effective for neonates with hypoxic ischaemic encephalopathy and adults after witnessed cardiopulmonary arrest with ventricular fibrillation. This review considered the clinical effectiveness of therapeutic hypothermia after paediatric cardiopulmonary arrest. The authors were unable to find any RCTs or quasi-RCTs meeting their criteria, but they found four on-going RCTs which may provide data for analysis in the future. They stated that, based on their review findings, they were unable to make any recommendations for clinical practice.


Pilot studies in humans, as well as animal studies, suggest that inducing mild hypothermia (cooling) after peripartum hypoxia-ischaemia in newborn infants may reduce neurological sequelae without adverse effects. This review considered the effect of therapeutic hypothermia in encephalopathic asphyxiated newborn infants on mortality and long-term neurodevelopmental disability and assessed whether there are clinically important side effects. The review included 11 RCTs involving 1505 term and late preterm infants with moderate/severe encephalopathy and evidence of intrapartum asphyxia. The results of these trials indicated that therapeutic hypothermia led to a statistically significant and clinically important reduction in the combined outcome of death or major developmental disability at age 18 months (typical relative risk 0.75 (95% CI 0.68 to 0.83); typical risk difference −0.15, 95% CI −0.20 to −0.10); number needed to treat for an additional beneficial outcome (NNTB) 7 (95% CI 5 to 10) (8 studies, 1344 infants). Cooling also produced statistically significant reductions in mortality (typical RR 0.75 (95% CI 0.64 to 0.88), typical RD −0.09 (95% CI −0.13 to −0.04); NNTB 11 (95% CI 8 to 25) (11 studies, 1468 infants) and in neurodevelopmental disability in survivors (typical RR 0.77 (95% CI 0.63 to 0.94), typical RD −0.13 (95% CI −0.19 to −0.07); NNTB 8 (95% CI 5 to 14) (8 studies, 917 infants). Adverse effects of cooling included an increase sinus bradycardia and a significant increase in thrombocytopenia. The authors concluded that therapeutic hypothermia is beneficial for term and late preterm infants with hypoxic ischaemic encephalopathy. It reduces mortality without increasing long term disability and the benefits outweigh the short term adverse effects. They stated that hypothermia should be instituted in term and late preterm infants with moderate-to-severe hypoxic ischaemic encephalopathy if this is identified before six hours of age.
Drooling is a common problem for children with CP and their parents and caregivers. It may lead to social rejection, damp and smelly clothing, chapped skin, mouth infections, dehydration, speech difficulties and damage to learning materials like books and computers. This review had three aims: firstly to evaluate the effectiveness and safety of interventions aimed at reducing or eliminating drooling in children with CP, secondly to provide the best available evidence to inform clinical practice and, thirdly, to assist with future research planning. The review included studies only if they were RCTs or controlled clinical trials (CCTs). The authors found six relevant studies, five RCTs and one CCT, with approximately 198 participants altogether. Four were of botulinum toxin A (BoNT-A) and there was one study on each of the pharmacological treatments benztrpine and glycopyrrolate. There was considerable heterogeneity between the BoNT-A studies and so a meta-analysis was not possible. All six studies showed some statistically significant effects of the intervention for up to one month following the intervention but the authors considered that all studies had methodological flaws. They concluded that it was not possible to reach a conclusion on the effectiveness and safety of either BoNT-A or the pharmaceutical interventions, benztrpine and glycopyrrolate, and that there is insufficient evidence to inform clinical practice on interventions for drooling in children with CP.


Oropharyngeal dysphagia (OD) encompasses problems with chewing and preparing the food for swallowing, moving the food or fluid posteriorly through the oral cavity with the tongue into the back of the throat, and swallowing the food or fluid and moving it through the pharynx to the oesophagus. It is commonly experienced by children with neurological impairment, including those with CP. The authors of this review considered the effectiveness of interventions for OD in children with neurological impairment. They found three small studies meeting their inclusion criteria, two of which they considered to have high risk of bias. Two studies compared oral sensorimotor interventions to standard care in children with CP and the other compared lip strengthening exercises to no treatment in children with myotonic dystrophy type 1. The two trials of oral sensorimotor interventions were too different for meta-analysis to be possible. The authors concluded that there is currently insufficient high-quality evidence from RCTs or quasi-RCTs to provide conclusive results about the effectiveness of any particular type of oral-motor therapy for children with neurological impairment and that more research with larger-scale RCTs is urgently needed.


Compared to infants born at term, infants born pre-term are at higher risk of developing cognitive and motor impairments. This review considered the effectiveness of early developmental intervention post-discharge from hospital for improving motor or cognitive development in preterm (< 37 weeks) infants with no major congenital abnormalities. Twenty-one studies met the review’s inclusion criteria (3133 randomised patients) but only ten were RCTs with appropriate allocation concealment. Meta-analysis indicated that intervention improved cognitive outcomes at infant age (developmental quotient (DQ): standardised mean difference (SMD) 0.31 standard deviations (SD); 95% confidence interval (CI) 0.13 to 0.50; P < 0.001; 13 studies; 2147 patients), and pre-school age (intelligence quotient (IQ): SMD 0.45 SD; 95% CI 0.34 to 0.57; P < 0.001; six studies; 1276 patients). However, this effect was not sustained at school age (IQ: SMD 0.25 SD; 95% CI -0.10 to 0.61; P = 0.16; five studies; 1242 patients). There was significant heterogeneity between studies for cognitive outcomes at infant and school ages. In respect of motor outcomes, meta-analysis of 10 studies showed a significant effect in favour of early developmental interventions but the effect was small (motor scale developmental quotient (DQ): SMD 0.10 SD; 95% CI 0.00 to 0.19; P = 0.04; 10 studies; 1745 patients). There was no effect on the rate of cerebral palsy in survivors: risk ratio (RR) 0.89; 95% CI 0.55 to 1.44; (4 studies; 737 patients). There was little evidence for a positive effect on motor outcomes in the long term, and only five studies reported outcomes at pre-school or school age. The authors concluded that early intervention programmes for preterm infants have a positive influence on cognitive and motor outcomes during infancy and the cognitive benefits persist into pre-school age. Substantial differences between studies make comparing intervention programmes hard.


A number of childhood disorders, including Down syndrome, cerebral palsy (CP), spina bifida and a broad range of other neuromuscular disorders are associated with delays in motor development in infancy. Treadmill training, in which the child is supported by a harness, can give children the opportunity to walk with support for long enough periods of time to acquire the motor skills necessary for independent walking. This review assessed the effectiveness of treadmill interventions on locomotor motor development in pre-ambulatory infants and children under six years of age who are at risk for neuromotor delay. The review included five studies (RCTs or controlled clinical trials) reporting on treadmill interventions. The studies had methodological limitations as outcome assessors were not blinded. In total 139 children were involved, 73 in the intervention groups and the rest in the control groups. Three studies involved children with Down syndrome (90 children in total), one involved children with CP (8 in total) and one involved children at risk of neuromotor delay. The studies were quite diverse with regard to the types of comparisons made, the time of evaluation and the parameters assessed, so meta-analysis was only possible for the results of three studies. The authors stated that, given the limited number of studies, and their heterogeneity, they could not provide any firm evidence for the clinical application of treadmill training but that, for children with Down syndrome, treadmill training may facilitate earlier walking and children with Down syndrome who received more intensive treadmill intervention may be more accomplished in their gait parameters that the children who received less intensive treadmill intervention.
Children with CP often have difficulties with speech, language and gesture due to motor, sensory and intellectual impairment. Speech and language therapy (SLT) is used to help children maximise their communication skills. It may involve augmentative and alternative communication systems, such as symbol charts or communication aids with synthetic speech, as well as enhancing children's natural forms of communication. This review considered the effectiveness of SLT focusing on the child or their familiar communication partners (as measured by changes in patterns of interaction) and whether some forms of SLT are better than others. The review criteria included any experimental study with an element of control and sixteen such studies were identified by the authors. Nine studies evaluated treatment given to the children and seven training for communication partners. The authors concluded that their review did not indicate firm evidence for the positive effect of SLT for children with CP but there were some positive trends in communication. They recommended further more methodologically rigorous research.


Children with CP often have spasticity (stiffness) of the legs due to involuntary muscle over-activity caused by brain or spinal cord damage. Spasticity is a major component of the disability and deformities associated with CP and it causes poor coordination, spasms, abnormal posture and pain. Botulinum toxin blocks the release of acetylcholine from the neuromuscular junction and weakens the muscle. This review considered whether botulinum toxin A (BoNT-A) is safe and effective treatment for lower limb spasticity in children with CP. The authors identified three small, short term, randomised studies with follow-up periods of between four and 26 weeks. They concluded that there was no strong evidence to either support or refute the use of BoNT-A for the treatment of leg spasticity in CP.


This review assessed the effectiveness of injections of Botulinum toxin A (BoNT-A), alone or in combination with occupational therapy, for the treatment of the upper limb in spasticity and hypertonia children with CP. The authors identified ten RCTs meeting their inclusion criteria, nine of which were considered to be of high quality. They concluded that there was high level evidence to support the use of BoNT-A as an adjunct to occupational therapy in managing the upper limbs in children with spastic CP and moderated evidence that BoTN-A alone is not effective. They recommended that practitioners follow the Fehlings guidelines:


**Katalinic OM, Harvey LA, Herbert RD, et al. 2010. Stretch for the treatment and prevention of contractures.**

Stretch is widely used for the prevention of contractures and the preservation of joint mobility in a range of neurological and musculoskeletal conditions, including CP. This review assessed the effectiveness of this practice. The authors identified 35 RCTs or controlled clinical trials meeting their criteria with 1391 participants in total. No study lasted longer than seven months. The authors found that in people with neurological conditions, there was moderate to high quality evidence to indicate that stretch does not have clinically important effects on joint mobility either immediately (mean difference 3°; 95% CI 0 to 7), in the short-term (mean difference 1°; 95% CI 0 to 3) or the long-term (mean difference 0°; 95% CI –2 to 2). The results were similar for people with non-neurological conditions. For all conditions, they found little or no effect of stretch on pain, spasticity, activity limitation, participation restriction or quality of life. They concluded that stretch does not have clinically important effects on joint mobility in people with, or at risk of, contractures if performed for less than seven months and they stated that the effects of stretch performed for periods longer than seven months have not been investigated.

**Smuelders JCM, Coester A, Kreulen M. 2009. Surgical treatment for the thumb-in-palm deformity in patients with cerebral palsy.**

Thumb-in-palm deformity impairs the ability of a person with CP to use their thumb and severely limits hand function. A variety of surgical procedures to correct this problem have been described. This review considered: the efficacy of surgical interventions for the thumb-in-palm deformity in patients with spastic CP; the selection criteria for surgical treatment of thumb-in-palm deformity in these patients; and the outcome assessment used in studies of these treatments. The authors did not find any relevant RCTs or controlled clinical trials. They did find 14 prospective studies and included the best nine of these, which compared preoperative and postoperative assessment, in their review. They concluded that because of the poor methodological quality of the studies it was not possible to provide a reliable judgement of the role of surgery for thumb-in-palm deformity in patients with CP but surgery appeared to improve hand function, to facilitate hygiene, and to improve appearance and quality of life.

Children with hemiplegic CP often neglect or fail to use their affected limb. Constraint-induced movement therapy (CIMT) is an emerging treatment for hemiplegic CP. It aims to increase spontaneous use of the affected upper limb and thereby limit the effects of developmental disregard. CIMT is based on two fundamental principles: constraint of the non-affected limb and massed practice of therapeutic tasks with the affected limb. This review examined the effectiveness of CIMT for children with upper limb hemiplegic CP. The authors identified three studies (RCTs or controlled clinical trials) meeting their inclusion criteria with 18, 41 and 31 children. The studies used a variety of constraint methods and outcome measures. One trial found a significant treatment effect using modified CIMT and another found a positive trend favouring modified CIMT and Forced Use. The authors concluded that, given the limited evidence, the use of CIMT, modified CIMT and Forced Use should be considered experimental.


This review evaluated the efficacy and safety of pharmacologic treatments for childhood spasticity due to cerebral palsy. The authors did not find any peer-reviewed studies on phenol, alcohol, or botulinum toxin type B that involved more than nine patients with CP aged 19 years or younger and had English language abstracts. They stated that there was insufficient evidence to either support or refute the use of BoNT-B, phenol, and alcohol injections as a treatment for spasticity in children with spastic CP.

Botulinum type A: They found 148 studies of botulinum toxin A (BoNT-A) fulfilling the same criteria. Fifteen of these were Class I and five were Class II according to the American Academy of Neurology criteria. One Class I trial was a randomized, controlled clinical trial with masked or objective outcome assessment in a representative population and a Class II trial was either a RCT that lacks one of the criteria for a class I trial or a high-quality prospective matched cohort study. A total of 573 children received BoNT-A in the Class I and II studies. All but three of these studies reported spasticity reduction but there was conflicting evidence regarding functional improvement. Seventeen studies reported transient adverse events, most commonly localized pain, excessive weakness, unsteadiness and increased falls, and fatigue. Five patients were reported to have developed urinary incontinence and two, dysphagia. The authors recommended that for children with CP who have localized or segmental spasticity in the upper and lower extremities, BoNT-A should be offered as an effective and generally safe treatment. They stated that there was insufficient evidence to either support or discourage the use of BoNT-A to improve motor function in these children.

Diazepam: Based on the results of one Class I study (n=180) and one Class II study, the authors concluded that diazepam is probably effective for the short-term treatment of spasticity in children with CP. They noted that no studies formally considered whether it improved motor function and that there was insufficient evidence to support or refute its use for this purpose. Most studies reported ataxia and drowsiness as side effects. They recommended that diazepam be considered a short-term anti-spasticity treatment and noted that adverse effects such as sedation, drowsiness, hypersalivation, and weakness limit its usefulness for long-term use and that prolonged use can produce physical dependence.

Dantrolene: The authors identified one Class I, two Class II and two Class IV studies. The Class I study and one of the Class II studies had conflicting results. Weakness, drowsiness, and irritability were frequent side effects. The authors stated that there was insufficient evidence to support or refute the use of dantrolene and noted that, in their experience, this agent is rarely used in clinical practice.

Baclofen (oral): There were two Class II studies (one with 20 children and one with 15) and one Class IV study. The results of the Class II studies were conflicting and some participants in the larger study suffered somnolence or sedation (20%) and hypotonia (15%). The authors concluded that there was insufficient evidence to support or refute the use of this medication but they noted that it is widely used in clinical practice.

Tizanidine: There was one small Class II placebo-controlled parallel study (10 children in the treatment group and 30 in the placebo group). There was a significant reduction in spasticity in the treatment group and no side effects were reported. The authors concluded that this agent is possibly effective and recommended that its use may be considered in children with CP and spasticity. They noted that this agent is used in adults with multiple sclerosis and spinal cord injury with reported adverse effects including hypotension, sedation, asthenia, dry mouth, dizziness, hallucinations, and hepatotoxicity.

Intrathecal baclofen pump: This device is a surgically implanted pump that delivers medication via a catheter into the spinal cord. There was one Class III and five Class IV studies and all reported reduced spasticity. Common adverse effects were: catheter malfunction (43%), wound infection (39%), seromas (29%) and CSF leaks (17%). Less common side effects included headache, vomiting, lethargy, disorientation, agitation, irritability, and meningitis. The authors concluded that the data regarding the use of continuous intrathecal baclofen in children with CP and spasticity are inadequate and that there is insufficient evidence to support or refute its use. They noted the impracticality of RCTs of this technology.

Other Relevant Publications


This concise guideline intended for parents provides an overview of cerebral palsy and its treatment.
**Autism Spectrum Disorder**

**Introduction**

The diagnostic criteria for autism have recently changed. In May 2013, the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) moved to a single overarching Autism Spectrum Disorder (ASD) diagnosis [59]. Previously, DSM-IV had allowed for four separate diagnoses: autistic disorder, Asperger disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified. Research suggested, however, that these diagnoses were not being consistently applied [60], and that many clinicians were finding it difficult to differentiate between these four categories [59]. A single umbrella category was thus seen as better reflecting current knowledge [60].

The new DSM-V uses two symptom dimensions to define ASD, with further sub-classifications being based on functional severity [59]. The first dimension is a persistent impairment in reciprocal social communication and interaction. This includes problems with social-emotional reciprocity (e.g. failure of normal back-and-forth conversation); problems with nonverbal communication (e.g. abnormalities in eye contact, body language, and understanding gestures); and problems with developing, maintaining, and understanding relationships (e.g. difficulties adjusting behaviour to suit different social contexts, difficulties with making friends) [61]. The second dimension relates to the presence of restricted and repetitive patterns of behaviour. This includes stereotyped or repetitive movements, use of objects, or speech; the insistence on sameness, including inflexible adherence to routines, and ritualized patterns of behaviour; the presence of highly restricted, fixated interests; and an unusual interest in the sensory aspects of the environment [61].

The current consensus is that early intervention improves outcomes for children with ASD. The aims of treatment are to foster growth in communication, cognitive abilities and social and daily living skills, and to reduce problem behaviours which interfere with learning [62]. Programmes often draw on procedures from special education, behavioural psychology and occupational therapy. While traditionally seen as adjuncts to other therapies, medications are occasionally used to manage problem behaviours and to enhance participation in educational programmes. [62]. Over time a large number of alternative treatments have been suggested, but evidence for the efficacy of many is limited, and some have actually caused direct harm [62].

The causes of ASD remain unknown. A genetic basis is supported by research which suggests an average concordance of 88% between identical twins. Other siblings of children with ASD are also at an increased risk, and the reported risk of recurrence in families is at least 4–7%. Single gene disorders (e.g. fragile X) are sometimes diagnosed in people with ASD, and the proportion with an abnormality on genetic evaluation is increasing, as more sophisticated tests become available. The majority of people with ASD, however, are thought to have idiopathic autism with no identifiable genetic abnormality. While this may reflect the limitations of current testing, the current view is that ASD develops when an array of genetic vulnerabilities, possibly in concert with epigenetic factors, or a gene-environment interaction, affects brain development [62].

At present there is no reliable information on the prevalence of ASD in New Zealand, although an estimate from the Statistics NZ Household Disability Survey suggested that 2,100 children may have ASD (personal communication Phillipa Clark 2006) giving a prevalence of 24.8 per 10,000. An estimate from Nelson Marlborough suggested a prevalence of 46 per 10,000 [63]. Some overseas estimates (based on parental report) have placed the prevalence as high as 110 per 10,000 [64].

In the absence of other more reliable data sources, the following section reviews hospital admissions for children and young people with any mention of autism or other pervasive developmental disorders in any of their first 15 diagnoses. Note: Given that DSM-V was only released in May 2013, the analysis uses ICD-10-AM codes based on the earlier definitions of autism and pervasive developmental disorders.
### Data Source and Methods

**Definition**

1. Hospital admissions for children and young people aged 0–24 years with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses

**Data Source**

1. National Minimum Dataset

**Numerator**: Hospital admissions for children and young people aged 0–24 years with autism or other pervasive developmental disorders (ICD-10-AM F84) listed in any of the first 15 diagnoses.

**Denominator**: Statistics New Zealand Estimated Resident Population (projected from 2007)

**Notes on Interpretation**

Note 1: This analysis focuses on hospital admissions for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses, rather than on the subset of admissions where a pervasive developmental disorder was the primary reason for admission. The rationale for this wider focus was the fact that the majority of those with these diagnoses were not hospitalised for their pervasive developmental disorder per se, but rather for a range of other conditions, some of which were potentially more likely as a result of their pervasive developmental disorder, and some of which were unrelated.

Note 2: As the majority of children and young people with pervasive developmental disorders are managed in the outpatient or primary care setting, it is likely that this analysis significantly underestimates the number of children and young people with these diagnoses. The rationale for the methodology used however, was the absence of other more reliable sources of information on the number of children and young people with autism or other pervasive developmental disorders in the community.

Note 3: If no mention of a pervasive developmental disorder was made in any of the first 15 diagnoses, then these cases have not been included, even if the patient was diagnosed with a pervasive developmental disorder on a previous admission.

### New Zealand Distribution and Trends

#### Distribution by Primary Diagnosis

In New Zealand during 2008–2012, autism and other pervasive developmental disorders were listed as the primary diagnosis in only 14.3% of hospitalisations for children and young people with a pervasive developmental disorder in any of the first 15 diagnoses. Of those with a pervasive developmental disorder listed as the primary diagnosis, 64.0% had childhood autism, 22.8% had Asperger syndrome and 13.3% had other pervasive developmental disorders. Overall, 23.8% of admissions in children and young people with pervasive developmental disorders were for dental caries or other oral health conditions, while a further 8.5% were for epilepsy or convulsions (Table 59).

#### Distribution by Age

In New Zealand during 2008–2012, hospital admissions for children and young people with pervasive developmental disorders increased rapidly during the preschool years, reached a peak at eight years of age, and then declined (Figure 32). During 2006–2010, five children or young people had a pervasive developmental disorder listed as the main underlying cause of death, or as a contributory cause, with all of these deaths occurring in those over ten years of age.
### Table 59. Hospital Admissions in Children and Young People Aged 0–24 Years with Autism or Other Pervasive Developmental Disorders by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Admissions in those with Pervasive Developmental Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pervasive Developmental Disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Childhood Autism</td>
<td>222</td>
<td>44.4</td>
<td>2.91</td>
<td>9.1</td>
</tr>
<tr>
<td>Asperger Syndrome</td>
<td>79</td>
<td>15.8</td>
<td>1.03</td>
<td>3.3</td>
</tr>
<tr>
<td>Other Pervasive Developmental Disorders</td>
<td>46</td>
<td>9.2</td>
<td>0.60</td>
<td>1.9</td>
</tr>
<tr>
<td>Total Autism and Other Pervasive Developmental Disorders</td>
<td>347</td>
<td>69.4</td>
<td>4.54</td>
<td>14.3</td>
</tr>
<tr>
<td><strong>Other Diagnoses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dental Caries</td>
<td>454</td>
<td>90.8</td>
<td>5.95</td>
<td>18.7</td>
</tr>
<tr>
<td>Other Dental and Oral Health Issues</td>
<td>124</td>
<td>24.8</td>
<td>1.62</td>
<td>5.1</td>
</tr>
<tr>
<td>Epilepsy and Status Epilepticus</td>
<td>138</td>
<td>27.6</td>
<td>1.81</td>
<td>5.7</td>
</tr>
<tr>
<td>Unspecified Convulsions</td>
<td>68</td>
<td>13.6</td>
<td>0.89</td>
<td>2.8</td>
</tr>
<tr>
<td>Mood Disorders</td>
<td>83</td>
<td>16.6</td>
<td>1.09</td>
<td>3.4</td>
</tr>
<tr>
<td>Schizophrenia, Schizotypal and Delusional Disorders</td>
<td>81</td>
<td>16.2</td>
<td>1.06</td>
<td>3.3</td>
</tr>
<tr>
<td>Other Mental and Behavioural Disorders</td>
<td>116</td>
<td>23.2</td>
<td>1.52</td>
<td>4.8</td>
</tr>
<tr>
<td>Respiratory Infections and Diseases</td>
<td>89</td>
<td>17.8</td>
<td>1.17</td>
<td>3.7</td>
</tr>
<tr>
<td>Constipation</td>
<td>45</td>
<td>9.0</td>
<td>0.59</td>
<td>1.9</td>
</tr>
<tr>
<td>Infectious and Parasitic Diseases</td>
<td>29</td>
<td>5.8</td>
<td>0.38</td>
<td>1.2</td>
</tr>
<tr>
<td>Respite Care</td>
<td>27</td>
<td>5.4</td>
<td>0.35</td>
<td>1.1</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>826</td>
<td>165.2</td>
<td>10.82</td>
<td>34.0</td>
</tr>
<tr>
<td>Total Other Diagnoses</td>
<td>2,080</td>
<td>416.0</td>
<td>27.24</td>
<td>85.7</td>
</tr>
<tr>
<td>Total</td>
<td>2,427</td>
<td>485.4</td>
<td>31.78</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Figure 32. Hospital Admissions for Children and Young People with Autism or Other Pervasive Developmental Disorders by Age, New Zealand 2008–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Distribution by Ethnicity and Gender

In New Zealand during 2008–2012, hospital admissions for those with autism or other pervasive developmental disorders were significantly higher for males. Admissions were also significantly higher for European/Other > Māori and Asian/Indian > Pacific children and young people (Table 60).

Similarly during 2000–2012, admission rates for European/Other children and young people with autism or other pervasive developmental disorders were consistently higher than for Asian/Indian, Māori and Pacific children and young people, although rates for all ethnic groups increased during this period (Figure 33).

Table 60. Hospital Admissions for Children and Young People Aged 0–24 Years with Autism or Other Pervasive Developmental Disorders by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prioritised Ethnicity</td>
<td>Gender</td>
<td></td>
<td></td>
<td>Prioritised Ethnicity</td>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Autism or Other Pervasive Developmental Disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>23.71</td>
<td>0.61</td>
<td>0.52–0.70</td>
<td>Female</td>
<td>19.19</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>39.17</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>43.75</td>
<td>2.28</td>
<td>2.09–2.49</td>
</tr>
<tr>
<td>Māori</td>
<td>23.70</td>
<td>0.61</td>
<td>0.54–0.67</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>16.29</td>
<td>0.42</td>
<td>0.34–0.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000
Figure 33. Hospital Admissions for Children and Young People 0–24 Years with Autism or Other Pervasive Developmental Disorders by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised

**Northern DHBs Distribution and Trends**

**Northern DHBs Distribution**

In the Northern region during 2008–2012, a total of 38 Northland, 246 Waitemata, 164 Auckland and 187 Counties Manukau children and young people were hospitalised with a diagnosis of autism or other pervasive developmental disorders. Admission rates per 100,000 population were *significantly* lower than the New Zealand rate in Northland and Counties Manukau but *significantly* higher in the Waitemata DHB. Rates in Auckland DHB however, were not *significantly* different from the New Zealand rate (Table 61).
### Table 61. Hospital Admissions for Children and Young People Aged 0–24 Years with Autism or Other Pervasive Developmental Disorders, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A*</td>
<td>B*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Autism and Other Pervasive Developmental Disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>38</td>
<td>38</td>
<td>45</td>
<td>0.24</td>
<td>16.65</td>
<td>0.52</td>
</tr>
<tr>
<td>Waitemata</td>
<td>243</td>
<td>246</td>
<td>367</td>
<td>0.30</td>
<td>38.88</td>
<td>1.22</td>
</tr>
<tr>
<td>Auckland</td>
<td>160</td>
<td>164</td>
<td>214</td>
<td>0.26</td>
<td>28.57</td>
<td>0.90</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>184</td>
<td>187</td>
<td>268</td>
<td>0.29</td>
<td>27.12</td>
<td>0.85</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1,528</td>
<td>2,427</td>
<td>0.32</td>
<td>31.78</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (i.e. DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (i.e. the sum of DHB totals exceeds New Zealand total); Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics.

### Northern DHBs Trends

In the Northern DHBs during 2000–2012, large variations in rates made trends in hospital admissions for those with autism or other pervasive developmental disorders difficult to interpret (Figure 34).
Figure 34. Hospital Admissions for Children and Young People Aged 0–24 Years with Autism or Other Pervasive Developmental Disorders, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Local Policy Documents and Evidence-Based Reviews Relevant to Autism Spectrum Disorder

In New Zealand there are a number of guidelines relevant to autism spectrum disorder and these are summarized in Table 62, along with a range of publications which consider ASD in the overseas context.

Table 62. Local Policy Documents and Evidence-Based Reviews Relevant to Autism

<table>
<thead>
<tr>
<th>Ministry of Health and Ministry of Education Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>This document, intended as a resource for practitioners in the health, disability and education sectors, contains a brief review of the available validated diagnostic instruments for autism spectrum disorder. It describes their basic characteristics including appropriate use and setting, statistical properties, requirements in terms of user qualifications and training, and licensing arrangements. It also sets out some potentially preferable combinations of instruments for screening and diagnosis of autism, and for screening for Asperger disorder.</td>
</tr>
<tr>
<td>This guideline provides evidence-based information for all those involved in providing services for people with autism and their families including health, disability and education professionals. The eight parts of the guideline cover Diagnosis and initial assessment of Autism Spectrum Disorder (ASD), Support for individuals, families and carers, Education for learners with ASD, Treatment and management of ASD, Living in the community, Professional learning and development, Māori perspectives and Pacific peoples’ perspectives. Recommendations in the guidelines are graded using NZ Guidelines Group Grading System. Grades are based on the quality, quantity, consistency, applicability and clinical impact of the studies forming the relevant body of evidence. The information in the guidelines should be read in conjunction with the supplementary papers produced by the Living Guidelines group to update the recommendations in the Guidelines according to new evidence. These reviews are:</td>
</tr>
</tbody>
</table>
**International Guidelines**


This abbreviated guideline covers the recognition, referral and diagnosis of autism in children and young people from birth up to 19 years. It does not cover the management of the condition. Its recommendations are listed under the following headings: Local pathway for recognition, referral and diagnostic assessment of possible autism. Recognising children and young people with possible autism. Referring children and young people to the autism team. After referral to the autism team. Autism diagnostic assessment for children and young people. After the autism diagnostic assessment. Medical investigations. Communicating the results from the autism diagnostic assessment. and Information and support for families and carers. There are also research recommendations relating to training professionals, gathering information in schools or nurseries, additional assessments and comparative genomic hybridisation array.

The full version of the above guideline is:


This guideline is intended for people working in, or using, the NHS in England and Wales, including professionals, service commissioners and planners, and children, young people and parents going through the referral and diagnosis process for autism. The guidelines address a series of clinical questions relating to the following main outcomes: signs and symptoms of autism, specificity and sensitivity of tools to identify an increased likelihood of autism and diagnostic tools, yield of medical and diagnostic tests, differential diagnoses, coexisting conditions, and children’s and young people’s views and the views of their parents and carers of the process of referral, assessment and diagnosis, and their support and information needs. For each clinical question there is an overview of the evidence and a table listing the relevant studies, and providing, for each study, a quality assessment and a summary of findings which includes numbers of cases and controls, and results with 95% confidence intervals. The evidence chapters are entitled: recognition, following referral, diagnostic assessment, differential diagnosis, assessment of coexisting conditions, medical investigations and information and support. The last chapter covers service descriptions and resource use.

The appendices for the above guideline can be found here:  

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**Guidelines from the James M. Anderson Center for Health Systems Excellence at Cincinnati Children’s Hospital**  

The Anderson Center at Cincinnati Children's Hospital has published a number of “Best Evidence Statements” related to care issues for children with autism. They contain brief details of studies relevant to the clinical questions addressed and care recommendations. Guidance on the following topics (with year of publication) can be found at these links:

- **Craniosacral Therapy for Children with Autism and/or Sensory Processing Disorder** (2011)
- **Use of Sensory Assessment Tools with Children diagnosed with Autism Spectrum Disorder (ASD)** (2009)
- **Speech Therapist Directed Use of Video Modeling for Patients with Autism Spectrum Disorder** (2012)
- **Use of a Weighted or Pressure Device to Modify Behavior in Children with a Sensory Processing Disorder** (2012)
- **Adding home based services to complement center based intervention for children with autism** (2013)

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[http://www.sign.ac.uk/pdf/sign98.pdf](http://www.sign.ac.uk/pdf/sign98.pdf)

This Scottish guideline aims to provide the evidence base and recommendations to inform clinical service provision, especially in regard to assessment, diagnosis and clinical intervention. It considers the evidence for working in partnership with children and young people and their parents and carers. It also considers the evidence relating to how multidisciplinary and multiagency working can best address the needs of people with ASD at all levels of care provision. It considers educational interventions which may influence clinical outcomes but does not examine educational and social opportunities offered to those with ASD which may add value to their lives and promote social inclusion.
This clinical report from the American Academy of Pediatrics reviews the educational strategies and associated therapies that are the primary treatments for children with ASD. It aims to assist paediatricians in educating parents and helping them to choose empirically supported interventions for their children. It also covers important health care issues including management of associated medical problems, pharmacologic and non-pharmacologic intervention for challenging behaviours or coexisting mental health conditions, and use of complementary and alternative medical treatments.


These guidelines address identification, assessment, diagnosis and access to early interventions for pre-school and primary school age children with ASD. The recommendations are A, B, or C as follows: Grade A (at least one RCT), Grade B (well conducted clinical trials but no RCTs) and Grade C (expert NIASA Working Group recommendation).

Recent Systematic and Other Reviews

Cheuk KLD, Wong V, Chen XW. 2013. Acupuncture for autism spectrum disorders (ASD). Cochrane Database of Systematic Reviews(7)

This review assessed the effectiveness of acupuncture for people with ASD in improving core autistic features, as well as communication, cognition, overall functioning and quality of life, and to determine if it has any adverse effects. The authors identified ten trials involving 390 children with ASD in Hong Kong, mainland China and Egypt. The children’s ages ranged from three to 18 years and the lengths of treatment from four weeks to nine months. The authors concluded that current evidence does not support the use of acupuncture for ASD since the trials in children have been inconclusive and there have been no RCTs in adults with ASD.


This review considered whether treatment with an SSRI: 1. improves the core features of autism (social interaction, communication and behavioural problems); 2. improves other non-core aspects of behaviour or function such as self-injurious behaviour; 3. improves the quality of life of adults or children and their carers; 4. has short- and long-term effects on outcome; and 5. causes harm. Nine RCTs (320 participants in total) were included, evaluating four different SSRIs: fluoxetine (three studies), fluvoxamine (two studies), fenfluramine (two studies) and citalopram (two studies). Five studies included only children and four, only adults. The studies varied in their inclusion criteria relating to diagnosis and IQ and also in their outcome measures so meta-analysis of study results was not possible except for the outcome ‘proportion improvement’. One large, high-quality study in children found no evidence of positive effect of citalopram. Three small studies in adults showed positive outcomes for Clinical Global Impression and obsessive-compulsive behaviour; one study showed improvements in aggression, and another in anxiety. The authors concluded that here is no evidence of beneficial effect of SSRIs in children and emerging evidence of harm and also that there is limited evidence of the effectiveness of SSRIs in adults from small studies in which risk of bias is unclear.


Young children with autism have impairments in social interaction and communication and often also exhibit repetitive and/or non-compliant behaviour. These characteristics are difficult for parents so helping parents develop interaction and behaviour management strategies are a common focus for early interventions in ASD. This review assessed the effectiveness of parent-mediated early interventions in terms of the benefits for both children with ASD and their parents and to explore some potential moderators of treatment effect. It included 17 studies (all RCTs) involving 919 children with ASD in six countries (USA, UK, Australia, Canada, Thailand and China). The studies differed in the theoretical basis underpinning interventions, the duration and intensity of interventions, and the way outcomes were measured but meta-analyses were possible for subsets of data from ten studies that evaluated interventions to enhance parent interaction style and thereby facilitate children's communication. The authors concluded that there was some evidence for the effectiveness of parent-mediated interventions, especially in proximal indicators within parent-child interaction such as improvement in parent-child synchrony in observed interactions, but also in more distal indicators of child language comprehension and reduction in severity of autism characteristics. The effect sizes were generally small but the authors stated that for a serious disorder like ASD, even small gains could serve as pointers to potentially effective approaches for managing early childhood autism. The evidence on whether such interventions may reduce parent stress was found to be inconclusive.

Tan LM, Ho JJ, Teh HK. 2012. Polyunsaturated fatty acids (PUFAs) for children with specific learning disorders. Cochrane Database of Systematic Reviews(12)

The authors of this review had hoped to assess the effects of polyunsaturated fatty acids (PUFAs) supplementation on learning outcomes for children with specific learning disorders (including those with co-existing developmental disorders such as ADHD and autism) but they were unable to find any relevant RCTs or quasi-RCTs. They concluded that there was insufficient evidence from which to draw any conclusions about the effectiveness of PUFAs for these children.

Early intensive behavioural intervention (EIBI) is one of the more well-established treatments for ASD. It is a treatment based on the principles of applied behaviour analysis and delivered for multiple years at an intensity of 20 to 40 hours per week. This review systematically reviewed the evidence for the effectiveness of EIBI in increasing the functional behaviours and skills of young children with ASD. The authors identified one RCT and four controlled clinical trials (203 participants in total) which met their inclusion criteria. They conducted meta-analyses using a random effects model on the four CCTs for the outcomes adaptive behaviour composite (behaviours that increase independence and the ability to adapt to the environment), IQ, communication and language skills, social competence, and daily living skills. The results (expressed as standard mean difference effect size) provided evidence that EIBI improves adaptive behaviour (SMD ES 0.69; 95% CI 0.69 to 1.01), IQ (SMD ES 0.76; 95% CI 0.40 to 1.11), expressive language (SMD ES 0.50; 95% CI 0.05 to 0.95) receptive language (SMD ES 0.57; 95% CI 0.20 to 0.94), everyday communication skills (SMD ES 0.20; 95% CI 0.13 to 0.28), everyday social competence (SMD ES 0.42; 95% CI 0.11 to 0.73), and daily living skills (SMD ES 0.55; 95% CI 0.24 to 0.87) for this population. The authors noted that the overall quality of the evidence was low as there is a high risk of bias due to the studies being non-randomised. They concluded that there was some limited evidence that EIBI is an effective behavioural treatment for some young children with ASD.


In 1998 secretin, a gastrointestinal hormone, was suggested as an effective treatment for autism spectrum disorders (ASD), based on anecdotal evidence. This review considered data from 14 RCTs of intravenous secretin compared to placebo involving over 900 children. The trials reported on twenty-five established standardised outcome measures and no more than four studies used any one outcome measure similarly. Outcomes were reported at between three and six weeks. The authors concluded that these trials provide no evidence that single or multiple dose intravenous secretin is effective and therefore at present it should not be recommended or administered as a treatment for ASD.

Reichow B, Steiner AM, Volkmar F. 2012. Social skills groups for people aged 6 to 21 with autism spectrum disorders (ASD). Cochrane Database of Systematic Reviews(7)

Social skills groups led by therapists aim to improve the social competence of people with ASD. They typically meet once per week over 12 or more weeks. Common topics covered include emotional recognition and regulation, social competence, social problem solving, and social communication. This review assessed the effectiveness of these groups for people with ASD. The authors found five relevant RCTs, all conducted in the U.S. There were 196 participants ranging in age from six to 21 years and, across all the studies, participants had a mean IQ in the average range. The authors conducted a random effects meta-analysis where this was possible and reported the results as standardized mean difference effect sizes (ES). They reported that there is some evidence that social skills groups improve overall social competence (ES = 0.47, 95% CI 0.16 to 0.78) and friendship quality (ES = 0.41, 95% CI 0.02 to 0.8) for this population. Data from two studies indicated no differences between treatment and control groups in relation to emotional recognition (ES = 0.34, 95% CI 0.20 to 0.88) and data from one study indicated no differences in social communication as related to the understanding of idioms (ES = 0.05, 95% CI −0.63 to 0.72). Two additional quality of life outcomes were evaluated, and results of single studies suggested decreases in loneliness (ES = −0.66, 95% CI 1.15 to −0.17) but no effect on child or parental depression. The authors concluded that there is some evidence that social skills groups can improve social competence for some children and adolescents with ASD but more research is needed.


Anti-psychotic drugs have been used to treat irritability associated with ASD. Aripiprazole, a third generation atypical antipsychotic, is a relatively new drug that has a unique mechanism of action different from other antipsychotics. This review examined the efficacy and safety of aripiprazole for people with ASD. There were two RCTs that had evaluated the use of aripiprazole in 316 children with ASD over a period of eight weeks. These trials were considered to be at low risk of bias. Meta-analysis of study results indicated a mean improvement of 6.17 points on the Aberrant Behavior Checklist (ABC) irritability subscale, 7.93 points on the ABC hyperactivity subscale, and 2.66 points in the stereotypy subscale in children treated with aripiprazole compared to children treated with a placebo. Adverse effects were a greater increase in weight (mean increase of 1.13kg relative to placebo) and a higher risk ratio for sedation (RR 4.28) and tremor (RR 10.26). The authors concluded that the evidence suggests that aripiprazole can be effective in treating some behavioural aspects of ASD in children, in particular irritability, hyperactivity, and stereotypies (repetitive, purposeless actions), but that the benefits need to be weighed against significant side effects such as weight gain, sedation, drooling and tremor. They stated that longer term studies are needed.
Tricyclic antidepressants (TCAs) block noradrenaline and serotonin reuptake, thereby increasing the availability of these neurotransmitters in the central nervous system. They have been used as in the treatment of autistic symptoms and comorbidities, especially anxiety and obsessive-compulsive type behaviours, in people with ASD. This review considered whether treatment of people with ASD using tricyclic antidepressants: 1. improves the core features of autism, including restricted social interaction, restricted communication and stereotypical and repetitive behaviours; 2. improves non-core features such as challenging behaviours; 3. improves comorbid states, such as anxiety and depression; and 4. causes adverse effects. The authors found three small RCTs, with between 12 and 32 participants, most of whom had significant intellectual disability. Two trials assessed clomipramine and one, tianeptine. One trial (of clomipramine) included both adults and children, the other two trials only children. Meta-analysis of study results was not possible due to the differences between trials in participant characteristics, medications investigated and outcomes measured. The authors concluded that clinicians contemplating prescribing these medications for people with ASD need to be aware of the limited and inconsistent evidence for the effectiveness of these drugs and the possibility of adverse effects, including interference with cardiac conduction, drowsiness and reduced activity levels.

It has been suggested that deficiency of omega-3 fatty acids may partially explain the impairments associated with ASD and that people with ASD may benefit from Omega-3 supplementation. This review assessed the efficacy of omega-3 fatty acids for improving core features of ASD (such as social interaction, communication, and stereotypies) and associated symptoms. It included two RCTs involving 37 children with ASD who received either omega-3 fatty acids supplementation or a placebo. These trials did not indicate that omega-3 supplements had an effect on social interaction (mean difference (MD) 0.82, 95% confidence interval (CI) −2.84 to 4.48, I² = 0%), communication (MD 0.62, 95% CI −0.89 to 2.14, I² = 0%), stereotypy (MD 0.77, 95% CI −0.69 to 2.22, I² = 8%), or hyperactivity (MD 3.46, 95% CI −7.79 to 7.70, I² = 0%). The authors concluded that there was no high quality evidence omega-3 fatty acids supplementation is effective for improving core and associated symptoms of ASD.

Risperidone is an atypical antipsychotic drug, commonly used to treat schizophrenia, which has been used for symptom relief and improvement of problem behaviours, such as self-injury or aggression, in people with autism. This review aimed to evaluate the efficacy and safety of risperidone in people with ASD. The authors identified three short-term (three months or less) RCTs comparing risperidone to placebo. Numbers of participants ranged from 31 to 101. One study involved adults only and the others 5–17 year olds and 5–12 year olds. Two studies had a high proportion of participants leaving the study (20% to 25%). Meta-analysis was possible for three outcomes: Clinical Global Impression Scale, Aberrant Behavior Checklist and weight gain. The authors found that there was some evidence for the benefits of risperidone in irritability, repetition and social withdrawal but there were also adverse effects, most notably weight gain (in the two studies of children, 2.7kg (95% CI 1.15 to 2.41) in the treatment group).

Auditory integration therapy, and the similar therapies, the Tomatis Method and Samonas Sound Therapy, are techniques for improving abnormal sound sensitivity in individuals with behavioural disorders including ASD. This review evaluated the effectiveness of auditory integration training and similar therapies in people with ASD. The authors identified six RCTs of auditory integration therapy and one of Tomatis therapy, involving a total of 182 individuals aged three to 39 years. Five trials had fewer than 20 participants and allocation concealment was inadequate in all of the studies. Across all the studies 20 different outcome measures were used and only two outcomes were measured in three or more studies. Meta-analysis was not possible due to the heterogeneity of the studies. Three small studies reported improvements at three months for the auditory integration therapy group based on the Aberrant Behaviour Checklist, but the authors noted that they used a total score rather than subgroup scores, which is questionable validity (it is an incorrect use of the checklist according to the checklist’s developer), and that the results of one of these studies did not reach statistical significance. Another study also reported improvements at three months in the auditory integration therapy group for the Aberrant Behaviour Checklist subgroup scores. The one small cross-over study of Tomatis therapy described an improvement in language that was similar in the treatment and placebo groups. The authors conclude that there is no evidence that auditory integration therapy or other sound therapies are effective as treatments for ASD but also not sufficient evidence to prove they are ineffective due to the disparate outcome measures between studies.

Music therapy for people with autism spectrum disorders aims to enhance communication and expression using music and its elements. This review assessed the effect of such therapy. The authors identified three very small studies (four to ten participants in each, 24 in total) meeting their inclusion criteria, all from the U.S. Two were RCTs and the other called itself “counterbalanced”. The trials were all very short, lasting from one to four weeks and the duration of treatment was only one week (with one session per day) in each study. Music therapy was found to be superior to “placebo” therapy with respect to verbal and gestural communicative skills (verbal: 2 RCTs, n = 20, SMD 0.36 CI 0.15 to 0.57; gestural: 2 RCTs, n = 20, SMD 0.50 CI 0.22 to 0.79). Effects on behavioural problems were not significant. The authors concluded that the studies included in their review were of limited applicability to clinical practice but their findings indicated that music therapy may help some children with ASD to improve their communication skills.

It has been suggested that peptides from the dietary proteins casein and gluten may have a role in the origins of autism and that gluten and/or casein free diets may be beneficial for people with ASD. This review identified only two small RCTs of a gluten and casein free diet vs. a normal diet, one with 15 children (lasting 12 weeks, with cross-over at six weeks) and one with 20 (lasting 12 months). Meta-analysis was not possible. The results of the longer study indicated a beneficial effect of the diet on reduction in autistic traits (mean difference (MD, intervention − control) = −5.60; 95% CI −9.02 to −2.18; z = 3.21; p=0.001), communication and interaction (MD = 1.70; 95% CI 0.50 to 2.90; z = 2.77; p = 0.006) and social isolation (MD = −3.20; 95% CI −5.20 to −1.20; z = 3.14; p = 0.002). The shorter study found no significant differences between the intervention and control groups. The authors noted that the outcome ‘social isolation’ was a component of the outcome ‘autistic traits’ (implying that these two outcomes are not independent). They pointed out that gluten and casein-free diets are costly to the family both in monetary terms and in convenience and they concluded that these diets could not be recommended on the basis of the limited evidence available.


For several decades, large doses of vitamin B6 and magnesium have been reported to be beneficial for people with ASD. This review assessed the efficacy of vitamin B6 and magnesium (B6-Mg) for improving behaviour in the areas of social interaction, communication, and behavioural responses to environmental stimuli in children and adults with ASD. Although there have been many published studies of this therapy which were not RCTs, the authors of this review identified only three small RCTs, with a total of 33 participants aged 3–18 years and intervention periods which varied from four to 20 weeks. They concluded that, due to the small number of studies, the methodological deficiencies of the studies, and the small sample sizes, no recommendations can be made based on their review regarding the use of B6-Mg as a treatment for autism. They stated that there is insufficient evidence to demonstrate efficacy of B6-Mg.

Other Relevant Publications


This study reported on interviews with the parents and whānau of 19 Māori children with ASD who shared their experiences of raising their children. It includes information on significant areas of unmet needs.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
**ECZEMA AND DERMATITIS**

**Introduction**

Eczema and dermatitis are terms that are often used interchangeably. They are common skin conditions affecting up to one in five people during their lifetime, with a wide clinical spectrum from mild to severe disease. They can be acute, chronic, or both [65]. Acute eczema/dermatitis is an inflammation of the skin that is usually itchy, red and swollen. Chronic eczema/dermatitis is a longer-term inflammation, resulting in skin that is usually darker, scratched and thicker (lichenified) than the surrounding skin. Specific types include: atopic dermatitis; allergic and contact dermatitis; and seborrhoeic dermatitis.

Atopic dermatitis usually appears early in infancy, and persists into adulthood in a significant proportion of people. Risk factors at the population level include increased urbanisation, family history of atopy, higher socio-economic status, and higher educational levels of families. At the individual level, common causes include allergies such as dust mites, pollen or foods. There is also a major underlying genetic association between the filaggrin gene and atopic dermatitis, which may provide further understanding of the role of environmental and lifestyle factors for some children. More recent evidence suggests there may different subtypes of atopic dermatitis. For some subtypes progression to asthma and allergic rhinitis may occur, but for others it may not [65]. Psychological stresses can provoke or aggravate dermatitis, presumably by suppressing normal immune mechanisms.

Risk factors for allergic contact dermatitis include atopic dermatitis, skin barrier defects and intense or repetitive contact with irritants such as metals, cleaning products and perfume. [66]. Patch testing is the gold standard diagnostic test for allergic contact dermatitis. The sensitisation rate increases with age as children are exposed to more environmental factors, but also appears to be more frequent in recent years [66].

New Zealand is among countries with the highest prevalence (15-20%) of eczema and dermatitis [67]. Most children will not need to be hospitalized for this condition. However, infected eczema, controlling of the itch/scratch cycle and intensification of topical therapy may be indications for more intensive treatment and management in hospital.

The following section briefly reviews hospitalizations for children aged 0–14 years with any mention of eczema or dermatitis in any of the first 15 diagnoses, before considering those children for whom eczema or dermatitis was the primary reason for admission. The rationale for the greater focus on the latter group was the finding that the majority of children with eczema or dermatitis were admitted for conditions unrelated to their eczema (e.g. bronchiolitis, gastroenteritis) and in such cases, it was unclear the extent to which the child’s eczema contributed to their admission.

<table>
<thead>
<tr>
<th>Data Source and Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
</tr>
<tr>
<td>1. Hospital admissions for children 0–14 years with eczema or dermatitis listed in any of their first 15 diagnoses</td>
</tr>
<tr>
<td>2. Hospital admissions for children 0–14 years with eczema or dermatitis listed as a primary diagnosis</td>
</tr>
<tr>
<td><strong>Data Source</strong></td>
</tr>
<tr>
<td>1. National Minimum Dataset</td>
</tr>
<tr>
<td><strong>Numerator</strong>: Hospital admissions for children aged 0–14 years with eczema or dermatitis (ICD-10-AM L01.1, L20–L26, L27.2, L28–L30, B000, H01.1) listed in any of their first 15 diagnoses</td>
</tr>
<tr>
<td><strong>Numerator</strong>: Hospital admissions for children aged 0–14 years with eczema or dermatitis (ICD-10-AM L01.1, L20–L26, L27.2, L28–L30, B000, H01.1) listed as a primary diagnosis</td>
</tr>
<tr>
<td><strong>Denominator</strong>: Statistics New Zealand Estimated Resident Population (projected from 2007)</td>
</tr>
<tr>
<td>Individual diagnoses include: Eczema Herpeticum (B00.0); Seborrhoeic Dermatitis (L21); Diaper Dermatitis (L22); Contact Dermatitis (L23–L25); Dermatitis due to Food Ingestions (L27.2); Atopic and Other Dermatitis (L20, L26, L28–L29, H01.1, L30.0–L30.2, L30.4–L30.5, L30.8–L30.9); and Infective Dermatitis (L01.1, L30.3).</td>
</tr>
<tr>
<td>Broader diagnostic groupings include: Infective Dermatitis (L01.1, L303); and Other Eczema and Dermatitis (L20L–L26, L28–L29, B00.0, H01.1, L27.2, L30.0–L30.2, L30.4–L30.5, L30.8–L30.9).</td>
</tr>
</tbody>
</table>
Notes on Interpretation
Apart from the first table in each section (NZ and DHB), which considers the primary diagnoses assigned to children hospitalised with eczema or dermatitis in any of their first 15 diagnoses, this analysis focuses on hospitalisations where eczema or dermatitis were the primary reasons for admission. The rationale for the narrower focus was the finding that the majority of children with eczema or dermatitis listed in their first 15 diagnoses were admitted for conditions unrelated to their eczema (e.g. bronchiolitis, gastroenteritis). In such cases, it was unclear how severe the child’s eczema was, how it contributed to their admission, or the extent to which the characteristics of those admitted for other reasons, reflected the demographic profiles of those with eczema or dermatitis. Admissions with a primary diagnosis of eczema or dermatitis were thus seen as a better reflection of the need for acute secondary health services in children with eczema or dermatitis.

New Zealand Distribution and Trends

Admissions with Eczema or Dermatitis in First 15 Diagnoses

Table 63. Hospital Admissions for Children Aged 0–14 Years with Eczema or Dermatitis, by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Eczema or Dermatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eczema and Dermatitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atopic and Other Dermatitis</td>
<td>1,631</td>
<td>326.2</td>
<td>36.57</td>
<td>12.6</td>
</tr>
<tr>
<td>Infective Dermatitis</td>
<td>1,394</td>
<td>278.8</td>
<td>31.26</td>
<td>10.8</td>
</tr>
<tr>
<td>Eczema Herpeticum</td>
<td>208</td>
<td>41.6</td>
<td>4.66</td>
<td>1.6</td>
</tr>
<tr>
<td>Contact Dermatitis</td>
<td>134</td>
<td>26.8</td>
<td>3.00</td>
<td>1.0</td>
</tr>
<tr>
<td>Dermatitis due to Food Ingestions</td>
<td>118</td>
<td>23.6</td>
<td>2.65</td>
<td>0.9</td>
</tr>
<tr>
<td>Diaper Dermatitis</td>
<td>111</td>
<td>22.2</td>
<td>2.49</td>
<td>0.9</td>
</tr>
<tr>
<td>Seborrhoeic Dermatitis</td>
<td>109</td>
<td>21.8</td>
<td>2.44</td>
<td>0.8</td>
</tr>
<tr>
<td>Total Eczema or Dermatitis</td>
<td>3,705</td>
<td>741.0</td>
<td>83.07</td>
<td>28.7</td>
</tr>
<tr>
<td>Total Other Diagnoses*</td>
<td>9,212</td>
<td>1,842.4</td>
<td>206.55</td>
<td>71.3</td>
</tr>
<tr>
<td>New Zealand Total</td>
<td>12,917</td>
<td>2,583.4</td>
<td>289.62</td>
<td>100.0</td>
</tr>
</tbody>
</table>

*Other Primary Diagnoses

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Eczema or Dermatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchiolitis</td>
<td>1,353</td>
<td>270.6</td>
<td>30.34</td>
<td>10.5</td>
</tr>
<tr>
<td>Asthma and Wheeze</td>
<td>1,074</td>
<td>214.8</td>
<td>24.08</td>
<td>8.3</td>
</tr>
<tr>
<td>Skin Infections</td>
<td>806</td>
<td>161.2</td>
<td>18.07</td>
<td>6.2</td>
</tr>
<tr>
<td>Prematurity and Low Birth Weight</td>
<td>776</td>
<td>155.2</td>
<td>17.40</td>
<td>6.0</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>617</td>
<td>123.4</td>
<td>13.83</td>
<td>4.8</td>
</tr>
<tr>
<td>Pneumonia and Unspecified LRTI*</td>
<td>510</td>
<td>102.0</td>
<td>11.44</td>
<td>3.9</td>
</tr>
<tr>
<td>Acute Upper Respiratory Infections</td>
<td>438</td>
<td>87.6</td>
<td>9.82</td>
<td>3.4</td>
</tr>
<tr>
<td>Injury and Poisoning</td>
<td>385</td>
<td>77.0</td>
<td>8.63</td>
<td>3.0</td>
</tr>
<tr>
<td>Other Perinatal Conditions</td>
<td>356</td>
<td>71.2</td>
<td>7.98</td>
<td>2.8</td>
</tr>
<tr>
<td>Other Infectious Diseases</td>
<td>329</td>
<td>65.8</td>
<td>7.38</td>
<td>2.5</td>
</tr>
<tr>
<td>Viral Infections Unspecified</td>
<td>251</td>
<td>50.2</td>
<td>5.63</td>
<td>1.9</td>
</tr>
<tr>
<td>Cancer Neoplasms</td>
<td>130</td>
<td>26.0</td>
<td>2.91</td>
<td>1.0</td>
</tr>
<tr>
<td>Epilepsy and Convulsions</td>
<td>114</td>
<td>22.8</td>
<td>2.56</td>
<td>0.9</td>
</tr>
<tr>
<td>Urinary Tract Infection</td>
<td>97</td>
<td>19.4</td>
<td>2.17</td>
<td>0.8</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>1,976</td>
<td>395.2</td>
<td>44.31</td>
<td>15.3</td>
</tr>
<tr>
<td>Total Other Primary Diagnoses</td>
<td>9,212</td>
<td>1,842.4</td>
<td>206.55</td>
<td>71.3</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children with eczema or dermatitis listed in any of their first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: "LRTI: Lower Respiratory Tract Infection"
Distribution by Primary Diagnosis
In New Zealand during 2008–2012, only 28.7% of hospitalisations in children with eczema or dermatitis listed in any of their first 15 diagnoses, had eczema or dermatitis listed as the primary reason for their admission. Atopic and other dermatitis (12.6%) and infective dermatitis (10.8%) were the most frequent primary diagnoses assigned to those with eczema or dermatitis, while bronchiolitis and asthma and wheeze were the most frequent non-eczema related reasons for admission (Table 63).

Admissions with Eczema or Dermatitis as a Primary Diagnosis

Distribution by Age
In New Zealand during 2008–2012, hospital admissions for infective dermatitis and other forms of eczema and dermatitis were highest in infants aged less than one year, with rates then tapering off during the preschool years. Admission rates were lowest amongst children over five years of age (Figure 35).

Figure 35. Hospital Admissions for Children with a Primary Diagnosis of Eczema or Dermatitis by Age, New Zealand 2008–2012

Distribution by Ethnicity and Gender
In New Zealand during 2008–2012, hospital admissions for those with a primary diagnosis of infective eczema, or other eczema and dermatitis were both significantly higher in males than females. Admission rates for both outcomes were also significantly higher for Māori and Pacific > Asian/Indian > European/Other children (Table 64).

Trends by Ethnicity
Similarly during 2000–2012, hospitalisations for infective eczema and other eczema and dermatitis were consistently higher for Māori and Pacific children, than for European/Other and Asian/Indian children. While admission rates for Asian/Indian and European/Other children were similar during the early 2000s, rates for Asian/Indian children became higher than for European/Other children from the mid to late 2000s onwards. Admission rates for both outcomes increased for children of all ethnic groups during this period (Figure 36).
### Table 64. Hospital Admissions for Children Aged 0–14 Years with a Primary Diagnosis of Eczema or Dermatitis by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infective Dermatitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>22.24</td>
<td>1.82</td>
<td>1.45–2.29</td>
<td>Female</td>
<td>27.45</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>12.19</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>34.87</td>
<td>1.27</td>
<td>1.14–1.41</td>
</tr>
<tr>
<td>Māori</td>
<td>66.24</td>
<td>5.43</td>
<td>4.75–6.22</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>55.66</td>
<td>4.57</td>
<td>3.86–5.41</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Other Eczema and Dermatitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>64.02</td>
<td>2.37</td>
<td>2.06–2.72</td>
<td>Female</td>
<td>48.24</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>27.04</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>55.22</td>
<td>1.14</td>
<td>1.05–1.24</td>
</tr>
<tr>
<td>Māori</td>
<td>81.69</td>
<td>3.02</td>
<td>2.73–3.34</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>98.48</td>
<td>3.64</td>
<td>3.23–4.11</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>All Eczema and Dermatitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>86.25</td>
<td>2.20</td>
<td>1.95–2.48</td>
<td>Female</td>
<td>75.69</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>39.23</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>90.09</td>
<td>1.19</td>
<td>1.12–1.27</td>
</tr>
<tr>
<td>Māori</td>
<td>147.93</td>
<td>3.77</td>
<td>3.48–4.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>154.14</td>
<td>3.93</td>
<td>3.56–4.33</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children with eczema or dermatitis listed as a primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised.

### Figure 36. Hospital Admissions for Children Aged 0–14 Years with a Primary Diagnosis of Eczema or Dermatitis by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children with eczema or dermatitis listed as a primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised.
Northern DHBs Distribution and Trends

Admissions with Eczema or Dermatitis in First 15 Diagnoses

Distribution by Primary Diagnosis

In the Northern DHBs during 2008–2012, less than a quarter (range 20.4%–24.2%) of children hospitalised with eczema or dermatitis in any of their first 15 diagnoses, had eczema or dermatitis listed as the primary reason for their admission. Atopic and other dermatitis was the most frequent primary diagnoses assigned in those with eczema or dermatitis in all four DHBs, followed by infective dermatitis (Table 65, Table 66).

Table 65. Hospital Admissions for Children 0–14 Years with Eczema or Dermatitis by Primary Diagnosis, Northland, Waitemata and Auckland DHBs 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>No. of Admissions: Total 2008–2012</th>
<th>No. of Admissions: Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Admissions in those with Eczema and Dermatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eczema and Dermatitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atopic and Other Dermatitis</td>
<td>79</td>
<td>15.8</td>
<td>45.66</td>
<td>11.5</td>
</tr>
<tr>
<td>Infective Dermatitis</td>
<td>70</td>
<td>14.0</td>
<td>40.46</td>
<td>10.2</td>
</tr>
<tr>
<td>Diaper Dermatitis</td>
<td>6</td>
<td>1.2</td>
<td>3.47</td>
<td>0.9</td>
</tr>
<tr>
<td>Seborrhoeic Dermatitis</td>
<td>5</td>
<td>1.0</td>
<td>2.89</td>
<td>0.7</td>
</tr>
<tr>
<td>Eczema Herpeticum</td>
<td>5</td>
<td>1.0</td>
<td>2.89</td>
<td>0.7</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>521</td>
<td>104.2</td>
<td>301.14</td>
<td>75.9</td>
</tr>
<tr>
<td>Northland Total</td>
<td>686</td>
<td>137.2</td>
<td>396.51</td>
<td>100.0</td>
</tr>
<tr>
<td>Waitemata</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atopic and Other Dermatitis</td>
<td>135</td>
<td>27.0</td>
<td>24.25</td>
<td>9.1</td>
</tr>
<tr>
<td>Infective Dermatitis</td>
<td>92</td>
<td>18.4</td>
<td>16.53</td>
<td>6.2</td>
</tr>
<tr>
<td>Dermatitis due to Food Ingestions</td>
<td>24</td>
<td>4.8</td>
<td>4.31</td>
<td>1.6</td>
</tr>
<tr>
<td>Contact Dermatitis</td>
<td>18</td>
<td>3.6</td>
<td>3.23</td>
<td>1.2</td>
</tr>
<tr>
<td>Eczema Herpeticum</td>
<td>16</td>
<td>3.2</td>
<td>2.87</td>
<td>1.1</td>
</tr>
<tr>
<td>Seborrhoeic Dermatitis</td>
<td>9</td>
<td>1.8</td>
<td>1.62</td>
<td>0.6</td>
</tr>
<tr>
<td>Diaper Dermatitis</td>
<td>9</td>
<td>1.8</td>
<td>1.62</td>
<td>0.6</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>1,184</td>
<td>236.8</td>
<td>212.71</td>
<td>79.6</td>
</tr>
<tr>
<td>Waitemata Total</td>
<td>1,487</td>
<td>297.4</td>
<td>267.15</td>
<td>100.0</td>
</tr>
<tr>
<td>Auckland DHB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atopic and Other Dermatitis</td>
<td>168</td>
<td>33.6</td>
<td>42.58</td>
<td>14.1</td>
</tr>
<tr>
<td>Infective Dermatitis</td>
<td>60</td>
<td>12.0</td>
<td>15.21</td>
<td>5.0</td>
</tr>
<tr>
<td>Eczema Herpeticum</td>
<td>22</td>
<td>4.4</td>
<td>5.58</td>
<td>1.9</td>
</tr>
<tr>
<td>Diaper Dermatitis</td>
<td>19</td>
<td>3.8</td>
<td>4.82</td>
<td>1.6</td>
</tr>
<tr>
<td>Seborrhoeic Dermatitis</td>
<td>8</td>
<td>1.6</td>
<td>2.03</td>
<td>0.7</td>
</tr>
<tr>
<td>Contact Dermatitis</td>
<td>7</td>
<td>1.4</td>
<td>1.77</td>
<td>0.6</td>
</tr>
<tr>
<td>Dermatitis due to Food Ingestions</td>
<td>4</td>
<td>0.8</td>
<td>1.01</td>
<td>0.3</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>901</td>
<td>180.2</td>
<td>228.34</td>
<td>75.8</td>
</tr>
<tr>
<td>Auckland DHB Total</td>
<td>1,189</td>
<td>237.8</td>
<td>301.33</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children with eczema or dermatitis listed in any of their first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 66. Hospital Admissions for Children 0–14 Years with Eczema or Dermatitis by Primary Diagnosis, Counties Manukau DHB 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>No. of Admissions: Total 2008–2012</th>
<th>No. of Admissions: Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Admissions in those with Eczema and Dermatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eczema and Dermatitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Counties Manukau</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atopic and Other Dermatitis</td>
<td>216</td>
<td>43.2</td>
<td>35.89</td>
<td>10.5</td>
</tr>
<tr>
<td>Infective Dermatitis</td>
<td>154</td>
<td>30.8</td>
<td>25.59</td>
<td>7.5</td>
</tr>
<tr>
<td>Eczema Herpeticum</td>
<td>37</td>
<td>7.4</td>
<td>6.15</td>
<td>1.8</td>
</tr>
<tr>
<td>Seborrhoeic Dermatitis</td>
<td>25</td>
<td>5.0</td>
<td>4.15</td>
<td>1.2</td>
</tr>
<tr>
<td>Diaper Dermatitis</td>
<td>18</td>
<td>3.6</td>
<td>2.99</td>
<td>0.9</td>
</tr>
<tr>
<td>Contact Dermatitis</td>
<td>17</td>
<td>3.4</td>
<td>2.83</td>
<td>0.8</td>
</tr>
<tr>
<td>Dermatitis due to Food Ingestions</td>
<td>9</td>
<td>1.8</td>
<td>1.50</td>
<td>0.4</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>1,575</td>
<td>315.0</td>
<td>261.73</td>
<td>76.8</td>
</tr>
<tr>
<td>Counties Manukau Total</td>
<td>2,051</td>
<td>410.2</td>
<td>340.83</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children with eczema or dermatitis listed in any of their first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Admissions with Eczema or Dermatitis as a Primary Diagnosis

Northern DHBs Distribution

In the Northern Region during 2008–2012, 116 Northland, 242 Waitemata, 223 Auckland and 379 Counties Manukau children were admitted with a primary diagnosis of eczema or dermatitis. Admission rates per 100,000 population in the Waitemata and Auckland DHBs were significantly lower than the New Zealand rate, while in Northland and Counties Manukau, rates were not significantly different from the New Zealand rate (Table 67).

Northern DHBs Trends

During 2000–2012, admissions for infective dermatitis exhibited an upward trend in all four DHBs, although rates in Northland tapered off after 2008–2009. While trends for other eczema and dermatitis were more variable, overall admissions for eczema and dermatitis increased in all four DHBs during 2000–2012 (Figure 37).

Table 67. Hospital Admissions for Children Aged 0–14 Years with a Primary Diagnosis of Eczema or Dermatitis, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A*</td>
<td>B*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eczema and Dermatitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>116</td>
<td>116</td>
<td>165</td>
<td>0.28</td>
<td>95.37</td>
<td>1.15</td>
</tr>
<tr>
<td>Waitemata</td>
<td>239</td>
<td>242</td>
<td>303</td>
<td>0.25</td>
<td>54.44</td>
<td>0.66</td>
</tr>
<tr>
<td>Auckland</td>
<td>220</td>
<td>223</td>
<td>288</td>
<td>0.26</td>
<td>72.99</td>
<td>0.88</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>371</td>
<td>379</td>
<td>476</td>
<td>0.25</td>
<td>79.10</td>
<td>0.95</td>
</tr>
<tr>
<td>New Zealand</td>
<td>2,781</td>
<td>3,705</td>
<td>3,705</td>
<td>0.27</td>
<td>83.07</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children with eczema or dermatitis listed as a primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (i.e. DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (i.e. the sum of DHB totals exceeds New Zealand total); Rate Ratios are compared to NZ rates and have not been adjusted for population demographics.
Figure 37. Hospital Admissions for Children Aged 0–14 Years with a Primary Diagnosis of Eczema or Dermatitis, Northern DHBs vs. New Zealand 2000–2012

Admissions per 100,000 (0–14 Years)

Source: Numerator: National Minimum Dataset, hospital admissions for children with eczema or dermatitis listed as a primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Local Policy Documents and Evidence-Based Reviews Relevant to the Diagnosis or Management of Eczema or Dermatitis

In New Zealand there are no Ministry of Health policy documents specific to eczema and dermatitis, although a small number of other local publications are relevant to this topic. These are reviewed in Table 68, along with a range of evidence-based reviews which consider these issues in the overseas context.

Table 68. Local Policy Documents and Evidence-Based Reviews Relevant to Eczema and Dermatitis

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>There are currently no national guidelines in New Zealand for preventing or managing eczema and dermatitis.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>International Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>This guideline provides recommendations for the management of atopic eczema in children and adults in primary care, based on current evidence for best practice.</td>
</tr>
<tr>
<td>This clinical guideline concerns the management of atopic eczema in children from birth up to the age of 12 years. It has been developed with the aim of providing guidance on the diagnosis and assessment of the impact of the condition, the management during and between flares, and information and education to children and their families/caregivers about the condition.</td>
</tr>
<tr>
<td>This clinical guideline aims to provide evidence-based guidance for the diagnosis and treatment of patients with contact dermatitis. It covers both children and adults. These guidelines have been prepared for dermatologists on behalf of the British Association of Dermatologists. They present evidence-based guidance for investigation and treatment, with identification of the strength of evidence available at the time of preparation of the guidelines, including details of relevant epidemiological aspects, diagnosis and investigation.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cochrane Systematic Reviews</th>
</tr>
</thead>
<tbody>
<tr>
<td>This systematic review of 26 studies with 1229 subjects from randomised controlled trials published between 1980 (EMBASE) or 2000 (MEDLINE) to 2009 assessed if atopic eczema could be improved by antistaphylococcal agents. Studies were generally poor quality and short term. There was no significant difference in global outcome for clinically infected eczema when oral antibiotics were compared with placebo [one study, relative risk (RR) 0.40, 95% confidence interval (CI) 0.13-1.24] or when topical steroid antibiotic combinations were compared with steroid alone (two studies, RR 0.52, 95% CI 0.23-1.16). One study of children with infected eczema that added bleach to bathwater showed a significant improvement in eczema severity when compared with bathwater alone, although the difference could have been explained by regression to the mean. Although antistaphylococcal interventions reduced S. aureus numbers in people with clinically uninfected eczema, none of the studies showed any clinical benefit. They concluded that there was no evidence that commonly used antistaphylococcal interventions are clinically helpful in people with eczema that is not clinically infected.</td>
</tr>
<tr>
<td>This review assessed the effects of dietary exclusions for the treatment of established atopic eczema. The general quality of the studies was poor. There was some evidence from one study for the use of an egg-free diet in infants with a suspected egg allergy who have positive specific IgE antibodies to eggs. There was little evidence to support the use of various exclusion diets in unselected people with atopic eczema. Lack of benefit may be because people were not allergic to those substances but may also be because the studies were too small and poorly reported.</td>
</tr>
</tbody>
</table>
The following Cochrane Systematic Reviews showed no conclusive or only limited evidence and therefore are not currently recommended for the prevention or management of eczema and dermatitis in children. This may have been due to poorly conducted studies or no convincing results. In some reviews, further high quality research is recommended.

Osborn DA & Sinn KHJ. 2013. **Prebiotics in infants for prevention of allergy.** Cochrane Database of Systematic Reviews(3).

Gu S, et al. 2013. **Chinese herbal medicine for atopic eczema.** Cochrane Database of Systematic Reviews(9).

Bamford TMJ, et al. 2013. **Oral evening primrose oil and borage oil for eczema.** Cochrane Database of Systematic Reviews(6).

Apfelbacher CJ, et al. 2013. **Oral H1 antihistamines as monotherapy for eczema.** Cochrane Database of Systematic Reviews(5).

Kramer MS & Kakuma R. 2012. **Maternal dietary antigen avoidance during pregnancy or lactation, or both, for preventing or treating atopic disease in the child.** Cochrane Database of Systematic Reviews(9).


Other Systematic Reviews


A review of 24 systematic reviews of atopic eczema (AE) published between 1 August 2010 and 31 December 2011. An update of published summaries from previous years.

Epidemiological evidence points to the protective effects of early day-care, endotoxin exposure, consumption of unpasteurized milk, and early exposure to dogs, but antibiotic use in early life may increase the risk for AE.

Prevention of AE: there is currently no strong evidence of benefit for exclusive breastfeeding, hydrolysed protein formulas, soy formulas, maternal antigen avoidance, omega-3 or omega-6 fatty-acid supplementation, or use of prebiotics or probiotics.

Treatment of AE: the most compelling new systematic review evidence was for proactive treatment with topical anti-inflammatory agents (topical corticosteroids and topical calcineurin inhibitors) for the prevention of AE flares in patients with moderate to severe AE. A meta-analysis of 4 trials confirmed the superiority of tacrolimus 0.1% over pimecrolimus for the treatment of AE, and a review of 17 trials found that tacrolimus (0.1% or 0.03%) was broadly similar in efficacy to mild/moderate topical corticosteroids.

Evidence for the role of education in the management of AE was less conclusive, with evidence from randomized controlled trials showing mixed results. Further work is needed in this area to conduct high-quality trials of educational interventions that are clearly described and reproducible.

There is no clear evidence for the efficacy of homeopathy, botanical extracts or Chinese herbal medicine in the treatment of AE, as large well-designed trials are lacking in these areas.


A review of 19 RCTs with 905 participants on phototherapy for the management of atopic dermatitis. Formal meta-analysis was not feasible due to heterogeneity. Due to small study sizes and varying quality conclusions are cautious.

Phototherapy can be a valid therapeutic option for the management of atopic dermatitis with preference to UVA1 and UVB. Further well-designed, adequately powered RCTs are needed.


This meta-analysis of randomised controlled trials of 18 publications from 14 studies investigated whether probiotic use during pregnancy and early life decreases the incidence of atopic dermatitis and immunoglobulin E (IgE)-associated atopic dermatitis in infants and young children. The meta-analysis demonstrated that probiotic use decreased the incidence of atopic dermatitis (RR=0.79, 95% CI 0.71-0.88). Studies were fairly homogeneous (I² = 24%). The corresponding RR of IgE-associated atopic dermatitis was 0.80 (95% CI 0.66–0.96). No appreciable difference emerged across strata, nor was there evidence of publication bias. This provides evidence of a moderate role of probiotics in the prevention of atopic dermatitis and IgE-associated atopic dermatitis in infants. The favourable effect was similar regardless of the time of probiotic use (pregnancy or early life) and who received probiotics (mother, child or both).


Systematic review and meta-analysis of 8 randomised controlled trials with 385 subjects to assess the efficacy of allergen-specific immunotherapy (allergen-SIT) for patients with atopic dermatitis. They found that SIT has a significant positive effect on atopic dermatitis (odds ratio [OR], 5.35; 95% CI, 1.61–17.77; number needed to treat, 3; 95% CI, 2–9). SIT also showed significant efficacy in long-term treatment (OR, 6.42; 95% CI, 1.50–27.52) for patients with severe atopic dermatitis (OR, 3.13; 95% CI, 1.31-7.48), and when administered simultaneously (OR, 4.27; 95% CI, 1.36–13.39). This meta-analysis provides moderate evidence for the efficacy of SIT against atopic dermatitis but these findings are based on a small number of randomised controlled trials with considerable heterogeneity.

This article systematically reviewed the evidence from controlled clinical trials of any type of homeopathic treatment for any type of eczema. Only one randomised and two non-randomised trials were included. All were methodologically weak. The evidence from controlled trials did not demonstrate the efficacy of homeopathy for the treatment of eczema.


A meta-analysis of seven randomised, double-blind trials published between 2001 and 2009 was conducted for comparison of the development of atopic eczema in children whose mothers took probiotics during pregnancy v. placebo. The meta-analysis showed a significant risk reduction for atopic eczema in children aged 2–7 years by the administration of probiotics during pregnancy (reduction 5.7%; P=0.022). However, this effect was only significant for lactobacilli (reduction 10.6%; P=0.045), but not for a mixture of various bacterial strains as probiotics (difference 3.06%, P=0.204).

In conclusion, the meta-analysis shows that the administration of lactobacilli during pregnancy prevents atopic eczema in children aged 2–7 years. However, a mixture of various bacterial strains does not affect the development of atopic eczema, independent of whether they contain lactobacilli or not.


A review of epidemiologic studies identified through Ovid Medline (1950–2009) and Embase (1980–2009) investigating the effect of maternal fish consumption during perinatal life on atopic outcomes in infants or children. Five studies were found: three prospective cohort, one retrospective cohort, and one case control study with three studies reporting on eczema or atopic dermatitis. Protective effect of high fish consumption during pregnancy on atopic dermatitis at one to five years varied from 25% to 43%.

**van den Oord RA & Sheikh A. 2009.** *Filaggrin gene defects and risk of developing allergic sensitisation and allergic disorders: systematic review and meta-analysis.* BMI, 339, b2433.

A systematic review and meta-analysis to investigate whether filaggrin gene defects increase the risk of developing allergic sensitisation and allergic disorders. 24 genetic epidemiological studies (family, case-control) were included. The odds of developing atopic eczema was 1.99 (1.72–2.31) in the family studies and 4.78 (3.31–6.92) in the case-control studies. Filaggrin gene defects increase the risk of developing allergic sensitisation, atopic eczema, and allergic rhinitis. Evidence of the relation between filaggrin gene mutations and atopic eczema was strong, with people manifesting increased severity and persistence of disease. Filaggrin gene mutations also increased the risk of asthma in people with atopic eczema. Restoring skin barrier function in filaggrin deficient people in early life may help prevent the development of sensitisation and halt the development and progression of allergic disease.

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### Other Relevant Publications and Websites

- [DermNet NZ is provided by the New Zealand Dermatological Society Incorporated](http://dermnetnz.org/dermatitis/dermatitis.html) to present authoritative facts about the skin for consumers and health professionals in New Zealand and throughout the world. It was launched in March 1996. This site gives specific information about dermatitis and eczema.

- A clinical guideline for managing children with eczema which outlines the differential diagnosis, indications for admission, inpatient treatment, flow chart for inpatient care, and outpatient treatment.


- An article outlining the best practice management for eczema in the New Zealand setting.


The kidshealth website has been created by a partnership between the Paediatric Society of New Zealand (PSNZ) and the Starship Foundation.

PSNZ is a multi-disciplinary Society committed to improving the health of children and young people. With its membership of a broad range of child and youth health professionals, PSNZ is well-placed to develop content for the kidshealth website that is useful for parents and is up-to-date, accurate and valid. kidshealth information is for: New Zealand parents, caregivers, family and whānau and anyone else involved in caring for New Zealand children; and for the range of professionals in New Zealand who work with parents every day - doctors, nurses, early childhood staff, teachers, mental health professionals and others.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Inflammatory Bowel Disease

Introduction

Inflammatory bowel disease (IBD) refers to a group of inflammatory conditions affecting the colon and small intestine. The two main types of IBD are ulcerative colitis, which affects the large intestine, and Crohn’s disease, which may occur in any part of the gastrointestinal tract [68]. The peak age of onset is between 15 and 30 years [69]. Those first presenting with ulcerative colitis typically have obvious symptoms, such as bloody diarrhoea, which usually lead to rapid medical assessment. In contrast, the small intestine inflammation associated with Crohn’s disease may initially lead to more non-specific symptoms such as abdominal pain, nausea and weight loss [70].

While the precise cause of IBD is unknown, it is generally thought to arise from an inappropriate immune response to the commensal organisms living within the intestines, with or without an element of autoimmunity. While the intestines normally contain a large number of immune cells, these are normally restrained from mounting a full immune response to commensal organisms and dietary antigens by immune system regulatory pathways. During infections, activation of the gut-associated lymphoid tissues occurs but this is rapidly damped. In IBD, it is thought this process may be abnormally regulated [69].

In New Zealand, there has been a large rise in the incidence of IBD over the past 50 years [70]. Rates in the South Island are higher than in the North. One possible explanation is the different ethnic composition of the South Island population, and the fact that IBD is much higher in European, than in Māori or Pacific peoples [70]. Gender differences are also evident, with many studies finding that Crohn’s disease is more common in males than in females until around 16–18 years of age, after which time rates become higher in females. The reason for this switch is not understood, although hormonal factors have been implicated [70].

Some children with IBD may have more aggressive disease than adults, with children with Crohn’s having more frequent inflammation of the proximal small intestine, and a high proportion also having perianal disease. These factors may increase the risk of strictures and penetrating disease, thereby increasing the need for medication and surgery. Inflammatory bowel disease in children may have a negative impact on nutrition, so that weight loss is common at diagnosis and impaired height is a concern over time [70].

The following section reviews hospital admission for children and young people aged 0–24 years, with Crohn’s disease or ulcerative colitis listed in any of their first 15 diagnoses.

Data Source and Methods

Definition

Hospital admissions for children and young people aged 0–24 years with Crohn’s disease or ulcerative colitis listed in any of their first 15 diagnoses

Data Source

National Minimum Dataset

Numerator: Hospital admissions for children and young people aged 0–24 years with Crohn’s disease (ICD-10-AM K50) or ulcerative colitis (ICD-10-AM K51) listed in any of the first 15 diagnoses.

Denominator: Statistics New Zealand Estimated Resident Population (projected from 2007)

Notes on Interpretation

Note 1: This analysis focuses on hospital admissions for children and young people with Crohn’s disease or ulcerative colitis listed in any of the first 15 diagnoses, rather than on the subset of admissions where these diagnoses were the primary reason for admission. The rationale for looking beyond the primary diagnosis was the need to highlight the full spectrum of health issues experienced by children and young people with inflammatory bowel disease, and their consequent requirement for health services.

Note 2: If no mention of Crohn’s disease or ulcerative colitis was made in any of the first 15 diagnoses, these cases were not included, even if the patient had been assigned an inflammatory bowel disease-related code on a previous admission.
Table 69. Hospital Admissions for Children and Young People Aged 0–24 Years with Crohn’s Disease by Admission Type and Primary Diagnosis or Procedure, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis or Procedure</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Admissions in Category</th>
<th>% of Admissions in those with Crohn’s Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute and Arranged Admissions by Primary Diagnosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crohn’s Disease: Large Intestine</td>
<td>601</td>
<td>120.2</td>
<td>7.87</td>
<td>20.9</td>
<td>15.7</td>
</tr>
<tr>
<td>Crohn’s Disease: Small Intestine</td>
<td>314</td>
<td>62.8</td>
<td>4.11</td>
<td>10.9</td>
<td>8.2</td>
</tr>
<tr>
<td>Crohn’s Disease: Other</td>
<td>581</td>
<td>116.2</td>
<td>7.61</td>
<td>20.2</td>
<td>15.2</td>
</tr>
<tr>
<td>Crohn’s Disease: Unspecified</td>
<td>863</td>
<td>172.6</td>
<td>11.30</td>
<td>30.0</td>
<td>22.6</td>
</tr>
<tr>
<td>Anal/Rectal Abscess</td>
<td>58</td>
<td>11.6</td>
<td>0.76</td>
<td>2.0</td>
<td>1.5</td>
</tr>
<tr>
<td>Intestinal Obstruction</td>
<td>45</td>
<td>9.0</td>
<td>0.59</td>
<td>1.6</td>
<td>1.2</td>
</tr>
<tr>
<td>Anal/Rectal Fissure and Fistula</td>
<td>21</td>
<td>4.2</td>
<td>0.28</td>
<td>0.7</td>
<td>0.6</td>
</tr>
<tr>
<td>Other Diseases of the Digestive System</td>
<td>119</td>
<td>23.8</td>
<td>1.56</td>
<td>4.1</td>
<td>3.1</td>
</tr>
<tr>
<td>Iron Deficiency Anaemia</td>
<td>52</td>
<td>10.4</td>
<td>0.68</td>
<td>1.8</td>
<td>1.4</td>
</tr>
<tr>
<td>Abdominal/Pelvic Pain</td>
<td>25</td>
<td>5.0</td>
<td>0.33</td>
<td>0.9</td>
<td>0.7</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>198</td>
<td>39.6</td>
<td>2.59</td>
<td>6.9</td>
<td>5.2</td>
</tr>
<tr>
<td>Total Acute and Arranged Admissions</td>
<td>2,877</td>
<td>575.4</td>
<td>37.68</td>
<td>100.0</td>
<td>75.4</td>
</tr>
<tr>
<td>Waiting List Admissions by Primary Procedure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Injection or Infusion of Substance</td>
<td>361</td>
<td>72.2</td>
<td>4.73</td>
<td>38.4</td>
<td>9.5</td>
</tr>
<tr>
<td>Fibreoptic Colonoscopy +/- Biopsy</td>
<td>259</td>
<td>51.8</td>
<td>3.39</td>
<td>27.6</td>
<td>6.8</td>
</tr>
<tr>
<td>Panendoscopy +/- Biopsy</td>
<td>84</td>
<td>16.8</td>
<td>1.10</td>
<td>8.9</td>
<td>2.2</td>
</tr>
<tr>
<td>Resection of Small Intestine</td>
<td>52</td>
<td>10.4</td>
<td>0.68</td>
<td>5.5</td>
<td>1.4</td>
</tr>
<tr>
<td>Colectomy/Hemicolectomy</td>
<td>23</td>
<td>4.6</td>
<td>0.30</td>
<td>2.4</td>
<td>0.6</td>
</tr>
<tr>
<td>Procedures on Anal Fistula</td>
<td>15</td>
<td>3.0</td>
<td>0.20</td>
<td>1.6</td>
<td>0.4</td>
</tr>
<tr>
<td>Procedures Involving Ileostomy</td>
<td>14</td>
<td>2.8</td>
<td>0.18</td>
<td>1.5</td>
<td>0.4</td>
</tr>
<tr>
<td>Transfusion of Gamma Globulin</td>
<td>12</td>
<td>2.4</td>
<td>0.16</td>
<td>1.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Other Procedures</td>
<td>99</td>
<td>19.8</td>
<td>1.30</td>
<td>10.5</td>
<td>2.6</td>
</tr>
<tr>
<td>No Procedure Listed</td>
<td>21</td>
<td>4.2</td>
<td>0.28</td>
<td>2.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Total Waiting List Admissions</td>
<td>940</td>
<td>188.0</td>
<td>12.31</td>
<td>100.0</td>
<td>24.6</td>
</tr>
<tr>
<td>Total Crohn’s Disease Admissions</td>
<td>3,817</td>
<td>763.4</td>
<td>49.99</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with Crohn’s disease listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 70. Hospital Admissions in Children and Young People Aged 0–24 Years with Ulcerative Colitis by Admission Type and Primary Diagnosis or Procedure, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis or Procedure</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Admissions in Category</th>
<th>% of Admissions in those with Ulcerative Colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcerative Colitis</td>
<td>487</td>
<td>97.4</td>
<td>6.38</td>
<td>85.0</td>
<td>56.8</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>17</td>
<td>3.4</td>
<td>0.22</td>
<td>3.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Iron Deficiency Anaemia</td>
<td>10</td>
<td>2.0</td>
<td>0.13</td>
<td>1.7</td>
<td>1.2</td>
</tr>
<tr>
<td>Crohn’s Disease</td>
<td>9</td>
<td>1.8</td>
<td>0.12</td>
<td>1.6</td>
<td>1.1</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>50</td>
<td>10.0</td>
<td>0.65</td>
<td>8.7</td>
<td>5.8</td>
</tr>
<tr>
<td>Total Acute and Arranged Admissions</td>
<td>573</td>
<td>114.6</td>
<td>7.50</td>
<td>100.0</td>
<td>66.9</td>
</tr>
<tr>
<td>Fibreoptic Colonoscopy +/- Biopsy</td>
<td>173</td>
<td>34.6</td>
<td>2.27</td>
<td>60.9</td>
<td>20.2</td>
</tr>
<tr>
<td>Injection or Infusion of Substance</td>
<td>45</td>
<td>9.0</td>
<td>0.59</td>
<td>15.8</td>
<td>5.3</td>
</tr>
<tr>
<td>Panendoscopy +/- Biopsy</td>
<td>18</td>
<td>3.6</td>
<td>0.24</td>
<td>6.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Procedures Involving Ileostomy</td>
<td>13</td>
<td>2.6</td>
<td>0.17</td>
<td>4.6</td>
<td>1.5</td>
</tr>
<tr>
<td>Total Proctocolectomy</td>
<td>12</td>
<td>2.4</td>
<td>0.16</td>
<td>4.2</td>
<td>1.4</td>
</tr>
<tr>
<td>Colectomy/Hemicolectomy</td>
<td>7</td>
<td>1.4</td>
<td>0.09</td>
<td>2.5</td>
<td>0.8</td>
</tr>
<tr>
<td>Other Procedures</td>
<td>14</td>
<td>2.8</td>
<td>0.18</td>
<td>4.9</td>
<td>1.6</td>
</tr>
<tr>
<td>No Procedure Listed</td>
<td>2</td>
<td>0.4</td>
<td>0.03</td>
<td>0.7</td>
<td>0.2</td>
</tr>
<tr>
<td>Total Waiting List Admissions</td>
<td>284</td>
<td>56.8</td>
<td>3.72</td>
<td>100.0</td>
<td>33.1</td>
</tr>
<tr>
<td>Total Ulcerative Colitis Admissions</td>
<td>857</td>
<td>171.4</td>
<td>11.22</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with ulcerative colitis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
New Zealand Distribution and Trends

**Distribution by Primary Diagnosis and Procedure**

*Crohn’s Disease:* In New Zealand during 2008–2012, 82.0% of acute and arranged hospitalisations in children and young people with Crohn’s disease listed in any of their first 15 diagnoses, had Crohn’s disease listed as the primary reason for admission. The remaining 18.0% of admissions were for a range of conditions, the majority of which were related to Crohn’s disease, including anal/rectal abscesses, intestinal obstructions and anal/rectal fissures and fistulae (Table 69). Of those admitted from the waiting list with a diagnosis of Crohn’s disease, injections or infusions of therapeutic substances (38.4%) and fibreoptic colonoscopies +/- biopsies (27.6%) were the most frequent primary procedures listed (Table 69).

*Ulcerative Colitis:* During the same period, 85.0% of acute and arranged hospitalisations in children and young people with ulcerative colitis listed in any of their first 15 diagnoses, had ulcerative colitis listed as their primary reason for admission. The remaining 15.0% were for a range of conditions, including gastroenteritis and iron deficiency anaemia (Table 70). Of those admitted from the waiting list with a diagnosis of ulcerative colitis, fibreoptic colonoscopies +/- biopsies (60.9%) and injections or infusions of therapeutic substances (15.8%) were the most frequent primary procedures listed (Table 70).

**Distribution by Age**

In New Zealand during 2008–2012, hospital admission for Crohn’s disease and ulcerative colitis were infrequent during childhood, but increased during adolescence, with the highest rates being seen amongst those in their early twenties (Figure 38).

**Figure 38.** Hospital Admissions for Children and Young People with Crohn’s Disease or Ulcerative Colitis by Age, New Zealand 2008–2012
Distribution by Ethnicity and Gender

In New Zealand during 2008–2012, hospital admissions for those with Crohn’s disease were significantly higher for males, although no significant gender differences were evident for ulcerative colitis. Admission rates for Crohn’s disease were also significantly higher for European/Other > Asian/Indian > Māori > Pacific children and young people, while admissions for ulcerative colitis were significantly higher for European/Other > Asian/Indian > Māori and Pacific children and young people (Table 71).

Table 71. Hospital Admissions for Children and Young People Aged 0–24 Years with Crohn’s Disease or Ulcerative Colitis by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Crohn’s Disease</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>39.56</td>
<td>0.52</td>
<td>0.46–0.58</td>
<td>Female</td>
<td>47.92</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>76.72</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>51.95</td>
<td>1.08</td>
<td>1.02–1.16</td>
</tr>
<tr>
<td>Māori</td>
<td>7.54</td>
<td>0.10</td>
<td>0.08–0.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>1.28</td>
<td>0.02</td>
<td>0.01–0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ulcerative Colitis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>7.53</td>
<td>0.44</td>
<td>0.35–0.57</td>
<td>Female</td>
<td>11.66</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>17.00</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>10.80</td>
<td>0.93</td>
<td>0.81–1.06</td>
</tr>
<tr>
<td>Māori</td>
<td>1.71</td>
<td>0.10</td>
<td>0.07–0.15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>0.57</td>
<td>0.03</td>
<td>0.01–0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with Crohn’s disease or ulcerative colitis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population

Figure 39. Hospital Admissions for Children and Young People Aged 0–24 Years with Crohn’s Disease or Ulcerative Colitis by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with Crohn’s disease or ulcerative colitis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised
Trends Ethnicity
In New Zealand during 2000–2012, hospital admissions for those with Crohn’s disease were higher for European/Other > Asian/Indian > Māori > Pacific children and young people, while admissions for ulcerative colitis were higher for European/Other > Asian/Indian > Māori and Pacific children and young people. Admissions for both conditions increased in European/Other, Asian/Indian and Māori children and young people during this period. Trends for Pacific children and young people however, were more variable (Figure 39).

Northern DHBs Distribution and Trends

Northern DHBs Distribution
In the Northern region during 2008–2012, 24 Northland, 140 Waitemata, 84 Auckland and 98 Counties Manukau children and young people were admitted with a diagnosis of Crohn’s disease. Hospital admission rates per 100,000 in the Waitemata DHB were significantly higher than the New Zealand rate, while in Counties Manukau rates were significantly lower. In the Northland and Auckland DHBs rates were not significantly different from the New Zealand rate (Table 72).

Similarly during 2008–2012, 16 Northland, 81 Waitemata, 29 Auckland and 40 Counties Manukau children and young people were admitted with a diagnosis of ulcerative colitis. Hospital admission rates per 100,000 in the Waitemata DHB were significantly higher than the New Zealand rate, while in Counties Manukau rates were significantly lower. In the Northland and Auckland DHBs rates were not significantly different from the New Zealand rate (Table 72).

Northern DHBs Trends
During 2000–2012 hospital admissions for those with Crohn’s disease increased in all four DHBs, although in Counties Manukau rates tapered off after 2008–2009. Trends for ulcerative colitis however, were more variable (Figure 40).

Table 72. Hospital Admissions for Children and Young People 0–24 Years with Crohn’s Disease or Ulcerative Colitis, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A*</td>
<td>B*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crohn’s Disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>21</td>
<td>24</td>
<td>146</td>
<td>1.22</td>
<td>54.03</td>
<td>1.08</td>
</tr>
<tr>
<td>Waitemata</td>
<td>134</td>
<td>140</td>
<td>583</td>
<td>0.83</td>
<td>61.76</td>
<td>1.24</td>
</tr>
<tr>
<td>Auckland</td>
<td>72</td>
<td>84</td>
<td>381</td>
<td>0.91</td>
<td>50.87</td>
<td>1.02</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>93</td>
<td>98</td>
<td>296</td>
<td>0.60</td>
<td>29.96</td>
<td>0.60</td>
</tr>
<tr>
<td>New Zealand</td>
<td>937</td>
<td>3,817</td>
<td>81</td>
<td>0.81</td>
<td>49.99</td>
<td>1.00</td>
</tr>
<tr>
<td>Ulcerative Colitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>15</td>
<td>16</td>
<td>24</td>
<td>0.30</td>
<td>8.88</td>
<td>0.79</td>
</tr>
<tr>
<td>Waitemata</td>
<td>78</td>
<td>81</td>
<td>193</td>
<td>0.48</td>
<td>20.44</td>
<td>1.82</td>
</tr>
<tr>
<td>Auckland</td>
<td>27</td>
<td>29</td>
<td>72</td>
<td>0.50</td>
<td>9.61</td>
<td>0.86</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>37</td>
<td>40</td>
<td>62</td>
<td>0.31</td>
<td>6.28</td>
<td>0.56</td>
</tr>
<tr>
<td>New Zealand</td>
<td>396</td>
<td>857</td>
<td>857</td>
<td>0.43</td>
<td>11.22</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with Crohn’s disease or ulcerative colitis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (i.e. DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (i.e. the sum of DHB totals exceeds New Zealand total); Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics
Figure 40. Hospital Admissions for Children and Young People 0–24 Years with Crohn’s Disease or Ulcerative Colitis, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with Crohn’s Disease or Ulcerative Colitis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Local Policy Documents and Evidence-Based Reviews Relevant to Inflammatory Bowel Disease

In New Zealand there is a paucity of policy documents relevant to inflammatory bowel disease in children and young people. Table 73 reviews the available New Zealand publications, along with a range of guidelines and reviews which consider these issues in the overseas context.

Table 73. Local Policy Documents and Evidence-Based Reviews Relevant to Inflammatory Bowel Disease

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>This guideline covers colonoscopic surveillance for people at increased risk of developing colorectal cancer, specifically, people who have undergone previous colorectal cancer resection, people with inflammatory bowel disease (IBD) and people with adenomatous polyps. This guidance was an adaptation of sections of the National Institute for Health and Clinical Excellence (NICE) guideline <em>Colonoscopic Surveillance for Prevention of Colorectal Cancer in People with Ulcerative Colitis, Crohn’s Disease or Adenomas</em>, 2011.</td>
</tr>
<tr>
<td>The recommendations for people with IBD include: Surveillance colonoscopy should be offered following 8–10 years of clinical management in order to stratify risk. This differs from existing practice where patients with IBD are risk stratified at the onset of inflammatory bowel symptoms.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>International Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>This NICE Guideline offers best practice advice on the care of adults (aged &gt; 18 years), children (aged &lt; 11 years) and young people (aged 12 to 17 years) with Crohn’s disease. Its major recommendations focus on:</td>
</tr>
<tr>
<td>- Patient education and support</td>
</tr>
<tr>
<td>- Inducing remission in Crohn's Disease, with a range of criteria being given for the use of monotherapy with glucocorticosteroids, enteral nutrition, add on treatments such as azathioprine or mercaptopurine and the use of Infliximab and Adalimumab</td>
</tr>
<tr>
<td>- Maintaining remission in Crohn's Disease</td>
</tr>
<tr>
<td>- Surgery, including where Crohn's disease is limited to the distal ileum and the management of strictures</td>
</tr>
<tr>
<td>Ulcerative colitis is a lifelong disease arising from an interaction between genetic and environmental factors, observed predominantly in the developed countries of the world. The precise aetiology is unknown and therefore medical therapy to cure the disease is not yet available. Within Europe there is a North–South gradient, but the incidence appears to have increased in Southern and Eastern countries in recent years. Despite randomised trials there will always be many questions that can only be answered by the exercise of judgement and opinion. This leads to differences in practice between clinicians, which may be brought into sharp relief by differences in emphasis between countries.</td>
</tr>
<tr>
<td>This document updates the previous European Consensus on the diagnosis and management of UC, and was finalised by the European Crohn's and Colitis Organisation (ECCO) at a meeting held in Dublin in February 2011. ECCO is a forum for specialists in inflammatory bowel disease from 31 European countries.</td>
</tr>
</tbody>
</table>
When deciding the appropriate treatment strategy for active ulcerative colitis one should consider the activity, distribution (proctitis, left-sided, extensive), and pattern of disease. The disease pattern includes relapse frequency, course of disease, response to previous medications, side-effect profile of medication and extra-intestinal manifestations. The age at onset and disease duration may also be important factors. Severe ulcerative colitis necessitating hospital admission needs to be distinguished from those with mild or moderately active disease who can generally be managed as outpatients. The simplest, best validated and most widely used index for identifying severe UC remains that of Truelove and Witts: any patient who has a bloody stool frequency ≥ 6/day and a tachycardia (> 90 bpm), or temperature > 37.8 °C, or anaemia (haemoglobin < 10.5 g/dL), or an elevated ESR (> 30 mm/h) has severe ulcerative colitis (Table 1.3). Only one additional criterion in addition to the bloody stool frequency ≥ 6/day is needed to define a severe attack.

It should be standard practice to confirm the presence of active colitis by sigmoidoscopy before starting treatment. Flexible sigmoidoscopy and biopsy may exclude unexpected causes of symptoms that mimic active disease such as cytomegalovirus colitis, rectal mucosal prolapse, Crohn's disease, malignancy, or even irritable bowel syndrome and haemorrhoidal bleeding. In addition, all patients with active disease require stool cultures with *Clostridium difficile* toxin assay to exclude enteric infection. Patients with an appropriate travel history should also have stool microscopy to exclude parasitic infections such as amoebiasis.

In addition, detailed treatment guidelines are outlined.


Detailed guidelines on the diagnosis and management of ulcerative colitis in special situations are given. These special situations include: anaemia; pouchitis; colorectal cancer surveillance; psychosomatic; and extra-intestinal manifestations.


Detailed guidelines on the diagnosis and management of Crohn’s disease in further special situations are given. These special situations include: Crohn’s disease in children and adolescents; Crohn’s disease and pregnancy; Crohn’s disease and psychosocial factors; Extra-intestinal manifestations of Crohn’s disease; and alternative therapies.


Pediatric ulcerative colitis (UC) shares many features with adult-onset disease but there are some unique considerations; therefore, therapeutic approaches have to be adapted to these particular needs. This document aims to formulate guidelines for managing UC in children based on a systematic review of the literature and a robust consensus process. It is a product of a joint effort by a group of 27 experts in pediatric IBD of the European Crohn's and Colitis Organization (ECCO) and the European Society for Paediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN).

A total of 40 formal recommendations and 68 practice points were endorsed on how to monitor disease activity, the role of endoscopic evaluation, medical and surgical therapy, timing and choice of each medication, the role of combined therapy, and when to stop medications. A management flowchart, based on the Pediatric Ulcerative Colitis Activity Index (PUCAI), is presented.


These Guidelines aim to offer best practice advice on the use of colonoscopic surveillance in adults with inflammatory bowel disease (IBD), which covers ulcerative colitis and Crohn's disease or adenomas. Recommendations include:

- Offer colonoscopic surveillance to people with IBD whose symptoms started 10 years ago and who have: Ulcerative colitis (but not proctitis alone) or Crohn's colitis involving more than one segment of colon
- Offer a baseline colonoscopy with chromoscopy and targeted biopsy of any abnormal areas to people with IBD who are being considered for colonoscopic surveillance to determine their risk of developing colorectal cancer.
- Offer colonoscopic surveillance to people with IBD as defined in the recommendation above based on their risk of developing colorectal cancer determined at the last complete colonoscopy: Low risk: offer colonoscopy at 5 years; Intermediate risk: offer colonoscopy at 3 years; High risk: offer colonoscopy at 1 year.
- For people with IBD who have been offered colonoscopic surveillance, continue to use colonoscopy with chromoscopy as the method of surveillance.
- Offer a repeat colonoscopy with chromoscopy if any colonoscopy is incomplete. Consider whether a more experienced colonoscopist is needed.
These guidelines provide advice on the most appropriate initial radiologic examinations for patients (adults and children) with known or suspected Crohn’s disease (CD). Major recommendations include:

- Cross-sectional (CT and MR) enterography are the preferred imaging tests for the initial diagnosis and surveillance of patients with suspected and known Crohn’s disease.
- High-quality MR enterography provides the opportunity to eliminate radiation exposure for children and young adults while maintaining similar sensitivity to that of CT enterography. Institutional preference will be determined by availability, experience, and expertise.
- Barium studies (small-bowel series and barium enema) are being used less frequently in the imaging of Crohn’s disease but may be extremely helpful in demonstrating anatomy and strictures for preoperative planning purposes. Nuclear medicine techniques may be helpful in certain scenarios but are not widely used. Utilization will be determined by institutional preference.

The following Cochrane Reviews cover individual drug therapies in the management of inflammatory bowel disease:

- **Chande N, et al. 2013. Azathioprine or 6-mercaptopurine for induction of remission in Crohn’s disease.** Cochrane Database of Systematic Reviews(5).

The following other reviews cover individual drug therapies in the management of inflammatory bowel disease:


This review assessed the effects of psychological interventions (psychotherapy, patient education, relaxation techniques) on health-related quality of life, coping, emotional state and disease activity in inflammatory bowel disease (IBD). Twenty-one randomised, quasi-randomised and non-randomised controlled studies of psychological interventions in children or adults with IBD with a minimum follow up of 2 months were included with 1745 participants; 19 studies in adults and 2 in adolescents. There is no evidence for efficacy of psychological therapy in adult patients with IBD in general. In adolescents, psychological interventions may be beneficial with positive short term effects of psychotherapy on most outcomes assessed including quality of life (2 studies, 71 patients, SMD 0.70; 95% CI 0.21 to 1.18) and depression (1 study, 41 patients, SMD -0.62; 95% CI -1.25 to 0.01), but the evidence is limited.

This systematic review examined inflammatory bowel disease guidelines for quality of evidence, methods of grading evidence and conflicts of interest (COI). Nineteen IBD guidelines published by the American College of Gastroenterology, American Gastroenterological Association, British Society of Gastroenterology, Canadian Association of Gastroenterology, Crohn's and Collitis Foundation of America and European Crohn's and Collitis Organisation as of 27 September 2012 were reviewed. Nearly half the IBD guideline recommendations are based on expert opinion or no evidence. The majority of the guidelines fail to disclose any COI, and when commenting, all have numerous COI. Furthermore, the guidelines are not updated frequently and there is a lack of consensus between societal guidelines. This study highlights the critical need to centralize and redesign the guidelines development process.


This review evaluated the diagnosis and management of moderate to severe Crohn disease, with a focus on newer treatments and goals of care. MEDLINE was searched from 2000 to 2011. Additional citations were procured from references of select research and review articles. Evidence was graded using the American Heart Association level-of-evidence guidelines. Although mesalamines are still often used to treat Crohn disease, the evidence for their efficacy is lacking. Corticosteroids can be effectively used to induce remission in moderate to severe Crohn disease, but they do not maintain remission. The mainstays of treatment currently include particularly anti-tumor necrosis factor. Immunomodulators and biologics are now the preferred treatment options for Crohn disease.


Whereas endoscopy is a well-established and uniformly performed diagnostic examination, the implementation of radiologic techniques for the assessment of inflammatory bowel disease is still heterogeneous; variations in technical aspects and the degrees of experience and preferences exist across countries in Europe. ECCO and ESGAR scientific societies jointly elaborated a consensus to establish standards for imaging in IBD using magnetic resonance imaging, computed tomography, ultrasonography, and including also other radiologic procedures such as conventional radiology or nuclear medicine examinations for different clinical situations that include general principles, upper Gl tract, colon and rectum, perineum, liver and biliary tract, emergency situation, and the postoperative setting. The statements and general recommendations of this consensus are based on the highest level of evidence available, but significant gaps remain in certain areas such as the comparison of diagnostic accuracy between different techniques, the value for therapeutic monitoring, and the prognostic implications of particular findings.


This systematic review and meta-analysis was conducted to establish the cumulative risk of surgery among patients with inflammatory bowel disease (IBD) and evaluated how this risk has changed over time. The analysis included population-based studies published as articles (n = 26) and abstracts (n = 4) that reported risks of surgery at 1, 5, or 10 years after a diagnosis of Crohn's disease and/or ulcerative colitis. The trend in risk of surgery over time was analysed by meta-regression using mixed-effect models. Based on all population-based studies, the risk of surgery 1, 5, and 10 years after diagnosis of Crohn's disease was 16.3% (95% confidence interval [CI], 11.4%–23.2%), 33.3% (95% CI, 26.3%–42.1%), and 46.6% (95% CI, 37.7%–57.7%), respectively. On all population-based studies, the risk of surgery 1, 5, and 10 years after diagnosis of ulcerative colitis was 4.9% (95% CI, 3.8%–6.3%), 11.6% (95% CI, 9.3%–14.4%), and 15.6% (95% CI, 12.5%–19.6%), respectively. The risk of surgery 1, 5, and 10 years after diagnosis of Crohn's disease and 1 and 10 years after diagnosis of ulcerative colitis has decreased significantly over the past 6 decades (P < 0.05).


Psychotherapy may be a useful intervention for inflammatory bowel disease (IBD) patients. This systematic review evaluated all randomized controlled trials that have been performed in psychotherapy for inflammatory bowel disease patients. Eighteen studies (19 papers) were included. Psychotherapy was found to have minimal effect on measures of anxiety, depression, QOL and disease progression although shows promise in reducing pain, fatigue, relapse rate and hospitalisation, and improving medication adherence. It may also be cost effective. The effects of psychotherapy on IBD is mixed: future studies should determine whether patient screening or measuring different dependent variables improves outcomes and whether particular psychotherapies are superior over others.


Fecal calprotectin (FC) is increasingly used during the diagnosis of inflammatory bowel disease, outperforming blood markers during investigation in children. Tests that reduce endoscopy rates in children with suspected gut inflammation would be beneficial. This systematic review and meta-analysis assessed the usefulness of FC in children undergoing their primary investigation for suspected IBD. Eight papers met the inclusion criteria (six prospective and two retrospective case-control studies). The 8 studies presented FC levels at presentation in 715 patients, 394 pediatric IBD patients, and 321 non-IBD controls. Pooled sensitivity and specificity for the diagnostic utility of FC during the investigation of suspected pediatric IBD were 0.978 (95% confidence interval [CI], 0.947–0.996) and 0.682 (95% CI, 0.502–0.863), respectively; the positive and negative likelihood ratios were 3.07 and 0.03. The review concluded that FC has a high sensitivity and a modest specificity for the diagnosis of suspected pediatric IBD. Further work is required to determine the effect of FC on endoscopy rates and its role during the re-evaluation of those with confirmed disease.

This review evaluates: key definitional, measurement, and conceptual challenges for understanding treatment regimen adherence in inflammatory bowel disease; published studies focused on interventions to enhance adherence in IBD; and synthesizes of practical adherence promotion strategies for use in IBD by health care providers. Strategies are distinguished by the level of evidence supporting their utility as well as by age group. Findings suggest that strategies including education, regimen simplification, and use of reminder systems and organizational strategies (e.g., pill boxes) are likely to be best suited for addressing accidental non-adherence. In contrast, addressing motivational issues, teaching problem-solving skills, and addressing problematic patterns of family functioning are more likely to benefit individuals displaying intentional non-adherence.


The traditional goals of Crohn’s disease (CD) therapy, to induce and maintain clinical remission, have not clearly changed its natural history. In contrast, emerging evidence suggests that achieving and maintaining mucosal healing may alter the natural history of CD, as it has been associated with more sustained clinical remission and reduced rates of hospitalization and surgical resection. Induction and maintenance of mucosal healing should therefore be a goal toward which therapy is now directed. This systematic review sought to: 1) explore the definition of mucosal healing; 2) review the relationship between clinical and endoscopic disease activity; 3) outline the impact of mucosal healing on short and long-term outcomes and the natural history of CD; 4) evaluate the efficacy of current therapeutics in inducing and maintaining mucosal healing; 5) review the practical issues and limitations of implementing mucosal healing strategies in clinical practice; 6) review the role of non-invasive markers of mucosal healing; and 7) propose an approach towards integrating mucosal healing as a therapeutic endpoint in clinical practice. Unresolved issues pertain to the benefit of achieving mucosal healing at different stages of the disease, the relationship between mucosal healing and transmural inflammation, the intensity of treatment needed to achieve mucosal healing when it has not been obtained using standard therapy, and the means by which mucosal healing is defined using current endoscopic disease activity indices. The main clinical challenge relates to defining the means of achieving high rates of mucosal healing in clinical practice.


This meta-analysis of controlled clinical trials was conducted to evaluate whether the use of antibacterial therapy improves the clinical symptoms of inflammatory bowel disease. Randomized, controlled trials in which antibiotic therapy was compared with placebo were investigated. A total of 10 randomized, placebo-controlled clinical trials for Crohn’s disease (CD) were included in the meta-analysis. The pooling of the data from these trials yielded an odds ratio (OR) of 1.35 [95% confidence interval (CI), 1.16–1.58] for antibiotic therapy compared with placebo in patients with CD. Furthermore, nine randomized placebo-controlled clinical trials for ulcerative colitis (UC) matched our criteria and were included in the analysis. The pooling of the data from these trials yielded an OR of 2.17 (95% CI, 1.54–3.05) in favour of antibiotic therapy. These results suggest that antibiotics improve clinical outcomes in patients with IBD.


This systematic review assessed the rationale for probiotics in inflammatory bowel disease and systematically reviewed clinical intervention studies with probiotics in the management of IBD in adults. The 41 clinical intervention studies were categorized on disease type (ulcerative colitis [UC] with/without an ileo-anal pouch and Crohn’s disease [CD]) and disease activity. Well-designed randomized controlled trials supporting the application of probiotics in the management of IBD are still limited. So far, no evidence is available to support the use of probiotics in CD. Further insight into the aetiology of IBD and the mechanisms of probiotic strains will aid in selecting probiotic strains for specific disease entities and disease locations.


This meta-analysis of prospective studies evaluated the predictive capacity of fecal calprotectin (FC) in inflammatory bowel disease (IBD) relapse. The capacity of FC to predict relapse was comparable between UC and CD. As a simple and non-invasive marker, FC is useful to predict relapse in quiescent IBD patients.


Recent data from the inflammatory bowel disease (IBD) literature support a need for transition clinics. The ideal model of a transition programme has not been established. Controlled trials are not available to measure the impact of a structured transition programme on clinically relevant endpoints such as disease control and hospital admissions. As local resources and availability of staffing and funding are highly variable, this review summarized some practical guidelines for the adult and paediatric gastroenterologist that can be used as an aid to help adolescents through the transition process even without the support of an established transition clinic.

A systematic review aimed to answer: What are the effects of medical treatments to induce remission in adults with Crohn's disease? What are the effects of surgical interventions to induce and maintain remission in adults with small-bowel and colonic Crohn's disease? What are the effects of medical interventions to maintain remission in adults with Crohn's disease; and to maintain remission following surgery? What are the effects of lifestyle interventions to maintain remission in adults with Crohn's disease? This review included 93 systematic reviews, RCTs, or observational studies that met the inclusion criteria. The systematic review presents information relating to the effectiveness and safety of the following interventions: aminosalicylates, antibiotics, azathioprine/mercaptopurine, ciclosporin, corticosteroids (oral), enteral nutrition, fish oil, infliximab, methotrexate, probiotics, resection, segmental colectomy, smoking cessation, and strictureplasty.

**Other Relevant Publications and Websites**

http://www.clinicalevidence.bmj.com/x/systematic-review/0416/guidelines.html

Guidelines on inflammatory bowel disease sourced from the National Guidelines Clearinghouse in the USA (a repository of guidelines from around the world), NICE in the UK, and other international government sources, professional medical organisations or medical specialty societies.

http://crohnsandcolitis.org.nz/

Crohn's and Colitis New Zealand is a charitable trust whose aims are to provide support, advice and information to interested individuals and people who have Crohn's disease or ulcerative colitis and their families and caregivers, and educational material to medical professionals and organisations within New Zealand. Information available includes a detailed web-based toilet map for New Zealand http://www.toiletmap.co.nz/

http://www.healthnavigator.org.nz/health-topics/crohns-disease/

The Health Navigator NZ website is a collaborative, non-profit initiative led by clinicians and consumers in response to the need for a central place to find reliable and trustworthy health information and self-help resources. This webpage gives an overview of Crohn's disease with links to various websites for both patients and health professionals.

http://www.nzsg.org.nz/cms2/research/ibd/

New Zealand Society of Gastroenterology. This webpage lists inflammatory bowel research that has been undertaken or is ongoing in New Zealand. It also lists units and contact details of the New Zealand IBD clinical trials network that have established facilities for conducting clinical trials in inflammatory bowel disease and are currently participating in trials of new treatments in New Zealand.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Cystic Fibrosis

Introduction

Cystic fibrosis (CF) is one of the most common genetic diseases in European populations. It results from mutations affecting a gene that controls a chloride channel in cell membranes called the cystic fibrosis transmembrane conductance regulator (CFTR). CFTR is essential for regulating salt and water movement across cell membranes [71].

Absent or reduced functioning of CFTR results in thickened secretions in a number of organs, including the lungs and digestive system. In the lungs, the airways become clogged with thick sticky mucus, which slows the clearance of bacteria, leading to frequent infections and scarring of the airways. In about 85% of cases, ducts in the pancreas also become blocked, leading to problems with digestion and intestinal malabsorption. In infants this may result in failure to thrive, while older children and young people may become malnourished. Other complications include male infertility; CF-related diabetes, which in older patients may require daily insulin injections; chronic liver disease and portal hypertension; joint problems; and psychological problems arising from having to cope with a severe long-term medical condition [71].

The outlook for those with CF has improved steadily over the past two decades, largely as a result of earlier diagnosis, more aggressive treatment, and the provision of care in specialised centres [72]. In the US, the median predicted survival for those with CF increased from 25 years in 1975, to 37 years in 2008, with the length of survival being directly correlated with the decade of birth [73]. Those born with CF today are expected to live into their sixth decade [72,73].

In New Zealand, babies are routinely screened for CF as part of the Newborn Metabolic Screening Programme [17]. Screening involves a heel prick blood spot test that measures immunoreactive trypsinogen (IRT). A very high IRT concentration suggests pancreatic injury consistent with, but not necessarily specific to, CF [72]. In older children and adults, the diagnosis is made when a clinical history suggestive of cystic fibrosis is accompanied by biochemical and genetic markers of CFTR dysfunction. This typically includes a sweat test, which looks for an elevated concentration of chloride in sweat [72].

In terms of management, people with CF benefit from treatment in specialised CF centres which have a dedicated multidisciplinary team and an emphasis on frequent visits, periodic testing and monitoring adherence to therapy [73]. Those centres which see patients more frequently, obtain more cultures, and use more oral and intravenous antibiotics, have been shown to achieve better lung function than centres with less aggressive approaches to care [72]. A range of other therapies are also available, and reviews of a number of these are summarised in the evidence-based review table at the end of this section.

The following section reviews hospitalisations for children and young people with any mention of CF in any of their first 15 diagnoses, as well as mortality for those with CF listed as the main underlying, or as a contributory cause of death.
Data Source and Methods

Definition
1. Hospital admissions for children and young people aged 0–24 years with cystic fibrosis listed in any of the first 15 diagnoses
2. Mortality in children and young people aged 0–24 years with cystic fibrosis listed as the main underlying cause of death, or as a contributory cause

Data Source
1. National Minimum Dataset
   Numerator: Hospital admissions for children and young people aged 0–24 years with cystic fibrosis (ICD-10-AM E84) listed in any of the first 15 diagnoses.
2. National Mortality Collection
   Numerator: Mortality in children and young people aged 0–24 years with cystic fibrosis (ICD-10-AM E84) listed as the main underlying cause of death, or as a contributory cause.

Notes on Interpretation
Note 1: This analysis focuses on hospital admissions and mortality for children and young people with cystic fibrosis listed in any of the first 15 diagnoses, or as the main underlying or a contributory cause of death. The rationale for looking beyond the primary diagnosis was the need to highlight the full spectrum of health issues experienced by those with cystic fibrosis, and their consequent requirement for acute health services. For example, during 2008–2012, while around 84% of hospitalisations for children and young people with cystic fibrosis had cystic fibrosis listed as the main reason for admission, a significant minority were admitted for infectious and respiratory diseases, digestive system problems, or other reasons. Further a review of the secondary diagnoses of those admitted with a primary diagnosis of cystic fibrosis found that a significant proportion were also for infections, respiratory, or digestive system complications.
Note 2: If no mention of cystic fibrosis was made in any of the first 15 diagnoses, these cases were not included, even if the patient had been assigned a diagnosis of cystic fibrosis on a previous admission.

New Zealand Distribution and Trends

Distribution by Primary and Secondary Diagnosis

Primary Diagnosis: In New Zealand during 2008–2012, 83.7% of hospitalisations in children and young people with cystic fibrosis listed in any of their first 15 diagnoses, had cystic fibrosis listed as the primary reason for admission. The remainder of admissions were for a variety of infectious and respiratory diseases, digestive system problems and other issues (Table 74).

Secondary Diagnosis: Of those children and young people with cystic fibrosis listed as the primary diagnosis, the majority (92.6%) also had a secondary diagnosis. Of these, unspecified acute lower respiratory tract infections were the most frequent secondary diagnosis listed, with a range of other infectious (e.g. pseudomonas, staphylococcus aureus, aspergillosis), respiratory (e.g. bronchiectasis, pneumonia) and other (e.g. diabetes and pancreatic problems) conditions also making a contribution (Table 75).

Distribution by Age
In New Zealand during 2008–2012, hospitalisations for children and young people with cystic fibrosis were relatively evenly distributed across the age range, although a small peak was evident during adolescence. Mortality however, was more common amongst those in their late teens and early twenties, with 20 young people having cystic fibrosis listed as the main underlying cause of death, or as a contributory cause, during 2006–2010 (Figure 41).
### Table 74. Hospital Admissions in Children and Young People Aged 0–24 Years with Cystic Fibrosis by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Admissions in those with Cystic Fibrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystic Fibrosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cystic Fibrosis with Pulmonary Manifestation</td>
<td>1,226</td>
<td>245.2</td>
<td>16.06</td>
<td>44.1</td>
</tr>
<tr>
<td>Cystic Fibrosis with Intestinal Manifestation</td>
<td>58</td>
<td>11.6</td>
<td>0.76</td>
<td>2.1</td>
</tr>
<tr>
<td>Cystic Fibrosis with Other Manifestation</td>
<td>878</td>
<td>175.6</td>
<td>11.50</td>
<td>31.6</td>
</tr>
<tr>
<td>Cystic Fibrosis Unspecified</td>
<td>164</td>
<td>32.8</td>
<td>2.15</td>
<td>5.9</td>
</tr>
<tr>
<td>Total Cystic Fibrosis-Related Diagnoses</td>
<td>2,326</td>
<td>465.2</td>
<td>30.46</td>
<td>83.7</td>
</tr>
<tr>
<td>Respiratory System Diseases</td>
<td>90</td>
<td>18.0</td>
<td>1.18</td>
<td>3.2</td>
</tr>
<tr>
<td>Factors Influencing Health Service Contact</td>
<td>86</td>
<td>17.2</td>
<td>1.13</td>
<td>3.1</td>
</tr>
<tr>
<td>Diseases Digestive System</td>
<td>57</td>
<td>11.4</td>
<td>0.75</td>
<td>2.1</td>
</tr>
<tr>
<td>Complications Surgical Medical Care</td>
<td>47</td>
<td>9.4</td>
<td>0.62</td>
<td>1.7</td>
</tr>
<tr>
<td>Infectious and Parasitic Diseases</td>
<td>37</td>
<td>7.4</td>
<td>0.48</td>
<td>1.3</td>
</tr>
<tr>
<td>All Other Diagnoses</td>
<td>137</td>
<td>27.4</td>
<td>1.79</td>
<td>4.9</td>
</tr>
<tr>
<td>Total Other Diagnoses</td>
<td>454</td>
<td>90.8</td>
<td>5.95</td>
<td>16.3</td>
</tr>
<tr>
<td>Total</td>
<td>2,780</td>
<td>556.0</td>
<td>36.41</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with cystic fibrosis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
### Table 75. Secondary Diagnosis in Children and Young People Aged 0–24 Years Hospitalised with Cystic Fibrosis as a Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Secondary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>% of Admissions with Cystic Fibrosis as a Primary Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Lower Respiratory Infection Unspecified</td>
<td>588</td>
<td>117.6</td>
<td>25.3</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>274</td>
<td>54.8</td>
<td>11.8</td>
</tr>
<tr>
<td>Influenza and Pneumonia</td>
<td>56</td>
<td>11.2</td>
<td>2.4</td>
</tr>
<tr>
<td>Acute Upper Respiratory Infections</td>
<td>54</td>
<td>10.8</td>
<td>2.3</td>
</tr>
<tr>
<td>Other Respiratory System Diseases</td>
<td>123</td>
<td>24.6</td>
<td>5.3</td>
</tr>
<tr>
<td>Pseudomonas Infection</td>
<td>207</td>
<td>41.4</td>
<td>8.9</td>
</tr>
<tr>
<td>Aspergillosis</td>
<td>133</td>
<td>26.6</td>
<td>5.7</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em> Infection</td>
<td>112</td>
<td>22.4</td>
<td>4.8</td>
</tr>
<tr>
<td>Other Infectious and Parasitic Diseases</td>
<td>113</td>
<td>22.6</td>
<td>4.9</td>
</tr>
<tr>
<td>Specific Diseases of Pancreas</td>
<td>117</td>
<td>23.4</td>
<td>5.0</td>
</tr>
<tr>
<td>Other Diseases Digestive System</td>
<td>81</td>
<td>16.2</td>
<td>3.5</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>68</td>
<td>13.6</td>
<td>2.9</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>227</td>
<td>45.4</td>
<td>9.8</td>
</tr>
<tr>
<td>No Secondary Diagnosis</td>
<td>173</td>
<td>34.6</td>
<td>7.4</td>
</tr>
<tr>
<td>Total</td>
<td>2,326</td>
<td>465.2</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by secondary diagnosis for children and young people with cystic fibrosis listed as their primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Distribution by Ethnicity and Gender

In New Zealand during 2008–2012, hospital admissions for children and young people with cystic fibrosis were significantly higher for females than for males. Admission rates were also significantly higher for European/Other > Māori > Pacific and Asian/Indian children and young people (Table 76). Similar ethnic differences were seen during 2000–2012 (Figure 42).

Table 76. Hospital Admissions for Children and Young People Aged 0–24 Years with Cystic Fibrosis by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cystic Fibrosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>2.25</td>
<td>0.04</td>
<td>0.03–0.06</td>
<td>Female</td>
<td>39.02</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>54.88</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>33.92</td>
<td>0.87</td>
<td>0.81–0.94</td>
</tr>
<tr>
<td>Māori</td>
<td>21.13</td>
<td>0.39</td>
<td>0.35–0.43</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>3.12</td>
<td>0.06</td>
<td>0.04–0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cystic fibrosis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population
Northern DHBs Distribution and Trends

Northern DHBs Distribution

In the Northern region during 2008–2012, a total of 8 Northland, 34 Waitemata, 39 Auckland and 27 Counties Manukau children and young people were hospitalised with a diagnosis of cystic fibrosis. Admission rates per 100,000 in the Auckland, Waitemata and Counties Manukau DHBs were significantly lower than the New Zealand rate while in Northland rates were not significantly different from the New Zealand rate (Table 77).

Northern DHBs Trends

In the Northern region during 2000–2012, while individual DHB’s trends varied, admission rates for those with cystic fibrosis in the Waitemata, Auckland and Counties Manukau DHBs were consistently lower than the New Zealand rate. In Northland, large declines during the mid-2000s saw rates become lower than the New Zealand rate during 2010–2012 (Figure 43).
Table 77. Hospital Admissions for Children and Young People Aged 0–24 Years with Cystic Fibrosis, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A*</td>
<td>B*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>8</td>
<td>8</td>
<td>82</td>
<td>2.05</td>
<td>30.35</td>
<td>0.83</td>
</tr>
<tr>
<td>Waitemata</td>
<td>28</td>
<td>34</td>
<td>175</td>
<td>1.03</td>
<td>18.54</td>
<td>0.51</td>
</tr>
<tr>
<td>Auckland</td>
<td>34</td>
<td>39</td>
<td>232</td>
<td>1.19</td>
<td>30.98</td>
<td>0.85</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>22</td>
<td>27</td>
<td>109</td>
<td>0.81</td>
<td>11.03</td>
<td>0.30</td>
</tr>
<tr>
<td>New Zealand</td>
<td>351</td>
<td>2,780</td>
<td>1.58</td>
<td>36.41</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cystic fibrosis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (i.e. DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (i.e. the sum of DHB totals exceeds New Zealand total); Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics.

### Local Policy Documents and Evidence-Based Reviews Relevant to Cystic Fibrosis

In New Zealand there are a small number of publications of relevance to cystic fibrosis. **Table 78** briefly summarises these, along with a number of guidelines and evidence-based reviews which consider these issues in the overseas context.
Figure 43. Hospital Admissions for Children and Young People Aged 0–24 Years with Cystic Fibrosis, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cystic fibrosis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 78. Local Policy Documents and Evidence-Based Reviews Relevant to Cystic Fibrosis

<table>
<thead>
<tr>
<th>New Zealand Guidelines and Useful Web Sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>This document sets out minimum standards of care for people with Cystic Fibrosis (PWCF) in New Zealand regardless of where they live. It recognises that New Zealand does not have the population to support the types of specialist CF centres found overseas. It recommends that care be provided at a clinic at a hospital near the home of the PWCF with additional at least annual reviews provided by a regional CF centre. It details the requirements for local and regional CF clinics in terms of facilities, services and staffing. It highlights the need for communication and cooperation between PWCF, their local clinic and the regional CF centre for this model of shared care to work effectively.</td>
</tr>
<tr>
<td>The website of the Cystic Fibrosis Association Of New Zealand has a number of useful publications aimed at parents, patients and teachers, which are available for download here: [online]. <a href="http://www.cfnz.org.nz/our-services/library/downloads/">http://www.cfnz.org.nz/our-services/library/downloads/</a> . They are arranged under the following headings: Ages &amp; Stages, Exercise, Infection Control, New Diagnosis, Nutrition, Travel, Treatments &amp; CF-related disorders, Care in New Zealand, CFANZ Annual Reports, and Other.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>International Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>These U.K. guidelines are intended for clinicians and other allied health professionals, service commissioners and providers, parents and carers of children with CF, older children and adults with CF, and their families. They are not an evidence-based guideline but rather a consensus document setting out best practice for cystic fibrosis care. They state that all patients with CF should be under the direct supervision of a Specialist CF Centre serving a minimum of 100 patients but that some child patients can receive some of their care from a Network CF Clinic which has clear lines of communication with a Specialist CF Centre at all levels. The guidelines set standards of care for: clinical care by specialist multidisciplinary teams; measures to prevent cross-infection from other patients; monitoring of lung function, microbial surveillance and treatment of airway infections; chest physiotherapy; nutritional support; identifying and managing other CF manifestations and complications; psychosocial support; transition to adult care; and transplantation, palliative care and end-of-life care. The U.K. Cystic Fibrosis Trust has also produced guidelines on pharmacy standards in CF care, physiotherapy, laboratory standards for processing microbiological samples, antibiotic treatment, mexitilin resistant Staphylococcus aureus, bone mineralisation, Pseudomonas infection, the Burkholderia cepacia complex, CF-related diabetes, nutritional management of CF, and nursing management of CF. These can be found here: [online]. <a href="https://www.cysticfibrosis.org.uk/about-cf/publications/consensus-documents.aspx">https://www.cysticfibrosis.org.uk/about-cf/publications/consensus-documents.aspx</a></td>
</tr>
<tr>
<td>These guidelines are the first such guidelines to be published in Australia. The ten chapters cover facilities and staffing, services, newly diagnosed children, newly diagnosed adolescents and adults, outpatient care, inpatient care, home therapy, transition care, outreach services and care, transplantation and end of life care and the role of the CF organisations. Each chapter includes a literature review and provides guidelines for clinical care as well as specifying the requirements for facilities, staffing and services. Where evidence from the literature is referred to it is graded according to the National Health and Medical Research Council guidelines.</td>
</tr>
</tbody>
</table>

| Guidelines from the American Cystic Fibrosis Foundation [online]. http://wwwcff.org/treatments/CFCareGuidelines/ |
| These guidelines are produced under the direction of The Cystic Fibrosis Foundation Guidelines Steering Committee the members of which represent various stakeholders including the different health disciplines providing care for people with CF as well as members of the CF community. The recommendations in the guidelines are informed by the systematic reviews performed by investigators at The Johns Hopkins University who perform explicit assessment of evidence and grade it according to the grading system developed by the U.S. Preventive Services Task Force. |
Guidelines from the American Cystic Fibrosis Foundation http://www.cff.org/treatments/CFCareGuidelines/


This concise (20 page) publication aims to provide a consensus on standards of care for CF patients in Europe. It is the result of the 2004 European Consensus Conference organized by the European Cystic Fibrosis Society which took place in Artimino in Italy, and involved 36 experts in Cystic Fibrosis. It details the necessary infrastructure for a CF centre (serving a minimum of 50 patients), the minimum standards for routine evaluation and assessment of patients, the management of complications and the documentation of results in a standard database. The appendix covers a series of 35 “important questions” the answers to which include a grading of the evidence on which they are based. A table explains the grading system used.
The European Cystic Fibrosis Society has produced the following guidelines and reports on issues related to optimising patient care and CF team work, which can be downloaded here: [https://www.ecfs.eu/ecfs_guidelines](https://www.ecfs.eu/ecfs_guidelines).


Also on the same site are guidelines relating to the coordination of clinical research, travelling with CF, neonatal screening for CF, management of pregnancy in women with CF and issues related to small and medium sized enterprises and the development of new therapies for CF.

The European Cystic Fibrosis Society (which publishes the Journal of Cystic Fibrosis) has also published a number of consensus documents which have been developed at meetings of leading workers (typically with about 30 participants) in the particular topics. These publications do not discuss the details of research studies but all the information and recommendations contained in them are very well referenced. These publications can be downloaded here: [https://www.ecfs.eu/publications/consensus_reports](https://www.ecfs.eu/publications/consensus_reports).


Cochrane Reviews with Good Evidence for Clinical Decision-Making

Warnock L, Gates A, van der Schans CP. 2013. Chest physiotherapy compared to no chest physiotherapy for cystic fibrosis. Cochrane Database of Systematic Reviews(9)


Cystic Fibrosis - 218
### Vaccines for preventing infection with Pseudomonas aeruginosa in cystic fibrosis

Almost all patients with CF eventually become colonised with *Pseudomonas aeruginosa*. This bacterium produces a biofilm which surrounds the bacterial colonies within the lungs and protects them from host defence mechanisms and antibiotics. The constant frustrated efforts of the immune system to clear the large bacterial colonies results in leakage of toxic enzymes from neutrophils which cause further lung damage. Because it is almost impossible to eradicate *P. aeruginosa* infection once it becomes established an effective vaccine would be very beneficial. This review included three RCTs of vaccines aimed at reducing infection with *P. aeruginosa*, with 483, 476 and 37 patients respectively. No data was published from one of the large trials and the company involved has stated that the trial failed to confirm results from an earlier study and that it had suspended further clinical development of the vaccine. The other large trial reported a relative risk (RR) of chronic infection of 0.91 (95% confidence interval 0.55 to 1.49). The small trial also reported a RR close to one. In the large trial one patient died in the observation period (from acute lymphatic leukaemia, considered to be unrelated to the vaccine), and there were four severe adverse events registered in the vaccine group compared to one in the control group. The authors concluded that the use of vaccines against *P. aeruginosa* cannot be recommended.

### Recombinant growth hormone therapy for cystic fibrosis in children and young adults

Children and young people with CF often have malnutrition and delayed growth which is not sufficiently improved with nutritional supplementation. This review aimed to evaluate the effectiveness and safety of recombinant human growth hormone therapy in improving lung function, quality of life and clinical status of children and young adults with CF. It included four RCTs (161 participants in total). The authors concluded that recombinant growth hormone therapy, compared to no treatment, is modestly effective in improving height, weight, lean tissue mass and functional vital capacity (one measure of lung function), but not quality of life or overall clinical status.

### Oral non-steroidal anti-inflammatory drug therapy for lung disease in cystic fibrosis

This review included five RCTs (334 participants aged five to 39 years; maximum follow up of four years). Three of these, all comparing ibuprofen to placebo, were deemed to be of good or adequate methodological quality. Based on combined data from the two largest ibuprofen trials, the authors concluded that high-dose ibuprofen can slow the progression of lung disease in people with CF, particularly in children.

### Non-invasive ventilation for cystic fibrosis

This review included seven RCTs with 106 participants in total. The authors concluded that non-invasive ventilation via face mask may be useful as an adjunct to other airway clearance techniques, especially in people with CF who have difficulty expectorating sputum and that non-invasive ventilation, used in addition to oxygen, may improve gas exchange during sleep to a greater degree than oxygen therapy alone in those with moderate to severe disease. They noted that these beneficial effects have largely been demonstrated in single treatment sessions with small numbers of participants. They stated that the impact of this therapy on pulmonary exacerbations and disease progression remains uncertain and that further research involving adequately powered long term RCTs is needed.

### Nebuliser systems for drug delivery in cystic fibrosis

There are many different types of nebuliser systems that are used to deliver medications to people with CF. This review aimed to evaluate these systems in regard to their effectiveness, safety, burden of treatment and patient adherence to nebulised therapy. This review included 20 studied which were RCTs or quasi-RCTs (1936 participants in total). The authors stated that there is variability in performance between the different nebuliser systems and that newer technologies such as adaptive aerosol delivery and vibrating mesh technology are superior to conventional systems in terms of treatment time, deposition as a percentage of prescribed dose, patient preference and adherence. They also stated that long-term RCTs of these technologies are needed to determine patient-relevant outcomes (such as quality of life and burden of care), safe and effective dosing levels of medications, clinical outcomes (such as hospitalisations and need for antibiotics) and an economic evaluation of their use.

### Macrolide antibiotics for cystic fibrosis

This review included ten studies (959 participants). Five studies with a low risk of bias examined six months' treatment with azithromycin vs. placebo and demonstrated consistent improvement in FEV<sub>1</sub> over six months (mean difference at six months 3.97% (95% confidence interval 1.74% to 6.19%; n = 549 from four studies). Patients treated with six months’ azithromycin were about twice as likely to be free of pulmonary exacerbation at six months: odds ratio 1.96 (95% confidence interval 1.15 to 3.33). They also had had a greater weight gain and a reduced need for oral antibiotics but those who followed a once-weekly high dose regimen had more frequent gastrointestinal adverse events. Azithromycin treatment was associated with reduced identification of *Staphylococcus aureus* on respiratory culture but also a significant increase in macrolide resistance. The authors concluded that: there was evidence of improved respiratory function after six months azithromycin therapy; beyond six months the benefits were less clear although the reduction in pulmonary exacerbations persisted; azithromycin therapy appeared safe although the emergence of macrolide resistance was a concern. They stated that a multi-centre long term trial of azithromycin treatment is needed.

Some centres give children with CF prophylactic antibiotics from the time of diagnosis, usually an antibiotic which is active against Staphylococcus aureus, such as flucl ox acillin. There are concerns that such long-term prophylaxis might lead to the emergence of antibiotic resistance and increased likelihood of colonisation with Pseudomonas aeruginosa. There are also short-term adverse effects such as diarrhoea or oral candidiasis (thrush). This review included four RCTs with a total of 401 participants aged zero to seven years at study enrolment. The authors noted that overall the studies were of poor quality. They concluded that anti-staphylococcal antibiotic prophylaxis, when commenced in infancy and continued up to six years of age, leads to fewer children having isolated of S. aureus but that the clinical importance of this finding is uncertain since there was no difference between the prophylaxis and the no-prophylaxis groups in infant or conventional lung function measures, nutrition, hospital admissions, additional courses of antibiotics or adverse effects. There were also no differences between groups in the number of isolates of P. aeruginosa, though there was a trend towards a lower cumulative isolation rate of P. aeruginosa in the prophylaxis group at two and three years and towards a higher rate from four to six years (although this last finding was based on data from only one study since none of the other studies had more than three years of follow up). Since all the reviewed studies lasted for six years or less, no conclusions could be drawn about the long-term effects of prophylaxis.


From the results of three trials (131 participants in total) lasting one month or more, the authors of this review concluded that "oral calorie supplements do not confer any additional benefit in the nutritional management of moderately malnourished children with CF over and above the use of dietary advice and monitoring alone. While nutritional supplements may be used, they should not be regarded as essential."

Ng MS, Francini AJ. 2012. Drug therapies for reducing gastric acidity in people with cystic fibrosis. Cochrane Database of Systematic Reviews.(4).

Gastric acid-reducing agents have been used as an adjunct to pancreatic enzyme therapy to improve fat absorption and gastro-intestinal symptoms in people with CF. This review included 16 RCTs or quasi-RCTs with a total of 256 participants. One trial found that these therapies improved gastro-intestinal symptoms such as abdominal pain, seven trials reported significant improvement in measures of fat malabsorption and two trials reported no significant improvement in nutritional status. Only one trial reported on measures of respiratory function and one trial reported an adverse effect (of misoprostol). The Cochrane reviewers did not identify any trials assessing the effectiveness of gastric acid-reducing agents for improving quality of life or survival or reducing complications of increased gastric acidity (e.g. heartburn or gastric ulcers). They concluded that there is limited evidence that these agents are associated with improvements in gastrointestinal symptoms and fat absorption but insufficient evidence to determine whether they are associated with improvement in nutritional status, lung function, quality of life, or survival.


The defective gene causing CF normally codes for a protein called the cystic fibrosis transmembrane conductance regulator (CFTR) which is responsible for the movement of salt across cell membranes. CFTR is a chloride channel but it also plays apart in reducing the activity of the epithelial sodium channel (ENaC). In CF the absence of CFTR leads to increased activity of ENaC leading to excessive absorption of sodium and water which leads to dehydration of the airway surface liquid resulting in sticky mucus that is difficult to clear and a lung environment that is very susceptible to infection. This review aimed to determine whether topical administration of drugs that block sodium transport improve lung function in people with CF. The authors identified five RCTs (with a total of 226 participants) of amiloride (a short-acting sodium channel blocker) vs. placebo. A meta-analysis of results from three six-month studies indicated a mean difference between the intervention and placebo groups in relative change in forced vital capacity over six months which was significant and favoured the placebo group (weighted mean difference −1.51%; 95% confidence interval −2.77 to −0.25), although there was significant heterogeneity between the studies. One two-week study showed that hypertonic saline with amiloride pre-treatment did not result in a significant improvement in respiratory function or mucus clearance, compared to hypertonic saline with placebo pre-treatment. There were no significant differences identified in other clinically relevant outcomes. The authors concluded that there was no evidence that topical administration of short-acting sodium channel blockers improves respiratory function in people with CF and some limited evidence that they may cause deterioration in lung function, especially when delivered before hypertonic saline.


This review aimed to determine whether a prescribed regimen of physical training produces improvement or prevents deterioration in physiological and clinical outcomes in CF compared to no training. The authors identified seven RCTs or quasi-RCTs meeting their inclusion criteria (with a total of 231 participants). They stated that there was some limited evidence from both short and long term studies that aerobic or anaerobic physical training has a beneficial effect on primary outcomes (exercise capacity, strength and lung function) although improvements are not consistent between studies. They reported that the studies in the review were mostly of small size and limited duration with incomplete reporting which limited the conclusions that could be drawn from them. They noted that most people with CF are already offered physical training as part of their care package and there is a lack of evidence to discourage this. They stated that further research is needed to assess the benefits of exercise programmes comprehensively and to determine the relative benefits of aerobic exercise, anaerobic exercise or a combination of both for people with CF.
This review aimed to compare long intravenous lines (percutaneous lines) with short intravenous lines with regard to lifespan of the line, ease of insertion, complication rates of the line and patient satisfaction in people with CF receiving intravenous antibiotics. Two randomised studies with a total of 67 participants were included in the review and both were considered to have potential for bias in several domains. One study with 20 participants found that long lines lasted longer than short lines and were preferred by participants. The other study with 47 participants found no difference in line lifespan or participant preference when comparing two different long intravenous lines (the Hydrocath and Vygon EC). Neither study was adequately powered to detect differences in rates of serious line complications. The authors concluded that there was some evidence that long line are superior to short lines in terms of line lifespan and patient satisfaction but no evidence that any one type of long line is better than any other.

### Antioxidant Micronutrients for Lung Disease in Cystic Fibrosis


Persistent airway infection in people with CF leads to progressive lung damage which is due, in part, to oxidative stress stemming from both the infectious agent and the body’s inflammatory immune response. It is thought that supplementation with the exogenous anti-oxidant micronutrients, vitamin E, vitamin C, β-carotene and selenium, also known as free-radical scavengers, may be helpful in maintaining the oxidant-antioxidant balance in people with CF. This review aimed to synthesise the existing knowledge about the effect of vitamin C, vitamin E, β-carotene and selenium in CF lung disease. The authors identified four RCTs and one quasi-RCT which compared vitamin C, vitamin E, β-carotene and selenium (individually or in combination) to placebo or standard care but only three trials (87 participants) had data suitable for analysis. Data from two trials indicated anti-oxidant supplementation did not produce improvement in lung function. One trial reported significant improvement in quality of life favouring control, mean difference −0.06 points on the quality of well-being scale (95% confidence interval −0.12 to −0.01). Based on two trials, selenium-dependent glutathione peroxidase enzyme levels significantly improved in favour of both combined supplementation, mean difference 1.60 units per gram of haemoglobin (95% CI 0.30 to 2.90) and selenium supplementation, mean difference 10.20 units per gram of haemoglobin (95% CI 2.22 to 18.18). Levels of all plasma antioxidants, except vitamin C, significantly improved with supplementation. The authors concluded that the evidence regarding the clinical effectiveness of anti-oxidant supplementation in CF was insufficient to be conflicting. Limited data from few trials indicates that antioxidants appear to decrease both quality of life and oxidative stress. They stated that further RCTs examining clinically important outcomes and elucidation of a clear biological pathway of oxidative stress in CF are necessary before any firm conclusions can be drawn regarding effects of antioxidant supplementation.

### Inhaled Bronchodilators for Cystic Fibrosis


Inhaled bronchodilators are commonly used by people with CF to treat wheeze and breathlessness. This review aimed to evaluate the effectiveness of these agents in people with CF. There were eighteen RCTs or quasi-RCTs, with 369 participants in total, meeting the review inclusion criteria. All but two used a cross-over design. Meta-analysis of study results was not possible. The trials were heterogeneous with varied conclusions. In three out of five trials, in the short term, compared to placebo, long-acting beta-2 agonists increased FEV₁ and FEF25%–75% in participants known to have bronchodilator responsiveness, but they produced inconsistent results in long-term trials. Four trials assessed ipratropium, a short-acting anticholinergic, vs. placebo. Results from these trials indicated no consistent effects on lung function tests in either the short or the long term. There were no trials of fenoterol, formoterol or tiotropium. The authors concluded that it was not possible to fully determine the effectiveness of inhaled bronchodilators in CF since a meta-analysis could not be done but that short and long-acting beta-2 agonists can be beneficial both in the short and the long term in individuals with demonstrable bronchodilator responsiveness or bronchial hyper-responsiveness. They found no evidence for the use of fenoterol, formoterol or tiotropium and so they stated that the use of these agents in people with CF cannot be supported.
Ryan G, Singh M, Dwan K. 2011. Inhaled antibiotics for long-term therapy in cystic fibrosis. Cochrane Database of Systematic Reviews(3). This review aimed to examine the evidence that inhaled antibiotics reduce the frequency of infectious exacerbations and improve lung function, quality of life and survival for people with CF. It also aimed to examine the adverse effects of such treatment. The review included nineteen RCTs or quasi-RCTs, with a total of 1724 participants, which compared an antibiotic with placebo or usual treatment over a period of between one and 32 months. Due to variability in study designs and the reporting of results, meta-analysis was not possible. Eight trials evaluated tobramycin and the results of these indicated that lung function (as measured by FEV₁) was higher and exacerbations of lung infection (according to various different measures) were fewer in the group treated with antibiotics. There was a greater increase in resistance to antibiotics in the antibiotic group than the placebo group. No trial subjects were found to have auditory or renal impairment but tinnitus, voice alteration, hemoptysis and cough were more frequent with tobramycin than placebo. One trial, with 115 participants, compared tobramycin with colistin and found that after one month the mean difference in FEV₁ was 6.33 (95% confidence interval -0.04 to 12.70) in favour of tobramycin. The authors concluded that inhaled antibiotic treatment probably reduces the rate of exacerbations and improves lung function but it was not possible to calculate a pooled estimate of the level of benefit. They stated that tobramycin is the agent with the best evidence of effectiveness and that more research is needed to determine whether the benefit of inhaled antibiotics is maintained in the longer term and the significance of the emergence of antibiotic-resistant organisms.

Langton Hewer SC, Smyth AR. 2010. Antibiotic strategies for eradicating Pseudomonas aeruginosa in people with cystic fibrosis. Cochrane Database of Systematic Reviews(11). Most people with CF eventually acquire background respiratory tract infection with Pseudomonas aeruginosa. Once chronic infection with this organism becomes established it is almost impossible to eradicate and it is associated with increased morbidity and mortality. This review aimed to determine whether antibiotic treatment of early P. aeruginosa infection in children and adults with CF eradicates the organism, improves clinical and microbiological outcomes and is superior to or more cost-effective than other strategies. Four RCTs (95 participants in total) met the review’s inclusion criteria and the authors identified another two on-going trials. Evidence from two trials (of low methodological quality) showed that treatment of early P. aeruginosa infection with inhaled tobramycin results in microbiological eradication of the organism from respiratory secretions more often than placebo, OR 0.15 (95% CI 0.03 to 0.65) and that this effect may persist for up to 12 months. The authors concluded that nebulised antibiotics, either alone or in combination with oral antibiotics, were better than no treatment for early infection with P. aeruginosa and that eradication may be sustained in the short term. They stated that overall, their review found insufficient evidence to indicate which antibiotic strategy should be used for the eradication of early P. aeruginosa infection in people with CF.

Jones AP, Wallis C. 2010. Dornase alfa for cystic fibrosis. Cochrane Database of Systematic Reviews(3). Dornase alfa (Pulmozyme®) is a highly purified solution of recombinant human deoxyribonuclease (rhDNase). Inhaled via a nebuliser it reduces viscosity in the lungs, promoting improved clearance of secretions. It is an expensive therapy, costing around £7000 (NZ$13500) per patient per year in the U.K. (in 2009). This review aimed to determine whether the use of dornase alfa in CF is associated with improved morbidity and mortality compared to placebo or other mucolytics and to identify any adverse events associated with its use. The authors found 15 trials (2469 participants) meeting their inclusion criteria. Twelve studies compared dornase alfa to placebo or no dornase alfa treatment; one compared daily dornase alfa with hypertonic saline and alternate day dornase alfa; and two compared daily dornase alfa to hypertonic saline. Study lengths ranged from six days to three years. There were no significant differences in mortality between treatment groups and the authors noted that this was not surprising given that most trials were short term. Lung function (as measured by FEV₁ and FVC) improved in the treatment groups and there were significant differences between the intervention and control groups in percentage changes from baseline in lung function measures at one month (data from four trials), three months, six months and two years; there was a non-significant difference at three years (data from one trial only for each of these periods). There was considerable heterogeneity between these trials. The only adverse effects reported more frequently in the intervention group (in one trial only) were voice alteration and rash. There was insufficient data for the reviewers to analyse differences in antibiotic use, days of inpatient treatment or quality of life. The authors concluded that there is evidence that dornase alfa over a one-month period is associated with improved lung function in people with CF and they noted that one trial lasting six months also found the same effect. They stated that one trial indicated that two years of therapy significantly improved FEV₁ in children and non-significantly reduced the risk of infective exacerbations, and that voice alteration and rash appeared to be the only adverse effects reported with increased frequency in intervention groups in the RCTs.

Wark P, McDonald VM. 2010. Nebulised hypertonic saline for cystic fibrosis. Cochrane Database of Systematic Reviews(6). Nebulised hypertonic saline (HS) is a salt water solution with a salt concentration of 3% or more inhaled as a fine mist through a mask or mouthpiece. This review included controlled trials comparing HS to placebo or other mucolytic therapy, for any duration or dose regimen in people with CF (of any age or disease severity). There were 12 such trials (442 participants aged 6 to 46 years) identified by the authors. Based on the results of these trials the authors concluded that treatment with 7% HS for 48 weeks was associated with a small improvement in FEV₁ at four weeks but that this was not sustained at 48 weeks (based on the primary outcome measure of the only long-term trial). They stated that although HS (unlike RhDNase) does not improve long term lung function it does improve quality of life and reduce pulmonary exacerbations and it appears to be inexpensive and safe and not to increase risk of infection.
This review considered whether early detection of Cystic Fibrosis via newborn screening results in improved clinical outcomes (by preventing or reducing irreparable organ damage), and greater quality of life and survival. The authors found two suitable randomised controlled trials and analysed the data from one of them (the 1998 Wisconsin trial). In this trial 650,341 neonates were screened for CF and the results of screening were withheld from half the families and investigators until the children were 4 years old to provide the control group (unless the parents requested the results). There were benefits for the screened group in improved growth and nutrition but the effect of screening on long-term pulmonary function was confounded by other factors. Screening was found to be cheaper than traditional diagnosis.

Cochrane reviews which found that there were some RCTs related to the topic of the review but these did not provide clear guidance for the use of the therapy in question

The following topics relating to the treatment of cystic fibrosis have been the subject of Cochrane reviews which found that, although there were some adequate quality RCTs of the intervention in question, these did not provide conclusive guidance regarding the use of the intervention (topics are followed by the year of the review): oxygen therapy (2013), nebulized and oral thiol derivatives (2013), comparison of long-acting insulins, short-acting insulins and oral hypoglycemic agents for CF-related diabetes (2013), comparison of antibiotic therapy based on conventional antimicrobial susceptibility testing to antibiotic therapy based on combination antimicrobial susceptibility testing in the treatment of acute pulmonary exacerbations in people with CF and chronic infection with P. aeruginosa (2013), timing of dornase alfa inhalation (2013), palivizumab for prophylaxis against respiratory syncytial virus infection 2013), antibiotic adjuvant therapy for pulmonary infection (2013), vitamin K supplementation (2013), topical nasal steroids for treating nasal polyposis (2013), inhaled antibiotics for pulmonary exacerbations (2013), active cycle of breathing technique (2012), ursodeoxycholic acid for CF-related liver disease (2013), standard versus biofilm antimicrobial susceptibility testing to guide antibiotic therapy (2012), inhaled corticosteroids (2012), conventional chest physiotherapy compared to other airway clearance techniques (2013), topical cystic fibrosis transmembrane conductance regulator gene replacement for CF-related lung disease (2012), single vs. combination intravenous antibiotic therapy (2012), home vs. hospital intravenous antibiotic therapy (2012), elective vs. symptomatic intravenous antibiotic therapy (2012), vitamin D supplementation (2012), omega-3 fatty acids (2011), inspiratory muscle training (2011), self-management education (2011), vaccines for preventing influenza (2011), oscillating devices for airway clearance (2011), oral anti-pseudomonal antibiotics (2010), psychological interventions for people with CF and their families (2009), positive expiratory pressure physiotherapy for airway clearance (2009), oral protein calorie supplementation for children with chronic disease (2009).

Cochrane reviews which found that there were no RCTs of adequate quality related to the topic of the review

The following topics relating to the treatment of cystic fibrosis have been the subject of Cochrane reviews which found that there was no high quality evidence which could be used to determine the efficacy or otherwise of the intervention (topics are followed by the year of the review): Anti-IgE therapy for allergic bronchopulmonary aspergillosis in people with CF (2013), duration of intravenous antibiotic therapy (2013), interventions for the eradication of methicillin-resistant Staphylococcus aureus (MRSA) (2013), enteral tube feeding (2012), chemical pleurodesis versus surgical intervention for persistent and recurrent pneumothoraces (2013), antibiotic treatment for nontuberculous mycobacteria lung infection (2012), antibiotic treatment for Burkholderia cepacia complex in people with CF experiencing a pulmonary exacerbation (2012), pneumococcal vaccination in adults or children with CF(2012), vitamin A supplementation (2012), disease modifying anti-rheumatic drugs in people with CF-related arthritis (2012), totally implantable vascular access devices (2012), antifungal therapies for allergic bronchopulmonary aspergillosis (2012), antibiotic treatment for Stenotrophomonas maltophilia (2012), timing of hypertonic saline inhalation (2012), anti-inflammatory drugs and analgesics for managing symptoms in people with CF-related arthritis(2012), neuraminidase inhibitors for the treatment of influenza infection in people with CF (2011), singing for children and adults with CF (2010).

Other Reviews

Towns SJ, Bell SC. 2011. Transition of adolescents with cystic fibrosis from paediatric to adult care. The clinical respiratory journal, 5(2), 64-75

Improvements in the treatment of CF over recent decades have led to increasing numbers of young people with CF requiring care from the adult health system. This review article by two Australian authors provides a good overview of the issues involved in the transition of adolescents with CF from paediatric to adult care. Important factors in successful transition are: early planning and preparation, facilitating self-management skills, having a coordinated approach including young people with CF, their families and the paediatric and adult teams, detailed communication with provision of a written referral report and documentation of prior CF complications, feedback between the paediatric and adult healthcare teams, and on-going audit of the transition process.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
**TYPE 1 DIABETES**

**Introduction**

Type 1 diabetes is the most common type of diabetes in children and young people. The majority of cases are thought to arise from an environmentally triggered autoimmune destruction of the beta cells of the pancreas, set against a background of genetic risk. However, it is thought that, in a small number of cases, beta cell failure may occur for other reasons [74]. The incidence of type 1 diabetes increases from birth, peaks at around 10–14 years of age, and then declines after puberty. The initial onset is typically acute, with symptoms of thirst, frequent urination and weight loss. Beta cell destruction is typically progressive, leading to an increasing and on-going need for exogenous insulin [74].

Internationally the incidence of type 1 diabetes has been increasing [74]. Local research suggests that similar trends are occurring in New Zealand. In one recent Auckland review [75], the incidence of type 1 diabetes in children aged 0–14 years, rose from per 10.9 per 100,000 in 1990, to 22.5 per 100,000 in 2009, with the most rapid rises occurring amongst older children (aged 10–14 years) [75]. A similar study, which reviewed type 1 diabetes in Canterbury young people aged 15–24 years, found that the prevalence had increased by 45 per 100,000 (12%) between 2003 and 2010. While this increase was not statistically significant, the authors noted that the absolute increase in the number of young people with type 1 diabetes had significant implications for health service demand [76].

In the same Auckland review [75] the incidence of type 1 diabetes was higher for European children and young people than for those from other ethnic groups. However another Auckland study during 1995–2005 [77] found that Māori and Pacific children and young people with type 1 diabetes had poorer metabolic control (as measured by HbA(1c)) than European children and young people, as well as higher rates of hypoglycaemia. Low socioeconomic status was also independently associated with poor glycaemic control [77].

Increases in the incidence and prevalence of type 1 diabetes have significant implications for service delivery, as optimal long term outcomes require intensive management by the patient, their family and their health care team [78]. In addition, there are also implications for schools. With estimates that 1 in 500 school children have type 1 diabetes, most secondary schools likely to have at least one diabetic child [78]. In the longer term, these increases may also lead to higher rates of microvascular (e.g. retinopathy and nephropathy) and macrovascular disease (e.g. coronary heart disease, stroke and peripheral vascular disease) as the current generation of children and young people with type 1 diabetes reaches adulthood [79].

The following section reviews hospital admissions for children and young people aged 0–24 years with any mention of type 1 diabetes in any of the first 15 diagnoses, as well as mortality for children and young people where type 1 diabetes was listed as the main underlying, or as a contributory cause of death.

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**Data Source and Methods**

**Definition**

1. Hospital admissions for children and young people aged 0–24 years with type 1 diabetes listed in any of the first 15 diagnoses

2. Mortality in children and young people aged 0–24 years with type 1 diabetes listed as the main underlying cause of death or as a contributory cause

**Data Source**

1. National Minimum Dataset

Numerator: Hospital admissions for children and young people aged 0–24 years with type 1 diabetes (ICD-10-AM E10) listed in any of the first 15 diagnoses.
2. National Mortality Collection

Numerator: Mortality in children and young people aged 0–24 years with type 1 diabetes (ICD-10-AM E10) listed as the main underlying cause of death, or as a contributory cause.


Notes on Interpretation

Note 1: This analysis focuses on hospital admissions and mortality for children and young people with type 1 diabetes listed in any of the first 15 diagnoses, or as the main underlying, or a contributory cause of death. The rationale for looking beyond the primary diagnosis was the need to highlight the full spectrum of health issues experienced by children and young people with type 1 diabetes, and their requirements for health services.

For example, during 2008–2012, while 70% of hospitalisations in children and young people with type 1 diabetes were for diabetes-related diagnoses, 30% were for other conditions. Some of these may have been more likely as a result of diabetes (e.g. some types of infection), or because their management may have been more complex in diabetic patients (e.g. acute gastroenteritis). However, the number of admissions in diabetic patients which were unrelated to their diabetes (e.g. viral infections), may slightly overinflate the impact diabetes has on acute service demand.

Note 2: The terminology used to describe diabetic complications differs to that used previously due to changes in the way ICD-10-AM Version 6 deals with coma and ketoacidosis. Previous ICD-10-AM versions included two sub-categories: diabetes with coma and diabetes with ketoacidosis without coma. In ICD-10-AM Version 6 ketoacidosis and lactic acidosis are grouped together, with additional digit extensions being used to identify the presence or absence of coma. Thus earlier reports grouped admissions into type 1 diabetes with coma and type 1diabetes with ketoacidosis, whereas in this report, these have been combined into the category Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma.

Note 3: The admission rates presented here may differ from those presented previously due to a change between ICD-10-AM Version 3 and 6 which tightened up the way diabetes was assigned as an additional diagnosis. With the introduction of Version 6 in July 2008, new criteria were introduced for coding diabetes as a secondary diagnosis in the presence of another condition e.g. cystic fibrosis. While the impacts were greatest for type 2 diabetes, it is likely that these changes were responsible for some of the drop in admissions for type 1 diabetes which occurred in 2008–09, immediately after the introduction of ICD-10-AM V6.

Note 4: If no mention of type 1 diabetes was made in any of the first 15 diagnoses, these cases were not included, even if the patient had been assigned a diabetes-related code on a previous admission.

New Zealand Distribution and Trends

Distribution by Primary Diagnosis

Primary Diagnosis: In New Zealand during 2008–2012, 70.0% of hospital admissions for children and young people with type 1 diabetes listed in any of their first 15 diagnoses had a diabetes-related primary diagnosis, with ketoacidosis/lactic acidosis +/- coma accounting for 33.3% and type 1 diabetes without complications for 17.7% of admissions. A further 30.0% of hospitalisations were for diagnoses other than diabetes, with gastroenteritis, injuries and poisoning, pregnancy and childbirth, and respiratory diseases being the most common reasons for non-diabetes related admissions (Table 79).

Distribution by Age

In New Zealand during 2008–2012, hospitalisations for children and young people with type 1 diabetes increased during childhood, reached a peak at 14 years of age, and then fluctuated. Mortality was highest amongst those in their late teens and early twenties, with 15 young people having type 1 diabetes listed as the main underlying cause of death, or as a contributory cause, during 2006–2010. However, none of these deaths occurred in children aged less than 13 years (Figure 44).
Table 79. Hospital Admissions in Children and Young People Aged 0–24 Years with Type 1 Diabetes by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Type 1 Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type 1 Diabetes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis other than Type 1 Diabetes*</td>
<td>2,490</td>
<td>498.0</td>
<td>32.61</td>
<td>30.0</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma</td>
<td>2,769</td>
<td>553.8</td>
<td>36.26</td>
<td>33.3</td>
</tr>
<tr>
<td>Type 1 Diabetes without Complications</td>
<td>1,470</td>
<td>294.0</td>
<td>19.25</td>
<td>17.7</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ophthalmic Complications</td>
<td>87</td>
<td>17.4</td>
<td>1.14</td>
<td>1.0</td>
</tr>
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<td>Type 1 Diabetes with Neurological Complications</td>
<td>41</td>
<td>8.2</td>
<td>0.54</td>
<td>0.5</td>
</tr>
<tr>
<td>Type 1 Diabetes with Renal Complications</td>
<td>22</td>
<td>4.4</td>
<td>0.29</td>
<td>0.3</td>
</tr>
<tr>
<td>Type 1 Diabetes with Multiple Complications</td>
<td>13</td>
<td>2.6</td>
<td>0.17</td>
<td>0.2</td>
</tr>
<tr>
<td>Type 1 Diabetes with Unspecified Complications</td>
<td>5</td>
<td>1.0</td>
<td>0.07</td>
<td>0.1</td>
</tr>
<tr>
<td>Type 1 Diabetes with Other Specified Complications</td>
<td>1,412</td>
<td>282.4</td>
<td>18.49</td>
<td>17.0</td>
</tr>
<tr>
<td>New Zealand Total</td>
<td>8,309</td>
<td>1,661.8</td>
<td>108.81</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Other Diagnoses</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>441</td>
<td>88.2</td>
<td>5.78</td>
<td>5.3</td>
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<tr>
<td>Injury and Poisoning</td>
<td>243</td>
<td>48.6</td>
<td>3.18</td>
<td>2.9</td>
</tr>
<tr>
<td>Pregnancy Childbirth Post-partum</td>
<td>229</td>
<td>45.8</td>
<td>3.00</td>
<td>2.8</td>
</tr>
<tr>
<td>Diseases of the Respiratory System</td>
<td>200</td>
<td>40.0</td>
<td>2.62</td>
<td>2.4</td>
</tr>
<tr>
<td>Skin Infections</td>
<td>133</td>
<td>26.6</td>
<td>1.74</td>
<td>1.6</td>
</tr>
<tr>
<td>Abdominal and Pelvic Pain</td>
<td>117</td>
<td>23.4</td>
<td>1.53</td>
<td>1.4</td>
</tr>
<tr>
<td>Viral Infection Unspecified Site</td>
<td>94</td>
<td>18.8</td>
<td>1.23</td>
<td>1.1</td>
</tr>
<tr>
<td>Complications Medical Surgical Care</td>
<td>53</td>
<td>10.6</td>
<td>0.69</td>
<td>0.6</td>
</tr>
<tr>
<td>Other Infectious Diseases</td>
<td>46</td>
<td>9.2</td>
<td>0.60</td>
<td>0.6</td>
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<tr>
<td>Cystic Fibrosis</td>
<td>32</td>
<td>6.4</td>
<td>0.42</td>
<td>0.4</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>902</td>
<td>180.4</td>
<td>11.81</td>
<td>10.9</td>
</tr>
<tr>
<td><strong>Total Diagnoses other than Type 1 Diabetes</strong></td>
<td>2,490</td>
<td>498.0</td>
<td>32.61</td>
<td>30.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Figure 44. Hospital Admissions (2008–2012) and Mortality (2006–2010) for New Zealand Children and Young People with Type 1 Diabetes by Age

Source: Numerator Admissions: National Minimum Dataset, hospital admissions for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Numerator Mortality: National Mortality Collection, deaths with type 1 diabetes listed as the main underlying or a contributory cause of death; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

**Distribution by Ethnicity and Gender**

In New Zealand during 2008–2012, hospital admissions for those with type 1 diabetes were significantly higher for females and for European/Other > Māori and Pacific > Asian/Indian children and young people (Table 80). Similar ethnic differences were seen during 2000–2012 (Figure 45).

Table 80. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 1 Diabetes by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian/Indian</td>
<td>19.44</td>
<td>0.14</td>
<td>0.12–0.16</td>
<td>Female</td>
<td>127.92</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>138.29</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>90.65</td>
<td>0.71</td>
<td>0.68–0.74</td>
</tr>
<tr>
<td>Māori</td>
<td>89.71</td>
<td>0.65</td>
<td>0.61–0.69</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>84.72</td>
<td>0.61</td>
<td>0.56–0.67</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population
Figure 45. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 1 Diabetes by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 1 diabetes in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Note: See Note 2 in Methods box regarding cautions in interpreting time series information as a result of a change in the way diabetes codes were assigned in 2008.

Northern DHBs Distribution and Trends

Northern DHBs Distribution
In the Northern region during 2008–2012, 95 Northland, 383 Waitemata, 252 Auckland and 319 Counties Manukau children and young people were hospitalised with a diagnosis of type 1 diabetes. Admission rates per 100,000 in the Northland, Auckland and Counties Manukau DHBs were significantly lower than the New Zealand rate, while rates in the Waitemata DHB were not significantly different from the New Zealand rate (Table 81).
Table 81. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 1 Diabetes, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>A* 89</td>
<td>B* 95</td>
<td>257</td>
<td>0.54</td>
<td>95.11</td>
<td>0.87</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waitemata</td>
<td>A* 363</td>
<td>B* 383</td>
<td>966</td>
<td>0.50</td>
<td>102.33</td>
<td>0.94</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland</td>
<td>A* 234</td>
<td>B* 252</td>
<td>558</td>
<td>0.44</td>
<td>74.50</td>
<td>0.68</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>A* 291</td>
<td>B* 319</td>
<td>744</td>
<td>0.47</td>
<td>75.30</td>
<td>0.69</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td>A* 2,916</td>
<td>B* 8,309</td>
<td>8,309</td>
<td>0.57</td>
<td>108.81</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (sum of DHB totals exceeds NZ total). Rate Ratios are compared to NZ rate and have not been adjusted for population demographics.

**Northern DHBs Trends**

During 2000–2012, admissions for those with type 1 diabetes increased in both the Waitemata and Auckland DHBs, although rates in both DHBs remained lower than the New Zealand rate throughout this period. Trends in Northland and Counties Manukau were more variable, although in Counties Manukau rates also remained lower than the New Zealand rate throughout this period (Figure 46).

**Distribution by Primary Diagnosis**

In the Northern region during 2008–2012, around two thirds (range 65.1%–66.7%) of hospital admissions in children and young people with type 1 diabetes listed in any of their first 15 diagnoses, had a diabetes-related primary diagnosis. Ketoacidosis/lactic acidosis +/- coma was the most frequent diabetes-related primary diagnosis in all four DHBs (Table 82, Table 83).
Figure 46. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 1 Diabetes, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: See Note 2 in Methods box regarding cautions in interpreting time series information as a result of a change in the way diabetes codes were assigned in 2008
Table 82. Hospital Admissions in Children and Young People Aged 0–24 Years with Type 1 Diabetes by Primary Diagnosis, Northland, Waitemata and Auckland DHBs 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>No. of Admissions: Total 2008–2012</th>
<th>No. of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Type 1 Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Northland</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis other than Type 1 Diabetes</td>
<td>88</td>
<td>17.6</td>
<td>32.57</td>
<td>34.2</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma</td>
<td>84</td>
<td>16.8</td>
<td>31.09</td>
<td>32.7</td>
</tr>
<tr>
<td>Type 1 Diabetes with Other Specified Complications</td>
<td>46</td>
<td>9.2</td>
<td>17.02</td>
<td>17.9</td>
</tr>
<tr>
<td>Type 1 Diabetes without Complications</td>
<td>37</td>
<td>7.4</td>
<td>13.69</td>
<td>14.4</td>
</tr>
<tr>
<td>Type 1 Diabetes with Unspecified Complications</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Northland Total</td>
<td>257</td>
<td>51.4</td>
<td>95.11</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Waitemata</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis other than Type 1 Diabetes</td>
<td>327</td>
<td>65.4</td>
<td>34.64</td>
<td>33.9</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma</td>
<td>287</td>
<td>57.4</td>
<td>30.40</td>
<td>29.7</td>
</tr>
<tr>
<td>Type 1 Diabetes without Complications</td>
<td>201</td>
<td>40.2</td>
<td>21.29</td>
<td>20.8</td>
</tr>
<tr>
<td>Type 1 Diabetes with Neurological Complications</td>
<td>7</td>
<td>1.4</td>
<td>0.74</td>
<td>0.7</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ophthalmic Complications</td>
<td>4</td>
<td>0.8</td>
<td>0.42</td>
<td>0.4</td>
</tr>
<tr>
<td>Type 1 Diabetes with Other Specified Complications</td>
<td>140</td>
<td>28.0</td>
<td>14.83</td>
<td>14.5</td>
</tr>
<tr>
<td>Waitemata Total</td>
<td>966</td>
<td>193.2</td>
<td>102.33</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis other than Type 1 Diabetes</td>
<td>186</td>
<td>37.2</td>
<td>24.83</td>
<td>33.3</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma</td>
<td>161</td>
<td>32.2</td>
<td>21.50</td>
<td>28.9</td>
</tr>
<tr>
<td>Type 1 Diabetes without Complications</td>
<td>105</td>
<td>21.0</td>
<td>14.02</td>
<td>18.8</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ophthalmic Complications</td>
<td>10</td>
<td>2.0</td>
<td>1.34</td>
<td>1.8</td>
</tr>
<tr>
<td>Type 1 Diabetes with Neurological Complications</td>
<td>3</td>
<td>0.6</td>
<td>0.40</td>
<td>0.5</td>
</tr>
<tr>
<td>Type 1 Diabetes with Multiple Complications</td>
<td>3</td>
<td>0.6</td>
<td>0.40</td>
<td>0.5</td>
</tr>
<tr>
<td>Type 1 Diabetes with Other Specified Complications</td>
<td>90</td>
<td>18.0</td>
<td>12.02</td>
<td>16.1</td>
</tr>
<tr>
<td>Auckland Total</td>
<td>558</td>
<td>111.6</td>
<td>74.50</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: s: suppressed due to small numbers
Table 83. Hospital Admissions in Children and Young People Aged 0–24 Years with Type 1 Diabetes by Primary Diagnosis, Counties Manukau DHB 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>No. of Admissions: Total 2008–2012</th>
<th>No. of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Type 1 Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis other than Type 1 Diabetes</td>
<td>260</td>
<td>52.0</td>
<td>26.32</td>
<td>34.9</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/− Coma</td>
<td>194</td>
<td>38.8</td>
<td>19.64</td>
<td>26.1</td>
</tr>
<tr>
<td>Type 1 Diabetes without Complications</td>
<td>134</td>
<td>26.8</td>
<td>13.56</td>
<td>18.0</td>
</tr>
<tr>
<td>Type 1 Diabetes with Ophthalmic Complications</td>
<td>5</td>
<td>1.0</td>
<td>0.51</td>
<td>0.7</td>
</tr>
<tr>
<td>Type 1 Diabetes with Other Specified Complications</td>
<td>151</td>
<td>30.2</td>
<td>15.28</td>
<td>20.3</td>
</tr>
<tr>
<td>Counties Manukau Total</td>
<td>744</td>
<td>148.8</td>
<td>75.30</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with type 1 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Local Policy Documents and Evidence-Based Reviews Relevant to Type 1 Diabetes

In New Zealand a small number of policy documents are relevant to the management of children and young people with Type 1 diabetes and these are reviewed in Table 84, along with a range of guidelines and reviews which consider these issues in the overseas context.

Table 84. Local Policy Documents and Evidence-Based Reviews Relevant to Type 1 Diabetes in Children and Young People

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>There are no specific Ministry of Health guidelines for Type 1 Diabetes in New Zealand. A small number of publications review different aspects of Diabetes care.</td>
</tr>
<tr>
<td>This systematic review update was performed at the request of the Ministry of Health and considers whether subcutaneous insulin pump therapy is effective, safe, and cost-effective compared with multiple daily injections. The update was based on the National Institute for Health and Clinical Excellence (NICE) guidance for the use of continuous subcutaneous insulin infusion (CSII) in diabetes <a href="http://publications.nice.org.uk/continuous-subcutaneous-insulin-infusion-for-the-treatment-of-diabetes-mellitus-ta151">http://publications.nice.org.uk/continuous-subcutaneous-insulin-infusion-for-the-treatment-of-diabetes-mellitus-ta151</a>. It concluded that compared to multiple daily injections, CSII produces a modest improvement in glycosylated haemoglobin levels in all patient groups assessed, including children and that based on the studies identified there is limited evidence to support the contention that CSII produces a reduction in the incidence of severe hypoglycaemic events and improved quality of life (due to greater flexibility of lifestyle).</td>
</tr>
<tr>
<td>These guidelines are to provide ophthalmologists, optometrists and those involved with photographic retinal screening with a nationally consistent approach to classifying and referring people with significant diabetic retinopathy for review by an ophthalmologist, using eye screening photographs, with the ability to measure and monitor grading and referrals against a national grading and referral standard.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>International Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Executive Summary: Standards of Medical Care in Diabetes—2013 Diabetes Care January 2013 36:S4-S10; doi:10.2337/dc13-S004 <a href="http://care.diabetesjournals.org/content/36/S14/full.pdf">http://care.diabetesjournals.org/content/36/S14/full.pdf</a></td>
</tr>
<tr>
<td>These standards of care are intended to provide clinicians, patients, researchers, payers, and other interested individuals with the components of diabetes care, general treatment goals, and tools to evaluate the quality of care. The recommendations included are screening, diagnostic, and therapeutic actions that are known or believed to favourably affect health outcomes of patients with diabetes. A large number of these interventions have been shown to be cost-effective. A grading system, developed by the American Diabetes Association (ADA) and modelled after existing methods, was utilised to clarify and codify the evidence that forms the basis for the recommendations. These standards of care are revised annually by the ADA’s multidisciplinary Professional Practice Committee, incorporating new evidence. For the current revision, committee members systematically searched Medline for human studies related to each subsection and published since 1 January 2011.</td>
</tr>
<tr>
<td>These evidence-based clinical practice guidelines are intended to support healthcare decisions based on the most current evidence available. They provide recommendations on screening, prevention, diagnosis, education, care and management of diabetes. Chapters 4, 12, and 34 have specific recommendations for type 1 diabetes.</td>
</tr>
</tbody>
</table>

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These recommendations come from the Italian Society of Pediatric Endocrinology and Diabetology after an extensive review of the literature. Recommendations for the following issues were given: self-monitoring blood glucose, continuous glucose monitoring, glycemic variability, glycosuria, ketonuria, ketonemia, glycated hemoglobin, fructosamine and glycated albumin, logbook, data downloading, lancing devices, carbohydrate counting, and glycemic measurements at school.

<table>
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<tbody>
<tr>
<td>This national evidence-based guideline provides a comprehensive resource for the healthcare professional team in the modern clinical care of people with type 1 diabetes in Australia. It was developed by an Expert Advisory Group (EAG) representing specialist societies and organisations, with the active participation of consumer groups and the community. The guidelines are to be used in both the hospital and ambulatory healthcare setting. The guideline produced recommendations – based on evidence from the systematic reviews; and practice points – based on consensus decision-making, where the systematic review found insufficient high-quality data to produce evidence-based recommendations, but clinicians require guidance to ensure good clinical practice. The technical report that presents the findings from the systematic reviews that underpin these guidelines can be found here:</td>
<td><a href="http://www.diabetessociety.com.au/downloads/guidelinesTechReport1.pdf">http://www.diabetessociety.com.au/downloads/guidelinesTechReport1.pdf</a></td>
</tr>
</tbody>
</table>

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<tbody>
<tr>
<td>This guideline is based on the ISPAD Clinical Practice Consensus Guidelines Compendium 2009 (Pediatr Diabetes 2009; 10 (Suppl 12): 1-210) using evidence from reports from key meta-analyses, evidence-based reviews, clinical trials, cohort studies, epidemiological studies, animal and basic science studies, position statements and guidelines. The guideline provides a practical approach to promote the implementation of cost-effective, evidence-based care for children and adolescents across settings where resources may vary. Three levels of care are outline: recommended care; limited care for very limited resource locations; and comprehensive care for settings with considerable resources.</td>
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<tbody>
<tr>
<td>These evidence-based practice guidelines were developed to determine settings where patients are most likely to benefit from the use of continuous glucose monitoring (CGM). Real-time CGM (RT-CGM) alone was not recommended for adults in intensive care units or operating rooms pending further research. RT-CGM was recommended for children, adolescents and adult outpatients with some provisos including the ability to use the device.</td>
<td></td>
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</tbody>
</table>

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<tbody>
<tr>
<td>This guideline updates a previous version (2001) and provides recommendations based on current evidence for best practice in the management of diabetes for children and adults with type 1 and type 2 diabetes mellitus, and for pregnant women with gestational diabetes. Major recommendations include: lifestyle management; psychosocial factors; and management of diabetes and related conditions. Management of type 1 diabetes includes: diagnosis and epidemiology; initiating therapy at diagnosis; continuing management; quality of life; and long term complications and screening. The full guidelines and a summary of recommendations can be found at the link above.</td>
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</tbody>
</table>

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</tr>
</thead>
<tbody>
<tr>
<td>This NICE guideline addresses the diagnosis and management of babies, children, adolescents, adults and older people with type 1 diabetes. It covers care in primary and secondary health care and addresses the interface between community and specialist care, and between paediatric and adult services. The guideline also addresses support from the health system to early childhood services, schools and other institutions in the UK context.</td>
<td></td>
</tr>
</tbody>
</table>
### Cochrane Systematic Reviews


Continuous glucose monitoring (CGM) systems measure interstitial fluid glucose levels to provide semi-continuous information about glucose levels. Currently, the use of CGM is not common practice and its reimbursement status is a point of debate in many countries. This systematic review assessed the effects of CGM systems compared to conventional self-monitoring of blood glucose (SMBG) in patients with diabetes mellitus type 1. Twenty-two RCTs were included with results of the meta-analyses (across all age groups) indicating benefit of CGM for patients starting on CGM sensor augmented insulin pump therapy compared to patients using multiple daily injections of insulin (MDI) and standard monitoring blood glucose (SMBG). After six months there was a significant larger decline in HbA1c level for real-time CGM users starting insulin pump therapy compared to patients using MDI and SMBG (mean difference (MD) in change in HbA1c level -0.7%, 95% CI -0.8% to -0.5%, 2 RCTs, 562 patients, I²=84%). The risk of hypoglycaemia was increased for CGM users, but CIs were wide and included unity (4/43 versus 1/35; RR 3.26, 95% CI 0.38 to 27.82 and 21/247 versus 17/248; RR 1.24, 95% CI 0.67 to 2.29). There is limited evidence for the effectiveness of real-time continuous glucose monitoring (CGM) use in children, adults and patients with poorly controlled diabetes. The largest improvements in glycaemic control were seen for sensor-augmented insulin pump therapy in patients with poorly controlled diabetes who had not used an insulin pump before. There are indications that higher compliance of wearing the CGM device improves glycosylated haemoglobin A1c level (HbA1c) to a larger extent.

**Misso ML, et al. 2010.** Continuous subcutaneous insulin infusion (CSII) versus multiple insulin injections for type 1 diabetes mellitus. Cochrane Database of Systematic Reviews (1).

Glycaemic control is maintained in type 1 diabetes by replacement of insulin and may be in the form of 'conventional' insulin therapy (multiple injections per day) or continuous subcutaneous insulin infusion (CSII). This systematic review assessed the effects of CSII compared to multiple insulin injections (MI) in people with type 1 diabetes mellitus. Twenty three randomised controlled trial studies (976 participants) comparing CSII with three or more insulin injections per day in people with type 1 diabetes were included in the analysis. There was a statistically significant difference in glycosylated haemoglobin A1c (HbA1c) favouring CSII (weighted mean difference -0.3% (95% confidence interval -0.1 to -0.4). There were no obvious differences between the interventions for non-severe hypoglycaemia, but severe hypoglycaemia appeared to be reduced in those using CSII. Quality of life measures suggest that CSII is preferred over MI. There is some evidence to suggest that CSII may be better than MI for glycaemic control in people with type 1 diabetes. Non-severe hypoglycaemic events do not appear to be reduced with CSII. There is insufficient evidence regarding adverse events, mortality, morbidity and costs.


This review found two trials (60 participants) which investigated the effect of metformin added to insulin therapy for three months in adolescents with poorly controlled type 1 diabetes. The authors concluded “There is some evidence suggesting improvement of metabolic control in poorly controlled adolescents with type 1 diabetes, on addition of metformin to insulin therapy. Stronger evidence is required from larger studies, carried out over longer time periods to document the long-term effects on metabolic control, health-related quality of life as well as morbidity and mortality in those patients.”


This systematic review assessed the effects of routine hospital admission compared to out-patient or home-based management in children newly diagnosed with type 1 diabetes mellitus. Seven studies were included in the review, including a total of 298 children in the out-patient/home group. There was only one high quality trial identified which suggested that home-based management of children with newly diagnosed type 1 diabetes may lead to slightly improved long term metabolic control (at two and three years follow-up). No differences between comparison groups were found in any of the psychosocial and behavioural variables or rates of acute diabetic complications within two years. Due to the generally low quality or limited applicability of the studies identified, the results of this review are inconclusive. Generally, the data seem to suggest that where adequate out-patient/home management of type 1 diabetes in children at diagnosis can be provided, this does not lead to any disadvantages in terms of metabolic control, acute diabetic complications and hospitalisations, psychosocial variables and behaviour, or total costs.


As part of their treatment regimen patients with type 1 diabetes require basal insulin replacement. This review compares the use of intermediate versus long acting insulin for this purpose. Twenty three randomised controlled trials involving 3872 patients in the intervention group and 2915 patients in the control group were analysed. Overall the differences between the two groups were small and there were no differences in the quantity or quality of severe adverse events or deaths but the authors noted that there appeared to be a beneficial effect of long acting insulin on nocturnal glucose levels.
### Other Systematic Reviews

<table>
<thead>
<tr>
<th>Reference</th>
<th>Title</th>
<th>Journal/Source</th>
</tr>
</thead>
</table>

This systematic review explored physical activity and/or sedentary behaviour randomised controlled intervention studies for youth (<18 years) with type 1 diabetes. Eleven studies were included with 8 improving physical activity and/or fitness. Meta-analysis of 10 studies showed the interventions have a significant beneficial reduction of HbA1c (%), indicating an improvement in glycaemic control [WMD, -0.85% (95% CI, -1.45 to -0.25%)]. Limited reporting made comparison of findings challenging. There was an overall significant beneficial effect of physical activity on HbA1c.

This systematic review and meta-analysis explored the evidence for a glycaemic benefit of exercise in type 1 diabetes, defined as an improvement in glycosylated haemoglobin (HbA1c). Eight randomised and five non-randomised controlled trials were included. A meta-analysis of 12 studies (452 patients) demonstrated a trend towards HbA1c reduction (standardised mean difference (SMD) -0.25; 95% CI, -0.59 to 0.09), with no reduction seen in the four adult studies. This meta-analysis does not reveal evidence for a glycaemic benefit of exercise as measured by HbA1c although exercise does have other proven benefits in type 1 diabetes, and remains an important part of its management.

This review was to identify studies that used a text message-based intervention for youth with type 1 diabetes. Seven studies were included (3 randomised controlled trials) with the majority aiming to evaluate the feasibility and effectiveness of a text message intervention to improve glycaemic control. The reviewed articles had small sample sizes and mixed results regarding the effectiveness of text message interventions with respect to daily type 1 diabetes management behaviours and glycaemic control. However, they did demonstrate their feasibility and high levels of participant satisfaction. Further research using experimental designs and grounded in behavioural theory is needed.

This review investigated the way that family stress influences glycaemic control among patients with diabetes less than 18 years of age. Six cohort and 3 cross-sectionalal studies, and 1 qualitative review were included. In most studies family stress was negatively correlated with patients' glycaemic control. Family function was strongly related to patients' glycaemic control, while family conflict was adversely associated with glycaemic control. Families of low socioeconomic status, those of adolescents with diabetes, and those of single parents were more prone to diabetes-related stress and thus more susceptible to worse glycaemic control. Therapeutic psychological interventions and educational programmes can help alleviate family diabetes-related stress and will likely improve glycaemic control.

This meta-analysis was conducted to determine the overall effects of exercise (acute bouts of exercise and chronic exercise [or training]) on acute and chronic glycaemic control in patients with type 1 diabetes, the effects of different types of exercise on glycaemic control and which conditions are required to obtain these positive effects. Thirty-three studies were included. Aerobic exercise, resistance exercise, mixed exercise (aerobic combined with resistance training) and high-intensity exercise acutely decreased blood glucose levels. To prevent late-onset hypoglycaemic episodes, the use of single bouts of sprints into an aerobic exercise can be recommended. Only regular aerobic training will improve the glycaated haemoglobin level of a patient with type 1 diabetes.

This systematic review was conducted to determine the benefits and harms of current modes of intensive insulin therapy (continuous subcutaneous insulin infusion [CSII] vs. multiple daily injections [MDI]) and modes of blood glucose monitoring (real-time continuous glucose monitoring [rt-CGM] vs. self-monitoring of blood glucose [SMBG]). Forty-one studies were included and clinical outcomes were assessed in individuals with type 1 diabetes and type 2 diabetes. Both CSII and MDI had similar effects on glycaemic control and rates of severe hypoglycaemia in children and adolescents with type 1 diabetes and adults with type 2 diabetes. In contrast, some studies suggested that CSII was superior to MDI for glycaemic control in adults with type 1 diabetes with no difference in hypoglycaemia and weight gain. Limited evidence suggested that measures of quality of life or treatment satisfaction improved in patients with type 1 diabetes. rt-CGM/CSII in the form of sensor-augmented pumps was superior to MDI/SMBG in lowering HbA1c in the research studies analysed in this review.

This comparative analysis considered whether continuous glucose monitoring (CGM) provides further efficacy and safety benefits beyond self-monitoring of blood glucose (SMBG) in the management of type 1 diabetes. Fourteen randomised controlled trials (1188 patients) were included. Compared with SMBG, the use of CGM was associated with a greater reduction in HbA1c [-0.3% (confidence interval: 0.4, -0.2), p < 0.0001]. The number of hypoglycaemic events was not significantly different between the CGM and SMBG groups (0.52 ± 0.52 versus 0.52 ± 0.63 events/day, p = 0.5), but duration of hypoglycaemia was shorter for the CGM group (75 ± 39 versus 89 ± 19 min/day). Continuous glucose monitoring also resulted in a shorter duration of hyperglycaemia than SMBG (172 ± 125 versus 217 ± 152 min/day, p = 0.04). The use of CGM is associated with improvement in metabolic control in T1DM, with significant short- and long-term reductions in HbA1c and reduction in the duration of periods of hypoglycaemia and hyperglycaemia versus SMBG.


This systematic review aimed to assess the effects of quality improvement (QI) strategies on glycated haemoglobin (HbA1c), vascular risk management, microvascular complication monitoring, and smoking cessation in patients with diabetes. Randomised controlled trials of patients with either type 1 or type 2 diabetes were included. Forty-eight cluster-randomised controlled trials (2538 clusters and 84,865 patients) and 94 patient randomised controlled trials (38,664 patients) were reviewed. Many trials of QI strategies showed improvements in diabetes care. Interventions targeting the system of chronic disease management along with patient-mediated QI strategies should be an important component of interventions aimed at improving diabetes management. Interventions solely targeting health-care professionals seem to be beneficial only if baseline HbA1c control is poor.


This systematic review examined the evidence on culturally competent interventions for the needs of people with diabetes from ethnic minority groups. Studies were included if they reported primary research on the impact of culturally competent interventions on outcome measures of any ethnic minority group with diabetes. Eleven studies were included with varying study designs. A consistent finding from 10 of the studies was that any structured intervention, tailored to ethnic minority groups by integrating elements of culture, language, religion and health literacy skills, produced a positive impact on a range of patient-important outcomes.


This statement provides a framework for health care delivery during the transition period from late adolescence to young adulthood (18-30 years of age) for individuals with type 1 or type 2 diabetes. The statement addresses issues including the transition between paediatric and adult diabetes care and gives evidence-based recommendations for approaches to improve care transition of emerging adults with diabetes.

Other Relevant Publications and Websites


This Expert Opinion from the New Zealand Society for the Study of Diabetes compares the current situation regarding the use of the long acting insulin glargine in New Zealand with that in other countries. The references used for this expert opinion are now outdated.

Footnote: Some changes in this area have occurred according to a media release from Pharmac (16th August, 2010) http://www.pharmac.govt.nz/2010/08/16/Enhance%20access%20to%20diabetes%20treatments.pdf

- Widening access to the long-acting insulin glargine (Lantus);
- Funding a new rapid-acting insulin glulisine (Apidra);
- Widening access to blood ketones testing strips (Optium); and
- Widening access to the diabetes treatment acarbose (Glucobay).

In this position statement the members of the executive of the NZSSD offer their recommendations on who should be eligible for Insulin Pump therapy and how such therapy should be administered.


Funding of New Zealand medical and scientific insulin pump and consumables was approved and available from 1 September 2012 under certain criteria as outlined in the following document:


Diabetes New Zealand is a non-government non-profit organisation that was established in 1962 to represent and support people affected by diabetes. Diabetes New Zealand provides local support, advocacy, education and information and support of research with 22 branches throughout New Zealand.


Diabetes Youth New Zealand is a voluntary society established to provide support for children with diabetes and their families. It provides information about diabetes, news about camps and events and gives links to useful New Zealand and international websites related to diabetes for young people and young adults with diabetes.


Paediatric Society of New Zealand (PCNZ) is a multi-disciplinary Society committed to improving the health of children and young people. The KidsHealth website is a joint initiative between the Starship Foundation and PCNZ and provides information for New Zealand parents, caregivers, family and whānau, and for health professionals. This page gives information about diabetes.


This website of the American Diabetes Association gives information about diabetes, advocacy and news and research. The American Diabetes Association funds research to prevent, cure and manage diabetes, delivers services to communities, provides objective information and advocates for those denied their rights because of diabetes.


This Diabetes Resource Centre is part of [http://www.aboutkidshealth.ca](http://www.aboutkidshealth.ca) from the Toronto Hospital for Sick Children. The resources are organised to follow the natural course of diabetes from symptoms, to diagnosis, to treatment, and to long-term conditions. It contains illustrations, animations and games to help children understand diabetes.

[http://www.aboutkidshealth.ca/En/ResourceCentres/Diabetes/Pages/default.aspx](http://www.aboutkidshealth.ca/En/ResourceCentres/Diabetes/Pages/default.aspx)

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Introduction

An epileptic seizure is defined as the manifestation of an abnormal or excessive discharge of neurons in the brain. Epilepsy is defined as recurrent seizures, specifically two or more seizures separated by 24 hours but within 18 months of one another. This is based on the finding that children who experience one seizure have a 50% chance of recurrence within two years. Febrile seizures are normally excluded from these definitions [80].

The incidence of childhood epilepsy (number of new cases) has been reported to be around 80 per 100,000, which is higher than in the adult population. The prevalence of epilepsy (existing cases at any point in time), ranges from 4–7 per 1,000 depending on the study cited [80].

Epilepsy is commonly classified into three categories according to aetiology: genetic, metabolic/structural and idiopathic/unknown. Genetic disorders include diseases with a known genetic defect, where seizures are the main manifestation. Metabolic/structural causes include head injuries, central nervous system infections, and tumours. Most children with epilepsy have epilepsy of unknown cause [80].

In developed countries, it has been consistently shown that those with epilepsy have a 2–3 fold increase in risk of mortality. Almost all of the excess risk in children with epilepsy is in those with severe neurological conditions. Risk factors which increase the risk of mortality include onset of seizures in the first year of life, status epilepticus before diagnosis and poor seizure control. In contrast, mortality risk is only minimally elevated in children with epilepsy who are neurologically normal [81].

An audit of epilepsy-related deaths in the UK found that 59% of deaths during childhood could have potentially or probably been avoided had sufficient attention been given to appropriate drug management, access to specialist care, or adequate investigations [82]. Good seizure control is one of the most important aspects of prevention [81]. The appropriate management of status epilepticus is also important, as it is associated with significant mortality (3% in paediatric population data, and up to 32% in patients with refractory status epilepticus in paediatric intensive care). In light of this, a set of Australasian practice guidelines outlining the optimal management of status epilepticus in the emergency department setting has recently been released [83].

The following section reviews hospital admissions in children and young people aged 0–24 years with any mention of epilepsy or status epilepticus in any of the first 15 diagnoses.

Data Source and Methods

Definition
1. Hospital admissions for children and young people aged 0–24 years with epilepsy or status epilepticus listed in any of the first 15 diagnoses
2. Mortality for children and young people aged 0–24 years with epilepsy or status epilepticus listed as the main underlying cause of death or as a contributory cause

Data Source
1. National Minimum Dataset
   Numerator: Hospital admissions for children and young people aged 0–24 years with epilepsy (ICD-10-AM G40) or status epilepticus (ICD-10-AM G41) in any of the first 15 diagnoses.
2. National Mortality Collection
   Numerator: Mortality in children and young people aged 0–24 years with epilepsy (ICD-10-AM G40) or status epilepticus (ICD-10-AM G41) listed as the main underlying cause of death, or as a contributory cause.
   Denominator: Statistics New Zealand Estimated Resident Population (projected from 2007)
Notes on Interpretation

Note 1: This analysis focuses on hospital admissions and mortality for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses, or as a main underlying or contributory cause of death. The rationale for looking beyond the primary diagnosis was the need to identify the full spectrum of health issues experienced by those with epilepsy and their consequent need for acute health services.

Note 2: A review of the secondary diagnoses in those admitted with a primary diagnosis of epilepsy or status epilepticus also highlighted the fact that a number had other underlying conditions (e.g. cerebral palsy, developmental delay, congenital anomalies) which may have increased their risk of developing epilepsy.

Note 3: Children and young people with febrile or unspecified convulsions were not included in the analysis unless they also had a diagnosis of epilepsy or status epilepticus, on the basis that for many, such seizures are one off events which do not lead to a subsequent diagnosis of epilepsy.

Note 4: If no mention of epilepsy or status epilepticus was made in any of the first 15 diagnoses, these cases were not included, even if the patient had been assigned an epilepsy-related code on a previous admission.

New Zealand Distribution and Trends

Distribution by Age

In New Zealand during 2008–2012, hospital admissions for those with epilepsy or status epilepticus were highest during the first four years of life, with rates declining during childhood, to reach their lowest point at 14 years. Rates then increased again slightly, to reach a plateau amongst those in their late teens and early twenties. Mortality during 2006–2010 occurred sporadically across the age range, although rates were generally higher for those in their early twenties, than for those in late childhood (Figure 47).

Figure 47. Hospital Admissions (2008–2012) and Mortality (2006–2010) for New Zealand Children and Young People with Epilepsy or Status Epilepticus by Age

Source: Numerator Admissions: National Minimum Dataset, hospital admissions for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Numerator Mortality: National Mortality Collection, Deaths with epilepsy or status epilepticus listed as the main underlying or contributory cause of death; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 85. Hospital Admissions in Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Admissions in those with Epilepsy or Status Epilepticus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy or Status Epilepticus</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnoses other than Epilepsy*</td>
<td>2,310</td>
<td>462.0</td>
<td>30.25</td>
<td>24.7</td>
</tr>
<tr>
<td>Generalized: Idiopathic</td>
<td>2,281</td>
<td>456.2</td>
<td>29.87</td>
<td>24.4</td>
</tr>
<tr>
<td>Unspecified Epilepsy</td>
<td>1,873</td>
<td>374.6</td>
<td>24.53</td>
<td>20.1</td>
</tr>
<tr>
<td>Status Epilepticus</td>
<td>763</td>
<td>152.6</td>
<td>9.99</td>
<td>8.2</td>
</tr>
<tr>
<td>Grand Mal Seizures NOS</td>
<td>599</td>
<td>119.8</td>
<td>7.84</td>
<td>6.4</td>
</tr>
<tr>
<td>Focal: Symptomatic with Complex Partial Seizures</td>
<td>520</td>
<td>104.0</td>
<td>6.81</td>
<td>5.6</td>
</tr>
<tr>
<td>Focal: Symptomatic with Simple Partial Seizures</td>
<td>402</td>
<td>80.4</td>
<td>5.26</td>
<td>4.3</td>
</tr>
<tr>
<td>Generalized: Other</td>
<td>370</td>
<td>74.0</td>
<td>4.85</td>
<td>4.0</td>
</tr>
<tr>
<td>Other Epilepsy</td>
<td>157</td>
<td>31.4</td>
<td>2.06</td>
<td>1.7</td>
</tr>
<tr>
<td>Focal: Idiopathic with Localized Onset Seizures</td>
<td>37</td>
<td>7.4</td>
<td>0.48</td>
<td>0.4</td>
</tr>
<tr>
<td>Petit Mal NOS</td>
<td>17</td>
<td>3.4</td>
<td>0.22</td>
<td>0.2</td>
</tr>
<tr>
<td>Special Epileptic Syndromes</td>
<td>9</td>
<td>1.8</td>
<td>0.12</td>
<td>0.1</td>
</tr>
<tr>
<td>Total Epilepsy-Related Diagnoses</td>
<td>7,028</td>
<td>1,405.6</td>
<td>92.04</td>
<td>75.3</td>
</tr>
<tr>
<td>Total Admissions</td>
<td>9,338</td>
<td>1,867.6</td>
<td>122.29</td>
<td>100.0</td>
</tr>
<tr>
<td>*Other Diagnoses</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diseases of the Respiratory System</td>
<td>424</td>
<td>84.8</td>
<td>5.55</td>
<td>4.5</td>
</tr>
<tr>
<td>Injury and Poisoning</td>
<td>316</td>
<td>63.2</td>
<td>4.14</td>
<td>3.4</td>
</tr>
<tr>
<td>Pregnancy, Childbirth, Puerperium</td>
<td>172</td>
<td>34.4</td>
<td>2.25</td>
<td>1.8</td>
</tr>
<tr>
<td>Other Diseases Nervous System</td>
<td>157</td>
<td>31.4</td>
<td>2.06</td>
<td>1.7</td>
</tr>
<tr>
<td>Other Infectious and Parasitic Diseases</td>
<td>104</td>
<td>20.8</td>
<td>1.36</td>
<td>1.1</td>
</tr>
<tr>
<td>Q00-Q99 Congenital Anomalies</td>
<td>97</td>
<td>19.4</td>
<td>1.27</td>
<td>1.0</td>
</tr>
<tr>
<td>Dental and Oral Health</td>
<td>92</td>
<td>18.4</td>
<td>1.20</td>
<td>1.0</td>
</tr>
<tr>
<td>Unspecified Convulsions</td>
<td>37</td>
<td>7.4</td>
<td>0.48</td>
<td>0.4</td>
</tr>
<tr>
<td>All Other Diagnoses</td>
<td>911</td>
<td>182.2</td>
<td>11.93</td>
<td>9.8</td>
</tr>
<tr>
<td>Total Other Diagnoses</td>
<td>2,310</td>
<td>462.0</td>
<td>30.25</td>
<td>24.7</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 86. Secondary Diagnoses in Children and Young People Aged 0–24 Years Hospitalised with Epilepsy or Status Epilepticus as a Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Secondary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>% of Admissions in those with Epilepsy or Status Epilepticus as Primary Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secondary Diagnosis in Admissions with Epilepsy or Status Epilepticus as a Primary Diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory Infections and Diseases</td>
<td>347</td>
<td>69.4</td>
<td>4.9</td>
</tr>
<tr>
<td>History of Non-Compliance with Treatment</td>
<td>310</td>
<td>62.0</td>
<td>4.4</td>
</tr>
<tr>
<td>Cerebral Palsy and Other Paralytic Syndromes</td>
<td>297</td>
<td>59.4</td>
<td>4.2</td>
</tr>
<tr>
<td>Other Diseases of the Nervous System</td>
<td>221</td>
<td>44.2</td>
<td>3.1</td>
</tr>
<tr>
<td>Tobacco Use</td>
<td>213</td>
<td>42.6</td>
<td>3.0</td>
</tr>
<tr>
<td>Injury and Poisoning</td>
<td>202</td>
<td>40.4</td>
<td>2.9</td>
</tr>
<tr>
<td>Other Congenital Anomalies</td>
<td>167</td>
<td>33.4</td>
<td>2.4</td>
</tr>
<tr>
<td>Developmental Delay</td>
<td>149</td>
<td>29.8</td>
<td>2.1</td>
</tr>
<tr>
<td>Congenital Anomalies Nervous System</td>
<td>121</td>
<td>24.2</td>
<td>1.7</td>
</tr>
<tr>
<td>Viral Infection, Unspecified</td>
<td>101</td>
<td>20.2</td>
<td>1.4</td>
</tr>
<tr>
<td>Mental Retardation</td>
<td>98</td>
<td>19.6</td>
<td>1.4</td>
</tr>
<tr>
<td>Autism and Other Pervasive Developmental Disorders</td>
<td>94</td>
<td>18.8</td>
<td>1.3</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>91</td>
<td>18.2</td>
<td>1.3</td>
</tr>
<tr>
<td>Sequeulae of Head Injuries</td>
<td>74</td>
<td>14.8</td>
<td>1.1</td>
</tr>
<tr>
<td>Mental and Behavioural Disorders due to Alcohol</td>
<td>55</td>
<td>11.0</td>
<td>0.8</td>
</tr>
<tr>
<td>Otitis Media</td>
<td>53</td>
<td>10.6</td>
<td>0.8</td>
</tr>
<tr>
<td>Other Infectious and Parasitic Diseases</td>
<td>52</td>
<td>10.4</td>
<td>0.7</td>
</tr>
<tr>
<td>Epilepsy or Status Epilepticus</td>
<td>41</td>
<td>8.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>1,241</td>
<td>248.2</td>
<td>17.7</td>
</tr>
<tr>
<td>No Secondary Diagnosis</td>
<td>3,101</td>
<td>620.2</td>
<td>44.1</td>
</tr>
<tr>
<td>Total</td>
<td>7,028</td>
<td>1,405.6</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by secondary diagnosis for children and young people with epilepsy or status epilepticus listed as their primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Distribution by Primary and Secondary Diagnosis

Primary Diagnosis: In New Zealand during 2008–2012, 75.3% of all hospital admissions in children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses, had an epilepsy-related diagnosis listed as the primary reason for admission. Generalised idiopathic epilepsy (24.4%) and unspecified epilepsy (20.1%) were the most frequent epilepsy-related diagnoses. A further 24.7% of admissions were for conditions unrelated to epilepsy, with respiratory conditions and injury and poisoning being the most frequent non epilepsy-related diagnoses (Table 85).

Secondary Diagnosis: During the same period, the secondary diagnoses assigned to children and young people admitted with epilepsy or status epilepticus as a primary diagnosis, fell into two main categories: those conditions which may have increased the risk of the child or young person developing epilepsy (e.g. cerebral palsy, congenital anomalies, and other diseases of the nervous system); and acute concurrent illnesses such as respiratory and viral infections (Table 86).

Distribution by Ethnicity and Gender

In New Zealand during 2008–2012, hospital admissions for those with epilepsy or status epilepticus were significantly higher for males. Admission rates were also significantly higher for Māori and Pacific children and young people, than for European/Other, and then Asian/Indian children and young people (Table 87).

Similarly during 2000–2012, hospitalisations for Asian/Indian children and young people with epilepsy or status epilepticus were consistently lower than for Māori, Pacific and European/Other children and young people. While rates for Māori, Pacific and European/Other children and young people were similar during the early 2000s, diverging trends saw rates for Māori and Pacific children and young people become higher than for European/Other children and young people from 2008–09 onwards (Figure 48).

Table 87. Hospital Admissions for Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate</td>
<td></td>
<td></td>
<td></td>
<td>Rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epilepsy or Status Epilepticus</td>
<td></td>
<td></td>
<td></td>
<td>Epilepsy or Status Epilepticus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>56.6</td>
<td>0.48</td>
<td>0.44–0.53</td>
<td>Female</td>
<td>119.4</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>117.6</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>125.0</td>
<td>1.05</td>
<td>1.01–1.09</td>
</tr>
<tr>
<td>Māori</td>
<td>153.5</td>
<td>1.31</td>
<td>1.25–1.37</td>
<td>Pacific</td>
<td>151.3</td>
<td>1.29</td>
<td>1.20–1.37</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population
Figure 48. Hospital Admissions for Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised

Northern DHBs Distribution and Trends

Northern DHBs Distribution

In the Northern region during 2008–2012, a total of 182 Northland, 523 Waitemata, 330 Auckland and 644 Counties Manukau children and young people were hospitalised with a diagnosis of epilepsy or status epilepticus. Admission rates per 100,000 population were significantly higher than the New Zealand rate in Northland and Counties Manukau and significantly lower in Auckland DHB. In the Waitemata DHB, admission rates were not significantly different from the New Zealand rate (Table 88).

Northern DHBs Trends

In all four Northern DHBs, admission rates for those with epilepsy or status epilepticus declined during the early to mid 2000s, but were more static (or even increased slightly) after 2006–2007. During this period, rates in Auckland DHB were consistently lower than the New Zealand rate, while in Northland rates were consistently higher. Rates in Waitemata and Counties Manukau DHBs were closer to the New Zealand rate (Figure 49).
Table 88. Hospital Admissions for Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A*</td>
<td>B*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northland</td>
<td>177</td>
<td>182</td>
<td>424</td>
<td>0.47</td>
<td>156.92</td>
<td>1.28</td>
</tr>
<tr>
<td>Waitemata</td>
<td>495</td>
<td>523</td>
<td>1,166</td>
<td>0.45</td>
<td>123.52</td>
<td>1.01</td>
</tr>
<tr>
<td>Auckland</td>
<td>308</td>
<td>330</td>
<td>645</td>
<td>0.39</td>
<td>86.12</td>
<td>0.70</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>621</td>
<td>644</td>
<td>1,324</td>
<td>0.41</td>
<td>134.00</td>
<td>1.10</td>
</tr>
<tr>
<td>New Zealand</td>
<td>4,163</td>
<td>9,338</td>
<td></td>
<td>0.45</td>
<td>122.29</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (sum of DHB totals exceeds NZ total); Rate Ratios are compared to NZ rate and have not been adjusted for population demographics

Distribution by Primary Diagnosis

In the Northern DHBs during 2008–2012, around three quarters (range 66.0%–79.6%) of all hospital admissions in children and young people with epilepsy or status epilepticus listed in the first 15 diagnoses had an epilepsy-related diagnosis listed as the primary reason for admission. Unspecified epilepsy and generalised idiopathic epilepsy were the most frequent epilepsy-related diagnoses in all four DHBs (Table 89, Table 90).
Figure 49. Hospital Admissions for Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 89. Hospital Admissions in Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Primary Diagnosis, Northland and Waitemata DHBs 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Northland</th>
<th>Waitemata</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Admissions: Total</td>
<td>Number of Admissions: Annual</td>
</tr>
<tr>
<td></td>
<td>2008–2012</td>
<td>Average</td>
</tr>
<tr>
<td>Diagnoses other than Epilepsy</td>
<td>144</td>
<td>28.8</td>
</tr>
<tr>
<td>Unspecified Epilepsy</td>
<td>80</td>
<td>16.0</td>
</tr>
<tr>
<td>Generalized: Idiopathic</td>
<td>77</td>
<td>15.4</td>
</tr>
<tr>
<td>Status Epilepticus</td>
<td>47</td>
<td>9.4</td>
</tr>
<tr>
<td>Focal: Symptomatic with Complex Partial Seizures</td>
<td>18</td>
<td>3.6</td>
</tr>
<tr>
<td>Focal: Symptomatic with Simple Partial Seizures</td>
<td>17</td>
<td>3.4</td>
</tr>
<tr>
<td>Other Epilepsy</td>
<td>16</td>
<td>3.2</td>
</tr>
<tr>
<td>Generalized: Other</td>
<td>13</td>
<td>2.6</td>
</tr>
<tr>
<td>Grand Mal Seizures NOS</td>
<td>12</td>
<td>2.4</td>
</tr>
<tr>
<td>Northland Total</td>
<td>424</td>
<td>84.8</td>
</tr>
<tr>
<td>Diagnoses other than Epilepsy</td>
<td>298</td>
<td>59.6</td>
</tr>
<tr>
<td>Generalized: Idiopathic</td>
<td>264</td>
<td>52.8</td>
</tr>
<tr>
<td>Unspecified Epilepsy</td>
<td>166</td>
<td>33.2</td>
</tr>
<tr>
<td>Grand Mal Seizures NOS</td>
<td>108</td>
<td>21.6</td>
</tr>
<tr>
<td>Status Epilepticus</td>
<td>104</td>
<td>20.8</td>
</tr>
<tr>
<td>Focal: Symptomatic with Complex Partial Seizures</td>
<td>78</td>
<td>15.6</td>
</tr>
<tr>
<td>Generalized: Other</td>
<td>59</td>
<td>11.8</td>
</tr>
<tr>
<td>Focal: Symptomatic with Simple Partial Seizures</td>
<td>58</td>
<td>11.6</td>
</tr>
<tr>
<td>Other Epilepsy</td>
<td>21</td>
<td>4.2</td>
</tr>
<tr>
<td>Petit Mal NOS</td>
<td>5</td>
<td>1.0</td>
</tr>
<tr>
<td>Focal: Idiopathic with Localized Onset Seizures</td>
<td>5</td>
<td>1.0</td>
</tr>
<tr>
<td>Waitemata Total</td>
<td>1,166</td>
<td>233.2</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
### Table 90. Hospital Admissions in Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Primary Diagnosis, Auckland and Counties Manukau DHBs 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Epilepsy or Status Epilepticus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnoses other than Epilepsy</td>
<td>137</td>
<td>27.4</td>
<td>18.29</td>
<td>21.2</td>
</tr>
<tr>
<td>Generalized: Idiopathic</td>
<td>208</td>
<td>41.6</td>
<td>27.77</td>
<td>32.2</td>
</tr>
<tr>
<td>Unspecified Epilepsy</td>
<td>80</td>
<td>16.0</td>
<td>10.68</td>
<td>12.4</td>
</tr>
<tr>
<td>Status Epilepticus</td>
<td>71</td>
<td>14.2</td>
<td>9.48</td>
<td>11.0</td>
</tr>
<tr>
<td>Focal: Symptomatic with Complex Partial Seizures</td>
<td>64</td>
<td>12.8</td>
<td>8.55</td>
<td>9.9</td>
</tr>
<tr>
<td>Generalized: Other</td>
<td>24</td>
<td>4.8</td>
<td>3.20</td>
<td>3.7</td>
</tr>
<tr>
<td>Grand Mal Seizures NOS</td>
<td>23</td>
<td>4.6</td>
<td>3.07</td>
<td>3.6</td>
</tr>
<tr>
<td>Focal: Symptomatic with Simple Partial Seizures</td>
<td>22</td>
<td>4.4</td>
<td>2.94</td>
<td>3.4</td>
</tr>
<tr>
<td>Focal: Idiopathic with Localized Onset Seizures</td>
<td>12</td>
<td>2.4</td>
<td>1.60</td>
<td>1.9</td>
</tr>
<tr>
<td>Other Epilepsy</td>
<td>4</td>
<td>0.8</td>
<td>0.53</td>
<td>0.6</td>
</tr>
<tr>
<td>Auckland DHB Total</td>
<td>645</td>
<td>129.0</td>
<td>86.12</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Counties Manukau</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnoses other than Epilepsy</td>
<td>270</td>
<td>54.0</td>
<td>27.33</td>
<td>20.4</td>
</tr>
<tr>
<td>Unspecified Epilepsy</td>
<td>376</td>
<td>75.2</td>
<td>38.06</td>
<td>28.4</td>
</tr>
<tr>
<td>Generalized: Idiopathic</td>
<td>355</td>
<td>71.0</td>
<td>35.93</td>
<td>26.8</td>
</tr>
<tr>
<td>Status Epilepticus</td>
<td>95</td>
<td>19.0</td>
<td>9.62</td>
<td>7.2</td>
</tr>
<tr>
<td>Focal: Symptomatic with Complex Partial Seizures</td>
<td>63</td>
<td>12.6</td>
<td>6.38</td>
<td>4.8</td>
</tr>
<tr>
<td>Focal: Symptomatic with Simple Partial Seizures</td>
<td>58</td>
<td>11.6</td>
<td>5.87</td>
<td>4.4</td>
</tr>
<tr>
<td>Grand Mal Seizures NOS</td>
<td>44</td>
<td>8.8</td>
<td>4.45</td>
<td>3.3</td>
</tr>
<tr>
<td>Generalized: Other</td>
<td>32</td>
<td>6.4</td>
<td>3.24</td>
<td>2.4</td>
</tr>
<tr>
<td>Other Epilepsy</td>
<td>15</td>
<td>3.0</td>
<td>1.52</td>
<td>1.1</td>
</tr>
<tr>
<td>Unspecified Convulsions</td>
<td>8</td>
<td>1.6</td>
<td>0.81</td>
<td>0.6</td>
</tr>
<tr>
<td>Special Epileptic Syndromes</td>
<td>4</td>
<td>0.8</td>
<td>0.40</td>
<td>0.3</td>
</tr>
<tr>
<td>Focal: Idiopathic with Localized Onset Seizures</td>
<td>4</td>
<td>0.8</td>
<td>0.40</td>
<td>0.3</td>
</tr>
<tr>
<td>Counties Manukau Total</td>
<td>1,324</td>
<td>264.8</td>
<td>134.00</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Policy Documents and Evidence-Based Reviews Relevant to the Diagnosis or Management of Epilepsy or Status Epilepticus

International guidelines, along with a range of other evidence-based reviews are briefly summarised in Table 91. (Note: It was beyond the scope of this review to consider publications which explored the efficacy of individual drugs in the management of epilepsy, with the focus of the table below being on broader management principles).

Table 91. Policy Documents and Evidence-Based Reviews Relevant to the Diagnosis or Management of Epilepsy or Status Epilepticus

<table>
<thead>
<tr>
<th>International Guidelines and Useful Websites</th>
</tr>
</thead>
<tbody>
<tr>
<td>These clinical guidelines, which are an abbreviated version of the full guideline below, provide guidance on best practice for the care of children, young people and adults with epilepsy. They do not contain details of the evidence on which the recommendations are based.</td>
</tr>
<tr>
<td>These very comprehensive 600-page guidelines cover, in detail, specific topics relating to the treatment and management of people with epilepsy. They are a partial update of the original guideline published in 2004. Each section of the guideline, apart from the introductory chapters, is laid out in the same way. There is an introduction followed by a series of clinical questions. For each question there is a brief answer followed evidence statements with grades to indicate the strength of the evidence and details of the relevant systematic reviews and primary papers which make up the evidence. There are summary tables to present key information regarding comparisons between treatments (where this is available).</td>
</tr>
<tr>
<td>The appendices to the guideline can be found here: <a href="http://guidance.nice.org.uk/CG137/Guidance/Appendices">http://guidance.nice.org.uk/CG137/Guidance/Appendices</a></td>
</tr>
<tr>
<td>This review addressed the following clinical questions:</td>
</tr>
<tr>
<td>1. Are other forms of corticosteroids as effective as ACTH for short-term treatment of infantile spasms?</td>
</tr>
<tr>
<td>2. Are low-dose ACTH regimens effective for short-term treatment of infantile spasms?</td>
</tr>
<tr>
<td>3. Is ACTH more effective than VGB for short-term treatment of infantile spasms?</td>
</tr>
<tr>
<td>4. Is there a role for the ketogenic diet or for AEDs other than VGB in managing infantile spasms?</td>
</tr>
<tr>
<td>5. Does the successful short-term treatment of infantile spasms lead to long-term improvement of neurodevelopmental outcomes or a decreased epilepsy incidence?</td>
</tr>
<tr>
<td>Twenty-six articles were included in the analysis. For each question, evidence was analysed sequentially and then summarized to determine the overall strength of the evidence and to formulate recommendations. The conclusions of the review process were as follows: There is insufficient evidence to determine whether other forms of corticosteroids are as effective as adrenocorticotropic hormone (ACTH) for short-term treatment of infantile spasms (IS) but low-dose ACTH is probably as effective as high-dose ACTH. ACTH is more effective than vigabatrin (VGB) for short-term treatment of children with IS (apart from those with tuberous sclerosis complex). There is insufficient evidence to show that other agents or combination therapy are effective for short-term treatment of IS. Short lag time to treatment leads to better long-term developmental outcome. Successful short-term treatment of cryptogenic IS with ACTH or prednisolone leads to better long-term developmental outcome than treatment with VGB. The review authors recommended that: Low-dose ACTH should be considered for treatment of IS. ACTH or VGB may be useful for short-term treatment of IS and ACTH is to be preferred over VGB. Hormonal therapy (ACTH or prednisolone) may be considered for use in preference to VGB in infants with cryptogenic IS, to possibly improve developmental outcome. A shorter lag time to treatment of IS with either hormonal therapy or VGB possibly improves long-term developmental outcomes.</td>
</tr>
</tbody>
</table>
Uses the evidence regarding the effect of inpatient support groups on families and parents of children hospitalised with intractable epilepsy. There were eleven relevant studies identified by the review authors and at least one was a RCT. The authors state that the evidence indicates that parent support groups can improve parental attitudes and knowledge and decrease parental anxiety. They recommended that mutual support groups for parents and families of vulnerable paediatric patients (i.e. children with intractable epilepsy) in the inpatient care setting should be developed.

The International League Against Epilepsy [http://www.ilae.org/]

This site has a number of useful evidence-based guidelines and reports which can be found here: [http://www.ilae.org/Visitors/Centre/Guidelines.cfm](http://www.ilae.org/Visitors/Centre/Guidelines.cfm). Among them are:

- **Updated ILAE evidence review of antiepileptic drug efficacy and effectiveness as initial monotherapy for epileptic seizures and syndromes**, (2013)
- **Guidelines for imaging infants and children with recent-onset epilepsy**, (2009)
- **Guidelines on Neonatal Seizures** (WHO, ILAE) (2011)


This web page, published by the journal Epilepsia, provides links to a variety of recent special reports and guidelines related to epilepsy. It states that "These reports offer guidelines, expert opinion, and state-of-the art position papers – from the International League Against Epilepsy and from other groups with special (and usually authoritative) expertise. The Editors hope that by gathering such reports from the past few years into a Virtual Issue, we make these reports more accessible to the Epilepsia readership".


These guidelines are the result of a review by the Subcommittee for Pediatric Neuroimaging of the International League Against Epilepsy (ILAE) which examined published series reporting the use of computed tomography (CT) and magnetic resonance imaging (MRI) for the evaluation of children with new-onset seizure(s). The reviewers reported that nearly 50% of imaging studies in these children were abnormal and they provide details on the findings on imaging (or lack of them) and their diagnostic, prognostic and management implications for particular types of epilepsy. They recommend imaging, preferably with MRI if this is available, (because of the lack of radiation and its superior resolution and versatility), when localization-related epilepsy is known or suspected, when there is doubt about the epilepsy classification, or when there is suspicion of an epilepsy syndrome with remote symptomatic cause.


These Scottish guidelines are aimed at health care professionals involved in the diagnosis and management of childhood epilepsies. They do not cover issues relating to babies less than one month of age, non-epileptic seizures, surgical treatments or reproductive issues. (These last are addressed in the adult guidelines). There is an evidence grading system for the research evidence on which the guidelines are based and each recommendation in the guidelines is accompanied by a grade reflecting the strength of the evidence on which it is based. SIGN recommends that these guidelines should be used with caution given their age.

Recent Systematic and other Reviews

Cochrane reviews of drug treatments

Cochrane reviews relating to the prevention of epilepsy in particular circumstances


Around 30% of patients with epilepsy still have seizures despite multiple trials of anti-epileptic drugs. It is believed that early surgical intervention may prevent seizures at a younger age and improve the intellectual and social status of children. This review aimed to determine to determine the benefits and adverse effects of subpial transection, one of many types of surgery for refractory epilepsy, for partial-onset seizures and generalised tonic-clonic seizures in children and adults. The review authors did not find any relevant RCTs or quasi-randomised parallel group studies and so they concluded that there is no evidence to support or refute this surgery.


Infantile spasms, also known as West's syndrome, is a relatively rare condition (0.16–0.42 per 1000 live births) that includes a peculiar type of seizure, a high risk of psychomotor retardation, and usually a characteristic electroencephalographic (EEG) pattern known as hypsarrhythmia. Seizures usually begin in the first year of life and in most cases they resolve by the age of three. This review aimed to compare the effects of single pharmaceutical therapies used to treat infantile spasms in terms of control of the spasms, resolution of the EEG abnormality, relapse rates, psychomotor development, subsequent epilepsy, side effects, and mortality. The review authors found 16 small RCTs (<100 participants) and two larger RCTs (>100 participants). These 18 studies had 916 participants in total, treated with a total of 12 different drugs. Overall the studies were of poor methodological quality, partly because it is considered unethical to give placebo injections to children. Two studies showed that active treatment was better than placebo. The strongest evidence suggested that hormonal treatment (prednisolone or tetracosactide depot) leads to resolution of spasms faster (results from 2 studies) and in more infants (results from 3 studies) than does vigabatrin. Combining the results of three studies showed that 45 out of 81 patients randomly assigned to vigabatrin had resolution of their spasms compared to 57 out of 77 randomly assigned to ACTH, tetracosactide, or high-dose prednisolone (Peto Odds Ratio 0.42, 95% CI 0.21–0.80). The review authors cautioned that it is not yet clear whether these short-term benefits will lead to better long-term outcomes. The review authors also stated that if prednisone or vigabatrin is used, a high dosage is recommended and that vigabatrin may be the treatment of choice in tuberous sclerosis.


The Lennox-Gastaut syndrome (LGS) is an age-specific disorder, characterised by epileptic seizures, a characteristic electroencephalographic (EEG), psychomotor delay and behavioural disorder such as hyperactivity, aggressiveness and autistic tendencies. Between one and ten per cent of children with epilepsy have LGS. It occurs more frequently in males and onset is usually before the age of eight years, with a peak incidence at between three and five years of age. This review aimed to compare the effects of pharmaceutical therapies used to treat LGS in terms of control of seizures and adverse effects. The review authors identified nine relevant RCTs but were not able to perform any meta-analysis because the trials differed in populations studied, drugs used and outcomes measured. They concluded that the best treatment for LGS remains uncertain and that no study so far has shown any one drug to be highly efficacious but rufinamide, lamotrigine, topiramate and felbamate may be helpful as add-on therapy, and clobazam may be helpful for drop seizures. They stated that clinicians should continue to treat each patient individually, weighing up the risks and benefits of each therapy.


Kleine-Levin syndrome (KLS) is a very rare disorder affecting around one person per million mainly adolescent men. It is characterised by recurrent episodes of hypersomnia (excessive sleepiness), hyperphagia (overeating) and abnormal behaviour. The authors of this review aimed to identify and evaluate RCTs studying the effectiveness of drug treatment for Kleine-Levin syndrome but they did not find any. They stated that more research is needed.


Around 2–4% of children aged one month or more experience a seizure during a febrile illness and 30% of them go on to have another. Rapid-acting antiepileptics and antipyretics given during subsequent fever episodes have been used to prevent further seizures and avoid the adverse effects of continuous antiepileptic drugs. This review aimed to assess the effectiveness and safety of this practice. It included 36 articles describing 26 RCTs with 2740 randomised participants assessing 13 interventions of continuous or intermittent prophylaxis compared to placebo or control treatment. Most studies were of moderate to poor methodological quality. No significant benefit was found for valproate, pyridoxine, intermittent phenobarbitone or ibuprofen vs. placebo or no treatment; nor for diclofenac vs. placebo followed by ibuprofen, acetaminophen or placebo; nor for intermittent rectal diazepam vs. intermittent valproate, nor phenobarbitone vs. intermittent rectal diazepam. Adverse effects were reported in up to 30% of children. The review authors concluded “given the benign nature of recurrent febrile seizures, and the high prevalence of adverse effects of these drugs, parents and families should be supported with adequate contact details of medical services and information on recurrence, first aid management and, most importantly, the benign nature of the phenomenon.”
Anti-epileptic drugs have long term adverse effects so when a person’s epilepsy is in remission it may be in their best interest to discontinue medication. This review aimed to determine the optimal time for discontinuing anti-seizure medication in adults and children. It included seven RCTs of early (after less than two seizure-free years) vs. late (after more than two seizure-free years) withdrawal of anti-epileptic drugs (AEDs) involving 924 randomised children. There were no eligible trials in adults. The pooled relative risk for seizure relapse in early vs. late AED withdrawal was 1.32 (95% CI 1.02 to 1.70). On the basis of this estimate, the number needed to harm is ten (indicating that ten people would need to stop their medication early for one person to have a relapse seizure). Early discontinuation was associated with greater relapse rates in people with partial seizures (pooled RR is 1.52 (95% CI 0.95 to 2.41)) or an abnormal EEG (pooled RR 1.67 (95% CI 0.93 to 3.00)). The review authors concluded that the evidence supports waiting at least two seizure-free years before withdrawing AEDs in children, particularly if they have an abnormal EEG and partial seizures but that there is not sufficient evidence to establish when to withdraw AEDs in children who have had generalized seizures and no evidence to guide the timing of withdrawal of AEDs in seizure free adults.

The ketogenic diet is low in carbohydrates and high in fat and it is believed to decrease seizure frequency in people with epilepsy. It has mostly been used in children who have seizures despite treatment with anti-epileptic drugs. This review includes four RCTs with a total of 289 child or adolescent participants. Only one of these studies compared a ketogenic diet to a normal diet. The others compared different ketogenic diets and/or different rates of introduction of the diet. The review authors stated that the results of these studies suggest that, in children, a ketogenic diet results in short to medium term benefits in seizure control, and has an effect which is comparable to modern antiepileptic drugs. They noted that one long term study reported a high drop-out rate in the diet group which suggests that many children find the diet difficult to tolerate. They concluded that, for those with medically intractable epilepsy or those for whom surgery is unsuitable, a ketogenic diet could improve seizure control but it is difficult to tolerate.

Yoga may reduce stress, induce relaxation and influence the electroencephalogram and the autonomic nervous system, thereby preventing or controlling seizures. If it were proved effective it would be an attractive therapeutic option with few adverse effects. This review considered two unblinded trials with a total of 50 subjects (18 who were treated with yoga and 32 who received either alternative treatment or no treatment). Due to the small size of the studies and the resulting very large confidence intervals associated with the outcome measures it was not possible to draw any reliable conclusions about the efficacy of yoga as a treatment for epilepsy.

It is well known that taking anti-epileptic drugs (AEDs) in pregnancy may have adverse effects on the fetus but the relative risks of specific antiepileptic drug exposures remain poorly understood. This review aimed to assess the adverse effects of commonly used antiepileptic drugs on maternal and fetal outcomes in pregnancy in women with epilepsy. It focussed on neurodevelopmental outcomes in children exposed to anti-epileptic drugs in utero. The review authors identified 31 eligible studies based on 18 independent cohorts. Most studies were prospective cohort studies. Fifteen included children born to mothers without epilepsy as controls and four compared children with different AED exposures to each other. Most studies were small although only two studies had recruited fewer than 25 children with AED exposure in utero. Only seven independent cohorts had more than 50 children exposed to AEDs. Due to the wide variety of outcomes measured and methodological approaches the review authors chose to present a descriptive analysis of the studies’ results. They reported that most studies were of limited quality and that there was little evidence about which specific drugs carry more risk than others to the development of children exposed in utero; that the results between studies are conflicting and that while most failed to find a significant detrimental outcome with in utero exposure to monotherapy with carbamazepine, phenytoin or phenobarbitone, this should be interpreted cautiously; that there were few studies of sodium valproate; that exposure to polytherapy in utero was more commonly associated with poorer outcomes as was exposure to any AEDs when analysis did not take into account type of AED (possibly because there was a high proportion of children in these studies who were in fact exposed to polytherapy). The review authors concluded that based on the best evidence currently available, it seems advisable for women to continue medication during pregnancy using monotherapy at the lowest dose required to achieve seizure control and that it seems best to avoid polytherapy if possible. They stated that adequately powered population studies are needed.

Based on the results of 16 RCTs with 1468 participants, 15 conducted in China and one in Norway, authors of this review concluded that the current evidence does not support acupuncture as a treatment for epilepsy. The results of five trials indicated that compared with control treatment, needle acupuncture was not effective decreasing seizure frequency. Two trials compared acupuncture with phenytoin and found that needle acupuncture may be better for achieving at least 75% or at least 25% reduction in seizure frequency. Two trials compared acupuncture with valproate and these indicated that needle acupuncture may be better for achieving at least 50% or at least 75% reduction in seizure frequency, and be associated with better quality of life, lower frequency of impaired concentration, and higher likelihood of at least 70% improvement in epilepsy score.

Poor adherence to anti-epileptic medication is associated with increased morbidity and mortality. This review aimed to determine the effectiveness of interventions intended to improve adherence to medication in adults and children with epilepsy. It included six RCTs of adherence-enhancing interventions. Five studies targeted adults (222 patients in total) and one, children (51 in total). Follow-up times ranged from one to six months. The main types of intervention were educational and behavioural modification and all studies compared an intervention to ‘usual care’. Because the studies differed in intervention type and how adherence was measured the review authors did not pool the studies’ results. They reported that education and counselling interventions had mixed success but behavioural interventions such as the use of intensive reminders and ‘implementation intention’ interventions were more effective. An example of an intensive intervention is keeping a diary of medication use and seizures, using a Dossett medication container (pill organiser) and receiving prescription refill and appointment-keeping reminders. An ‘implementation intervention’ could be getting the patient to complete a simple worksheet linking taking medication with a particular time, place and other routine activity, such as brushing their teeth. The review authors concluded that intensive reminders and ‘implementation intention’ interventions seem promising for enhancing adherence to antiepileptic medications but further research involving well-designed RCTs is needed before a firm conclusion can be reached.


People with epilepsy sometimes have various types of immunological abnormalities such as low serum IgA level, lack of IgG subclass and the presence of antibodies which are pathogenic or secondary to the primary disease. For this reason intravenous immunoglobulin (IVIg) treatment may be beneficial for some people with epilepsy. This review aimed to examine the effects of IVIg, either as alone or as an add-on treatment, on the frequency and duration of seizures, quality of life in people with epilepsy and also the adverse effects of IVIg. The review authors included only one study in the review, with 61 participants aged from two to 51 years. This study was a randomized, double-blind, placebo-controlled, multi-centre trial which compared the treatment efficacy of IVIg as an add-on with placebo add-on in patients with refractory epilepsy over a period of six weeks. This study reported no significant difference between IVIg and placebo in 50% or greater reduction in seizure frequency but it did report a statistically significant effect for global assessment in favour of IVIg. No adverse effects were noted. The review authors stated that no reliable conclusions could be drawn about the efficacy of IVIg as a treatment for epilepsy.


Psychological interventions such as relaxation therapy, cognitive behaviour therapy, bio-feedback and educational interventions have been used alone or in combination in the treatment of epilepsy, with the aim of reducing seizure frequency and improving quality of life. This review aimed to evaluate such interventions. The review authors found only three small trials (with a total of 50 participants) and considered these to have poor methodological quality therefore they did not perform meta-analysis. No study found a significant effect of relaxation therapy on seizure frequency. The review authors concluded that there was no reliable evidence to support the use of psychological treatments.


It is common for people with intellectual disabilities to develop epilepsy. Seizures in intellectually disabled people are often complex and unresponsive to treatment. Antiepileptic drugs (AEDs) may have a profound effect upon behaviour in this patient group. This review aimed to consider RCTs of antiepileptic drug interventions in people with epilepsy and intellectual disabilities. The review authors identified 12 RCTs, with 761 randomised participants in total, comparing eight different pharmacological agents. Due to the heterogeneity between studies the review authors presented a descriptive analysis. They stated that, in general, the AEDs that have been proven effective in the general population are also effective for refractory epilepsy in people with intellectual disability and that it was not possible to comment on the relative efficacy of different medications. Side effects seemed to be similar to those observed in the non intellectually disabled population. Most studies either did not assess behavioural exacerbation or used unreliable measures of it but those that did measure it found little obvious behaviour disorder. The review authors concluded that the evidence broadly supports the use of AEDs to reduce seizure frequency in people with refractory epilepsy and intellectual disability and that side effect are similar to those in the general population, behavioural side effects leading to discontinuation are rare and other effects are under-researched.


People with intellectual disability and epilepsy are more likely than people with epilepsy but no intellectual disability to continue to experience seizures whilst taking one or more antiepileptic drugs. The authors of this review were unable to identify any RCTs of non-pharmacological interventions for people with epilepsy and intellectual disabilities except for one study still in progress. They concluded that there is a need for well-designed RCTs to assess the effect of non-pharmacological interventions on seizure and behavioural outcomes in an intellectually disabled epilepsy population.
Various models of service delivery for children with epilepsy have been developed in response to perceived inadequacies in quality of care but it is unclear what the best kind of service for children with epilepsy is. This review aimed to assess the effectiveness of specialist or dedicated teams or individuals for the care of children with epilepsy compared to usual care services. Studies were eligible for inclusion in this review if they were RCTs, controlled or matched trials, cohort studies or other prospective studies with a control group, or time series studies. Four studies and five reports were included. They reported on four different education and counselling programmes for children, children and parents, or teenagers and parents. All of the programmes showed some benefits for the wellbeing of children with epilepsy but all of the trials had methodological flaws and no single programme was evaluated by more than one study. The review authors noted that the impacts of the programmes were extremely variable and no programme showed benefits across the full range of outcomes. They stated that there were no detrimental effects demonstrated for any programme in the studies and they concluded that the evidence in favour of any single programme is insufficient to make it possible to recommend one programme over another.


Vagus nerve stimulation (VNS) is used as an adjunct treatment for some types of epilepsy. The procedure involves implanting a pacemaker-type device under the skin of the chest and connecting a stimulator wire to the left vagus nerve in the neck. The frequency, intensity and duration of nerve stimulation can be varied by programming the device. This review aimed to determine the effects of high-level VNS compared to low-level (presumed sub-therapeutic dose) VNS in adults and children with drug-resistant partial seizures by reviewing relevant RCTs. The review authors identified two RCTs sponsored by Cyberonics as part of their pre-approval programme for VNS and involving 312 randomised participants. All participants had a stimulator implanted, but the control group received less frequent and lower intensity stimulation. In addition, those in the control group did not receive any electrical current when the device was activated by the hand-held magnet. The outcomes assessed in the studies were: (1) 50% or greater reduction in seizure frequency; (2) treatment withdrawal (any reason); (3) adverse effects. Summary odds ratios (ORs) were estimated for each outcome. The overall odds ratio for 50% responders across all studies was 1.93 (95% CI 1.1 to 3.3), implying that those in the intervention group were almost twice as likely to experience a 50% or greater reduction in seizure frequency. The overall odds ratio for withdrawal for any reason was 1.08 (95% CI 0.97 to 17.51) indicating that those in the intervention group were no more likely to withdraw than those in the control group. The statistically significant adverse effects associated with implantation (compared to baseline) were hoarseness, cough, pain, and paresthesia. Statistically significant adverse effects associated with stimulation (high vs. low) were hoarseness and dyspnea, suggesting the implantation is associated with hoarseness, but the stimulation produces additional hoarseness. From these results the review authors concluded that VNS for partial seizures appears to be effective and well tolerated (since dropouts were rare). They noted that VNS does not produce the side effects typically associated with anti-epilepsy drugs such as ataxia, dizziness, fatigue, nausea, and somnolence.


Convulsive status epilepticus (currently defined as a grand mal convulsion lasting at least 30 minutes) and tonic-clonic (grand mal) convulsions are medical emergencies and demand urgent and appropriate anticonvulsant treatment. First choice drugs in these situations include benzodiazepines, (diazepam, lorazepam, midazolam), phenobarbitone, phenytoin and paraldehyde. This review aimed to review the evidence comparing the efficacy and safety of these drugs for treating children with tonic-clonic convulsions and convulsive status epilepticus in hospital. It included four trials involving 383 participants (all RCTs or quasi-RCTs comparing one drug with another). The main results were as follows: (1) Intravenous lorazepam is as effective as intravenous diazepam in the treatment of acute tonic clonic convulsions, 19/27 (70%) versus 22/34 (65%), RR 1.09 (95% CI 0.77 to 1.54), has fewer adverse events and rectal lorazepam may be more effective than rectal diazepam, 6/6 versus 6/19 (31%), RR 3.17 (95% CI 1.63 to 6.14), (2) Buccal midazolam controlled seizures in 61/109 (56%) compared with 30/110 (27%) of rectal diazepam treated episodes with acute tonic-clonic convulsions, RR 2.05 (95% CI 1.45 to 2.91), (3) Intranasal midazolam is as effective as intravenous diazepam in the treatment of prolonged febrile convulsions, 23/26 (88%) versus 24/26 (92%), RR 0.96 (95% CI 0.8 to 1.14), (4) There is moderate evidence that intranasal lorazepam is more effective than intramuscular paraldehyde for acute tonic-clonic convulsions and patients treated with intranasal lorazepam are significantly less likely to require further anticonvulsants to control continuing seizures, 8/90 (10%) versus 21/80 (26%), RR 0.58 (95% CI 0.42 to 0.79). The review authors concluded that, since the previous Cochrane review on this topic (which included only one study), the situation has changed and there is now evidence that intravenous lorazepam is at least as effective as intravenous diazepam for the treatment of acute tonic-clonic convulsions and is associated with fewer adverse events. They stated that where intravenous access is unavailable there is evidence from one trial that buccal midazolam is the treatment of choice.

This review aimed to assess the evidence regarding the effects of therapeutic drug monitoring upon outcomes in epilepsy. The review authors identified only one relevant RCT, with 180 participants aged six to 65 years who were taking one of a variety of anti-epileptic drugs as monotherapy. Sixty per cent of the patients randomised to therapeutic drug monitoring (intervention group) achieved 12-month remission from seizures as did 61% in the control group. A total of 56% in the intervention group and 58% in the control group were seizure free during the last 12 months of follow up. Adverse effects were reported by 48% in the intervention group and 47% in the control group. Of those randomised to therapeutic drug monitoring, 62% completed the two-year follow up compared with 67% of the control group. The review authors concluded that there was no clear evidence to support routine measurement of serum concentration of antiepileptic drugs with the aim of reaching predefined target ranges in order to optimise treatment of patients with newly-diagnosed epilepsy who are receiving antiepileptic drug monotherapy. They stated that this does not exclude the possibility that therapeutic drug monitoring of specific antiepileptic drugs may be useful during polytherapy, in special situations, or in selected patients, although evidence is lacking.

Other Relevant Publications


This paper reports on a study that reviewed clinical practice guidelines and reported physician practice concerning the management of paediatric status epilepticus in the largest Australian (n=9) and New Zealand (n=2) paediatric emergency departments within the Paediatric Research in Emergency Departments International Collaborative (PREDICT) network. There were seven different guidelines in use in ten of the sites and one site didn’t have a guideline. Initial management of seizure was similar at all sites with benzodiazepines being the first line strategy, consistent with Advanced Paediatric Life Support (APLS) guidelines. There was more variation in the second and third line strategies used for persistent seizures. The authors noted that there is a lack of evidence comparing the efficacy of different second and third line agents and regarding the use or non-use of rapid sequence intubation and that this is reflected in the variations in practice. They considered that further research in this area would be beneficial.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
Cancer

Introduction

In 2010, children and young people aged 0–24 years accounted for 1.5% of all New Zealand cancer registrations, with the most commonly registered cancers being leukaemia and testicular cancer in males and leukaemia and Hodgkin lymphoma in females [84]. During the same period, there were 51 deaths from cancer among children and young people, which accounted for 0.6% of all New Zealand cancer deaths. Amongst males, leukaemia and brain cancers were the most common causes of cancer mortality, while leukaemia was the most common cause for females [84].

Known risk factors for childhood cancer include family history, as the risk doubles if a sibling has been previously diagnosed with a malignancy in childhood. A number of genetic mutations (e.g. RB1 gene mutation which is associated with retinoblastoma) and environmental exposures have also been associated with specific childhood cancers. Examples of environmental risk factors include ionising radiation (e.g. antenatal x-ray exposure), ultraviolet radiation from sunlight (for melanoma and skin cancers) and infectious agents (e.g. Epstein-Barr virus for lymphomas; human papilloma virus for cervical cancer). However, the causes of the vast majority of childhood cancers remain unknown, despite intensive investigation since the mid-20th century [85].

In New Zealand the five year survival ratio for children with cancer is relatively high, although no significant changes in survival occurred between 1998–1999 (0.72) and 2008–2009 (0.80) [86]. Over the longer term, however, there have been significant improvements in child cancer mortality, largely as a result of the intensification of therapy using varying combinations of chemotherapy, radiotherapy, surgery and haematopoietic stem cell transplantation [87]. While these therapies are very successful in preventing death in most cases, the families of children newly diagnosed with cancer can still expect multiple hospital admissions, treatments with severe side effects (including immune suppression and an increased risk of infection), and significant disruption to many aspects of their everyday lives [88].

Fortunately, advances in supportive care have paralleled the intensification of cancer treatments, and careful attention is now paid to the hospital environment (e.g. separate rooms and careful hand washing), antibiotic prophylaxis, immunisation, antiemetics, pain management and the use of blood products [89]. In addition, it is increasingly recognised that the psychosocial care of children and their families is crucially important, and that comprehensive family-centred psychosocial support is necessary at each step in the care pathway, from the initial diagnosis, through the various rounds of treatment, to remission and any recurrence [90].

The following section uses data from the New Zealand Cancer Registry and the National Mortality Collection to review the incidence of, and mortality from, cancer in New Zealand children and young people.
Data Source and Methods

Definition
1. New Zealand Cancer Registry notifications for children and young people aged 0–24 years
2. Cancer deaths for children and young people aged 0–24 years

Data Source
1. New Zealand Cancer Registry

Numerator: NZ Cancer Registry notifications for children and young people aged 0–24 years. Cancer site was assigned using ICD-10-AM as follows: Carcinoma in Situ of Cervix (D06), Melanoma in Situ (D03), Hodgkin disease (C81), Non-Hodgkin Lymphomas (C82–C85), Acute Myeloid Leukaemia (C92.0), Other Myeloid Leukaemias (C92.1–C92.9), Acute Lymphoblastic Leukaemia (C91.0), Other Neoplasms Lymphoid and Haematopoietic Tissues (Remainder C81–C96). Malignant Neoplasms of the: Brain (C71); Testis (C62); Melanoma of Skin (C43); Bone and Cartilage (C40–41); Kidney (Excluding Renal Pelvis) (C64); Adrenal Gland (C74); Ovary (C56); Thyroid (C73); Cervix (C53); Retina (C69.2), Other Malignant Neoplasms (Remainder C00–C97), Other In Situ Neoplasms (Remainder D00–D09), Benign Neoplasms (D10–D36), Neoplasms of Uncertain Behaviour (D37–D48).

2. National Mortality Collection

Numerator: Cancer deaths in children and young people aged 0–24 years where the main underlying cause of death was in the ranges outlined above.


Notes on Interpretation
For the majority of analyses, rates per 100,000 children and young people aged 0–24 years have been used. For cancers of the reproductive organs however, gender-specific denominators have been used (malignant neoplasms of the testis, rates per 100,000 males 0–24 years, malignant neoplasms of the ovaries and cervix, rates are per 100,000 females 0–24 years). For carcinoma in situ of the cervix, rates per 100,000 females 0–24 years have been presented in the NZ and DHB tables to allow comparisons with other cancer types. However, in the rate ratio table, which compares rates by ethnicity, rates per 100,000 females 15–24 years have been presented, as the vast majority of notifications are in this age group.

New Zealand Distribution by Cancer Type

NZ Cancer Registry Notifications
In New Zealand during 2002–2011, acute lymphoblastic leukaemia was the most frequent malignant neoplasm notified to the NZ Cancer Registry in children and young people aged 0–24 years, followed by malignant melanoma of the skin. Carcinoma in situ of the cervix however, was the most frequent reason for a NZ Cancer Registry notification, accounting for 61.3% of all notifications during this period (Table 92).

Cancer Deaths
In New Zealand during 2001–2010, cancers of the brain were the leading cause of cancer mortality in children and young people aged 0–24 years, followed by acute lymphoblastic leukaemia (Table 93).
Table 92. NZ Cancer Registry Notifications for Children and Young People Aged 0–24 Years by Cancer Type, New Zealand 2002–2011

<table>
<thead>
<tr>
<th>Cancer Registry Notifications</th>
<th>Total No. 2002–2011</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Malignant Neoplasms</th>
<th>% of All Notifications</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>New Zealand</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancers of Lymphoid and Haematopoietic Tissues</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>425</td>
<td>42.5</td>
<td>2.85</td>
<td>14.9</td>
<td>5.3</td>
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<tr>
<td>Acute Myeloblastic Leukaemia</td>
<td>107</td>
<td>10.7</td>
<td>0.72</td>
<td>3.7</td>
<td>1.3</td>
</tr>
<tr>
<td>Other Myeloid Leukaemias</td>
<td>52</td>
<td>5.2</td>
<td>0.35</td>
<td>1.8</td>
<td>0.6</td>
</tr>
<tr>
<td>Hodgkin Disease</td>
<td>213</td>
<td>21.3</td>
<td>1.43</td>
<td>7.5</td>
<td>2.7</td>
</tr>
<tr>
<td>Non-Hodgkin Lymphomas</td>
<td>177</td>
<td>17.7</td>
<td>1.19</td>
<td>6.2</td>
<td>2.2</td>
</tr>
<tr>
<td>Other Lymphoid/Haematopoietic Neoplasms</td>
<td>54</td>
<td>5.4</td>
<td>0.36</td>
<td>1.9</td>
<td>0.7</td>
</tr>
<tr>
<td>Cancers of Reproductive Organs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>227</td>
<td>22.7</td>
<td>2.98</td>
<td>7.9</td>
<td>2.8</td>
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<tr>
<td>Malignant Neoplasm of Ovary</td>
<td>53</td>
<td>5.3</td>
<td>0.73</td>
<td>1.9</td>
<td>0.7</td>
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<tr>
<td>Malignant Neoplasm of Cervix</td>
<td>41</td>
<td>4.1</td>
<td>0.56</td>
<td>1.4</td>
<td>0.5</td>
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<tr>
<td>Other Malignant Neoplasms</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Melanoma of Skin</td>
<td>298</td>
<td>29.8</td>
<td>2.00</td>
<td>10.4</td>
<td>3.7</td>
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<tr>
<td>Malignant Neoplasm of Brain</td>
<td>259</td>
<td>25.9</td>
<td>1.74</td>
<td>9.1</td>
<td>3.2</td>
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<tr>
<td>Malignant Neoplasm of Bone and Cartilage</td>
<td>165</td>
<td>16.5</td>
<td>1.11</td>
<td>5.8</td>
<td>2.1</td>
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<td>Malignant Neoplasm of Connective Tissue</td>
<td>121</td>
<td>12.1</td>
<td>0.81</td>
<td>4.2</td>
<td>1.5</td>
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<tr>
<td>Malignant Neoplasm of Thyroid</td>
<td>96</td>
<td>9.6</td>
<td>0.64</td>
<td>3.4</td>
<td>1.2</td>
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<tr>
<td>Malignant Neoplasm of Kidney*</td>
<td>72</td>
<td>7.2</td>
<td>0.48</td>
<td>2.5</td>
<td>0.9</td>
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<td>Malignant Neoplasm of Retina</td>
<td>46</td>
<td>4.6</td>
<td>0.31</td>
<td>1.6</td>
<td>0.6</td>
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<tr>
<td>Malignant Neoplasm of Adrenal Gland</td>
<td>43</td>
<td>4.3</td>
<td>0.29</td>
<td>1.5</td>
<td>0.5</td>
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<tr>
<td>Other Malignant Neoplasms</td>
<td>409</td>
<td>40.9</td>
<td>2.75</td>
<td>14.3</td>
<td>5.1</td>
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<tr>
<td>Total Malignant Neoplasms</td>
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<td>285.8</td>
<td>100.0</td>
<td>35.7</td>
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<tr>
<td>In Situ Neoplasms or Neoplasms of Uncertain Behaviour</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Carcinoma In Situ of Cervix</td>
<td>4,911</td>
<td>491.1</td>
<td>67.39</td>
<td>61.3</td>
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<td>Melanoma In Situ</td>
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<td>15.5</td>
<td>1.04</td>
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<tr>
<td>Other In Situ Neoplasms</td>
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<td>5.0</td>
<td>0.34</td>
<td>0.6</td>
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<tr>
<td>Neoplasm Uncertain/Unknown Behaviour</td>
<td>31</td>
<td>3.1</td>
<td>0.21</td>
<td>0.4</td>
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<tr>
<td>Total In Situ or Uncertain Behaviour</td>
<td>5,147</td>
<td>514.7</td>
<td>64.3</td>
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<td></td>
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<tr>
<td>Total: All Cancer Registry Notifications</td>
<td>8,005</td>
<td>800.5</td>
<td>100.0</td>
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</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: *Malignant Neoplasm of Kidney excluding renal pelvis; Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years)
Table 93. Cancer Deaths in Children and Young People Aged 0–24 Years by Cancer Type, New Zealand 2001–2010

<table>
<thead>
<tr>
<th>Cancer Deaths</th>
<th>Total No. 2001–2010</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Cancer Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand</td>
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<td></td>
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<tr>
<td>Cancers of Lymphoid and Haematopoietic Tissues</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>79</td>
<td>7.9</td>
<td>0.53</td>
<td>13.2</td>
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<td>Acute Myeloblastic Leukaemia</td>
<td>38</td>
<td>3.8</td>
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<td>6.4</td>
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<td>Other Myeloid Leukaemias</td>
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<td>0.6</td>
<td>0.04</td>
<td>1.0</td>
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<td>Hodgkin Disease</td>
<td>9</td>
<td>0.9</td>
<td>0.06</td>
<td>1.5</td>
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<td>Non-Hodgkin Lymphomas</td>
<td>22</td>
<td>2.2</td>
<td>0.15</td>
<td>3.7</td>
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<tr>
<td>Other Lymphoid/Haematopoietic Neoplasms</td>
<td>15</td>
<td>1.5</td>
<td>0.10</td>
<td>2.5</td>
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<td>Cancers of Reproductive Organs</td>
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<td></td>
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<tr>
<td>Malignant Neoplasm of Testis</td>
<td>11</td>
<td>1.1</td>
<td>0.15</td>
<td>1.8</td>
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<tr>
<td>Malignant Neoplasm of Cervix</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
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<tr>
<td>Malignant Neoplasm of Ovary</td>
<td>4</td>
<td>0.4</td>
<td>0.06</td>
<td>0.7</td>
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<tr>
<td>Other Malignant Neoplasms</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
<td>127</td>
<td>12.7</td>
<td>0.86</td>
<td>21.3</td>
</tr>
<tr>
<td>Malignant Neoplasms Bone and Cartilage</td>
<td>73</td>
<td>7.3</td>
<td>0.49</td>
<td>12.2</td>
</tr>
<tr>
<td>Malignant Neoplasm of Connective and Soft Tissue</td>
<td>39</td>
<td>3.9</td>
<td>0.26</td>
<td>6.5</td>
</tr>
<tr>
<td>Malignant Neoplasm of Adrenal Gland</td>
<td>22</td>
<td>2.2</td>
<td>0.15</td>
<td>3.7</td>
</tr>
<tr>
<td>Malignant Melanoma of Skin</td>
<td>17</td>
<td>1.7</td>
<td>0.12</td>
<td>2.8</td>
</tr>
<tr>
<td>Malignant Neoplasm of Kidney*</td>
<td>6</td>
<td>0.6</td>
<td>0.04</td>
<td>1.0</td>
</tr>
<tr>
<td>Other Malignant Neoplasms</td>
<td>110</td>
<td>11.0</td>
<td>0.74</td>
<td>18.4</td>
</tr>
<tr>
<td>Benign Neoplasms or Neoplasms of Uncertain Behaviour</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasm Uncertain/Unknown Behaviour</td>
<td>12</td>
<td>1.2</td>
<td>0.08</td>
<td>2.0</td>
</tr>
<tr>
<td>Benign Neoplasms</td>
<td>6</td>
<td>0.6</td>
<td>0.04</td>
<td>1.0</td>
</tr>
<tr>
<td>Total: All Cancer Deaths</td>
<td>597</td>
<td>59.7</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: *Malignant Neoplasm of Kidney excluding renal pelvis; Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years); s: suppressed due to small numbers

Cancers of the Lymphoid and Haematopoietic Tissues

New Zealand Trends

In New Zealand during 2000–2011, acute lymphoblastic leukaemia made the greatest contribution to NZ Cancer Registry notifications for neoplasms of the lymphoid and haematopoietic tissues, followed by Hodgkin disease. Trends for individual neoplasms in this category varied during this period (Figure 50).

Distribution by Age

In New Zealand during 2002–2011, NZ Cancer Registry notifications for acute lymphoblastic leukaemia increased during infancy, reached a peak at three years of age and then declined, with the highest rates being seen in those aged 2–5 years. In contrast, notifications for Hodgkin disease were more frequent amongst those in their late teens and early twenties (Figure 51).
Figure 50. NZ Cancer Registry Notifications for Cancers of the Lymphoid/Haematopoietic Tissues in Children and Young People Aged 0–24 Years, New Zealand 2000–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Figure 51. NZ Cancer Registry Notifications for Cancers of the Lymphoid/Haematopoietic Tissues in Children and Young People by Age, New Zealand 2002–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Distribution by Prioritised Ethnicity and Gender

In New Zealand during 2002–2011, there were no significant ethnic or gender differences in NZ Cancer Registry notifications for acute lymphoblastic leukaemia, although notifications for Hodgkin disease were significantly higher for European/Other children and young people than for those from other ethnic groups (Table 94).

Table 94. NZ Cancer Registry Notifications for Acute Lymphoblastic Leukaemia and Hodgkin Disease in Children and Young People Aged 0–24 Years by Gender and Ethnicity, New Zealand 2002–2011

<table>
<thead>
<tr>
<th>Variable</th>
<th>Notifications: Total Number 2002–2011</th>
<th>Notifications: Annual Average</th>
<th>Notifications per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>192</td>
<td>19.2</td>
<td>2.63</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>233</td>
<td>23.3</td>
<td>3.06</td>
<td>1.16</td>
<td>0.96–1.41</td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>44</td>
<td>4.4</td>
<td>2.78</td>
<td>0.99</td>
<td>0.72–1.37</td>
</tr>
<tr>
<td>European/Other</td>
<td>242</td>
<td>24.2</td>
<td>2.80</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>85</td>
<td>8.5</td>
<td>2.53</td>
<td>0.90</td>
<td>0.70–1.15</td>
</tr>
<tr>
<td>Pacific</td>
<td>49</td>
<td>4.9</td>
<td>3.72</td>
<td>1.33</td>
<td>0.98–1.80</td>
</tr>
<tr>
<td>Hodgkin Disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>106</td>
<td>10.6</td>
<td>1.45</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>107</td>
<td>10.7</td>
<td>1.41</td>
<td>0.97</td>
<td>0.74–1.26</td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>8</td>
<td>0.8</td>
<td>0.51</td>
<td>0.27</td>
<td>0.13–0.55</td>
</tr>
<tr>
<td>European/Other</td>
<td>161</td>
<td>16.1</td>
<td>1.86</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>28</td>
<td>2.8</td>
<td>0.83</td>
<td>0.45</td>
<td>0.30–0.67</td>
</tr>
<tr>
<td>Pacific</td>
<td>10</td>
<td>1.0</td>
<td>0.76</td>
<td>0.41</td>
<td>0.21–0.77</td>
</tr>
</tbody>
</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Malignant Melanoma and Melanoma In Situ

New Zealand Trends

In New Zealand during 2000–2011, malignant melanoma of the skin was more frequently notified to the NZ Cancer Registry than melanoma in situ. Notification rates for malignant melanoma of the skin were variable during the early 2000s, but became relatively static from 2004–05 onwards (Figure 52).

Distribution by Age

In New Zealand during 2002–2011, NZ Cancer Registry notifications for malignant melanoma and melanoma in situ were infrequent during childhood but increased during adolescence, with the highest rates being seen amongst those in their late teens and early twenties (Figure 53).

Distribution by Prioritised Ethnicity and Gender

In New Zealand during 2002–2011, NZ Cancer Registry notifications for malignant melanoma and melanoma in situ were significantly higher for females and for European/Other children and young people, than for children and young people from other ethnic groups (Table 95).
Figure 52. NZ Cancer Registry Notifications for Malignant Melanoma and Melanoma In Situ in Children and Young People Aged 0–24 Years, New Zealand 2000–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Figure 53. NZ Cancer Registry Notifications for Malignant Melanoma and Melanoma In Situ in Children and Young People by Age, New Zealand 2002–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 95. NZ Cancer Registry Notifications for Malignant Melanoma and Melanoma In Situ in Children and Young People Aged 0–24 Years by Gender and Ethnicity, New Zealand 2002–2011

<table>
<thead>
<tr>
<th>Variable</th>
<th>Notifications: Total Number 2002–2011</th>
<th>Notifications: Annual Average</th>
<th>Notifications per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignant Melanoma and Melanoma In Situ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>268</td>
<td>26.8</td>
<td>3.68</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>185</td>
<td>18.5</td>
<td>2.43</td>
<td>0.66</td>
<td>0.55–0.80</td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>6</td>
<td>0.6</td>
<td>0.38</td>
<td>0.08</td>
<td>0.04–0.19</td>
</tr>
<tr>
<td>European/Other</td>
<td>390</td>
<td>39.0</td>
<td>4.52</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>15</td>
<td>1.5</td>
<td>0.45</td>
<td>0.10</td>
<td>0.06–0.17</td>
</tr>
<tr>
<td>Pacific</td>
<td>3</td>
<td>0.3</td>
<td>0.23</td>
<td>0.05</td>
<td>0.02–0.16</td>
</tr>
</tbody>
</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Cancers of the Cervix and Ovaries

Distribution by Age

In New Zealand during 2002–2011, NZ Cancer Registry notifications for carcinoma in situ of the cervix were relatively infrequent during early adolescence, but increased rapidly thereafter, with the highest rates being seen amongst those in their early twenties. Similarly, the majority of notifications for cancer of the cervix were for those in their early twenties. Notifications for cancers of the ovaries occurred from 11 years of age onwards (Figure 54).

Figure 54. NZ Cancer Registry Notifications for Cancer and Carcinoma In Situ of the Cervix and Cancer of the Ovary in Children and Young People by Age, New Zealand 2002–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Female Population (projected from 2007).
Distribution by Prioritised Ethnicity

In New Zealand during 2002–2011, NZ Cancer Registry notifications for carcinoma in situ of the cervix were significantly higher for European/Other > Māori > Pacific > Asian/Indian women (Table 96). However, caution should be taken when interpreting these figures as it is unclear whether they reflect ethnic differences in the uptake of cervical screening, or the underlying distribution of carcinoma in situ of the cervix in the community.

Table 96. NZ Cancer Registry Notifications for Carcinoma In Situ of the Cervix in Young Women Aged 15–24 Years by Ethnicity, New Zealand 2007–2011

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total Number</th>
<th>Annual Average</th>
<th>Notifications per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian/Indian</td>
<td>45</td>
<td>9.0</td>
<td>20.70</td>
<td>0.10</td>
<td>0.07–0.13</td>
</tr>
<tr>
<td>European/Other</td>
<td>1,845</td>
<td>369.0</td>
<td>208.16</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>512</td>
<td>102.4</td>
<td>166.50</td>
<td>0.80</td>
<td>0.73–0.88</td>
</tr>
<tr>
<td>Pacific</td>
<td>48</td>
<td>9.6</td>
<td>37.78</td>
<td>0.18</td>
<td>0.14–0.24</td>
</tr>
</tbody>
</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Female Population (projected from 2007)

Other Cancers

New Zealand Trends

In New Zealand during 2000–2011, trends for different cancer types varied (Figure 55).

Distribution by Age

In New Zealand during 2002–2011, NZ Cancer Registry notifications for cancers of the retina were more frequent for those under four years of age, while cancers of the brain were more evenly distributed throughout childhood and adolescence. Cancers of the bone and cartilage were more common after 6 years of age, with the highest rates being seen amongst those in their late teens (Figure 56).

Similarly, notifications for cancers of the kidney and adrenal were more common amongst those under eight years of age, while notifications for cancers of the thyroid were more frequent amongst those in their late teens and early twenties (Figure 57).

Distribution by Prioritised Ethnicity and Gender

In New Zealand during 2002–2011, there were no significant gender differences in NZ Cancer Registry notifications for cancers of the brain, although rates were significantly lower for Asian/Indian children and young people, than for European/Other and Pacific children and young people (Table 97).
Figure 55. NZ Cancer Registry Notifications for Selected Other Cancers in Children and Young People Aged 0–24 Years, New Zealand 2000–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: *Malignant Neoplasm of Kidney excludes renal pelvis

Figure 56. NZ Cancer Registry Notifications for Cancers of the Brain, Retina and Bone and Cartilage in Children and Young People by Age, New Zealand 2002–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Figure 57. NZ Cancer Registry Notifications for Cancers of the Adrenal, Kidney and Thyroid in Children and Young People by Age, New Zealand 2002–2011

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: *Malignant Neoplasm of Kidney excludes renal pelvis

Table 97. NZ Cancer Registry Notifications for Cancers of the Brain in Children and Young People Aged 0–24 Years by Gender and Ethnicity, New Zealand 2002–2011

<table>
<thead>
<tr>
<th>Variable</th>
<th>Notifications: Total Number 2002–2011</th>
<th>Notifications: Annual Average</th>
<th>Notifications per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>126</td>
<td>12.6</td>
<td>1.73</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>133</td>
<td>13.3</td>
<td>1.75</td>
<td>1.01</td>
<td>0.79–1.29</td>
</tr>
<tr>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>12</td>
<td>1.2</td>
<td>0.76</td>
<td>0.40</td>
<td>0.22–0.72</td>
</tr>
<tr>
<td>European/Other</td>
<td>163</td>
<td>16.3</td>
<td>1.89</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>50</td>
<td>5.0</td>
<td>1.49</td>
<td>0.79</td>
<td>0.57–1.08</td>
</tr>
<tr>
<td>Pacific</td>
<td>33</td>
<td>3.3</td>
<td>2.50</td>
<td>1.33</td>
<td>0.91–1.93</td>
</tr>
</tbody>
</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population.

Northern DHBs Distribution

Cancer Registry Notifications

In all four Northern DHBs during 2002–2011, acute lymphoblastic leukaemia was the most frequent malignant neoplasm notified to the NZ Cancer Registry in children and young people aged 0–24 years. Other frequently notified malignant neoplasms included non-Hodgkin lymphomas, malignant melanomas, and malignant neoplasms of the testis and brain. Carcinoma in situ of the cervix however, was the most frequent reason for a NZ Cancer Registry notification in all four DHBs (Table 98–Table 101).
Table 98. NZ Cancer Registry Notifications for Children and Young People Aged 0–24 Years by Cancer Type, Northland 2002–2011

<table>
<thead>
<tr>
<th>Cancer Registry Notifications</th>
<th>Total No. 2002–2011</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Malignant Neoplasms</th>
<th>% of All Notifications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cancers of Lymphoid and Haematopoietic Tissues</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>16</td>
<td>1.6</td>
<td>2.98</td>
<td>16.5</td>
<td>9.8</td>
</tr>
<tr>
<td>Acute Myeloblastic Leukaemia</td>
<td>7</td>
<td>0.7</td>
<td>1.31</td>
<td>7.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Non-Hodgkin Lymphomas</td>
<td>10</td>
<td>1.0</td>
<td>1.86</td>
<td>10.3</td>
<td>6.1</td>
</tr>
<tr>
<td>Other Neoplasms Lymphoid/Haematopoietic Tissues</td>
<td>5</td>
<td>0.5</td>
<td>0.93</td>
<td>5.2</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>10</td>
<td>1.0</td>
<td>3.63</td>
<td>10.3</td>
<td>6.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Ovary</td>
<td>5</td>
<td>0.5</td>
<td>1.92</td>
<td>5.2</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Melanoma of Skin</td>
<td>5</td>
<td>0.5</td>
<td>0.93</td>
<td>5.2</td>
<td>3.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
<td>10</td>
<td>1.0</td>
<td>1.86</td>
<td>10.3</td>
<td>6.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Bone and Cartilage</td>
<td>7</td>
<td>0.7</td>
<td>1.31</td>
<td>7.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Other Malignant Neoplasms</td>
<td>22</td>
<td>2.2</td>
<td>4.10</td>
<td>22.7</td>
<td>13.5</td>
</tr>
<tr>
<td>Total Malignant Neoplasms</td>
<td>97</td>
<td>9.7</td>
<td>100.0</td>
<td></td>
<td>59.5</td>
</tr>
<tr>
<td><strong>In Situ Neoplasms or Neoplasms of Uncertain Behaviour</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carcinoma In Situ of Cervix</td>
<td>62</td>
<td>6.2</td>
<td>23.77</td>
<td></td>
<td>38.0</td>
</tr>
<tr>
<td>Other In Situ Neoplasms</td>
<td>3</td>
<td>0.3</td>
<td>0.56</td>
<td></td>
<td>1.8</td>
</tr>
<tr>
<td>Neoplasm Uncertain/Unknown Behaviour</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td></td>
<td>s</td>
</tr>
<tr>
<td>Total In Situ or Uncertain Behaviour</td>
<td>66</td>
<td>6.6</td>
<td></td>
<td></td>
<td>40.5</td>
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<tr>
<td>Total Cancer Registry Notifications</td>
<td>163</td>
<td>16.3</td>
<td></td>
<td></td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years); s: suppressed due to small numbers
Table 99. NZ Cancer Registry Notifications for Children and Young People Aged 0–24 Years by Cancer Type, Waitemata 2002–2011

<table>
<thead>
<tr>
<th>Cancer Registry Notifications</th>
<th>Total No. 2002–2011</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Malignant Neoplasms</th>
<th>% of All Notifications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cancers of Lymphoid and Haematopoietic Tissues</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>56</td>
<td>5.6</td>
<td>3.12</td>
<td>17.0</td>
<td>6.3</td>
</tr>
<tr>
<td>Acute Myeloblastic Leukaemia</td>
<td>10</td>
<td>1.0</td>
<td>0.56</td>
<td>3.0</td>
<td>1.1</td>
</tr>
<tr>
<td>Other Myeloid Leukaemias</td>
<td>4</td>
<td>0.4</td>
<td>0.22</td>
<td>1.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Hodgkin Disease</td>
<td>35</td>
<td>3.5</td>
<td>1.95</td>
<td>10.6</td>
<td>3.9</td>
</tr>
<tr>
<td>Non-Hodgkin Lymphomas</td>
<td>19</td>
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<td>1.06</td>
<td>5.8</td>
<td>2.1</td>
</tr>
<tr>
<td>Other Neoplasms Lymphoid/Haematopoietic Tissues</td>
<td>8</td>
<td>0.8</td>
<td>0.45</td>
<td>2.4</td>
<td>0.9</td>
</tr>
<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>28</td>
<td>2.8</td>
<td>3.04</td>
<td>8.5</td>
<td>3.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Ovary</td>
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<td>0.46</td>
<td>1.2</td>
<td>0.4</td>
</tr>
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<td></td>
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</tr>
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<td>34</td>
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<td>1.89</td>
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<td>3.8</td>
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<td>Malignant Neoplasm of Brain</td>
<td>31</td>
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<td>1.73</td>
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<td>3.5</td>
</tr>
<tr>
<td>Malignant Neoplasm of Bone and Cartilage</td>
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<td>0.72</td>
<td>3.9</td>
<td>1.5</td>
</tr>
<tr>
<td>Malignant Neoplasm Connective/Soft Tissue</td>
<td>7</td>
<td>0.7</td>
<td>0.39</td>
<td>2.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Malignant Neoplasm of Thyroid</td>
<td>10</td>
<td>1.0</td>
<td>0.56</td>
<td>3.0</td>
<td>1.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Kidney*</td>
<td>7</td>
<td>0.7</td>
<td>0.39</td>
<td>2.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Malignant Neoplasm of Retina</td>
<td>10</td>
<td>1.0</td>
<td>0.56</td>
<td>3.0</td>
<td>1.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Adrenal Gland</td>
<td>9</td>
<td>0.9</td>
<td>0.50</td>
<td>2.7</td>
<td>1.0</td>
</tr>
<tr>
<td>Other Malignant Neoplasms</td>
<td>39</td>
<td>3.9</td>
<td>2.17</td>
<td>11.8</td>
<td>4.4</td>
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<td>330</td>
<td>33.0</td>
<td>100.0</td>
<td>37.1</td>
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</tr>
</tbody>
</table>

| **In Situ Neoplasms or Neoplasms of Uncertain Behaviour**          |                     |                |                  |                          |                        |
| Carcinoma In Situ of Cervix                                       | 537                 | 53.7           | 61.29            | 60.4                     |                        |
| Melanoma In Situ                                                  | 12                  | 1.2            | 0.67             | 1.3                      |                        |
| Other In Situ Neoplasms                                           | 8                   | 0.8            | 0.45             | 0.9                      |                        |
| Neoplasm Uncertain/Unknown Behaviour                               | <3                  | s              | s                | s                       |                        |
| Total In Situ or Uncertain Behaviour                               | 559                 | 55.9           |                  | 62.9                     |                        |
| Total Cancer Registry Notifications                                | 889                 | 88.9           |                  | 100.0                    |                        |

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: * Malignant Neoplasm of Kidney excluding renal pelvis; Rates are per 100,000 0–24 years, except for gender-specific cancers of reproductive organs (i.e. per 100,000 males or females 0–24 years); s: suppressed (small numbers)
### Table 100. NZ Cancer Registry Notifications for Children and Young People Aged 0–24 Years by Cancer Type, Auckland DHB 2002–2011

<table>
<thead>
<tr>
<th>Cancer Registry Notifications</th>
<th>Total No. 2002–2011</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Malignant Neoplasms</th>
<th>% of All Notifications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cancers of Lymphoid and Haematopoietic Tissues</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>36</td>
<td>3.6</td>
<td>2.44</td>
<td>11.7</td>
<td>5.3</td>
</tr>
<tr>
<td>Acute Myeloblastic Leukaemia</td>
<td>9</td>
<td>0.9</td>
<td>0.61</td>
<td>2.9</td>
<td>1.3</td>
</tr>
<tr>
<td>Other Myeloid Leukaemias</td>
<td>8</td>
<td>0.8</td>
<td>0.54</td>
<td>2.6</td>
<td>1.2</td>
</tr>
<tr>
<td>Hodgkin Disease</td>
<td>23</td>
<td>2.3</td>
<td>1.56</td>
<td>7.4</td>
<td>3.4</td>
</tr>
<tr>
<td>Non-Hodgkin Lymphomas</td>
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<td>1.22</td>
<td>5.8</td>
<td>2.7</td>
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<tr>
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<td>0.6</td>
<td>0.41</td>
<td>1.9</td>
<td>0.9</td>
</tr>
<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>25</td>
<td>2.5</td>
<td>3.35</td>
<td>8.1</td>
<td>3.7</td>
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<tr>
<td>Malignant Neoplasm of Ovary</td>
<td>4</td>
<td>0.4</td>
<td>0.55</td>
<td>1.3</td>
<td>0.6</td>
</tr>
<tr>
<td>Malignant Neoplasm of Cervix</td>
<td>3</td>
<td>0.3</td>
<td>0.41</td>
<td>1.0</td>
<td>0.4</td>
</tr>
<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Melanoma of Skin</td>
<td>32</td>
<td>3.2</td>
<td>2.17</td>
<td>10.4</td>
<td>4.7</td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
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<td>2.5</td>
<td>1.70</td>
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<td>3.7</td>
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<tr>
<td>Malignant Neoplasm of Bone and Cartilage</td>
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<td>2.0</td>
<td>1.36</td>
<td>6.5</td>
<td>3.0</td>
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<tr>
<td>Malignant Neoplasm Connective/Soft Tissue</td>
<td>11</td>
<td>1.1</td>
<td>0.75</td>
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<td>1.6</td>
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<tr>
<td>Malignant Neoplasm of Thyroid</td>
<td>12</td>
<td>1.2</td>
<td>0.82</td>
<td>3.9</td>
<td>1.8</td>
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<tr>
<td>Malignant Neoplasm of Kidney*</td>
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<td>0.48</td>
<td>2.3</td>
<td>1.0</td>
</tr>
<tr>
<td>Malignant Neoplasm of Retina</td>
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<td>0.5</td>
<td>0.34</td>
<td>1.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Malignant Neoplasm of Adrenal Gland</td>
<td>6</td>
<td>0.6</td>
<td>0.41</td>
<td>1.9</td>
<td>0.9</td>
</tr>
<tr>
<td>Other Malignant Neoplasms</td>
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<td>5.9</td>
<td>4.01</td>
<td>19.1</td>
<td>8.7</td>
</tr>
<tr>
<td>Total Malignant Neoplasms</td>
<td>309</td>
<td>30.9</td>
<td>100.0</td>
<td>45.8</td>
<td></td>
</tr>
</tbody>
</table>

| **In Situ Neoplasms or Neoplasms of Uncertain Behaviour** | | | | | |
| Carcinoma In Situ of Cervix    | 346| 34.6| 47.63| 51.3| |
| Melanoma In Situ               | 12 | 1.2 | 0.82 | 1.8 | |
| Other In Situ Neoplasms        | 4  | 0.4 | 0.27 | 0.6 | |
| Neoplasm Uncertain/Unknown Behaviour | 4  | 0.4 | 0.27 | 0.6 | |
| Total In Situ or Uncertain Behaviour | 366| 36.6| 54.2| |
| Total Cancer Registry Notifications | 675| 67.5| 100.0| | |

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: * Malignant Neoplasm of Kidney excluding renal pelvis; Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years)
Table 101. NZ Cancer Registry Notifications for Children and Young People Aged 0–24 Years by Cancer Type, Counties Manukau 2002–2011

<table>
<thead>
<tr>
<th>Cancer Registry Notifications</th>
<th>Total No. 2002–2011</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Malignant Neoplasms</th>
<th>% of All Notifications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cancers of Lymphoid and Haematopoietic Tissues</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>50</td>
<td>5.0</td>
<td>2.69</td>
<td>16.6</td>
<td>10.5</td>
</tr>
<tr>
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<td>0.38</td>
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<tr>
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<td>0.54</td>
<td>3.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Hodgkin Disease</td>
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<td>1.5</td>
<td>0.81</td>
<td>5.0</td>
<td>3.2</td>
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<tr>
<td>Non-Hodgkin Lymphomas</td>
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<td>2.4</td>
<td>1.29</td>
<td>7.9</td>
<td>5.1</td>
</tr>
<tr>
<td>Other Neoplasms Lymphoid/Haematopoietic Tissues</td>
<td>11</td>
<td>1.1</td>
<td>0.59</td>
<td>3.6</td>
<td>2.3</td>
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<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>12</td>
<td>1.2</td>
<td>1.27</td>
<td>4.0</td>
<td>2.5</td>
</tr>
<tr>
<td>Malignant Neoplasm of Ovary</td>
<td>6</td>
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<td>0.66</td>
<td>2.0</td>
<td>1.3</td>
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<tr>
<td>Malignant Neoplasm of Cervix</td>
<td>3</td>
<td>0.3</td>
<td>0.33</td>
<td>1.0</td>
<td>0.6</td>
</tr>
<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Melanoma of Skin</td>
<td>14</td>
<td>1.4</td>
<td>0.75</td>
<td>4.6</td>
<td>2.9</td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
<td>31</td>
<td>3.1</td>
<td>1.67</td>
<td>10.3</td>
<td>6.5</td>
</tr>
<tr>
<td>Malignant Neoplasm of Bone and Cartilage</td>
<td>31</td>
<td>3.1</td>
<td>1.67</td>
<td>10.3</td>
<td>6.5</td>
</tr>
<tr>
<td>Malignant Neoplasm Connective/Soft Tissue</td>
<td>11</td>
<td>1.1</td>
<td>0.59</td>
<td>3.6</td>
<td>2.3</td>
</tr>
<tr>
<td>Malignant Neoplasm of Thyroid</td>
<td>10</td>
<td>1.0</td>
<td>0.54</td>
<td>3.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Malignant Neoplasm of Kidney*</td>
<td>11</td>
<td>1.1</td>
<td>0.59</td>
<td>3.6</td>
<td>2.3</td>
</tr>
<tr>
<td>Malignant Neoplasm of Retina</td>
<td>3</td>
<td>0.3</td>
<td>0.16</td>
<td>1.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Malignant Neoplasm of Adrenal Gland</td>
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<td>0.3</td>
<td>0.16</td>
<td>1.0</td>
<td>0.6</td>
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<tr>
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<td>5.0</td>
<td>2.69</td>
<td>16.6</td>
<td>10.5</td>
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<td>100.0</td>
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<tr>
<td><strong>In Situ Neoplasms or Neoplasms of Uncertain Behaviour</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Carcinoma In Situ of Cervix</td>
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<tr>
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</tr>
<tr>
<td>Other In Situ Neoplasms</td>
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<td>0.27</td>
<td>1.1</td>
<td></td>
</tr>
<tr>
<td>Neoplasm Uncertain/Unknown Behaviour</td>
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<td>0.32</td>
<td>1.3</td>
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</tr>
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</table>

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: * Malignant Neoplasm of Kidney excluding renal pelvis; Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years)
Cancer Deaths

In the Northern DHBs during 2001–2010, cancers of the brain, cancers of bone and cartilage and acute lymphoblastic leukaemia were the leading causes of cancer-related mortality in children and young people aged 0–24 years (Table 102, Table 103).

Table 102. Cancer Deaths in Children and Young People Aged 0–24 Years by Cancer Type, Northland and Waitemata DHBs 2001–2010

<table>
<thead>
<tr>
<th>Cancer Deaths</th>
<th>Total No. 2001–2010</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Cancer Deaths</th>
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<tr>
<td><strong>Northland</strong></td>
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<td></td>
</tr>
<tr>
<td>Cancers of Lymphoid and Haematopoietic Tissues</td>
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<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>7</td>
<td>0.7</td>
<td>1.31</td>
<td>24.1</td>
</tr>
<tr>
<td>Other Lymphoid/Haematopoietic Neoplasms</td>
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<td>0.3</td>
<td>0.56</td>
<td>10.3</td>
</tr>
<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
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<td>Malignant Neoplasms Bone and Cartilage</td>
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<td>13.8</td>
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<tr>
<td>Other Malignant Neoplasms</td>
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<td>0.7</td>
<td>1.31</td>
<td>24.1</td>
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<td></td>
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</tr>
<tr>
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<td>s</td>
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<td>2.9</td>
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</tr>
<tr>
<td><strong>Waitemata</strong></td>
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<tr>
<td>Cancers of Lymphoid and Haematopoietic Tissues</td>
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<td></td>
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</tr>
<tr>
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<td>0.8</td>
<td>0.45</td>
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<td>0.23</td>
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<td>Other Lymphoid/Haematopoietic Neoplasms</td>
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<td>0.17</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
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<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
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<td>Malignant Neoplasm of Brain</td>
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<td>0.79</td>
<td>24.1</td>
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<tr>
<td>Malignant Neoplasms Bone and Cartilage</td>
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<td>0.8</td>
<td>0.45</td>
<td>13.8</td>
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<tr>
<td>Malignant Neoplasm of Adrenal Gland</td>
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<td>0.3</td>
<td>0.17</td>
<td>5.2</td>
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<tr>
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<td><strong>Benign Neoplasms or Neoplasms of Uncertain Behaviour</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Benign Neoplasms</td>
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<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Total: All Cancer Deaths</strong></td>
<td>58</td>
<td></td>
<td>5.8</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years); s: suppressed due to small numbers
Table 103. Cancer Deaths in Children and Young People Aged 0–24 Years by Cancer Type, Auckland and Counties Manukau DHBs 2001–2010

<table>
<thead>
<tr>
<th>Cancer Deaths</th>
<th>Total No. 2001–2010</th>
<th>Annual Average</th>
<th>Rate per 100,000</th>
<th>% of Cancer Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Auckland DHB</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cancers of Lymphoid and Haematopoietic Tissues</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>7</td>
<td>0.7</td>
<td>0.48</td>
<td>11.7</td>
</tr>
<tr>
<td>Other Leukaemias</td>
<td>5</td>
<td>0.5</td>
<td>0.34</td>
<td>8.3</td>
</tr>
<tr>
<td>Hodgkin and Non-Hodgkin Lymphomas</td>
<td>4</td>
<td>0.4</td>
<td>0.27</td>
<td>6.7</td>
</tr>
<tr>
<td>Other Lymphoid/Haematopoietic Neoplasms</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Ovary</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
<td>18</td>
<td>1.8</td>
<td>1.23</td>
<td>30.0</td>
</tr>
<tr>
<td>Malignant Neoplasm of Connective and Soft Tissue</td>
<td>3</td>
<td>0.3</td>
<td>0.21</td>
<td>5.0</td>
</tr>
<tr>
<td>Other Malignant Neoplasms</td>
<td>17</td>
<td>1.7</td>
<td>1.17</td>
<td>28.3</td>
</tr>
<tr>
<td><strong>Benign Neoplasms or Neoplasms of Uncertain Behaviour</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasm Uncertain/Unknown Behaviour</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Benign Neoplasms</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total: All Cancer Deaths</td>
<td>60</td>
<td>6.0</td>
<td></td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Counties Manukau</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cancers of Lymphoid and Haematopoietic Tissues</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute Lymphoblastic Leukaemia</td>
<td>8</td>
<td>0.8</td>
<td>0.44</td>
<td>12.1</td>
</tr>
<tr>
<td>Acute Myeloblastic Leukaemia</td>
<td>5</td>
<td>0.5</td>
<td>0.28</td>
<td>7.6</td>
</tr>
<tr>
<td>Hodgkin and Non-Hodgkin Lymphomas</td>
<td>4</td>
<td>0.4</td>
<td>0.22</td>
<td>6.1</td>
</tr>
<tr>
<td>Other Lymphoid/Haematopoietic Neoplasms</td>
<td>3</td>
<td>0.3</td>
<td>0.17</td>
<td>4.5</td>
</tr>
<tr>
<td><strong>Cancers of Reproductive Organs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Testis</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td><strong>Other Malignant Neoplasms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant Neoplasm of Brain</td>
<td>9</td>
<td>0.9</td>
<td>0.50</td>
<td>13.6</td>
</tr>
<tr>
<td>Malignant Neoplasms Bone and Cartilage</td>
<td>15</td>
<td>1.5</td>
<td>0.83</td>
<td>22.7</td>
</tr>
<tr>
<td>Malignant Neoplasm of Connective and Soft Tissue</td>
<td>5</td>
<td>0.5</td>
<td>0.28</td>
<td>7.6</td>
</tr>
<tr>
<td>Other Malignant Neoplasms</td>
<td>13</td>
<td>1.3</td>
<td>0.72</td>
<td>19.7</td>
</tr>
<tr>
<td><strong>Benign Neoplasms or Neoplasms of Uncertain Behaviour</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasm Uncertain/Unknown Behaviour</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>Total: All Cancer Deaths</td>
<td>66</td>
<td>6.6</td>
<td></td>
<td>100.0</td>
</tr>
</tbody>
</table>

Source: Numerator: National Mortality Collection; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Rates are per 100,000 0–24 years, except for cancers of reproductive organs which are gender-specific (i.e. per 100,000 males or females 0–24 years); s: suppressed due to small numbers
Local Policy Documents and Evidence-Based Reviews Relevant to Cancer in Children and Young People

In New Zealand, a small number of policy documents are relevant to the management of children and young people with cancer and these are reviewed in Table 104, along with a range of international guidelines and reviews which consider these issues in the overseas context. Note: The efficacy of particular therapeutic agents in the management of specific types of cancer in children and young people is beyond the scope of this review, with the focus of the table below being on broader management principles only.

Table 104. Local Policy Documents and Evidence-Based Reviews Relevant to Cancer in Children and Young People

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>This guidance was commissioned by the Ministry of Health to help improve the integration of palliative care service delivery to children and young people in New Zealand, especially to those who live outside Auckland (where many specialist services for children are concentrated). It aims to provide a starting point starting point for the development of paediatric palliative care services, and to act as a resource. It examines paediatric palliative care services, both internationally and in New Zealand and, and uses the insights gained as the basis for a proposed framework for the development of a coherent, integrated and co-ordinated system of paediatric palliative care service delivery.</td>
</tr>
<tr>
<td>Children and young people receiving cancer treatment are especially vulnerable to illnesses such as measles because chemotherapy greatly reduces their immunity and means their previous vaccinations are no longer effective. They cannot be re-vaccinated during their treatment because the measles vaccine contains live virus. Measles has around a 50% fatality rate in children with low immunity. This booklet contains the stories of four young people with cancer and the impact the 2011 Auckland measles outbreak has had on them and their families plus viewpoints from two paediatric oncologists. The booklet stresses the point that the whole community needs to be immunised to protect these children.</td>
</tr>
<tr>
<td>The National Child Cancer Services Plan was developed with the aim of strengthening services by achieving national agreement on the service delivery model for child cancer services. It recognises the challenges presented by New Zealand’s relatively small and dispersed population. The plan recommends that there be two specialist paediatric oncology centres, at Starship Children's Hospital in Auckland DHB and Christchurch Hospital in Canterbury DHB and that these centres make shared care arrangements with other DHBs. This service arrangement is considered to achieve the best balance between the need for access for families and whānau, and the need for consolidation to support a scarce paediatric oncology workforce and best clinical practice.</td>
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New Zealand Guidelines

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<th>New Zealand Guidelines</th>
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<tr>
<td>This guideline is intended for primary care practitioners to help them to recognise the signs and symptoms suggestive of a cancer diagnosis, make a timely referral to specialist services when cancer is suspected, and be aware of the investigations that may be appropriate to undertake in the primary care setting. Chapter 14 deals with cancer in children and young people. It provides general recommendations for the specific cancers leukaemia, lymphoma, brain and central nervous system (CNS) tumours, neuroblastoma, Wilms’ tumour, soft tissue sarcoma, bone sarcomas and retinoblastoma. A brief 2-page summary of key evidence-based recommendations and good practice points for selected site-specific cancers can be found here: <a href="http://www.health.govt.nz/system/files/documents/publications/suspected_cancer.pdf">http://www.health.govt.nz/system/files/documents/publications/suspected_cancer.pdf</a></td>
</tr>
<tr>
<td>This guideline draws on the 2005 NICE guideline <strong>Clinical Guideline 27: Referral Guidelines for Suspected Cancer</strong>, which can be found at <a href="http://guidance.nice.org.uk/CG27/Guidance">http://guidance.nice.org.uk/CG27/Guidance</a>. The section on ovarian cancer was updated in 2011.</td>
</tr>
</tbody>
</table>
Chapter 25 in this guideline provides brief guidance on melanoma in children and Chapter 26, on melanoma and pregnancy, including hormone replacement therapy and oral contraceptives.

**International Guidelines**


This paper reports on an international initiative to promote collaboration and avoid duplication of effort in the development of clinical practice guidelines for long-term follow-up of childhood and young adult cancer survivors, known as the International Late Effects of Childhood Cancer Guideline Harmonization Group. It states that currently there are four such guidelines, independently developed and published by the North American Children’s Oncology Group (2008, below), the Dutch Childhood Oncology Group (2008), the United Kingdom’s Children’s Cancer and Leukaemia Group (2005 [http://cclg.org.uk/dynamic_files/LTFU-full.pdf] and the Scottish Intercollegiate Guidelines Network (2013, below).


The five-year survival rate for child cancer is now around 80% and this has led to an increasing population of adult survivors of childhood cancer. These survivors are at risk of premature death and a range of physical and psychosocial problems as a result of their cancer and its treatment. Lifelong follow-up is considered to be best practice. This guideline is an update and substantial revision of an earlier SIGN guideline. It is intended for primary care staff and those who work in long-term follow-up clinics. It provides recommendations based on current evidence for best practice in identification, assessment and management of late effects in childhood cancer survivors. The recommendations are graded according to the strength of the supporting evidence (not their clinical importance) and grouped under the following headings: subsequent primary cancers, fertility issues, cardiac effects, bone health and metabolic syndrome.


This guideline is an update of the 2006 American Society of Clinical Oncology (ASCO) guideline. It addresses four clinical questions: (1) Are patients with cancer interested in interventions to preserve fertility? (2) What is the quality of evidence supporting current and forthcoming options for preservation of fertility in males? (3) What is the quality of evidence supporting current and forthcoming options for preservation of fertility in females? (4) What is the role of the oncologist in advising patients about fertility preservation options? The guideline also gives special attention to the fertility preservation needs of children. It is based on a systematic review of the literature published from March 2006 through to January 2013. In total there were 222 new publications meeting the review’s inclusion criteria: 18 RCTs, 16 systematic reviews, meta-analyses or previous guidelines and many narrative reviews, case series, case studies and editorials. The guidelines recommend that, where infertility is a potential risk of cancer therapy, fertility preservation should be discussed with all patients of reproductive age and with the parents or guardians of children and adolescents, and a referral to a reproductive specialist should be offered. They state that, at present, fertility preservation (sperm cryopreservation and oocyte cryopreservation) is an established technology only for post-pubertal individuals and that methods for pre-pubertal children are still experimental.


Antineoplastic-induced nausea and vomiting (ANIV) is a common problem for children being treated for cancer. This guideline deals with prevention of ANIV in the acute phase (occurring within 24 hours of administration of an antineoplastic agent). It is intended for healthcare personnel who care for children aged 1 month to 18 years who are receiving antineoplastic medication. It is based on a review of existing guidelines and a comprehensive literature search for relevant primary pediatric studies and adapted from two existing guidelines primarily focussed on adult patients:


Meta-analysis of primary study results was performed when feasible. For each of the six clinical questions addressed there is a summary table giving a list of recommendations. Each recommendation is accompanied by an indication of the strength of the recommendation and the quality of the evidence. Where there were suitable relevant studies identified, there is also a table giving a summary of the results of synthesis of studies.

A brief summary of the guideline can be found here: [http://www.pogo.ca/_media/Files/guidelines/POGO_Acute_AINV_Guideline_Quick_Summary_Feb_21_2013.pdf]

This Canadian guideline provides detailed recommendations for influenza immunisation in adult and paediatric patients with cancer and also a discussion of the research evidence. The guideline states that, in general, there is scant evidence from well-controlled studies on influenza immunisation in adult and child cancer patients and that recommendations are based, in part, on data extrapolated from healthy populations together with the best practices and opinions of experts in Alberta. Appendix B provides tables summarising the research evidence. These guidelines recommend that all paediatric cancer patients aged six months or older should have annual administration of the inactivated influenza vaccine. They strongly recommend annual influenza immunisation of family members, out-of-home caregivers, and hospital or clinic staff in contact with child cancer patients.

<table>
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<tr>
<td>Neutropenic sepsis is a potentially fatal complication of cancer treatment, particularly chemotherapy. It has a higher mortality in children and young people than in adults. It occurs when a person whose natural defences against infection (the white blood cells produced by the bone marrow) have been suppressed by chemotherapy or radiotherapy acquire an infection that their body cannot fight off. This guideline aims to improve management of this complication by providing evidence-based recommendations for prevention, identification and management. It is an abbreviated version of the full guideline:</td>
</tr>
<tr>
<td>Within these guidelines are a series of clinical questions relating to key clinical issues. For each question there is a summary of the research evidence (relating to both clinical and cost effectiveness) and recommendations. Where this was possible data from multiple studies was combined in a meta-analysis and synthesised into a GRADE &quot;evidence profile&quot; (for more information on this process, see <a href="http://www.gradeworkinggroup.org/">http://www.gradeworkinggroup.org/</a>). The evidence profile shows, for each outcome, an overall assessment of both the quality of the evidence as a whole (low, moderate or high) as well as an estimate of the size of effect. For each question there is also a LETR (Linking Evidence to Recommendations) statement which is intended to explain to the reader how the final recommendations were arrived at. The LETR statement usually covers: the relative value placed on the outcomes considered; the strength of evidence about benefits and harms for the intervention being considered; the costs and cost-effectiveness of an intervention; the quality of the evidence (see GRADE); the degree of consensus within the Guideline Development Group; and other considerations such as equalities issues.</td>
</tr>
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</table>

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<tbody>
<tr>
<td>Febrile neutropenia (FN, fever in association with a low white blood cell count) is a common complication in children receiving cancer treatment. These guidelines were produced by The International Pediatric Fever and Neutropenia Guideline Panel which included representatives from oncology, infectious disease, nursing, and pharmacy, as well as a patient advocate, from 10 different countries. They address a number of clinical questions related to assessing the level of risk associated with an episode of FN, on-going management of FN from 24 to 72 hours after initiation of empiric antibacterial treatment, and empiric antifungal treatment more than 96 hours after initiation of empiric antibacterial treatment. For each question members of the panel undertook a literature review and used the Grades of Recommendation Assessment, Development, and Evaluation (GRADE) approach to generate summaries and classify the evidence as high, moderate, low, or very low based on methodological considerations. Each recommendation is accompanied by grades indicating the strength of the recommendation and the quality of the evidence.</td>
</tr>
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<tbody>
<tr>
<td>This guidance is intended for health sector personnel and others who have a role in, and responsibility for, preventing skin cancer, for example GPs, pharmacists, practice nurses, school nurses and those responsible for employee health and wellbeing. The recommendations in the guidance aim to raise awareness and maintain knowledge about the risks of UV exposure and to influence attitudes and prompt behaviour change. They cover: delivery of national mass-media campaigns and local information provision; how to develop and evaluate information campaigns and interventions; the factual content of information; the tone of messages and how to tailor them for specific audiences; the workplace, and especially protecting children; young people and outdoor workers; and provision of shade as part of the design of new buildings Appendix C lists the evidence statements underpinning the recommendations.</td>
</tr>
</tbody>
</table>
Antineoplastic-induced nausea and vomiting (AINV) is distressing for all cancer patients, including children. This guideline is intended to provide physicians, nurses, pharmacists and other health care providers who care for children (aged 1 month to 18 years) receiving antineoplastic medication with a way to assess the potential of antineoplastic regimens to produce acute ANIV, i.e. ANIV within 24 hours of administration. It is most applicable to children who are receiving their first course of anti-neoplastic drugs. It considers the emetogenic potential of both single agents and multi-agent regimens given over either single or multiple days. The guidelines are based on National Comprehensive Cancer Network’s (NCCN) guideline for adults “Antiemesis v.2 2008”, and a comprehensive literature review. For each clinical question there is a summary table with brief details of the relevant studies and an assessment of the level of evidence and a grade of recommendation (the grading scheme is explained in Appendix C).

A short summary version of this guideline can be found here: [http://www.pogo.ca/_media/File/guidelines/POGO%20Emetogenicity%20Classification%20Guideline%20-%2020250111.pdf](http://www.pogo.ca/_media/File/guidelines/POGO%20Emetogenicity%20Classification%20Guideline%20-%2020250111.pdf)


These guidelines were developed as a resource for clinicians who provide continuing healthcare for survivors of childhood cancer. They are based on a thorough literature review and consensus from a panel of experts. They provide recommendations for screening and management of the late effects that may follow exposure to anti-cancer therapies including surgery, blood and serum products, radiation, chemotherapy, and haematopoietic cell transplant during treatment for paediatric cancer. The authors of this guideline noted that there were no randomised clinical trials (and none forthcoming in the foreseeable future) on which to base recommendations for periodic screening evaluations in this population; therefore, the guidelines should not be misconstrued as representing conventional "evidence-based clinical practice guidelines" or "standards of care".

Recent Systematic Reviews


Decreased physical fitness and impaired social functioning have been reported in childhood cancer survivors. This may be due to the effects of the cancer itself, cancer treatment or the behavioural, social and psychological consequences of the whole cancer experience. This review evaluated the effect of a physical exercise training intervention, offered either at home, at a physical therapy centre or at a hospital, on the physical fitness of children with cancer in comparison to that of children with cancer in a care-as-usual group. Intervention studies were eligible for this review if the intervention was offered within five years from cancer diagnosis and they were RCTs or controlled clinical trials (CCTs). Four RCTs (14, 14, 28, and 51 participants) and one CCT (24 participants) were included. In total there were 131 participants, all being treated for childhood acute lymphoblastic leukaemia (ALL). All the interventions were home-based exercise programmes implemented during chemotherapy treatment but they differed in duration of the entire intervention, the duration of each training session, the timing and the type of the interventions, and the outcomes assessed. Most outcomes were assessed by only one study, except for BMI (two studies). The authors considered that, overall, the studies were of low or very low quality with very small numbers and inadequate methodologies. They concluded that, based on the currently available evidence, they could not draw any conclusions about the best physical exercise training intervention for childhood cancer survivors, nor about the best timing for such interventions, but they noted that the studies indicated that physical training is feasible for children with ALL and that the intervention groups had some outcomes that were better than control groups, such as body composition, flexibility, and cardiorespiratory fitness, although for other outcomes, including muscle strength/endurance, the level of daily activity, health-related quality of life, fatigue, and adverse events, there were no differences between groups.


The 1989 UN Convention on the Rights of the Child gives children the right to have their views heard in matters that affect their lives. Children with cancer generally prefer to be involved in decisions relating to their healthcare and even in those relating to their end-of-life issues. It is therefore important for health professionals to know the best ways of involving children with cancer in shared decision making (SDM). This review aimed to examine the effects of SDM interventions on the process of SDM for children with cancer aged from four to 18 years. Studies were eligible for inclusion in the review if they were RCTs of SDM interventions for children with cancer aged four to 18 years. The authors were unable to find any studies meeting their criteria and so they stated that they were unable to draw any conclusions about on the effects of interventions to promote SDM for children with cancer aged four to 18 years. They stated that they plan to include non-randomised studies in future reviews to expand the range of evidence.
Human papilloma virus (HPV) is generally recognised to be the cause of cervical cancer. Vaccination against HPV is included in the immunisation programmes of many countries. There is increasing recognition that HPV has a role in the development of other neoplasms including condylomas (genital warts) and penile, anal, vulvar, vaginal, and oropharyngeal cancers. Men are affected by HPV almost as much as women but they receive no screening for HPV-related disease and no vaccination, and they are the main route for transmission of the virus from person to person. This open-access review presents the consensus of a panel of experts who met at three workshops organized and moderated by the Fondazione Giovanni Lorenzini Medical Science Foundation (Milan, Italy and Houston, TX, USA). It focuses on scientific, medical, and economic studies, and on the achievements from health organizations’ intervention programmes for dealing with HPV infection. The consensus reached was that “the reviewed studies on the natural history of HPV infection and related diseases in women and men, the increasing experience of HPV vaccination in women, the analysis of clinical effectiveness vs. economic efficacy of HPV vaccination, are even more supportive of the economic sustainability of vaccination programmes both in women and men. Those achievements address increasing and needed attention to the issue of social equity in healthcare for both genders”.


Childhood cancer survivors are at risk for subsequent central nervous system (CNS) neoplasms which have poor survival rates. This review aimed to answer three questions: (1) what is the risk of CNS tumours after radiation to the cranium (skull) for a paediatric cancer, compared with the risk in the general population; (2) what are the outcomes in children who received CNS-directed radiation for a paediatric cancer and develop subsequent neoplasms of the CNS; (3) are outcomes of subsequent neoplasms different from those of primary neoplasms of the same histology? It included 18 studies. Fourteen retrospective cohort studies (150,000 survivors of childhood or young adult cancer from 1940 to 2005, among who there were 959 subsequent CNS tumours) assessed the risk of subsequent CNS tumours after treatment for a childhood or young adult malignancy. The numbers of studies relating to outcomes following subsequent CNS neoplasms varied with the outcome in question. The studies indicated that childhood cancer survivors have an incidence of CNS neoplasms that is between 8-1 and 52.3-times higher than that of the general population. Nearly all the cancer survivors who developed a subsequent CNS neoplasm had been exposed to cranial radiation and some studies showed a correlation between CNS cancer risk and radiation dose. Five year survival rates for subsequent high-grade gliomas were poor, ranging from 0-19.5% while survival rate for subsequent meningiomas, were similar rates to those observed in patients with primary gliomas or meningiomas (57.3-100%). The authors noted that quality of evidence was limited by variations in study design and details about treatment and outcomes, and by limited follow-up and small sample sizes. They stated that current literature is insufficient to draw conclusions about the potential harms and benefits of routine screening for subsequent CNS neoplasms in child cancer survivors.


Cryptorchidism (undescended testis/testes) affects around six per cent of all newborn boys and is one of the most common congenital abnormalities. Testicular cancer is the most common malignancy in men aged between 20 and 45 years. Cryptorchidism is widely regarded as the most significant risk factor for testicular cancer but published estimates of the magnitude of the increased risk range from threefold to almost 50-fold. The aim of this review was to perform a meta-analysis to clarify the magnitude if this risk, for those without genetic syndromes or other conditions associated with an increased predisposition to the development of cryptorchidism. The authors identified nine relevant case control studies (2281 cases and 4811 controls) and three cohort studies. The cohort studies included 2,177,941 boys, of whom 345 later developed testicular cancer. (The total length of follow-up was 58,270,679 person-years). The pooled relative risk was 2.90 (95% CI 2.21 to 3.82) with significant heterogeneity (p=0.00001; I²=89%). The authors concluded that boys with isolated cryptorchidism are three times more likely to develop testicular cancer than other boys. They stated that there were some limitations to their review, especially possible publication bias and the lack of high-quality evidence focusing on the risk of malignancy in boys with isolated cryptorchidism.


Communicating effectively with children and adolescent with cancer about their condition, its treatment and its implications is an important part of good quality care. This review, which is an update of an earlier 2003 review, aimed to assess the effects of improving communication with children and adolescents about their cancer. In total ten studies met the review’s inclusion criteria: four RCTs, two non-randomised controlled trials, one non-randomised controlled trial with an historical control group, one before-and-after study with an historical control group and two uncontrolled before and after studies. Only one study, a RCT, was new since the earlier review. The studies varied considerably in interventions evaluated, study designs, types of participants and outcomes measured. The authors concluded that interventions to enhance communication with children and adolescents with cancer have not been widely or rigorously evaluated but the weak evidence that exists suggests that some children and adolescents with cancer may get some benefit from specific information-giving programmes, from support before and during particular procedures, and from interventions that aim to facilitate their reintegration into school and social activities.
Malnutrition can be a consequence of both cancer itself and cancer treatment. To combat this problem, nutritional liquids can be delivered through a central or peripheral vein (parenteral nutrition, PN); or nutritional liquids or solids can be delivered to the gut, either orally or via a tube (enteral nutrition, EN). This nutritional support can be either instead of, or additional to, normal eating. This review aimed to determine the effects of any form of parenteral or enteral nutritional support in children and young people with cancer undergoing chemotherapy. The authors identified eight RCTs (159 participants aged < 21 years with leukaemia or solid tumours) meeting their criteria, all of low quality. One small trial found that compared to EN, PN significantly increased weight (mean difference (MD) 4.12; 95% CI 1.91 to 6.33), serum albumin levels (MD 0.70; 95% CI 0.14 to 1.26), calorie intake (MD 22.00; 95% CI 5.12 to 38.88) and protein intake (MD 0.80; 95% CI 0.45 to 1.15). One trial comparing peripheral PN and EN with central PN found that mean daily weight gain (MD –27.00; 95% CI −43.32 to −10.68) and energy intakes (MD −15.00; 95% CI −26.81 to −3.19) were significantly less for the peripheral PN and EN group, whereas mean change in serum albumin was significantly greater for that group (MD 0.47; 95% CI 0.13 to 0.81, P = 0.008). The authors concluded that there was limited evidence from individual trials suggesting that PN is more effective than EN in well-nourished children and young people with cancer undergoing chemotherapy but the evidence regarding other comparisons and nutritional support in malnourished children remains unclear. They stated that “further research, incorporating larger sample sizes and rigorous methodology utilising valid and reliable outcome measures, is essential”.


Neutropenia (low white blood cell count) is a common side effect of chemotherapy and a risk for life-threatening infections. It has been suggested that a low bacterial diet (LBD) can prevent infections and infection-related mortality in people who are neutropenic as a result of cancer therapy. This review aimed to examine the evidence for this and also to assess the time to first febrile episode, the need for empirical antibiotic therapy, diet acceptability and quality of life. The authors identified three RCTs comparing intervention and control diets. In total 192 patients (children and adults) with various malignancies were included. The studies differed in regard to intervention and control diets, outcome definitions, and co-interventions (e.g. protective environment, antimicrobial prophylaxis, central venous catheter care, oral care, hygiene practices and colony-stimulating factors). All studies had serious methodological limitations and pooling of results was not possible. The authors conclude that currently there is no evidence from individual RCTs in children and adults that supports the use of LBDs for the prevention of infection and related outcomes in neutropenic cancer patients. They noted, however, that “no evidence of effect” is not the same as “evidence of no effect”. They stated that they were unable to make any recommendations for clinical practice and that more high-quality research is needed.


The objective of this review was to determine whether there was any difference in efficacy between outpatient and inpatient management of children with low-risk febrile neutropenia (FN) as a result of cytotoxic chemotherapy. The authors identified 16 relevant studies for inclusion in the review. All of them dealt only with low-risk patients. Eight were RCTs and eight were prospective non-randomised studies. None of the RCTs directly compared inpatient to outpatient treatment. Five of the eight RCTs compared oral to parenteral (IV) antibiotics. In total, 16 studies described the outcomes of 24 different treatment regimens. Results of two meta-analyses indicated that treatment failure, including antibiotic modification, was less likely to occur in the outpatient setting than the inpatient setting (15% vs. 28%, p=0.68), but was not significantly different between oral and parenteral antibiotic regimens (20% vs. 22%, p=0.68). There were no infection-related deaths in either the 953 episodes treated in the outpatient setting or the 676 episodes treated with oral antibiotics. This review did not report confidence intervals for between-group differences. The authors concluded that, based on the combination of results from all prospective studies to date, both outpatient and oral antibiotic management of low-risk FN in children are effective and should be made a part of clinical care where feasible.
This paper reports on a systematic review and meta-analysis assessing the efficacy and safety of vaccines against the oncogenic types of human papilloma virus. The review included seven RCTs of HPV vaccines involving 44,142 females. The authors synthesised efficacy data using fixed-effect models, and evaluated for heterogeneity using the I² statistic. In the per-protocol population (PPP, only those trial participants who adhered to the trial protocol), the fixed-effect relative risk (RR) and 95% confidence intervals were 0.04 (0.01 to 0.11) and 0.10 (0.03 to 0.38) for HPV-16 and HPV-18-related high-grade cervical lesions or worse (CIN2+). In the intention-to-treat population (ITT, all trial participants including those who dropped out, were lost to follow-up or did not receive some or all of their vaccine doses if they were randomised to a treatment group) the corresponding RR was 0.47 (0.36 to 0.61) and 0.16 (0.08 to 0.34). Overall, the vaccines also highly efficacious against 6-month persistent infection with HPV 16 and 18, both in the PPP cohort (RR: 0.06, [95% CI 0.04–0.09] and 0.05, [95% CI 0.03–0.09], respectively), and the ITT cohorts (RR: 0.15 [0.10–0.23] and 0.24 [0.14–0.42], respectively). The vaccine had limited prophylactic effect against CIN2+ and 6-month persistent infections associated with non-vaccine oncogenic HPV types. There were no differences between the vaccine and control groups in the risk of serious adverse events (RR: 1.00, 0.91–1.09) or vaccine-related serious adverse events (RR: 1.82; 0.79–4.20). The most common serious adverse effects were abnormal pregnancy outcomes but these were under-reported since only three out of the seven trials reported on them. The authors concluded that prophylactic HPV vaccines are safe, well tolerated, and highly efficacious in preventing persistent infections and cervical diseases associated with vaccine-HPV types among young women but that future trials are needed to address long-term efficacy and safety.


This guideline is an update of the 2003 U.S. Preventive Services Task Force (USPSTF) recommendation statement. It is based on a targeted literature search for new evidence that counselling patients about sun protection reduces skin cancer or intermediate outcomes (such as sunburn). The USPSTF found that there was convincing evidence linking UV radiation exposure during childhood and youth to a moderately increased risk for skin cancer later in life, and adequate evidence linking adult UV radiation exposure to a small increase in risk for skin cancer risk. It stated that there is moderate certainty that counselling has a moderate net benefit for children, adolescents and young adults aged 10 to 24 years with fair skin. The USPSTF made no recommendations for children under the age of 10 years because there were few trials available to determine the effectiveness of counselling parents or guardians to prevent children’s UV exposure. It found adequate evidence that, for young people and adults, there are no appreciable harms associated with counselling or sun-protective behaviours.


This review was undertaken for the USPSTF to assist them with updating their 2003 recommendation on behavioural counselling to prevent skin cancer. The authors identified no trials meeting their criteria that directly examined whether behavioural counselling can reduce skin cancer but they did find 11 fair or good quality RCTs examining the effect of counselling interventions on sun-protective behaviours. Four trials in university-age young adults used “appearance-based” behavioural interventions that emphasised the aging effect of ultraviolet light on skin and norms about tanning and looking tanned instead of a “health-based” message about skin cancer. Three of these trials (897 participants) found that the intervention reduced indoor tanning among women who had the intention to tan indoors, by up to 35%, although follow up was only three to six months. The other trial (133 participants) used a brief video intervention, with or without an ultraviolet facial photograph, and found it led to a moderate decrease in objectively measured skin pigmentation (using skin reflectance spectrophotometry) at 12 months, based on Cohen’s d statistic. One trial in adolescents (n = 819) found that computer support can increase sunscreen use and decrease midday sun exposure. The reviewers identified thirty-five observational studies, of mainly fair quality, examining the relationship between ultraviolet exposure or sunscreen use and skin cancer. Increased intermittent sun exposure in childhood is associated with increased risk for basal cell carcinoma, squamous cell carcinoma, and melanoma. The results of one fair-quality RCT (1621 participants) suggested that regular sunscreen use can prevent squamous cell carcinoma, but not basal cell carcinoma. The evidence from cohort and case-control studies regarding the effect of sunscreen on the risk of melanoma was equivocal. There is evidence from one fair quality case-control and a number of fair quality cohort studies that regular or early use of indoor tanning may increase melanoma risk. The reviewers note that observational studies are limited by the complexity of measuring ultraviolet exposure and sunscreen use, and inadequate adjustment for important confounders. They concluded that RCTs suggest that primary care-relevant counselling can increase sun-protective behaviours and decrease indoor tanning. The publications above, and related material, can be found on this web page:

Influenza can be (but is not usually) a serious illness in children with cancer, therefore vaccination against influenza is generally recommended. There is conflicting data about the immune response to influenza vaccination in children with cancer and so the value of vaccination for these children is unclear. This review aimed to assess the efficacy of influenza vaccination in stimulating immune response (compared to control groups) and in preventing influenza-like illness (compared to placebo), in children (aged one to 18 years) receiving chemotherapy for cancer. It also aimed to determine the adverse effects of influenza vaccination in these children. The review included one RCT (comparing responses to two different influenza vaccination protocols) and eight controlled clinical trials. In total there were 708 participants. None of the studies compared influenza vaccine to placebo and none reported on clinical outcomes such as confirmed influenza, hospitalisation, delay in chemotherapy or mortality, but all reported on influenza immunity and adverse reactions. The studies’ results indicated that paediatric cancer patients receiving chemotherapy are able to generate an immune response to influenza vaccination but their immune response is weaker than that of healthy children, children with asthma or children with cancer who completed chemotherapy one month or more before vaccination. These differences between trial groups in immune response were found irrespective of the method used to assess immune response. Adverse effects were mild local reaction and low grade fever. There were no serious adverse reactions. The authors concluded that paediatric cancer patients receiving chemotherapy do generate an immune response to influenza vaccination but it is unclear whether this protects them from influenza infection or its complications and therefore it is uncertain if vaccination has any clinical benefits for this population. They stated that further well-designed RCTs are needed to address this issue.


Nausea and vomiting are common problems in children being treated with chemotherapy for cancer. This review aimed to assess the effectiveness and adverse effects of pharmacological interventions in controlling anticipatory, acute and delayed nausea and vomiting in children and young people (aged < 18 years) either about to receive or receiving chemotherapy. It included 28 RCTs which examined a range of different antiemetic drugs, used different doses and comparators, and reported a variety of outcomes Most studies were small. Most of the quantitative data from these studies related to the control of acute (within 24 hours) vomiting (22 studies). Twenty-four studies reported on adverse events and ten on nausea. Two studies assessed the addition of dexamethasone to 5-HT3 antagonists for complete control of vomiting and found that the two drugs together were superior: pooled ratio 2.03, 95% confidence interval 1.35 to 3.04 (indicating that patients receiving the combination were about twice as likely not to have vomiting). Three studies compared granisetron 20 mcg/kg with 40 mcg/kg for complete control of vomiting (pooled RR 0.93; 95% CI 0.80 to 1.07). No other pooled analyses could be done. It appeared that 5-HT3 antagonists are more effective than older antiemetic agents, even when those agents are combined with a steroid and that, of the 5-HT3 receptor antagonists, granisetron may be more effective at higher doses. Cannabinoids appear to be effective but frequently cause side effects (particularly drowsiness and dizziness). The authors concluded that our knowledge about the best anti-emetics for preventing chemotherapy-induced nausea and vomiting in children with cancer is limited. They stated that 5-HT3 antagonists with added dexamethasone are effective but noted that the use of steroids as an anti-emetic is somewhat controversial because some in-vitro studies have suggested that glucocorticoids (such as dexamethasone) reduce the sensitivity of a wide range of cell lines to chemotherapy agents (although no studies have found an association between steroids as an antiemetic and worsened outcomes).


Infection with the sexually transmitted human papilloma virus (HPV) is the key risk factor for cervical cancer. This review aimed to assess the effectiveness of behavioural interventions for young women intended to encourage safer sexual practices to prevent transmission of HPV, cervical cancer and other sexually transmitted infections (STIs). It included 23 RCTs, most conducted in the U.S. in healthcare settings such as family planning clinics. Most of the interventions provided information about STIs and taught safer sex skills such as communication. Some also provided resources, for example free sexual health services. There was considerable variation (i.e. heterogeneity) among the trials in behavioural aims, provider, contact time, duration and outcomes, so meta-analysis was not considered appropriate. The trials addressed a variety of STIs including HIV and chlamydia but none explicitly mentioned HPV or cervical cancer. Trials commonly reported statistically significant effects on behavioural outcomes such as increased condom use, although this was not universal and varied with type of outcome. No trials reported statistically significant effects on reducing or abstaining from sexual activity. There were few statistically significant effects on biological outcomes related to STIs. The authors concluded that behavioural interventions for young women aimed at promoting behaviours that reduce the risk of acquiring a STI can be effective, primarily at encouraging condom use. They stated that future evaluations should have a greater focus on HPV and its link to cervical cancer and involve long-term follow up to assess impact on behaviour change, rates of HPV infection and progression to cervical cancer.
Cervical screening is used to detect pre-cancerous changes in the cervix so that they can be dealt with before they develop into invasive cervical cancer. Increasing the uptake of cervical screening is important to reduce the number of women who develop cervical cancer but there is increasing debate about ‘informed uptake’ in recognition of the fact that screening has both positive and negative effects for individual women and that increasing uptake at all costs may not be justified. This review aimed to assess the effectiveness of interventions aimed at increasing uptake, and informed uptake of cervical screening. It included 27 RCTs and eight quasi-RCTs of variable quality. Interventions studied included invitation letters and telephone calls, face-to-face assessments, mass letter campaigns, educational interventions, counselling, risk factor assessment, procedures (for example revealing the gender of the smear taker and using a health promotion nurse), and economic incentives (such as free transport or parking). Due to the heterogeneity between the studies only limited pooling of data was possible and the review’s conclusions are largely based on a narrative synthesis. The authors concluded that there was evidence from good quality RCTs to support the use of invitation letters for increasing the uptake of cervical screening via Pap smears and some evidence for the use of educational interventions. They noted that no studies considered that uptake of informed cervical screening.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2857928/?report=classic
This review aimed to answer three clinical questions: 1) what is the incidence and excess risk for breast cancer in women after chest radiation for paediatric or young adult cancer? 2) for these women, are the clinical characteristics of breast cancer and the outcomes after therapy different from those of women with sporadic breast cancer in the general population? and 3) what are the potential benefits and harms associated with breast cancer surveillance among women exposed to chest radiation? For question 1, the authors identified eleven retrospective cohort and three case-control studies meeting their eligibility criteria. The cohort studies included over 14,000 young women, of whom 7000 were treated with chest radiation, mostly for Hodgkin’s lymphoma, and 422 subsequently developed breast cancer. All of the studies reported a significantly increased incidence and/or absolute excess risk of breast cancer in women who had received chest radiation. Among the higher quality cohort studies, the standardised incidence ratios ranged from 13.3 to 55.5 and the absolute excess risk ranged from 18.6 to 79.0 per 10,000 person-years. The cumulative incidence of breast cancer by age 40–45 years ranged from 13–20% and the risk of breast cancer increased linearly with chest radiation dose. The evidence for questions 2) and 3) was of limited quality due to substantial study heterogeneity, variations in study design, and small sample size but suggested that characteristics of the breast cancers in these women and the outcomes following diagnosis are similar to those in the general population and that these breast cancers can be detected by mammography, though sensitivity is limited.

Cochrane Reviews relating to more specific aspects of childhood cancer treatment
The following Cochrane review relate to specific aspects of childhood cancer treatment. There is not space to summarise the contents of all of them here, but the following list of titles is provided to indicate the topics that have been the subject of recent Cochrane reviews.


Note: The publications listed were identified using the search methodology outlined in Appendix 1.
OBESITY, NUTRITION AND PHYSICAL ACTIVITY
**Introduction**

According to the World Health Organization, childhood obesity is one of the most serious public health challenges of the 21st century \[91\]. Over the past thirty to forty years there has been a marked increase in the prevalence of childhood obesity in almost all countries for which data is available \[92\].

New Zealand is part of this global trend. The New Zealand Health Survey 2011/12 found that, among children aged 2–14 years, 20.7% were overweight and 10.2% were obese \[2\]. In boys, but not in girls, there was a statistically significant increase in the obesity rate in 2–14 year olds from the 2006/07 survey (8%) to the 2011/12 survey (10%). For 5–14 year old children, although the rate of obesity did not change from 2002 (9%) to 2006/07 (8%), from 2006/07 to 2011/12 there was a statistically significant increase (to 11%).

Childhood obesity carries significant physical and mental health risks both in the short term and the long term \[5\]. In the short term, childhood obesity may be associated with asthma \[93\], sleep apnoea \[94,95,96\], slipped femoral capital epiphysis (slipped upper femoral epiphysis) \[4,97\], being bullied, teased and socially marginalised \[98,99,100,101,102\], and having low self-esteem \[103,104\]. Childhood obesity has been associated with emotional and behavioural problems even in pre-school children \[105\]. Many studies in children have documented the association between childhood obesity and most of the major risk factors for later cardiovascular disease: high blood pressure, dyslipidaemia, hyperinsulinaemia and/or insulin resistance, abnormalities in left ventricular mass and/or function, and abnormalities in endothelial function \[5\].

A number of large, long-running cohort studies have found that childhood overweight and obesity is associated with premature mortality in adulthood and with an increased risk of type 2 diabetes, stroke, coronary heart disease, and hypertension later in life \[3\]. It has been argued that this is largely because childhood overweight and obesity is a strong predictor of adult obesity \[106,107\]. Obese children have a moderately high probability of becoming obese adults (in the range 40–70%) \[5\] and adult obesity is associated with substantially increased risks of hypertension, dyslipidaemia, type 2 diabetes, coronary heart disease and stroke \[108\]. This is the major reason why childhood obesity is considered to be a public health crisis \[109\].

Adult obesity is also associated with an increased risk of gallbladder disease, osteoarthritis, sleep apnoea and respiratory problems and some types of cancer \[108\]. The most convincing evidence for body fatness as a cause as a cause of cancer is for cancer of the oesophagus, pancreas, colon and rectum, breast (in post-menopausal women), uterus (endometrium), and kidney \[110\].

There is a general consensus among obesity experts that the obesity problem is not simply a personal issue of eating too much and doing too little but a problem that has its roots in a mismatch between basic human biology, the product of thousands of years of evolution, and modern society. It therefore requires tackling complex social and economic issues and changing public policy in many areas including food production, manufacturing and retailing, trade, urban planning, transport, healthcare, education and culture \[109\].

This in-depth topic aims to examine the determinants and consequences of childhood obesity and also to provide background information on the trends in childhood obesity prevalence (both globally and in New Zealand), defining and measuring obesity, and the natural history of obesity over the lifespan.

The sections covering these topics are arranged as follows:

- Factors contributing to childhood obesity
• Defining and measuring obesity
• The prevalence of childhood obesity
• The natural history of obesity over the life course
• The consequences of childhood obesity

Factors contributing to childhood obesity

Humans, like all animals, have a powerful biological drive to consume the food necessary for survival and rarely choose not to eat when they are hungry. They often, however, eat when they are not hungry and continue eating when they have already eaten enough. At a fundamental level, obesity is the result of mismatch between appetite, the drive to eat, and satiety, the drive to stop eating when sufficient food has been consumed. Eating behaviour is influenced by complex interactions between genetic, physiological, psychological and environmental factors that are incompletely understood but have been, and continue to be, the subject of much research.

From very early in life there are both genetic and environmental factors operating to influence a child’s chances of becoming overweight or obese [111, 112, 113].

Genetics and parental weight status

Parental obesity is a strong predictor of a child’s weight status [114]. The Avon longitudinal study of parents and children, which included 8234 children born in the U.K. in 1991 and 1992, found that a child was ten times more likely to be obese at age seven if both parents were obese than if neither parent was obese (adjusted odds ratio 10.4, 95% CI 5.1 to 21.3) [115]. Since parents are largely responsible for their child’s food environment in early life it is difficult to separate out the genetic and environmental components of the influence of parental obesity [116].

Maes et al. reviewed over thirty twin, adoption and family studies and concluded that genetic factors are significant and explain between 20% and 90% of the variation in body mass index (BMI, defined as weight/height$^2$ with weight measured in kg and height in metres) [117]. It seems that there are many interacting genes involved in the predisposition to obesity, each with a small effect [118]. Single gene mutations causing morbid obesity in humans have been identified [119] but they are exceedingly rare [120].

There are a number of very rare syndromes with a Mendelian (single gene) pattern of inheritance, such as Prader-Willi syndrome and Bardet-Biedl syndrome, which include obesity as a clinical feature, often in association with mental retardation, dysmorphic features and organ-specific developmental abnormalities [121].

The hormone leptin, discovered in 1994, is secreted by adipocytes (fat cells) and is essential for regulating body weight through its effects on food intake and energy expenditure. [122] A few people who were homozygous for mutations in the gene encoding leptin, and consequently had congenital leptin deficiency, were subsequently identified in two families, in Pakistan [123] and in Turkey [124]. The clinical phenotype associated with congenital leptin deficiency includes hyperphagia (overeating), severe obesity, hypogonadism, and impaired immunity. Treating leptin-deficient people with daily injections of recombinant human leptin produces dramatic reductions in food intake and obesity [122, 125]. Subsequent research has indicated that there are many different genetic defects affecting the downstream pathways from leptin signalling in the brain. All of these are associated with hyperphagia. This research has made it clear that human appetite and food intake are in part biologically determined and there are hopes it may lead to therapeutic options for regulating food intake in obese people [122].

O’Rahilly and Farooqi observed that all known monogenetic causes of obesity are associated with disruption of the hypothalamic pathways in the central nervous system [126] and have a profound effect on food intake and satiety [127]. They suggest that the evidence indicates that the major effect of genes on obesity is just as likely to be via an impact on hunger, satiety and food intake as via an impact on metabolic rate or fat deposition.
Prenatal influences
There is increasing evidence from both human and animal studies that prenatal and neonatal exposures can influence a child’s risk of becoming an obese adult, developing metabolic syndrome (the combination of hypertension, insulin resistance, type 2 diabetes, dyslipidaemia and obesity) and developing cardiovascular disease [128,129]. The fetal origins hypothesis [130], also known as the developmental origins hypothesis, is that cardiovascular disease and type 2 diabetes may have their origins in under or over-nutrition in utero. The fetus makes adaptive responses to cues from the mother about her health or physical state. These responses may include changes in metabolism, hormone production and tissue sensitivity to hormones which may affect tissue and organ development and lead to persistent alterations in metabolic and physiologic set points [131]. Epigenetic changes in DNA (changes that do not involve changes in the DNA sequence but involve either methylation of DNA or modifications of chromatin) occurring in response to environmental stimuli alter gene expression and are believed to be a mechanism by which the fetus is “programmed” for later cardiac and metabolic disease, mostly on the basis of evidence from animal studies [132].

Whether being born to a mother with gestational diabetes increases the risk of childhood overweight and obesity may depend on whether or not the mother is also overweight or obese. A high pre-pregnancy BMI increases the risk of developing gestational diabetes. A 2009 systematic review and meta-analysis found that, compared to women with normal BMI, the unadjusted pooled odds ratios of developing gestational diabetes for overweight, moderately obese and morbidly obese women were 1.97 (95% CI 1.77 to 2.19), 3.01 (95% CI 2.34 to 3.87) and 5.55 (95% CI 4.27 to 7.21) respectively [133]. A 2011 systematic review considered 12 studies examining the association between gestational diabetes and childhood overweight and obesity [134]. The review authors found that most studies had methodological limitations. Only three studies adjusted for confounders. The two studies that adjusted for pre-pregnancy BMI found that, after this adjustment, there was no statistically significant association between gestational diabetes and offspring overweight and obesity.

Birth weight and early weight gain
Three recent systematic reviews have examined the relationship between birthweight and long term risk of overweight or obesity. Schellong et al. performed a meta-analysis of 66 studies (with various definitions of overweight and obesity, mostly related to BMI) and found a positive linear relationship between birthweight and later (in childhood, adolescence or adulthood) risk of overweight [135]. Low birthweight (<2500g) was associated with a decreased risk of overweight (Odds ratio 0.67; 95% CI 0.59–0.76) while high birthweight (>4000g) was associated with an increased risk of overweight (OR = 1.66; 95% CI 1.55–1.77). Zhao et al. performed a meta-analysis of fifteen studies reporting on the relationship between birthweight and overweight or obesity in adults [136]. They found that low birthweight (<2500g) was not associated with an increased risk of overweight/obesity (OR 1.17; 95% CI 0.94–1.46) but high birthweight (≥ 4000g) was associated with an increased risk of both overweight/obesity (OR = 1.46; 95% CI 1.27–1.68) and obesity (OR 1.43; 95% CI 1.25–1.64). Yu et al. included 33 studies in their review focusing on the association of birthweight with obesity in children and adolescents, most of which had been conducted in China [137]. They performed a meta-analysis of the results of 20 of them. They also concluded that high birthweight (>4000g) was associated with obesity (14 studies, OR = 2.07; 95% CI 1.91–2.24) and that low birthweight (<2500g) had a negative association with obesity (10 studies, OR = 0.61; 95% CI 0.46–0.80). The odds ratio for obesity increased gradually with increasing birthweight across all birthweight categories and 3500g was the threshold value above which the risk of obesity was increased.

The association between birthweight and the risk of type 2 diabetes in adulthood has been extensively studied. Whincup et al. carried out a quantitative systematic review of this issue using data from 30 reports (31 populations; 6090 diabetes cases; 152,084 individuals) [138]. After data from two Native American population with a high prevalence of maternal diabetes and one other population of young adults was excluded, the pooled
odds ratio for type 2 diabetes after adjustment for age and sex was 0.75 (95% CI, 0.70–0.81) per kilogram. The odds ratio was a little greater after adjustment for current BMI and adjustment for socio-economic status made almost no difference. Birthweight is therefore inversely related to type 2 diabetes risk across the normal range of birth weight.

Low birth weight may not be a risk factor for metabolic syndrome and subsequent type 2 diabetes if it is due to prematurity alone rather than failure to grow in utero. A recent systematic review by Parkinson et al. considered studies of adults (mean age 19.6 years, range 18–45 years) who were born pre-term babies (mean gestational age 32 weeks) that had measured markers of metabolic syndrome, including BMI, blood pressure, fasting glucose and lipid profiles [139]. There were no differences between preterm and term-born adults in most markers, including BMI, waist-to-hip ratio and per cent fat mass, but the adults born pre-term had higher blood pressure, more so in women. Young adults born pre-term had increased plasma low-density lipoprotein which could indicate an increased risk of later atherosclerosis and cardiovascular disease.

Low birthweight babies who have early rapid “catch-up” growth in their first two years are fatter than other children by age five [140]. Many large cohort studies have indicated that, among all children, after adjustment for age, sex and birthweight, more rapid weight gain in infancy is associated with an increased risk of subsequent obesity [141,142,143].

A possible explanation for the apparently paradoxical finding that the higher the birthweight the lower the risk of diabetes and cardiovascular disease but the higher the risk of adult overweight and obesity (which are risk factors for both diabetes and cardiovascular disease) is that a higher birthweight programmes greater lean mass in later life and that at given level of adult BMI, having had a low birthweight is associated with a higher percentage of body fat [144,145,146]. Further studies investigating body composition in infants may help to clarify this issue [147].

Breastfeeding
It is difficult to assess the effect of breast feeding on obesity risk. It is unethical to conduct a randomised controlled trial (RCT) of breast feeding versus formula feeding and observational studies are subject to numerous confounders. There has been a large RCT (17,046 infants) of a breastfeeding promotion intervention in Belarus which found that, although the intervention led to substantial increases in the duration and exclusivity of breastfeeding, it did not reduce measures of adiposity at age 6.5 years in the intervention group, or increase stature, or reduce blood pressure [148].

There are plausible reasons to believe breastfeeding should lower obesity risk: breast milk contains hormones such as leptin, adiponectin and ghrelin which may affect long term appetite regulation; infants being breast fed are typically better than those being bottle fed at signalling to their mothers that they are full and so may develop better self-regulation of energy intake; breastfed infants gain weight more slowly than formula fed infants; infant formula contains more protein than breast milk and randomised controlled trials have shown that protein-enriched formulae produce more rapid weight gain, increased adiposity and some later adverse cardiometabolic consequences in childhood and adolescence; and many cohort studies have shown an association between having been breastfed or longer duration of breastfeeding and reductions in obesity risk [149].

Owen et al. reviewed a large number of observational studies and found that breastfeeding was associated with a slightly lower mean BMI in later life but adjustment for socioeconomic status, maternal smoking in pregnancy and maternal BMI in 11 studies abolished the effect [150]. They concluded that mean BMI is slightly lower in those who were breastfed but this result is likely to be strongly influenced by publication bias and confounding factors and that, while there are good reasons for mothers to breastfeed, doing so is not likely to reduce their children’s mean BMI in later life. Another 2004 systematic review by Arenz et al. included a meta-analysis of data from nine studies (over 69,000 children) that adjusted for at least three of the factors birthweight, parental overweight, parental smoking, dietary factors, physical activity and socioeconomic status [151]. The adjusted odds ratio calculated from the data of these studies was 0.78 (95% CI
0.71–0.85) and the study authors concluded that there was a small but consistent protective effect of breastfeeding against obesity risk in later childhood. Weng et al. performed a meta-analysis of ten studies published between 2003 and 2009, five of which had found a protective effect of breast feeding and five of which had not, to obtain a pooled odds ratio for the effect of having been ever breast fed on being overweight in childhood of 0.85 (95% CI 0.74–0.99) [152].

Sleep

There is evidence from many cross-sectional studies around the world of a link between sleep duration and obesity in all age groups [153]. A 2008 systematic review by Chen et al. reported on three cohort studies, 12 cross-sectional studies and two case-control studies in children and adolescents [154]. Six had been conducted in the U.S., five in Europe, four in Asia and one each in Australia and Canada. Numbers of participants varied from 150 to 8,941 but most studies had over 1,000. The review authors found that there was strong evidence of an association between short sleep duration and childhood obesity and that early life shortness of sleep seems to be associated with greater risk. The association between short sleep and obesity may be stronger in boys as this was found in large studies in Japan and Australia. In general, the studies in children aged < 10 years found an inverse association between sleep duration (according to parental report) and obesity but those in adolescents were inconsistent. Based on a meta-analysis of 11 studies, the pooled odds ratio of overweight/obesity for shorter vs. longer sleep duration (based on each individual study’s criteria) was 1.58 (95% CI 1.26–1.98). Based on three studies, gender-specific odds ratios were 2.50 (95% CI 1.91–3.26) for boys and 1.24 (95% CI 1.07–1.45) for girls. Another systematic review by Patel and Hu, which included ten of the same studies and also three smaller studies, two of which did not adjust for any confounders, stated that the studies’ results suggest that, in children, short sleep is strongly and consistently associated with current and future adiposity but that major limitations in study design precluded definitive conclusions [155].

An Otago study investigating the relationship between sleep duration and measures of adiposity in adolescents found an effect in boys, but not in girls. In boys each hour increase in average nightly sleep duration was associated with decreases of 1.2% for waist circumference, 0.9% for waist-to-hip ratio 4.5% for fat mass index and 1.4% for fat-free mass index in multivariate models. Results were similar for weekday and weekend night sleep duration [156]. The Prevention of Overweight in Infancy (POI.nz) study is currently in progress in Dunedin. This is a four-arm randomised controlled trial of food, activity and sleep interventions for preventing the development of overweight from infancy [157].

Environmental factors in childhood

Many of the changes in family lifestyles that have occurred over the last few decades are likely to be contributing to childhood overweight and obesity but it is difficult to assess the impact of any one factor in isolation. Factors that are probably significant contributors to what has been termed the “obesogenic environment” are: increased consumption of food away from home, processed food, fast food, sugar-sweetened beverages and fruit juices; fewer children walking or biking to school; increased television watching, computer use and video game playing (“screen time”); and decreased participation in organised sport [158]. Reduced physical activity and increased sedentary behaviour may be both causes and consequences of obesity [113].

It is unclear what relative contributions excess consumption and insufficient physical activity make to childhood obesity. A recent review identified 26 cross-sectional and longitudinal studies examining the relationship of factors related to energy intake and energy expenditure to childhood obesity [159]. The reviewers found that there was wide variation in data quality between studies and concluded that, on the basis of current evidence, there is no consensus on the main driver of the increase in childhood obesity prevalence over recent decades.

A recent Australian study used data from the Longitudinal study of Australian Children to examine whether patterns of behaviour, based on physical activity, diet, screen time and sleep time, were associated with obesity in 1833 children aged 6–7 years [160]. Using
latent class analysis the researchers identified three behavioural profiles: healthy (27.7%), sedentary (24.8%) and short sleepers/unhealthy eaters (47.5%).

The healthy profile was associated with the lowest rates of sleeping <10 hours per night, low levels of screen time and high levels of physical activity, and it also had the highest intake of fruit and vegetables and the lowest intake of high fat foods and high sugar drinks. The sedentary profile was characterised by long screen time (> 2 hours per day) and low rates of physical activity (< 1 hour per day). The short sleepers/unhealthy eaters profile was characterised by the highest rate of short sleep (27.2%) and greater consumption on high fat foods and sugary drinks. Compared to the healthy profile, at two year follow up both the sedentary profile (odds ratio = 1.59, 95% CI 1.06–2.38) and short sleepers/unhealthy eaters profile (odds ratio = 1.47; 95% CI 1.03–2.13) had elevated odds of obesity.

The cost of healthy eating
It has been claimed that healthy food is more expensive than “junk food” and that cost is a barrier to families being able to provide nutritionally adequate meals for their children [161]. A 2011 study by Regional Public Health in Wellington, entitled Food Costs for Families: Analysis of the proportion of the minimum wage and income support benefit entitlements that families need to purchase a healthy diet, found that in order to purchase a “basic” healthy diet families needed to spend 43%–89% of their net income after deduction of rent costs [162]. Others have claimed, however, that it is possible to achieve substantial improvements in nutrition without spending a great deal more money [163].

Food insecurity has been defined as: “the inability to acquire or consume an adequate diet quality or sufficient quantity of food in socially acceptable ways or the uncertainty that one will be able to do so” [164]. The 2008/09 New Zealand Adult Nutrition Survey classified 59.1% of New Zealanders as living in households that were fully/almost food secure, 33.7% as living in households that were moderately food secure and 7.3% as living in households that had low food security, based on survey participants’ answers to questions on eight facets of food security related to food affordability. This survey did not report on households with children specifically [165].

The 2002 National Children’s Nutrition Survey asked adult members of the children’s households about their household’s food security. Overall, 77.8% of children’s households could “always” afford to “eat properly” and 20.1% could “sometimes” afford to do so. There were marked increases in the proportion of households who could only sometimes afford to eat properly with increasing number of children (from 15.1% in those with ≤ 2 children to 37.3% in those with ≥ 5 children) and with increasing deprivation (from 5.5% in NZDep quintile I to 37.5% in NZDep quintile 5). The proportion of Māori children’s households who could only sometimes afford to eat properly was 33.6%. The proportion of Pacific children’s households in this category was 47.9% while the proportion of European/Other children’s households was 12.1% [166].

While it may seem counter-intuitive, a link between food insecurity and obesity has been found in a number of studies. Several recent reviews have looked at this issue in relation to children and adolescents [167,168,169,170]. There is a consensus that there is an association between food insecurity and obesity for adult women but for children the evidence is inconclusive.

There are particular challenges faced by Pacific people wishing to choose more healthy eating patterns. In Pacific societies food plays an important social and cultural role and the ability to provide plenty of food for the family and visitors is an integral part of Pacific identity [171]. Lanumata and others reported on a series of focus groups that they facilitated with Māori, Pacific and low-income New Zealanders to learn about their views on food security and physical activity [172]. They stated that there was “unanimous agreement amongst participants about the desire for better access to nutritious food in order to live healthier and longer lives”. The Tongan participants reported that huge feasts were part of cultural functions such as birthdays and funerals. Along with cultural expectations regarding hospitality, cost was reported to be a major barrier to healthy eating.
for many Pacific people. Participants also talked about the convenience of fast food and some people lacking the time or the knowledge and skills to prepare healthy food.

A recent report from the Office of the Auditor General comments on four focus groups which discussed the issue of child obesity and programmes to address it with Māori and Pasifika families of children aged five to fourteen in Auckland [173]. Māori parents reported poverty as a major obstacle to providing healthy food for their families. This was also a concern for Pasifika families but they saw the main issue as food and eating to excess being an integral part of their culture and a customary belief that being big was a sign of health, strength and being well cared for.

The prevalence of obesity in children and young people

A global perspective

Until relatively recently, most of humanity struggled against food shortage, disease and a hostile environment [174]. People lived in small groups and obtained most of their food from sources close to where they lived. Obesity was confined to a few high status individuals. In the poorest and least developed countries this is still the situation today but in developed and developing countries obesity is pervasive and is increasingly a greater problem for the lower socio-economic groups in society. This shift from the higher to the lower socio-economic groups occurred first in the U.S.A. and Europe but it is now occurring in developing countries also, most obviously in women [175,176]. In developing countries in the midst of the “nutritional transition” where diets are changing from locally produced fruits, vegetables and whole grains to highly processed, low cost, energy dense foods there is a “nutrition paradox” where over nutrition and under nutrition co-exist and overweight mothers may have stunted children [177].

From the 1970s to the end of the 1990s the prevalence of overweight or obesity in school age children doubled or trebled in many countries including Canada, the United States, Australia, Japan, the U.K., Finland, Germany, Greece, Spain, Brazil and Chile [92]. There is, however, some evidence that, over recent years, in a number of countries, obesity rates in children are no longer increasing and the prevalence of overweight and obesity may have plateaued [178,179,180].

New Zealand children: trends over time, ethnic and socio-economic disparities

Not only are there more obese children than there were but, on average, all children are heavier. Participants in the Family Lifestyle, Activity, Movement and Eating Study, born in Dunedin in 2001–2002 had a mean body mass index at age seven that was 0.84 kg/m² (95% CI 0.61 to 1.06) greater than that of the participants in the Dunedin Multidisciplinary Health and Development Study, born in 1972–1973 [181]. A comparison of body mass index in two groups of 12 year old Hawke’s Bay schoolchildren who were part of an asthma prevalence study indicated that, over the 11 year period from 1989 to 2000, the geometric mean BMI increased from 18.1 kg/m² (95% CI 17.9 to 18.3) to 19.8 kg/m² (95% CI 19.6 to 20.0) in 2000, a relative increase of 9.2% (95% CI 7.6 to 10.9) [182].

As has been found in other developed countries [183,184,185], the New Zealand Health Survey 2011/12 found marked social and ethnic inequalities in childhood (2–14 years) obesity rates [2]. Pacific and Māori children (compared to European/other children) and children living in the most deprived areas (compared to those living in the least deprived areas) had significantly higher rates of both overweight and obesity. Similar disparities were found in the Youth ’07 survey of 9,107 secondary school students [186]. More detail on the prevalence of overweight and obesity in New Zealand children can be found in the Overweight and Obesity chapter, beginning on page 301.

Defining and measuring obesity

In order to be able to make meaningful comparisons between obesity rates in different population groups and to monitor trends in obesity over time it is essential that a consistent definition of obesity is used. The World Health Organization defines overweight and obesity as “abnormal or excessive fat accumulation that may impair health” [187]. It is not possible to measure total body fat precisely in a living person so various measures are
used as proxies. The techniques that have been developed to measure body fat are complex and have mostly been used only in research settings. They include hydrodensitometry (under water weighing), air displacement plethysmography, CT and MRI scanning, dual-energy X-ray absorptiometry (DEXA/DXA), and bioelectrical impedance methods [188].

For clinical use and for population surveys and screening, the measures used to assess body fatness include skinfold thickness, waist circumference, waist-to-hip ratio and body mass index [188]. Body mass index (BMI), defined as weight/height$^2$ (with weight is measured in kg and height in metres) is the most widely used tool for monitoring obesity. Definitions of overweight and obesity based on BMI values depend on specified cut-off points above which BMI values indicate overweight or obesity. For adults, the World Health Organization has defined obesity as a BMI $\geq 30$ kg/m$^2$ and overweight as a BMI $\geq 25$ kg/m$^2$.

A child’s BMI is typically plotted on a BMI-for-age reference chart which has percentile lines marked on it to indicate the percentage of children in a reference standard (non-obese) population whose BMI is at or below a given level at each age. Whether or not a child is labelled as being overweight or obese depends somewhat on the reference standard used and the percentiles chosen as cut-offs [189]. Children who have a BMI greater than or equal to the 95th percentile for their age are commonly classified as being obese, and those whose BMI is at or above the 85th percentile but less than the 95th percentile as being overweight [190] but there is no universal agreement on BMI cut-off points for defining obesity in children [191,192].

The World Health Organization’s Child Growth standards are widely used for monitoring obesity in children aged 0–5 years [189,193]. The growth charts that are used by Well Child/Tamariki Ora providers (and are in the Well Child/Tamariki Ora Healthbook) are based on the 2006 World Health Organization’s standards and were originally developed for use in the U.K. [194,195,196,197].

For older children widely used reference standards (especially for research purposes) are the BMI-for-age percentile charts of the World Health Organization [198,199] and the BMI-for-age obesity and overweight cut-off charts and tables of the International Obesity Taskforce [200]. Some countries, including the U.S.[201] and the U.K. [202] use their own reference standards.

Research studies often report results as BMI z-scores, also known as BMI SDS (standard deviation scores), which indicate how may units of the standard deviation a child’s BMI is above or below the average BMI for their age group and sex [203]. For example, a child with a z-score of 2.0 has a BMI that is 2.0 standard deviations above the average. Charts, tables or software can be used to convert children’s BMI values into BMI percentiles or z-scores [204]. Although z-scores and percentiles can be converted to each other the commonly used cut-off points are not precisely comparable: a z-score of +2 corresponds to the 97.7th percentile and the 85th percentile corresponds to a z-score of 1.04 [205]. The advantage of using z-scores is that they are useful for monitoring changes in weight in patients with BMI values above the 99th percentile, and in a research setting they enable the calculation of a meaningful average result for a group that includes children of different ages.

**Should the same BMI values be used to define obesity in different ethnic groups?**

There had been considerable debate about whether the same BMI cut-off points should be used to define obesity in different ethnic groups, both internationally [206] and in New Zealand [207,208]. Tyrrell et al. measured obesity rates in 2273 Auckland according to both BMI and bioelectrical impedance measures in the late 1990s. These children attended primary schools with a high proportion of Māori and Pacific Island pupils. There were clear differences in obesity rates between different ethnic groups: Pacific Island 24.1%, Māori 15.8%, and European 8.6%, but no clinically relevant ethnic differences in the relationship between BMI and body composition in children who had BMIs in the normal range (<30). The small, but statistically significant effect of ethnicity on this
relationship was attributable to the number of Pacific Island children with BMI>30. Tyrrell et al. stated that this skewing did not justify the use of different BMI percentiles for the different ethnic groups [208].

Using a subsample of participants in the 2002 New Zealand Child Nutrition Survey (643 children aged 5–14 years), Rush et al. investigated the relationships between BMI, body fatness, ethnicity, age and blood lipids. They found that Māori and Pacific Island girls had a lower per cent body fat (as determined by bioimpedance analysis) for the same BMI compared to European girls, but for boys there were no ethnic differences [209]. A later Auckland study of 1,247 primary school children, which included 147 East Asian and 117 South Asian children, found that, although the level of body fat at a given BMI appeared similar among Māori, European and East Asian boys and girls, South Asian children averaged 3.0–5.1% (boys) and 4.2–6.1% (girls) more body fat than the remaining four ethnic groups. When adjustment was made for this difference in body fat percentage, the prevalence of obesity in South Asian children rose from 5.1% to 21.4% [210]. Using a larger sample of 1676 girls with a wider age range (5–16 years), the same authors found that, compared to European girls, for a fixed BMI and age, South and East Asians averaged 4.2% and 1.3% more body fat, while Pacific Islanders averaged 1.8% less [211]. Using stepwise multiple regression they developed a series of ethnic-specific BMI cut-off points which they considered would provide a more accurate indication of overweight or obesity in New Zealand girls [212].

The limitations of BMI in clinical practice

While BMI is very useful for making comparisons between different population groups or for monitoring a population over time, it has some limitations as an indicator of body fatness at the individual level [192]. It does not take account of where on the body fat is deposited. It is high visceral fat that is associated with insulin resistance and risk of diabetes and cardiovascular disease [213]. A high BMI may be due to high muscle mass in an athletic child or adolescent who does not have excess body fat [190]. Body mass index in older children is affected by degree of sexual maturity. Early maturing girls tend to have a higher BMI than less mature girls of the same age while the opposite is the case for boys [214].

In the clinical setting, for identifying children who may be candidates for weight management intervention, BMI is a complement to, and not a substitute for, clinical examination. Visual inspection alone may be unreliable for identifying obese, and particularly overweight, children because, as being overweight is increasingly the norm, children with BMIs above the 85th or 95th percentile may not appear very different from other children [215]. Plotting BMI on a BMI-for-age chart is useful for tracking a child’s weight status over time since it can take account of the fact that a child is growing in height but improvements in health status following adopting a healthier diet and increasing physical activity may not lead to improvements in BMI if fat is replaced with muscle.

The natural history of obesity over the life course

Cross-sectional population surveys, including the most recent New Zealand Health Survey [216], indicate that the proportion of people who are overweight or obese increases with age up until old age, where it levels off or declines [217,218].

Longitudinal cohort studies provide the best methods for studying the tracking of children’s weight status over time and investigating the childhood predictors of adult overweight and obesity and adult cardiovascular disease and diabetes [116,219]. In the epidemiological literature, “tracking” is the name often given to the concept of persistence or relative stability of a risk factor over time [220,221,222].

A 2008 systematic review by Singh et al. reported on longitudinal studies investigating the persistence of childhood and adolescence overweight [219]. The review included 25 longitudinal studies of retrospective or prospective design, 13 of which were considered to be of high quality. Most of the high quality studies were published after 2001.

All of the studies included in the review reported that overweight or obese children and adolescents had an increased risk of becoming overweight or obese adults. Four studies
included analyses stratified for different levels of body composition (BMI) and these studies showed that persistence of overweight increased with increasing degree of overweight. Most of the studies that included several measurements during childhood and adolescence showed that persistence of weight status into adulthood increased with age. Some studies reported that greater persistence of weight status in girls than in boys but others reported contrasting findings. Overall, the high quality studies indicated that the risk of overweight children becoming overweight or obese adults is at least twice that of children of normal weight with the highest odds ratio reported by a high quality study being 10. For obese children, the relative risks or odds ratios were generally greater. For overweight adolescents, one high quality study reported a relative risk for adult overweight of 4.3 and one high quality study reported odds ratios for adult obesity of 15 for boys and 12 for girls. Three high quality studies reported percentages of overweight adolescents who became overweight adults which ranged from 22% to 58%. Overall the percentage of obese adolescents who became overweight or obese adults was higher than the percentage of obese children. In three high quality studies it ranged from 24% to 90%.

The review authors concluded that there was strong evidence for “moderate” persistence of childhood and adolescent overweight into adulthood but there was considerable variation in predictive values. They noted that the study subjects had grown up in a less obesogenic environment than the current one which may mean that the persistence of overweight will be different for today’s children. They also noted that the prevalence of overweight in adults is much higher than it is in children indicating that most overweight adults were not overweight as children.

An earlier 1993 review by Serdula et al. also concluded that obese children are at increased risk of becoming obese adults [223]. Differences between studies in study design, definitions of obesity and analytic methods made it difficult to quantify the correlation between childhood and adult obesity.

The consequences of childhood obesity
There are both short term and long term consequences of childhood obesity. The short term consequences that may affect a child’s quality of life are largely limited to severely obese children. They include sleep problems, asthma, type 2 diabetes, polycystic ovary syndrome, orthopaedic disorders (including slipped upper femoral epiphysis) and psychological and social distress [224]. In addition, a number of markers for subclinical coronary artery disease and atherosclerosis are measurable in obese children including elevated blood pressure and serum concentrations of total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol [225]. The long term consequences of childhood obesity are probably mostly related to the fact that obese children have a high probability of becoming obese adults with increased risks of metabolic syndrome, type 2 diabetes and cardiovascular disease, since adults who have normal BMI but were obese as children have similar cardiovascular risk profiles to those who have always had normal BMI [226].

New Zealand data on children’s and young people’s hospital admissions for slipped upper femoral epiphysis, type 2 diabetes and bariatric surgery is presented in the consequences of obesity chapter beginning on page 316.

Sleep problems
Obstructive sleep apnoea is one of the most serious problems that can affect obese children and it is more common among those who are severely obese. Parents may notice loud snoring with pauses in breathing, restless sleep and daytime sleepiness. Disturbed sleep can result in poor attention, poor school performance and bed-wetting. Obstructive sleep apnoea can lead to right ventricular hypertrophy and pulmonary hypertension. Diagnosis is made via polysomnography. Treatment options include removal of tonsils and adenoids if these are enlarged and continuous positive airway pressure (CPAP) therapy during sleep [227].
Obesity hypoventilation syndrome occurs in severely obese patients when the weight of fat on the chest and abdomen impairs breathing. The symptoms are similar to those of obstructive sleep apnoea [227].

**Asthma**

According to a 2013 review by Papoutsakis et al., the recent evidence from the epidemiological literature indicates that there is a weak but significant association between high body weight and asthma in children [93]. The association appears to be more pronounced where there is central obesity and where the asthma is not related to allergies. Some prospective studies have found that high body weight precedes asthma symptoms. A 2012 Cochrane review identified no published RCTs on the effect of either weight loss or weight gain on asthma symptoms in children but stated that a few studies were in progress [228].

**Type 2 Diabetes**

Type 2 diabetes is one of the most serious complications of childhood obesity [227]. Most overweight children have significant metabolic abnormalities due to insulin resistance even if they have no evidence of type 2 diabetes [229]. It has been reported that the incidence of type 2 diabetes in children < 15 years in Auckland increased five-fold from 1995 to 2007, from 0.5 per 100,000 to 2.5 per 100,000 (representing about 10% of all new cases of diabetes among children and adolescents in the Auckland region) [230]. The average annual incidence over the period was 1.3 per 100,000 overall and there were significant ethnic disparities with an average annual incidence (per 100,000) of 0.1 in Europeans and 3.4 in both Māori and Pacific children. Unlike type 1 diabetes, type 2 diabetes is often asymptomatic and therefore diagnosis requires laboratory testing. Obese or overweight children with a family history of diabetes, especially Māori and Pacific children, are at particular risk of developing type 2 diabetes.

**Polycystic Ovary syndrome**

Obese adolescent women may develop Polycystic Ovary syndrome (POCS) which is the combination of polycystic ovaries (visible on ultrasound) with hyperandrogenism (which can result in severe acne), anovulatory menstrual cycles or oligomenorrhea, and hirsutism (excess facial and body hair). Not only does POCS lead to fertility and cosmetic problems, it is also a risk factor for future metabolic syndrome, type 2 diabetes and cardiovascular disease [229].

**Orthopaedic disorders, including slipped upper femoral epiphysis**

Children's cartilaginous bones and unfused growth plates predispose them to obesity-related orthopaedic problems [224]. Dislocation of the femoral growth plate, known as slipped upper femoral epiphysis (SUFE), can lead to permanent damage to the femoral head. At least 50% of patients with SUFE are obese and SUFEs occur at significantly younger ages among obese children than non-obese children [224]. Blount’s disease involves bowing of the legs in response to unequal or early excess weight bearing. Treatment can require multiple surgical osteotomies. Severe infantile (under three years) Blount’s disease seems to be associated with obesity with one study reporting that 12 out of 18 patients with Blount’s disease at the Boston Children’s Hospital Medical Center had weights above 120% of ideal body weight for their age and sex and all of the “obese” patients had infantile Blount’s disease [231].

**Psychological distress, stigmatisation and bullying**

Parents of obese children often worry most about the emotional consequences of childhood obesity [173,232] and research indicates that there are good reasons for this. Obesity has been said to be one of the “most stigmatising and least socially acceptable conditions in childhood” [233]. One cross-sectional study of 106 obese children and adolescents (mean BMI 34.7 kg/m²) referred to an academic children’s hospital found that obese children and adolescents were more likely to have impaired health-related quality of life (QOL) than healthy children (odds ratio 5.5; 95% CI 3.4–8.7) and were similar to children and adolescents diagnosed with cancer (OR, 1.3; 95% CI, 0.8–2.3). Compared to healthy children, the obese children had impaired quality of life in all five QOL domains both by self-report and parent proxy report, as shown by unadjusted odds ratios which
ranged from 4.0 (95% CI 2.4–6.5) for self-reported school functioning to 13.6 (95% CI 8.2–22.5) for parent-reported overall psychosocial health (comprising emotional, social and school functioning) [233].

From a review of 17 studies, Tang-Peronard and Heitman concluded that overweight girls seemed to experience a greater degree of stigmatisation than overweight boys and were more often teased about their weight and bullied verbally, relationally and physically and also more socially marginalised with respect to friendships and romantic relationships [234]. A British prospective cohort study also found gender differences with respect to bullying [101]. At the age of 8.5 years, after adjustment for social class, obese boys were 1.66 (95% CI 1.04–2.66) times more likely to be overt bullies and 1.54 (95% CI 1.12 to 2.13) times more likely to be overt victims. Compared to average weight girls, obese girls were 1.53 (95% CI 1.09–2.15) times more likely to be overt victims. The authors of this study suggested that the physical dominance of the obese boys in their peer group allowed some of them to become the perpetrators rather than the victims of bullying.

Childhood obesity may be associated with emotional and behavioural problems from a very young age. The British Millennium Cohort Study (11,202 children) used the Strengths and Difficulties Questionnaire to assess the relationship between obesity and behavioural problems in three and five year old children [105]. Adjusted linear and multinomial regression analyses indicated that at age three, compared to normal weight children, obese boys had more conduct problems, and obese girls had more prosocial behaviours. At age five, obese boys had more conduct problems, hyperactivity and inattention problems, peer relationship problems and total difficulties. Obese girls only had more peer relationship problems.

**Later life consequences of childhood obesity**

Most of the concern about rising rates of childhood obesity is based on the belief that society is facing a future tidal wave of obesity-related health problems that will overwhelm the health system [235,236]. As has been discussed earlier, obese children are at higher risk of becoming obese adults than normal weight children and this risk is greater for obese older children and adolescents than obese young children and for the most severely obese children [237]. Most overweight or obese adults, however, were of normal weight in childhood but gradually gained weight through adulthood to become overweight or obese in later life [238,239], so clearly childhood obesity is not the only predictor of adult obesity.

Reilly and Kelly conducted a systematic review of the more recent evidence (published from January 2002–June 2010) on the long term impact of child and adolescent obesity on premature mortality and physical morbidity in adulthood [3]. They identified five studies on the association between child or adolescent overweight or obesity and premature mortality, four of which found a significantly increased risk. All of the eleven studies on cardiometabolic morbidity reported that overweight and obesity were associated with increased risk of adult cardiometabolic disease (diabetes, heart disease, hypertension and stroke) with the hazard ratios ranging from 1.1 to 5.1. Nine studies examined other adult morbidity. Associations between child or adolescent overweight and cancer were inconsistent, but there were associations with increased adult risk of receiving a disability pension, asthma and symptoms of polycystic ovary syndrome. Reilly and Kelly stated that many of the studies in their review attempted to consider the issue of the relative contributions of child and adolescent obesity per se and the tendency of childhood obesity to persist into adult life by adjustment for current (adult) weight status. Some studies found that the associations between child and adolescent obesity and adult outcomes were attenuated after adjustment for adult weight status but others found such adjustment had a negligible effect.

**Does childhood obesity have adverse consequences if adult weight is normal?**

Although many studies have demonstrated positive associations between childhood obesity and adult cardiovascular risk Lloyd et al. have argued that it is uncertain whether there is an effect of child adiposity on adult cardiovascular risk that is independent of the degree of adult adiposity or whether the observed associations merely reflect the tracking
of childhood obesity into adulthood [106]. These authors undertook a systematic review of longitudinal studies (published prior to July 2008) investigating the association between childhood BMI and adult cardiovascular disease (CVD) with two objectives: firstly to report on the strength of the association between childhood obesity and adult CVD risk, and, secondly, to investigate whether the effects of childhood obesity are independent of adult BMI status. They identified 16 studies meeting their inclusion criteria, all but one published in the ten years prior to 2008. Most studies took account of age and gender but not socioeconomic status and most treated childhood BMI as a continuous variable. There was considerable variation in age of childhood BMI measurements (ranging from two to 18 years) and adult outcome measurements (18 to 71 years).

Eight studies considered the relationship between childhood BMI and adult blood pressure, six the relationship with carotid intima-media thickness (CIMT, a marker of atherosclerosis and increased risk of coronary heart disease and stroke), and three the impact on incidence of coronary or ischaemic heart disease and stroke. Lloyd et al. stated that only two studies reported evidence of an independent positive relationship between childhood BMI and adult blood pressure and neither of them included the relevant adjusted data (i.e. confidence intervals or correlation statistics). One of them was the study with the fewest participants and it measured adult blood pressure at a young age (18 to 26 years). For these reasons, Lloyd et al. considered that the evidence for an independent association between childhood BMI and adult blood pressure was weak. In contrast, three studies which adjusted for adult BMI provided evidence of a negative relationship between childhood BMI and adult blood pressure, and two of them measured adult blood pressure at older ages (45 and 50 years) and could therefore be considered to provide a better indication of lifetime risk of hypertension. Lloyd et al. stated that the findings of these studies suggest that it is those who had low BMI in childhood but became overweight as adults who have the greatest risk of high blood pressure. Two studies, both using data from the Bogalusa Heart Cohort, reported a positive independent association between childhood BMI and adult CIMT but Lloyd et al. considered that these studies had weaknesses in their design and interpretation. The remaining studies showed no association between childhood BMI and adult CIMT after adjustment for adult BMI although all except one measured CIMT in young adulthood. Lloyd et al. therefore concluded that there was little evidence for an independent relationship between childhood BMI and adult CIMT and the limited existing evidence should be considered weak.

Two studies reported a positive association between childhood BMI and mortality from coronary or ischaemic heart disease but neither adjusted for adult BMI and so did not provide evidence for an independent effect of childhood BMI. Another study showed that men who died from coronary heart disease had a higher childhood BMI than those who did not, but these men had a mean BMI on the sixtieth centile for the population as a whole and therefore were well within the normal weight range.

In summary, Lloyd et al. found that there was little evidence that childhood obesity is an independent risk factor for increased blood pressure, CIMT or cardiovascular disease morbidity or mortality and that what evidence existed was weak. They stated that the approach and targeting of obesity interventions requires careful consideration and that while it is important for interventions to target the stages of life which offer the greatest long-term benefits, it is important to avoid the potential for negative consequences of obesity prevention or treatment programmes if they coincide with critical stages of neurological, behavioural and physical development.

In another systematic review, Lloyd et al. looked at studies investigating the associations between childhood BMI and markers of adult metabolic syndrome and whether or not any associations were independent of adult BMI [107]. They identified eleven studies published up until July 2010 which fulfilled their inclusion and exclusion criteria and most of these (seven studies) did not adjust for adult BMI. While several studies found positive associations between childhood BMI and adult total cholesterol, low density lipoprotein cholesterol, triglyceride and insulin concentrations, these associations were attenuated or reversed after adjustment for adult BMI or body fatness. None of the four studies that
considered adult metabolic syndrome as an endpoint found evidence of an independent association with childhood obesity. Lloyd et al. concluded that there was little evidence that childhood obesity is an independent risk factor for adult metabolic syndrome or its markers. They noted that data from studies which adjusted for adult BMI showed a weak negative association between childhood BMI and adult metabolic variables with those who were at the lower end of the BMI in childhood, but obese in adulthood, at particular risk.

Juonala et al. analysed data from four prospective cohort studies (6328 subjects) that followed participants from childhood into adulthood (mean length of follow up 23 years): the Bogalusa Heart Study (conducted in the U. S.), the Muscatine Study (U.S.), the Childhood Determinants of Adult Health Study (Australia), and the Cardiovascular Risk in Young Finns Study (Finland) to determine whether childhood obesity increases cardiovascular risk independent of adult BMI [226]. They found that, compared to those who had normal BMI as children and became non-obese adults, subjects who had a consistently high BMI from childhood through adulthood had an increased risk of type 2 diabetes (relative risk, 5.4; 95% confidence interval 3.4 to 8.5), hypertension (RR 2.7; 95% CI 2.2 to 3.3), elevated low-density lipoprotein cholesterol levels (RR 1.8; 95% CI 1.4 to 2.3), reduced high-density lipoprotein cholesterol levels (RR 2.1; 95% CI 1.8 to 2.5), elevated triglyceride levels (RR 3.0; 95% CI 2.4 to 3.8), and carotid artery atherosclerosis (increased intima-media thickness of the carotid artery) (RR 1.7; 95% CI 1.4 to 2.2). In contrast, subjects who were overweight or obese during childhood but were non-obese as adults had risks of the outcomes that were similar to those of subjects who had a consistently normal BMI from childhood to adulthood (p >0.20 for all comparisons).

**Conclusions**

The prevalence of childhood obesity has increased significantly over the last thirty years, in New Zealand and in other developed countries. This is seen by many as a public health crisis, heralding a future epidemic of adult obesity and a tidal wave of obesity-related health problems, including diabetes (and consequent renal failure), hypertension, cardiovascular disease and cerebrovascular disease, which could overwhelm the health system. This is because childhood obesity, particularly severe childhood obesity, is a strong predictor of adult obesity, although it must be remembered that most currently overweight and obese adults were not obese as children. While there are obesity-related physical health problems for children, these are mostly confined to the relatively few severely obese children. It is the social and mental health consequences of childhood obesity which are of most concern to parents. Obesity is a stigmatised condition and obese children may face social exclusion, teasing and bullying.

There are both genetic and environmental factors which contribute to a child’s chances of being obese. The many different contributing factors interact to form a complex web of potential determinants of childhood obesity [113]. These factors include maternal under or over nutrition, maternal smoking, in-utero growth restriction, maternal diabetes, high birth weight, low birth weight followed by rapid catch-up growth, lack of breast feeding, short sleep duration, parental overweight and obesity, low parental education and/or socio-economic status, low physical activity, high screen time, unhealthy food and beverages, food marketing practices, food prices, food insecurity and a built environment that discourages outdoor play and walking and cycling.

Many children start on the pathway to obesity very early in life. Obesity is not evenly distributed among the child population. Children of obese parents are at high risk of obesity for both genetic and environmental reasons, since parents are largely responsible for the food available to young children. Obesity also disproportionately affects poor and Māori and Pacific children. The cost of healthy food is a barrier to healthy eating for many families.
THE DISTRIBUTION OF OVERWEIGHT AND OBESITY IN CHILDREN AND YOUNG PEOPLE

Introduction

Increasing rates of childhood obesity are of concern, both in New Zealand and internationally [2,91]. This is because childhood obesity has significant adverse effects on health in childhood and beyond [3,5]. During childhood, obesity has been associated with adverse psychological effects, lower educational achievement [5,240], asthma, and slipped upper femoral epiphysis [4,93,97]. Childhood obesity has also been strongly associated with the clustering of cardiovascular risk factors which tend to persist thus increasing the risk of cardiovascular disease in later life [5]. In the longer term, childhood overweight and obesity may also increase the risk of cardio-metabolic diseases (diabetes, hypertension, ischaemic heart disease and stroke), premature death, and polycystic ovary syndrome for those whose obesity persists into adulthood [3].

Obesity is determined by a complex mix of environmental factors, life course factors, intergenerational factors and underlying biology [109]. Risk factors linked to childhood obesity and overweight include early rapid weight gain, high birth weight, and maternal pre-pregnancy overweight, maternal smoking in pregnancy, short sleep duration and prolonged television viewing [113,152]. Parental obesity is also a strong predictor of childhood obesity [109,115]. Prolonged breastfeeding (> 4 months) and delaying the introduction of solids until six months are probably modestly protective factors [241]. Societal changes in the production and marketing of food, transportation, and work patterns have also been implicated in the creation of an ‘obesogenic environment’ that predisposes children to weight gain [109,115].

The following section begins with a brief review of the measurement of overweight and obesity, before exploring their prevalence in New Zealand children and young people using information from two data sources: The 2011/12 New Zealand Health Survey, and the Youth’12 Survey of secondary school students.

Notes on the Measurement of Overweight and Obesity

**Obesity**: Obesity is defined as an excess in adiposity or body fat mass. Measures of adiposity in current use include weight, weight for height, skin fold thickness (e.g. triceps/sub-scapular) and circumferences/diameters (e.g. waist-hip/waist-thigh ratios, mid-upper arm circumferences), each of which has its own reference standards and cut-off points [242]. Of these, the most popular is the Body Mass Index (BMI) which is calculated using the formula

\[
\text{BMI} = \frac{\text{weight (kg)}}{\text{height (m)}^2}
\]

Using height and weight to assess adiposity is generally viewed as being reliable, reproducible, non-intrusive and cheap, making BMI one of the most popular measures for obesity, both in New Zealand and overseas. In adults, cut-offs are based on mortality risk or other criteria, with those having a BMI of 25–29.9 kg/m2 being traditionally classified as overweight and those with a BMI of 30 kg/m2 or over being seen as obese. Using BMI to assess obesity in children however has a number of drawbacks, including the changes in body composition that occur as part of normal growth and with the onset of puberty, and ethnic differences in body composition for a given BMI [243]. These issues are discussed in more detail below.

**Changes in Body Composition with Age: The Need for BMI Percentile Charts**

Assessing obesity during childhood and adolescence is more complex than in adults, as both height and body composition change progressively with development. In particular, the proportion of fat mass to total body weight changes significantly during childhood, beginning at around 13–15% in term newborn infants and increasing progressively during the first year of life, to a maximum of 25–26% at 12 months of age. From 12 months to 4–6 years, the proportion of body fat then declines, to a nadir of around 12–16%, before increasing again between the ages of 6-10 years. By early adulthood, the proportion of fat mass is 20–25% for women and 15–20% for men [243]. As a result of these changes, when assessing the level of obesity in an individual child, BMI for age percentile charts are usually used, which extrapolate back the traditional adult cut-off points of 25–29.9 kg/m2 and ≥30 kg/m2, to the same points on the BMI distribution during the childhood years e.g. a male child with a BMI > 19.3 at the age of 5 years, is on the same point in the percentile charts as an 18 year old with a BMI of >30, and thus will be classified as obese [200]. As New Zealand to date has not developed its own BMI percentile charts for children, overseas standards must be used. Of these, the most popular were...
developed by the International Obesity Taskforce (see Cole [200,244]) using pooled survey data from a number of different countries.

**Ethnic Differences in BMI**

With no BMI-for-age percentile charts specifically designed for New Zealand use, there remains a significant amount of debate about the appropriateness of the traditional BMI-for-age cut-offs for New Zealand children of different ethnic groups. While a number of studies have suggested that, for a given BMI, Māori and Pacific children have a lower percentage of body fat [208,245,246], others have argued that while statistical differences may exist, there are no clinically significant ethnic differences in the relationship between BMI and body composition and that a common standard should be used for children of all ethnic groups [208]. Overseas research also suggests that ethnic differences in body composition may increase during puberty, with differences being much less marked amongst children <8 years of age [247]. Similarly, ethnic differences in the onset of puberty may also make utilisation of a common BMI cut-off difficult, with puberty occurring, on average, earlier amongst Māori and Pacific groups [166]. Such differences need to be kept in mind when interpreting ethnic specific obesity rates calculated using overseas percentile charts, as these rates may tend to overestimate obesity rates amongst Māori and Pacific children slightly.

**The 2011/12 New Zealand Health Survey**

The 2011/12 NZ Health Survey (2011/12 NZHS) [2] was a cross sectional survey carried out between July 2011 and August 2012, which collected information on 4,478 children aged from birth to 14 years. Information was collected in a similar way to the earlier 2006/07 NZ Health Survey, making it possible to compare overweight and obesity rates during these two periods. The following section briefly reviews changes in the prevalence of overweight and obesity in children aged 2–14 years between the 2006/07 and 2011/12 Surveys, before exploring the distribution of overweight and obesity by a range of socio-demographic factors in the most recent 2011/12 NZHS.

**Data Sources and Methods**

**Definitions**

- The proportion of children and young people aged 2–24 years who are overweight or obese by age
- The proportion of children aged 2–14 years who are overweight or obese by gender, ethnicity and NZDep06

**Data Sources**

The 2011/12 New Zealand Health Survey

The data on children aged 2–14 years in this section were derived from The Health of New Zealand Children 2011/12: Key Findings of the New Zealand Health Survey, and its associated data tables, downloadable at http://www.health.govt.nz/publication/health-new-zealand-children-2011-12 Regional results were sourced from http://www.health.govt.nz/publication/regional-results-2011-12-new-zealand-health-survey while data for young people aged 15–24 years was derived from The Health of New Zealand Adults 2011/12: Key Findings of the New Zealand Health Survey, and its associated data tables, which are downloadable at http://www.health.govt.nz/publication/health-new-zealand-adults-2011-12

**Notes on Interpretation**

**Sample Size and Weighting:** The 2011/12 NZHS [2] was a cross sectional survey carried out between July 2011 and August 2012, which collected information on 4,478 children aged from birth to 14 years, and 12,370 adults aged 15 years and over. The child survey included 2,968 European/Other, 1,592 Māori, 730 Pacific and 432 Asian children, with the data being weighted (e.g. to take into account response rates and individual item non-responses) to ensure that the results were representative of the total New Zealand child population. The weighted response rate for the child questionnaire was 85%, with the primary caregiver answering the questionnaire on their child’s behalf. In addition, height and weight measurements were taken on all children aged 2–14 years using standardised equipment and procedures.

**Ethnicity:** In the NZHS, four ethnic groups were reported: Māori, Pacific, Asian and European/Other. Total response ethnicity was used throughout, with an individual being included in the analysis for each ethnic group to which they affiliated. As a result, ethnic groups cannot be directly compared with each other, with adjusted rate ratios which compare children by ethnicity comparing those in a particular group (e.g. Pacific) with those who are not in that ethnic group (e.g. non-Pacific) [2].

**Age Standardisation:** Age is an important determinant of health status. In the NZHS, all time trend results have been age-standardised, so that populations can be compared over time, and so that any differences reported are not due to changes in the population age structure. Similarly all rate ratio comparisons by gender, ethnicity and NZ Deprivation Index (NZDep) decile have been age adjusted, so that any differences identified cannot be attributed to differing age structures between the population groups. The method of age standardisation used was the direct method, using the World Health Organization world population age distribution [2]. Regional rates however, are presented as unadjusted prevalences, so that the actual prevalence of those affected in each region can be assessed, including by age group.
Other Standardisation and the Relative Index of Inequality: In addition to age standardisation, any ethnic comparisons have also been adjusted for gender. For neighbourhood deprivation comparisons (i.e. by NZDep06) rate ratios refer to the relative index of inequality [2]. This compares neighbourhood deprivation after adjusting for age, sex and ethnic differences. The relative index of inequality can be interpreted in the same way as adjusted rate ratios, although it is calculated in a slightly different way.

Regional Results: NZHS results are reported by region, with DHBs being grouped as follows: Northern Region: Northland, Waitemata, Auckland, Counties Manukau; Midland Region: Waikato, Bay of Plenty, Lakes, Tairawhiti, Taranaki; Central Region: Hawke's Bay, Whanganui, MidCentral, Hutt Valley, Capital and Coast, Wairarapa; Southern Region: Nelson Marlborough, Canterbury, South Canterbury, West Coast, Southern. It is anticipated that results will become available by DHB in future years, as more survey data is collected.

Measurement of Overweight and Obesity: Overweight and obesity in the NZHS was measured using body mass index (BMI). BMI is calculated by dividing weight in kilograms by the square of height in metres (kg/m2). In the NZHS, for respondents aged 2–17 years, the age and sex specific BMI cut-off points developed by the International Obesity Taskforce (IOTF) were used to classify underweight, normal range, overweight and obesity. These BMI-cut-offs were designed to coincide with the adult cut-offs at 18 years [2].

Differences between estimates are said to be statistically significant when the confidence intervals for each rate do not overlap. Sometimes, however, even when there are overlapping confidence intervals, the difference between the groups can be statistically significant. Any differences between two variables where the confidence intervals overlap are tested using a t-test. The significance of a t-test is represented by the p-value. If a p-value is below 0.05, then we are 95 percent confident the difference between the two estimates is statistically significant [248].

Trends in Overweight and Obesity

Obesity Trends

Overall: The proportion of New Zealand children aged 2–14 years who were obese increased significantly (p=0.02) between NZ Health Surveys, with rates rising from 8.4% (95% CI 7.5–9.4) in 2006/07, to 10.3% (95% CI 8.9–11.9) in 2011/12.

By Gender: When broken down by gender, the proportion of boys aged 2–14 years who were obese increased significantly (p=0.05), with rates rising from 8.0% (95% CI 6.9–9.3) in 2006/07 to 10.2% (95% CI 8.4–12.3) in 2011/12. Rates however, did not increase significantly (p=0.17) for girls, being 8.8% (95% CI 7.3–10.4) in 2006/07 and 10.4% (95% CI 8.6–12.5) in 2011/12 (Figure 58).

By Ethnicity: When broken down by ethnicity, the proportion of Māori children aged 2–14 years who were obese increased significantly (p=0.03), with rates rising from 11.9% (95% CI 10.0–13.9) in 2006/07 to 16.4% (95% CI 12.5–20.9) in 2011/12. Obesity rates for children of other ethnic groups however did not increase significantly between 2006/07 and 2011/12: Pacific 23.1% (95% CI 19.7–26.8) → 25.5% (95% CI 20.7–30.7), Asian 5.9% (95% CI 3.7–8.8) → 7.6% (95% CI 4.6–11.7) and European/Other 5.6% (95% CI 4.5–6.9) → 6.2% (95% CI 5.0–7.6) (Figure 58).

Overweight (but not Obese) Trends

Overall: The proportion of New Zealand children aged 2–14 years who were overweight (but not obese) did not change significantly (p=0.81) between NZ Health Surveys, with rates being 21.0% (95% CI 19.4–22.8) in 2006/07, and 20.7% (95% CI 19.0–22.5) in 2011/12.

By Gender: When broken down by gender, the proportion of boys and girls aged 2–14 years who were overweight did not change significantly between NZ Health Surveys, with rates for boys being 20.6% (95% CI 18.5–22.8) in 2006/07 and 19.0% (95% CI 16.6–21.5) in 2011/12. For girls rates were 21.5% (95% CI 19.2–24.1) in 2006/07 and 22.6% (95% CI 20.1–25.2) in 2011/12 (Figure 58).

By Ethnicity: When broken down by ethnicity, the proportion of Māori, Pacific, Asian and European/Other children who were overweight did not change significantly between NZ Health Surveys. Rates for Māori children were 25.8% (95% CI 22.8–29.0) in 2006/07 and 27.2% (95% CI 23.5–31.2) in 2011/12, while rates for Pacific children were 31.3% (95% CI 27.8–35.0) in 2006/07 and 28.1% (95% CI 23.2–33.3) in 2011/12. Rates for Asian children were 14.6% (95% CI 11.3–18.5) in 2006/07 and 15.4% (95% CI 11.3–20.2) in 2011/12, while rates for European/Other children were 19.4% (95% CI 17.5–21.4) in 2006/07 and 18.8% (95% CI 16.8–20.9) in 2011/12 (Figure 58).
Figure 58. Proportion of Children Aged 2–14 Years who were either Overweight or Obese by Gender and Ethnicity, 2006/07 and 2011/12 New Zealand Health Surveys

Source: 2006/07 and 2011/12 New Zealand Health Surveys; Note: Rates have been age-standardised
Obesity: Distribution by Region

When broken down by region, the proportion of children aged 2–14 years in the Midland region who were obese increased *significantly* \((p=0.00)\) between NZ Health Surveys, with rates rising from 7.5\% (95\% Cl 5.9–9.3) in 2006/07, to 14.7\% (95\% Cl 11.1–18.9) in 2011/12. Obesity rates for Northern \((p=0.22)\), Central \((p=0.34)\) and Southern \((p=0.90)\) children however, did not change *significantly*. Rates for Northern children were 9.5\% (95\% Cl 8.1–11.0) in 2006/07 and 11.2\% (95\% Cl 9.0–13.7) in 2011/12. Rates for Central children were 8.6\% (95\% Cl 6.6–10.9) in 2006/07 and 10.3\% (95\% Cl 7.6–13.7) in 2011/12, while rates for Southern children were 7.2\% (95\% Cl 4.8–10.2) in 2006/07 and 7.4\% (95\% Cl 4.7–10.9) in 2011/12 (*Figure 59*).

*Figure 59. Proportion of Children Aged 2–14 Years Who Were Obese by Region, 2006/07 and 2011/12 New Zealand Health Surveys*

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age

**Current Distribution of Overweight and Obesity**

**Distribution by Age**

In the children’s component of the 2011/12 NZHS, there were no *significant* differences in the (unadjusted) prevalence of obesity by age, with rates being 9.2\% (95\% Cl 6.5–12.5) in those aged 2–4 years, 10.6\% (8.6–12.9) in those aged 5–9 years and 10.8\% (95\% Cl 8.7–13.2) in those aged 10–14 years. In the adult component of the 2011/12 NZHS however, obesity rates were *significantly* higher for those aged 18–24 years (22.9\% (95\% Cl 19.6–26.6\%)) than for those aged 15–17 years (12.0\% (95\% Cl 8.0–17.2\%)).

There were also no *significant* age differences in the (unadjusted) prevalence of children who were overweight (but not obese) in the child component of the 2011/12 NZHS. Nor were there any *significant* differences between those aged 15–17 years and 18–24 years in the adult component of the NZHS.

In addition, there were also no *significant* gender differences in the proportion of children age 2–14 years who were overweight or obese in the 2011/12 NZHS, once rates were adjusted for age. *Figure 60* reviews the (unadjusted) prevalence of children and young people aged 2–24 years who were either overweight or obese by gender and age in the 2011/12 NZHS.
Figure 60. Proportion of Children and Young People Aged 2–24 Years who were either Overweight or Obese by Gender and Age, 2011/12 New Zealand Health Survey

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted

Figure 61. Proportion of Children Aged 2–14 Years who were either Overweight or Obese by Gender and Ethnicity, 2011/12 New Zealand Health Survey

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age
**Distribution by Ethnicity**

In the 2011/12 NZHS, Māori children aged 2–14 years were 2.10 (95% CI 1.64–2.68) times more likely to be obese than non-Māori children, while Pacific children were 3.08 (95% CI 2.41–3.93) times more likely to be obese than non-Pacific children, once rates were adjusted for age and gender. There were no significant differences however, in obesity rates between Asian and non-Asian children.

Similarly, Māori children aged 2–14 years were 1.40 (95% CI 1.18–1.67) times more likely to be overweight (but not obese) than non-Māori children, while Pacific children were 1.44 (95% CI 1.17–1.76) times more likely to be overweight than non-Pacific children, once rates were adjusted for age and gender. In contrast, Asian children were significantly less likely (RR 0.68 95% CI 0.49–0.94) to be overweight than non-Asian children.

**Figure 61** reviews the proportion of children aged 2–14 years who were either overweight or obese by gender and ethnicity in the 2011/12 NZHS.

**Distribution by NZ Deprivation Index Decile**

In the 2011/12 NZHS, children aged 2–14 years who were living in the most deprived (NZDep06 deciles 9–10) areas were 2.33 (95% CI 1.37–3.93) times more likely to be obese than children living in the least deprived (NZDep06 deciles 1–2) areas, once rates were adjusted for age, sex, an ethnic group. When rates were further broken down by gender, these differences remained statistically significant only for girls.

Similarly, children aged 2–14 years who were living in the most deprived (NZDep06 deciles 9–10) areas were 1.72 (95% CI 1.20–2.46) times more likely to be overweight (but not obese) than children living in the least deprived (NZDep06 deciles 1–2) areas, once rates were adjusted for age, sex, an ethnic group. When rates were further broken down by gender, these differences remained statistically significant only for boys.

**Figure 62** reviews the proportion of children aged 2–14 years who were either overweight or obese by gender and NZ Deprivation Index decile in the 2011/12 NZHS.

![Figure 62. Proportion of Children Aged 2–14 Years Who Were Either Overweight or Obese by Gender and NZ Deprivation Index Decile, 2011/12 New Zealand Health Survey](image-url)
The Youth’12 Survey

Youth’12 was the third national survey of Year 9–15 students (Years 14 and 15 are those students who are repeating Years 12 and 13) in New Zealand, with previous surveys being undertaken in 2001 and 2007. The Youth’12 sample included 8,500 students aged 13–17+ years, which was approximately 3% of the 2012 New Zealand secondary school roll [249].

Data Sources and Methods

Definitions
The proportion of secondary school students aged 13–17+ years who were overweight or obese

Data Sources
The data on overweight and obesity in this section is derived from The Youth’12 Overview: The Health and Wellbeing of New Zealand Secondary School Students in 2012 [250], and its companion document the Youth’12 Prevalence Tables [249].

Notes on Interpretation

Survey Methodology and Sample: Youth’12 is the third national health and wellbeing survey of secondary school students in New Zealand, produced by the Adolescent Health Research Group, with previous surveys being undertaken in 2001 and 2007. The Youth’12 Survey was a random survey of composite and secondary schools. For schools with more than 150 Year 9–15 students, a random sample of 20% of the roll were invited to participate, while for schools with a roll of <150, 30 students were randomly asked to participate. Overall, 73% of invited schools took part, with 68% of students who were invited to participate agreeing to do so. This resulted in total sample of 8,500 students (3% of the 2012 New Zealand secondary school roll). Students were asked to provide their address to determine their census meshblock (NZDep 2006), and the student’s height and weight was measured using standardised measurement protocols [249].

Body Mass Index: Body mass index (BMI) was calculated using measured height and weight. The percentage of students who were overweight and obese was determined using age and sex specific BMI definitions for children and adolescents, as recommended by the International Obesity Taskforce [251].

Ethnicity Reporting: The Youth’12 ethnicity question was based on the 2001/2006 NZ Census ethnicity question, which asked students which ethnic group they belonged to, with multiple responses being possible. Students who had selected more than one ethnic group were also asked “Which is your main ethnic group (the one you identify with the most)”? Possible options included “I can’t choose only one ethnic group”. For the purposes of comparing ethnic groups, Statistics NZ’s ethnicity prioritisation methods were used [252], which reported five ethnic groups: Māori, Pacific, Asian, European and Other.

Comparison Between 2007 and 2012 Surveys

In the Youth’12 survey, 24.1% (95% CI 22.8–25.4) of students were overweight, and 12.6% (95% CI 10.1–15.1) were obese, as compared to the Youth’07 survey where 24.2% (95% CI 22.7–25.6) of students were overweight and 10.4% (95% CI 8.8–11.9) obese.

Distribution by Gender

In both the Youth’07 and Youth’12 surveys, there were no significant differences in the proportion of males and females that were overweight or obese. In the Youth’12 survey, 23.4% (95% CI 21.9–24.8) of males were overweight, and 12.1% (95% CI 9.9–14.2) were obese, while 24.7% (95% CI 22.8–26.5) of females were overweight, and 13.1% (95% CI 9.6–16.6) were obese (Figure 63).

Distribution by Age, NZ Deprivation Index Decile and Geography

In the Youth’12 survey, there were no significant age differences (by single year of age) in the proportion of students who were overweight or obese. However, the proportion of students who were overweight was significantly higher for those from the most deprived (high=NZDep deciles 8–10) areas (28.2% (95% CI 26.1–30.4)) than for those from the least deprived (low=NZDep06 deciles 1–3) areas (21.4% (95% CI 19.6–23.3)). Similarly, the proportion who were obese was significantly higher for those from the most deprived areas (21.9% (95% CI 17.2–26.6)) than for those from the least deprived areas (7.0% (95% CI 5.8–8.2)). There were no significant rural vs. urban differences in the proportion of students who were overweight or obese. The proportion who were overweight was 24.6% (95% CI 23.2–26.0) in urban areas and 21.3% (95% CI 18.6–24.0) in rural areas. Similarly the proportion who were obese was 13.3% (95% CI 10.4–16.2) in urban areas and 9.1% (95% CI 7.4–10.9) in rural areas (Figure 64).
Figure 63. Proportion of Secondary School Students Aged 13–17+ Years who were Underweight, a Healthy Weight, Overweight or Obese by Gender, New Zealand Youth’07 and Youth’12 Surveys

Source: Youth’07 and Youth’12 Surveys

Figure 64. Proportion of Secondary School Students Aged 13–17+ Years who were Overweight or Obese by Gender, Age and NZ Deprivation Index, New Zealand Youth’12 Survey

Source: Youth’12 Survey; Note: NZDep2006: Low = Deciles 1–3; Medium = Deciles 4–7; High = Deciles 8–10

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Local Policy Documents and Evidence-Based Reviews Relevant to Overweight and Obesity in Children and Young People

In New Zealand a number of policy documents and reviews consider the prevention and management of overweight and obesity. These are briefly summarised in Table 105, along with a range of guidelines and reviews which consider these issues in the overseas context. In addition an in-depth topic commencing on Page 287 reviews The Determinants and Consequences of Overweight and Obesity, while a second in-depth topic commencing on Page 332 reviews The Treatment of Obesity in Children and Adolescents.

Table 105. Local Policy Documents and Evidence-Based Reviews Relevant to Overweight and Obesity in Children and Young People

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
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<tr>
<td>The Healthy Eating – Healthy Action strategic framework (Minister of Health 2003) and Implementation Plan (2004) addressed three of the New Zealand Health Strategy priorities: to improve nutrition, reduce obesity and increase the level of physical activity. The documents provide a planning tool and integrated policy framework for achieving the objectives, aimed at a variety of sectors and service providers including the Ministry of Health, District Health Boards and the Health Research Council. There are suggestions for action, milestones and progress measures in the key priority areas of lower socioeconomic groups, children, young people and their whānau (including older people), environments, communication and workplace. The background document (Ministry of Health 2003) provides the scientific evidence that underpins the Strategy. It is based on a review of the literature and extensive public consultation. It provides a summary of the issues identified in the areas of nutrition, physical activity, obesity, and includes chapters focusing on these issues for Māori, Pacific and South Asian populations.</td>
<td>258</td>
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<td>This paper provides an analysis of the usefulness and feasibility of an indicator to monitor obesity in children and young people and the effectiveness of strategies and interventions for the prevention and management of childhood obesity. It was intended as a guide for policy and funding decisions in the Ministry of Health, other government departments and District Health Boards. The report includes a literature review and consultation with experts to identify the best indicator: age-related body mass index (BMI) percentile. Ethical considerations including consent, privacy, psychosocial risks and costs are discussed and practical issues including the setting, the personnel requirements and the data collection process are described. The report concludes with a discussion of how the introduction of a systems performance indicator of obesity in childhood can contribute to the overall goal of reducing the prevalence of childhood obesity, and thus obesity and related disease in adulthood.</td>
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This updated review examined the effectiveness of interventions intended to prevent obesity in children, assessed by change in Body Mass Index (BMI). Fifty-five controlled trials (with and without randomisation) were included in the review, mostly targeting children aged six to 12 years. The meta-analysis (37 studies, 27,946 children) demonstrated that, overall, programmes were effective in reducing adiposity (standardised mean difference in BMI/zBMI -0.15kg/m², 95% CI -0.21 to -0.09), although not all programmes were effective and there was a high degree on unexplained heterogeneity. Intervention effects by age subgroups were: -0.26kg/m² (95% CI; -0.53 to 0.00) (0–5 years), -0.15kg/m² (95% CI; -0.23 to -0.08) (6–12 years), and -0.09kg/m² (95% CI; -0.20 to 0.03) (13-18 years). Eight studies reported on adverse outcomes but there was no evidence of adverse effects including unhealthy dieting or increased prevalence of overweight, or increased inequalities in health (assessed in fewer studies). The authors conclude that there is strong evidence to support targeted obesity prevention programmes, particularly those targeted at six to 12 year olds, although the heterogeneity and likelihood of small study bias means that results should be interpreted with caution. It was not possible to identify specific beneficial components but a number of promising strategies are identified including: school curriculum that includes healthy eating, physical activity and body image; increased sessions for physical activity and movement skills throughout the school week; improvements in nutritional quality of the food supply in schools; environments and cultural practices that support children eating healthier foods and being active; staff support and training; and parent support and home based activities. Further research needs to focus on how effective intervention components can be embedded in health and education systems to achieve long-term, sustainable benefits.
This review sought to identify the most effective behavioural models and behaviour change strategies used in preschool
and school-based interventions aimed at preventing obesity in four to six year olds. Twelve studies with at least six
month follow-up (4 RCTs, 5 cluster RCTs and 3 controlled trials), three of which did not have full data available, were
included in the review. The most commonly used model was social cognitive theory (SCT)/social learning theory (SLT)
(where behaviour change is viewed as an interaction between personal, e.g. skills and self-efficacy, behavioural, e.g.
modelling and rewarding/reinforcement, and environmental factors, e.g. provision of fruit). A narrative synthesis was
undertaken due to study variability. Interventions that were based on SCT/SLT, that combined high levels of parental
involvement with interactive school-based learning, that targeted physical activity and dietary change, and that had long-
term follow-up, were most effective. The authors recommended that in addition to high parental involvement and
programmes targeting both dietary and physical activity changes, interventions should also focus on developing
children’s (and parents’) perceived competence at making dietary and physical changes. The conclusions should be
interpreted with caution given that most of the data were provided by parents and carers, and were open to positive
reporting bias.

Parental involvement was widely advocated as important for school-based interventions. This review examined the
effectiveness of parental involvement in school-based obesity prevention interventions in children and adolescents. Only
five studies (4 cluster RCTs and 1 RCT), four of which focused on both nutrition and physical activity behaviours and one
targeted physical activity in combination with alcohol use prevention, met inclusion criteria. Some positive effects of
parental involvement were identified, in particular interventions which used parental modules including different
strategies and addressing several home-related determinants and parenting practices concerning eating and physical
activity behaviours. However, no conclusive evidence was identified concerning the added value of parent involvement,
due to the paucity of studies testing this hypothesis. The authors conclude that further research, comparing interventions
with and without a parental, is required.

This review assessed the effectiveness of child and adolescent weight-related health interventions with a focus on levels
of parental participation. Thirty-six RCTs (7,455 participants, range six to 1,029) with 42 interventions were included in
the review. Interventions included education on nutrition, physical activity or behaviour, physical activity sessions,
behaviour therapy, or a combination of these. Seventeen were preventive interventions, and 25 were treatment
interventions for overweight or obese children or adolescents. Ten interventions did not include parents, nine
interventions provided the option for parents to participate, and 23 required parents’ participation. Study quality was not
reported. Interventions that required parental participation (24 interventions) had greater success than interventions with
no parent participation (11 studies) \( (p=0.027) \). No significant differences in success rates were found between
interventions with different age groups, prevention compared to treatment and single compared to multiple activity
interventions. Among the prevention studies only, parent participation \( (p=0.01) \) and intervention duration \( (p=0.006) \) were
significant positive predictors of intervention effectiveness. The authors conclude that interventions that include parental
participation are likely to be more effective in reducing BMI in children and adolescents than those without.

This review examined the effectiveness of lifestyle interventions incorporating a dietary component on both weight
change and cardio-metabolic risks in overweight/obese children. Thirty-eight controlled trials were included in the review,
33 of which had complete data for meta-analysis on weight change; 15 reported serum lipids, fasting insulin, or blood
pressure. Most of the studies were conducted in children and/or adolescents aged over five years. The number of
participants ranged 16 to 258 (median 72). Lifestyle interventions produced significant weight loss compared with no-
treatment control conditions (12 studies, 899 participants); pooled BMI reduction 1.25 kg/m\(^2\) (95% CI 0.32 to 2.18) and
BMI z score reduction of 0.10 (95% CI 0.02 to 0.18). Studies comparing lifestyle interventions to usual care also resulted
in significant immediate and post-treatment effects on BMI up to one year from baseline. Weight loss was greater when
the duration of treatment was longer than six months. Significant improvements were also identified in low-density
lipoprotein cholesterol, triglycerides, fasting insulin and blood pressure up to one year from baseline. No differences were
found for high-density lipoprotein cholesterol. The heterogeneity of the included studies made it difficult to give definitive
recommendations for practice, although a variety of components appeared to be more effective, including family
involvement and a structured exercise training programme. The longer term effectiveness of interventions, particularly on
cardiovascular outcomes, remains unknown.

This review assessed the effectiveness of interventions aimed at preventing overweight and obesity in pre-adolescent girls. Thirty studies met the inclusion criteria (4 cluster RCTs, 14 RCTs, 11 controlled trials and 1 cohort pre–post trial), most of which were set in schools and included a combination of physical activity and nutrition components. Trials lasted at least three months and 19 lasted at least 12 months. Results for girls had to be reported separately. Data extraction for the calculation of effect sizes was possible for 21 studies. There were 66 effect sizes of less than 0.2 (no effect), 56 categorised as low, 16 as medium and two as high. While interventions (including reducing sedentary behaviours and modifying school food provision) aimed at pre-adolescent girls had potential to reduce the risk factors associated with childhood overweight and obesity, the sustainability of intervention effects was unclear and it was difficult to arrive at simple recommendations for best practice from the existing evidence.


This review assessed the effectiveness of interactive electronic media (CD-ROM, internet and emails, internet only and telemedicine) interventions for the prevention or treatment of obesity and/or obesity-related behaviours in children and adolescents (18 years and under). Twenty-four studies of 21 interventions (11 RCTs, 5 non-RCTs and 5 other study designs) were included in the review (5,812 participants, study size from 35 to 2,840 participants). Most of the studies were conducted in the US. Among children (4 studies) three studies showed limited benefits on the prevention of obesity and one study demonstrated modest benefits of home-based internet behaviour programmes on treating obesity. Among adolescents (20 studies) six studies showed modest benefits on prevention; six studies were poor quality and did not provide adiposity outcomes, and eight studies showed benefits on treating obesity in terms of body mass index, body fat, psychosocial factors, diet and physical activity. The authors conclude that while electronic intervention appeared to show promise for prevention and treatment of obesity in children and adolescents, the results of the review should be viewed with caution due to the poor quality of studies and further research is required.


This review assessed the effectiveness and cost-effectiveness of weight management schemes for the under fives. Inclusion criteria were restricted to controlled trials with objective measures, eliciting only four effectiveness RCTs for inclusion in the review. There were no treatment or cost-effectiveness studies. One trial showed a statistically significant difference between groups with significantly smaller increases in BMI in children in the intervention arm (0.48 versus 1.14; p=0.008). None of the trials reported any statistically significant differences in weight or physical activity. Although there were positive trends in weight measures these changes failed to reach statistical significance and the authors conclude the further RCTs are required. Potential important intervention features which require further assessment are identified, including cultural sensitivity, sustained moderate to vigorous exercise, active engagement of the parents in the programme and as role models of healthy living, and active engagement of the children in nutrition education.


This review assessed the effect of immersion treatment (weight loss camps and residential programmes) on changes in weight status among obese children under 18 years of age. The review included 22 studies, only one of which had a control group (obese waiting list children) and five had a comparison group and only one study randomly assigned participants. Interventions lasted at least 10 days, and included controlled diet, activities, nutrition education, and therapy and/or education regarding behaviour change. Eleven programmes included long-term follow-up evaluations at four months to 3.6 years. All the studies achieved weight loss among participants, with an average reduction in per cent overweight of 23.9% from pre- to post-immersion and 20.6% from pre-immersion to follow-up. The authors compared these results with the results of a separate meta-analysis published in 2007 of outpatient programmes targeting lifestyle changes which found reductions in per cent-overweight of 8.2% during treatment and 8.9% at follow-up [254], stating that immersion programmes produced an average of 191% greater reductions in per cent-overweight at post-treatment and 130% greater reduction at follow-up. These results should be interpreted with extreme caution given that these interventions have not been directly compared and there is no indication that the population is the same. It is also of note that both authors are employed by a company that provides immersion treatment for obese children.


This review assessed the evidence for interventions designed to prevent or reduce overweight and obesity in children younger than two years. Twelve articles representing 10 studies (5 RCTs and 7 non-randomised) were included in the review, assessing educational interventions to promote dietary behaviours or a combination of nutrition education and physical activity. Intervention durations were generally less than six months. Studies were assessed as poor or fair quality. Overall, studies found modest success in affecting outcome measures such as dietary intake and parental attitudes and knowledge about nutrition; however, no intervention improved child weight status. The authors conclude that further rigorous research is required to assess whether intervention targeting children in this age group have clinically important and sustainable effects.
This review assessed the effectiveness of bariatric surgery in paediatric patients. Eighteen studies were included in the review, assessing laparoscopic adjustable gastric banding (LAGB) (8 studies, 352 participants), Roux-en-Y gastric bypass (RYGB) (6 studies, 161 participants) and other surgical procedures (5 studies, 158 participants). The average age of participants was 16.8 years (range 9–21). All but one of the studies were retrospective and only one study had a (non-similar) control group, limiting the reliability of the findings. Meta-analysis indicated sustained and significant BMI at longest follow up for LAGB and RYGB but both procedures were associated with complications, which were more severe for RYGB. Three issues arising when considering bariatric surgery in paediatric patients are identified: informed consent; potential for interference with physical growth and/or sexual maturation; and compliance with postsurgical diets. Further higher quality studies, with improvements in long term data, are required.

Other Relevant Evidence


These guidelines are intended for service commissioners and providers in the U.K. NHS, as well as health professionals and interested members of the public. They provide recommendations on lifestyle weight management services for overweight and obese children and young people aged under 18. The recommendations cover planning services, commissioning programmes, core components of lifestyle weight management programmes, developing a tailored programme plan to meet individual needs, encouraging adherence, raising awareness of programmes, formal referrals to programmes, providing ongoing support, programme staff: training, knowledge and skills, training in how to make programme referrals, supporting programme staff and those making programme referrals, and monitoring and evaluating programmes. Further details on the evidence on which the recommendations are based can be found here: [http://publications.nice.org.uk/managing-overweight-and-obesity-among-children-and-young-people-lifestyle-weight-management-ph47/the-evidence-2](http://publications.nice.org.uk/managing-overweight-and-obesity-among-children-and-young-people-lifestyle-weight-management-ph47/the-evidence-2) and here: [http://guidance.nice.org.uk/PH47/SupportingEvidence](http://guidance.nice.org.uk/PH47/SupportingEvidence).


These Australian guidelines have been designed primarily for management of overweight and obesity at the individual level in primary health care. They are based on the 2010 evidence-based guideline from the Scottish Intercollegiate Guideline Network (SIGN): Management of obesity: a national clinical guideline as well as on systematic literature review (see below). They cover both adults and children and follow the 5As approach: Ask and Assess, Advise, Assist, Arrange. Recommendations are graded according to the SIGN grading system.


This systematic review was commissioned to inform the Australian guidelines above. It addresses two questions: What are the health outcomes associated with weight loss in individuals with overweight or obesity? and What are the impacts of weight reduction interventions on degree and duration of weight loss? The review covers literature published in English since 2007. It includes 70 systematic reviews/meta-analyses and reports of individual studies (RCTs) that had a follow up period of longer than 12 months.


This report identifies a broad range of population-level actions that governments can take to prevent childhood obesity. It suggests that a comprehensive childhood obesity prevention strategy should incorporate aspects of each of the key components. The first component is the structures within government, including leadership, “health-in-all” policies, dedicated funding for health promotion, non-communicable disease monitoring systems, workforce capacity, and networks and partnerships to support childhood obesity prevention policies and interventions. The second component is population-wide policies and initiatives that help to create environments that support healthy diets and physical activity, such as regulations, taxes and subsidies, and social marketing campaigns that affect the population as a whole (or large population groups). The third component is multi-component community-based interventions, typically applied across multiple settings (including early childcare settings, schools and other community settings), tailored to the local environment and implemented locally.
In 2010 the American Academy of Pediatrics joined the White House and federal departments to launch the Let's Move initiative to reduce rates of childhood obesity. This Task Force report identifies a comprehensive set of recommendations aimed at reducing childhood obesity, based on literature reviews, and consultation with experts and the broader public. Recommendations include: getting children a healthy start in life (including good prenatal care, support for breastfeeding, quality child care and limited “screen time”); empowering parents and caregivers (improved health messages and labelling, reduced marketing of unhealthy products, improved healthcare including BMI measurement for all children); providing healthy food in schools; improving access to healthy, affordable food; and getting children more physically active. The website [http://www.letsmove.gov/](http://www.letsmove.gov/) has a variety of resources for families and professionals.


This updated statement about screening for overweight in children and adolescents from the US Preventive Services Task Force includes an examination of the evidence for the effectiveness of interventions to address overweight and obesity in children and adolescents, and the magnitude of potential harms of treatment. The Task Force concludes that there is moderate certainty that the net benefit is moderate for screening for obesity in children aged six years and older and for offering or referring children to moderate- to high-intensity interventions (>25 hours of contact with the child and/or the family over a 6-month period) to improve weight status. There was not sufficient evidence to support screening children under the age of six and no evidence was found regarding appropriate intervals for screening. There is adequate evidence that the harms of behavioural interventions are no greater than small and harms of screening were judged to be minimal. The net benefit of screening was judged to be at least moderate.


These evidence-based guidelines include a systematic literature review examining the management of obesity in children (available at [http://guidance.nice.org.uk/CG43/Guidance/Section/5a/pdf/English](http://guidance.nice.org.uk/CG43/Guidance/Section/5a/pdf/English)). Multicomponent interventions that include behaviour change strategies to increase physical activity levels or decrease inactivity, improve eating behaviour and dietary intake, are identified as the treatment of choice in clinical care. Recommendations for children include: addressing lifestyle with the family; using BMI, adjusted for age and gender, as a practical estimate of overweight, but interpreted with caution as it is an indirect measure of adiposity; and consideration of referral to specialist service for children with significant comorbidity or complex needs.


This UK Government Foresight Programme report sought to examine how society might deliver a sustainable response to obesity in the UK over the next 40 years. The report includes an evidence-based review to identify the broad range of factors that influence obesity and identify effective interventions, an examination of the relationships between key factors influencing levels of obesity and their relative importance, and an analysis of future levels of obesity and the most effective future responses. Given that once gained, weight is difficult to lose, the report focuses on prevention. Behaviour change, changing the environment, changing biology (e.g., through promoting breastfeeding) and the role of technology are examined. The report concludes that evidence-based evidence supports the need for a substantial degree of intervention to affect an impact on the rising trend in obesity. It identifies a case for a national debate on the appropriate level for policy intervention and the apportioning of responsibility. Five core principles are identified: a system-wide approach, redefining the nation’s health as a societal and economic issue; higher priority for the prevention of health problems, with clearer leadership, accountability, strategy and management structures; engagement of stakeholders within and outside Government; long-term, sustained interventions; and ongoing evaluation and a focus on continuous improvement.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
THE CONSEQUENCES OF OBESITY

Introduction
Childhood obesity has both short and long term consequences. The short term consequences that affect children's quality of life are largely limited to severely obese children. They include sleep problems, asthma, type 2 diabetes, orthopaedic disorders (including slipped upper femoral epiphysis) and psychological and social distress [224]. In addition, a number of early markers for coronary artery disease are measureable in obese children including elevated blood pressure and cholesterol [225]. The long term consequences are mainly related to the fact that obese children have a high probability of becoming obese adults, with the consequent increased risks of metabolic syndrome, type 2 diabetes and cardiovascular disease [226].

There is no routinely collected information on the consequences of obesity in children and young people. However, a small number of children and young people do come into contact with the hospital system for obesity-related conditions including those hospitalised for type 2 diabetes, slipped upper femoral epiphysis and bariatric surgery. The following section reviews the data available for each of these conditions.

TYPE 2 DIABETES

Introduction
Type 2 diabetes is one of the most serious complications of childhood obesity [227]. Most overweight children have significant metabolic abnormalities due to insulin resistance, even if they have no evidence of type 2 diabetes [229]. In Auckland, the incidence of type 2 diabetes in children 0–14 years increased five-fold between 1995 and 2007, from 0.5 per 100,000 to 2.5 per 100,000 (representing about 10% of all new cases of diabetes among children and adolescents in the Auckland region) [230]. The average annual incidence over the period was 1.3 per 100,000, with rates being 0.1 per 100,000 for European children and 3.4 per 100,000 for Māori and Pacific children [230]. Unlike type 1diabetes, type 2 diabetes is often asymptomatic and therefore diagnosis requires laboratory testing.

The following section reviews hospital admissions in children and young people aged 0–24 years with type 2 diabetes mentioned in any of their first 15 diagnoses.

Data Source and Methods
Definition
1. Hospital admissions for children and young people aged 0–24 years with type 2 diabetes listed in any of their first 15 diagnoses
2. Mortality for children and young people aged 0–24 years with type 2 diabetes listed as the main underlying cause of death or as a contributory cause

Data Source
1. National Minimum Dataset
   Numerator: Hospital admissions for children and young people aged 0–24 years with type 2 diabetes (ICD-10-AM E11) listed in any of the first 15 diagnoses.
2. National Mortality Collection
   Numerator: Mortality in children and young people aged 0–24 years with type 2 diabetes (ICD-10-AM E11) listed as the main underlying cause of death, or as a contributory cause.

Notes on Interpretation
Note 1: This analysis focuses on hospital admissions and mortality for children and young people with type 2 diabetes listed in any of the first 15 diagnoses, or as the main underlying, or a contributory cause of death. The rationale for looking beyond the primary diagnosis was the need to highlight the spectrum of health issues experienced by those with type 2 diabetes, and their consequent requirement for health services.
New Zealand Distribution and Trends

Distribution by Age

In New Zealand during 2008–2012, hospitalisations for children and young people with type 2 diabetes were infrequent during childhood, but increased thereafter, with the highest rates being seen in those in their early twenties (Figure 65). During 2006–2010, four young people had type 2 diabetes listed as the main underlying cause of death, or as a contributory cause, with all deaths being in young people over 15 years of age.

Figure 65. Hospital Admissions for Children and Young People with Type 2 Diabetes by Age, New Zealand 2008–2012

Table 106. Hospital Admissions in Children and Young People Aged 0–24 Years with Type 2 Diabetes by Primary Diagnosis, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Type 2 Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 2 Diabetes</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Diagnoses other than Type 2 Diabetes*</td>
<td>616</td>
<td>123.2</td>
<td>8.07</td>
<td>78.8</td>
</tr>
<tr>
<td>Type 2 Diabetes without Complications</td>
<td>32</td>
<td>6.4</td>
<td>0.42</td>
<td>4.1</td>
</tr>
<tr>
<td>Type 2 Diabetes with Multiple Complications</td>
<td>29</td>
<td>5.8</td>
<td>0.38</td>
<td>3.7</td>
</tr>
<tr>
<td>Type 2 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma</td>
<td>16</td>
<td>3.2</td>
<td>0.21</td>
<td>2.0</td>
</tr>
<tr>
<td>Type 2 Diabetes with Renal Complications</td>
<td>6</td>
<td>1.2</td>
<td>0.08</td>
<td>0.8</td>
</tr>
<tr>
<td>Type 2 Diabetes with Ophthalmic Complications</td>
<td>6</td>
<td>1.2</td>
<td>0.08</td>
<td>0.8</td>
</tr>
<tr>
<td>Type 2 Diabetes with Unspecified Complications</td>
<td>3</td>
<td>0.6</td>
<td>0.04</td>
<td>0.4</td>
</tr>
<tr>
<td>Type 2 Diabetes with Other Specified Complications</td>
<td>74</td>
<td>14.8</td>
<td>0.97</td>
<td>9.5</td>
</tr>
<tr>
<td>Total Type 2 Diabetes Admissions</td>
<td>782</td>
<td>156.4</td>
<td>10.24</td>
<td>100.0</td>
</tr>
</tbody>
</table>

*Conditions Contributing to Diagnoses other than Type 2 Diabetes

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number of Admissions: Total 2008–2012</th>
<th>Number of Admissions: Annual Average</th>
<th>Rate per 100,000 Population</th>
<th>% of Admissions in those with Type 2 Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy Childbirth Post-Partum</td>
<td>134</td>
<td>26.8</td>
<td>1.75</td>
<td>17.1</td>
</tr>
<tr>
<td>Skin Infections</td>
<td>54</td>
<td>10.8</td>
<td>0.71</td>
<td>6.9</td>
</tr>
<tr>
<td>Diseases of the Respiratory System</td>
<td>50</td>
<td>10.0</td>
<td>0.65</td>
<td>6.4</td>
</tr>
<tr>
<td>Injury and Poisoning</td>
<td>36</td>
<td>7.2</td>
<td>0.47</td>
<td>4.6</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>23</td>
<td>4.6</td>
<td>0.30</td>
<td>2.9</td>
</tr>
<tr>
<td>Other Mental Health Issues</td>
<td>25</td>
<td>5.0</td>
<td>0.33</td>
<td>3.2</td>
</tr>
<tr>
<td>Cardiovascular Diseases</td>
<td>24</td>
<td>4.8</td>
<td>0.31</td>
<td>3.1</td>
</tr>
<tr>
<td>Abdominal and Pelvic Pain</td>
<td>21</td>
<td>4.2</td>
<td>0.28</td>
<td>2.7</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>20</td>
<td>4.0</td>
<td>0.26</td>
<td>2.6</td>
</tr>
<tr>
<td>Other Infectious Diseases</td>
<td>18</td>
<td>3.6</td>
<td>0.24</td>
<td>2.3</td>
</tr>
<tr>
<td>Complications Medical Surgical Care</td>
<td>17</td>
<td>3.4</td>
<td>0.22</td>
<td>2.2</td>
</tr>
<tr>
<td>All Other Diagnoses</td>
<td>194</td>
<td>38.8</td>
<td>2.54</td>
<td>24.8</td>
</tr>
<tr>
<td>Total Other Diagnoses</td>
<td>616</td>
<td>123.2</td>
<td>8.07</td>
<td>78.8</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with type 2 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Consequences of Obesity

Distribution by Primary Diagnosis

In New Zealand during 2008–2012, 21.2% of hospital admissions for children and young people with type 2 diabetes in any of their first 15 diagnoses had a diabetes-related code listed as their primary diagnosis. The remaining 78.8% of admissions had non-diabetes related primary diagnoses, with pregnancy and childbirth (17.1%), skin infections (6.9%) and diseases of the respiratory system (6.4%) being the leading non-diabetes related reasons for admission (Table 106).

Table 107. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 2 Diabetes by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian/Indian</td>
<td>4.16</td>
<td>1.07</td>
<td>0.75–1.53</td>
<td>Female</td>
<td>14.51</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>3.89</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>6.18</td>
<td>0.43</td>
<td>0.37–0.50</td>
</tr>
<tr>
<td>Māori</td>
<td>17.76</td>
<td>4.56</td>
<td>3.78–5.51</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>36.83</td>
<td>9.46</td>
<td>7.79–11.49</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 2 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population

Figure 66. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 2 Diabetes by Ethnicity, New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 2 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Note: See Note 2 in Methods box regarding cautions in interpreting time series information as a result of a change in the way diabetes codes were assigned in 2008.
**Distribution by Ethnicity and Gender**

In New Zealand during 2008–2012, hospital admissions for those with type 2 diabetes were *significantly* higher for females and for Pacific > Māori > Asian/Indian and European/Other children and young people (Table 107). Similar ethnic differences were seen during 2000–2012, with admission rates increasing for Pacific and Māori children and young people during this period (Figure 66).

**Northern DHBs Distribution and Trends**

**Northern DHBs Distribution**

In the Northern region during 2008–2012, 25 Northland, 43 Waitemata, 64 Auckland and 114 Counties Manukau children and young people were hospitalised with a diagnosis of type 2 diabetes. Admission rates per 100,000 in the Northland, Auckland and Counties Manukau DHBs were *significantly* higher than the New Zealand rate, while rates in the Waitemata DHB were not *significantly* different from the New Zealand rate (Table 108).

**Northern DHBs Trends**

Large year to year variations in rates made trends in type 2 diabetes admissions difficult to interpret during 2000–2012. However, rates in Northland and Counties Manukau remained higher than the New Zealand rate throughout this period, while rates in the Waitemata DHB remained similar to the New Zealand rate (Figure 67).

Table 108. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 2 Diabetes, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>A* 23</td>
<td>B* 25</td>
<td>0.45</td>
<td>20.72</td>
<td>2.02</td>
<td>1.54–2.65</td>
</tr>
<tr>
<td>Waitemata</td>
<td>39</td>
<td>43</td>
<td>0.41</td>
<td>9.43</td>
<td>0.92</td>
<td>0.74–1.15</td>
</tr>
<tr>
<td>Auckland</td>
<td>57</td>
<td>64</td>
<td>0.39</td>
<td>16.82</td>
<td>1.64</td>
<td>1.36–1.98</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>111</td>
<td>114</td>
<td>0.31</td>
<td>18.02</td>
<td>1.76</td>
<td>1.49–2.07</td>
</tr>
<tr>
<td>New Zealand</td>
<td>439</td>
<td>782</td>
<td>0.36</td>
<td>10.24</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 2 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (sum of DHB totals exceeds NZ total). Rate Ratios are compared to NZ rate and have not been adjusted for population demographics.
Figure 67. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 2 Diabetes, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with type 2 diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: See Note 2 in Methods box regarding cautions in interpreting time series information as a result of a change in the way diabetes codes were assigned in 2008.
SLIPPED UPPER FEMORAL EPIPHYSIS

Introduction

The long bones in children’s arms and legs grow from areas of cartilage near the ends of the bones, known as the growth plates or physis (singular physis). The upper femoral epiphysis is the rounded upper end of the thigh bone that forms part of the hip joint. There is a growth plate between the upper femoral epiphysis and the femoral shaft (the long part of the bone, known as the diaphysis). A slipped upper femoral epiphysis (SUFE), occurs when the femoral head is displaced from the shaft at the growth plate to a variable degree [255]. The term SUFE is a little misleading since the head of the femur remains in its normal position in the acetabulum (socket) of the pelvis while the rest of the femur has moved upwards, forwards and laterally from its normal position.

SUFE is one of the most common hip disorders in adolescents and it affects 10–60 per 100,000 children and adolescents per year [256,257]. The peak age of incidence is around 13 years for boys and 11 years for girls, and rates are higher in boys. It is more common in the left hip than the right, although in 20–40% of cases both hips are affected. A study of 211 children admitted to Starship Children’s Hospital with SUFEs between 1988 and 2000 estimated that, compared to European children, Māori children had admission rates 4.2 times higher and Pacific children 5.6 times higher [258].

The causes of SUFE are unclear but obesity is a significant risk factor, especially for bilateral SUFEs [259]. One study done in New York found that 81.1% of 106 children with a SUFE on x-ray had a BMI above the 95th percentile, compared to 41.1% of the 46 children who had a hip x-ray for hip pain but did not have a SUFE [260].

SUFE usually develops gradually with no apparent precipitating injury but it may follow a fall or sports injury or occur acutely with severe pain [256]. The signs and symptoms include pain in the hip, groin or knee, altered gait and limping. “Stable” SUFEs (>90% of all SUFEs) permit the patient to walk with or without crutches but a patient with an “unstable” SUFE cannot walk at all [261].

Treatment involves orthopaedic surgery to ensure stability across the growth plate by fixing the epiphysis in situ with pins or a screw [261]. The most severe complication of SUFE treatment, which is mostly associated with unstable SUFEs, is avascular necrosis of the femoral head (death of part of the bone as a result of interruption to its blood supply) which leads to early development of severe osteoarthritis of the hip, ultimately necessitating hip replacement. Treatment may also be associated with damage to the articular cartilage (chondrolysis) in the hip joint. The incidence of this complication has reportedly decreased with improvements in surgical techniques.

The following section reviews hospital admissions in children and young people aged 0–24 years with a slipped upper femoral epiphysis mentioned in any of their first 15 diagnoses.

Data Source and Methods

Definition

1. Hospital admissions for children and young people aged 0–24 years with a slipped upper femoral epiphysis (SUFE) listed in any of their first 15 diagnoses

Data Source

1. National Minimum Dataset

Numerator: Hospital admissions for children and young people aged 0–24 years with a slipped upper femoral epiphysis (non-traumatic) (ICD-10-AM M93.0) listed in any of the first 15 diagnoses.

New Zealand Distribution and Trends

Distribution by Primary Diagnosis and Procedure

In New Zealand during 2008–2012, 96.3% of hospitalisations for children and young people aged 0–24 years with a slipped upper femoral epiphysis listed in any of their first 15 diagnoses, had SUFE listed as the primary reason for admission.

Of the 748 hospitalisations during 2008–2012 where SUFE was listed in any of the first 15 diagnoses, 705 (94.3%) were acute (same day) or arranged (within seven days of referral) admissions, while 43 (5.7%) were drawn from the waiting list.

During 2008–2012, 95.9% of hospitalisations in children and young people with SUFE listed in any of the first 15 diagnoses, also had a primary procedure recorded, with closed reductions of a slipped capital femoral epiphyses (47.6%) and epiphysiodesis of the femur (21.5%) being the most frequently listed primary procedures (Table 109).

Distribution by Age

In New Zealand during 2008–2012, hospitalisations for children and young people with SUFE were infrequent during early childhood, but increased rapidly after eight years of age. Admissions reached a peak at 11 years in females and 12 years in males, before declining again during the early-mid teens. During this period, the rapid increases seen in late childhood, the peak in early adolescence, and the subsequent decline in the early-mid teens, occurred on average, one year earlier in females than in males (Figure 68).

Figure 68. Hospital Admissions for Children and Young People with a Slipped Upper Femoral Epiphysis by Age and Gender, New Zealand 2008–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with SUFE listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
Table 109. Hospital Admissions in Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis by Primary Procedure, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Procedure</th>
<th>Number: Total 2008–2012</th>
<th>Number: Annual Average</th>
<th>Admission Rate per 100,000 Population</th>
<th>% of Admissions in those with SUFE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Closed Reduction of Slipped Capital Femoral Epiphysis</td>
<td>356</td>
<td>71.2</td>
<td>4.66</td>
<td>47.6</td>
</tr>
<tr>
<td>Epiphysiodesis of Femur</td>
<td>161</td>
<td>32.2</td>
<td>2.11</td>
<td>21.5</td>
</tr>
<tr>
<td>Open Reduction of Slipped Capital Femoral Epiphysis</td>
<td>108</td>
<td>21.6</td>
<td>1.41</td>
<td>14.4</td>
</tr>
<tr>
<td>Insertion of Internal Fixation Device, Not Elsewhere Classified</td>
<td>31</td>
<td>6.2</td>
<td>0.41</td>
<td>4.1</td>
</tr>
<tr>
<td>Osteotomy of Proximal Femur with Internal Fixation</td>
<td>15</td>
<td>3.0</td>
<td>0.20</td>
<td>2.0</td>
</tr>
<tr>
<td>CT and MRI Scans</td>
<td>7</td>
<td>1.4</td>
<td>0.09</td>
<td>0.9</td>
</tr>
<tr>
<td>Open Reduction of Fracture of Femur with Internal Fixation</td>
<td>6</td>
<td>1.2</td>
<td>0.08</td>
<td>0.8</td>
</tr>
<tr>
<td>Arthrodesis of Hip</td>
<td>5</td>
<td>1.0</td>
<td>0.07</td>
<td>0.7</td>
</tr>
<tr>
<td>Ostectomy of Proximal Femur with Internal Fixation</td>
<td>4</td>
<td>0.8</td>
<td>0.05</td>
<td>0.5</td>
</tr>
<tr>
<td>Forage of Neck and/or Head of Femur</td>
<td>3</td>
<td>0.6</td>
<td>0.04</td>
<td>0.4</td>
</tr>
<tr>
<td>Other Procedures</td>
<td>21</td>
<td>4.2</td>
<td>0.28</td>
<td>2.8</td>
</tr>
<tr>
<td>No Listed Procedure</td>
<td>31</td>
<td>6.2</td>
<td>0.41</td>
<td>4.1</td>
</tr>
<tr>
<td><strong>Total Admissions</strong></td>
<td><strong>748</strong></td>
<td><strong>149.6</strong></td>
<td><strong>9.80</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions by primary procedure for children and young people with SUFE listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
**Distribution by Ethnicity and Gender**

In New Zealand during 2008–2012, there were no significant gender differences in hospital admissions for SUFE, although rates were significantly higher for Pacific and Māori > European/Other > Asian/Indian children and young people (Table 110). Similar ethnic differences were seen during 2000–2012, with SUFE rates for Pacific children and young people also being higher than for Māori children and young people for the majority of this period (Figure 69).

Table 110. Hospital Admissions for Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
<th>Variable</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slipped Upper Femoral Epiphysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>1.57</td>
<td>0.36</td>
<td>0.21–0.61</td>
<td>Female</td>
<td>9.43</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>European/Other</td>
<td>4.43</td>
<td>1.00</td>
<td></td>
<td>Male</td>
<td>10.14</td>
<td>1.08</td>
<td>0.93–1.24</td>
</tr>
<tr>
<td>Māori</td>
<td>20.44</td>
<td>4.62</td>
<td>3.87–5.50</td>
<td>Pacific</td>
<td>25.78</td>
<td>5.82</td>
<td>4.75–7.13</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with SUFE listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: Ethnicity is Level 1 Prioritised; Rate is per 100,000 population

Figure 69. Hospital Admissions for Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis by Ethnicity, New Zealand 2000–2012
Northern DHBs Distribution and Trends

Northern DHBs Distribution

In the Northern DHBs during 2008–2012, 40 Northland, 39 Waitemata, 47 Auckland and 118 Counties Manukau children and young people were admitted with a slipped upper femoral epiphysis. Hospital admission rates per 100,000 in Northland and Counties Manukau were significantly higher than the New Zealand rate, while rates in the Auckland and Waitemata DHBs were significantly lower (Table 111).

Northern DHBs Trends

While there was considerable year to year variability in rates, SUFE admissions in Northland and Counties Manukau remained higher than the New Zealand rate throughout 2000–2012, while rates in the Waitemata DHB remained lower. Rates in the Auckland DHB were lower than the New Zealand rate for the majority of this period (Figure 70).

Table 111. Hospital Admissions for Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis, Northern DHBs vs. New Zealand 2008–2012

<table>
<thead>
<tr>
<th>DHB</th>
<th>Total Number of Individuals 2008–2012</th>
<th>Total Number of Admissions 2008–2012</th>
<th>Average Number of Admissions per Individual per Year</th>
<th>Admission Rate per 100,000 Population</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>39</td>
<td>40</td>
<td>56</td>
<td>0.28</td>
<td>20.72</td>
<td>2.12</td>
</tr>
<tr>
<td>Waitemata</td>
<td>38</td>
<td>39</td>
<td>47</td>
<td>0.24</td>
<td>4.98</td>
<td>0.51</td>
</tr>
<tr>
<td>Auckland</td>
<td>47</td>
<td>47</td>
<td>53</td>
<td>0.23</td>
<td>7.08</td>
<td>0.72</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>118</td>
<td>118</td>
<td>135</td>
<td>0.23</td>
<td>13.66</td>
<td>1.39</td>
</tr>
<tr>
<td>New Zealand</td>
<td>617</td>
<td>748</td>
<td>0.24</td>
<td>9.80</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with SUFE listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: A*: Each individual only counted once in DHB in which they first reside (i.e. DHB total = NZ total); B*: Each individual counted once in each DHB in which they reside (i.e. the sum of DHB totals exceeds New Zealand total); Rate Ratios are compared to New Zealand rates and have not been adjusted for population demographics.
Figure 70. Hospital Admissions for Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis, Northern DHBs vs. New Zealand 2000–2012

Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with SUFE listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)
**Bariatric Surgery**

**Introduction**

While surgery for obesity is not generally recommended for obese children and young people, it has increasingly been used for the treatment of those with extreme obesity and obesity-related comorbidities, when more conservative treatment methods have failed [213]. In this context, guidelines from Australia’s NHMRC [112] suggest that a post-pubertal adolescent with a BMI of >40 kg/m², or >35 kg/m² plus significant severe comorbidities such as type 2 diabetes or obstructive sleep apnoea, may be considered for bariatric surgery, if other interventions have been unsuccessful.

There are a number of different surgical procedures used, all of which are usually done laparoscopically. They include the Roux-en-Y gastric bypass, the adjustable gastric band, biliopancreatic diversion and the sleeve gastrectomy [262]. The best-studied procedure in adolescents is the Roux-en-Y gastric bypass. In this procedure, the stomach is stapled to exclude almost all of the stomach volume and create a small pouch at the top of the stomach. This is separated from the main body of the stomach and attached to the small intestine. Weight loss ensues from restriction of food intake and malabsorption [263]. Adverse effects that may follow the procedure include anastomotic leak, small bowel obstruction, dumping syndrome (symptoms that may include nausea, vomiting, bloating, cramps, diarrhoea and/or other symptoms), protein-calorie malnutrition, and micronutrient deficiency related to malabsorption [264].

The following section reviews hospital admission for bariatric surgery in children and young people aged 0–24 years using data from the National Minimum Dataset.

<table>
<thead>
<tr>
<th>Data Source and Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
</tr>
<tr>
<td>1. Hospital admissions for young people aged 15–24 years with bariatric surgery listed in any of their first 15 procedures</td>
</tr>
<tr>
<td><strong>Data Source</strong></td>
</tr>
<tr>
<td>1. National Minimum Dataset</td>
</tr>
<tr>
<td><strong>Numerator</strong></td>
</tr>
<tr>
<td>Hospital admissions for young people aged 15–24 years with bariatric surgery listed in any of their first 15 procedures</td>
</tr>
<tr>
<td><strong>Specific procedures (ACHI codes) included:</strong></td>
</tr>
<tr>
<td><strong>Denominator</strong></td>
</tr>
<tr>
<td>Statistics New Zealand Estimated Resident Population (projected from 2007)</td>
</tr>
<tr>
<td><strong>Notes on Interpretation</strong></td>
</tr>
<tr>
<td>As only one procedure occurred in a young person less than 15 years of age, the analysis in this section has been restricted to young people aged 15–24 years.</td>
</tr>
</tbody>
</table>

**New Zealand Distribution and Trends**

**Distribution by Primary Diagnosis and Procedure**

*Primary Diagnosis:* In New Zealand during 2008–2012, obesity was the most frequent primary diagnosis in young people aged 15–24 years admitted for bariatric surgery, accounting for 65.9% of admissions. Type 2 diabetes and mechanical complications of gastrointestinal prosthetic devices made a smaller contribution (Table 112).

*Primary Procedure:* During the same period, laparoscopic gastric reductions (41.5%) were the most frequent primary procedure listed in young people admitted for bariatric surgery, followed by gastric bypasses for morbid obesity (29.3%) (Table 113).
### Table 112. Hospital Admissions for Bariatric Surgery by Primary Diagnosis in Young People Aged 15–24 Years, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Diagnosis</th>
<th>Number: 2008–2012</th>
<th>Percent of Admissions (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bariatric Surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>27</td>
<td>65.9</td>
</tr>
<tr>
<td>Type 2 Diabetes</td>
<td>4</td>
<td>9.8</td>
</tr>
<tr>
<td>Mechanical Complications Gastrointestinal Prosthetic Devices</td>
<td>4</td>
<td>9.8</td>
</tr>
<tr>
<td>Other Diagnoses</td>
<td>6</td>
<td>14.6</td>
</tr>
<tr>
<td>Total</td>
<td>41</td>
<td>100.0</td>
</tr>
</tbody>
</table>


### Table 113. Hospital Admissions for Bariatric Surgery by Primary Procedure in Young People Aged 15–24 Years, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Primary Procedure</th>
<th>Number: 2008–2012</th>
<th>Percent of Admissions (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bariatric Surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laparoscopic Gastric Reduction</td>
<td>17</td>
<td>41.5</td>
</tr>
<tr>
<td>Gastric Bypass for Morbid Obesity</td>
<td>12</td>
<td>29.3</td>
</tr>
<tr>
<td>Revision of Gastric Band</td>
<td>4</td>
<td>9.8</td>
</tr>
<tr>
<td>Biliopancreatic Diversion</td>
<td>3</td>
<td>7.3</td>
</tr>
<tr>
<td>Other Procedures</td>
<td>5</td>
<td>12.2</td>
</tr>
<tr>
<td>Total</td>
<td>41</td>
<td>100.0</td>
</tr>
</tbody>
</table>


### Figure 71. Hospital Admissions for Bariatric Surgery in Young People Aged 15–24 Years, New Zealand 2000–2012

- **Bariatric Surgery Number (Annual Average)**
- **Bariatric Surgery Rate**

New Zealand Trends
In New Zealand, bariatric surgery admissions in young people aged 15–24 years increased from on average 0.5 admissions per year in 2000–01, to 10 per year during 2010–2012 (Figure 71).

Distribution by Age
In New Zealand during 2008–2012, bariatric surgery admissions were infrequent during the early teens, but increased thereafter, with the highest rates being seen amongst those in their early twenties (Figure 72).

Figure 72. Hospital Admissions for Bariatric Surgery in Young People Aged 15–24 Years by Age, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Number of Admissions per 100,000</th>
<th>Bariatric Surgery Number (Annual Average)</th>
<th>Bariatric Surgery Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>16</td>
<td>0.2</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>17</td>
<td>0.4</td>
<td>0.4</td>
<td>0.2</td>
</tr>
<tr>
<td>18</td>
<td>0.6</td>
<td>0.6</td>
<td>0.3</td>
</tr>
<tr>
<td>19</td>
<td>0.8</td>
<td>0.8</td>
<td>0.4</td>
</tr>
<tr>
<td>20</td>
<td>1.0</td>
<td>1.0</td>
<td>0.5</td>
</tr>
<tr>
<td>21</td>
<td>1.2</td>
<td>1.2</td>
<td>0.6</td>
</tr>
<tr>
<td>22</td>
<td>1.4</td>
<td>1.4</td>
<td>0.7</td>
</tr>
<tr>
<td>23</td>
<td>1.6</td>
<td>1.6</td>
<td>0.8</td>
</tr>
<tr>
<td>24</td>
<td>1.8</td>
<td>1.8</td>
<td>0.9</td>
</tr>
</tbody>
</table>


Table 114. Hospital Admissions for Young People Aged 15–24 Years for Bariatric Surgery by Ethnicity and Gender, New Zealand 2008–2012

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number: Total 2008–2012</th>
<th>Rate</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bariatric Surgery</td>
<td>Prioritised Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>&lt;3</td>
<td>s</td>
<td>s</td>
<td>s</td>
</tr>
<tr>
<td>European/Other</td>
<td>28</td>
<td>1.52</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>4</td>
<td>0.64</td>
<td>0.42</td>
<td>0.15–1.20</td>
</tr>
<tr>
<td>Pacific</td>
<td>6</td>
<td>2.29</td>
<td>1.51</td>
<td>0.62–3.64</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>34</td>
<td>2.20</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>7</td>
<td>0.43</td>
<td>0.20</td>
<td>0.09–0.44</td>
</tr>
</tbody>
</table>

Source: Numerator: National Minimum Dataset; Denominator: Statistics New Zealand Estimated Resident Population (projected from 2007); Note: Rate is per 100,000 15–24 years; Ethnicity is Level 1 Prioritised; s: suppressed due to small numbers
Distribution by Ethnicity and Gender
In New Zealand during 2008–2012, while bariatric surgery admissions were higher for Pacific > European/Other > Māori young people, these differences did not reach statistical significance. Admission rates however, were significantly higher for females than for males (Table 114).

Northern DHBs Distribution
In the Northern DHBs during 2008–2012, there were 23 admissions for bariatric surgery in young people aged 15–24 years (<3 in Northland, 11 in Waitemata, <3 in Auckland DHB and 8 in Counties Manukau).

Local Policy Documents and Evidence-Based Reviews Relevant to the Consequences of Obesity in Children and Young People
Obesity significantly increases the risk of developing type 2 diabetes, or a slipped upper femoral epiphysis. Interventions aimed at obesity prevention and management may therefore reduce the risk of these conditions and the need for bariatric surgery.

In New Zealand, a number of policy documents and reviews consider the prevention and management of obesity. These are briefly summarised in Table 105 on Page 310, along with a range of guidelines and reviews which consider these issues in the overseas context. In addition an in-depth topic commencing on Page 287 reviews The Determinants and Consequences of Overweight and Obesity, while an in-depth topic commencing on Page 332 reviews The Treatment of Obesity in Children and Adolescents.
IN DEPTH TOPIC: THE TREATMENT OF OBESITY IN CHILDREN AND ADOLESCENTS

Introduction

The main reason why the rising prevalence of childhood obesity is an important public health issue is that obese children are likely to become obese adults at high risk of developing diabetes and cardiovascular disease and it is feared that the future cost of healthcare for obesity-related illnesses will be beyond the nation’s resources [158,265].

From the perspective of the individual obese child and his or her family, however, more immediate consequences of obesity, such as having low self-esteem, being bullied, teased or socially marginalised, being unable to participate in physical activities and sport or to wear fashionable clothes, tend to be of greater importance. There is evidence that many parents of overweight or obese children are unaware of their child’s weight status although the reasons for this have not been thoroughly explored [266]. Raising awareness of the significance of childhood obesity, as the Lets Move! campaign started by Michelle Obama has done in the U.S. [267,268], is important as unless parents are motivated to change their families’ habits to improve their children’s weight there is little point in offering intervention.

There is a general consensus among obesity experts that tackling the obesity problem requires a whole of society approach to prevention, and that this involves tackling complex social and economic issues and changing public policy in many areas including food production, manufacturing and retailing, trade, urban planning, transport, healthcare, education and culture [109].

Nevertheless, those who work in healthcare want to be able to help individual obese children and their families in the here and now. This in-depth topic aims to provide information on evidence-based interventions for the treatment of established overweight and obesity in children and adolescents. It is organised into five sections as follows:

- Identifying and engaging children (and their parents) who are candidates for weight management interventions
- Insights from a 2009 Cochrane review of obesity interventions in children and adolescents
- Insights from other reviews of obesity interventions in children and adolescents
- New Zealand interventions
- Primary care interventions, including recent RCTs addressing obesity in primary care

In addition, there are a number of evidence-based guidelines for the management of overweight and obesity in children and young people, including those published by the N.Z. Ministry of Health (2009) [269], the U.K. National Institute for Health and Care Excellence (2013) [270], the Scottish Intercollegiate Guidelines Network (2010) [271], and the Australian National Health and Medical Research Council (2013) [112]. Readers wanting more detailed information than is provided here might like to refer to these guidelines.

Identifying and engaging children (and their parents) who are candidates for weight management interventions

It cannot be assumed that the parents of overweight and obese children are greatly concerned about their child’s weight status and its implications for future health and will therefore seek assistance from health professionals. If interventions are to reach the children and young people most in need then those working in the health system may need to actively seek out and attempt to engage families of overweight and obese children.
[2], while being mindful that dealing with a child’s overweight may not be a high priority in families who are struggling with more urgent problems.

**Identifying children who are candidates for weight management interventions**

Body mass index (BMI) is defined as weight/height² with weight measured in kg and height in metres. Plotting a child’s BMI on a BMI-for-age reference chart allows easy assessment of a child’s weight status in relation to that of other children of the same age and sex. Commonly used BMI charts for children are those of the World Health Organization [199] and the U.S. Centers for Disease Control and Prevention [201]. Children who have a BMI greater than or equal to the 95th percentile for their age are commonly classified as being obese, and those whose BMI is at or above the 85th percentile but less than the 95th percentile as being overweight [190]. A particular advantage of using BMI-for-age charts is that they allow monitoring of a child’s BMI over time and allow the identification of children at risk of obesity because their BMI is increasing more rapidly than would be expected for their age (sometimes referred to as “upward percentile crossing”).

All of the guidelines mentioned above agree that BMI percentile is the best indicator for identifying overweight and obesity in children over the age of two years (although they do not agree precisely on the BMI percentile values that indicate a need for intervention). This consensus is supported by a 2010 systematic review [272], (which recommended the use of national BMI-for-age reference data if this was available).

Severely obese children can have BMI values that are off the chart so the standard charts produced by the CDC or the WHO cannot be used to characterise or monitor the weight status of these children. Since severely obese children are at the greatest risk of obesity related health problems it is important to be able to identify the most severely obese children so that they can be given priority for intervention efforts and also to be able to monitor the effectiveness of any interventions attempted with these children [273]. To address the limitations of the standard BMI charts for these purposes Flegal et al. proposed that the BMI of a very obese child be described as “the percentage of the 95th percentile” [274]. Gulati et al. have used this idea to develop growth charts that have additional lines above the 95th percentile line corresponding to 110%, 120%, 130% and so on, of the 95th percentile BMI values [275]. They have used these charts to track the weight status of their severely obese child patients over time and have suggested that their charts may also be useful in research.

**Screening for overweight and obesity in children**

It is recommended that both routine and health concern related child health provider contacts should include measurement of BMI-for-age-and-sex, provided the child’s parent or carer agrees [112,269,276]. This could be regarded as “opportunistic screening”. There is no New Zealand data on the degree to which this is happening in primary care other than as part of Well Child/Tamariki Ora services. A recent study from the U.S. used data from the National Health and Nutrition Examination Survey for the years 1999 through 2008 to examine trends in parental report of health professional notification of childhood overweight [277]. Parents were asked: “Has a doctor or health professional ever told you that your child is overweight?” The percentage of parents of children with BMIs ≥ the 85th percentile who recalled ever having been told that their child was overweight increased only slightly (from 19.4% to 23.2%) over the 1999–2006 period but increased to 29.1% in the 2007–2008 period. Even among the parents of very obese children (≥ the 99th percentile), on average only 58% recalled ever having been told that their child was overweight. The authors stated that “further research is necessary to determine where and why communication of weight status breaks down and how effective appropriate communication of weight status is in motivating families toward healthier living”.

Whether more systematic screening is desirable is doubtful. Westwood et al. were commissioned by the U.K. Health Technology Assessment Programme to conduct a systematic review on whether or not primary school children should be routinely screened for obesity [278]. They found that there had been (up till July 2005) no trials assessing the effectiveness of monitoring or screening for identifying obesity in children and there was
“extremely sparse” information on the attitudes of children, parents and health professionals to monitoring. The authors stated that “there is currently little evidence that weight reduction interventions are effective and without this evidence any move towards identifying individual children appears difficult to justify”. Another systematic review on this topic for the U.S. Preventive Services Task Force (2005) reached similar conclusions [279].

In the U.K. the National Child Measurement Programme (NCMP) measures the height and weight of all children in reception (ages four and five) and year six (ages 10 and 11) classes, except those of parents who have chosen to opt their children out [280,281]. The NMCP is not a screening programme in the accepted sense of the term (since its primary aims are not to identify individual children at risk of obesity early so that they can be treated more effectively than would be possible if they were identified later on). The aims of the NMCP are: to inform local planning and delivery of services, to gather population-level data for monitoring trends in growth patterns and obesity, and to increase public and professional understanding of weight issues in children and be a vehicle for engagement with children and families regarding weight issues and healthy lifestyles [280]. Some, but not all, Local Authorities inform parents of their child’s results by letter [281].

**Parental perceptions of children’s weight status**

Since screening cannot yet be recommended as a method of identifying obese children, it is worth considering how good parents are at recognising that their child is overweight or obese, how likely they are to seek help if they recognise that their child has a weight problem, and how they perceive health professionals’ attitudes to them and their children.

Parry et.al undertook a systematic review of 23 studies (3864 children aged 2–12 years) which had assessed parental perceptions of their child’s weight status and compared these to their child’s actual weight status according to a recognised standard for defining overweight such as BMI centiles or International Obesity Taskforce cut-offs [282]. The percentage of parents who recognised their child’s overweight status ranged from 6.2% to 73%, but in 19 of the 23 studies it was less than 50%. A more recent systematic review, by Rietmeijer-Mentink et.al., included 51 publications (35,103 children) which were of variable methodological quality [283]. The pooled results from these studies indicated that, according to objective criteria, 11,530 children were overweight and, of these, 62.4% (7191) were incorrectly perceived by their parents as being of normal weight.

As part of the Pacific Island Families Study, when their children were four and six years old, 569 parents were asked, “How concerned are you about your child becoming overweight?” and their responses were compared with their child’s BMI [284]. At four and six years the majority of parents were not concerned about their child’s weight (62% and 69.1%) yet at four years only 40.1% of children were considered to be of normal weight but 34.1% were overweight and 25.8% obese. At six years the proportions were: 41.3% normal weight, 31.1% overweight and 27.6% obese. Parents were more likely to be concerned about their child’s future weight status if their child was overweight or obese. At six years the percentage of parents who were concerned was 20% for normal weight children, 28% for overweight children and 51% for obese children. The study authors stated that their findings raised the concern that there is normalisation of overweight and obesity among Pacific parents and/or their children. They suggested that attention be paid to addressing the socio-economic environment of Pacific families and raising parents’ awareness of the links between obesity and eating and activity patterns.

**Parents’ perceptions of health professionals’ attitudes**

Even if parents do recognise that their child has a weight problem they may be reluctant to seek help because of fear they will be “blamed and shamed” and they fear adverse effects on their child’s mental well-being [285]. A number of studies have explored parent’s views and experiences of their children’s obesity-related encounters with the health system, either in primary care [285,286,287] or in specialist clinics [288,289,290,291,292]. Parents have often attempted their own dietary and physical activity strategies before seeking help so they are unlikely to be satisfied with general advice about eating less and exercising more [287]. If they have a weight problem of their own and they believe their GP has not
helped them with it then they may think he or she will not be able to help their child either [285]. One researcher who interviewed a self-selected group of parents who had concerns about their child’s weight (parents of 40 children in south-west England), found that professional responses to parental help seeking had ranged from positive, but not very helpful, to negative and dismissive [287]. According to the parents interviewed in a later English study, it is important for practitioners to be non-judgmental and empathetic, to have sufficient knowledge and skills to treat childhood obesity, and to pay attention to broader issues such as low self-esteem and behavioural problems [285].

The long term health consequences of obesity may not be a major concern for many parents. They may be more concerned about their child being teased or bullied, or being unable to participate in physical activities and sport or to buy clothes that fit [285,289,293,294]. Health professionals need to be aware of this so they can focus on weight loss goals that have meaning for parents and children.

Engaging the families of obese children
It can be difficult to engage families of obese children with services that facilitate long term weight management, even if they are free as they are in the U.K. Banks et al. reported on a project which aimed to identify obese children (BMI ≥ 98th percentile) from the databases of 12 general practices in Bristol, U.K. and invite them for a primary care consultation and possible referral to a specialist secondary care clinic [295]. Invitation letters were sent to 285 families, 134 patients consulted their GP within the follow up period (minimum 3 months) and the child’s weight was discussed at 42 of these consultations. Nineteen patients received a secondary care referral and six received an alternative weight management referral. The authors noted that children's weight is a sensitive issue, about which parents may feel guilt and shame, and it is therefore a difficult area for parents and health professionals to discuss. They also cite research which has found that many parents do not recognise their child’s overweight or obesity as a health problem that needs attention.

A recently-published New Zealand study investigated what factors influence participation in a family-based weight management programme for overweight and obese four to eight year-old children identified through participation in by-invitation screening [296]. A key aim of this study was to determine whether motivational interviewing for feedback was an appropriate way to inform parents that their young child was overweight. All parents in the study received feedback consisting of a neutral presentation of their child’s weight status. Parents of overweight (BMI 85th to <95th percentile) or obese children (BMI ≥95th percentile) were randomised to receive or not receive motivational interviewing before their child’s weight status information was presented. Out of the 1093 children screened 24.8% were overweight or obese. Of these, 72.7% agreed to participation in the intervention. Overall there were few differences between participating and non-participating parents but non-participating parents more often came from homes in more deprived areas (p=0.039); participating mothers tended to be more highly educated (p=0.051); and fewer non-participating parents believed their child to be overweight (23% vs. 49%, p <0.001) or were concerned about it (16% vs. 43%, p <0.001), despite their children having an average body mass index close to the 95th percentile. The type of feedback received did not appear to influence participation rates (p=0.221). The authors of this study speculated that the reason why they achieved much higher uptake rates than the Bristol study could be because the parents in their study received face-to-face feedback about their child’s weight status rather than being informed by letter.

Interventions for treating obesity in children and adolescents
Interventions for treating obesity in individual children and adolescents fall into three broad categories: lifestyle (diet, physical activity and behavioural therapy, often in combination), drug treatment and surgery. There has been a considerable amount of research into various lifestyle interventions and a number of systematic reviews of lifestyle interventions have been published [254,297,298,299] but there is relatively little research on drug
interventions or bariatric surgery both of which are considered appropriate only for obese adolescents.

**Insights from a 2009 Cochrane review of obesity interventions in children and adolescents**

A 2009 Cochrane review aimed to determine the efficacy of lifestyle, drug and surgical interventions for the treatment of obesity in children from a review of all relevant randomised controlled trials which had a follow up duration of at least six months [299]. Lifestyle interventions were divided into three types, dietary, physical activity and behavioural, and discussed in two age categories, those for children under 12 years old and those for children 12 years old and older. The section which follows discusses some of the insights gained from this 2009 review.

**Dietary interventions in children under 12 years old**

There were four studies of dietary interventions in children under 12 years old. Two fulfilled all the criteria for meta-analysis but they made different comparisons so meta-analysis was not possible. One of these studies, involving children aged from three to 12 years, found a beneficial effect of a dietary intervention comprising a specific calorie-controlled diet, detailed guidelines regarding physical activity and parental commitment and the provision of a food diary with instructions for its use compared to provision of general obesity information leaflets on obesity risks, healthy eating and physical activity, both at six and 12 months follow up [300]. The intervention group (72 children, mean age 8.6 ± 1.9 years) had a significantly higher reduction in percentage overweight than the control group (n=114, mean age 8.6 ± 2.1 years): −8.8% at six months and −8.5% at 12 months vs. −2.9% at six months and −2.9% at 12 months. (Percentage overweight was defined in this study as ((actual weight – ideal weight) / ideal weight ) *100, where ideal weight was the 50th percentile weight for the age at which the child’s height was at the 50th percentile.) BMI values at baseline, six and 12 months were 23.8 ± 2.7, 22.5 ± 2.5, 23.0 ± 2.4 in the intervention group and 22.4 ± 1.9, 22.2 ± 1.9, 22.7 ± 2.1 in the control group.

The other study compared a “making healthy food choices” intervention (n=21) to a “decrease high energy foods” intervention (n=20) [301]. The interventions lasted for five months and results were assessed at six, 12 and 24 months. Using a mixed effects regression model the study authors found that the children in the increase healthy foods group had greater reductions in BMI z scores than those in the reduce high energy foods group, both at 12 months (−0.30 vs. −0.15, p=0.01) and at 24 months (−0.36 vs. −0.13, p=0.04).

**Physical activity interventions in children under 12 years old**

Nine studies in children under 12 years old focussed mainly on the physical activity component of the intervention. Four studies fulfilled the reviewers’ quality criteria but had incomparable study designs and interventions and so were unsuitable for meta-analysis. One study randomised 90 families with obese 8–12 year old children to receive either emphasis on discouraging sedentary behaviours or on encouraging physically active behaviours as part of a comprehensive family-based behavioural weight control programme that included dietary and behaviour change information [302]. The study results indicated that targeting physical activity or sedentary behaviours was associated with similar decreases in per cent overweight and increases in physical fitness during the two-year observation period. Across all four groups in this study (high and low dose increasing physical activity and decreasing sedentary behaviour interventions) the change in per cent overweight was −25.5% ± 10.6% at six months and −12.9% ± 17.0% at 24 months. Changes in BMI z scores were not reported.

Another study randomised 192 families with at least one 7–14 year old child who was overweight or at risk of overweight to either an “America on the move” group or a self-monitoring only group [303]. Both groups were asked to use pedometers to record daily physical activity and, in addition, the “America on the move” group were asked to walk an extra 2000 steps per day above baseline and to eliminate 420 kJ per day from their diet by
replacing dietary sugar with a non-caloric sweetener. At six months, the “America on the move” group had a significantly higher percentage of children who maintained or reduced their BMI-for-age (67% vs. 53%, p < 0.05) and a significantly lower percentage of children who increased their BMI for age (33% vs. 47%, p< 0.05). Children in both groups had groups had small decreases in BMI-for-age z-scores which the study authors (rather optimistically) stated were “clinically meaningful and statistically significant”. The “America on the move” group had a greater decrease in BMI-for age z score (−0.066 ± 0.166) than the self-monitoring group (−0.039 ± 0.169) but the difference between the two groups was not statistically significant (−0.027, 95% confidence interval −0.075 to 0.022, p= 0.282 ). There was no change in parent BMI in either group. The authors stated that their “small changes” approach could be useful for addressing childhood obesity by preventing excess weight gain in families.

**Behavioural interventions in children under 12 years**

There were 24 studies of behavioural interventions in children under 12 years. Behavioural interventions included family therapy, problem-solving approaches, cognitive-behavioural treatment and multi-component behavioural programmes incorporating a variety of behavioural techniques. Meta-analysis of the results from four studies (301 participants) showed a small positive effect for parent-focused behavioural group intervention compared to standard care at six months: the BMI-SDS (z-score) difference was −0.06 (95% CI −0.12 to −0.01), indicating that the average BMI in the intervention group was 0.06 of a standard deviation (based on BMI-for-age-and-sex reference values) below that of the control group. A second meta-analysis pooled the results of the three of these four studies that had also reported on 12 month follow up (264 participants) and found that at 12 months there was no benefit from the parent-focused behavioural group intervention compared to standard care: the change in BMI-SDS was −0.04 (95% CI −0.12 to 0.04).

**Dietary interventions in children 12 years and older**

Two studies explored dietary interventions in children 12 years and older, but only one of them reported an intention-to-treat analysis. This small RCT (16 participants, 14 of whom completed) compared two dietary interventions, one with a reduced glycaemic index and one which was a standard dietary intervention with reduced fat load, both in combination with behavioural therapy [304]. At 12 months follow there was a significantly greater decrease in absolute BMI (mean ± std. error: −1.3 ± 0.7 vs. 0.7± 0.5, p 0.02) and fat mass (−3.0 ± 1.6 vs. 1.8 ±1.0 kg, p=0.01) in the reduced glycaemic index group compared to the reduced fat load group. Compared to baseline values, there was a significant favourable effect on absolute BMI and fat mass at 12 months for the reduced glycaemic index group but not for the reduced fat load group.

**Physical activity-based interventions for children 12 years and older**

Three studies compared an experimental activity programme to an “active placebo” or control intervention. Only one of these three fulfilled the review’s quality criteria. This study compared an after school activity programme to an exercise placebo (light body conditioning/stretching exercises) or usual care. It found that, at six months follow up, there were no significant changes in BMI-SDS from baseline or between any of the groups, but there were significant changes (favouring the exercise group) in physical self-worth, associated measures of self-esteem and physical activity [305].

**Behavioural interventions in children 12 years and older**

There were 12 lifestyle interventions in adolescents with a behavioural component as the main focus of the intervention. Seven of these studies were of sufficient quality for their results to be pooled in a meta-analysis but only four reported similar outcomes at six months. A meta-analysis of pooled data from three studies at six months follow up indicated an overall effect of a behavioural intervention on BMI-SDS (291 participants’ data) of −0.14 (95% CI −0.17 to −0.12) and an overall effect on absolute BMI (362 participants’ data) of−3.04 (95% CI −3.14 to −2.94) kg/m², in comparison to standard care or control condition.

One study found a non-significant decrease in BMI-SDS in adolescents who participated in a four month behavioural intervention initiated in primary care (phone and email contact),

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compared to a non-significant increase in BMI-SDS for adolescents receiving standard single physician care. This meant that at the end of the intervention (four months) there was a significant difference in change from baseline between the groups. At seven months, however, there were no longer any differences between groups. The 20 intervention subjects’ mean BMI (SD) values were 31.0 (3.5) at baseline, 30.9 (3.8) post-treatment and 31.1 (4.5) at follow up and the 19 people in the control group had BMI values of 30.7 (3.1) at baseline, 31.8 (3.4) post-treatment and 32.1 (3.8) at follow up [306].

Another RCT compared two additions to cognitive behavioural therapy: ‘peer-enhanced adventure therapy’ (similar to Outward Bound) and aerobic exercise [307]. Adolescents in both interventions lost significant amounts of weight at the end of treatment (16 weeks) but there was no significant difference in weight loss between groups. At 10 months from randomisation significantly more adolescents in the adventure therapy group had maintained a minimum 4.5 kg weight loss: 35% vs. 12% in the aerobic exercise group.

Three studies had 12 months follow up data. One study showed no effect of adding coping skills training to a four-month behavioural programme for 7–17 year old children. Neither change from baseline in absolute BMI nor differences between groups was significant. A meta-analysis of 12 month follow up data from two studies (321 participants) showed that changes in BMI-SDS and absolute BMI in favour of the behavioural management programme that were significant at six months were still significant at 12 months. The difference in BMI-SDS between the behavioural management groups and the control groups at 12 months was −0.14 (95% CI −0.18 to −0.10), and the difference in absolute BMI was −3.17 kg/m² (95% CI −3.38 to −3.17).

One study which had found that, in teenage girls, an internet-based behavioural programme was significantly superior to an internet-based control programme at six months, found that at 24 months follow up there were no longer any significant differences between groups since the girls in the intervention group had regained weight.

**Drug interventions for obese adolescents**

The Cochrane review identified ten studies reporting on drug trials for three medications: metformin (2 studies), sibutramine (5 studies), and orlistat (3 studies).

**Metformin**

Neither of the two Metformin studies reported an analysis based on intention to treat, therefore the reviewers did not consider the effectiveness or otherwise of this drug.

**Orlistat**

Orlistat works by inhibiting the enzymes (lipases) responsible for absorption of dietary fat leading to increased excretion of undigested fat in the stools and creating an energy deficit which promotes weight loss [308].

There were two RCTs of orlistat (trade name Xenical®) which fulfilled Cochrane criteria for meta-analysis. A pooled meta-analysis of data from 579 participants indicated that, in combination with a lifestyle intervention, orlistat (compared to placebo) had an effect on absolute BMI at six months follow up: −0.76 kg/m², (95% CI −1.07 to −0.44, p< 0.00001).

In all three of the orlistat studies withdrawals due to adverse events were higher in the orlistat intervention groups compared to the placebo group, with withdrawal rates ranging from 3.4% to 31.8%. The most common types of adverse events reported in all three studies were associated with the gastrointestinal tract. They included oily spotting, fatty/oily stools or evacuation, increased defecation, cramps and abdominal pain.

One study measured additional adverse effects: cardiovascular effects, gallbladder structure, bone mineral content/density, renal structure and other non-GIT effects. Ten patients in the orlistat group and one in the placebo group developed ECG abnormalities but an independent cardiologist did not consider that these were medication-related. At the end of the study six orlistat patients and one placebo patient were found to have asymptomatic gallstones that had not been seen at baseline and another orlistat patient had multiple gallstones at day 167, after a 15.8 kg weight loss, and later had a cholecystectomy. Ultrasound identified two new renal abnormalities in the orlistat group.
The most common other adverse events that were more common in the orlistat group were headache, upper respiratory tract infection and nasopharyngitis.

Orlistat is listed in the New Zealand Formulary as a medication for adults [309] but the Ministry of Health's 2009 publication *Clinical Guidelines for Weight Management in New Zealand Children and Young People* suggests it may be considered in addition to lifestyle modification to assist weight control in obese young people (BMI ≥ 95th percentile) but only if a lifestyle change programme has failed and specialist services with experience in the use of anti-obesity drugs supervise its use [269]. Orlistat is not a funded medication and is relatively expensive, costing around $180 for a 1-month supply [310].

**Sibutramine**

The Cochrane review found a favourable effect of sibutramine (trade name Reductil®) plus lifestyle interventions compared to placebo plus lifestyle interventions at six months. Sibutramine has been withdrawn from sale in a number of countries, including New Zealand, because a major study found it increased the risks of heart attack and stroke [311].

**Bariatric surgery**

There were no studies of surgical interventions in adolescents that were eligible for inclusion in the Cochrane review. Another 2009 Cochrane review looked at surgery for obesity in adults [312]. This review included 26 studies: 20 RCTs comparing different bariatric procedures and three RCTs and three prospective cohort studies comparing surgery with non-surgical management. The authors concluded that surgery results in greater weight loss than conventional treatment, both in moderate (BMI > 30 kg/m²), and severe obesity (BMI > 40 kg/m²), that the weight loss from surgery persists for at least ten years, and that surgery also leads to reductions in comorbidities such as diabetes and hypertension. There were improvements in health-related quality of life at two years post-surgery but effects at ten years were mixed with improvements in some quality of life domains but not others. Surgery is associated with significant complications, including pulmonary embolism, and there have been deaths following surgery.

### Conclusions from the Cochrane Review

The 2009 Cochrane review made a number of useful observations. They stated that:

- Family-based lifestyle interventions that include a behavioural component aimed at changing thinking patterns regarding diet and physical activity produce significant and clinically meaningful reductions in overweight in children and adolescents, compared to self-help or standard care in the short and long term.

- Parental involvement is important, particularly for pre-adolescent children,

- Consideration may be given to the adjunctive use of orlistat in adolescents but this therapy needs to be carefully weighed against possible adverse side effects.

- It was not possible to determine whether any one lifestyle intervention was better than another.

The authors also noted that most of the studies included in the review were small (44 out of 64 randomised <30 children to at least one group), most did not account for missing data, many had high dropout rates, and less than half performed an analysis based on intention to treat. Many studies were based in specialist clinics and some studies reported that transportation difficulties were a barrier to participation. Most of the lifestyle intervention studies (36 out of 54) did not report on measures of harm but 18 reported on adverse effects such as disordered eating, depression or anxiety and these studies reported no adverse effects on eating behaviours or psychological well-being. Lifestyle studies commonly reported on reasons for dropout and changes in linear height growth. No lifestyle studies reported an adverse effect of the intervention on linear height growth.

### Insights from other reviews of obesity interventions

This section presents information from a number of recent systematic reviews investigating the effectiveness of various obesity interventions in different age groups, plus the results of a few recent randomised controlled trials.
Timing of solid food introduction for infants
There is much debate about the appropriate time to introduce solid foods into an infant’s diet. The World Health Organization recommends exclusive breastfeeding for the first six months [313]. A 2010 systematic review by Moorcroft et al. considered whether there was an association between the timing of introducing solid foods in infancy and obesity in childhood [314]. Studies were included only if they were undertaken in developed countries and measured obesity in infancy and/or childhood using an appropriate measure such as BMI or skinfold thickness or circumference measures, and if they were randomised, observational or case-control studies. The authors identified 24 studies that met their criteria (mostly cohort studies), with a total of over 34,000 participants. No clear association was found between the timing of introduction of solid food and the risk of overweight and obesity in infancy and childhood. The authors concluded that, when the whole complex situation regarding childhood obesity is considered, a whole family approach to the prevention of childhood obesity is necessary and that concentrating on a range of modifiable factors is likely to be more effective than concentrating on any single factor in isolation.

Physical activity interventions
Many interventions to treat childhood overweight and obesity incorporate physical activity components [315]. As part of their 2011 review on interventions for childhood obesity, Canoy and Bundred assessed the effect of physical activity interventions alone for helping children lose weight. They identified two systematic reviews on this topic [298,299] and two subsequent RCTs [316,317]. One of the reviews was the 2009 Cochrane review discussed earlier. The other, by McGovern et al. included 20 RCTs of physical activity interventions, five of which were also included in the Cochrane review [298]. The authors stated that the 17 trials with complete data yielded inconsistent results. When the trials were combined in two separate meta-analyses according to whether they had measured intervention effects as changes in BMI or changes in fat mass, physical activity interventions had an effect on fat mass (6 trials, 358 participants, SMD= −0.52, 95% CI −0.73 to −0.30) but not on BMI (11 trials, 433 participants, SMD= −0.02, 95% CI −0.21 to 0.18) although the authors stated that reporting bias may explain this finding.

Dietary interventions
Since the publication of the 2009 Cochrane review there have been no new systematic reviews comparing the effectiveness of different dietary interventions for treating (as opposed to preventing) childhood obesity. Two earlier systematic reviews [318,319], were both published in 2006. Gibson et al. identified nine studies, seven of which were RCTs [319]. They reported that “low carbohydrate and low-glycaemic index diets appeared to be at least as effective as energy-restricted low fat diets for short-term weight loss, but most studies were too small to be informative, and none provided evidence on long-term weight control”. They concluded that there was little evidence to support current dietary recommendations for weight reduction in children and adolescents and that there was an urgent need for well-designed RCTs to evaluate the long term effectiveness of alternative dietary interventions.

A review by Collins et al. [318,320] reported on RCTs that included a dietary component either alone or in combination with lifestyle changes and/or psychological therapies. The authors identified 37 RCTs (2262 participants in total). Only seven studies compared a dietary intervention alone with a non-intervention control group or a different treatment approach. Seventeen studies contained enough information to be included in a Forest plot of standardised effects but only a minority had an adequate control group and the treatments studied were highly diverse so the authors did not consider a meta-analysis appropriate. They did, however, perform meta-analyses of the results of the eight studies that included both a dietary component and an adequate control group and of the results of the four of these studies which had follow-up data (at ≤ 15 months). While the authors stated that their results should be viewed with caution because diet was only a component of the interventions they suggested that the results of the meta-analyses indicated that dietary components were effective in achieving weight loss but that the effects of 340
interventions diminished over time. They stated that the two studies with the greatest standardised effect, neither of which reported follow-up data, reported reductions in the per cent body fat in adolescents of between three and six per cent. Overall, the authors concluded that “It is not possible to evaluate the effectiveness of dietary treatment for childhood obesity because of the lack of high-quality studies and the heterogeneity of designs, treatment combinations, outcome measures, and follow-up”. They stated that there was an urgent need to improve the quality of studies in this area.

In another review, Collins et al. highlighted some of the difficulties in measuring children’s dietary intake for research studies and discuss how they contribute to the current limitations of the evidence base for dietary interventions [321]. Often studies rely on the child’s or the parent’s recall of what has been eaten and this information may be biased for a number of reasons: study participants may give inaccurate responses that they feel are socially desirable or likely to meet with approval, children tend to be less accurate at identifying portion sizes than adults, overweight children may be sensitive about their food intake and under report what they have eaten, and children of different ethnic or cultural backgrounds may differ in how accurately they recall their food intake. The use of doubly labelled water (DLW) provides a technique for accurately measuring total energy expenditure, which is close to dietary energy intake since only 1–2% of a child’s energy intake is used for growth, but this method is expensive, technically demanding and of limited availability. Studies which have compared energy intake from reported food intake with the doubly labelled water method have shown that in younger children there can be large individual differences between parent reported energy intake and energy expenditure as measured by DLW and that in older children and adolescents under-reporting of energy intake using food records increases with age, females are more likely to under-report than males, and obese children are more likely to under-report than lean children.

**Family-based interventions**

The family is a key component of obesity interventions since the family is the major determinant of a child’s eating and lifestyle habits and obese children frequently have obese parents [322].

Sung-Chan et al. conducted a systematic review of RCTs that had investigated family-based models for interventions to treat childhood obesity [322]. They included 15 RCTs of family-based lifestyle interventions for children and adolescents aged 2–19 years (published from 1975 to 2010), 3 of which were also included in the 2009 Cochrane review [299] discussed earlier.

They considered that overall these RCTs were of satisfactory methodological quality. Almost all studies (93%) had a sample size of less than 40 and only 66% reported follow-up results of the effects of treatment. Sixty per cent (9 of the 15) made follow-up measurements at 6–12 months after treatment and one study reported follow-up measurements at three months.

They classified the interventions into four categories based on the two underlying theoretical frameworks for the interventions: behavioural approach (8 studies), behavioural approach plus additional training in parenting and child management (5 studies), family approach (1 study) and a combination of behavioural and family therapy approaches (1 study). They assigned outcome scores ranging from 1 to 4 to each study according to whether the weight reductions in the treatment group (compared to the control group) were not significantly better (score=1), marginally better (score=2), significantly better, but not maintained at follow-up or there was no follow-up (score=3) or significantly better and largely maintained in the follow-up period (score =4).

Interventions based on behaviour theory aim to reduce the risk of child obesity by encouraging the adoption of a healthy lifestyle, particularly in regard to diet and exercise. Parents and children are taught behavioural knowledge about self-monitoring, goal setting for eating and physical activity, behavioural contracting and relapse prevention. Some behaviour theory-based interventions also include parent education aimed at improving authoritative parenting styles. Sun-Chen et al. found that, of the 15 studies that used a
behavioural approach, the eight RCTs that focussed on healthy eating and exercising and involved one family member or the whole family were more effective (mean score = 3.5) than the five RCTs that incorporated child management and parenting style components in addition to a family-based healthy lifestyle intervention (mean score = 2.6).

Interventions based on family therapy draw on the perspective of family systems theory which maintains that family dynamics are the key to understanding how the family, as a basic social system, influences children’s behaviour via patterns of interaction between family members. According to family systems theory, child obesity is an expression of dysfunctional family dynamics, maintained via the development of an unhealthy lifestyle. Well-functioning families can adapt easily if lifestyle changes are needed whereas poorly-functioning families become more rigid in the face of change, making it difficult for them to adopt new patterns of diet and exercise. In one of the few examples of this approach to treating childhood obesity, Flodmark et al. [323] offered brief family therapy (six sessions spread over one year) in addition to dietary counselling and medical check-ups over a period of 14 to 18 months. During therapy sessions, family therapists tried to reinforce the families’ resources and create an optimal emotional climate for helping the obese child. This three-arm RCT found that one year after the end of treatment, there was a significantly smaller increase in BMI in the family therapy group compared to the control (no intervention) group (mean +5.1% vs. +12.0%, p=0.022) but none of the differences between the family therapy and conventional treatment groups, or between the conventional treatment and the untreated control group, were significant.

One of the studies identified by Sun-Chen et al. could be classified as having used a hybrid approach, incorporating elements from both Family Systems and Social Cognitive Theories to enhance family variables (family competence, nurturance, conflict resolution and cohesion) and to help participants gain knowledge and self-esteem, understand the benefits of not being obese, and develop skills in self-monitoring, goal setting, substituting healthful alternatives, and enlisting social support [324]. This study randomised 42 adolescent girls (with BMI ≥ 95th percentile) and their families into three groups: a multifamily therapy plus psycho-education group (n=14), a psycho-education only group (n=13) and a control (wait list) group (n=8). At the conclusion of the 16 week trial, none of the participants had significant changes in BMI but those in the psycho-education only groups showed a greater decrease in energy intake (based on a dietitian-administered structured interview to determine 24 hour diet recall) compared to the multifamily therapy plus psycho-education group (p<0.01). There was an association between positive changes in family nurturance and lower levels of adolescent energy intake (p< 0.05) and the authors stated that this indicated nurturance can be an important family variable to target in adolescent dietary and weight loss programmes.

The use of Health Information Technology in the treatment of childhood obesity

The 2009 US Congressional Act, Health Information Technology for Economic and Clinical Health, includes incentives for using IT to facilitate delivery of BMI screening and counselling on diet and physical activity, e.g. by using computerised growth charts [325,326].

A recently published systematic review by Smith et al. examined the effect of health IT (electronic health records, telemedicine, text messaging or telephone support) on care processes and patient outcomes in paediatric obesity management [327]. This review identified five treatment studies (4 RCTs and one before-and-after study) that reported patient outcomes, with sample sizes ranging from 17 to 475 participants, at one to ten practice sites. Three of the treatment studies focussed on obese children aged 8–12 years, one on obese younger children aged 2–6.9 years, and one on overweight adolescents aged 13–16 years.

Of the two telemedicine studies, one was a RCT (17 participants) of group counselling and one a before-and after study (294 participants) of individual counselling. The group counselling study did not demonstrate any improvement in patient outcomes, including BMI z-score but the individual counselling study found that 64% of children counselled by
telephone had decreased BMI percentile at one year (compared to 69% of children counselled in person).

Three studies looked at the effects of text messaging and telephone support on BMI and other clinical outcomes. One RCT (220 participants) involving group counselling offered some families an additional 10 maintenance sessions using automated telephone counselling. Those children whose families completed 6–10, but not those who completed 0–5, telephone sessions had greater decreases in BMI z-scores at one year than children whose families received group counselling alone. Another RCT (151 participants) compared adolescents who received group counselling followed by text message, telephone, or e-mail contact every other week to adolescents who received group counselling alone and found no difference in mean changes in BMI, waist circumference, or blood pressure at one year, but there was low adolescent engagement since <22% of messages marked “please reply” were replied to. In the largest RCT (475 participants) no difference in BMI or BMI z-score was found between children who received enhanced weight management including three 15-minute phone calls, and those who received usual care at 1 year, but although all intervention participants were offered three clinic visits and three phone calls less than half of families completed two or more calls or visits.

The authors considered that health IT interventions increase access to obesity treatment and can decrease travel costs for families but their impact on weight loss and other outcomes has been insufficiently studied and inconsistent.

This review was reviewed by the NHS Centre for Reviews and Dissemination (CRD) [328]. The CRD reviewers commented that, “Given the potential for bias in the review, poor quality of the included studies and limited evidence synthesis, the authors’ conclusions regarding treatment access and adherence to guidelines may be overstated”.

**Interventions for children under the age of two years**

As previously discussed, there is evidence that a child’s weight status and weight gain trajectory early in life may have implications for future obesity status. For this reason, Ciampa et al. conducted a systematic review to assess the evidence for interventions to prevent or reduce overweight and obesity in children under the age of two years [329]. They identified 10 studies of poor to fair quality, eight of which used educational interventions to promote healthy dietary behaviours and two of which used a combination of nutrition education and a guided programme of physical activity.

There were a variety of study settings: home (n=2), classroom (n=4), clinic (n=3) and a combination (n=1). The interventions generally lasted for less than six months and had only modest success in altering measures such as dietary intake and parent’s attitudes and knowledge about nutrition. None of the studies improved child weight status.

The authors concluded that few published studies had attempted preventive or therapeutic obesity interventions in very young children but there was limited evidence that interventions may improve parent’s knowledge and attitudes about nutrition for young children.

The Centre for Reviews and Dissemination commentary on this review stated: “The substandard quality of included studies and potential methodological limitations in the review process mean that the authors' conclusion might not be reliable” [330].

**Interventions for pre-school children**

Noting that two previous systematic reviews of weight management schemes for the under-fives had included studies of uncontrolled design and with potentially biased self-reported outcomes, Bond et al. restricted their 2009 systematic review to controlled trials with objective outcome measures [331]. They found four RCTs assessing the effectiveness of preventive interventions but no treatment or cost-effectiveness studies.

Only one of the prevention trials (in a Latino community) showed a statistically significant advantage from the intervention in terms of a slower rate of increase in BMI but in the other three studies trends in decrease in BMI and weight loss favoured the intervention groups. Bond et al. hypothesised that important components to include in future
interventions might be effective training for staff involved in delivering the intervention, cultural sensitivity, sustained moderate to vigorous exercise, active engagement of parents as participants in the programme and as role models for healthy lifestyles, and nutritional education for children.

The Centre for Reviews and Dissemination considered that this was a well-conducted review and that the authors’ cautious conclusions reflected the scarce and disparate evidence for obesity interventions in the under-fives [332].

Since the publication of the Bond et al. review in 2009, there have been a few RCTs of interventions for pre-school children. The “High Five for Kids” and the “Buffalo Healthy Tots” studies are discussed in a later section on primary care, while the “LAUNCH” study is reviewed in the text box below.

The LAUNCH study
Stark et al. conducted a pilot RCT to evaluate the efficacy of a 6-month clinic and home-based intervention, known as LAUNCH (Learning about Activity and Understanding Nutrition for Child Health), for obese (BMI ≥95<sup>th</sup> percentile) pre-schoolers [333]. The Launch intervention consisted of two phases. Phase one consisted of 12 weekly sessions that alternated between group-based clinic sessions (concurrent groups for parents and children) and individual home visits. The parent group sessions were conducted by a licensed clinical psychologist following a written manual. Phase two (the maintenance phase) consisted of 12 weeks of every other week sessions alternating between group sessions and home visits. The 90-minute parent group sessions addressed dietary education, physical activity and parenting skills. Parents were taught techniques to manage child behaviour including: praise and attention to increase healthy eating and physical activity; ignoring and time-out to manage tantrums; contingency management; and modelling. The child group sessions included nutrition education via games and art activities, trying new foods during a structured meal and 15 minutes vigorous physical activity. The home visits were carried out by psychology postdoctoral fellows and were designed to support generalisation of clinic learning to the home and help parents eliminate unhealthy foods from the home and set up a safe place to play.

Fifty-six eligible families were identified from records of a large U.S paediatric practice, with 38 declining to participate. Eighteen families were randomised to either LAUNCH or a control intervention consisting of a single session of paediatrician counselling (PC) with recommendations for diet and physical activity. At six months, there were statistically significant differences in weight outcomes between the LAUNCH children and the PC children as follows: BMI z-score (−0.59 ± 0.17), BMI percentile (−2.4 ± 1.0), and weight gain (−2.7 kg ± 1.2) and these differences were increased at 12-month follow-up. The difference in weight loss between the LAUNCH parents and the PC parents was also significant: (−5.5 kg ± 0.9) at month 6 and (−8.0 kg ± 3.5) at month 12.

The authors concluded that, based on the data from their small sample, an intensive intervention including child behaviour management strategies to improve healthy eating and activity appeared to be more promising for reducing preschool obesity than a low intensity intervention that was typical of treatment that could be delivered in primary care.

Weight loss camps and other residential interventions
In some countries, including the U.S. and U.K, children’s weight loss camps are an option for some obese children. These camps typically combine dietary restriction, physical activity and behaviour modification [334]. They may be for-profit commercial enterprises or non-profits run in association with academic institutions or children’s hospitals [335]. They are usually only accessible to children from relatively wealthy families since the fees are normally paid by parents, but in the U.K. the National Health Service has paid for some children to attend the Carnegie Weight Management residential camp [336], now known as More-life [337].

Kelly and Kirschenbaum have reviewed published studies on “immersion treatment” (weight loss camps and other residential programmes) [338]. These authors, who are both employees of Wellspring, a leading provider of weight loss camps in the U.S. [339], identified 22 published studies of interventions which targeted and assessed change in weight status and involved a minimum stay of 10 days and nights. The interventions typically included controlled diet, activities, therapy and/or education regarding behaviour change and nutrition education. The authors stated that: “compared with results
highlighted in a recent meta-analysis of out-patient treatments, these immersion programmes produced an average of 191% greater reduction in per cent-overweight at post treatment and 130% greater reduction at follow-up. They also stated that their review showed that interventions which included cognitive behaviour therapy seemed to have better outcomes and “outperform the non-CBT interventions by a wide margin” despite the CBT studies generally having longer follow-up periods which tend to be associated with poorer outcomes.

This review was reviewed by the NHS Centre for Reviews and Dissemination (CRD) at the University of York, [340]. The CRD reviewer(s) noted a number of limitations to the review: the studies appeared to be heterogeneous in terms of intervention, design and outcomes; the authors did not state that quality assessment of included studies was performed and it appeared that the study designs were at high risk of bias since although six studies used control or comparison groups, only one reported randomised assignment of participants and only one reported an intention-to-treat analysis; a limited number of databases were searched for published studies in English and therefore publication bias and language bias could not be ruled out. The CRD reviewers considered that the methodological limitations of the review and the considerable risk of bias meant that the conclusions should be interpreted cautiously.

**Lifestyle interventions: impact on weight change and cardio-metabolic risk factors**

A recent systematic review by Ho et al. examined the impact of lifestyle interventions with a dietary component on both weight change and cardio-metabolic risk factors, such as blood pressure, serum lipids and fasting insulin, in overweight and obese children [341]. The review included 38 RCTs, published between 1975 and 2010 and of variable quality, comparing the effectiveness of a lifestyle intervention including a dietary or nutrition component with wait-list or no treatment control, usual care, written diet and physical activity education materials or minimal advice. The number of participants per study ranged from 16 to 258. Thirty three studies had adequate data for meta-analysis on weight change and 15 reported on lipids, fasting insulin or blood pressure.

Compared to no treatment, lifestyle interventions produced significant weight loss (at latest point of follow-up) as indicated by both pooled BMI (−1.25 kg/m², 95% CI −2.18 to −0.32) and BMI z-score (−0.10, 95% CI −0.18 to −0.02). Lifestyle interventions also led to significant weight loss compared to usual care as measured by pooled BMI, both immediately at the end of active treatment (−1.30kg/m², 95% CI −1.58 to −1.03), and at subsequent follow up at 7–12 months (−0.92 kg/m², 95% CI −1.31 to −0.54). Lifestyle interventions led to significant improvements in low-density lipoprotein cholesterol (−0.30 mmol/L, 95% CI 20.45 to 20.15), triglycerides (−0.15 mmol/L, 95% CI −0.24 to −0.07), fasting insulin (−55.1 pmol/L, 95% CI −71.2 to −39.1) and blood pressure up to one year from baseline but for high-density lipoprotein cholesterol no differences were found. The authors noted that, without having individual participants’ data, it wasn’t possible to determine the relationship between the extent of weight loss and changes in the various cardio-metabolic outcomes. They also noted that the heterogeneity of the studies included in the review made it difficult to provide definitive recommendations for practice but they stated that almost all of the effective interventions, especially those in children under 12 years old, reported including a family component that included separate education sessions for parents and children.

The authors concluded that lifestyle interventions which include a dietary component together with an exercise or behavioural component are effective for treating childhood obesity and improving cardio-metabolic outcomes. The Centre for Reviews and Dissemination regarded this review as being generally well-conducted but noted that variations in intervention settings, constituent components, and duration meant that the evidence did not provide a clear indication on which intervention format was likely to be most effective in practice and in the long term [342].

**Metformin for the treatment of overweight and obesity in adolescents**

Metformin is an oral hypoglycaemic agent and is the most widely used drug for the treatment of type 2 diabetes in adults. Its primary action seems to be the inhibition of
hepatic glucose production. At high concentrations, it also increases peripheral insulin sensitivity and glucose uptake [343]. As a consequence of the increase in prevalence of obesity in children and adolescents, there has been an increase in the number of children and adolescents with type 2 diabetes. Since insulin resistance related to excessive weight gain is a first step on the pathway to type 2 diabetes, metformin has been used in obese children and adolescents who are not diabetic to reduce overweight and prevent or delay the onset of type 2 diabetes.

Two recent reviews have examined the use of metformin in overweight or obese non-diabetic children or adolescents [343,344].

A 2012 review by Brufani et al. identified 11 trials with duration of six months or more. The number of participants ranged from 16 to 151. All except one focussed mostly on adolescents. Eight were double-blind placebo RCTs and three compared metformin plus lifestyle intervention to lifestyle intervention alone without placebo. The trials differed in inclusion criteria, the use (or not) and type of lifestyle interventions, the indicators of insulin resistance/sensitivity, metformin dosage and participant ethnicity so the authors did not consider meta-analysis to be justified. Most of the trials (nine out of eleven) found a small but significant benefit of metformin in decreasing BMI by from 1.1 to 2.7 kg/m² compared to placebo or lifestyle intervention alone. The authors concluded that metformin has a very modest effect as an anti-obesity drug and noted that the trials in children and adolescents with severe obesity (BMI ≥ 32) had mean BMI reduction of from 1.1 to 1.7 kg/m² which is clinically insignificant.

Bouza et al. included nine RCTs in their review of the use of metformin in overweight and obese adolescents (498 participants, mean age 14.2 years, and mean BMI 36.4 kg/m²). All but one compared metformin plus lifestyle intervention to placebo plus lifestyle intervention. Meta-analysis indicated that metformin reduced mean BMI by 1.42 kg/m² (95% CI −2.18 to 0.66) and also had favourable effects on fasting insulin and the HOMA index (the homeostasis model assessment – a method of assessing β-cell function). Bouza et al. concluded that the available evidence indicated that, in the short term, metformin in addition to lifestyle intervention is relatively effective at reducing BMI and hyperinsulinaemia in obese adolescents without “related morbidity” (presumably without diabetes), and has an acceptable safety profile, but its long term effects are unknown. The NHS Centre for Reviews and Dissemination (CRD) commented on this review and pointed out that the estimate of change in BMI was very small and it was unclear whether it would be clinically significant among obese people [345]. The CRD reviewer(s) stated that: “Overall, the authors' conclusions reflect the evidence presented but cannot be considered reliable due to limitations of the evidence base”.

**Bariatric surgery**

While surgery for obesity is not generally recommended for obese children or young people it has increasingly been used for treatment of those with extreme obesity and obesity-related comorbidities when more conservative treatment methods have failed [346]. The 2006 guidelines from the U.K. National Institute for Clinical Excellence [346] noted that there were (at that time) only three published guidelines that contained recommendations relating to bariatric surgery in adolescents: NHMRC Australian guidelines for the management of overweight and obese children and adolescents, the Singapore Ministry of Health clinical guidelines and the Institute for Clinical Systems Improvement (ICSI) guidelines. The Singapore guidelines [347] are now out of date but updated guidelines from the NHMRC [112] and the ICSI [264] suggest that a post-pubertal adolescent with a BMI of > 40 kg/m², or > 35 kg/m² plus significant severe comorbidities such as type 2 diabetes or obstructive sleep apnoea, may be considered for bariatric surgery if other interventions have been unsuccessful. A working party from the Royal Australasian College of Physicians made similar recommendations [348].

There are a number of different surgical procedures used in bariatric surgery and they are all usually done laparoscopically. They include the Roux-en-Y gastric bypass, the adjustable gastric band, biliopancreatic diversion and the sleeve gastrectomy [262]. The best-studied procedure in adolescents is probably the Roux-en-Y gastric bypass. In this
procedure, the stomach is stapled to exclude almost all of the stomach volume and create a small pouch at the top of the stomach. This is separated from the main body of the stomach and attached to the small intestine bypassing the duodenum and the proximal 20–39 cm of jejunum. The bypassed intestine coming from the main body of the stomach is then joined to the intestine beyond the new stomach outlet to allow drainage of gastric secretions. Weight loss ensues from restriction of food intake and malabsorption [263]. Adverse effects that may follow the procedure include anastomotic leak, small bowel obstruction, dumping syndrome (symptoms that may include nausea, vomiting, bloating, cramps, diarrhoea and/or other symptoms), protein-calorie malnutrition, and micronutrient deficiency related to malabsorption [264].

**Research on bariatric surgery outcomes in adolescents**

There has been limited research on the effectiveness of bariatric surgery for obese adolescents. Treadwell et al. reviewed studies (published in English up until December 2007) that had reported outcomes on three or more patients aged ≤21 years who represented at least 50% of the paediatric surgical patients enrolled at a centre, and had followed up patients for at least one year. There were eight studies (352 patients, mean BMI 45.8 kg/m²) on laparoscopic adjustable gastric banding (LAGB), six studies (131 patients, mean BMI 51.8 kg/m²) on Roux-en-Y gastric bypass (RYGB), and five studies (158 patients, mean BMI 48.8 kg.m²) of other surgical procedures. The patients had an average age of 16.8 years and the age range was nine to 21 years. Of the total of eighteen studies only one reported on a control group of patients not treated by bariatric surgery and the patients in the control group were significantly different to the surgery patients since they had a lower BMI and no reported comorbidities. Most studies were retrospective and therefore possibly biased towards favourable outcomes since centres with unfavourable outcomes are less likely to choose to publish their results. There was considerable heterogeneity between studies.

Treadwell et.al conducted meta-analyses of the results of data on BMI reduction from six studies of LAGB and four studies of RYGB [349]. They reported that, for LAGB, the 95% confidence interval for change in BMI post-surgery was from −10.6 to −13.7 BMI units, and for RYGB it was −17.8 to −22.3 BMI units. Eight per cent (28/352) of the LAGB patients required re-operation because of various complications, and, in addition, there were eight cases of iron deficiency and five cases of mild hair loss. The RYGB studies reported that, although there were no in-hospital deaths, one patient died nine months after surgery due to severe *Clostridium difficile* colitis, and three other patients died from causes considered to be unrelated to the bariatric surgeries. The most frequently reported complication of RYGB was protein-calorie malnutrition and micronutrient deficiency but there were some potentially life-threatening complications including shock, pulmonary embolism, post-operative bleeding, severe malnutrition and gastrointestinal obstruction. Treadwell et al. concluded that bariatric surgery in paediatric patients produces clinically significant weight loss, but can have serious complications.

In their commentary on Treadwell et al.’s review, the NHS Centre for Reviews and Dissemination stated that the quality of the studies included in the review was low (almost all were case series), and the reporting of the review process and results was poor so the reliability of the authors’ conclusions was unclear [350].

There has been a prospective RCT of gastric banding in adolescents which was conducted in Melbourne during 2004–08 [351]. It involved 50 patients between the ages of 14 and 18 years, with mean BMI > 35 kg/m², who were randomised to either gastric banding or a supervised lifestyle intervention and followed up for two years. Those in the gastric banding group at follow up (data from 24/25 patients) had a mean weight loss of 34.6 kg (95% CI 30.2 to 39.0kg ), representing an excess weight loss of 78.8% (95% CI 66.6% to 91.0%), 12.7 BMI units (95% CI 11.3 to 14.2), and a BMI z-score change from 2.39 (95% CI, 2.05 to 2.73) to 1.32 (95% CI, 0.98 to 1.66) while the lifestyle group (18/25) had a mean weight loss of 3.0 kg (95% CI 2.1 to 8.1), representing excess weight loss of 13.2% (95% CI 2.6% to 21.0%), 1.3 BMI units (95% CI 0.4 to 2.9), and a BMI z-score change from 2.41 (95% CI 2.21 to 2.66) to 2.26 (95% CI, 1.91 to 2.43). At baseline, nine in the
gastric banding and ten in the lifestyle group had metabolic syndrome and at follow up none in the gastric banding but four (out of 18) in the lifestyle group did. Eight of those in the gastric banding group required subsequent operations for either proximal pouch dilation or tubing injury. The authors concluded that gastric banding, compared to lifestyle intervention, resulted in a greater percentage of subjects achieving a loss of 50% of excess weight and greater improvements to health and quality of life.

Padwall et al. identified 31 RCTs of bariatric surgery in adults (2,619 patients, mean age 30–48 years, mean BMI 42–58 kg/m²) [352]. Compared to standard care, data from 15 trials (1103 participants) showed the following mean differences (MD) in BMI from baseline at one year: jejunoileal bypass (MD: −11.4 kg/m²), mini-gastric bypass (−11.3 kg/m²), biliopancreatic diversion (−11.2 kg/m²), sleeve gastrectomy (−10.1 kg/m²), Roux-en-Y gastric bypass (−9.0 kg/m²), horizontal gastroplasty (−5.0 kg/m²), vertical banded gastroplasty (−6.4 kg/m²), and adjustable gastric banding (−2.4 kg/m²). Padwall et al. concluded that, although data from large, adequately powered long term RCTs was lacking, bariatric surgery appeared substantially more efficacious than standard care for reducing BMI and that, compared to Roux-en-Y gastric bypass, adjustable gastric banding produces less weight loss but has fewer serious adverse effects. The NHS Centre for Reviews and Dissemination reviewed this review and considered that, based on the limited evidence available, the authors’ conclusions were likely to be reliable [353].

One of the key reasons for encouraging weight loss in obese children and adolescents is the belief that weight loss will reduce the risk of developing metabolic syndrome, diabetes and cardiovascular disease later in life, but, according to a 2013 systematic review produced by the Australian National Health and Medical Research Council, “there are no longer term data available from high quality studies that assess the impact of bariatric surgery on cardio-metabolic outcomes in adolescent patients” so the effect of adolescent bariatric surgery on future disease risk is as yet unknown [354]. While most obese adults who have bariatric surgery lose substantial amounts of weight, many initially have such high BMIs that even after substantial weight loss following surgery they still have BMIs in the obese range. This observation has been used as an argument for intervening earlier in life [355,356].

<table>
<thead>
<tr>
<th>Conclusions from other reviews of obesity interventions</th>
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<tr>
<td>The key findings from the other reviews of obesity interventions discussed above are:</td>
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<tr>
<td>• There is no clear association between the timing of introduction of solid food and the risk of overweight and obesity in infancy and childhood</td>
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<tr>
<td>• Physical activity interventions alone probably decrease fat mass but may not result in decreases in BMI</td>
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<tr>
<td>• There is a lack of evidence for the effectiveness of dietary treatment because of a lack of high quality studies</td>
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<tr>
<td>• Family-based interventions are more effective if they include a behavioural component dealing with self-monitoring, goal setting for eating and physical activity, problem solving, behavioural contracting and relapse prevention. Enhancing family competence, nurturance, conflict resolution and cohesion may also be helpful</td>
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<tr>
<td>• The use of IT could increase access to obesity interventions, especially for those in more remote areas, but it is unknown if child or adolescent obesity treatment via IT is effective</td>
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<tr>
<td>• There is little evidence regarding interventions for very young children</td>
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<tr>
<td>• Weight loss camps and other residential interventions may be effective but have not been evaluated via RCTs and their long-term effects are largely unknown</td>
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<tr>
<td>• Lifestyle interventions which include a dietary component together with an exercise or behavioural component are effective for treating childhood obesity and improving cardio-metabolic outcomes for at least a year from the end of the intervention</td>
</tr>
<tr>
<td>• Metformin produces small and clinically insignificant reductions in BMI in obese adolescents who do not have obesity-related comorbidities (i.e. diabetes) in the short term</td>
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**New Zealand interventions**

Reports of a number of interventions for preventing or reducing the prevalence of childhood overweight and obesity in New Zealand children have been published in the international literature [357,358,359], but none of these interventions have been targeted at obese children alone. There are no published RCTs of New Zealand interventions that are specifically for obese or overweight children only, such as Bodywise and Green Prescription Active Families, although there are published reports of other types of evaluations of these interventions.

Waikato DHB funds Project Energise, a population-based intervention which aims to increase the quality and quantity of physical activity and improve nutrition for primary school children [360,361]. Trained “Energisers” work with schools to develop and support programmes for healthy eating and physical activity. The project included an evaluation of the intervention programme via a RCT which ran from 2004–2006 [360]. Schools were randomised with stratification by rurality and socio-economic status (SES) to receive the intervention (62 schools) or act as controls (62 schools). Children aged five and ten years had weight, height, body fat (by bioimpedance) and resting blood pressure (BP) measured at baseline and two years later. Over the two years, after adjustment for baseline measure, rurality and school decile there was no difference between the intervention and control groups in BMI standard deviation score (SDS, = z score) in either the younger or the older children but the 5–7 year olds in the intervention group had improved % body fat SDS (intervention – control = −0.14, 95% confidence interval −0.26 to −0.01) and the 10–12 year olds in the intervention group had an improvement in systolic blood pressure SDS (intervention – control = −0.23, 95% CI −0.43 to −0.02) and, to a lesser extent, diastolic BP SDS (intervention – control = −0.14, 95% CI −0.30 to 0.04).

Subsequently the intervention was extended to more schools and, by 2011, all but two of the 235 school in the Waikato region were engaged [361]. In 2011, to assess the effect of the programme, researchers compared indices of obesity and physical fitness in seven year old and ten year old children in schools that had been “energised” for at least 18 months with historical measurements. Obesity indices were compared with the 2004 measurements of control children in the earlier RCT and fitness measures (time taken to run 550m) with those of Canterbury children obtained between 2001 and 2007. The 2011 children were a little younger than the historical control children (on average 1.5 months for the younger and 3.6 months for the older children) but of the same average height. The 2011 children had lower prevalence of overweight and obesity combined, (younger children 19.5 vs. 23.8%, older children 26.6 vs. 27.8%). They also had lower BMI (younger children 16.83 vs. 17.36, older children 18.7 vs. 19.3 kg/m²), and had faster times for the 550m run (younger children 183.2 vs. 212.7, older children 164.2 vs. 179.8 seconds). After adjusting for age, sex, ethnicity (not for the run times), SES and school cluster effects, the odds ratios for combined obesity and overweight were: younger children: 0.69 (95% confidence interval 0.54–0.88) and older children: 0.85 (95% CI 0.72–1.00). The adjusted relative (%) differences in mean BMI were: younger children: −3.0 (95% CI −4.3 to −1.7), older children: −2.4 (BF 95% CI −3.7 to −1.1), and the adjusted relative (%) differences in mean time to run 550m were: younger children: −13.7 (BF 95% CI −15.8 to −12.3), older children: −11.3 (95% CI −14.1 to −9.0).

The project evaluation report indicates that children who participated in Project Energise became fitter, had decreased waist measurements compared to earlier cohorts of Waikato children of the same age, and had good knowledge about healthy eating and physical activity [362]. The programme is reported to be affordable, costing around $45 per child per year in 2010, and cost effective [363].

Waikato DHB is currently running Bodywise, a family-focussed 12-month intervention for children aged 5–12 years who require weight management [364]. Bodywise involves an
initial appointment at the hospital children’s clinic, followed by a six week group programme at Sport Waikato (attending twice per week) and monthly follow-up home visits by a dietician and active families coordinator. A formal evaluation of this programme has yet to be published but preliminary results were presented at the CAMHS conference in 2007 [365] and these indicated modest decreases in BMI z-scores but marked improvements in the percentage of programme participants who met food and nutrition guidelines and increases in time spent in physical activity and time spent outdoors. The intervention was well-received by parents and children.

Green Prescription Active Families is an initiative (introduced in 2004 as an offshoot from the Green Prescription for adults) which aims to increase physical activity for children, young people and their families [366]. It is funded by the Ministry of Health. On July 1st 2012 funding and management was devolved to DHBs who currently contract eighteen providers to deliver the initiatives. Criteria for referral to the programme are inactive overweight or obese children who have a family motivated to make lifestyle changes. Priority is given to children aged 5–12 years. The programme includes group sessions with physical activity components, where participants work on individual goals, plus information and education about general well-being, healthy eating and physical activity. People participate for up to 12 months and the long term goal is for each child to have a minimum of 60 minutes of moderate-intensity physical activity on most days.

The latest survey of participants in the Green Prescription Active Families programme involved 133 families (61% of the total) who participated in the programme between July 2012 and May 2013 and it found that the contract holders exceeded all nine of the key performance indicators measured [367]. Eighty-four per cent of participating families reported that they had noticed positive changes in their child’s health since joining the programme particularly that they had more energy, were more willing to try new activities, and were more confident. Seventy-seven per cent said their child was more active and almost all children understood the benefits of healthy eating (87%) and being physically active (83%). Eighty-five per cent said their family had dietary changes, most commonly generally eating more healthily including eating less takeaways or junk food (26%), having less sugar or sugary food and drinks (19%), eating smaller meals (14%) and eating more fruit and vegetables (13%). Forty-one per cent said that their child had either lost weight or noticed their clothes being looser. Overall most survey respondents said they were either satisfied (28%) or very satisfied (68%) with the programme.

Key points about New Zealand Interventions

- Project Energise, a DHB-funded school-based intervention for primary school children in the Waikato, is a promising population-based preventive intervention
- Bodywise (funded by Waikato DHB) and Green Prescription Active Families (funded by all DHBs) are interventions for overweight and obese children that are well regarded by families. There is no clear evidence that they improve children’s BMIs but participants in these interventions report improvements in behaviours related to diet and physical activity

Primary care interventions

While much of the research into treatment programmes for obese children has been done in specialist hospital clinics, given the large number of children who are now overweight or obese there is a clear need for interventions that are based in primary care or other community settings [368]. Vine et al. recently reviewed the published literature from 2006 to 2012 to provide U.S. examples of the range of roles that primary care providers can play in the prevention and treatment of childhood obesity and to synthesise evidence concerning the important characteristics, strategies or features of successful community-based models [368]. They noted that a U.S. nationwide survey of primary care providers (PCPs) found that fewer than half were assessing BMI percentiles regularly in children despite this being recommended by the White House Taskforce on Childhood Obesity [158], the American Academy of Pediatrics [276] and the American Heart Association [369], and only 18% reported referring children for further evaluation or management [370].
This review identified seven studies relating to primary care treatment of overweight and obesity in American children and adolescents, none of which were RCTs although one was related to a RCT (it examined the correlates of participation in a trial of an obesity intervention). Vine et al. reported that treatment interventions that involved individual case management or patient-centred counselling over multiple sessions showed some evidence of success. Examples of these kinds of interventions included private, age-appropriate conversations with clinicians about achieving a healthy weight; goal setting; motivational interviewing; and discussions with registered dieticians about patient readiness for long term behavioural change, diet, and exercise.

Vine et al. stated that there is a need for primary care providers to move beyond measuring patients' height and weight and treating health problems and become involved in advocacy, modelling and promoting healthy behaviours in the community, participating in multi-sector community initiatives and counselling individuals and families about obesity prevention. This requires development of clinician skills in evidence-based assessment and counselling techniques and changes to clinical infrastructure and care models.

An example: Healthy weight clinics in Massachusetts
Anand et al. have reported on the development of “Healthy Weight Clinics” established within eight community health centres in Massachusetts serving predominantly poor minority patients among whom child rates of overweight or obesity range from 32% to 47% [371]. There are three key components to this care model: designated condition-specific visits that allow more time than standard primary care visits, multidisciplinary, team-based care, and specialised knowledge and training for members of the primary care team. Patients are seen in a series of one-hour visits by a three-person team consisting of a clinical champion, a dietician and a case manager. The clinical champion, or team leader, deals with the medical assessment of obesity, including reviewing laboratory results, family history and other health conditions such as sleep apnoea. The dietician reviews intake of sweetened beverages, fruit and vegetable consumption, and the ability of the patient (or patient's caregiver) to recall what the patient ate in the previous 24 hours. The case manager assesses sedentary activity, such as watching TV or playing video games, and physical activity. The team helps each family develop a self-management plan that is culturally appropriate and achievable with the available family and community resources, and behaviour modification techniques are used to set treatment goals that are agreed on by the patient, the family and the team. Children are typically seen every one to two months for a total of six visits.

The healthy weight clinic staff meet with staff at other healthy weight clinics via monthly teleconferences and two face-to-face meetings annually to solve common problems and share best practices. All clinics use a web-based quality monitoring system to report on key process and outcome measures including BMI, diet and physical activity. Preliminary results from 174 patients who had more than one clinic visit for the period June 2008–August 2009 were considered promising: 100% had a self-management plan, 79.8% had made any lifestyle change, 29.9% had reduced screen time, 45.5% had increased physical activity, 32.2% had decreased sweetened beverages, 33.3% had increased fruit and vegetables, and 50% had decreased BMI (but it was not reported by how much). Anand et al. consider that the Healthy Weight Clinics provide an example of an effective, efficient and family-centred model of secondary (referral-based) care within primary care, which is easier for patients to access and less costly than hospital-based programmes.

The 2010 USPSTF review of primary care interventions
Whitlock et al. conducted a systematic review for the U.S. Preventive Services Taskforce on the effectiveness of primary care weight management interventions for children and adolescents [372]. The review included controlled trials in primary care-relevant settings of interventions designed to promote weight loss or weight maintenance in overweight (BMI 84th–94th percentile) or obese (BMI ≥ 95th percentile) two to 18 year olds (published in English from 1985 to June 2008). Trials had to report outcomes at least six months from the beginning of treatment and have at least 10 participants in each trial arm. The USPSTF review used different terminology from the 2009 Cochrane review and used the term “behavioural interventions” in a way that corresponds to what the Cochrane review calls “lifestyle interventions” to mean multi-faceted interventions that involve encouraging patients and families to adopt healthier patterns of eating and physical activity and, optimally, also include cognitive and behavioural management techniques to help change thinking patterns about food and the body.
This review included 11 behavioural intervention trials (1099 participants in total, six trials rated good quality, and five fair quality) which measured short term outcomes (6–12 months) in overweight or obese children and adolescents (4–18 years). All except three of these were also included in the 2009 Cochrane review. The three that were not included in the Cochrane review were one study of an internet intervention for adolescents which was not included because it was not published until August 2008, one study excluded because it was not a RCT, and one excluded because it did not have sufficiently long follow-up.

The results of all eleven trials were consistent with a beneficial effect on BMI, BMI SDS (z-score) or percentage overweight although not all effects were statistically significant. In these 11 studies differences between intervention and control groups ranged from 0.3 to 3.3 kg/m², reflecting weight loss as well as weight gain prevention for those in the intervention groups. The largest effects BMI differences of 1.9 to 3.3 kg/m² were seen in three comprehensive weight management programmes (these included diet or weight loss counselling, physical activity counselling or programme, and behavioural management techniques to aid behavioural change), with at least medium (26 to 75 contact hours) or high (≥ 76 contact hours) intensity. Meta-analyses of the results of all eleven weight management programmes in four categories (medium-to-high, low and very low intensity comprehensive interventions and focussed interventions), confirmed the superior effects of medium to high intensity interventions compared to all the other interventions for short term weight change and compared to the other comprehensive interventions for maintenance of weight change. The authors pointed out that the largest reported difference in BMI, 3.3 kg/m² over 6–12 months, would equate to a weight difference (assuming a height on the 50th percentile for age) of about 13 pounds (5.9 kg) for an eight year old boy, 17–18 pounds (7.9 kg) for a 12-year old boy or girl, 19 pounds (8.6 kg) for a 16 year old girl and 22–23 pounds (10.2 kg) for a 16 year old boy.

Owing to the small number of trials, the diversity of intervention components and the fact that most interventions included multiple components Whitlock et.al were not able to judge what the most beneficial elements of weight management programmes were, other than to say that it seemed that interventions with more hours of participant contact were better. While programmes that included organised physical activity appeared to be better than those that encouraged participants to exercise at home, this effect was confounded with treatment intensity and so it was impossible to determine whether it was the exercise programme or the overall treatment intensity that was responsible for the greater likelihood of successful treatment. Whitlock et al. noted that all the medium-to-high intensity interventions reviewed had been conducted in specialty healthcare settings and that the lower intensity (or focussed) interventions that might be feasible in primary care had more modest and less consistent effects on reducing BMI.

The best of the interventions conducted in a primary care setting, involving 44 adolescents aged 12–16 years, assessed a “Healthy Habits” intervention [306]. Participants used a computer programme which assessed participants’ responses questions on eating, physical activity and sedentary behaviour and used this information to produce a personalised plan for improving habits in these areas. They were also given a non-personalised manual on behavioural skills for weight control. A paediatrician discussed the computer-generated plan with each participant and then telephone counsellors contacted the participants weekly for eight weeks and then biweekly for three more calls to help them implement their plan. The telephone counsellors used detailed scripts to ensure their calls covered all the key elements of the plan. At the beginning of the intervention, participants had an average BMI of 31.0 kg/m², well above the 95th percentile. At the end of the four-month treatment phase, the average BMI had fallen to 30.7 kg/m² and three months after that it was 31.1 kg/m². In comparison, the control group’s average BMIs were 30.7 kg/m² at baseline, 31.8 kg/m² at four months and 32.1 kg/m² at follow-up. The difference between the two groups’ baseline to follow up changes in BMI was not statistically significant (but the sample size was quite small, 44 participants in total). Whitlock et al. stated that for a 14 year old girl of height 5′4″ (163 cm) who grew 1″ (2.54 cm) over the study period and who had a BMI equal to the average for the study participants, these BMI differences would mean that over the seven months she would have gained seven pounds (3.2 kg, from 81.6
to 84.8 kg) if she had been in the intervention group and 14 pounds (6.4 kg, from 81.2 to 87.5 kg) if she had been in the control group. Whitlock et al. stated that further research on less intensive interventions suitable for use in primary care was greatly needed.

**Recent RCTs addressing childhood obesity in primary care**

There have been a number of RCTs of obesity interventions for children or adolescents in primary care. The text box below reviews a number of recent RCTs of primary care interventions in pre-adolescent children, which were published after the 2008 cut-off for inclusion in the USPSTF review.

### Examples of primary care interventions to treat obesity in pre-adolescent children

#### The “High Five For Kids” study

The “High Five For Kids” study, is a cluster RCT involving 10 paediatric primary care offices of a multi-site group practice in Massachusetts [373]. In the trial, 475 children aged 2–6.9 years with either a BMI ≥ the 95th percentile, or a BMI ≥ 85th and < 95th percentile and at least one overweight (BMI ≥ 25) parent, were randomised to either usual care or an intervention carried out by paediatric nurse practitioners trained in motivational interviewing. This consisted of four 25-minute in person chronic-disease visits and three 15-minute telephone calls in the first year. The behavioural goals were less than one hour per day television/video viewing, no television in rooms where children sleep, one serving or less per week of fast food, and one serving or less per day of sugar-sweetened beverages.

After one year, the difference in mean BMI between the usual care group and the intervention group was not significant (−0.21; 95% confidence interval −0.50 to 0.07; p=0.15). Differences in consumption of fast-food and sweetened beverages were also non-significant but there was a significant difference in television viewing (−0.36 hours/day; 95% CI, −0.64 to −0.09; p=0.01). The authors noted that their observed BMI differences were of similar magnitude to those seen in the LEAP trial (see below) and they offered four possible reasons for the lack of a significant effect on BMI: it involved only the primary care setting and not the children’s environments; adherence to the intervention was relatively low with only a little over half of participants completing at least two of the six visits/telephone calls; the motivational interviewing technique used allowed parents to choose to work on behaviours that could have had a lesser effect on BMI, such as increasing fruit and vegetable intake; and it is possible that BMI changes might lag behind behavioural changes.

Taveras et al. reported on the correlates of participating (475 parents) and refusing to participate (329 parents) in the above trial [374]. Parents were less likely to participate if they had a college degree and if their child was overweight rather than obese. Among the refusers with an obese child, 21% said they wouldn’t participate as their child did not have a weight problem as did 30% of the refusers with an overweight child. Other reasons for not participating included: “study will take up too much time” (60%), “things (being) too difficult in the family right now—illness, divorce, new baby etc.” (9%), and “clinical site too far away” (5%). Taveras et al. suggested that to prevent and manage obesity in pre-school children it is necessary to raise parental awareness of their child’s weight status and the potential health risks associated with obesity, and also to address parental concerns about the time commitment required to participate in an obesity intervention.

#### Buffalo Healthy Tots

Quattrin et al. reported on a RCT designed to test the efficacy of a family-based primary care behavioural intervention for weight control, known as Buffalo Healthy Tots [375]. In this study, 105 children aged 2–5 years with a BMI ≥ 85th percentile and an overweight parent were recruited at a well- or sick-child visit to one of four suburban practices and randomised to receive either the intervention or an information-only control.

Both the intervention and control groups were offered ten 60-minute group meetings over six months and eight phone calls between meetings from an assigned “coach”. The meetings for both groups involved a group leader delivering education on diet and physical and sedentary activities to the parents and trained staff engaging the children in active ball games. In addition, at the intervention group meetings, the group leader stressed behavioural and parenting strategies to promote parent and child behaviour change, such as selective ignoring, time out, praising, rewarding and contracting, and strategies aimed at changing parent behaviour in areas that would facilitate child and parent change, such as pre-planning, stimulus control, shaping, modelling, self-monitoring, changing the home environment, social support and changing black and white thinking. Either before or after the group meeting, each parent in the intervention group also had a one-to-one meeting with an assigned coach, who helped the parent with shaping behavioural goals after reviewing the parent’s and child’s (parent-kept) food, activity and weight diaries.
Ninety six of the 105 randomised families started the programme and there were no baseline differences between the intervention (46 children) and control groups (50 children). The authors expressed the changes in children’s weights in units of %0BMI, defined as ((actual BMI – 50th percentile BMI)/ 50th percentile BMI)*100. Adjusted mean (±SD) estimates for child %0BMI at baseline, three and six months were 30.6 ± 9.7, 26.0 ± 9.9, and 24.2 ± 10.1 for the intervention group and 30.5 ± 9.3, 28.7 ± 9.4, and 28.3 ± 9.5 for the control group. The difference in %0BMI between the groups was statistically significant at three and six months. In both intervention and control groups, the children with a higher baseline %0BMI were more likely to have greater weight loss over time. There was a significant correlation between child %0BMI and parent BMI changes at six months.

This study demonstrates the benefits of concurrently targeting toddlers and parents for weight control and provides a model of an intervention that can be implemented in primary care setting. The authors stated it is not always easy to convince parents that their child needs weight management, but if the focus is shifted to the whole family then parents can model health lifestyle behaviours for their children.

Healthy Living Today!

Arauz Boudreau et al. reported on a pilot RCT of a family-centred primary care-based intervention for overweight or obese Latino children in a predominantly low-income community in the U.S. [376]. The trial involved 41 children aged 9–12 years with a BMI > the 85th percentile who were randomised to an intervention group (23 children) or a wait-list control group (18 children). The intervention consisted of six interactive group classes focussed on nutrition, physical activity and stress management, followed by monthly culturally-sensitive health coaching in-person or by telephone for six months. The coaching was aimed at empowering families to incorporate learned lifestyle changes and address the family and social barriers to making changes. The 1.5 hour classes were conducted in five consecutive weekly sessions at the health centre, with a sixth session three months later. Fourteen of the intervention group (61%), and 12 of the control group (67%) attended the first the second visit and so provided (some) pre- and post-intervention data.

Health-related quality of life, as measured by both child self-report and parent proxy using PedsQL™, improved in both groups but there was greater, though not significantly greater, improvement in the intervention children. Post-intervention, there were no differences between the intervention and control children for BMI, physical activity (as measured by accelerometers worn around the hip) or metabolic markers of obesity.

The authors noted that many caregivers cited factors outside their control as barriers to adopting healthy lifestyles such as the inability to find physical activities suitable for the whole family, inability to control what their child ate, children’s emerging independence, and social stressors such as family conflict, time pressures and financial stress. They also noted that all participants had low quality of life scores suggesting that obesity has a substantial effect on children’s quality of life although, given that the study was conducted in a low-income community, the effects of financial stress, racism and bias could not be discounted. They cited two possible reasons for the lack of statistically significant results: the small sample size and the possibility that families may require a more intensive intervention that includes scheduled coaching and/or changes to the environment.

Helping HAND

O’Connor et al. reported on a pilot RCT of Helping HAND (Healthy Activity and Nutrition Directions), an obesity intervention targeting five to eight year old ethnic minority children in primary care clinics in Houston, Texas [377]. The six-month intervention was delivered by trained allied health staff in the child’s community paediatric clinic. The 25 hours training for the five Health Advisors (HAs), three of whom were fluent in Spanish, covered the obesity intervention strategies recommended in the report of an Expert Committee of representatives from 15 national health care organisations [227], national recommendations for age-appropriate diet, physical activity and television viewing, authoritative parenting and effective behaviour-specific parenting strategies, patient-centred communication, and how to implement the helping HAND programme and worksheets. Each family was assigned an HA who met with the family once a month and encouraged them to self-select one behaviour to target from a menu of healthy behaviours which included: ‘Watch less TV’, ‘Be more active’, ‘Eat more fruit’, ‘Eat more vegetables’, ‘Eat healthy snacks’, ‘Drink less sweet drinks’, and ‘Drink more water’. Worksheets for children and parents were used to help with goal setting, making plans to reach the goal by the end of the month and making goal-specific behaviour changes. Parents and children signed the worksheets so they functioned as a behaviour change contract. Two weeks after the meeting the HA phone the family to assess progress and help solve any problems. At the next meeting families could chose to either continue working on the same goal for one more month or select a new behaviour to target.
The study randomised 40 families (parent-child dyads) to either the intervention or a waitlist control group. Eighty-two per cent of the families were Hispanic, 80% had girl, and 65% reported an income of US$30,000 or less. Eighty per cent of families attended four of more of the six sessions (a 20% attrition rate). During the six month study period, families selected an average of 4.75 (SD 1.75) behaviours to target and each of the seven target behaviours was chosen by between 45% and 80% of families. At the end of the intervention there were no differences between the intervention and the control group for child’s BMI z-score, dietary intake or physical activity but the intervention group watched less television (14.9 (SE 2.3) vs. 23.3 (SE 2.4) hours/week, p< 0.05). The authors concluded that Helping HAND was a feasible intervention for evaluation with a fully-powered RCT since it had a low attrition rate, appropriate content, overall participant satisfaction and was associated with improvements in some clinically relevant child and parenting behaviours.

**A lifestyle intervention for Mexican youth**

Diaz et al. conducted a 12-month RCT of lifestyle intervention in a primary care setting for obese Mexican youth [378]. The trial randomised 76 young people, aged 9–17 years with either a BMI >95th percentile or both BMI and waist circumference > 90th percentile, to either an intervention or a control group. Participants in the control group (n=22, mean age 11.7 years) attended 10–15 minute monthly consultations with a primary care physician who had received brief training on obesity. Participants in the intervention group (n=21, mean age 11.6 years) attended a family-centred programme consisting of 12 consecutive weekly two-hour group sessions at the clinic, led by a registered dietician (RD). They also had weekly consultations with the RD for the first 12 weeks and then monthly thereafter and monthly 10–15 minute consultations with a primary care physician. The curriculum for the group sessions had a behaviour modification focus. Initially the programme focussed mainly on children’s perceptions of susceptibility, severity, benefits, and barriers. The second part of the programme covered dealing with emotions, self-esteem, communication skills, information about body weight regulation, energy intake, nutrition, and physical activity, and the use of behaviour modification techniques. During the group sessions participants were encouraged to set their own goals for diet, physical activity and sedentary activity and these goals were revised and renewed at every session. There were six education sessions for parents, who were encouraged to lose weight if they were overweight.

Forty-three participants (57%) completed 12 months in the study. At 12 months, for those completing the study, mean changes in body weight were −0.8 kg (95% CI −3.2 to 1.5) in the intervention group and +5.6 kg (95% CI 3 to 8.2; p<0.001) in the control group. An intention-to-treat analysis confirmed significant differences in weight and BMI in favour of the intervention group: weight −3.5 kg, p<0.02; BMI −1.2 kg/m², p< 0.03.

The authors stated that theirs was the first long term study to show significant effects on obesity parameters in a primary care setting, although compared to other studies, it had a relatively high attrition rate (43%). They also stated that it is possible that only high-intensity interventions, such as their study, can produce changes in obesity parameters in our obesogenic environment and that cost-effectiveness analyses are needed to assess the utility of such interventions in primary care.

**Live, Eat and Play (LEAP)**

Two RCTs of a primary care intervention for overweight or mildly obese children have been conducted in Melbourne. The intervention was nested within a baseline cross-sectional BMI survey and known as Live, Eat and Play (LEAP). In the first trial, 163 overweight or mildly obese children (BMI z-score <3 ) aged 5– 9 years were randomised to either an intervention or control group [379]. Families in the control group were notified of their child’s weight status by letter. The intervention group received four standard GP consultations over 12 weeks, targeting changes in nutrition, physical activity and sedentary behaviour. Participants in the intervention group watched less television (14.9 (SE 2.3) vs. 23.3 (SE 2.4) hours/week, p< 0.05).

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The GPs delivering the intervention attended three evening group educational sessions. The core component of these sessions was training in brief solution-focussed therapy techniques and the sessions also included didactic and reflective teaching on childhood obesity. Prior to the child’s first GP appointment, the LEAP team provided the GP with the child’s personalised folder, BMI, and a two-page summary of parent responses to the baseline questionnaire relating to current nutrition, physical activity patterns and concern about their child’s weight status. During the four intervention consultations, GPs did not weigh or measure the child since the intervention was focussed on behavioural change rather than weight change. GPs recorded discussion content, contracts made and visit dates on a LEAP form in the child’s medical record.
Outcomes were measured at nine and 15 months. Attrition was 10%. The adjusted mean difference (intervention–control) in BMI was not significant at either follow-up time: −0.2 kg/m² (95% CI −0.6 to 0.1; p=0.25) at 9 months and −0.0 kg/m² (95% CI −0.5 to 0.5; p=1.00) at 15 months. There was a significant improvement in nutrition scores at both nine and 15 months, due to a reduction in consumption of high-fat milk and an increase in low-fat milk and water consumption. There was weak evidence of an improvement in physical activity. The authors concluded that this intervention did not result in sustained reductions in BMI, despite the parent-reported improved nutrition. They suggested two possible reasons: that brief individual solution-focused approaches may not be an effective method of dealing with child overweight or that the intervention might not have been intensive enough and the GP’s training insufficient. They stated that, based on this trial, they could not recommend that GPs adopt brief solution-focused behavioural strategies to deal with their overweight child patients.

The second LEAP trial, had the same enrolment criteria and intervention design as the first and randomised 258 children, 139 to either the intervention (139 children) or a control (119 children) group [380]. Outcomes were measured at six months and 12 months and attrition was 3.1% at six months and 6.2% at 12 months. The primary outcome was BMI and secondary outcomes were mean activity count/min by 7-day accelerometry, nutrition score from 4-day abbreviated food frequency diary, and child health related quality of life. Differences were adjusted for socioeconomic status, age, sex, and baseline BMI. Adjusted mean differences (intervention – control) at 6 and 12 months were, for BMI, −0.12 (95% CI −0.40 to 0.15, p=0.4) and −0.11 (−0.45 to 0.22, p=0.5); for physical activity in counts/min, 24 (−4 to 52, p=0.09) and 11 (−26 to 49, p=0.6); and, for nutrition score, 0.2 (−0.03 to 0.4, p=0.1) and 0.1 (−0.1 to 0.4, p=0.2). None of these differences were statistically significant. There was no evidence of harm to the children.

The authors concluded that, “primary care screening followed by brief counselling did not improve BMI, physical activity, or nutrition in overweight or mildly obese 5–10 year olds, and it would be very costly if universally implemented”. They noted that only a third of families with an eligible child chose to take up the intervention. This suggests that the majority of families are not concerned about their child’s weight status or have other priorities. The authors stated that health system resources for obesity interventions might be better spent divided between primary prevention at the population and community levels and improvement of treatment options for children with established obesity.

There were significant costs associated with the LEAP intervention for both the families and the health system [381]. A cost-consequence analysis indicated that the costs to the health system were AU$ 873 per intervention family and AU$ 64 per control family, a difference of AU$ 809 (p<0.001). These costs excluded the initial development cost of the LEAP intervention.

Key points from the reviews and recent RCTs of primary care interventions:

Overall, the reviews and individual RCTs of primary care obesity interventions suggested that:

- There is no evidence that brief interventions in primary care are effective
- The few effective interventions that have been carried out in primary care settings have largely replicated the care model of a specialist obesity clinic and offered both a series of group sessions for parents and children (usually separately) and multiple individual consultations over an extended period, either in person or by phone

Conclusions

There is considerable evidence indicating that childhood obesity has its origins very early in life, even before birth. It therefore seems that childhood obesity is best tackled early but there are a number of difficulties. Parents need to recognise that their overweight or obese pre-schooler has a problem about which something needs to be done. Evidence suggests that parents are not very good at recognising that their young child has a weight problem. Even if they recognise the problem, or it is pointed out to them by a health professional, they may feel that their child will grow out of it, that denying their child treats that everyone else gets is just too hard, or that there are other more pressing problems in their life. There is very little evidence regarding effective obesity interventions for pre-schoolers although two recently published trials of relatively high intensity interventions have shown promising results [333,375].

Given the high proportion of children who are currently overweight or obese there is no way that all of them can be treated by specialist paediatric services. There is a clear need
for effective low-cost interventions that can be delivered in primary care. Unfortunately, there is little current evidence that such interventions exist. Most of the research has been conducted in specialist clinics. The interventions that are effective tend to be resource intensive, involving at least 25 contact hours, and, although they may result in significant reductions in excess weight (i.e. significant reductions in BMI percentile or z-score), they do not usually make an obese child into a child of normal weight. Effective interventions include attention to diet and physical activity and also behavioural components aimed at changing thinking patterns regarding diet and physical activity, goal setting, and improving self-esteem. There is currently insufficient evidence to indicate which particular dietary, physical activity or behavioural interventions are the best. Effective interventions also usually involve addressing parents’ overweight or obesity, since the likelihood of a child successfully losing excess weight is improved if the whole family adopts a healthier lifestyle.

The few small studies that have demonstrated good results in primary care settings have used a similar treatment model to those used in specialist clinics and offered both a series of group sessions for parents and children (usually separately) and multiple individual consultations over an extended period, either in person or by phone.

It seems likely that the health system cannot afford high intensity interventions for any but the most severely obese children. Most obesity experts believe that dealing with the obesity epidemic requires a whole of society approach to prevention. Cultural change is required to make healthy lifestyles the norm, but there are powerful commercial interests behind the provision of cheap but unhealthy food. It is unrealistic to expect that the health system can solve the problem of childhood obesity on its own.
Breastfeeding and Solids

Introduction

The Ministry of Health recommends that babies be exclusively breastfed until they are ready for and need extra food at around six months of age [2]. This is because breastfeeding confers considerable health benefits for both baby and mother. Breastfed babies have lower rates of common childhood infections such as diarrhoea, respiratory infections and otitis media, and lower rates of sudden infant death syndrome [382]. Mothers who breastfeed have lower rates of post-partum haemorrhage, breast cancer and ovarian cancer, lose their extra pregnancy weight faster, and are less likely to become pregnant soon after their baby’s birth [382,383,384].

Research suggests that formula feeding may be associated with higher risks for major chronic conditions such as type 2 diabetes, asthma and obesity, all of which are becoming more common [385]. Introducing solid foods prior to four months of age may increase the risk of conditions such as eczema, asthma, food allergies and gastroenteritis [2]. Introducing solid food after six months, however, may be associated with an increased risk of iron deficiency and malnutrition [2].

In New Zealand all lead maternity carers are required to promote breastfeeding [386]. Yet, while NZ Breastfeeding Authority data suggests that New Zealand has a relatively high rate of breastfeeding initiation (in 2005, 80.5% of infants born in Baby Friendly Hospitals were exclusively breastfed on discharge [384]), there is a significant decline in the prevalence of exclusive breastfeeding through the first six months of life [384]. While the decision to breastfeed or not is a personal one, and mothers should not be made to feel guilty if they are unable to, or choose not to breastfeed, the success rate among mothers who wish to breastfeed can be improved if there is active support from families, friends, communities, clinicians, health care leaders, employers and policymakers [382].

The following section reviews breastfeeding rates at <6 weeks, 3 months and 6 months using Plunket data. The proportion of babies who were given solid food prior to four months of age is also reviewed, using data from the 2011/12 NZ Health Survey.

Data Sources and Methods

Indicator

1. Exclusive/full breastfeeding rates in Plunket babies at <6 weeks, 3 months and 6 months of age

Data Source

Plunket Client Information System

Numerator: The number of Plunket babies exclusively/fully breastfed at <6 weeks (range: 2 weeks to 5 weeks, 6 days), 3 months (range: 10 weeks to 15 weeks, 6 days) 6 months (range: 16 weeks to 7 months, 4 weeks)

Denominator: The number of babies in contact with Plunket at these ages

Notes on Interpretation

Note 1: Plunket currently enrol more than 88% of the new baby population, although Māori and Pacific mothers may be under-reported in these samples. Plunket have breastfeeding data dating back to 1922, with more detailed information being available in recent years.

Note 2: Plunket’s breastfeeding definitions, which are similar to the World Health Organization are:

Exclusive Breastfeeding: The infant has never had any water, formula or other liquid or solid food. Only breast milk, from the breast or expressed, and prescribed medicines have been given from birth.

Fully Breastfed: The infant has taken breast milk only and no other liquids or solids except a minimal amount of water or prescribed medicines, in the past 48 hours.

Partially Breastfed: The infant has had some breast milk and some infant formula or other solid food in the past 48 hours.

Artificially Fed: The infant has had no breast milk, but has had an alternative liquid such as infant formula, with or without solid food in the past 48 hours.
Exclusive/Full Breastfeeding Rates in Plunket Babies

New Zealand Distribution and Trends

New Zealand Trends by Age

In New Zealand during the years ending June 2006–2012, the proportion of Plunket babies who were exclusively or fully breastfed remained relatively static. Exclusive/full breastfeeding rates in the year ending June 2012 were 66.1% at <6 weeks, 54.6% at 3 months and 24.9% at 6 months of age (Figure 73).

Figure 73. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age, New Zealand, Years Ending June 2006–2012

Source: Plunket Client Information System

New Zealand Trends by Ethnicity

In New Zealand during the years ending June 2006–2012, exclusive/full breastfeeding rates at <6 weeks of age were consistently higher for European babies than for babies of other ethnic groups. At 3 and 6 months of age however, exclusive/full breastfeeding rates were generally higher for European > Asian > Māori and Pacific babies, with differences between Asian and European babies decreasing as the period progressed (Figure 74).

New Zealand Distribution by NZ Deprivation Index Decile

In New Zealand during the year ending June 2012, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were lower for babies from the most deprived (NZDep decile 10) areas, than for babies from average or less deprived areas (Figure 75).
Breastfeeding and Solids

Figure 74. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age and Ethnicity, New Zealand, Years Ending June 2006–2012

Source: Plunket Client Information System

Figure 75. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age and NZ Deprivation Index Decile, New Zealand, Year Ending June 2012

Source: Plunket Client Information System
Northern DHBs Distribution and Trends

Northern DHBs vs. New Zealand

In Northland and the Waitemata DHB during the years ending June 2006–2012, exclusive/full breastfeeding rates at <6 weeks and 3 months were generally higher than the New Zealand rate, while rates at 6 months were similar. In Counties Manukau however, breastfeeding rates were much lower than the New Zealand rate at all three ages. In Auckland DHB, breastfeeding rates at 3 months were higher than the New Zealand rate, although at <6 weeks rates were more similar (Figure 76).

Northern DHBs Distribution by Ethnicity

In Northland during the years ending June 2006–2012, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were higher for European babies than for Māori babies. In the Waitemata, Auckland and Counties Manukau DHBs, breastfeeding rates at <6 weeks were much higher for European babies than for babies of other ethnic groups. In the Waitemata and Auckland DHBs at 3 and 6 months however, breastfeeding rates were generally higher for European > Asian > Māori and Pacific babies (Figure 77, Figure 78).

Northern DHBs Distribution by NZDep Decile

In all four Northern DHBs during the year ending June 2012, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were lower for babies living in the most deprived (NZDep decile 10) areas, than for babies living in the least deprived (NZDep decile 1) areas. Rates for those living in average (NZDep decile 5) areas however were more variable (Figure 79).
Figure 76. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age, Northern DHBs vs. New Zealand, Years Ending June 2006–2012

Source: Plunket Client Information System
Figure 77. Proportion of Plunket Babies who were Exclusively or Fully Breastfed at <6 Weeks by Ethnicity, Northern DHBs vs. New Zealand, Years Ending June 2006–2012

Source: Plunket Client Information System
Figure 78. Proportion of Plunket Babies who were Exclusively or Fully Breastfed at 3 Months and 6 Months by Ethnicity, Northern DHBs vs. New Zealand, Years Ending June 2006–2012

Source: Plunket Client Information System
Figure 79. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age and NZ Deprivation Index Decile, Northern DHBs, Year Ending June 2012

Source: Plunket Client Information System
Babies Given Solid Food Before Four Months of Age

In the 2011/12 NZ Health Survey [2], the parents of children aged under five years were asked at what age their child was first given solid food. Information was collected in a similar way to the 2006/07 NZ Health Survey, making it possible to compare changes over time. The following section thus briefly reviews changes in the proportion of children given solid food prior to four months of age between the 2006/07 and 2011/12 NZ Health Surveys, before exploring the distribution of early solids by a range of socio-demographic factors, in the most recent 2011/12 NZHS.

### Data Sources and Methods

<table>
<thead>
<tr>
<th>Indicator</th>
<th>The proportion of children aged 4 months to 4 years who were given solid food before four months of age</th>
</tr>
</thead>
</table>

**Data Source**

The 2011/12 New Zealand Health Survey (NZHS)


**Notes on Interpretation**

The 2011/12 NZ Health Survey [2] was a cross sectional survey carried out between July 2011 and August 2012, which collected information on 4,478 children aged from birth to 14 years. In this survey the parents of children aged less than five years (four months to four years) were asked at what age their child was first given solid food. Further detail on the 2011/12 NZ Health Survey is available in the Data Sources and Methods section of the Overweight and Obesity Section commencing on page 287.

Differences between estimates are said to be statistically significant when the confidence intervals for each rate do not overlap. Sometimes, however, even when there are overlapping confidence intervals, the difference between the groups can be statistically significant. Any differences between two variables where the confidence intervals overlap are tested using a t-test. The significance of a t-test is represented by the p-value. If a p-value is below 0.05, then we are 95 percent confident the difference between the two estimates is statistically significant [248].

### Trends in Giving Solids Before Four Months

#### Trends by Gender and Ethnicity

**Overall:** The proportion of children aged 4 months to 4 years given solid food before four months of age decreased *significantly* (p=0.00) between NZ Health Surveys, with rates falling from 15.8% (95% CI 13.7–18.1) in 2006/07, to 9.5% (95% CI 7.9–11.4) in 2011/12.

**By Gender:** When broken down by gender, the proportion of boys aged 4 months to 4 years given solid food before four months declined *significantly* (p=0.01), from 15.4% (95% CI 12.4–18.9) in 2006/07 to 10.2% (95% CI 8.0–12.9) in 2011/12. Rates for girls also declined *significantly* (p=0.00) from 16.2% (95% CI 13.3–19.5) in 2006/07 to 8.8% (95% CI 6.4–11.7) in 2011/12 (**Figure 80**). Once adjusted for age, there were no *significant* gender differences in the proportion of babies given solid foods before four months of age in the 2011/12 NZHS.

**By Ethnicity:** When broken down by ethnicity, the proportion of Māori children aged 4 months to 4 years given solid food before four months declined *significantly* (p=0.04), from 21.7% (95% CI 17.8–26.0) in 2006/07 to 15.6% (95% CI 11.7–20.2) in 2011/12. Rates for Asian children fell (p=0.00) from 9.5% (95% CI 6.7–12.9) in 2006/07 to 2.9% (95% CI 1.0–6.4) in 2011/12, while rates for European/Other children fell (p=0.00) from 14.4% (95% CI 11.9 –17.1) in 2006/07 to 8.1% (95% CI 6.2–10.3) in 2011/12. While rates for Pacific children also fell, from 20.7% (95% CI 14.7–27.9) in 2006/07 to 14.3% (95% CI 8.8–21.5) in 2011/12, the differences did not reach statistical significance (p=0.15) (**Figure 80**).

#### Distribution by Region

When broken down by region, the proportion of children aged 4 months to 4 years in the Northern region given solid food before four months of age decreased *significantly* (p=0.00) between NZ Health Surveys, with rates falling from 14.1% (95% CI 10.9–17.8) in 2006/07, to 7.5% (95% CI 4.9–10.9) in 2011/12. Rates in the Central region also fell *significantly* (p=0.02), from 16.8% (95% CI 11.9–22.6) in 2006/07 to 9.0% (95% CI 5.6–
13.5) in 2011/12. While rates in the Midland (p=0.05) and Southern (p=0.10) regions also fell, differences for these regions did not reach statistical significance (Figure 81).

Figure 80. Proportion of Babies and Children Aged 4 Months to 4 Years Who Were Given Solid Food Before Four Months of Age by Gender and Ethnicity, 2006/07 and 2011/12 New Zealand Health Surveys

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age

Figure 81. Proportion of Babies and Children Aged 4 Months to 4 Years Who Were Given Solid Food Before Four Months of Age by Region, 2006/07 and 2011/12 New Zealand Health Surveys

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age
Current Distribution of Giving Solids Before Four Months

Distribution by Ethnicity and NZ Deprivation Index Decile

In the 2011/12 NZHS, Māori children aged 4 months–4 years were 2.23 (95% CI 1.56–3.19) times more likely to be given solid food before four months of age than non-Māori children, while Pacific children were 1.67 (95% CI 1.09–2.56) times more likely to be given solid food before four months than non-Pacific children, once rates were adjusted for age and gender. In contrast, Asian children were significantly less likely to be given solid food before four months (aRR 0.28 (95% CI 0.11–0.68)) than non-Asian children. There were however, no significant differences in the proportion of children from the most and least deprived NZDep06 areas being given solid food before four months, once rates were adjusted for age, sex and ethnic group (Figure 82).

Figure 82. Proportion of Babies and Children Aged 4 Months to 4 Years Who Were Given Solid Food Before Four Months of Age by Gender, Ethnicity and NZ Deprivation Index Decile, 2011/12 New Zealand Health Survey

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age
Local Policy Documents and Evidence-Based Reviews Relevant to Breastfeeding and Infant Nutrition

In New Zealand there are a range of policy documents and reviews relevant to breastfeeding and infant nutrition and these are briefly summarised in Table 115, along with a number of evidence-based reviews which consider these issues in the overseas context.

Table 115. Local Policy Documents and Evidence-Based Reviews Relevant to Breastfeeding and Infant Nutrition

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>This publication (the Plan) contains the advice of the National Breastfeeding Committee to the Director General of Health. The Plan recognises that the influences on breastfeeding rates are complex and that cultural change is required to improve breastfeeding rates. While the health sector has the leading role in the protection, promotion and support if breastfeeding all sectors of society need to be involved. The Plan proposes objectives to describe what needs to be done and a list of desired outcomes in each of the following settings: government, family and community, health services, and workplaces, childcare and early childhood education.</td>
</tr>
<tr>
<td>The purpose of this paper is to provide up to date policy advice and information on nutrition and physical activity for infants and toddlers to be used: as a basis for education programmes to support families and children, to guide and support health practitioners in their work, to provide a basis for preparing policies on the protection, promotion and support of breastfeeding and to identify inequalities so that education and support can be targeted at reducing inequalities related to nutrition and physical activity. Chapter 3 includes concise but comprehensive guidelines on breastfeeding. Exclusive breastfeeding is recommended until an infant is six months of age. Chapters 7 and 8 relate specifically to Māori, Pacific and Asian infants and toddlers. Some sections have been partially revised because of changes in policy. Updated recommendations include those to decrease the risk of food-related choking in babies and young children, and minor changes to the information on infant formula.</td>
</tr>
<tr>
<td>This document provides a New Zealand Interpretation of International Code of Marketing of Breast-Milk Substitutes (WHO 1981) and subsequent relevant World Health Assembly resolutions (to which NZ is a signatory). It includes the Code of Practice for Health Workers in New Zealand and the Code of Practice for the Marketing of Infant Formula.</td>
</tr>
<tr>
<td>This literature review was commissioned by the National Breastfeeding Advisory Committee to inform the development of the National Strategic Plan of Action for Breastfeeding. It covers the context and history of breastfeeding in New Zealand, the local and global legislative and policy context for breastfeeding, and social and clinical issues influencing breastfeeding in New Zealand. It includes a literature review of the evidence for interventions supporting breastfeeding and concludes with a concise summary of common interventions undertaken both in New Zealand and internationally. It briefly assesses the quality of these interventions according to the evidence from the reviewed literature. Interventions of proven effectiveness are listed as follows:</td>
</tr>
<tr>
<td>• Training health professionals in the psycho-social and physiological elements of breastfeeding and lactation management</td>
</tr>
<tr>
<td>• Accreditation to the Baby Friendly Hospital Initiative and implementation of the 10 Steps to successful breastfeeding, particularly: kangaroo care, training of staff, early initiation of breastfeeding, the promotion of exclusive breastfeeding and limitation of any form of supplementation, and on-demand breastfeeding;</td>
</tr>
<tr>
<td>• Skilled peer support provided by well-trained and knowledgeable peers;</td>
</tr>
<tr>
<td>• Home visitation as a service delivery mechanism;</td>
</tr>
<tr>
<td>• The provision of adequate workplace facilities in which to express breast milk or to breastfeed; and</td>
</tr>
<tr>
<td>• Childcare that is supportive of breastfeeding.</td>
</tr>
<tr>
<td>Promising interventions identified include prenatal education, biological nurturing approaches, social marketing, support for fathers, family/whānau and friends, and developing breastfeeding friendly business and public spaces.</td>
</tr>
</tbody>
</table>
This background paper (revised in 2008 and updated in 2009) provides evidence-based policy advice on the nutrition, physical activity, lifestyle and environmental determinants for achieving and maintaining the best possible health for healthy pregnant and breastfeeding women, and the best possible pregnancy outcome. It provides information that can be used as a basis for programmes and education to support healthy pregnant and breastfeeding women and guidance for all health practitioners involved in their care. It also aims to identify health inequalities relating to nutrition and physical activity so that education and support for healthy pregnant and breastfeeding women can be targeted to reduce health inequalities between population groups, and includes sections that focus on the needs of Māori and Pacific women. It includes food and nutrition guidelines for pregnant and breastfeeding women, a review of energy intakes and weight changes in pregnant and breastfeeding women, and infant birthweight and the determinants of infant birthweight, and recommended sources and dietary intakes of nutrients.

### International Guidelines


These guidelines provide recommendations for safe and appropriate use of pharmacologic agents for anaesthesia and pain relief in breastfeeding women during labour and postpartum and for lactating women during surgery, and examine the evidence currently available for various approaches to labour pain management on breastfeeding outcomes. They make recommendations for prudent practice.


These evidence-based guidelines for health professionals, managers, commissioners and all those involved in pre-school childcare are aimed at pregnant women and those who are planning to become pregnant, mothers and other carers of children aged under 5 and their children, particularly aimed at those on a low income or from a disadvantaged group. The report includes key priorities and recommendations and a review of the evidence base. Recommendations include: to provide women with information and advice on the benefits of taking a vitamin D supplement during pregnancy and while breastfeeding; to provide ‘Healthy Start’ vitamin supplements (folic acid and vitamin C and D) for eligible pregnant women; to implement a structured programme to encourage breastfeeding, including training for health professionals, within NHS organisations; to encourage breastfeeding by providing information, practical advice and ongoing support, including the help of breastfeeding peer supporters; and once infants are aged six months, to assist parents and carers to progressively introduce them to a variety of nutritious foods, in addition to milk.


These guideline’s objectives include: To promote, support, and sustain breastfeeding in the late preterm infant; to maintain optimal health of the infant and mother; to allow the late preterm infant to breastfeed and/or breastmilk feed to the greatest extent possible; to heighten awareness of difficulties that late preterm infants and their mothers may experience with breastfeeding; to offer strategies to anticipate, identify promptly, and manage breastfeeding problems that the late preterm infant and mother may experience in the inpatient and outpatient settings; to prevent medical problems such as dehydration, hypoglycemia, hyperbilirubinemia, and failure to thrive in the late preterm infant; to maintain awareness of mothers’ needs, understanding of current plans, and ability to cope.


This updated guideline targets all pregnant women, new mothers, and their support people to promote a philosophy and practice of maternal-infant care that advocates breastfeeding; to support the normal physiologic functions involved in the establishment of this process; and to assist families choosing to breastfeed with initiating and developing a successful and satisfying experience. The document outlines a model breastfeeding policy for institutions based on recommendations from the most recent breastfeeding policy statements published by the Office on Women's Health of the U.S. Department of Health and Human Services, the American Academy of Pediatrics, the American College of Obstetricians and Gynecologists, the American Academy of Family Physicians, the World Health Organization, the Academy of Breastfeeding Medicine, and the UNICEF/WHO evidence-based “Ten Steps to Successful Breastfeeding.”


This guideline provides information on safe human milk storage in the home for full-term infants for breastfeeding mothers and family members and caregivers. It outlines preparation, storage and use of stored human milk.


This guideline offers best practice advice on the operation of donor breast milk bank services and protecting the safety of donor milk. The guideline gives recommendations on: quality assurance; recruiting donors; screening and selecting donors; serological testing; consent and continued eligibility; stopping or suspending milk donations; handling donor milk at home, during transportation and at the milk bank; and tracking and tracing.
This systematic review assessed the effectiveness of support for breastfeeding mothers and included randomised or quasi-randomised controlled trials comparing extra support for healthy breastfeeding mothers of healthy term babies with usual maternity care. Of the 67 studies that were eligible for inclusion, 52 contributed outcome data to the review (56,451 mother-infant pairs). All forms of extra support analysed together showed an increase in duration of any breastfeeding (includes partial and exclusive breastfeeding) (risk ratio (RR) for stopping any breastfeeding before six months 0.91, 95% confidence interval (CI) 0.88 to 0.96). All forms of extra support together also had a positive effect on duration of exclusive breastfeeding (RR at six months 0.86, 95% CI 0.82 to 0.91; RR at four to six weeks 0.74, 95% CI 0.61 to 0.89). Extra support by both lay and professionals had a positive impact on breastfeeding outcomes. Strategies that rely mainly on face-to-face support are more likely to succeed. Support that is only offered reactively, in which women are expected to initiate the contact, is unlikely to be effective; women should be offered ongoing visits on a scheduled basis so they can predict that support will be available. Support should be tailored to the needs of the setting and the population group.


This systematic review assessed the effects on child health, growth, and development, and on maternal health, of exclusive breastfeeding for six months versus exclusive breastfeeding for three to four months with mixed breastfeeding (introduction of complementary liquid or solid foods with continued breastfeeding) thereafter through six months. All internally-controlled clinical trials and observational studies were selected and studies were stratified according to study design (controlled trials versus observational studies), provenance (developing versus developed countries), and timing of compared feeding groups (three to seven months versus later). Twenty-three independent studies meeting the selection criteria were identified: 11 from developing countries (two of which were controlled trials in Honduras) and 12 from developed countries (all observational studies). Infants who are exclusively breastfed for six months experience less morbidity from gastrointestinal infection than those who are partially breastfed as of three or four months, and no deficits have been demonstrated in growth among infants from either developing or developed countries who are exclusively breastfed for six months or longer. Moreover, the mothers of such infants have more prolonged lactational amenorrhea. Although infants should still be managed individually so that insufficient growth or other adverse outcomes are not ignored and appropriate interventions are provided, the available evidence demonstrates no apparent risks in recommending, exclusive breastfeeding for the first six months in developing and developed-countries.

Abdulwadud OA & Snow EM. 2012. Interventions in the workplace to support breastfeeding for women in employment. Cochrane Database of Systematic Reviews(10).

This systematic review assessed the effectiveness of workplace interventions to support and promote breastfeeding among women returning to paid work after child birth, and its impact on process outcomes pertinent to employees and employers. No trials have evaluated the effectiveness of workplace interventions in promoting breastfeeding among women returning to paid work after the birth of their child. The impact of such intervention on process outcomes is also unknown. Randomised controlled trials are required to establish the benefits of various types of workplace interventions to support, encourage and promote breastfeeding among working mothers.


This review assessed the effect of unrestricted versus restricted pacifier use in healthy full-term newborns whose mothers have initiated breastfeeding and intend to exclusively breastfeed, on the duration of breastfeeding, other breastfeeding outcomes and infant health. Three trials (involving 1915 babies) were identified but only two trials were included (involving 1302 healthy full-term breastfeeding infants) in the analysis. Pacifier use in healthy term breastfeeding infants, started from birth or after lactation is established, did not significantly affect the prevalence or duration of exclusive and partial breastfeeding up to four months of age.


This document provides evidence-based guidelines for the evaluation and management of the drug-dependent woman choosing to breastfeed. Infants of drug-dependent women, at risk for multiple health and developmental difficulties, stand to benefit substantially from breastfeeding and human milk, as do their mothers. An antenatal plan preparing the mother for parenting, breastfeeding, and postpartum substance abuse treatment should be formulated for each woman.


This guideline evaluates the state of evidence on the prevention, recognition, and management of breast engorgement to encourage successful breastfeeding.

Cochrane Systematic Reviews


Infected mastitis is commonly caused by *Staphylococcus aureus*. The prevalence of mastitis in breastfeeding women may reach 33%. Effective milk removal, pain medication and antibiotic therapy have been the mainstays of treatment. This review examines the effectiveness of antibiotic therapies in relieving symptoms for breastfeeding women with mastitis with or without laboratory investigation. Two randomised controlled trials met the inclusion criteria but the numbers were small and the quality poor in one of the studies. There is insufficient evidence to confirm or refute the effectiveness of antibiotic therapy for the treatment of lactational mastitis.


This review assessed the effectiveness of support for breastfeeding mothers and included randomised or quasi-randomised controlled trials comparing extra support for healthy breastfeeding mothers of healthy term babies with usual maternity care. Of the 67 studies that were eligible for inclusion, 52 contributed outcome data to the review (56,451 mother-infant pairs). All forms of extra support analysed together showed an increase in duration of any breastfeeding (includes partial and exclusive breastfeeding) (risk ratio (RR) for stopping any breastfeeding before six months 0.91, 95% confidence interval (CI) 0.88 to 0.96). All forms of extra support together also had a positive effect on duration of exclusive breastfeeding (RR at six months 0.86, 95% CI 0.82 to 0.91; RR at four to six weeks 0.74, 95% CI 0.61 to 0.89). Extra support by both lay and professionals had a positive impact on breastfeeding outcomes. Strategies that rely mainly on face-to-face support are more likely to succeed. Support that is only offered reactively, in which women are expected to initiate the contact, is unlikely to be effective; women should be offered ongoing visits on a scheduled basis so they can predict that support will be available. Support should be tailored to the needs of the setting and the population group.


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This review assessed the effect of unrestricted versus restricted pacifier use in healthy full-term newborns whose mothers have initiated breastfeeding and intend to exclusively breastfeed, on the duration of breastfeeding, other breastfeeding outcomes and infant health. Three trials (involving 1915 babies) were identified but only two trials were included (involving 1302 healthy full-term breastfeeding infants) in the analysis. Pacifier use in healthy term breastfeeding infants, started from birth or after lactation is established, did not significantly affect the prevalence or duration of exclusive and partial breastfeeding up to four months of age.
Early skin-to-skin contact (SSC) involves placing the naked baby (ideally soon after birth) prone on the mother’s bare chest and covered across the back with a blanket. It is thought that this elicits innate mammalian behaviours from both the mother and the neonate and promotes the release of maternal oxytocin which increases maternal skin temperature (thus warming the neonate) and also decreases maternal anxiety and enhances mother-infant bonding and the likelihood of spontaneous breastfeeding. This review included thirty studies (1925 mother-infant dyads) which were either RCTs of quasi-RCTs comparing early SSC with usual hospital care however only 8 out of 64 outcome measures had data from more than two of the trials which limited the possibilities for meta-analysis. SCC had statistically significant positive effects on breastfeeding at one to four months post birth (10 trials; 552 participants) (odds ratio (OR) 1.82, 95% CI 1.08 to 3.07), and breastfeeding duration (seven trials; 324 participants) (weighted mean difference (WMD) 42.55, 95% CI -1.69 to 86.79). There were trends found for improved summary scores with early SCC for maternal affectionate love/touch during observed breastfeeding (four trials; 314 participants) (standardized mean difference (SMD) 0.52, 95% CI 0.07 to 0.98) and maternal attachment behaviour (six trials; 396 participants) (SMD 0.52, 95% CI 0.31 to 0.72). One trial (44 participants) found that SCC infants cried for a shorter length of time (WMD -8.01, 95% CI -8.98 to -7.04). Late preterm infants with early SCC had better cardio-respiratory stability (one trial; 35 participants) (WMD 2.88, 95% CI 0.53 to 5.23). No adverse effects from SCC were found. The review authors concluded “Based on the available evidence, SSC appears to have some clinical benefit, especially for breastfeeding and for temperature and cardio-respiratory stability in late preterm infants”.

Systematic and Other Reviews From the International Literature


There is increasing attention to the topic of prevention and continued debate as to whether breastfeeding (BF) is protective against childhood obesity. Previous systematic reviews on this topic were done in 2005 showing that BF was protective against childhood obesity but, because of confounding variables, the evidence was weak. The majority of studies identified in this article showed a relationship between BF and obesity prevention, but because of confounding maternal, child, cultural, genetic, and environmental variables, the relationship remains unclear.


This article reviews how hospital newborn services can be constructed or restructured to support the breastfeeding mother-infant dyad so that they can achieve high levels of breastfeeding success. These include: Patient education and preparation: skin-to-skin contact and early, unrestricted breastfeeding; The Baby-Friendly Hospital Initiative’s “Ten Steps”; and support of breastfeeding through common medical problems in the newborn period such as hypoglycemia and hyperbilirubinemia, and in the late preterm infant. Important positive and negative factors from the prenatal period, and the preparation for hospital discharge are also discussed.


This review explores common personal and societal barriers to exclusive breastfeeding and offers evidence-based strategies to support mothers to breastfeed exclusively, such as ensuring antenatal education, supportive maternity practices, timely follow-up, and management of lactation challenges. The article also addresses common reasons nursing mothers discontinue exclusive breastfeeding, including the perception of insufficient milk, misinterpretation of infant crying, returning to work or school, early introduction of solid foods, and lack of support.


This review, mainly focused on the USA, discusses trends in breastfeeding, influences on the reacceptance of a breastfeeding norm, and breastfeeding as a social and public health issue. The goal is to create an enabling environment for optimal breastfeeding in health care and social norms, and to adjust the social and political realities to support an economic milieu that favours breastfeeding.


This review of 38 papers focuses on strategies to support breastfeeding. The findings included collaboration with community and family members; confidence building; appropriate ratio of staffing levels; development of communication skills; and ‘closing the gap’ in inequalities in health. Mothers benefit from strategies that encourage breastfeeding, with guidance that supports self-efficacy and feelings of being capable and empowered, and is tailored to individual needs.


The systematic review describes peer support interventions supporting breastfeeding during pregnancy and the postnatal period. It included studies from Europe, North America, Australia and New Zealand. During pregnancy, hospitalisation and the postnatal period, individual support and education were used most commonly. Peer support was strongly associated with the postnatal period. The combination of professional support and peer support by trained and experienced peer supporters was effective in ensuring the continuation of breastfeeding.
Breastfeeding has many health benefits for women and their babies, but particularly if the woman is obese and/or had a pregnancy affected with gestational diabetes mellitus (GDM). Women who have had GDM are at high risk for developing metabolic syndrome or type 2 diabetes, and their offspring are at greater risk for these metabolic disorders both in childhood and later in adulthood. There is considerable evidence that breastfeeding may attenuate these risks. This article presents the most recent evidence on what is known about how breastfeeding can mitigate the adverse metabolic effects of obesity and GDM on both mother and child, and describes best practices that can support and sustain breastfeeding, particularly in racial/ethnic communities at risk.

### References


This document does not represent NICE guidance but is the culmination of work commissioned by the former Health Development Agency (whose functions were transferred to the National Institute for Clinical Excellence when it became the National Institute for Health and Clinical Excellence). It sets out a series of evidence-based actions for promoting both the initiation and the continuation of breastfeeding, particularly among population groups where breastfeeding rates are low. These were developed from a list of interventions for which there is international research evidence of effectiveness which became a list of "what will really work in practice in England". The evidence-based actions are:

- Baby Friendly Initiative (BFI) in the maternity and community services
- Education and/or support programmes
- Changing policy and practice within community and hospital settings in order to support effective positioning and attachment, encourage baby-led feeding, and encourage women with “insufficient milk” through supportive care, teaching technique, providing sound information and reassurance.
- Abandoning the following policies in hospitals and the community: restricting timing and/or frequency of breastfeeding in immediate post-natal care, restricting mother-baby contact from birth onwards, routine or medically unjustified supplementary feeding, separating babies from mothers for the treatment of jaundice, and the provision of hospital discharge packs containing promotional material for formula.
- Complementary telephone peer or volunteer support
- Education and support from one professional (targeted particularly to low income women)
- Education and support throughout the first year
- Media programmes targeting teenagers to improve attitudes towards breastfeeding

The briefing is largely based on one evidence briefing and three systematic reviews but appendix B provides brief details of the individual studies relating to each of the effective and the harmful interventions and the full list of references is contained in appendix D.

The publications on which this briefing is largely based are:


Under section 69Y of the Employment Relations Act 2000, since 1 April 2009 employers have been required, as far as it is reasonable and practicable, to provide appropriate breaks and facilities for employees who wish to breastfeed their infants or express milk during working hours. This publication provides guidance for employers on this issue.

### The 2008 Infant Feeding Amendment to the Employment Relations Act 2000


The purpose of this Act includes: (i) require facilities and breaks to be provided, so far as is reasonable and practicable in the circumstances, for employees who wish to breastfeed in the workplace or during work periods; and (ii) require employees to be provided with rest breaks and meal breaks. Breastfeeding breaks are in addition to breaks an employee is already entitled to, however, if an employee and employer agree, the same break may be taken for both purposes. Section 69Y of the Act (relating to breastfeeding facilities and breaks in the workplace) came into force on 1 April 2009.


The Ministry of Health has contracted the New Zealand Breastfeeding Authority Board (NZBA) to develop and manage the Baby Friendly Hospital Initiative (BFHI) which is a global effort launched by the WHO and UNICEF in 1991 to implement practices that protect, promote and support breastfeeding. The New Zealand BFHI documents have been developed from the WHO BFHI documents (see below) to reflect the unique circumstances of New Zealand’s health system and acknowledge the Treaty of Waitangi principles of protection, partnership and participation. The format of the documents differs somewhat from that of the WHO documents. The New Zealand BFHI documents, which can be found on the NZBA website (select BFHI resources from the drop down list under the resources tab) are:

- Forward
- Part 1: Background and Baby Friendly Implementation in New Zealand
- Part 2: The NZBA Criteria for BFHI
- Part 3: Self-Appraisal Questionnaire
- Part 4: BFHI Assessment Manual
- Part 5: BFHI Assessment Summary
- Part 6: Resources for Aotearoa New Zealand
- Part 7: BFHI Annual Self-Appraisal Questionnaire


The Ministry of Health has contracted the New Zealand Breastfeeding Authority Board (NZBA) to facilitate the implementation of the Baby Friendly Community Initiative (BFCI) in health services in the community. The BFCI consists of a seven point plan for the protection, promotion and support of breastfeeding in the community (details of which can be found on the website) in order to achieve three objectives: to increase the proportion of babies who are breastfed, to increase the duration of exclusive breastfeeding, and to sustain breastfeeding beyond six months alongside feeding with appropriate, adequate and safe complementary foods. The BFCI also included standards of care for the non-breastfeeding mother and her baby.


These documents are revisions of the original 1992 BFHI guidelines and the first four of the five sections of the revised BFHI package are available on the website. The five sections are 1. Background and Implementation, 2. Strengthening and Sustaining the BFHI: A course for decision-makers, 3. Breastfeeding Promotion and Support in a Baby-friendly Hospital: a 20-hour course for maternity staff, 4. Hospital Self-Appraisal and Monitoring, and 5. External Assessment and Reassessment. Section 5 is for limited distribution only to external assessors.

### Webpage from Equal Employment Opportunities Trust, Women’s Health Action on implementing breastfeeding support in the workplace.

http://www.eeotrust.org.nz/content/docs/breastfeeding_sheets.pdf

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
OTHER NUTRITIONAL INDICATORS

Introduction

Optimal nutrition during childhood is vital for good health, growth and development, and the prevention of obesity [387]. Children’s dietary patterns and food choices are strongly influenced by their parents and caregivers. For example, parental modelling is associated with healthy food intake, and family/whānau meals taken together are associated with a higher intake of fruit and vegetables, highlighting the importance of involving parents in efforts to improve children’s diets[388,389,390]. Children’s diets are also influenced by a complex interplay of personal, social, cultural, and economic factors [387,390]. The wider food environment has been implicated in the food choices families make, including the increased availability of highly palatable, relatively inexpensive, energy dense and nutrient poor food in increased portion sizes [387]. Understanding the roles all these factors play is important, as patterns of diet and physical activity established during childhood continue to influence health on into adulthood.

Food insecurity, defined as the inability to acquire nutritionally adequate and safe food that meets cultural needs, is influenced by family income, the number of people living in a household and the location of households in relation to food sources. Food insecurity can result in both under-nutrition and over-nutrition [165,387,391]. New Zealand research also suggests that food insecurity is more common for Māori and Pacific families and households with lower incomes. For example, data from the longitudinal Survey of Families, Income and Employment (SoFIE, n=18,950) found that households in the lowest income quintile were significantly more likely to be food insecure than those in the highest quintile (OR 4.9, 95%CI 4.0 to 5.9) [391]. Another study found that families on low incomes needed to spend between 43% and 89% of their income (once rent was deducted) to purchase a ‘basic’ healthy diet [162].

The following section reviews a range of nutritional indicators of relevance to children and young people using data from two different sources: The 2011/12 New Zealand Health Survey, and the Youth’12 Survey of secondary school students.

The 2011/12 New Zealand Health Survey

Data Sources and Methods

Definitions

The proportion of children aged 2–14 years who ate breakfast at home every day in the past week

The proportion of children aged 2–14 years who ate fast food three or more times in the past week

The proportion of children aged 2–14 years who had fizzy drinks three or more times in the past week

Data Source

The 2011/12 New Zealand Health Survey

The data on children aged 2–14 years in this section were derived from The Health of New Zealand Children 2011/12: Key Findings of the New Zealand Health Survey, and its associated data tables, downloadable at http://www.health.govt.nz/publication/health-new-zealand-children-2011-12 Regional results were sourced from http://www.health.govt.nz/publication/regional-results-2011-12-new-zealand-health-survey

Notes on Interpretation

Sample Size and Weighting: The 2011/12 NZHS [2] was a cross sectional survey carried out between July 2011 and August 2012, which collected information on 4,478 children aged from birth to 14 years, and 12,370 adults aged 15 years and over. The child survey included 2,968 European/Other, 1,592 Māori, 730 Pacific and 432 Asian children, with the data being weighted (e.g. to take into account response rates and individual item non-responses) to ensure that the results were representative of the total New Zealand child population. The weighted response rate for the child questionnaire was 85%, with the primary caregiver answering the questionnaire on their child’s behalf.

Ethnicity: In the Survey, four ethnic groups were reported: Māori, Pacific, Asian and European/Other. Total response ethnicity was used throughout, with an individual being included in the analysis for each ethnic group to which they affiliated. As a result, ethnic groups cannot be directly compared with each other, with adjusted rate ratios which compare children by ethnicity comparing those in a particular group (e.g. Pacific) with those who are not in that ethnic group (e.g. non-Pacific) [2].
**Age Standardisation**: Age is an important determinant of health status. In the NZHS, all time trend results have been age-standardised, so that populations can be compared over time, and so that any differences reported are not due to changes in the population age structure. Similarly all rate ratio comparisons by gender, ethnicity and NZ Deprivation Index (NZDep) decile have been age adjusted, so that any differences identified cannot be attributed to differing age structures between the different population groups. The method of age standardisation used was the direct method using the World Health Organization world population age distribution [2]. Regional rates however, are presented as unadjusted prevalences, so that the actual prevalence of those affected in each region can be assessed, including by age group.

**Other Standardisation and the Relative Index of Inequality**: In addition to age standardisation, any ethnic comparisons have also been adjusted for gender. For neighbourhood deprivation comparisons (i.e. by NZDep) rate ratios refer to the relative index of inequality [2]. This compares neighbourhood deprivation after adjusting for age, sex and ethnic differences. The relative index of inequality can be interpreted in the same way as adjusted rate ratios, although it is calculated in a slightly different way.

**Regional Results**: NZHS results are reported by region, with DHBs being grouped as follows: Northern Region: Northland, Waitemata, Auckland, Counties Manukau; Midland Region: Waikato, Bay of Plenty, Lakes, Tairawhiti, Taranaki; Central Region: Hawke's Bay, Whanganui, MidCentral, Hutt Valley, Capital and Coast, Wairarapa; Southern Region: Nelson Marlborough, Canterbury, South Canterbury, West Coast, Southern. It is anticipated that results will become available by DHB in future years, as more survey data is collected.

Differences between estimates are said to be statistically significant when the confidence intervals for each rate do not overlap. Sometimes, however, even when there are overlapping confidence intervals, the difference between the groups can be statistically significant. Any differences between two variables where the confidence intervals overlap are tested using a t-test. The significance of a t-test is represented by the p-value.

If a p-value is below 0.05, then we are 95 percent confident the difference between the two estimates is statistically significant [248].

**Breakfast Eaten at Home**

Eating breakfast every day was used in the 2011/12 NZ Health Survey as a proxy for healthy eating behaviour, as children who eat breakfast at home are less likely to eat high fat or high sugar snacks [2]. Further, the 2002 National Children’s Nutrition Survey [166] found that children who usually eat breakfast at home have, on average, a lower BMI than those who do not, even once other potentially confounding risk factors are taken into account.

Figure 83. Proportion of Children Aged 2–14 Years Who Ate Breakfast at Home Every Day in the Past Week by Gender and Ethnicity, 2006/07 and 2011/12 New Zealand Health Surveys

Source: 2011/12 New Zealand Health Survey; Note: Rates have been age-standardised
Trends in Proportion of Children Who Ate Breakfast at Home

**Overall:** The proportion of children aged 2–14 years who ate breakfast at home every day in the last week did not change significantly \((p=0.52)\) between NZ Health Surveys, with rates being 87.9\% (95\% CI 86.6–89.0) in 2006/07 and 87.3\% (95\% CI 85.7–88.7) in 2011/12.

**By Gender:** When broken down by gender, the proportion of boys and girls aged 2–14 years who ate breakfast at home every day in the last week did not change significantly between the 2006/07 and 2011/12 Surveys (**Figure 83**). In the 2011/12 NZHS, once adjusted for age, there were also no significant gender differences in the proportion of children aged 2–14 years who ate breakfast at home every day in the last week, with rates being 88.6\% (95\% CI 86.6–90.4) for boys and 85.9\% (95\% CI 83.5–88.0) for girls.

**By Ethnicity:** When broken down by ethnicity, there were no significant changes in the proportion of Māori, Pacific, Asian and European/Other children who ate breakfast at home every day in the last week, between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 81.8\% (95\% CI 79.1–84.2) of Māori, 81.9\% (95\% CI 77.7–85.7) of Pacific, 90.2\% (95\% CI 85.9–93.6) of Asian and 88.9\% (95\% CI 87.0–90.6) of European/Other children ate breakfast at home every day in the last week (**Figure 83**).

**Distribution by Region**

When broken down by region, there were no significant changes in the proportion of Northern, Midland, Central or Southern children who ate breakfast at home every day in the last week, between the 2006/07 and 2011/12 NZ Health Surveys. In the 2011/12 NZHS, 87.7\% (95\% CI 84.9–90.2) of Northern, 87.0\% (95\% CI 83.3–90.1) of Midland, 83.4\% (95\% CI 79.4–86.8) of Central and 90.5\% (95\% CI 87.0–93.3) of Southern children ate breakfast at home every day in the last week (**Figure 84**).

**Figure 84. Proportion of Children Aged 2–14 Years Who Ate Breakfast at Home Every Day in the Past Week by Region, 2006/07 and 2011/12 New Zealand Health Surveys**

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age
Current Distribution of Children Who Ate Breakfast at Home

**Distribution by Age**
In the 2011/12 NZ Health Survey, children aged 10–14 years (81.2% (95% CI 78.3–83.9)) were significantly less likely to eat breakfast at home every day in the last week than children aged 2–4 years (93.9% (95% CI 91.7–95.6)). Similar age-related differences were evident when rates were broken down by gender (Figure 85).

**Distribution by Ethnicity**
In the 2011/12 NZ Health Survey, once adjusted for age and gender, Māori children were significantly less likely (aRR 0.92 (95% CI 0.88–0.95)) than non-Māori children to have eaten breakfast at home every day in the last week, with rates also being significantly lower for Pacific children (aRR 0.93 (95% CI 0.89–0.98)) than for non-Pacific children. No significant differences however were evident between Asian and non-Asian children.

**Distribution by NZ Deprivation Index Decile**
In the 2011/12 NZ Health Survey, once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly less likely to eat breakfast at home every day in the last week than children in the least deprived (NZDep06 deciles 1–2) areas (aRR 0.94 (95% CI 0.88–1.00)).

Figure 85. Proportion of Children Aged 2–14 Years Who Ate Breakfast at Home Every Day in the Past Week by Gender, Age Group, Ethnicity and NZ Deprivation Index Decile, 2011/12 New Zealand Health Survey

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age

**Fast Food Consumption**

**Trends in Proportion of Children Who Ate Fast Food**
**Overall:** The proportion of children aged 2–14 years who ate fast food three or more times in the past week did not change significantly (p=0.39) between NZ Health Surveys, with rates being 7.2% (95% CI 6.3–8.2) in 2006/07 and 6.5% (95% CI 5.3–7.9) in 2011/12.
Figure 86. Proportion of Children Aged 2–14 Years Who Ate Fast Food Three or More Times in the Past Week by Gender and Ethnicity, 2006/07 and 2011/12 New Zealand Health Surveys

Source: 2011/12 New Zealand Health Survey; Note: Rates have been age-standardised

By Gender: When broken down by gender, the proportion of boys and girls aged 2–14 years who had eaten fast food three or more times in the past week did not change significantly between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, once adjusted for age, there were also no significant gender differences in the proportion of children aged 2–14 years who had eaten fast food three or more times in the past week, with rates being 6.2% (95% CI 4.7–8.0) for boys and 6.8% (95% CI 5.2–8.9) for girls (Figure 86).

By Ethnicity: When broken down by ethnicity, there were also no significant changes in the proportion of Māori, Pacific, Asian and European/Other children who had eaten fast food three or more times in the past week between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 10.3% (95% CI 8.1–13.0) of Māori, 15.7% (95% CI 11.7–20.5) of Pacific, 5.9% (95% CI 3.3–9.5) of Asian and 4.5% (95% CI 3.5–5.7) of European/Other children had eaten fast food three or more times in the past week (Figure 86).

Current Distribution of Children Who Ate Fast Food

Distribution by Age
In the 2011/12 NZ Health Survey, while a higher proportion of children aged 10–14 years (8.2% (95% CI 6.2–10.6)) had eaten fast food three or more times in the past week than children aged 2–4 years (5.0% (95% CI 3.3–7.2)) these differences did not reach statistical significance. A similar pattern was seen when age specific rates were broken down by gender (Figure 87).

Distribution by Ethnicity
In the 2011/12 NZ Health Survey, once adjusted for age and gender, Māori children were significantly more likely (aRR 1.96 (95% CI 1.45–2.64)) than non-Māori children to have eaten fast food three or more times in the past week, with rates also being significantly higher for Pacific children (aRR 3.20 (95% CI 2.34–4.36)) than for non-Pacific children. No significant differences however were evident between Asian and non-Asian children.
Figure 87. Proportion of Children Aged 2–14 Years Who Ate Fast Food Three or More Times in the Past Week by Gender, Age, Ethnicity and NZ Deprivation Index Decile, 2011/12 New Zealand Health Survey

**Distribution by NZ Deprivation Index Decile**
In the 2011/12 NZ Health Survey, once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly more likely to have eaten fast food three or more times in the past week than children in the least deprived (NZDep06 deciles 1–2) areas (aRR 3.23 (95% CI 1.66–6.29)).

**Fizzy Drink Consumption**

**Trends in the Proportion of Children Who Consumed Fizzy Drinks**

**Overall:** The proportion of children aged 2–14 years who had consumed fizzy drinks three or more times in the past week did not change significantly (p=0.98) between NZ Health Surveys, with rates being 19.6% (95% CI 18.0–21.2) in 2006/07 and 19.6% (95% CI 17.9–21.4)) in 2011/12.

**By Gender:** When broken down by gender, the proportion of boys and girls aged 2–14 years who had consumed fizzy drinks three or more times in the past week did not change significantly between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS however, once adjusted for age, boys (22.1% (95% CI 19.6–24.7)) were significantly more likely than girls (16.9% (95% CI 14.8–19.3)) to have consumed fizzy drinks three or more times in the past week (Figure 88).

**By Ethnicity:** When broken down by ethnicity, there were no significant changes in the proportion of Māori, Pacific, Asian and European/Other children who had consumed fizzy drinks three or more times in the past week between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 25.0% (95% CI 21.6–28.6) of Māori, 26.9% (95% CI 20.8–33.8) of Pacific, 27.9% (95% CI 21.3–35.4) of Asian and 16.4% (95% CI 14.7–18.2) of European/Other children had consumed fizzy drinks three or more times in the past week (Figure 88).
Current Distribution of Children Who Consumed Fizzy Drinks

Distribution by Age
In the 2011/12 NZ Health Survey, children aged 10–14 years (28.4% (95% CI 25.4–31.6)) were significantly more likely have consumed fizzy drinks three or more times in the past week than children aged 2–4 years (12.9% (95% CI 9.5–16.9)). Similar age-related differences were evident when rates were broken down by gender (Figure 89).

Distribution by Ethnicity
In the 2011/12 NZ Health Survey, once adjusted for age and gender, Māori children were significantly more likely (aRR 1.40 (95% CI 1.19–1.65)) than non-Māori children to have consumed fizzy drinks three or more times in the past week. Rates were also significantly higher for Pacific children (aRR 1.49 (95% CI 1.23–1.81)) than for non-Pacific children and for Asian children (aRR 1.49 (95% CI 1.17–1.91)) than for non-Asian children.

Distribution by NZ Deprivation Index Decile
In the 2011/12 NZ Health Survey, once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly more likely have consumed fizzy drinks three or more times in the past week than children in the least deprived (NZDep06 deciles 1–2) areas (aRR 1.81 (1.30–2.51)).
Figure 89. Proportion of Children Aged 2–14 Years Who Had Fizzy Drinks Three or More Times in the Past Week by Gender, Age, Ethnicity and NZ Deprivation Index Decile, 2011/12 New Zealand Health Survey

Source: 2011/12 New Zealand Health Survey; Note: Rates are unadjusted for age

Youth’12 Survey

Youth’12 was the third national survey of Year 9–15 students (Years 14 and 15 are those students who are repeating Years 12 and 13) in New Zealand, with previous surveys being undertaken in 2001 and 2007. The Youth’12 sample included 8,500 students aged 13–17+ years, which was approximately 3% of the 2012 New Zealand secondary school roll. In the survey students were asked how often they ate breakfast, as well as about their daily fruit and vegetable consumption [249].

Data Sources and Methods

Definitions
1. Frequency of eating breakfast in secondary school students aged 13–17+ years
2. Proportion of secondary school students aged 13–17+ years who ate 2+ fruit and 3+ vegetables per day

Data Sources

The Youth’12 Survey

In this section, data on nutrition in secondary school students was derived from The Youth’12 Overview: The Health and Wellbeing of New Zealand Secondary School Students in 2012 [250], and its companion document the Youth’12 Prevalence Tables [249].

Notes on Interpretation

Survey Methodology and Sample: Youth’12 is the third national health and wellbeing survey of secondary school students in New Zealand, with previous surveys being undertaken in 2001 and 2007. The Youth’12 Survey was a random survey of composite and secondary schools. For schools with more than 150 Year 9–15 students, a random sample of 20% of the roll were invited to participate, while for schools with a roll of <150, 30 students were randomly asked to participate. Overall, 73% of invited schools took part, with 68% of students who were invited to participate agreeing to do so. This resulted in total sample of 8,500 students (3% of the 2012 New Zealand secondary school roll) [249].

Ethnicity Reporting: The Youth’12 ethnicity question was based on the NZ Census 2001/2006 ethnicity question, which asked students which ethnic group they belonged to, with multiple responses being possible. For the purposes of comparing ethnic groups, Statistics NZ’s ethnicity prioritisation methods were used [252], which reported five ethnic groups: Māori, Pacific, Asian, European and Other.
How Often Students Eat Breakfast

In the Youth’12 Survey, 16.7% (95% CI 15.1–18.2) of secondary school students said they hardly ever ate breakfast, with the proportion of females (20.8% (95% CI 18.4–23.3)) being significantly higher than for males (11.7% (95% CI 10.6–12.8)).

While there were no age-related differences in the proportion of students who hardly ever ate breakfast, a significantly higher proportion of students from the most deprived (NZDep deciles 8–10) areas (21.8% (95% CI 19.4–24.3)) said they hardly ever ate breakfast, than students from the least deprived (NZDep06 deciles 1–3) areas (12.7% (95% CI 11.0–14.3)).

There were no significant urban (17.2% (95% CI 15.5–19.0) vs. rural (13.8% (95% CI 11.5–16.0) differences in the proportion of students who said they hardly ever ate breakfast (Figure 90).

Figure 90. Frequency of Eating Breakfast in Secondary School Students Aged 13–17+ Years by Gender, Age, NZ Deprivation Index Decile and Geography, New Zealand Youth’12 Survey

Fruit and Vegetable Consumption

In the Youth’12 survey, 30.0% (95% CI 28.4–31.6) of secondary school students said that they ate 2+ fruit and 3+ vegetables per day. There were no significant gender or age differences in the proportion of students who ate 2+ fruit and 3+ vegetables per day. Rates were also not significantly different between those living in the most and least deprived NZDep06 areas, or in urban and rural areas (Figure 91).
Figure 91. Fruit and Vegetable Consumption in Secondary School Students Aged 13–17+ Years by Gender, Age, NZ Deprivation Index Decile and Geography, New Zealand Youth’12 Survey

Source: Youth’12 Survey; Note: NZDep2006: Low = Deciles 1–3; Medium = Deciles 4–7; High = Deciles 8–10

Local Policy Documents and Evidence-Based Reviews Relevant to Nutrition in Children and Young People

In New Zealand a number of local policy documents relate to nutrition in children and young people, and these are briefly summarised in Table 116, along with a range of systematic and other reviews which consider these issues in the overseas context.

In addition, Table 115 on Page 372 summarises publications relevant to breastfeeding and infant nutrition, while Table 105 on Page 310, reviews publications relevant to the prevention and management of obesity. An in-depth topic commencing on Page 287 also reviews The Determinants and Consequences of Overweight and Obesity, while the in-depth topic commencing on Page 332 reviews The Treatment of Obesity in Children and Adolescents.

Table 116. Local Policy Documents and Evidence-Based Reviews Relevant to Nutrition

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<th>Ministry of Health Policy Documents</th>
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This background paper provides evidence-based technical information and best practice recommendations on nutrition and physical activity for health practitioners working in clinical and population health settings, and is used as the basis health education resources for the public. The paper includes a set of guideline statements on food, nutrition and physical activity, and recommendations for meal patterns, measurement of body size and nutrient intake. The paper includes reviews of meal patterns, physical activity and body size among New Zealand children and adolescents, and the wider determinants of health and the food environment. Further chapters address food and nutrition among tamariki and rangatahi Māori and their whānau, Pacific children and young people and their families, and Asian and other populations.
particularly US based, controlled trials were included in the review. None of the trials investigated intervention with fruits, price manipulation of vegetables, or feedback that was a tangible non-food reward. A narrative review was undertaken. The studies addressing price incentives suggested that such incentives may be more successful. Further research is required to assess the effect and that post-intervention follow-up on maintenance of change is needed and studies should also include objective physical measurements where possible.

**Cochrane Systematic Reviews**

Wolfenden L, et al. 2012. *Interventions for increasing fruit and vegetable consumption in children aged 5 years and under*. Cochrane Database of Systematic Reviews doi:10.1002/14651858.CD008552.pub2

This review assessed the effectiveness, cost-effectiveness and associated adverse events of interventions designed to increase the consumption of fruit and/or vegetables amongst children aged five years and under. Only five trials (2 RCTs and 3 cluster RCTs, 3967 participants) were included in the review. Two trials examined the impact of specific feeding practices (e.g. repeated food exposure) in increasing child intake of a target vegetable. Two trials assessed the effectiveness of home visiting programmes implemented in disadvantaged communities and one trial investigated the effect of a preschool-based intervention in increasing child fruit and vegetable intake. Meta-analysis of two trials examining repeated food exposure versus a no intervention comparison found no significant difference in target vegetable consumption in the short term (mean difference 1.37, 95% CI -2.78 to 5.52). One trial found that coupling repeated food exposure with a tangible non-food or social reward was effective in increasing targeted vegetable consumption in the short term. The home visiting programmes did not significantly increase overall fruit intake in the short term (standardised mean difference 0.01, 95% CI -0.09 to 0.11), and the multi-component preschool-based intervention failed to significantly increase child consumption of vegetables, but did report a small significant increase in mean child consumption of fruit, six months following baseline assessment. None of the trials investigated intervention cost-effectiveness or reported information regarding any adverse events or unintended adverse consequences. The authors conclude that the review highlights the paucity of evidence for effective interventions.

**Other Systematic Reviews**


This review assessed the effectiveness of computer- and web-based interventions on improving eating behaviour (e.g. increasing fruit and vegetable consumption; decreasing fat consumption) and/or diet-related physical outcomes (e.g. body mass index) among children and adolescents. Fifteen, mostly US based, controlled trials were included in the review. All the studies were directed towards improving some type of eating behaviour. While a majority of interventions resulted in statistically significant positive changes in eating behaviour and/or diet-related physical outcomes, interventions that included post intervention follow-up (ranging from 3 to 18 months) showed that changes were not maintained. The authors suggest that interventions delivered in schools, and incorporating individually tailored feedback may be more successful. Further research is required to assess the effect and that post-intervention follow-up on maintenance of change is needed and studies should also include objective physical measurements where possible.


This review aimed to quantify the impact of school-based interventions on fruit and vegetable intake in children aged five to 12 years. Twenty seven (mostly cluster RCTs) studies were included in the review, 21 of which met the criteria for meta-analysis. The meta-analyses indicated an improvement of 0.25 portions (95% CI 0.06 to 0.43 portions) of fruit and vegetable daily intake if fruit juice was excluded and an improvement of 0.32 portions (95% CI 0.14 to 0.50 portions) if fruit juice was included. Improvement was mainly due to increases in fruit consumption: the results for fruit (excluding juice) and vegetables separately indicated an improvement of 0.24 portions (95% CI 0.05 to 0.43 portions) and 0.07 portions (95% CI 0.03 to 0.16 portions), respectively. The overall quality of the studies was poor with a high risk of bias. The authors conclude that while school-based interventions can moderately increase fruit intake, they appear to have minimal effect on vegetable intake and further research is required to address the barriers to improving vegetable intake.


This review assessed the effectiveness of economic incentives, such as free fruit and vegetables, price manipulation of healthy and energy-dense snack foods and rewards for tasting fruit and vegetables, for improving nutritional behaviour in schools. Twenty-eight studies, using a variety of methodologies including four RCTs, were included in the review. Outcome measures included self-reports, monitoring of sales and observation by researchers. The majority of studies took place in the US. A narrative review was undertaken. The studies addressing price incentives suggested that such incentives are effective for altering consumption in the school setting. Other types of economic incentives have been included in combined intervention schemes, but the inclusion of other intervention elements made it difficult to draw conclusions about the effectiveness of these economic incentive instruments and further research is suggested.

This review assessed the effect of school-based interventions in Europe to promote a healthy diet in children and adolescents aged six to 18 years. Forty-two studies, mostly conducted in the UK, were included for review (n=72,600): 29 studies of children (n=42,060) and 13 studies of adolescents (n=30,540). Included studies evaluated educational programmes, environmental modifications (school lunch modifications, increased availability of healthy foods, fruit and vegetable distribution) or a combined educational and environmental modification programme. Most interventions included some family involvement and were classroom based. Intervention duration ranged from two weeks to five years in children and from one week to two years in adolescents. Some studies targeted low socioeconomic status groups or ethnic minority groups. Given the heterogeneity of the studies a narrative synthesis was undertaken. In children there was limited evidence that educational interventions improved dietary behaviour, six of 12 studies found no effect. The evidence for the effect on anthropometric outcomes was inconclusive (4 studies). Six studies found positive effects of fruit and vegetable distribution or breakfast initiatives on dietary behaviour; however, all studies were of moderate or weak quality. All nine of the studies of interventions that combined fruit and vegetable distribution and education improved fruit and vegetable intake reported positive effects. In adolescents there was moderate evidence that educational interventions in adolescents improved dietary intake (5 studies). Evidence for beneficial effects of education interventions on anthropometric outcomes was inconclusive. Two weak trials reported conflicting results for the effect of environmental interventions on dietary behaviour in adolescents. There was limited evidence for the impact of environmental intervention combined with nutrition education on dietary behaviour in adolescents (4 weak studies). The authors conclude that multicomponent interventions that promoted a healthy diet in school-age children in European Union countries had a positive impact on self-reported dietary behaviour, but evidence of effectiveness on anthropometric outcomes is lacking.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
**Introduction**

Participation in physical activity is important for children and young people’s growth and development. It has been linked to the prevention of type 2 diabetes and improvements in skeletal health, self-esteem and depression [392]. Physical activity also plays an important role in preventing overweight and obesity, and the decline in physical activity in recent decades is thought to have contributed to rising obesity levels [393].

Worldwide, a range of environmental factors have been associated with the decline in physical activity, including reductions in active transport such as walking and cycling, and increases in sedentary leisure behaviours such as television watching [393,394,395]. Physical activity also appears to decline as children transition through adolescence [396].

The New Zealand Physical Activity Guidelines developed in 2007 by the Ministries of Health and Education, and Sport New Zealand, recommend that children and young people aged five to 18 years do sixty minutes or more of moderate to vigorous physical activity each day [397]. This should consist of a variety of activities, and it is recommended that less than two hours each day (out of school hours) should be spent in front of television, computers, and game consoles.

The following section reviews a range of physical and sedentary activities in children and young people using data from two sources: The Youth’12 Survey of secondary school students and the 2011/12 NZ Health Survey.

**Youth’12 Survey**

Youth’12 was the third national survey of Year 9–15 students (Years 14 and 15 are those students who are repeating Years 12 and 13) in New Zealand, with previous surveys being undertaken in 2001 and 2007. The Youth’12 sample included 8,500 students aged 13–17+ years, which was approximately 3% of the 2012 New Zealand secondary school roll. In the survey, students were asked a range of questions about their level of physical activity and how they spent their leisure [250].

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**Data Sources and Methods**

**Definitions**

1. Proportion of secondary school students aged 13–17+ years who engaged in more than 20 minutes of vigorous physical activity on three or more occasions in the past 7 days
2. Proportion of secondary school students aged 13–17+ years who did 60+ minutes physical activity daily
3. Proportion of secondary school students aged 13–17+ years who participated in sports teams and clubs outside of school and the reasons given by those not participating, for non-participation
4. Time spent on selected sedentary leisure activities in secondary school students aged 13–17+ years

**Data Sources**

The Youth’12 Survey

The data in this section are derived from The Youth’12 Overview: The Health and Wellbeing of New Zealand Secondary School Students in 2012 [250], and its companion document the Youth’12 Prevalence Tables [249].

**Notes on Interpretation**

**Survey Methodology and Sample:** Youth’12 is the third national health and wellbeing survey of secondary school students in New Zealand, produced by the Adolescent Health Research Group (AHRG), with previous surveys being undertaken in 2001 and 2007. For composite and secondary schools with more than 150 Year 9–15 students, a random sample of 20% of the roll were invited to participate, while for schools with a roll of less than 150, 30 students were randomly asked to participate. Overall, 73% of invited schools took part, with 68% of students who were invited to participate agreeing to do so. This resulted in total sample of 8,500 students (3% of the 2012 New Zealand secondary school roll). Students were asked to provide their address to determine their census meshblock (NZDep 2006) [250].
Students Participation in Physical Activity

In the Youth’12 survey, while 61.9% (95% CI 59.9–64.0) of students had participated in more than 20 minutes vigorous physical activity on three or more occasions in the past seven days, only 9.6% (95% CI 8.7–10.5) reported achieving the recommended 60+ minutes of physical activity daily.

The proportion of males (68.5% (95% CI 66.2–70.8) undertaking more than 20 minutes vigorous physical activity on three or more occasions in the past seven days was significantly higher than for females (56.6% (95% CI 53.9–59.1). Rates were also significantly higher for younger students (aged 15 years and younger) than for older students (16 years and over), and for students from less deprived (NZDep06 decile1–3) areas, than for students from more deprived (NZDep06 deciles 8–10) areas (66.3% (95% CI 63.5–69.1). vs. 56.7% (95% CI 54.2–59.3)). Rates were also significantly higher for students from rural (68.0% (95% CI 64.6–71.3) than urban (61.0% (95% CI 58.9–63.2) areas (Figure 92).

The proportion of males (13.6% (95% CI 12.4–14.7)) achieving the recommended 60+ minutes of physical activity a day was also significantly higher than for females (6.3% (95% CI 5.6–7.0). No significant differences were evident however, by NZDep06 decile or rural/urban area of residence (Figure 92).

Figure 92. Proportion of Secondary School Students Aged 13–17+ Years Who Engaged in More than 20 Minutes of Vigorous Physical Activity on 3+ Occasions in Past 7 Days, or Who Did 60+ Minutes Physical Activity Daily, New Zealand Youth’12 Survey
Travel to School by Active Means
In the Youth’12 survey, 32.7% (95% CI 29.5–35.9) of students usually travelled to school by active means (walk, bike or skate) six or more times in the past seven days. Note: walking to school and home again on one day was counted as two times; walking to school and driving home was counted as one time.

While there were no significant gender differences in the proportion of students who usually travelled to school by active means, rates were significantly higher for students aged 15 years or less, than for those aged 17 years or older. In addition, a significantly higher proportion of students from more deprived (NZDep06 deciles 8–10) areas (37.2% (95% CI 33.0–41.5) usually travelled to school by active means, than did students from less deprived (NZDep06 decile1–3) areas (25.7% (95% CI 21.4–30.0). Rates were also significantly higher for students from urban (35.3% (95% CI 31.9–38.8) than rural (18.3 (95% CI 13.6–23.0)) areas (Figure 93).

Figure 93. Proportion of Secondary School Students Aged 13–17+ Years Who Walked, Biked or Skated To/From School 6+ Times in Past 7 Days, New Zealand Youth’12 Survey

Source: Youth’12 Survey; Note: Walking to school and home again on one day is counted two times; walking to school and driving home is counted once

Participation in Sports Teams and Clubs
In the Youth’12 Survey, 58.7% (95% CI 55.9–61.4) of students participated in sports teams or clubs outside of school hours, with participation rates being lowest for older students (17 or more years), those from more deprived (NZDep06 deciles 8–10) areas, and for students from urban areas. A range of reasons were given for non-participation amongst those who did not belong to a sports team or club, with the most frequent being not being interested, having other responsibilities, not being good enough at sport, and feeling shy, nervous or embarrassed (Table 117).
Table 117. Participation in Sports Teams and Clubs Outside of School and Reasons for Non-Participation, Secondary School Students Aged 13–17+ Years, New Zealand Youth’12 Survey

<table>
<thead>
<tr>
<th>Participation or Reasons Given for non-Participation</th>
<th>Percent (%)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participates in a Sports Team or Club Outside of School</td>
<td>58.7</td>
<td>55.9–61.4</td>
</tr>
<tr>
<td>Reasons For Not Participating in Sports Teams or Clubs Outside School*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>It costs too much</td>
<td>14.3</td>
<td>12.6–16.1</td>
</tr>
<tr>
<td>I’m not good enough at sport</td>
<td>20.8</td>
<td>19.2–22.4</td>
</tr>
<tr>
<td>I’m not interested</td>
<td>39.4</td>
<td>37.2–41.6</td>
</tr>
<tr>
<td>It takes too much time</td>
<td>15.1</td>
<td>13.1–16.8</td>
</tr>
<tr>
<td>None of my friends are in sports</td>
<td>9.1</td>
<td>8.0–10.3</td>
</tr>
<tr>
<td>The sports I’m interested in aren’t available</td>
<td>11.3</td>
<td>10.1–12.4</td>
</tr>
<tr>
<td>Can’t get there</td>
<td>12.1</td>
<td>10.9–13.4</td>
</tr>
<tr>
<td>I would feel shy, nervous or embarrassed</td>
<td>17.5</td>
<td>16.0–19.0</td>
</tr>
<tr>
<td>I have other responsibilities</td>
<td>23.2</td>
<td>21.6–24.7</td>
</tr>
<tr>
<td>My parents wouldn’t let me</td>
<td>4.7</td>
<td>3.5–5.8</td>
</tr>
<tr>
<td>I don’t know</td>
<td>10.8</td>
<td>9.5–12.1</td>
</tr>
<tr>
<td>There are no sports facilities in my area</td>
<td>14.6</td>
<td>13.3–16.0</td>
</tr>
<tr>
<td>Other</td>
<td>3.9</td>
<td>3.1–4.6</td>
</tr>
</tbody>
</table>

Source: Youth’12 Survey; Note: *Reasons given are for students not involved in sports teams or clubs; students could choose more than one response so numbers do not sum to 100%

Time Spent in Sedentary Leisure

In the Youth’12 Survey 28.2% (95% CI 25.9–30.4) of students spent three or more hours each day watching TV, while 19.5% (95% CI 17.4–21.7) spent three or more hours playing computer games, and 34.8% (95% CI 33.2–36.4) spent three or more hours on the internet.

While there were no significant gender differences in the proportion of students who spent three or more hours watching TV, or going on the internet, the proportion of males (31.9% (95% CI 29.6–34.2)) who spent three or more hours playing computer games was significantly higher than for females (9.4% (95% CI 7.6–11.3)) (Figure 94, Figure 95).

When broken down by age, a significantly higher proportion of students 14 years or less spent three or more hours playing computer games than did those 17 years or older. In contrast, a significantly lower proportion of students aged 13 years or less spent three hours or more hours on the internet, than did those age 16 years or older. There were no significant age differences however, in the proportion that spent three or more hours watching TV (Figure 94, Figure 95).

A significantly higher proportion of students from more deprived (NZDep06 deciles 8–10) areas spent three or more hours playing computer games, watching TV and going on the internet, than did those from less deprived (NZDep06 deciles 1–3) areas. Similarly a significantly higher proportion of students from urban areas spent three or more hours playing computer games and going on the internet, than did students from rural areas (Figure 94, Figure 95).
Figure 94. Time Spent Per Day on Selected Sedentary Leisure Activities, Secondary School Students Aged 13–17+ Years, New Zealand Youth’12 Survey

Source: Youth’12 Survey; Note: Computer games excludes physically interactive computer games like Wii

Figure 95. Proportion of Secondary School Students Aged 13–17+ Years who Spend 3+ Hours Each Day on Selected Sedentary Leisure Activities, New Zealand Youth’12 Survey

Source: Youth’12 Survey; Note: Computer games excludes physically interactive computer games like Wii
The 2011/12 NZ Health Survey

In the 2011/12 NZ Health Survey [2], the parents of children aged under 15 years were asked about their child’s television viewing habits, as well as whether they travelled to school (if aged five and over) by active means. Information on school transport was collected in a similar way to the 2006/07 NZ Health Survey, making it possible to compare changes over time. The following section briefly reviews changes in the proportion of children travelling to school by active means between the 2006/07 and 2011/12 NZ Health Surveys, as well as the proportion of children who usually watched two or more hours of television per day in the most recent 2011/12 NZHS.

Data Sources and Methods

Definitions

- The proportion of children aged 5–14 years who usually use active transport to and from school
- The proportion of children aged 2–14 years who usually watch two or more hours of television per day

Data Sources

The 2011/12 New Zealand Health Survey (NZHS)

In this section, the data on children aged 2–14 years were derived from The Health of New Zealand Children 2011/12: Key Findings of the New Zealand Health Survey, with data tables from this report being downloadable at http://www.health.govt.nz/publication/health-new-zealand-children-2011-12

Notes on Interpretation

Sample Size and Weighting: The 2011/12 NZHS [2] was a cross sectional survey carried out between July 2011 and August 2012, which collected information on 4,478 children aged from birth to 14 years, and 12,370 adults aged 15 years and over. The child survey included 2,968 European/Other, 1,592 Māori, 730 Pacific and 432 Asian children, with the data being weighted (e.g. to take into account response rates and individual item non-responses) to ensure that the results were representative of the total New Zealand child population. The weighted response rate for the child questionnaire was 85%, with the primary caregiver answering the questionnaire on their child’s behalf.

Ethnicity: In the Survey, four ethnic groups were reported: Māori, Pacific, Asian and European/Other. Total response ethnicity was used throughout, with an individual being included in the analysis for each ethnic group to which they affiliated. As a result, ethnic groups cannot be directly compared with each other, with adjusted rate ratios which compare children by ethnicity comparing those in a particular group (e.g. Pacific) with those who are not in that ethnic group (e.g. non-Pacific) [2].

Age Standardisation: Age is an important determinant of health status. In the NZHS, all time trend results have been age-standardised, so that populations can be compared over time, and so that any differences reported are not due to changes in the population age structure. Similarly all rate ratio comparisons by gender, ethnicity and NZ Deprivation Index (NZDep) decile have been age adjusted, so that any differences identified cannot be attributed to differing age structures between the different population groups. The method of age standardisation used was the direct method using the World Health Organization (WHO) world population age distribution [2].

Other Standardisation and the Relative Index of Inequality: In addition to age standardisation, any ethnic comparisons have also been adjusted for gender. For neighbourhood deprivation comparisons (i.e. by NZDep) rate ratios refer to the relative index of inequality [2]. This compares neighbourhood deprivation after adjusting for age, sex and ethnic differences. The relative index of inequality can be interpreted in the same way as adjusted rate ratios, although it is calculated in a slightly different way.

Differences between estimates are said to be statistically significant when the confidence intervals for each rate do not overlap. Sometimes, however, even when there are overlapping confidence intervals, the difference between the groups can be statistically significant. Any differences between two variables where the confidence intervals overlap are tested using a t-test. The significance of a t-test is represented by the p-value. If a p-value is below 0.05, then we are 95 percent confident the difference between the two estimates is statistically significant [248].

Travel to School by Active Means

Trends in Proportion of Children Travelling to School by Active Means

Overall: The proportion of children aged 5–14 years who usually travelled to school by active means did not change significantly (p=0.51) between NZ Health Surveys, with rates being 46.1% (95% CI 43.3–48.8) in 2006/07 and 47.5% (95% CI 44.2–50.7) in 2011/12.

By Gender: When broken down by gender, the proportion of boys and girls aged 5–14 years who usually travelled to school by active means did not change significantly between the 2006/07 and 2011/12 Surveys (Figure 96). In the 2011/12 NZHS, once adjusted for age, there were also no significant gender differences in the proportion of children aged 5–

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14 years who usually travelled to school by active means, with rates being 48.2% (95% CI 43.9–52.5) for boys and 46.7% (95% CI 42.7–50.8) for girls.

Figure 96. Proportion of Children Aged 5–14 Years Who Usually Use Active Transport to and From School by Gender and Ethnicity, 2006/07 and 2011/12 NZ Health Surveys

Figure 97. Proportion of Children Aged 5–14 Years Who Usually Use Active Transport to and From School by Gender, Age, Ethnicity and NZ Deprivation Index Decile, 2011/12 NZ Health Survey

Source: 2011/12 New Zealand Health Survey
By Ethnicity: When broken down by ethnicity, there were no significant changes in the proportion of Māori, Pacific, Asian and European/Other children who usually travelled to school by active means, between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 52.6% (95% CI 48.1–57.0) of Māori, 55.9% (95% CI 49.4–62.2) of Pacific, 47.5% (95% CI 40.5–54.6) of Asian and 44.5% (95% CI 40.3–48.7) of European/Other children usually travelled to school by active means (Figure 96).

Current Distribution of Children Travelling to School by Active Means

Distribution by Age
In the 2011/12 NZ Health Survey, there were no significant differences in the proportion of younger (5–9 years (44.7% (95% CI 40.8–48.7))) and older (10–14 years (50.2% (95% CI 46.2–54.3))) children who usually travelled to school by active means. Similarly, no age-related differences were evident when rates were broken down by gender (Figure 97).

Distribution by Ethnicity
In the 2011/12 NZ Health Survey, once adjusted for age and gender, Māori children were significantly more likely (aRR 1.15 (95% CI 1.03–1.27)) than non-Māori children to travel to school by active means, with rates also being significantly higher for Pacific children (aRR 1.22 (95% CI 1.09–1.38)) than for non-Pacific children. No significant differences however were evident between Asian and non-Asian children.

Distribution by NZ Deprivation Index Decile
In the 2011/12 NZ Health Survey, once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly more likely to travel to school by active means than children in the least deprived (NZDep06 deciles 1–2) areas (aRR 1.26 (95% CI 1.04–1.52)).

Current Distribution of Children Watching 2+ Hours of Television per Day

Distribution by Age
In the 2011/12 NZ Health Survey, children aged 5–9 years (49.1% (95% CI 45.2–53.1)) were significantly less likely to watch two or more hours of television per day than children aged 10–14 years (57.8% (95% CI 54.5–60.9)). While still evident, these differences were no longer statistically significant when rates were broken down by gender (Figure 98).

Distribution by Ethnicity
In the 2011/12 NZ Health Survey, once adjusted for age and gender, Māori children were significantly more likely (aRR 1.24 (95% CI 1.15–1.34)) than non-Māori children to watch two or more hours of television per day, with rates also being significantly higher for Pacific children (aRR 1.14 (95% CI 1.03–1.25)) than for non-Pacific children. No significant differences however were evident between Asian and non-Asian children.

Distribution by NZ Deprivation Index Decile
In the 2011/12 NZ Health Survey, once adjusted for age, ethnicity and gender, children from the most deprived (NZDep06 deciles 9–10) areas were significantly more likely to watch two or more hours of television per day than children in the least deprived (NZDep06 deciles 1–2) areas (aRR 1.19 (95% CI 1.03–1.39)).
Figure 98. Proportion of Children Aged 2–14 Years Who Usually Watch 2+ Hours of Television per Day by Gender, Age, Ethnicity and NZ Deprivation Index Decile, 2011/12

New Zealand Health Survey

Source: 2011/12 New Zealand Health Survey
Local Policy Documents and Evidence-Based Reviews Relevant to Physical Activity in Children and Young People

In New Zealand a number of local policy documents relate to physical activity in children and young people, and these are briefly summarised in Table 118, along with a range of systematic and other reviews which consider these issues in the overseas context.

Table 118. Local Policy Documents and Evidence-Based Reviews Relevant to Physical Activity

<table>
<thead>
<tr>
<th>Ministry of Health Policy Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>This health information leaflet, based on Ministry of Health physical activity guidance (<a href="http://www.health.govt.nz/our-work/preventative-health-wellness/physical-activity">http://www.health.govt.nz/our-work/preventative-health-wellness/physical-activity</a>), provides advice to parents and carers on physical activity for five to 18 year olds. At least 60 minutes of moderate to vigorous physical activity each day is recommended. Using a mixture of activities to encourage aerobic fitness, strength and flexibility is advised and examples of moderate and vigorous intensity activities are given. There are no specific guidelines for children under five years, but Sport New Zealand provides active movement guides for nought to five year olds (<a href="http://www.sportnz.org.nz/en-nz/young-people/Ages-0-5-Years/Active-Movement-Resources/">http://www.sportnz.org.nz/en-nz/young-people/Ages-0-5-Years/Active-Movement-Resources/</a>).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Other Government Publications</th>
</tr>
</thead>
<tbody>
<tr>
<td>This report provides guidelines for organisations and individuals that run sport and recreation programmes for children and young people (aged 0–24 years). The guidelines aim to encourage the development of a child/young person centred philosophy that ensures that children and young people receive the greatest possible value from their participation and are encouraged to continue to participate over time.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cochrane Systematic Reviews</th>
</tr>
</thead>
<tbody>
<tr>
<td>This review assessed the effectiveness of school-based interventions in promoting physical activity and fitness in children and adolescents. The review included 26 studies that were deemed of sufficient quality, most of which were conducted in the United States, with a smaller number from Europe and Australia. Participants ranged in age from six to 18 years. All the projects had a control group that represented either a school or group of schools from a different community, city or state that did not receive the intervention. Overall, school-based interventions had positive effects on duration of physical activity, television viewing, VO2 max (maximal oxygen uptake or aerobic capacity, reflects the physical fitness level of an individual and generally increases as fitness levels improve), and blood cholesterol, but no effect on leisure time physical activity rates, systolic and diastolic blood pressure, body mass index, and pulse rate. There was no evidence of harmful effects and the authors recommend ongoing physical activity promotion in schools.</td>
</tr>
</tbody>
</table>

| This review sought to determine whether exercise alone or exercise as part of a comprehensive intervention can improve self-esteem among children and young people. Twenty-three RCTs (1,821 participants, aged 3 to 20 years) were included in the review. The trials were mostly small in size and of low quality. Thirteen trials compared exercise alone with no intervention, eight of which were included in the meta-analysis, which found heterogeneous results. Twelve trials compared exercise as part of a comprehensive programme with no intervention, only four of which provided data sufficient to calculate overall effects, indicating a moderate short-term difference in self-esteem in favour of the intervention (SMD 0.51, 95% CI 0.15 to 0.88). The authors conclude that the evidence suggests that exercise has positive short-term effects on children’s self-esteem, but further well-designed research is required. |

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This meta-analysis assessed the effects of interventions, implemented in schools and the general population, aimed at preventing high levels of sedentary behaviour (e.g., television/DVD watching) on the amount of sedentary behaviour and BMI. Thirty-four studies (4 controlled trials and 30 RCTs) reporting 33 different interventions, from the USA, Europe and Australasia lasting seven days to four years, were included in the analysis. Follow-up was post-intervention in the majority of studies. Significant reductions in sedentary behaviour and BMI were identified. The post-intervention mean difference in sedentary behaviour was −17.95 min/day (95% CI -26.61 to -9.28) and the change-from-baseline mean difference was −20.44 min/day (95% CI -30.69 to -10.20). The post-intervention mean difference in BMI was −0.25 kg/m² (95%CI -0.40 to -0.09). No significant differences were found between single and multiple health behaviour interventions. The authors conclude that school and population based health behaviour intervention can lead to significant reductions in sedentary behaviour, although longer term follow-up is needed to assess the sustainability of such interventions.

This review sought to determine whether, and to what extent, physical activity interventions affect the overall activity levels of children. Thirty studies, with 14,326 participants (range 18 to 2,840) were included in the review (27 RCTs and 3 controlled clinical trials); a subgroup of 6,153 children (range 18 to 1,138) was assessed using accelerometers across these studies. Sixteen studies were judged to be of high quality. Most interventions were school based or conducted in the family home and provided activity or exercise sessions. Meta-analysis across all 30 studies showed a statistically significant effect in favour of the intervention group for both total physical activity (SMD 0.12, 95% CI 0.04 to 0.20) and moderate or vigorous physical activity (SMD 0.16, 95% CI 0.08 to 0.24). These differences were considered to be small and of limited clinical significance. Meta-regression indicated that the pooled intervention effect did not differ significantly between any of the subgroups. The authors conclude that the review provides strong evidence that physical activity interventions have had only a small effect (approximately 4 minutes more walking or running per day) on children’s overall activity levels. It is suggested that this may help to explain why such interventions have had limited success in reducing the body mass index or body fat of children, although these outcomes were not assessed in the review.

This review assessed the effectiveness of interventions aiming to decrease sedentary behaviour. International Journal of Behavioral Nutrition and Physical Activity, 9(1), 61.

Other Systematic Reviews


This review assessed the effectiveness of interventions that focus on reducing sedentary behaviour and BMI. Thirty-four studies (4 controlled trials and 30 RCTs) reporting 33 different interventions, from the USA, Europe and Australasia lasting seven days to four years, were included in the analysis. Follow-up was post-intervention in the majority of studies. Significant reductions in sedentary behaviour and BMI were identified. The post-intervention mean difference in sedentary behaviour was −17.95 min/day (95% CI -26.61 to -9.28) and the change-from-baseline mean difference was −20.44 min/day (95% CI -30.69 to -10.20). The post-intervention mean difference in BMI was −0.25 kg/m² (95%CI -0.40 to -0.09). No significant differences were found between single and multiple health behaviour interventions. The authors conclude that school and population based health behaviour intervention can lead to significant reductions in sedentary behaviour, although longer term follow-up is needed to assess the sustainability of such interventions.


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This review assessed the effectiveness of school-based interventions with a physical activity component on psychological determinants, physical activity, and health outcomes. The review included 129 studies, most of which included at least 250 children and were of between four and 12 months duration. Only ten studies were assessed to be of high quality. Meta-analysis was not possible due to heterogeneity of the studies and the review just presents numbers and/or percentages of studies with or without significant findings without an indication of effect sizes or confidence intervals. Seventy-five studies investigated BMI as a health and fitness outcome and 28% found a positive effect of the intervention, 2.7% found a negative effect and 69.3% found no effect. Seventy-four studies investigated physical activity and 56.8% found a positive effect, 6.8% found a negative effect and 36.4% found no effect. A positive intervention effect was found in 87.5% of 16 studies that investigated knowledge as a psychological determinant. Seven of 16 studies that investigated attitudes reported positive treatment effect and two reported a negative effect, and four of 14 studies that investigated motivation or enjoyment found a positive effect. Studies of adolescents more frequently reported significant differences in BMI between intervention and control group than did studies of children, and studies that combined a physical activity programme with a cognitive approach were more likely to report reduced BMI than studies that investigated an activity intervention only. Low quality studies reported significant results more frequently than studies of moderate or high methodological quality. It is difficult to draw conclusions from this review given the lack of effect sizes or confidence intervals.


This review assessed the effectiveness of interventions that focus on reducing sedentary behaviour (SB) among school-age children. Twelve randomised trials, which lasted at least 12 weeks, aimed at decreasing SB among children aged 6 to 19 years were included. Most of the studies were conducted in school settings. Heterogeneity of the studies meant that a quantitative analysis was not possible. Overall, interventions that focused on decreasing SB were associated with reduction in time spent on SB and/or improvements in anthropometric measurements related to childhood obesity. Several of the studies considered elements related to the potential for translation of the intervention into practice settings. However, only five of the studies incorporated post-intervention follow-up measures, which ranged from 5 to 12 months, so the long-term sustainability of these interventions is unknown.

This review assessed the effectiveness of physical education in promoting participation in physical activity, enjoyment of physical activity and movement skill proficiency in children and adolescents. Twenty-three studies (13 RCTs and 10 controlled trials) were included in the review, 19 of which assessed physical activity participation, four assessed movement skill proficiency, and seven enjoyment of physical activity. Two (of the 10 controlled trials and six of the RCTs were judged to be of high methodological quality. Heterogeneity in interventions and outcomes precluded meta-analysis. The most effective strategies to increase children’s levels of physical activity and improve movement skills in physical education were direct instruction teaching methods and providing teachers with sufficient and ongoing professional development in using these instruction methods. Interventions aimed at improved enjoyment lacked statistical power and were of insufficient quality to draw conclusion. Overall, the lack of effect size and confidence intervals makes it difficult to draw conclusion from the review and further research is needed.


This review assessed active school transport intervention studies with the aim of guiding future research. Fourteen studies (3 RCTs and 11 quasi-experimental/observational design studies) involving at least 10,605 children, were included in the review. Most of the studies were conducted in elementary schools in urban settings in the USA, UK and Australia. The interventions included elements of preparation, promotion, programmes, projects and/or policy strategies. Quality was weak in all the included studies, including problems of confounding, representativeness and validity of outcome measures. Twelve studies reported a 3% to 64% increase in percentage of active transportation to school following the intervention and two studies showed no difference. Interventions with a specific goal seemed to be more effective than interventions with a broader focus. While intervention to increase active transport to school was deemed promising, the evidence base was limited and further research, including long term outcomes to assess sustainability, is needed.


This review assessed levels of metabolic expenditure and changes in activity patterns associated with active video game (AVG) play (e.g. Wi, Sony EyeToy) in children and young people (≤21 years) and to provide directions for future research. Seventeen small studies (11 to 60 participants), including three RCTs, were included in the review. Activity levels during AVG play were highly variable; percentage increases in energy expenditure from rest ranged from 100% to 400% (mean 222%); percentage increases in heart rate from 26% to 98% (mean 64%). Percentage increases in heart rate and energy expenditure were significantly lower for games that primarily used upper body movements compared with those that engaged the lower body (difference, −148%; 95% CI −231% to −66%; p=0.001). Drop-out rates after 12 weeks ranged from zero to 41%. The authors conclude that while AVGs appear to enable light to moderate physical activity, there is limited evidence to draw conclusions on their long-term efficacy for physical activity promotion.

Other Relevant Evidence


These evidence-based guidelines are aimed at all those involved in promoting physical activity among children, including health and education providers, parents and carers. Recommendations are provided on: how to promote the benefits of physical activity and encourage participation; high level strategic planning; the importance of consultation with children and young people and how to set about it; planning and providing spaces, facilities and opportunities; training people to run programmes and activities; and how to promote physically active travel such as cycling and walking. The guidelines were reviewed in 2012 and it was concluded that no update was required.


These evidence-based guidelines (reviewed in 2011) provided recommendations on how to improve the physical environment to encourage physical activity, to all those with influence on or responsibility for the built or natural environment. Seven recommendations cover strategy, policy and plans, transport, public open spaces, buildings and schools, including: ensuring planning applications for new developments always prioritise the need for people (including those whose mobility is impaired) to be physically active as a routine part of their daily life; ensuring pedestrians, cyclists and users of other modes of transport that involve physical activity are given the highest priority when developing or maintaining streets and roads; planning and providing a comprehensive network of routes for walking, cycling and using other modes of transport involving physical activity; and ensuring public open spaces and public paths can be reached on foot, by bicycle and using other modes of transport involving physical activity.

Note: The publications listed were identified using the search methodology outlined in Appendix 1.
CHILDREN OF PARENTS WITH MENTAL ILLNESS AND ALCOHOL AND OTHER ADDICTIONS
In-Depth Topic: Children of Parents with Mental Illness and Alcohol and Other Addictions (COPMIA)

Introduction

The recent report on Health Loss in New Zealand [398] observed that “we are living longer, but not all of this time is spent in good health” and noted that mental health disorders were the third leading condition group contributing 11.1% of health loss. Within this group the main conditions were anxiety and depressive disorders, alcohol use disorders and schizophrenia. Among youth (15–24 years) and young adults (25–44 years) mental disorders were the leading cause of health loss, and for women of reproductive age (15–44 years) over 25% of health loss was attributed to mental disorders.

In New Zealand and internationally, improvements in pharmacotherapy and a move from institutional to community care for adults with mental illness has resulted in an increase in children living with a parent with mental illness [399]. An estimated 15–20% of young people live in families with a parent who has mental illness or addiction [400,401]. Data on the actual number of children living with parents with alcohol or other addictions is scant.

The 2007/08 Alcohol Use in New Zealand Report noted that over one quarter (29%) of women consumed alcohol during their pregnancy [402]. Babies exposed to alcohol in utero are at risk of a range of developmental abnormalities collectively termed Fetal Alcohol Spectrum Disorder. The more severe form is termed Fetal Alcohol Syndrome, and includes growth deficiency, facial anomalies and neurological abnormalities [403]. International reviews estimate that fetal alcohol exposure conservatively affects nearly one in every 100 births [404]. Currently in New Zealand there is no reliable prevalence data for fetal alcohol spectrum disorders or fetal alcohol syndrome.

The risks for children with a mentally ill parent have been recognised for several decades [405] but the children’s needs have often been neglected or hidden. Furthermore, families affected by mental illness are more likely than other families to be affected by other adversity such as financial stress, parental conflict and social isolation [406]. Despite this, services for children of parents with mental health and addiction (COPMIA) in New Zealand are scarce and have only recently received focus from mental health services. Negative impacts on children of parents with mental illness and addiction are not necessarily inevitable, however the psycho-social context of many parents with mental illness put many children at risk of adverse outcomes [407].

This in-depth topic considers the current issues experienced by the children of parents with mental health issues and alcohol and other addictions in New Zealand and identifies evidence-based effective programmes that could be implemented to reduce risk and enhance resilience in these children. The following questions are addressed:

1. How many children and families are affected by having a parent with mental illness and/or addiction in New Zealand?
2. What are the outcomes for these children?
3. What are the optimal services and effective models that could be implemented?
4. What health and support services are currently being implemented to support these children and families internationally?
5. What is the New Zealand situation for services for these children and what are the potential ways forward?
In answering these questions, this in-depth topic is broken into five parts.

1. Part 1 reviews the New Zealand prevalence of children of parents with mental illness and addiction issues.
2. Part 2 reviews the health and support needs of children of parents with mental illness and addiction.
3. Part 3 reviews outcomes for these children including health, development and psycho-social impacts.
4. Part 4 reviews optimal service delivery models from an international perspective based on the best evidence currently available followed by a best practice systems model.
5. Part 5 reviews New Zealand strategies and plans followed by an overview of services for children of parents with mental illness and addiction in New Zealand and the implications for paediatric and adult services for these children.

**The Prevalence of Children of Parents with Mental Illness and Addiction in New Zealand**

Exact data on children of parents with mental illness and addiction in New Zealand is not available. Adult mental health services currently do not routinely collect data or report on the numbers of children of their clients [408]. However, we can obtain some estimates from other surveys.

**Parental mental illness**

The New Zealand General Social Survey (2012) [401] reports that 15% of households with children, have parents with moderate to severe poor mental health. A report from Hutt Community and Mental Health Service (C-AMH) notes that in 2008, 42% of children and adolescents in the CAMH service had a parent with mental illness (6% of those parents were known and receiving treatment via the adult CAMH service, a further 10% were under GP care, 10% were not receiving treatment and 15% did not answer or their status was not known) (Dr H Thabrew, personal communication).

Te Rau Hinengaro: The New Zealand Mental Health Survey 2003/2004, a national household survey of residents aged 16 years and over reported that 39.5% met criteria for a mental disorder (DSM-IV) at any time in their life, and 21% had experienced a disorder in the past 12 months. These disorders included: anxiety, mood, substance use, and eating disorders. Māori and Pacific people had a higher prevalence of disorders and were less likely to attend services for treatment [409]. The 2011/2012 NZ Health Survey reported that 16% of adults aged 15 years and over in New Zealand had been diagnosed with depression, bipolar disorder and/or anxiety in their lifetime. Depression was the most common diagnosis at 14%. Women were 1.7 times more likely than men (20% vs. 12%) to have been diagnosed with a common mental disorder after adjusting for age, with a peak prevalence of diagnosed mental health disorders in women aged 35–44 years of 24%.

The lifetime prevalence of mental disorders (50.7%), and in the past 12 months (29.5%), was higher for Māori. Pacific people also had a higher lifetime prevalence (46.5%) and past 12 month prevalence (25.0%) compared to the overall New Zealand population. In addition, both Māori and Pacific people were less likely than other groups to access treatment when severity was accounted for (9.4%, 8.0% and 12.6% respectively) indicating unmet need for mental health services for Māori and Pacific people [410].

International reviews are consistent with New Zealand data. The World Health Organization World Mental Health Survey Initiative [411] reported an estimated lifetime prevalence of having one or more mental health disorders including anxiety, mood, impulse control and substance use from 12% to 47.4%. Countries reported between one sixth and one third of respondents being affected [411]. The Australian National Survey of Mental Health and Wellbeing reported that 45% of adult Australians have a lifetime mental disorder including anxiety, affective, and substance use disorders, and one in five had a mental disorder in the previous 12 months [412]. About 40% of adults with any 12-month
A 2008 Cochrane Review on Antenatal psychosocial assessment for reducing perinatal mental health morbidity [417] recognises mental health problems associated with pregnancy, childbirth and the first postnatal year as a major public health issue, with up to 15% of childbearing women likely to develop a new episode of major or minor depression between conception and the first three months postpartum [418]. Disorders include minor and major depression, anxiety, post-traumatic stress, bipolar, schizophrenia, and puerperal psychoses. Comorbid disorders are also common in this population with mental illness commonly complicated by drug and alcohol abuse and domestic violence [417].

**Parental Alcohol and Substance Use**

The Alcohol Liquor Advisory Council Drinking Behaviours report 2009–10 [419] reported that the majority of adults (84%) 18 years or older in New Zealand drink alcohol to some extent. There were 21% of adults classified as ‘Binge drinkers’ defined as those who consumed seven or more standard drinks on the last occasion they drank alcohol. In this survey 40% of drinking adults and 45% of binge drinkers lived in families with children aged under 15.

The New Zealand Alcohol and Drug Use Survey 2007/8 reported that among women who had been pregnant in the previous three years, almost 30% had consumed alcohol while pregnant [402]. A recently published study of 723 post-partum women in New Zealand reported that overall 34% of women drank alcohol at some time during their pregnancy, and 12% of pregnancies were at high risk of heavy alcohol exposure early in the pregnancy [420]. The pregnancies that were most at risk were those of younger women, less educated women, Māori women, Pacific women, smokers and drug users. Almost one-quarter of women also continued consuming alcohol after pregnancy recognition [420].

Heavy episodic drinking (binge drinking) is the pattern of drinking most harmful to the fetus [421] although low amounts of alcohol such as one standard drink (10 g alcohol) have been associated with adverse child behaviours [421,422]. The true incidence and prevalence of Fetal Alcohol Spectrum Disorder in New Zealand is unknown. As yet there are no nationally consistent definitions or diagnostic criteria, specific services, or routine screening and preventive protocols to protect the unborn child [423]. The New Zealand Paediatric Surveillance Unit briefly monitored the new incidence of Fetal Alcohol Syndrome (July 1999–December 2001) and found an incidence of 2.9 per 100,000 children aged under 15 years per year [424]. Fetal alcohol syndrome persists progressively through childhood, adolescence and adulthood with lifelong associated physical, mental and behavioural problems [425].

Mental illness and addiction thus affects a considerable proportion of adults at any one time, many of whom are parents. It is likely that 15–20% of children in New Zealand are living in families where parents are affected by mental illness and addiction.

**Health and Support Needs of these Children**

The relationship and impact of parental mental illness on children has been well recognised for several decades [405]. While parental mental illness does not universally lead to adverse effects, mental illness can hinder parenting and parent-child interactions [426], and children are at higher risk of developing psychopathology and adjustment problems [427]. Furthermore, families affected by mental illness are more likely than other families to be affected by other adversity such as financial stress, parental conflict and social isolation [406]. Several studies have also shown that severity and chronicity of parental mental illness may confer additional risk to their children [428]. Added factors that may deter families from seeking adequate support are stigma of mental illness [429,430].
and fear that children may be removed by authorities, which often occurs for mothers with serious mental illness [431]. Parents are also often concerned about the impact of their mental illness on their children and may perceive children’s ‘normal behaviour’ as signs of developing psychological problems due to their illness [432].

Maybery et al. [428] summarizes the needs of children of parents with mental illness:

1. Ensuring that appropriate systems are in place for the identification, assessment, referral and/or intervention for these children from primary health care settings such as GPs or community mental health or welfare settings
2. Involving the other parent or supportive adults/wider family in intervention
3. Educating parents/caregivers regarding attachment, connectedness, impact of illness on children and parenting behaviours
4. Providing support and education to the other parent (without the mental illness) and providing support to enhance relationships between parents
5. Developing a plan to manage the circumstance of ill parent hospitalization
6. Encouraging open and age appropriate discussion and education about parental mental illness
7. Ensuring adequate family financial circumstances

Children of one parent families are at higher risk and will need more support from other supportive adults.

In addition, a review of children’s needs conducted in Tasmania revealed two main themes: their struggle to understand the illness and recognise the signs of mental illness; and managing the illness and the impact of their parent’s hospitalization [427].

Many children act as carers for a parent with mental illness, as well as looking after themselves and younger siblings. This is often a ‘hidden’ or unacknowledged role and there is a need in New Zealand for firstly identifying young carers of parents with mental illness and addiction, and looking at outcomes of caring to identify those whose role is inappropriate or placing them at ‘risk’ [433]. How children are supported or not supported, and helped or not helped to understand their parent’s mental illness can have serious consequences for their own mental health later in life [434].

Outcomes

The impact of parenting behaviours has been suggested as a link between parental psychosocial functioning and child outcomes, with worsening parental mental health and parental conflict associated with lower parenting capacity [435] and children’s internalizing and externalizing problems. Thus there is the potential to minimize risk and enhance resilience through developing individual and family skills and improving social and other supports [399]. Programmes that will be described later are based on this premise. The following section however briefly describes outcomes for children of parents with mental illness, before considering those with parents with alcohol and substance use.

Outcomes for Children of Parents with Mental Illness

Parental depression, particularly maternal depression is associated with a wide range of adverse outcomes for their children including behavioural problems, depressive and emotional disorders and interpersonal difficulties [405,407,436,437]. There is evidence of a genetic link between parental mental illness and that of their offspring for conditions including schizophrenia [438], and major affective disorders [439]. However, researchers have concluded that the family context with multiple risk factors may be more significant than biological vulnerability in accounting for children’s outcomes [440,441]. Thus the genetic contribution and direct effects of parental mental illness may be less harmful to children than the adversity that often accompanies mental illness [437].
Risk and Resilience Factors

A number of reviews discuss factors that increase the risk of psychosocial problems in children of parents with mental illness and those factors that promote resilience [399,407,442,443,444]. Devlin and O'Brien's [399] review usefully describes these factors by different domains: child, family, parental illness, and social factors.

Factors that increase the risk of mental health problems in children of parents with mental illness:

1. Child factors including gender and temperament [405,445,446]
2. Family factors including family break-up; marital and family discord [447]; impaired parenting skills [443]; impaired relationship with mother [448]; and emotional deprivation and neglect
3. Parental illness including involvement in symptomatology (e.g. as part of parent's delusion); chronicity [442]; child's age at time of illness onset [442]; nature of disorder/comorbidity; parental hospitalization (especially if frequent, maternal illness or results in alternative care) [449];
4. Socio-economic and other adversity; and stigma and social isolation.

Conversely, factors that enhance psychosocial resilience in children of parents with mental illness include:

1. Child factors: ability to sustain psychological separation from parental illness; ability to resist over-identification with ill parent; social competence; intellectual competence; and low risk temperament
2. Family factors: effective parenting practices; child has a good relationship with at least one parent; presence of a supportive well other parent; and intact family
3. Parental illness: parental symptomatology does not involve the child; and illness is mild, brief or transient
4. Social factors: external adult role model; quality peer relationships; extended support system; and compensatory social activity.

Perinatal Exposure to Parental Mental Illness

Maternal mental illness can have an adverse impact on the cognitive, emotional, social, and behavioural development of infants [417]. Associations with adverse physical outcomes have also been found in infants and young children [450].

Research has found that antenatal maternal mood impacts on in utero fetal development, with significant associations between the levels of maternal distress during pregnancy and child behavioural outcomes [451,452].

A meta-analytic review reports that maternal depression was significantly related to higher levels of internalizing (e.g. mood, anxiety, or social withdrawal), externalizing (e.g. aggression, conduct disorders, or oppositional defiant disorder), general psychopathology and negative affect/behaviour (e.g. angry, sad, anxious or fearful) in their children although effects were small. The effects were significantly modified by other variables including the severity and duration of parental illness, age of the child and other sociodemographic factors [453]. The younger the child at first exposure to their mother’s depression, the stronger the negative impact. [442]

Children of mothers with post-natal depression show: more behaviour problems in early childhood, especially if the maternal depression persists; lower IQ scores in later childhood; and increased rates of affective disorders in adolescence [454]. Depression in mothers can affect their parenting by being more likely to be inconsistent, ineffective or negatively toned with their children [455]. Although parental treatment for depression is associated with improvements in child psychopathology [456,457], recent reviews have found that the treatment of maternal postnatal depression alone may not be adequate to improve cognitive development, attachment, temperament and other development in
infants and toddlers, but that probably a clear treatment focus on the mother-infant relationship is also required [456,458].

**Childhood and Adolescent Exposure to Parental Mental Illness**

Children who are older when they are first exposed to parental mental illness may have had more years of healthy development and therefore may not be as vulnerable as those exposed at a younger age. In addition, older children are less exclusively dependent on their mothers which may lessen some of the effects of living with a depressed mother as fathers, teachers and peers have more influence. Older children may also be better able to understand parental symptoms and be better able to emotionally and socially regulate and process their situation [453].

**Outcomes for Children of Parents with Alcohol and Substance Use**

Parental substance misuse can have a profound effect on child health and development, with children exhibiting higher rates of externalizing and internalizing problems including conduct disorders, emotional difficulties, underachievement at school, social isolation, antisocial activity, early alcohol and drug use, and ‘precocious maturity’ [459,460]. Both antenatal and postnatal exposure to a mother with substance abuse puts children at high risk for poor outcomes, with mother’s having difficulties in providing nurturing environments, intensified by concomitant economic and social problems. Maternal substance abuse has also been associated with child abuse and neglect [461]. Some of the problems of childhood and adolescence can continue into adulthood including a much higher risk of substance misuse.

As noted earlier, antenatal alcohol use impacts on the developing fetus and may result in Fetal Alcohol Spectrum Disorder or other cognitive and behavioural problems. Even low levels of alcohol consumption are adversely related to child behaviour with externalizing and aggressive behaviours. Moderate to heavy levels of alcohol exposure are associated with higher delinquent behaviour scores, with children 3.2 times more likely to have delinquent behaviour scores in the clinical range compared to non-exposed children [422]. Child cognition is affected for children born of binge drinking mothers.

A review by Broning et al. [462] reported that children of parents with substance abuse problems often have an earlier onset of substance consumption, earlier drunken experiences, increased rates of binge drinking, and are at higher risk of developing substance use disorders themselves. They noted that family issues including relationship problems, conflict, or absence of a supportive parent contribute to the transmission of substance problems to their children. Protective factors include a nurturing parent with good parental attachment, monitoring, and communication of positive family values and expectations.

The Christchurch Health and Developmental Study is a longitudinal study of a birth cohort of 1265 New Zealand children born in 1977. At aged 15 years Fergusson et al. reported on the childhoods of the three percent of adolescents who had multiple problems including conduct disorder, police contact, substance abuse behaviours, early onset of sexual activity, suicidal ideation, mood disorders and lowered self-esteem. These children tended to come from disadvantaged homes with the accumulative risk of parental substance abuse, impaired parenting, family instability and martial conflict adversely impacting the children as adolescents [463].

**Optimal Service Delivery Models**

Given the high prevalence of mental health issues and hazardous alcohol use among parents and the potentially serious consequences for their children, the section which follows reviews the international literature for service delivery models and interventions that may meet the needs of COPMIA. The section is divided into two parts, with the first reviewing interventions specifically developed for children of parents with mental illness, and/or parents with alcohol or other addiction issues. The second part then describes interventions of a more general nature, which may be of benefit to parents and children in
families affected by mental illness and/or addiction but which have not been specifically developed for or evaluated in COPMIA.

**International Models**

**Programmes with a Focus on Children of Parents with Mental Illness**

A review published in 2012 by Reupert et al. [464] on intervention programmes for children whose parents have a mental illness explored programmes from Australia, Europe and North America. They focused on programmes that targeted children aged 5–18 years and collated them into (i) family interventions, (ii) peer-support programmes, (iii) online interventions and (iv) bibliotherapy. They found that the core component across programmes was the provision of psychosocial education to children about mental illness.

(i) **Family interventions** ranged from two to 20 sessions and focused on reducing family dysfunction and enhancing children's support networks and competencies. Overall the evaluations, mainly randomised controlled trials, showed positive results in reducing psychosocial symptoms and improving coping mechanisms.

(ii) **Peer-support programmes** were offered as school holiday programmes, after-school programmes or camps and targeted children aged 7–18 years. They aimed to increase children's knowledge about mental illness and enhance coping skills using a strengths-based preventive approach. To date evaluations of these programmes has not been rigorous and longitudinal data are not often available so outcomes remain unclear. However, evidence shows that the programmes are increasing knowledge about mental illness, enhancing levels of self-esteem and decreasing psychosocial symptoms.

(iii) **Online interventions** targeted older children and young adults and found an increase in knowledge, and self-reported life skills, but one found no change in coping skills. There were only two such programmes reviewed and future evaluations need to focus on child outcomes.

(iv) **Bibliotherapy** uses literature involving characters in a similar position to the children, allowing the children to ‘normalise their situation, gain insight into the problem-solving techniques of those characters and apply this learning to their own lives’. However, a certain level of literacy is required and there is the potential for misinterpretation. To date there is no evidence that bibliotherapy is effective in children of parents with mental illness.

**Parenting Programmes for Children of Parents with Mental Illness Issues**

A systematic review and meta-analysis of preventive interventions in mentally ill parents on the mental health of their offspring by Siegenthaler et al. reported that cognitive, behavioural, or psycho-educational interventions appear to be effective, decreasing the risk on mental health problem in children by 40% [465]. Beardslee et al. [466] reviewed preventive interventions for children of parents with depression and reported on two systematic national programmes (see Table 119 below). Programmes reviewed included:

1. The Effective Child and Family Programme (ECFP), implemented with families with psychiatric problems, substance use issues, physical health problems, and more recently poverty and criminality issues. Under Finnish health and child welfare law, services for adult patients must also attend to the needs of their children [467,468,469]. Included in the methods is a low-threshold intervention “Let’s Talk About Children” described in the table below.

2. In Australia, the COPMIA National Initiative has developed resources to support families where a parent has depression. The Australia COPMIA Initiative has identified primary care settings as a focus for time-limited, evidence-based interventions including The Family Focus intervention based on the Family Talk Intervention (described in the table below). This includes a DVD for families and an online workforce education resource for training mental health professionals which is currently being piloted [466]. Beardslee et al. note that most parents seeking help for depression will present to primary care physicians so key factors for physicians are: recognising
depression; appropriate treatment; understanding children’s concerns and needs; offering psycho-education; providing parental guidance; and follow up.

Table 119: Preventive interventions for children of parents with depression (adapted from Beardslee et al. [466])

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Development and implementation</th>
<th>Target group</th>
<th>Description</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family Talk</td>
<td>Developed in the USA by Beardslee et al. [470] and implemented in the US, Finland, Holland, Sweden, Norway, Costa Rica</td>
<td>Families where a parent has depression</td>
<td>Family Talk (6–11 sessions) and a two-session public health lecture – focusing on providing education to parents.</td>
<td>Long-term RCT showed positive sustained effects for Family Talk and lecture.</td>
</tr>
<tr>
<td>Family group cognitive behavioural intervention</td>
<td>Developed and implemented in the US by Compas et al. [471, 472]</td>
<td>Family groups where a parent has experienced depression</td>
<td>10-sessions family group model seen in parent or family groups. Participants taught to understand and cope with depression</td>
<td>At 24-month follow up significant benefits for parents and children including few depressive episodes in children.</td>
</tr>
<tr>
<td>Family cognitive behavioural preventive intervention</td>
<td>Developed and implemented in the US by Garber et al. [473]</td>
<td>Adolescent groups that have a parent with depression</td>
<td>8-session cognitive behavioural preventive intervention</td>
<td>Fewer depressive episodes in adolescents unless the parent currently depressed. Longer term follow up underway.</td>
</tr>
<tr>
<td>Let’s Talk About Children</td>
<td>Developed and implemented in Finland by Solantaus [467, 468]</td>
<td>Parents/families where a parent has depression/other psychiatric problems</td>
<td>Manual-based, two-session intervention with parents</td>
<td>RCT comparing Family Talk and Let’s Talk About Children – not effective. Family Talk more effective in reducing emotional symptoms.</td>
</tr>
</tbody>
</table>

Research suggests behavioural family interventions founded on social learning models that target family relationships and parenting have the greatest empirical support [474].

Lundahl et al’s meta-analysis of 63 peer-reviewed studies of parent training report that generally, parent training for modifying disruptive child behaviour is effective with moderate effect sizes immediately following treatment. However, parents and children facing higher levels of adversity did not benefit as much as non-disadvantaged families, with parent training the least effective for economically disadvantaged families [475]. Further investigation of factors associated with success among financially disadvantaged families showed that individually delivered behavioural parent training was far better than group delivered training [475].

Home Visiting Programmes for Children at Risk of Adverse Childhood Experiences Including Having Parents with Mental Health and Addiction Issues

Health Families America (Home Visiting for Child Well-being) (HFA) is a home visiting programme for families who are at risk for child abuse, neglect and other adverse childhood experiences. It is designed to work with families who may have mental health and/or substance abuse issues, histories of trauma or intimate partner violence. A review of 33 HFA evaluations by Harding et al. [476] reported consistent positive impacts on parenting outcomes such as parental attitudes but mixed results for other domains including child health and development, maternal life course, and child maltreatment.
A Cochrane Review by Dogget et al. on *Home visits during pregnancy and after birth for women with an alcohol or drug problem* [477] found six studies comparing home visits (mostly after birth) with no home visits. Visitors included health professionals, trained counsellors, paraprofessional advocates and lay visitors. Although individual studies reported a significant reduction in involvement with child protective services, there were no other positive differences found. Many of the studies had methodological concerns. They concluded that there was insufficient evidence to recommend the routine use of home visits for pregnant or postpartum women with a drug or alcohol problem and recommended further large, high-quality trials.

**Programmes for Children of Parents with Substance Abuse Problems**

Broning et al.’s [462] review of preventive programmes for children of substance-affected families including family-based, school-based and one community-based intervention considered outcomes such as knowledge, self-worth, coping and social behaviour. Program-related knowledge increased substantially in all interventions however other outcomes had some promising but mixed results. Family-based programmes showed the value of including children and parents in the intervention compared with school-based interventions where only children were involved. The authors recommend carefully planned programme evaluations and more longitudinal research to evaluate long-term outcomes of programmes.

Niccols et al. [460] review integrated treatment programmes for mothers with substance abuse issues and their children that included on-site pregnancy, parenting, or child-related services with addiction services. They included thirteen studies and reported that most pre-post design studies showed improvements in children’s development, with large effect sizes on improvements of emotional and behavioural functioning. In addition, comparison group studies showed higher scores for development and most growth parameters (length, weight, and head circumference). In addition, integrated programmes were slightly better than non-integrated programmes for emotional and behavioural functioning.

One programme for parents on methadone developed *Focus on Families* (Families Facing the Future), a preventive intervention focused on relapse prevention and parenting skills training to reduce substance use disorders among their children. Shorter-term benefits of the programme trended towards reducing children’s drug use and delinquent behaviour were shown one to two years following the intervention, although the children were still relatively young (age 11 years) at that time [478,479]. Haggerty et al studied the long-term effects of this group parent-training programme 12–14 years after the intervention programme and found a significant reduction in risk of developing a substance use disorder only for males compared to the control group [480].

**Key Points: Evidence-based interventions for supporting children of parents with mental illness and addiction – What works?**

**For young children**
- Good evidence for family interventions including parenting programmes although disadvantaged families may benefit more from individual rather than group parenting programmes.
- Good evidence for integrated treatment programmes including pregnancy, parenting and child services for mothers with substance abuse issues
- Some evidence for home visiting programmes with appropriately trained staff

**For older children and teenagers**
- Some evidence for peer support groups
General programmes that may be beneficial children of parents with mental illness

General Parenting Programmes

Parenting programmes have been shown to have a positive impact on the emotional and behavioural outcomes of children under three years of age [481], and of conduct and behaviour problems in children aged three to 10 years [482], although these reviews were focused on child problems and outcomes rather than parental issues. However, a recent Cochrane review with 48 randomised controlled trials conducted in a wide range of countries and settings including the USA, Australia, Canada, UK, China, Germany, Japan, the Netherlands and New Zealand has shown that group based parenting programmes are effective in improving parental psychosocial wellbeing [483].

A Cochrane review by Barlow et al. on Group-based parent-training programmes for improving emotional and behavioural adjustment in children from birth to three years old found that parenting programmes may impact on parental psychosocial wellbeing by being strengths-based, and aimed at changing parental attitudes and practices in a supportive and non-judgemental manner thus enhancing parental capacity [483]. In addition, parenting programmes may also provide strategies that directly improve parental psychological functioning as well as enhancing their parenting practices, although it must be noted that these reviews do not include programmes provided to parents with clinical mental health or psychiatric problems [483]. The findings also suggest that the benefits are short-term and that additional support to parents may be needed to sustain the improvements.

The text box below reviews a number of parenting programmes which have been evaluated overseas but which are also available in New Zealand.

The Incredible Years Programme

The Incredible Years programme was developed by Clinical Psychologist Dr Webster-Stratton in, Washington. The programme has been extensively researched over the past 30 years, and is now being delivered in many parts of the world, including Europe, Scandinavia, Australasia, and the United States. Evaluations have shown that the programme is effective across a range of ethnicities and cultures [484]. The programme is a parent management training programme developed for parents of children with conduct problems and is based on Social Learning Theory where parents share their experiences in a non-judgemental environment, view video examples and practice new parenting skills in the session. Groups of 10–15 parents with two trained group leaders usually meet for about 2 hours in 8 to 20 weekly sessions. There are specific programmes for different ages including mother and baby; toddler; pre-school and school-age groups.

Triple P Positive Parenting Program

Triple P Positive Parenting Program aims to prevent severe behavioural, emotional and developmental problems in children through improving knowledge, skills and confidence in parenting. Triple P is a multi-level family support intervention of varying intensity and with differing delivery formats that is used in more than 20 countries and has been translated into 18 languages. Triple P was developed by Professor Sanders and colleagues from the Parent and Family Support Centre at the University of Queensland, Australia and has been researched with more 150 international trials and studies showing it to be effective across different family structures, cultures and socio-economic groups. A comprehensive meta-analysis found that the programme was effective across settings, initial severity of problem from mild to severe, and across countries – for child behaviour problems, parenting behaviour, and parental wellbeing [485]. Two reviews with meta-analysis found positive benefits of the programme for severe behavioural difficulties [486,487]. A more recent review article however, challenges the overwhelmingly positive outcomes of the Triple P Parenting Program with concerns that the results have a high risk of bias and ‘no convincing evidence that the interventions work across the whole population or that any benefits are long-term’ [488]. This was strongly refuted by Sanders et al. [489], however a further commentary by Coyne and Kwakkenbos highlight the over-reliance on positive but underpowered Triple P trials that are particular susceptible to risks of bias. [490]. These authors call on clinicians and policymakers to adequately monitor and evaluate Triple P Programs to ensure resources are wisely spent.

Triple P Positive Parenting Program has been adapted by the Australian Central Coast Children and Young People’s Mental Health team to the Mental Health Positive Parenting Programme.
In Depth Topic COPMIA

(MHPPP). It retains the basic sessions and adds two more sessions on ‘The impact of mental health on parenting’ and ‘Children’s fears, friendships and schooling’. It also follows up with four weekly home visits allowing facilitators to give direct assistance to parents to apply their learnt skills, and talk with their children, where appropriate, about their parent’s mental illness. A pilot study and a subsequent before- and after- intervention evaluation found a reduction in the number of dysfunctional parenting strategies and parent-reported child behaviour problems [491].

Parents as First teachers (PAFT)

Parents as First Teachers (PAFT) is a targeted programme enabling eligible families with young children from birth to 3 years to access practical support and guidance. PAFT is a low intensity home visitation programme where parent educators make visits to families to share information, practical ideas and give guidance as the child grows and develops. It was originally developed in Missouri, USA over 20 years ago and has been renamed Parents as Teachers with over 2,600 programmes throughout the USA and in six other countries [492].

Cost Effectiveness of General Parenting Programmes

A systematic review of the cost effectiveness of general parenting programmes by Charles et al. [493] noted that parenting programmes should be recognised as ‘complex interventions’ that therefore need complex evaluations in order to account for the actual effects and potential ripple effects of parenting programmes. Cunningham et al. [494] conducted a randomized trial comparing a large group community-based parenting programme to a clinic-based individual parenting programme. They found that parents of children with severe behaviour problems were more likely to enrol in community-based parenting programmes and that they reported greater improvements in behaviour problems. In addition the community-based programme was six times as cost effective as the clinic/individual programme. A study investigating the cost effectiveness of the Triple-P Positive Parenting Programme implemented at a population level in Queensland Australia reported that it was likely to be worthwhile, although further research is required to confirm the results [495]. An Australian economic analysis of prevention in mental health programmes reported that screening children/adolescents for symptoms of depression with subsequent therapy, and parenting interventions for childhood anxiety prevention are cost effective [496].

Review of Components Associated with General Parent Training Programme Effectiveness

A meta-analytic review by Kaminski et al. [497] of components associated with parent training programme effectiveness noted that following numerous meta-analyses and systematic reviews, parent training approaches to enhance parenting behaviours and skills, and child behaviour and adjustment can be effective. Kaminski et al. then sought to disentangle the various components of parenting programmes to discover which components contributed greater effects. They found that positive parent-child interactions and emotional communication skills, teaching parents to use time out and the importance of parenting consistency, and requiring parents to practice new skills with their children during parent training sessions were associated with larger effects. Smaller effects were found for programme components such as teaching parents problem solving, teaching parents to promote children’s cognitive, academic, or social skills, and providing additional services as part of the parenting programme. They concluded that existing programmes could consider changing, adding or discarding components associated with larger or smaller effects to enhance the effectiveness of the programme.

General Parent Child Interaction Therapy

Parent child interaction therapy is derived from social and developmental theories and is an individualised intervention for children aged 4–7 years with externalizing problems and their parents. It is delivered through initial didactic presentations to parents (usually two sessions) followed by direct coaching sessions of parents with the parent and child in a play therapy room being observed and coached by a therapist through a one-way mirror and a listening device in the ear of the parent [498]. A review and meta-analysis by Thomas et al. [498] evaluated and compared the outcomes of Parent Child Interaction
Therapy and Triple-P Positive Parenting Program. They found moderate to large effects for both standard programmes but smaller effects for abbreviated versions or Media Triple-P.

**General Home Visiting Programmes**

A meta-analytic review by Sweet and Appelbaum of programmes whose primary service delivery strategy was home visits conducted in the United States, reviewed sixty such programmes. Child outcomes included cognitive, socio-emotional, and prevention of child abuse while parent outcomes included enhanced childrearing (including parenting behaviours and attitudes), and enhancement of maternal life course. In general the children and parents in families in home visiting programmes had better outcomes than the control groups. However, using Cohen’s guidelines [499] for interpreting standardized effect sizes where a small effect size was defined as 0.20 or lower, a medium effect size was defined as 0.50, and a large effect size was defined as 0.80 or larger, the effect sizes were small (lower than 0.25) for child outcomes, and even smaller for parent outcomes (lower than 0.14). Further analysis on programme design, populations targeted, and primary programme goals had mixed results.

Fergusson et al. [500] reported on a nine-year follow-up of a home visitation programme (Early Start) randomised trial of 443 families in New Zealand on child abuse, child behaviour, and parental and family-level benefits. They found that families in the Early Start programme showed significant benefits in reduced risk of hospital attendance for unintentional injury, lower risk of parent-reported harsh punishment, lower levels of physical punishment, higher parenting competence scores, and more positive child behavioural adjustment scores. However, the effect sizes were small to moderate (median 0.25), and there were no significant differences on parental behaviour and family outcomes including maternal depression, parental substance use, intimate partner violence, adverse economic outcomes and life stress for families in Early Start compared to control families.

Other reviews have suggested that home visiting programmes can benefit both infant and maternal health, particularly when the home-visitor was a well-trained nurse [501], the visits were frequent, and a trusting relationship was built with coaching on maternal-infant interaction [501,502,503]. One review found the results of home visiting programmes was similar for lay workers and health professionals [503].

**Best Practice Service Models for Children of Parents with Mental Illness and Addiction**

The entry point to services for many children of parents with mental illness will be either through primary care, or secondary adult mental health services.

In 2004, following an extensive literature search and wide-ranging consultations across Australia, the Australian Infant, Child, Adolescent and Family Mental Health Association produced the *Principles and Actions for Services and People Working with Children of Parents with a Mental Illness*. They identified several subgroups of children among families in which a parent has a mental illness including: children who appear ‘well’; children who appear resilient but in need of support; children who are vulnerable and in need of services; and lastly, children who are vulnerable and in need of protection owing to risk of injury [504]. They note that these children may move in any direction along this spectrum of ‘risk’ during their lifetime. Therefore, the challenge to service providers is to:

- ‘Strengthen and support families and children to enhance protective factors that contribute to the parents’ and children’s mental health, and
- Identify and reduce risk factors in parents with a mental illness, their family and community that contribute to their children’s health and wellbeing’.

Although ideally, enhancing mental health and wellbeing should take a broad health promotion approach, the document focuses on actions targeted to the group of the population deemed at higher risk than average.

In view of that target to the higher risk population, the areas of focus for service provision for children of parents with mental illness and addiction were:
• Early identification, in systematically identifying the parental role of many adult mental health service consumers, as well as other groups that may be at higher risk of mental health issues including refugees and migrants
• Family preservation and support for family members
• Addressing grief and loss issues
• Access to information, education and decision-making processes
• Care and protection of children
• Partnerships and cross-agency processes
• Recognition of diversity (culturally and linguistically)
• Workforce development and service reorientation; and research and evaluation

Adapted from Mrazek and Haggerty [505], as outlined in the Australian National Action Plan for Promotion, Prevention and Early Intervention for Mental Health, 2000 [506].

Service Action areas
Service action areas in the document are divided into those provided by individual workers and/or teams, and system responses. The following system responses are taken from the Australian Principles and Actions for Services and People Working with Children of Parents with a Mental Illness [504] as an example of best practice recommendations for services that would be relevant to New Zealand. The development of service requirements for individual workers or teams would also need to be developed for New Zealand use. For further detailed information please see: http://www.copmi.net.au/

System Responses Required for COPMIA Services
(Abbreviated from Principles and Actions for Services and People Working With Children of Parents With a Mental Illness. 2004, Australian Infant Child Adolescent and Family Mental Health Association [504])

Promoting wellbeing and reducing risk
Mental health services and child and family health services can support the identification of risk factors relating to children of parents with a mental illness and the promotion of wellbeing by:
• Putting in place procedures for the identification of the parental role of people who have a mental illness, including ‘expectant’ parents
• Assisting information services to provide appropriate information regarding referral to services for families affected by parental mental illness
• Developing and implementing policies and procedures to support workers in the promotion of the wellbeing of families and the identification and reduction of risk factors for children affected by parental mental illness

Support for families and children
Support for families is enhanced when community service providers, child and family health services, mental health services and child protection services work together to ensure that:
• Parental support groups and parenting skill programmes are available in the community that can respond to the needs of parents with a mental illness
• Planned care and flexible respite care services are available for both children and parents during parental crisis and at other times
• Supported, targeted and evidence-based early intervention programmes of sufficient duration and intensity are available to prevent or minimise the longer term consequences of disrupted or dysfunctional child-parent relationships

Addressing grief and loss issues
To prevent or minimise the feelings of grief and loss often experienced by parents with a mental illness and their family members, mental health services in association with child protection/child welfare services (and the justice sector where applicable) can:
• Ensure policy, practice and procedures recognise and support the importance of secure attachment for infant’s health and future wellbeing
- Provide information, counselling and financial support to informal and formal temporary carers who care for the children during periods of parental illness or as a preventative strategy to maintain the parent’s health

Mental health services (and the justice sector where applicable) can also provide family friendly facilities within adult mental health and justice sectors

**Access to information, education and decision-making**
The education sector, child and family health services and child/youth information services can assist in meeting children’s information needs by:

- Providing information and supporting universal access for children regarding mental health, mental illness and relevant support services which is non-stigmatising and culturally and linguistically appropriate (e.g. via curriculum, help-lines, websites, library resources)

**Care and protection of children**
Adult mental health services can play a key role in the care and protection of their consumer’s children by:

- Supporting family-oriented and family sensitive practice, through workforce development, resource allocation and organizational policy
- Ensuring parents have access to legal advice regarding child protection

The justice sector and child protection services can support children of parents with a mental illness with identified care and protection needs by:

- Ensuring advice/evidence regarding the comprehensive strengths-based assessment of parenting competence of individuals with a mental illness

Mental health services can provide valuable support to child protection services by:

- Working collaboratively and providing mental health expertise to assist in assessment of parenting ability and family capacity where the parent has a mental illness and a child’s safety, development and/or wellbeing are at risk

**Partnerships and cross-agency processes**
Government can facilitate high quality service provision for families and children affected by parental mental illness in partnership with all relevant stakeholders by:

- Developing, supporting and resourcing the implementation of protocols to enhance partnerships between mental health services, community service providers, child protection services, the justice sector, the education sector, families and other key stakeholders regarding enhancement of family and individual mental health and wellbeing in families where a parent has a mental illness and the care and protection of children (where concerns are identified)

**Workforce development and service reorientation**
Children of parents with a mental illness could benefit from the development of workforce standards in the child protection, education sector (student support staff), child and family health and community services areas.

Undergraduate, post-graduate and in-service education and training for those whose work includes the care and protection of children, and those whose work relates to the mental and physical health and wellbeing of children and families (e.g. GPs, teachers, police officers, midwives, childcare workers, paediatricians, child and maternal health nurses, psychiatric trainees, psychologists, social workers, physiotherapists, occupational therapists and speech pathologists). Such support improves outcomes for children of parents with a mental illness when it includes:

- Information regarding the identification of potential risk factors and burdens associated with having a parent with a mental illness
- Education about enhancing and strengthening family wellbeing and how to access supports for children and their families affected by parental mental illness

Parents, children and families affected by parental mental illness would benefit from:

- Increased education of the adult mental health workforce in the area of family-focused and family-sensitive practice, strengths-based approaches and the changing needs of the parent over time
Research and evaluation

To enhance the efficacy and efficiency of services to children of parents with a mental illness, governments and other funding bodies can:

- Request and fund service providers to ensure process and outcome evaluation of programmes developed specifically for children, parents and other carers where the parent has a mental illness.
- Adopt and build upon child and family enhancement and intervention programmes that have been evaluated and found to be both effective and consistent with best practice resource utilisation (including funding and policy development).

Governments can also support research to assist service providers to improve their support, care and protection for children and families where a parent has a mental illness by:

- Identifying factors that enhance positive health outcomes for children and parents.
- Identifying children’s risk status.
- Developing knowledge, tools and mechanisms regarding identification of appropriate levels of intervention for children who appear ‘well’, those who appear to be resilient but in need of support, those who are vulnerable and in need of resources, and those who are vulnerable and in need of protection.
- Identifying effective interventions for children and families using a range of child and family-oriented measures (e.g. schooling attendance and retention, and social connectedness).
- Developing models of effective collaboration between families, child and adolescent and adult mental health services, child protection services and other key stakeholders with the aim of ensuring the safety and wellbeing of children who have a parent with a mental illness.
- Developing information and models to provide culturally appropriate services and information to children and families.

COPMIA Services in New Zealand

Services for children of parents with mental health and addiction in New Zealand are scarce and have only recently received focus from mental health services.

The Ministry of Health notes that there is no consistency in the delivery of services to COPMIA despite pockets of good practice. It is therefore currently working on: establishing New Zealand statistics; DHB/NGO/workforce consultation; and exploration of best practice interventions leading to the development of a national framework.

The Werry Centre for Child and Adolescent Mental Health (Auckland) has been contracted by the Ministry of Health to undertake a stock-take of current COPMIA initiatives in New Zealand; identify expectations regarding COPMIA capability in current competency frameworks; and develop a strategy document for the Ministry of Health and Workforce New Zealand for COPMIA in New Zealand.

There are also a number of strategic frameworks that are driving change in New Zealand with regards to COPMIA. These include: Blueprint II: Mental Health Commission; Service Development Plan: Rising to the Challenge (Ministry of Health); and the White Paper for Vulnerable Children (Ministry of Social Development) all of which lay the groundwork for COPMIA service development.

The following sections outline current New Zealand strategies and plans related to COPMIA; programme examples of current COPMIA services; and based on international evidence and experience, programmes that may have the potential to provide COPMIA services in New Zealand.

New Zealand strategies and plans

*Healthy Beginnings: Developing perinatal and infant mental health services in New Zealand* was published by the Ministry of Health in early 2012 to provide guidance to DHB and other planners, funders and providers of services on ways to address the mental health and addiction needs of mothers and their infants. This comprehensive
document gives an overview of current services in New Zealand including universal preventative, primary care, secondary and tertiary care for perinatal and infant mental health. Guidelines for the development of services are proposed and workforce development issues are discussed. The document also recognises that:

- Perinatal and infant mental health and AOD services cannot be effective unless they are delivered in collaboration with other maternal, family and child health and social services
- Services for Māori will be based on whānau ora – Māori families supported to achieve their maximum health and wellbeing – as the overall vision for Māori health
- It is not desirable or necessary to create a new health ‘silo’ to improve perinatal and infant mental health and AOD services
- The current constrained fiscal environment demands that joining up service provision and sharing resources effectively rather than new funding are required to develop existing services
- Services for infants are less well developed than maternal services and each region will start from a different point.

The guiding principles of Healthy Beginnings are based on those set out by Te Raukura – Mental health and alcohol and other drugs: Improving outcomes for children and youth [512]. The report also notes that currently no DHB is providing the full range of mental health services for mothers and infants that is required.

In addition a range of other publications are relevant to COPMIA in New Zealand.

As early as 1998, New Futures: A strategic framework for specialist mental health services for children and young people in New Zealand [513] recognised that children of parents with severe mental health problems faced a much higher risk for developing mental health problems themselves. The framework was aligned with the New Zealand Mental Health Strategy: Looking Forward (1994) [514] and Blueprint for Mental Health Services in New Zealand (1998) [515] and recognised that some groups of children and young people were ‘lost’ in the current specialist mental health service provision and highlighted the need to focus on developing appropriate consultation and liaison services for children of parents with severe mental health problems [513].

The mental health and addiction strategy Te Tāhuhu: Improving Mental Health (2005–2015) [516] and its associated action plan, Te Kökiri: the Mental Health and Addictions Action Plan 2006–2015 [517] again highlighted the need for services for children of parents with mental illness and/or addiction. The action plan calls for increased services that are funded for children and young people and for a review and update of the framework for child and youth mental health and addiction service provision (New Futures) based on good evidence and best practices, addressing gaps, reflecting specific population needs and considering children of parents, whānau with mental illness and maternal and infant mental health [517]. Te Raukura – Mental health and alcohol and other drugs: Improving outcomes for children and youth [512] was therefore developed, again highlighting the gaps in service for children of parents with a mental illness of addiction.

Blueprint II: How Things Need To Be [518] notes the significant increase in mental health and addiction initiatives since 2005 and introduces a ‘life course’ approach focusing on early responses at key moments in order to have a positive impact. They advocate a priority area focusing on mothers/infants, and children and young people from vulnerable families/whānau with mental health and addiction issues. The companion document Blueprint II: Improving Mental Health and Wellbeing for all New Zealanders – Making Change Happen [400] outlines the priority of ‘Providing a good start’ with a specific action to reduce the impact of parental mental health and addiction issues on infant and child development and to increase access for vulnerable families to effective developmental assessments, parenting support and mental health and addiction responses. They note the need to build workforce capacity for the development of mental health services for children as target access rates have not been achieved, particularly for Māori and Pacific children.
Along with *Blueprint II*, the *White Paper for Vulnerable Children Volume 1* [510] also calls for building on firm foundations including services for primary mental healthcare for mothers suffering from depression, drug and alcohol interventions for parents whose substance abuse puts their child at risk, and parenting and relationship support for struggling parents. The Children’s Action Plan [519] also focuses on identifying and supporting vulnerable children by working across sectors, using evidence-based programmes, and providing a safe and competent workforce.

*Rising to the Challenge: The Mental Health and Addiction Service Development Plan 2012–2017* [509] calls for building resilience and averting future adverse outcomes for infants, children and youth. Among the priority services to be implemented are programmes for children of parents with mental health and addiction issues using reprioritised existing funding or new demographic funding led by DHBs. ‘DHBs will implement and evaluate targeted, group-based psycho-education programmes that provide the children of parents with mental health and addiction issues (COPMIA) with information, peer support and tools to promote resilience, self-esteem and coping strategies. These services will work in conjunction with services that support parents with mental health and addiction issues’ [509].

**Current Programmes in New Zealand that may address COPMIA**

Currently in New Zealand there are only scattered ad hoc services provided specifically for children of parents with mental illness and addiction issues. Some examples are listed in the text box below:

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### Examples of COPMIA Services in New Zealand

#### Supporting Families in Mental Illness – 19 branches throughout New Zealand

**Auckland**

- Supporting Children in Families Where There Is Parental Mental Illness Tu Tangata Tonu – a Pilot Project at the Kari Centre (Child & Adolescent Mental Health Service at Auckland District Health Board) to provide support for children in families where there is a parental mental illness
- Kidzone and Youthzone are peer support and education groups for children and adolescents whose parent or caregiver has a diagnosis of mental illness run by Tu Tangata Tonu for families who reside within the Auckland District Health Board area.
- Kids Club for children aged 8–12 years who have a parent, family or whānau member with mental illness in Mount Eden, Auckland

**Canterbury**

- Familial Trust: a service for families to learn about the effects another’s addiction has had on them
- Stepping Stone Trust: one-on-one support and recreational programmes for children and adolescents of parents with a mental illness; Children Understanding Mental Illness group programme
- Purapura Whetu Trust: a kaupapa Māori mental health services to adults, adolescents, children, and their whānau.

**Think Parent, Think Family (Matua Raki National Addiction Workforce Development)**

Matua Raki is the National Addiction Workforce Development Centre funded by Health Workforce New Zealand (Ministry of Health) whose projects include supporting children of people with mental health and addiction issues in conjunction with the Werry Centre. Matua Raki and Kina Trust have developed a resource for services called ‘Think Parent Think Family’ for responding to parents and their children in alcohol and other drug services. This includes practical ideas and a family inclusive practice organizational checklist. The resource encourages service providers of parents with mental health and addiction problems to welcome children and whānau, and invite parents to share parenting concerns and worries about their children.

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**Other New Zealand programmes of relevance to COPMIA**

A number of other services in New Zealand may be of benefit to children of parents with mental health and alcohol and other addictions. These are outlined in the sections below:
Parenting Programmes
There is a wide range of parenting programmes currently offered throughout New Zealand, with two examples being included in the text box below. In New Zealand, none of these programmes are specifically targeting families with parents with mental health and/or addiction issues.

The Incredible Years Programme
The Incredible Years programme is endorsed by the Ministries of Health, Social Development and Education as a highly effective evidence-based intervention in New Zealand [520]. Training for group leaders is conducted by the Werry Centre with government and community organizations throughout New Zealand offering the Incredible Years programme. Currently the programme is delivered by Ministry of Education, Special Education staff and by 51 non-government organisations (NGOs) contracted to deliver the programme in partnership with the Ministry. Eleven of the NGOs are Whānau Ora providers. Many are also providing a range of social services to families funded through the Ministries of Health or Social Development or other agencies. In 2009, Fergusson et al. [521] reviewed the Incredible Years Basic Parent Programme in New Zealand and concluded that it was an effective and culturally appropriate programme with significant improvements in behaviour and social competence scores and high parental satisfaction. The effects were similar for Māori and non-Māori children and parents. Although these outcomes are very promising, this was a preliminary retrospective review based on agency records and the authors recommend a wait-list randomized design study to fully evaluate the effectiveness of the programme in New Zealand.

Triple P Positive Parenting Program
In New Zealand Triple P Positive Parenting courses are currently being rolled out in four North Island regions (Bay of Plenty, MidCentral, Counties Manukau, and Waitemata) through a government initiative that aims to improve parenting skills and raise awareness of parenting support. The programmes are being implemented as either one-on-one consultations accessed through primary health care, or in discussion groups [250]. The Werry Centre is coordinating this Ministry of Health funded pilot project in conjunction with more intensive parenting supports such as Incredible Years. Qualitative and quantitative evaluation is inbuilt in the pilot project including the training and support of practitioners, delivery of interventions, and family outcomes [522].

Home Visiting and Other Programmes in New Zealand
A number of home visiting programmes are also available in New Zealand with two examples being provided in the text box below.

Parents as First Teachers (PAFT)
The PAFT home visiting and parenting programme began in New Zealand in 1991. Adapted from the original PAFT curriculum, now called ‘Born to Learn’, ‘Ahuru Mowai’ – the Māori dimension, is based on traditional child bearing and rearing beliefs and practices. The programme is managed by the PAFT National Centre, now part of the Ministry of Social Development, and monitors 25 contracts with various organizations who deliver the PAFT programme in 36 locations through New Zealand. The personal visits allow the parent educator to individualise the programme providing parenting and child development information specific to their child. A recent evaluation found a mixed picture of effectiveness with modest impacts noting that poverty and other stressors such as family violence, drug and alcohol abuse and parental mental health would need to be addressed for the parenting programme to have greater effects [523].

Family Start
Family Start is a New Zealand initiative which was first established in 1998 as a family-focused, home-based early intervention programme for the 15% most at-risk families. Its goal is to achieve better wellbeing and development outcomes for the family and children. The programme is designed to deliver an integrated package of services that are relevant to the family over a long duration (up to five years) based on inter-agency collaboration (Ministries of Health, Social Development, and Education, and Child Youth and Family Services). Potential clients are referred to a contracted Family Start provider through an approved referral agency including Lead Maternity Carers, hospital maternity services and Well Child/Tamariki Ora providers from six months before to six months after the baby is born. Family Start takes a strengths-based approach and includes developing needs assessments and case plans; home visiting and coordinating access to services; and information on parenting (Ahuru Mowai/Born to Learn programme). An early evaluation of the Family Start programme had methodological limitations and mixed outcomes [524]. Improvements were subsequently made to strengthen the programme management and outcomes.
Other programmes
Other programmes in New Zealand include: PEPE (Parenting Education Programme) delivered by Plunket; SAGES – older people as mentors; Strategies with Kids, Information for Parents (SKIP); Whānau Toko I Te Ora (WTITO); HIPPY (Home Interaction Programme for Parents and Youngsters); Anau Ako Pasifika; Whanāu Toko/Te Ora; Parents Inc; Barnados; Presbyterian Support Services; Parents Centres; and Early Start.

Some of these programmes, including all the key government funded parenting programmes, were evaluated by the Families Commission in 2005 [525]. They noted that all communities have some access to parent support and development services, however there are gaps in services within specific communities (for example, small rural communities). They concluded that parenting programme effectiveness was very difficult to determine given the range of delivery mechanisms, content of the programmes, engagement of parents and other adversities such as chronic stress and struggle to meet basic needs that may hinder parental involvement in such programmes. They recommended more rigorous evaluations to generate further knowledge about the effectiveness of parenting programmes in New Zealand.

Vulnerable Pregnant Women’s Programmes
Some District Health Boards have set up Vulnerable Pregnant Women’s programmes to provide wrap-around services to ensure that every pregnancy has the healthiest outcome for both mother and baby. An evaluation of the Hawke’s Bay District Health Board Vulnerable Pregnant Women’s Multidisciplinary Team [526] noted the need for interagency collaboration to facilitate solutions to complex issues that pregnant women may face including socio-economic, health, community and cultural issues.

Whānau Ora
Whānau Ora is an interagency approach to providing health and social services empowering whānau as a whole rather than focusing separately on individual family members. Whānau Ora providers provide support to whānau to make plans to improve their lives working with hapū, iwi or a non-government organisation. Other whānau can receive wrap-around services specifically tailored to their needs from specialist Whānau Ora providers. These whānau will have a ‘navigator’ to work with them to identify their needs, develop a plan to address those needs and facilitate their access to health and social services. These services will include mental health and addiction services and include initiatives for youth mental health through the Prime Minister’s Youth Mental Health project. A whānau ora approach will also be taken for addressing mental health services for mothers and infants [511].

Universal Programmes: Well Child Tamariki Ora
Well Child/Tamariki Ora is a free national service for all New Zealand children from birth to five years. The programme offers screening through regular wellness checks, health promotion and education, health protection and support. The first ‘well baby’ check is provided by the lead maternity carer, who then facilitates the choice of a Well Child provider for the remainder of the checks. Well Child providers include: Plunket; Māori health providers; Pacific Island health providers; public health services including public health nurses or community workers; or a general practice team [527]. Plunket is the largest provider of Well Child services providing care for about 92% of babies born in New Zealand. Plunket also delivers Parents as First Teachers and PEPE (Parenting Education Programme) in locations throughout New Zealand [528].

A recent report by the Families Commission on Early-Intervention Support and Vulnerable Families and Whānau [529] included families and whānau with mental health problems and multiple other needs as ‘vulnerable’. They noted that vulnerable families and whānau are less likely to take up services on offer and more likely to drop out, although trusted relationships with outreach workers, case workers or home visitors could often successfully ‘bridge’ these families to access services.

Access to universal services such as Well Child/Tamariki Ora would allow for screening and identification of families with needs without stigma. They note that although group-
based parent education programmes are usually recommended, there is evidence that individual-based programmes can be more effective where the family needs are complex [475].

Ways Forward for Children of Parents with Mental Illness and Addiction in New Zealand

New Zealand has recognised the need for services for children of parents with mental illness and addiction for the last 15 years. Various strategies and action plans have further highlighted the gaps and Blueprint II [518] outlines the priority of providing a good start with specific action to reduce the impact of parental mental health and addiction issues on infant and child development, and to increase access for vulnerable families to effective developmental assessments, parenting support, and mental health addiction responses. The Children’s Action Plan [519] also calls for identification of vulnerable children and integrated services to meet their needs.

Mental Health and Addiction services in New Zealand recognise the high prevalence of co-morbidity. People with addiction problems also have a high prevalence of mental health problems and vice versa, so an entrance point via any service is needed to ensure they receive comprehensive support for all their problems. Mental health disorders are more common among Māori and Pacific people [530]. Mental health and addiction disorders are also more common among those with the least education, lowest household incomes, high unemployment rates, and low access to services [531]. Te Rau Matatini, the Māori health workforce agency is a key player in ensuring workforce development and strengthening to particularly address the mental health and addiction needs of Māori to improve outcomes for their children [532].

Rising to the Challenge: The Mental Health and Addiction Service Development Plan 2012–2017 [509] calls for priority services to be implemented for children of parents with mental health and addiction issues led by DHBs. ‘DHBs will implement and evaluate targeted, group-based psycho-education programmes that provide the children of parents with mental health and addiction issues (COPMIA) with information, peer support and tools to promote resilience, self-esteem and coping strategies. These services will work in conjunction with services that support parents with mental health and addiction issues’.

Applying the evidence and international experience of the most effective services, the following best practice system response recommendations for DHBs to consider are outlined in the text box below.

Best Practice System Responses for DHBs to Consider

Early identification: systematically identifying the parental role of many adult mental health service consumers, and other groups that may be at higher risk of mental health issues

- Identification of babies and young children at primary health care level through universal programmes such as Well Child/Tamariki Ora, Māori and Pacific health providers, and general practice teams antenatally and in early childhood
- Identification of children at secondary and tertiary health care levels through adult and child mental health services
- Identification of the needs for children who appear ‘well’; children who appear resilient but in need of support; children who are vulnerable and in need of services; and children who are vulnerable and in need of protection owing to risk of injury. Recognise that these children may move in any direction along this spectrum of ‘risk’
- Clear referral pathways to support services for those children identified

Family preservation and support for family members

- Support, targeted and evidence-based early intervention programmes of sufficient duration and intensity being available to prevent or minimise the longer term consequences of disrupted or dysfunctional child-parent relationships
- Parenting skill programmes that could be tailored for parents with mental illness and addiction issues for example Incredible Years, Triple P Positive Parenting Program
- Family home-visiting programmes that could be tailored for parents with mental illness and addiction issues for example Family Start, Parents as First Teachers
- Identify psychosocial factors which increase the health risks often associated with parents with a mental illness (e.g. poverty and social isolation) which also impact on their children, and advocate for action to address these issues
- Facilitate collaborative multi-agency support for families according to risk and need for example through Whānau Ora
- Ensure policy, practice and procedures recognise and support the importance of secure attachment for infant’s health and future wellbeing

**Access to information, education and decision-making processes**
- Provide information and support universal access for children regarding mental health, mental illness and relevant support services which is non-stigmatising and culturally and linguistically appropriate (via curriculum, help-lines, websites, library resources, child/teen support groups)
- Provide education for relevant support staff regarding parental mental illness, its potential impact on children and age-appropriate responses, resources and supports that may be required by children where a parent has a mental illness

**Care and protection of children**
- Support family-oriented and family sensitive practice, through workforce development, resource allocation, organisational policy, and services that meet the needs of Māori and Pacific families.
- Ensure parents have access to legal advice regarding child protection
- Work collaboratively and provide mental health expertise to assist in assessment of parenting ability and family capacity where the parent has a mental illness and a child’s safety, development and/or wellbeing are at risk

**Partnerships and cross-agency processes**
- Develop, support and resource the implementation of protocols to enhance partnerships between mental health services, community service providers, child protection services, the justice sector, the education sector, families and other key stakeholders regarding enhancement of family and individual mental health and wellbeing in families where a parent has a mental illness and/or addiction and the care and protection of children (where concerns are identified)
- Establish, build upon and implement local protocols, formal linkages, coordination and provision of education across all sectors involved with children of parents with a mental illness and/or addiction to enable agencies to identify and respond appropriately, flexibly and at the earliest opportunity to children and families who would benefit from support
- Establish communication processes within the mental health sector, across agencies and in partnership with families, to ensure coordinated support, assessment (as required) and care planning for families
- Work with disability and key addiction services (drug, alcohol and gambling) to ensure a coordinated approach to parents with co-morbidities and their families

**Recognition of diversity (culturally and linguistically)**
- Improve access to culturally appropriate information for families, provided in a range of languages, on the services available to support families in which a parent has a mental illness
- Develop culturally appropriate services to meet needs

**Workforce development and service reorientation**
- Develop workforce standards in child protection, the education sector, child and family health and community services for working with children of parents with mental illness and addiction
- Develop undergraduate, post-graduate and in-service education and training for those whose work includes the care and protection of children, and those whose work relates to the mental and physical health and wellbeing of children and families (e.g. GPs, teachers, police officers, midwives, childcare workers, paediatricians, child and maternal health nurses, psychiatric trainees, psychologists, social workers, community workers) to support improved outcomes for children of parents with a mental illness and/or addiction
Research and evaluation

- Ensure process and outcome evaluation of programmes developed specifically for children, parents and other carers where the parent has a mental illness and/or addiction
- Adopt and build upon child and family enhancement and intervention programmes that have been evaluated and found to be both effective and consistent with best practice resource utilisation (including funding and policy development)

Conclusions

Although the issues of children of parents with mental health and addiction in New Zealand have been raised over the last 15 years and are embedded in high level strategy and policy documents, current services are scarce. There is a lack of a well-planned national service, including an agreed service model to ensure the needs of these children are met. The systematic identification and assessment of the needs of COPMIA and referral to appropriate service does not currently occur. The actual numbers of children who need support are not known, however estimates from prevalence rates of mental health and addiction problems indicate that 15–20% of children live in households where a parent has mental health and/or addiction issues.

The international literature suggests that services required to best support children of parents with mental health and addiction will include: early identification in systematically identifying the parental role of many adult mental health service consumers, as well as other groups that may be at higher risk of mental health issues; family preservation and support for family members; addressing grief and loss issues; access to information, education and decision-making processes; care and protection of children; partnerships and cross-agency processes; recognition of diversity (culturally and linguistically); workforce development and service reorientation; and research and evaluation.

Family interventions that are the most likely to benefit children of parents with mental health and addiction include evidence-based effective parenting and home visiting programmes, and peer support programmes. The positive impacts resulting from these programmes are considered to be cost effective. Yet, COPMIA services also need family support to address the adverse socio-economic determinants of health and wellbeing commonly associated with parental mental illness and addiction. Current parenting and family support services available for all families in need including COPMIA include a range of parenting programmes funded by Ministries of Health, Social Development and Education, all of which show some positive impacts. However, these must be rigorously evaluated to ensure this most vulnerable group of children are receiving the best support available to enhance their health and wellbeing. The proportion of parents with mental illness and addiction currently accessing parenting support services is not known.

Initiatives to support parents with mental health and addiction issues to achieve broader socio-economic goals are also recognised as a priority. Initiatives to support parents into employment are now being trialled and Whānau Ora is showing some promise.

Realising the gaps, the Ministry of Health is beginning to focus on this important issue by establishing New Zealand data on COPMIA, consulting with DHBs and non-government agencies on workforce capabilities and requirements, and exploring best practice interventions in order to develop a national framework for addressing COPMIA. The Ministry of Health has contracted the Werry Centre for Child and Adolescent Mental Health to provide advice on agreed models of service that will need to be funded at a level appropriate to meet demand.
APPENDICES AND REFERENCES
One of the features of this reporting series is the inclusion of sections which briefly review local policy documents (e.g. Ministry of Health Strategies and Toolkits) and international evidence-based reviews that are relevant to the prevention and or management of child and youth health issues. The approaches taken in these sections borrow heavily from the principles of the Evidence-Based Medicine (EBM) movement, which has emerged in recent years as a means of providing busy clinicians with up to date overviews of the evidence in particular areas [533,534]. Such overviews generally rely on reviewers collating all of the available evidence (published and unpublished trials, observational studies etc.), evaluating it in a rigorous manner, and then publishing the resulting synthesis of the evidence in a format which allows clinicians to evaluate quickly the effectiveness of the intervention(s) reviewed. While the evidence base for population level interventions is much less developed than that for individual patient therapies (as such interventions often have longer follow up times, more diffuse outcomes, and less readily identifiable “control” groups [535]), there is nevertheless a reasonable body of evidence emerging about the effectiveness of specific population level interventions.

The brief overviews presented in this report therefore aim to provide busy DHB staff with a logical starting point from which to consider the types of interventions available to address particular child and youth health issues. In preparing these overviews the methodology used was not exhaustive but rather involved searching a number of EBM journals and databases (e.g. the Cochrane Library) as well as Ovid MEDLINE and PubMed for systematic reviews of population level interventions in child and youth health (see Text Box below).

In addition New Zealand or other websites relevant to the topic were assessed and included if they were thought to be useful to DHB or clinical staff working in that area.

### Methodology Used in Preparing Policy/Evidence-Based Review Sections

**New Zealand (Health) Policy Documents**

Each review section aims to provide an overview of Ministry of Health (or where appropriate, other Government Agency) policy documents and strategies relevant to the area. The Ministry of Health’s website (http://www.moh.govt.nz/moh.nsf) was searched for key documents. All identified documents were then reviewed and the most relevant summarised, focusing on those which provided strategic guidance to DHBs on the prevention/population level management of the issues in question.

**Evidence-Based and Other Reviews**

The five databases listed below were searched for reviews considering the effectiveness of population level interventions to prevent and/or manage each of the issues in question. While this list is not exhaustive, the databases were selected on the basis of the calibre of the institutions publishing the reviews. In addition, the search strategy concentrated on publications which attempted to synthesise all of the available evidence, thereby providing as broad as possible coverage of the relevant literature. In general, only literature from 2005 onwards was searched, with a focus on literature published since 2010. Earlier publications were included if there was a paucity of more recent information. While individual trials and protocols were not specifically sought, if there was no other relevant information available, an attempt was made to locate individual research reports or recommendations. While they are not totally comprehensive, it is nevertheless hoped that these brief overviews will provide a useful starting point for DHBs wishing to explore strategies to address particular child and youth health issues.

**Evidence Based Medicine Reviews:** This database allows seven EBM resources to be searched at once including The Database of Reviews of Effects (DARE), Health Technology Assessments (HTA) and the NHS Economic Evaluation Database (NHSEED) all produced by National Health Services’ Centre for Reviews and Dissemination at the University of York, U.K., The Cochrane Database of Systematic Reviews, and the ACP Journal Club.

**National Guideline Clearinghouse:** http://www.guideline.gov/ This is a searchable database of evidence-based clinical practice guidelines maintained by the Agency for Healthcare Research and Quality in the United States.

**Centre for Reviews and Dissemination (CRD):** This is a Department of the University of York and is part of the National Centre for Health Research (NCHR) (http://www.york.ac.uk/inst/crd/). While CRD produces the database of Review Effects (DARE), captured in the Evidence Based Medicine Review Database, searching the CRD site identifies other reviews not captured by DARE. This database is available through most local...
library services. The CRD provides a commentary on many of the reviews in their database. Where there were a number of relevant reviews on a topic identified, those which the CRD considered to have been well conducted and to have reliable conclusions were given priority for inclusion in this report.

National Institute for Health and Clinical Excellence (NICE): This is an independent organisation based in the United Kingdom which provides national guidance on the promotion of good health and the prevention and treatment of ill health. (http://www.nice.org.uk/)

Guide to Community Preventive Services: Systematic Reviews and Evidence Based Recommendations: This guide was developed by the non-federal Task Force on Community Preventive Services whose members are appointed by the Director of the Centre for Disease Control and Prevention (CDC). The Community Guide summarises what is known about the effectiveness, economic efficiency, and feasibility of interventions to promote community health and prevent disease (http://www.thecommunityguide.org/about/).

While undertaking this task it quickly became apparent that the quality of evidence varied considerably depending on the issue reviewed. In addition, in many cases, the research provided reasonably strong guidance about what did not work (for example, current evidence suggests additional social support is ineffective in preventing preterm birth in high-risk women), but little advice on effective interventions.

Thus in many cases these brief overviews serve to highlight the current paucity of evidence on population level interventions to address child and youth health needs (although the absence of systematic/other reviews does not rule out the existence of individual studies in particular areas). In this context, the search strategy utilised did not primarily aim to identify individual studies or reviews of individual patient therapies. In cases where such studies were identified and where no other systematic reviews were available, they were included under the heading of “Other Relevant Publications”. In such cases the reader needs to be aware that these studies were identified in a non-systematic manner and that their findings should therefore not be given the same weight as systematic reviews (e.g. Cochrane reviews) where all of the available evidence has been rigorously evaluated. The evidence-based review tables also include some topical New Zealand research publications.
Appendix 2: Statistical Significance Testing and its Use in this Report

Understanding Statistical Significance Testing

Inferential statistics are used when a researcher wishes to use a sample to draw conclusions about the population as a whole (e.g. weighing a class of 10 year old boys, in order to estimate the average weight of all 10 year old boys in New Zealand). Any measurements based on a sample however, even if drawn at random, will always differ from that of the population as a whole, simply because of chance. Similarly, when a researcher wishes to determine whether the risk of a particular condition (e.g. lung cancer) is truly different between two groups (smokers and non-smokers), they must also consider the possibility that the differences observed arose from chance variations in the populations sampled.

Over time, statisticians have developed a range of measures to quantify the uncertainty associated with random sampling error (e.g. to quantify the level of confidence we can have that the average weight of boys in our sample reflects the true weight of all 10 year old boys, or that the rates of lung cancer in smokers are really different to those in non-smokers). Of these measures, two of the most frequently used are:

P values: The p value from a statistical test tells us the probability that we would have seen a difference at least as large as the one observed, if there were no real differences between the groups studied (e.g. if statistical testing of the difference in lung cancer rates between smokers and non-smokers resulted in a p value of 0.01, this tells us that the probability of such a difference occurring if the two groups were identical is 0.01 or 1%. Traditionally, results are considered to be statistically significant (i.e. unlikely to be due to chance) if the probability is <0.05 (i.e. less than 5%) [536].

Confidence Intervals: A 95% Confidence Interval suggests that if you were to repeat the sampling process 100 times, 95 times out of 100 the confidence interval would include the true value. In general terms, if the 95% confidence intervals of two samples overlap, there is no significant difference between them (i.e. the p value would be ≥0.05), whereas if they do not overlap, they can be assumed to be statistically different at the 95% confidence level (i.e. the p value would be <0.05) [536].

The Use of Statistical Significance Testing in this Report

In the preparation of this report a large range of data sources were used. For the purposes of statistical significance testing however, these data sources can be considered as belonging of one of two groups: Population Surveys and Routine Administrative Datasets. The relevance of statistical testing to each of these data sources is described separately below:

Population Surveys: A number of indicators in this report utilise data derived from national surveys (e.g.2011/12 New Zealand Health Survey), where information from a sample has been used to make inferences about the population as a whole. In this context statistical significance testing is appropriate, and where such information is available in published reports, it has been incorporated into the text accompanying each graph or table (i.e. the words significant, or not significant in italics are used to imply that a test of statistical significance has been applied to the data and that the significance of the associations are as indicated). In a small number of cases however information on statistical significance was not available in published reports, and in such cases any associations described do not imply statistical significance.

Numbers and Rates Derived from Routine Administrative Data: A large number of the indicators in this report are based on data derived from New Zealand’s administrative datasets (e.g. National Minimum Dataset, National Mortality Collection), which capture
information on all of the events occurring in a particular category. Such datasets can thus be viewed as providing information on the entire population, rather than a sample and as a consequence, 95% confidence intervals are not required to quantify the precision of the estimate (e.g. the number of leukaemia deaths in 2003–2007 although small, is not an estimate, but rather reflects the total number of deaths during this period). As a consequence, 95% confidence intervals have not been provided for any of the descriptive data (numbers, proportions, rates) presented in this report, on the basis that the numbers presented are derived from the total population under study.

Rate Ratios Derived from Routine Administrative Data: In considering whether statistical significance testing is ever required when using total population data Rothman [537] notes that if one wishes only to consider descriptive information (e.g. rates) relating to the population in question (e.g. New Zealand), then statistical significance testing is probably not required (as per the argument above). If, however, one wishes to use total population data to explore biological phenomena more generally, then the same population can also be considered to be a sample of a larger super-population, for which statistical significance testing may be required (e.g. the fact that SIDS in New Zealand is 10 times higher in the most deprived NZDep areas might be used to make inferences about the impact of the socioeconomic environment on SIDS mortality more generally (i.e. outside of New Zealand, or the 5 year period concerned)). Similarly, in the local context the strength of observed associations is likely to vary with the time period under study (e.g. in updating 5-year asthma admission data from 2004–2008 to 2005–2009, rate ratios for Pacific children are likely to change due to random fluctuations in annual rates, even though the data utilised includes all admissions recorded for that particular 5-year period). Thus in this report, whenever measures of association (i.e. rate ratios) are presented, 95% confidence intervals have been provided on the assumption that the reader may wish to use such measures to infer wider relationships between the variables under study [537].

The Signalling of Statistical Significance in this Report
In order to assist the reader to identify whether tests of statistical significance have been applied in a particular section, the significance of the associations presented has been signalled in the text with the words *significant*, or *not significant* in italics. Where the words *significant* or *not significant* do not appear in the text, then the associations described do not imply statistical significance or non-significance.
APPENDIX 3: THE NATIONAL MINIMUM DATASET

Mode of Data Collection

The National Minimum Dataset (NMDS) is New Zealand’s national hospital discharge data collection and is maintained by the Ministry of Health. The information contained in the dataset has been submitted by public hospitals in a pre-agreed electronic format since 1993. Private hospital discharges for publicly funded events (e.g. births, geriatric care) have been submitted since 1997. The original NMDS was implemented in 1993, with public hospital information back loaded to 1988 [538]. Information contained in the NMDS includes principal and additional diagnoses, procedures, external causes of injury, length of stay and sub-specialty code and demographic information such as age, ethnicity and usual area of residence.

Dataset Quality and Changes in Coding Over Time

There are a number of key issues which must be taken into account when interpreting information from the NMDS. Many of these issues arise as a result of regional differences in the way in which data are coded and uploaded to the NMDS. These include:

1. Inconsistencies in the way in which different providers upload day cases to the NMDS, and how this has changed over time.
2. The changeover from the ICD-9 to ICD-10 coding system, and irregularities in the way in which diagnoses and procedures are allocated ICD codes.
3. Changes in the way in which ethnicity information has been collected over time and across regions (Appendix 6).

The following sections discuss the first two if these issues, while the third is discussed in Appendix 6, which reviews the way in which ethnicity information is collected and coded within the health sector.

1. Inconsistencies in the Uploading of Day-Cases to the NMDS

One of the key issues with time series analysis using hospital discharge data is the variability with which different providers upload day cases to the NMDS. Day cases are defined as cases that are admitted and discharged on the same day, with the “three hour rule” (treatment time >3 hours) traditionally being utilised to define an admission event. In contrast, patients who spend at least one (mid)night in hospital are classified as inpatients irrespective of their length of stay [539].

In the past, there have been significant regional variations in the way in which different providers have uploaded their day cases to the NMDS, leading to problems with both time series analysis and regional comparisons. These inconsistencies have included

1. During the mid 1990’s, a number of providers began to include A&E events as day cases if the total time in the Emergency Department (including waiting time) exceeded 3 hours, rather than uploading only those whose actual treatment time exceeded 3 hours [539]. NZHIS provided feedback which rectified this anomaly and since January 1995 the correct procedure has been used (these additional cases were coded using medical and surgical sub-specialty codes and are thus difficult to filter out using traditional Emergency sub-specialty filters).
2. Over time, a number of providers have become more efficient at recording the time of first treatment within the Emergency Department (rather than time of attendance) and thus during the late 1990s and early 2000s have become more efficient in identifying emergency department cases which meet the 3-hour treatment rule and are thus eligible to be uploaded to the NMDS. This has resulted in a large number of additional cases being uploaded to the NMDS, particularly in the upper North Island.
3. In addition, some providers admit cases to their short stay observation units while other providers do not, leading to regional variations in the appearance of day cases in the NMDS [540].

**Previous Attempts to Address Inconsistent Uploading at the Analytical Stage**

When producing their annual Hospital Throughput reports, the Ministry of Health has adopted the following filter to ensure regional and time series comparability with respect to day patient admissions [540]. In its analyses it excludes all cases where:

1. the admission and discharge date are the same (length of stay = 0), and
2. the patient was discharged alive, and
3. the health specialty code on discharge is that of Emergency Medicine (M05, M06, M07, and M08).

While this coding filter succeeds in ensuring a degree of comparability between regions and across time (although it fails to correct the anomalies occurring during the mid 1990s when A&E cases were uploaded using medical sub-specialty codes), the exclusion of emergency day cases from time series analysis has a number of limitations including:

1. Exclusion of only those with a length of stay of 0 days means that those emergency cases who begin their treatment late at night and are discharged in the early hours of the following morning (up to a quarter of emergency cases have a length of stay of 1 day in some DHBs) are included as genuine hospital admissions, whereas those who begin their treatment early in the morning and are discharged late in the afternoon or the evening of the same day are excluded.

2. With a move towards the development of specialist paediatric emergency departments in larger urban centres (e.g. Auckland), there remains the possibility that some larger DHBs are now seeing and treating a number of acute medical patients within the emergency setting, while in regional centres similar patients continue to be assessed on the paediatric medical ward/assessment unit and thus receive a paediatric medical specialty code. The exclusion of all emergency presentations from time series and sub-regional analysis may thus differentially exclude a large portion of the workload occurring in large urban centres where access to specialist advice and treatment is available within the Emergency Department setting.

The potential impact of inconsistent uploading of day cases to the NMDS is likely to be greatest for those conditions most commonly treated in the emergency department setting. Analysis of 2001–2003 hospital admission data suggests that more than a third of NMDS emergency department discharges for those aged 0–24 years were due to injury, with another third due to ambulatory sensitive conditions (e.g. asthma, gastroenteritis, respiratory infections). In contrast, only 2% of those presenting with bacterial meningitis and 4% of those with septic arthritis were discharged with an emergency sub-specialty code.

Further sub-analysis of these two admission categories however demonstrated that inclusion/exclusion of emergency department admissions had quite different effects depending on the category of admission under study (injury vs. ambulatory sensitive admissions) and whether the region had access to a specialist Paediatric Emergency Department. In this analysis the Wider Auckland Region, (comprising one third of the NZ population and whose residents have access to specialist Paediatric Emergency Departments) was compared to the rest of NZ. For ambulatory sensitive admissions, exclusion of emergency department cases resulted in Auckland's admission rates being consistently lower than in the rest of New Zealand. It was only when emergency cases were included in this analysis that Auckland’s admission rates began to approximate those of the rest of NZ. In contrast for injuries, inclusion of emergency department cases resulted in hospital admissions in the Auckland Region consistently exceeding the rest of New Zealand. It was only when emergency cases were excluded from the analysis that Auckland’s injury admission rates began to approximate those of the rest of NZ. (These findings occurred despite Auckland having a similar proportion of children living in the most deprived NZDep small areas as the rest of NZ).
Loosely interpreted, the findings of this analysis suggest that the workload of large specialist Paediatric Emergency Departments must not be discounted when examining trends in ambulatory sensitive or other medical admissions, as it is only when emergency cases are included in the analysis that the admission rates of the Wider Auckland Region (with its access to specialist Paediatric Emergency care) begin to approximate the rest of NZ. In contrast, it is possible that specialist Paediatric Emergency Departments have much less of an influence on admission thresholds for injury, with these being handled in a similar manner by different emergency departments across the country. Thus for injury data, the greater tendency for some emergency departments to upload their cases to the NMDS must be taken into account in any analysis.

**Implications for Interpreting Time Series Analyses in these Reports**

Throughout this report, analysis of time series and other information has been undertaken using unfiltered hospital admission data. The exceptions are the injury and poisoning sections where emergency department discharges have been filtered out of the dataset in an attempt to address some of the inconsistencies discussed above. Despite such an approach, there remains the potential for the inconsistent uploading of day cases to significantly influence the time series analyses presented in this report. In particular, such practices may lead to an over estimate of the number of medical admissions commonly treated in the emergency department setting (e.g. asthma, skin infections, respiratory tract infections), while at the same time the filtering out of injury and poisoning emergency cases may lead to undercounting for a number of more minor types of injury. Nevertheless, the filtering processes used in this report are thought to provide the best balance when considering hospital admissions amongst those 0–24 years. Despite this, the reader must bear in mind that a potential for significant residual bias remains, when interpreting the time series analyses presented in this report.

2. **Data Quality and Coding Changes over Time (ICD-9 and ICD-10)**

**Change Over from ICD-9 to ICD-10 Coding**

From 1988 until June 1999, clinical information in the NMDS was coded using versions of the ICD-9 classification system (ICD-9 CM until June 1995, then ICD-9-CM-A until June 1999). From July 1999 onwards, the ICD-10-AM classification system has been used, although for time series analysis, back and forward mapping between the two classification systems is possible fusing pre-defined algorithms [538].

The introduction of ICD-10-AM represents the most significant change in the International Classification of Diseases (ICD) in over 50 years and uses an alphanumeric coding system for diseases in which the first character of the code is always a letter followed by several numbers. This has allowed for the expansion of the number of codes to provide for recently recognised conditions and to provide greater specificity about common diseases (there are about 8,000 categories in ICD-10-AM as compared to 5,000 in ICD-9). While for most conditions there is a reasonable 1:1 correspondence between ICD-9 and ICD-10 codes, for some this may lead to some irregularities in time series analysis [541]. Where possible such irregularities will be highlighted in the text, although care should still be taken when interpreting time series analysis across the 1999–2000 period as some conditions may not be directly comparable between the two coding systems.

**Accuracy of ICD Coding**

In recent years the Ministry of Health has undertaken a number of reviews of the quality of ICD coding in the NMDS. In the latest audit 2,708 events were audited over 10 sites during a 3 month period during 2001/2002. Overall the audit found that 22% of events required a change in coding, although this also included changes at the fourth and fifth character level. The average ICD code change was 16%, with changes to the principal diagnosis being 11%, to additional diagnoses being 23% and to procedure coding being 11%. There were 1625 external causes of injury codes, of which 15% were re-coded differently [542]. These findings were similar to an audit undertaken a year previously.

While the potential for such coding errors must be taken into consideration when interpreting the findings of this report, it may be that the 16% error rate is an overestimate,
as in the majority of the analyses undertaken in this report, only the principal diagnosis (with an error rate of 11%) is used to describe the reason for admission. In addition, for most admissions the diagnostic category (e.g. lower respiratory tract infections) is assigned using information at the 3 digit level (with the 16% error rate also including issues with coding at the 4th or 5th digit level).

3. Ethnicity Information in the NMDS
The reader is referred to Appendix 6 for a discussion of this issue.

Conclusion
In general the inconsistencies outlined above tend to make time series and (regional) comparative analyses based on the NMDS less reliable than those based on Mortality or Birth Registration data (where legislation dictates inclusion criteria and the type of information collected). While hospital discharge data still remains a valuable and reasonably reliable proxy for measuring the health outcomes of children and young people in this country, the reader is cautioned to take into consideration the biases discussed above, when interpreting the findings outlined in this report.
APPENDIX 4: THE BIRTH REGISTRATION DATASET

Mode of Data Collection

Since 1995 all NZ hospitals and delivering midwives have been required to notify Internal Affairs (within 5 working days of delivery), of the birth of a live or stillborn baby 20+ weeks gestation or weighing >400g. Prior to 1995, only stillborn babies reaching 28+ weeks of gestation required birth notification. Information on the hospital’s notification form includes maternal age, ethnicity, multiple birth status, and baby’s sex, birth weight and gestational age. In addition, parents must complete a Birth Registration Form within two years of delivery, duplicating the above information with the exception of birth weight and gestational age, which are supplied only on hospital notification forms. Once both forms are received by Internal Affairs, the information is merged into a single entry. This two-stage process it is thought to capture 99.9% of births occurring in New Zealand and cross-checking at the receipting stage allows for the verification of birth detail [543].

Interpretation of Information Derived from the Birth Registration Dataset

Because of the two-stage birth registration process, the majority of variables contained within the birth registration dataset are >98% complete, and cross-checking at the receipting stage (with the exception of birth weight and gestational age) allows for the verification of birth details. In addition, the way in which ethnicity is collected in this dataset confers a number of advantages, with maternal ethnicit

y being derived from the information supplied by parents on their baby’s birth registration form. This has the advantage of avoiding some of the ambiguities associated with hospital and mortality data, which at times have been reported by third parties. Changes in the way ethnicity was defined in 1995 however make information collected prior to this date incomparable with that collected afterwards. For births prior to 1995, maternal ethnicity was defined by ancestry, with those having half or more Māori or Pacific blood meeting ethnic group criteria, resulting in three ethnic groups, Māori, Pacific and non-Māori non-Pacific. For births after 1995 maternal ethnicity was self identified, with an expanded number of ethnic categories being available and parents being asked to tick as many options as required to show which ethnic group(s) they belonged to. For those reporting multiple ethnic affiliations a priority rating system was introduced, as discussed Appendix 6 of this report.

Because this dataset captures 99.9% of births occurring in NZ, is >98% complete for most variables, collects self-reported ethnicity in a standard manner and is collated and coded by a single agency, information derived from this dataset is likely to be of higher quality than that derived from many of NZ’s other data sources. Limitations however include the relatively restricted number of variables contained within the dataset (e.g. it lacks information on maternal smoking, BMI or obstetric interventions) and the lack of cross-checking for birth weight and gestational age (which is supplied only on the hospital notification form). The changeover in ethnicity definition during 1995 also prohibits time series analysis by ethnicity over the medium to long term. Finally, since the last report, the Ministry of Health has stopped providing stillbirth data in the Birth Registration Dataset, and thus all analyses based on this set are restricted to live births only. Each of these factors must thus be taken into account when interpreting information in this report that has been derived from the Birth Registration Dataset.
APPENDIX 5: NATIONAL MORTALITY COLLECTION

Mode of Data Collection
The National Mortality Collection is a dataset managed by the Ministry of Health which contains information on the underlying cause(s) of death as well as basic demographic data for all deaths registered in New Zealand since 1988. Data pertaining to fetal and infant deaths are a subset of the Mortality Collection, with cases in this subset having additional information on factors such as birth weight and gestational age [544].

Each month the Births, Deaths and Marriages service of the Department of Internal Affairs sends the Ministry of Health electronic death registration information, Medical Certificates of Cause of Death, and Coroner’s reports. Additional information on the cause of death is obtained from the National Minimum Dataset (NMDS), private hospital discharge returns, the NZ Cancer Registry (NZCR), the Department of Courts, the Police, the Land Transport Authority (LTSA), Water Safety NZ, Media Search and from writing letters to certifying doctors, coroners and medical records officers in public hospitals. Using information from these data sources, an underlying cause of death (ICD-10-AM) is assigned by Ministry of Health staff using the World Health Organization’s rules and guidelines for mortality coding [545].

Data Quality Issues Relating to the National Mortality Collection
Unlike the NMDS, where information on the principal diagnosis is coded at the hospital level and then forwarded electronically to the Ministry of Health, in the National Mortality Collection each of the approximately 28,000 deaths occurring in New Zealand each year is coded manually by Ministry of Health staff. For most deaths the Medical Certificate of Cause of Death provides the information required, although coders also have access to the information contained in the NMDS, NZ Cancer Registry, LTSA, Police, Water Safety NZ and ESR [545]. As a consequence, while coding is still reliant on the accuracy of the death certificate and other supporting information, there remains the capacity for a uniform approach to the coding which is not possible for hospital admissions data.

While there are few published accounts of the quality of coding information contained in the National Mortality Collection, the dataset lacks some of the inconsistencies associated with the NMDS, as the process of death registration is mandated by law and there are few ambiguities as to the inclusion of cases over time. As a consequence, time series analyses derived from this dataset are likely to be more reliable than that provided by the NMDS. One issue that may affect the quality of information derived from this dataset however is the collection of ethnicity data, which is discussed in more detail in Appendix 6 of this report.
APPENDIX 6: MEASUREMENT OF ETHNICITY

The majority of rates calculated in this report rely on the division of numerators (e.g., hospital admissions, mortality data) by Statistics NZ Estimated Resident Population denominators. Calculation of accurate ethnic-specific rates relies on the assumption that information on ethnicity is collected in a similar manner in both the numerator and the denominator, and that a single child will be identified similarly in each dataset. In New Zealand this has not always been the case, and in addition the manner of collecting information on ethnicity has varied significantly over time. Since 1996 however, there has been a move to ensure that ethnicity information is collected in a similar manner across all administrative datasets in New Zealand (Census, Hospital Admissions, Mortality, Births). The following section briefly reviews how information on ethnicity has been collected in national data collections since the early 1980s and the implications of this for the information contained in this report.

1981 Census and Health Sector Definitions

Earlier definitions of ethnicity in official statistics relied on the concept of fractions of descent, with the 1981 census asking people to decide whether they were fully of one ethnic origin (e.g. Full Pacific, Full Māori) or if of more than one origin, what fraction of that ethnic group they identified with (e.g. 7/8 Pacific + 1/8 Māori). When prioritisation was required, those with more than 50% of Pacific or Māori blood were deemed to meet the ethnic group criteria of the time [546]. A similar approach was used to record ethnicity in health sector statistics, with birth and death registration forms asking the degree of Pacific or Māori blood of the parents of a newborn baby/the deceased individual. For hospital admissions, ancestry-based definitions were also used during the early 1980s, with admission officers often assuming ethnicity, or leaving the question blank [547].

1986 Census and Health Sector Definitions

Following a review expressing concern at the relevance of basing ethnicity on fractions of descent, a recommendation was made to move towards self-identified cultural affiliation. Thus the 1986 Census asked the question “What is your ethnic origin?” and people were asked to tick the box or boxes that applied to them. Birth and death registration forms however, continued to use the “fractions of blood” question until 1995, making comparable numerator and denominator data difficult to obtain [546]. For hospital admissions, the move from an ancestry-based to a self-identified definition of ethnicity began in the mid-80s, although non-standard forms were used and typically allowed a single ethnicity only [547].

1991 Census and Health Sector Definitions

A review suggested that the 1986 ethnicity question was unclear as to whether it was measuring ancestry or cultural affiliation, so the 1991 Census asked two questions:

1. Which ethnic group do you belong to? (tick the box or boxes which apply to you)
2. Have you any NZ Māori ancestry? (if yes, what iwi do you belong to?)

As indicated above however, birth and death registrations continued with ancestry-based definitions of ethnicity during this period, while a number of hospitals were beginning to use self-identified definitions in a non-standard manner [547].

1996 Census and Health Sector Definitions

While the concepts and definitions remained the same as for the 1991 census, the ethnicity question in the 1996 Census differed in that:

- The NZ Māori category was moved to the top of the ethnic categories
- The 1996 question made it more explicit that people could tick more than one box
- There was a new “Other European” category with 6 subgroups

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As a result of these changes, there was a large increase in the number of multiple responses, as well as an increase in the Māori ethnic group in the 1996 Census [546]. Within the health sector however, there were much larger changes in the way in which ethnicity information was collected. From late 1995, birth and death registration forms incorporated a new ethnicity question identical to that in the 1996 Census, allowing for an expansion of the number of ethnic groups counted (previously only Māori and Pacific) and resulting in a large increase in the proportion of Pacific and Māori births and deaths. From July 1996 onwards, all hospitals were also required to inquire about ethnicity in a standardised way, with a question that was compatible with the 1996 Census and that allowed multiple ethnic affiliations [547]. A random audit of hospital admission forms conducted by Statistics NZ in 1999 however, indicated that the standard ethnicity question had not yet been implemented by many hospitals. In addition, an assessment of hospital admissions by ethnicity over time showed no large increases in the proportions of Māori and Pacific admissions after the 1996 “change-over”, as had occurred for birth and death statistics, potentially suggesting that the change to a standard form allowing for multiple ethnic affiliations in fact did not occur. Similarities in the number of people reporting a “sole” ethnic group pre- and post-1996 also suggest that the way in which information on multiple ethnic affiliations was collected did not change either. Thus while the quality of information available since 1996 has been much better than previous, there remains some concern that hospitals continue to undercount multiple ethnic identifications and as a result, may continue to undercount Pacific and Māori peoples [547].

2001 Census and Health Sector Definitions

The 2001 Census reverted back to the wording used in the 1991 Census after a review showed that this question provided a better measure of ethnicity based on the current statistical standard [546]. The health sector also continued to use self-identified definitions of ethnicity during this period, with the Ethnicity Data Protocols for the Health and Disability Sector providing guidelines which ensured that the information collected across the sector was consistent with the wording of the 2001 Census (i.e. Which ethnic groups do you belong to (Mark the space or spaces that apply to you)?)

2006 Census and Health Sector Definitions

In 2004, the Ministry of Health released the Ethnicity Data Protocols for the Health and Disability Sector [548] with these protocols being seen as a significant step forward in terms of standardising the collection and reporting of ethnicity data in the health sector [549]. The protocols stipulated that the standard ethnicity question for the health sector was the 2001 Census ethnicity question, with respondents being required to identify their own ethnicity, and with data collectors being unable to assign this on respondent’s behalf, or to transfer this information from another form. The protocols also stipulated that ethnicity data needed to be recorded to a minimum specificity of Level 2 (see below) with systems needing to be able to store, at minimum, three ethnicities, and to utilise standardised prioritisation algorithms, if more than three ethnic groups were reported. In terms of outputs, either sole/combination, total response, or prioritised ethnicity needed to be reported, with the methods used being clearly described in any report [548].

The following year, Statistics New Zealand’s Review of the Measurement of Ethnicity (RME), culminated in the release of the Statistical Standard for Ethnicity 2005 [550], which recommended that:

1. The 2006 Census ethnicity question use identical wording to the 2001 Census
2. Within the “Other” ethnic group, that a new category be created for those identifying as “New Zealander” or “Kiwi”. In previous years these responses had been assigned to the European ethnic group
3. All collections of official statistics measuring ethnicity have the capacity to record and report six ethnicity responses per individual, or at a minimum, three responses when six could not be implemented immediately
4. The practice of prioritising ethnicity to one ethnic group should be discontinued.
At the 2006 Census however, a total of 429,429 individuals (11.1% of the NZ population) identified themselves as a New Zealander, with further analysis suggesting that 90% of the increase in those identifying as New Zealanders in 2006, had arisen from those identifying as New Zealand European at the 2001 Census [551]. In 2009 Statistics NZ amended the Standard to reflect these issues [552] with the current recommendation being that future Censuses retain the current ethnicity question (i.e. that New Zealander tick boxes not be introduced) but that alongside the current standard outputs where New Zealander responses are assigned to the Other Ethnicity category, an alternative classification be introduced which combines the European and New Zealander ethnic groups into a single European and Other Ethnicity category for use in time series analysis (with those identifying as both European and New Zealanders being counted only once in this combined ethnic group [552].

The Current Recording of Ethnicity in New Zealand’s National Datasets

In New Zealand’s national health collections (e.g. National Minimum Dataset, Mortality Collection and NZ Cancer Registry), up to three ethnic groups per person are stored electronically for each event, with data being coded to Level 2 of Statistics New Zealand’s 4-Level Hierarchical Ethnicity Classification System [538]. In this Classification System increasing detail is provided at each level. For example [548]:

- Level 1 (least detailed level) e.g. code 1 is European
- Level 2 e.g. code 12 is Other European
- Level 3 e.g. code 121 is British and Irish
- Level 4 (most detailed level) e.g. code 12111 is Celtic

Māori however, are identified similarly at each level (e.g. Level 1: code 2 is Māori vs Level 4: code 21111 is Māori).

For those reporting multiple ethnic affiliations, information may also be prioritised according to Statistics New Zealand’s protocols, with Māori ethnicity taking precedence over Pacific > Asian/Indian > Other > European ethnic groups [548]. This ensures that each individual is counted only once and that the sum of the ethnic group sub-populations equals the total NZ population [547]. The implications of prioritisation for Pacific groups however are that the outcomes of those identifying as both Māori and Pacific are only recorded under the Māori ethnic group.

For those reporting more than 3 ethnic affiliations, the ethnic groups recorded are again prioritised (at Level 2), with Māori ethnicity taking precedence over Pacific > Asian/Indian > Other > European ethnic groups (for further details on the prioritisation algorithms used see [548]). In reality however, less than 0.5% of responses in the National Health Index database have three ethnicities recorded, and thus it is likely that this prioritisation process has limited impact on ethnic-specific analyses [548].

Undercounting of Māori and Pacific Peoples in National Collections

Despite significant improvements in the quality of ethnicity data in New Zealand’s national health collections since 1996, care must still be taken when interpreting the ethnic-specific rates presented in this report, as the potential still remains for Māori and Pacific children and young people to be undercounted in our national data collections. In a review that linked hospital admission data to other datasets with more reliable ethnicity information (e.g. death registrations and Housing NZ Corporation Tenant data), the authors of Hauora IV [553] found that on average, hospital admission data during 2000–2004 undercounted Māori children (0–14 years) by around 6%, and Māori young people by around 5–6%. For cancer registrations, the undercount was in the order of 1–2% for the same age groups. While the authors of Hauora IV developed a set of adjusters which could be used to minimise the bias such undercounting introduced when calculating population rates and rate ratios, these (or similar) adjusters were not utilised in this report for the following reasons:

1. Previous research has shown that ethnicity misclassification can change over time, and thus adjusters developed for one period may not be applicable to other periods [554].
2. Research also suggests that ethnic misclassification may vary significantly by DHB [554], and thus that adjusters developed using national level data (as in Hauora IV) may not be applicable to DHB level analyses, with separate adjusters needing to be developed for each DHB.

Further, as the development of adjusters requires the linkage of the dataset under review with another dataset for which more reliable ethnicity information is available, and as this process is resource-intensive and not without error (particularly if the methodology requires probabilistic linkage of de-identified data), the development of a customised set of period and age specific adjusters was seen as being beyond the scope of the current project. The reader is thus urged to bear in mind that the data presented in this report may undercount Māori and Pacific children to a variable extent (depending on the dataset used) and that in the case of the hospital admission dataset for Māori, this undercount may be as high as 5–6%.

**Ethnicity Classifications Utilised in this Report and Implications for Interpretation of Results.**

Because of inconsistencies in the manner in which ethnicity information was collected prior to 1996, all ethnic-specific analysis presented in this report are for the 1996 year onwards. The information thus reflects self-identified concepts of ethnicity, with Statistics NZ’s Level 1 Ethnicity Classification being used, which recognises 5 ethnic groups: European (including New Zealander), Māori, Pacific, Asian (including Indian) and Other (including Middle Eastern, Latin American and African). In order to ensure that each health event is only counted once, prioritised ethnic group has been used unless otherwise specified.
APPENDIX 7: NZ DEPRIVATION INDEX

The NZ Deprivation Index (NZDep) is a small area index of deprivation, which has been used as a proxy for socioeconomic status in this report. The main concept underpinning small area indices of deprivation is that the socioeconomic environment in which a person lives can confer risks/benefits which may be independent of their own social position within a community [555]. They are thus aggregate measures, providing information about the wider socioeconomic environment in which a person lives, rather than about their individual socioeconomic status.

The NZDep was first created using information from the 1991 census, but has since been updated following each census. The NZDep2006 combines 9 variables from the 2006 census which reflect 8 dimensions of deprivation (Table 120). Each variable represents a standardised proportion of people living in an area who lack a defined material or social resource (e.g. access to a car, income below a particular threshold), with all 9 variables being combined to give a score representing the average degree of deprivation experienced by people in that area. While the NZDep provides deprivation scores at meshblock level (Statistics NZ areas containing approx 90 people), for the purposes of mapping to national datasets, these are aggregated to Census Area Unit level (≈1,000–2,000 people). Individual area scores are then ranked and placed on an ordinal scale from 1 to 10, with decile 1 reflecting the least deprived 10% of small areas and decile 10 reflecting the most deprived 10% of small areas [556].

Table 120. Variables used in the NZDep2006 Index of Deprivation [557]

<table>
<thead>
<tr>
<th>No</th>
<th>Factor</th>
<th>Variable in Order of Decreasing Weight in the Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Income</td>
<td>People aged 18–64 receiving means tested benefit</td>
</tr>
<tr>
<td>2</td>
<td>Employment</td>
<td>People aged 18–64 unemployed</td>
</tr>
<tr>
<td>3</td>
<td>Income</td>
<td>People living in households with income below an income threshold</td>
</tr>
<tr>
<td>4</td>
<td>Communication</td>
<td>People with no access to a telephone</td>
</tr>
<tr>
<td>5</td>
<td>Transport</td>
<td>People with no access to a car</td>
</tr>
<tr>
<td>6</td>
<td>Support</td>
<td>People aged &lt;65 living in a single parent family</td>
</tr>
<tr>
<td>7</td>
<td>Qualifications</td>
<td>People aged 18–64 without any qualifications</td>
</tr>
<tr>
<td>8</td>
<td>Owned Home</td>
<td>People not living in own home</td>
</tr>
<tr>
<td>9</td>
<td>Living Space</td>
<td>People living in households below a bedroom occupancy threshold</td>
</tr>
</tbody>
</table>

The advantage of NZDep is its ability to assign measures of socioeconomic status to the elderly, the unemployed and to children (where income and occupational measures often don’t apply), as well as to provide proxy measures of socioeconomic status for large datasets when other demographic information is lacking. Small area indices have limitations however, as not all individuals in a particular area are accurately represented by their area’s aggregate score. While this may be less of a problem for very affluent or very deprived neighbourhoods, in average areas, aggregate measures may be much less predictive of individual socioeconomic status [555]. Despite these limitations, the NZDep has been shown to be predictive of mortality and morbidity from a number of diseases in New Zealand.
## APPENDIX 8: CONGENITAL ANOMALY CODES

Table 121. ICD-10-AM Congenital Anomaly Coding Used in this Report (Table 1 of 2)

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q00</td>
<td>Anencephaly</td>
</tr>
<tr>
<td>Q01</td>
<td>Encephalocele</td>
</tr>
<tr>
<td>Q02</td>
<td>Microcephaly</td>
</tr>
<tr>
<td>Q03</td>
<td>Congenital Hydrocephalus</td>
</tr>
<tr>
<td>Q04</td>
<td>Other Brain Malformations</td>
</tr>
<tr>
<td>Q05</td>
<td>Spina Bifida</td>
</tr>
<tr>
<td>Q06</td>
<td>Other Spinal Cord Malformations</td>
</tr>
<tr>
<td>Q07</td>
<td>Other CNS Malformations</td>
</tr>
<tr>
<td>Q10</td>
<td>Eyelid/Lacrimal/Eye/Orbit Malformations</td>
</tr>
<tr>
<td>Q16</td>
<td>Ear Malformations Impairing Hearing</td>
</tr>
<tr>
<td>Q170</td>
<td>Accessory Auricle</td>
</tr>
<tr>
<td>Q171</td>
<td>Other Ear Malformations</td>
</tr>
<tr>
<td>Q18</td>
<td>Other Face/Neck Malformations</td>
</tr>
<tr>
<td>Q20</td>
<td>Malformations Cardiac Chambers/Connections</td>
</tr>
<tr>
<td>Q210</td>
<td>Ventricular Septal Defect</td>
</tr>
<tr>
<td>Q211</td>
<td>Atrial Septal Defect</td>
</tr>
<tr>
<td>Q212</td>
<td>Atrioventricular Septal Defect</td>
</tr>
<tr>
<td>Q213</td>
<td>Tetralogy of Fallot</td>
</tr>
<tr>
<td>Q214</td>
<td>Other Cardiac Septal Malformations</td>
</tr>
<tr>
<td>Q22</td>
<td>Pulmonary/Tricuspid Valve Malformations</td>
</tr>
<tr>
<td>Q23</td>
<td>Aortic/Mitral Valve Malformations</td>
</tr>
<tr>
<td>Q24</td>
<td>Other Heart Malformations</td>
</tr>
<tr>
<td>Q250</td>
<td>Patent Ductus Arteriosus</td>
</tr>
<tr>
<td>Q251</td>
<td>Malformations Great Arteries Excluding PDA</td>
</tr>
<tr>
<td>Q26</td>
<td>Malformations Great Veins</td>
</tr>
<tr>
<td>Q27</td>
<td>Other Peripheral Vascular Malformations</td>
</tr>
<tr>
<td>Q28</td>
<td>Other Circulatory Malformations</td>
</tr>
<tr>
<td>Q30</td>
<td>Nose Malformations</td>
</tr>
<tr>
<td>Q31</td>
<td>Larynx Malformations</td>
</tr>
<tr>
<td>Q32</td>
<td>Trachea/Bronchus Malformations</td>
</tr>
<tr>
<td>Q33</td>
<td>Lung Malformations</td>
</tr>
<tr>
<td>Q34</td>
<td>Other Respiratory Malformations</td>
</tr>
<tr>
<td>Q35</td>
<td>Cleft Palate</td>
</tr>
<tr>
<td>Q36</td>
<td>Cleft Lip</td>
</tr>
<tr>
<td>Q37</td>
<td>Cleft Palate and Lip</td>
</tr>
<tr>
<td>Code</td>
<td>Description</td>
</tr>
<tr>
<td>-------</td>
<td>-------------------------------------------------------------------</td>
</tr>
<tr>
<td>Q38–45</td>
<td>Other Congenital Malformations of the Digestive System</td>
</tr>
<tr>
<td>Q381</td>
<td>Ankyloglossia Tongue Tie</td>
</tr>
<tr>
<td>Q380, Q382–Q388</td>
<td>Tongue/Mouth/Pharynx Malformations</td>
</tr>
<tr>
<td>Q39–Q40</td>
<td>Oesophagus/Upper Alimentary Malformations</td>
</tr>
<tr>
<td>Q41–Q43</td>
<td>Intestinal Malformations</td>
</tr>
<tr>
<td>Q44–Q45</td>
<td>Other Digestive Malformations</td>
</tr>
<tr>
<td>Q50–56</td>
<td>Malformations of the Genital Organs</td>
</tr>
<tr>
<td>Q50–Q52</td>
<td>Female Genital Malformations</td>
</tr>
<tr>
<td>Q53</td>
<td>Undescended Testicle</td>
</tr>
<tr>
<td>Q54</td>
<td>Hypospadias</td>
</tr>
<tr>
<td>Q55</td>
<td>Other Male Genital Malformations</td>
</tr>
<tr>
<td>Q56</td>
<td>Indeterminate Sex/Pseudohermaphroditism</td>
</tr>
<tr>
<td>Q60–64</td>
<td>Malformations of the Urinary System</td>
</tr>
<tr>
<td>Q60</td>
<td>Renal Agenesis/Reduction Defects</td>
</tr>
<tr>
<td>Q61</td>
<td>Cystic Kidney Disease</td>
</tr>
<tr>
<td>Q62</td>
<td>Renal Pelvis Obstruction/Ureter Malformations</td>
</tr>
<tr>
<td>Q63–Q64</td>
<td>Other Kidney/Urinary Malformations</td>
</tr>
<tr>
<td>Q65–79</td>
<td>Malformations of the Musculoskeletal System</td>
</tr>
<tr>
<td>Q650–Q652</td>
<td>Congenital Dislocation Hip</td>
</tr>
<tr>
<td>Q653–Q655</td>
<td>Congenital Subluxation Hip</td>
</tr>
<tr>
<td>Q656, Q658–Q659</td>
<td>Other Deformities Hip</td>
</tr>
<tr>
<td>Q66</td>
<td>Foot Deformities</td>
</tr>
<tr>
<td>Q67–Q68, Q79</td>
<td>Other Musculoskeletal Malformations</td>
</tr>
<tr>
<td>Q69</td>
<td>Polydactyly</td>
</tr>
<tr>
<td>Q70</td>
<td>Syndactyly</td>
</tr>
<tr>
<td>Q71–Q74</td>
<td>Reduction Defects/Other Limb Malformations</td>
</tr>
<tr>
<td>Q75–Q76</td>
<td>Skull/Facial Bones/Spine/Thorax Malformations</td>
</tr>
<tr>
<td>Q77–Q78</td>
<td>Osteochondrodysplasia</td>
</tr>
<tr>
<td>Q80–89</td>
<td>Other Congenital Malformations</td>
</tr>
<tr>
<td>Q80</td>
<td>Ichthyosis</td>
</tr>
<tr>
<td>Q81</td>
<td>Epidermolysis Bullosa</td>
</tr>
<tr>
<td>Q825</td>
<td>Non-Neoplastic Naevus</td>
</tr>
<tr>
<td>Q820–Q824, Q828–Q829</td>
<td>Other Skin Malformations</td>
</tr>
<tr>
<td>Q83</td>
<td>Breast Malformations</td>
</tr>
<tr>
<td>Q84</td>
<td>Other Integument Malformations</td>
</tr>
<tr>
<td>Q85–Q87, Q89</td>
<td>Other Malformations</td>
</tr>
<tr>
<td>Q90–99</td>
<td>Chromosomal Abnormalities</td>
</tr>
<tr>
<td>Q90</td>
<td>Down Syndrome</td>
</tr>
<tr>
<td>Q91</td>
<td>Edwards and Patau Syndromes</td>
</tr>
<tr>
<td>Q92</td>
<td>Other Autosomal Trisomies</td>
</tr>
<tr>
<td>Q93, Q95</td>
<td>Monosomies and Autosomal Deletions/Other Rearrangements</td>
</tr>
<tr>
<td>Q96</td>
<td>Turners Syndrome</td>
</tr>
<tr>
<td>Q97</td>
<td>Other Sex Chromosome Anomalies Female Phenotype</td>
</tr>
<tr>
<td>Q98</td>
<td>Sex Chromosome Anomalies Male Phenotype</td>
</tr>
<tr>
<td>Q99</td>
<td>Other Chromosome Anomalies</td>
</tr>
</tbody>
</table>

Table 122. ICD-10-AM Congenital Anomaly Coding Used in this Report (Table 2 of 2)
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