TE OHONGA AKE

THE HEALTH OF MĀORI CHILDREN AND YOUNG PEOPLE WITH CHRONIC CONDITIONS AND DISABILITIES IN NEW ZEALAND

SERIES TWO

Te Ohonga Ake

The Health of Māori Children and Young People with Chronic Conditions and Disabilities in New Zealand Series Two

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Te Ohonga Ake

The literal translation of Te Ohonga Ake is the Awakening. In the context of this report it refers to an awakening towards the reality of Māori child and youth health status in New Zealand. While many of us have been acutely aware of poor outcomes for Māori children and young people in this country, this report confirms our concerns and provides strong evidence for everyone to wake up, pay attention and take action to improve the lives of our most precious asset, our mokopuna.

Cover Artwork: *Whakapapa*—Family Tree by Coree Te Whata-Col

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INTRODUCTION

EDITORIAL: MĀORI CHILDREN AND YOUNG PEOPLE WITH CHRONIC CONDITIONS AND DISABILITIES—ISSUES AND PRIORITIES

Associate Professor Joanne Baxter

This report, *Māori Children and Young People with Chronic Conditions and Disabilities,* contains a wealth of information. The report's focus on disability, chronic disease and overweight and obesity encompasses health issues and conditions impacting on many Māori babies, tamariki and rangatahi. In addition, these conditions often have a broader impact on education, on whānau and across the life course.

This editorial provides an overview of selected findings from this report, and positions these findings within the context of previous Māori child and youth health reports produced by the Child and Youth Epidemiology Service. The focus is on conditions with marked ethnic inequality, that have a high impact and/or are areas where there are gaps in knowledge.

Issues and priorities identified

Section 1: Conditions Arising in the Perinatal Period

This section commences with findings for fetal death and for preterm birth among Māori babies. Analysis shows that for Māori babies there were higher rates of fetal death, particularly late fetal death (28+ weeks' gestation) when compared with non-Māori. Among Māori there were also higher rates of premature birth particularly at lower gestation (20–27 weeks). The leading causes for both outcomes are 'unspecified' and it remains uncertain as to what are the causes or strategies to reduce these concerning outcomes. This is an area for further exploration.

With one in six Māori children living with disability, consideration of disability is integral to any analysis of Māori child and youth health needs and priorities. Around 1 in 25 Māori babies (4.2%) have one or more congenital anomalies identified at birth with rates among Māori babies being lower than for non-Māori non-Pacific babies. A broad range of anomalies is presented with the greatest number in Māori babies occurring in circulatory, musculoskeletal and digestive systems. There remains little published literature on these issues in Māori babies. The gaps in evidence include the impact of conditions and the effectiveness of services for Māori children with specific disability needs.

The report highlights the impact that increasing maternal age has on rates of congenital anomalies, including those in the cardiovascular system, present at birth. Younger maternal age likely contributes to lower rates of birth anomalies among Māori.

Section 2: Other Disabilities

This section includes permanent hearing loss, cerebral palsy and autism spectrum disorder. In 2012, 73 Māori children were identified in the Deafness Notification Database (DND). Age peaks occur in the under 1 year olds and in 5 and 6 year olds, consistent with newborn screening and B4 school checks. This reinforces the importance of screening for the detection of hearing loss. Newborn screening data for Māori does show 74.5% of Māori babies are screened by 3 months of age, which is lower than the total rate of 85.9% and indicates an area for further improvement. The finding that between April 2010 and March 2012 Māori babies made up 39.5% of the 81 babies identified from newborn screening as having permanent congenital hearing loss provides powerful evidence for both the need to ensure high rates of screening coverage, and also the significant impact of hearing loss as an issue for Māori children and whānau.

There are similar rates of hospitalisation for cerebral palsy for Māori when compared with non-Māori non Pacific children and young people; however, there are lower rates for diagnoses of autism or other pervasive development disorders. It remains uncertain as to

whether this indicates a lower incidence of autism among Māori or whether hospitalisations are lower.

Section 3: Chronic Medical Conditions

This section includes findings for morbidity for chronic medical conditions, specifically eczema and dermatitis, inflammatory bowel disease, cystic fibrosis, Type I diabetes, epilepsy and cancer. Analysis shows Māori children have lower rates of hospitalisation for inflammatory bowel disease, cystic fibrosis and type 1 diabetes, when compared with non-Māori non-Pacific children; with differences in patterns of familial genetic vulnerability suggested as reasons for these lower rates. For cancer, the rates of hospitalisation are similar. Although these conditions are of relatively low prevalence, they are serious and impact significantly on children, young people and their whānau. There is an ongoing need to ensure community and hospital services are accessible and responsive to the needs of Māori children and whānau.

The pattern of hospitalisation is quite different for a primary diagnosis of eczema and dermatitis: Māori children and young people were over 3 times more likely to be hospitalized than non-Māori non-Pacific children and young people. This is consistent with other research showing higher rates of severe eczema among Māori. Of importance, Figure 18 shows growing hospitalisation rates and inequalities, for eczema and dermatitis hospitalisations between 2000 and 2012. Hospitalisation rates for epilepsy are also significantly higher among Māori compared with non-Māori non-Pacific children and young people. Analysis of trends over time also suggests this rate difference is growing over the past decade. There is a paucity of information on the causes, management and impact of epilepsy among Māori and this may be an area for further research.

Section 4: Obesity, Nutrition and Physical Activity

This section includes data on overweight and obesity, the consequences of obesity, nutrition and physical activity. Findings are very consistent with high levels of concern about the increasing prevalence and consequences of obesity and overweight and the evidence of marked ethnic inequalities. In 2011/2012 almost 1 in 6 (16.4%) of Māori children were obese with a further 1 in 4 (27.2%) overweight. Similar levels of overweight and obesity were also found in Māori youth. Childhood figures constituted a significant increase from 2006/2007 for Māori children. This contrasts with European / Other children where there were no significant increases over time. Child and youth overweight and obesity thus appear to be an area of marked and growing inequality between Māori and non-Māori non-Pacific children and young people.

Of extreme concern, this inequality is very marked for specific consequences. Māori children were 4.36 times more likely to be hospitalized for Type II diabetes and 5.09 times more likely to be hospitalized for slipped upper femoral epiphysis (SUFE) than non-Māori non-Pacific young people. Findings for nutritional indicators (breakfast eating, fizzy drink consumption, family meals, fruit and vegetable eating) reinforce the importance of continuing to strive for strategies to increase the affordability and accessibility of healthy, nutritious food and the contribution that socio-economic factors play in contributing to nutritional outcomes.

Previous reports

The Child and Youth Epidemiology Service released its first Māori child-focused report in 2012: *Māori Children and Young People with Chronic Conditions and Disabilities*. That report reinforced the importance of healthy pregnancy and childbirth for Māori child health; the impact of chronic conditions, in particular those that have an infectious disease origin (rheumatic fever, bronchiectasis), on Māori children; and the ongoing and growing concern about inequity in childhood and adolescent overweight and obesity. The commentary (Joanne Baxter, Emma Wyeth) recognized the importance of understanding and meeting the needs of Māori children and young people living with disability and the gaps in knowledge across a number of areas [1].

In 2013, the *Health Status of Māori Children and Young people* report was released. This report outlined Māori child and youth mortality and morbidity and reinforced concerns

about persisting disparities in mortality for sudden unexpected death in infancy (SUDI), transport-related deaths, medical deaths with a social gradient, and suicide. Inequities in hospitalisations for infectious, respiratory and oral disease in particular were very evident. Commentary from Dr David Tipene-Leach on issues associated with SUDI discussed the importance of continued perseverance in initiatives aimed at safe-sleep and a need for persistent focus on addressing smoking in pregnancy [2].

In 2014, the report released on the *Determinants of Health for Māori Children and Young People* was consistent with the previous two reports in reiterating the direct and indirect impacts of social and economic determinants, and their consequences on the health of Māori children and young people. Ethnic inequalities in childhood poverty, household overcrowding and other indicators were evident. The report also in highlighted ethnic inequalities in secondary school education outcomes and for mental health. Editorial commentary by Dr Bev Lawton focused on pregnancy and the need to ensure that Māori mothers have timely access to culturally responsive, appropriate and effective antenatal care, optimized to meet Māori needs and positive outcomes for Māori babies and mothers [3].

Of note, the Determinants report included findings on immunisation coverage showing the elimination of ethnic inequality for full immunisation at 24 months. It further showed a persistent reduction of smoking among Māori Year 10 students. These are more positive outcomes that lead to reduced inequalities.

Summary of issues

The combined picture of Māori child outcomes across the three previous reports is concerning. The reports encompass common themes including the important role that differential exposure to socioeconomic determinants plays in driving Māori child and youth health outcomes across many areas.

In this current report, *Māori Children and Young People with Chronic Conditions and Disabilities,* key challenges and issues align with those identified in the earlier reports. Ethnic inequalities exist, most notably for pre-term birth and low birth weight, hearing impairment and loss, eczema and dermatitis and epilepsy. The growing rate of Māori childhood and adolescent overweight and obesity remains significant; coupled with the alarming statistics for Māori child and youth Type II diabetes morbidity and slipped upper femoral epiphysis, this issue must be considered a high priority for exploration of effective strategies for prevention, treatment and mitigation.

Across all reports, gaps in knowledge exist that would inform the understanding of Māori child and youth health needs, and areas for further analyses and research are evident. The importance of robust and consistent approaches to ethnicity data collection across different data sources is an ongoing challenge for measuring prevalence and monitoring trends in health service access and outcomes.

This report and conclusions from earlier reports reinforce the persistent recommendations for ensuring accessible, appropriate and effective prevention (healthy pregnancy and childbirth, injury prevention), early detection (sensory disability) and disability support services. These are all required. There is an important need to gain further understanding of the drivers for, and effective strategies to address, areas of preventable morbidity and mortality, and most importantly for tackling the socio-economic drivers of ethnic inequality. It is hoped that much can be learned and taken from specific successes (e.g. immunisation at 24 months) in reducing inequality.

BACKGROUND AND OVERVIEW

Report Structure and Content

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

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

As previously, this report is based on an *Indicator Framework* [4] 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.

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) for Māori 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, Māori 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) that may lead to greater health and disability support service demand during childhood and adolescence
2. Identifying the numbers of Māori 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 Māori children and young people

Ethnicity Coding, Data Quality Issues and the Signalling of Statistical Significance

When the authors prepared this report, high quality data were not always available in areas of public health importance. In a number of cases, the authors have opted to use data of lesser quality, in order to ensure that such issues do not fall below the public health radar. The cautions on interpretation that accompany each indicator will give readers a better understanding of the strengths and weaknesses of the data used. The text box below outlines a number of more specific data quality issues.

Ethnicity Coding and the Ethnicity Classifications Used in this Report

All of the ethnic-specific analyses presented in this report are from 1996 onwards, and thus reflect self-identified concepts of ethnicity (see **Appendix 5** for a more detailed review). In New Zealand's national health collections, up to 3 ethnic groups are stored electronically for each event [5]. However, inconsistencies in the way ethnicity information was collected before 1996 mean the data cannot easily be compared. Further, unless otherwise specified, this report uses total response ethnicity to identify ethnicity. That is, the term Māori refers to those children and young people identifying as Māori in any of their first three ethnic groups. The term non-Māori non-Pacific refers to those children and young people who did not identify as being either Māori or Pacific in any of their first three ethnic groups.

Note: The non-Māori reference group is often used in the Health Sector for rate ratio comparisons. However, the Te Ohonga Ake Advisory Group selected the non-Māori non-Pacific reference group on the basis that, as a group, these children and young people had the lowest documented exposures to health disparities.

Undercounting of Māori in National Health Collections

When reviewing the hospital admission data in sections that follow, the reader must bear in mind that none of the ethnic-specific rates have been adjusted for undercounting. Therefore, the rate ratios presented may underestimate, to a variable extent, the magnitude of any ethnic inequalities present. Despite significant improvements in the quality of ethnicity data since 1996, national data collections may still undercount Māori children and young people. The authors of Hauora Māori Standards of Health IV [6] linked hospital admission and cancer registry data to other more reliable data sources. They found that, on average, hospital admission data during 2000–2004 undercounted Māori children by 6%, and Māori young people by 5–6%. For cancer registrations, the undercount was in the order of 1–2% (**see Appendix 5**).

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 that accompany these indicators, in order to gain a better understanding of the strengths and weaknesses of the data used.

Further, **Appendix 1** outlines the rationale for the use of statistical significance testing in this report and **Appendix 2–Appendix 4** 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 1**), 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

Table 1. Overview of the Health of Māori Children and Young People with Chronic Conditions and Disabilities

Stream	Indicator	Distribution in Māori Children and Young People
Conditions Arising in the Perinatal Period		
Perinatal Conditions	Fetal Deaths	• During 2006–2010, unspecified cause was the most frequently listed main fetal cause of intermediate fetal death (IFD 20–27 weeks' gestation) in Māori babies, followed by congenital and chromosomal anomalies and prematurity/low birth weight. • Unspecified cause was also the most frequently listed main fetal cause of late fetal death (LFD 28+ weeks' gestation) in Māori babies, followed by malnutrition/slow fetal growth, congenital anomalies and intrauterine hypoxia. • During 2006–2010, there were on average 71 IFDs per year in Māori babies, with rates not being *significantly* different from those of non-Māori non-Pacific babies (RR 0.90 95% CI 0.80–1.02). • In addition, there were on average 67 LFDs per year, with rates for Māori babies being *significantly* higher than for non-Māori non-Pacific babies (RR 1.21 95% CI 1.06–1.39).
	Preterm Birth: Singletons	• During 2008–2012, preterm birth rates (20–36 weeks) for Māori babies were *significantly* higher than for non-Māori non-Pacific babies (RR 1.15 95% CI 1.11–1.18). When viewed by gestational age however, the magnitude of the excess risk for Māori babies was greatest at lower gestations (20–27 weeks RR 1.54 95% CI 1.35–1.75 vs. 32–36 weeks RR 1.11 95% CI 1.07–1.15). • During 2000–2012, preterm birth rates for Māori babies were relatively static. However, the actual number of preterm Māori babies born increased, as the result of a rising birth rate, with most of this increase occurring during 2002–2008. The largest increases were seen at 32–36 weeks, with numbers in this category rising from 861 in 2000 to 967 in 2012.
	Preterm Birth: Multiple Pregnancies	• During 2008–2012, preterm birth rates for Māori twins were *not significantly* different from those of non-Māori non-Pacific twins (RR 0.97 95% CI 0.93–1.01). On average, 273 Māori preterm babies were born each year as a result of a twin pregnancy. • Overall, 93.4% of singleton Māori babies were born after 36 weeks' gestation, as compared to 45.4% of twins and 6.0% of triplets, with the gestational age curve shifting towards younger gestational ages as the number of babies increased. • During 2000–2012, preterm birth rates were relatively static in Māori singletons and triplets. Preterm birth rates for Māori twins however increased, from 48.7 in 2000 to 56.3 in 2000, with the majority of this increase occurring after 2008.
Congenital Anomalies Evident at Birth	Congenital Anomalies Evident at Birth	• During 2008–2012, a large number of congenital anomalies were identified at birth in Māori babies, with these ranging in severity from minor skin conditions (e.g. non-neoplastic nevus), through to anomalies that were incompatible with life (e.g. anencephaly/ encephalocele). • On average, 559 Māori babies per year (4.2% of all Māori births), had one or more congenital anomalies identified at birth, with rates being *significantly* lower than for non-Māori non-Pacific babies (RR 0.89 95% CI 0.85–0.92).

Stream	Indicator	Distribution in Māori Children and Young People
Congenital Anomalies Evident at Birth	Congenital Heart Disease	• During 2008–2012, patent ductus arteriosus (PDA) was the most frequent cardiovascular (CVS) anomaly identified at birth in Māori babies, with 65.6% of PDAs being in preterm babies who had no other CVS anomalies. Atrial septal and ventricular septal defects were the next most frequent CVS anomalies identified. • On average, 72 Māori babies per year (excluding isolated preterm PDAs) had one or more CVS anomalies identified at birth, with rates not being *significantly* different from those of non-Māori non-Pacific babies (RR 0.96 95% CI 0.85–1.08).
	Down Syndrome	• During 2008–2012, on average 9 Māori babies per year had Down syndrome identified at birth, with rates, while lower, not being *significantly* different from those of non-Māori non-Pacific babies (RR 0.79 95% CI 0.58–1.09).
	Neural Tube Defects	• During 2008–2012, on average 4.2 Māori babies per year had a neural tube defect identified at birth, with rates, while higher, not being *significantly* different from those of non-Māori non-Pacific babies (RR 1.41 95% CI 0.84–2.37).
Other Disabilities		
Permanent Hearing Loss	Deafness Notification Database (DND)	• During 2012, 73 children notified to the DND identified as Māori. As total response ethnicity was used, it was not possible to provide a breakdown of the proportions of children notified by ethnicity.
	Newborn Hearing Screening	• During 1st Oct. 2011–31st Mar. 2012, of those Māori babies who completed newborn hearing screening, 89.8% did so within one month, with 2.3% of those completing screening receiving an audiology referral. Of those who passed screening, a further 6.8% had risk factors for delayed onset/progressive hearing loss that warranted follow up. During this period, 12 Māori babies were identified as having a permanent congenital hearing loss and 32 as having a conductive hearing loss.
Other Disabilities	Cerebral Palsy	• During 2008–2012, 384 individual Māori children and young people were hospitalised with a diagnosis of cerebral palsy, with admission rates not being *significantly* different from those of non-Māori non-Pacific children and young people (RR 0.97 95% CI 0.91–1.04). • Acute and arranged admissions made up 53.3% of hospitalisations for Māori children and young people with cerebral palsy, while 46.7% were from the waiting list. • Only 7.5% of acute and arranged admissions had cerebral palsy listed as the primary reason for admission, with 13.9% being for epilepsy or convulsions and 15.8% for influenza, pneumonia or unspecified lower respiratory infections. • Injections into ligaments, tendons, or soft tissue accounted for 35.9% of waiting list admissions, with orthopaedic procedures collectively being the leading reasons for waiting list admission.
	Autism Spectrum Disorder	• During 2008–2012, 284 individual Māori children and young people were hospitalised with a diagnosis of autism or other pervasive developmental disorders, with admission rates being *significantly* lower than for non-Māori non-Pacific children and young people (RR 0.65 95% CI 0.58–0.72). • Autism and other pervasive developmental disorders were listed as the primary diagnosis in only 10.8% of hospitalisations for Māori children and young people with a pervasive developmental disorder in any of the first 15 diagnoses. 27.0% of admissions were for dental caries or other oral health conditions, while a further 9.6% were for epilepsy or convulsions.

Stream	Indicator	Distribution in Māori Children and Young People
Chronic Medical Conditions		
Chronic Medical Conditions	Eczema and Dermatitis	• During 2008–2012, only 32.4% of hospitalisations in Māori children with eczema or dermatitis listed in any of their first 15 diagnoses, had these diagnoses as the primary reason for admission. Infective dermatitis (14.5%) and atopic and other dermatitis (14.3%) were the most frequent primary diagnoses in those with eczema or dermatitis, while bronchiolitis, asthma and wheeze and skin infections were the most frequent non-eczema related diagnoses. • During this period, 1,155 individual Māori children were hospitalised with a primary diagnosis of eczema or dermatitis, with admission rates being *significantly* higher than for non-Māori non-Pacific children (RR 3.16 95% CI 2.94–3.39).
	Inflammatory Bowel Disease	• During 2008–2012, 41 individual Māori children and young people were hospitalised with Crohn's disease, while 21 were hospitalised with ulcerative colitis (UC). Admission rates per for both outcomes were *significantly* lower than for non-Māori non-Pacific children and young people (Crohn's disease RR 0.11 95% CI 0.09–0.13; UC RR 0.11 95% CI 0.07–0.16) • 84.0% of acute and arranged hospitalisations in Māori children and young people with Crohn's disease had Crohn's disease listed as the primary reason for admission. Of those admitted from the waiting list, injections or infusions of therapeutic substances (58.8%) and fibreoptic colonoscopies +/− biopsies (15.7%) were the most frequent primary procedures listed. • 60.0% of acute and arranged hospitalisations in Māori children and young people with ulcerative colitis, had ulcerative colitis listed as their primary reason for admission. Of those admitted from the waiting list, fibreoptic colonoscopies +/− biopsies (80.0%) were the most frequent primary procedures listed.
	Cystic Fibrosis (CF)	• During 2008–2012, 37 individual Māori children and young people were hospitalised with a diagnosis of CF, with admission rates being *significantly* lower than for non-Māori non-Pacific children and young people (RR 0.46 95% CI 0.41–0.51). • 83.0% of hospitalisations in Māori children and young people with CF had CF listed as the primary reason for admission. Of those with CF listed as the primary diagnosis, the majority (92.5%) also had a secondary diagnosis. In 25.1% of cases, the secondary diagnosis was influenza, pneumonia or an unspecified lower respiratory infection, while a further 14.0% had bronchiectasis.
	Type 1 Diabetes	• During 2008–2012, 429 individual Māori children and young people were hospitalised with a diagnosis of Type 1 Diabetes, with admission rates being *significantly* lower than for non-Māori non-Pacific children and young people (RR 0.76 95% CI 0.72–0.80). • 72.8% of hospital admissions for Māori children and young people with Type 1 Diabetes had a diabetes-related primary diagnosis, with ketoacidosis/lactic acidosis +/− coma accounting for 38.3% and Type 1 Diabetes without complications for 13.0% of admissions. A further 27.2% of admissions were for diagnoses other than diabetes, with pregnancy and childbirth, gastroenteritis, and respiratory diseases being the most common reasons for non-diabetes related admissions.

Stream	Indicator	Distribution in Māori Children and Young People
Chronic Medical Conditions	Epilepsy	• During 2008–2012, 1,161 individual Māori children and young people were hospitalised with a diagnosis of epilepsy or status epilepticus, with admission rates being *significantly* higher than for non-Māori non-Pacific children and young people (RR 1.42 95% CI 1.36–1.49) • 74.3% of all hospital admissions in Māori children and young people with epilepsy or status epilepticus, had an epilepsy-related primary diagnosis. Generalised idiopathic epilepsy (24.2%) and unspecified epilepsy (21.9%) were the most frequent epilepsy-related diagnoses. A further 25.7% of admissions were for conditions unrelated to epilepsy, with respiratory conditions, injury and poisoning, and pregnancy and childbirth being the most frequent non epilepsy-related reasons for admission.
	Cancer	• During 2002–2011, acute lymphoblastic leukaemia was the most frequent malignant neoplasm notified to the NZ Cancer Registry in Māori children and young people, followed by cancers of the testis. Carcinoma in situ of the cervix however, was the most frequent reason for a NZ Cancer Registry notification, accounting for 60.8% of notifications in Māori children and young people. • During 2001–2010, cancers of the brain were the leading cause of cancer mortality in Māori children and young people, followed by cancers of the bone and cartilage.
Obesity, Nutrition and Physical Activity		
Distribution of Overweight and Obesity	NZ Health Surveys	• The proportion of Māori children aged 2–14 years who were obese increased *significantly* between NZ Health Surveys, 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. • The proportion of Māori children who were overweight (but not obese) did not change *significantly* between NZ Health Surveys however, with rates being 25.8% (95% CI 22.8–29.0) in 2006/07 and 27.2% (95% CI 23.5–31.2) in 2011/12. • In the 2011/12 NZHS, Māori children were 2.10 (95% CI 1.64–2.68) times more likely to be obese than non-Māori children, and 1.40 (95% CI 1.18–1.67) times more likely to be overweight once rates were adjusted for age and gender.
	Youth'12 Survey	• There were no *significant* changes in the proportion of Māori students who were overweight or obese between Youth surveys, with rates being 46.5% in Youth'07 and 43.9% in Youth'12. • In the Youth'12 Survey, 54.5% of Māori students were a healthy weight, while 26.7% were overweight and 17.2% were obese.
Consequences of Overweight and Obesity	Type 2 Diabetes	• During 2008–2012, 157 individual Māori children and young people were hospitalised with a diagnosis of Type 2 Diabetes, with admission rates being *significantly* higher than for non-Māori non-Pacific children and young people (RR 4.36 95% CI 3.66–5.19). • 19.0% of hospital admissions for Māori children and young people with Type 2 Diabetes had a diabetes related primary diagnosis. The remaining 81.0% of admissions had non-diabetes related primary diagnoses, with pregnancy and childbirth (14.8%), skin infections (7.7%) and diseases of the respiratory system (5.8%) being the leading non-diabetes related reasons for admission.
	Slipped Upper Femoral Epiphysis (SUFE)	• During 2008–2012, 289 individual Māori children and young people were hospitalised with a slipped upper femoral epiphysis, with admissions being *significantly* higher than for non-Māori non-Pacific children and young people (RR 5.09 95% CI 4.29–6.04).

Stream	Indicator	Distribution in Māori Children and Young People
Consequences of Overweight and Obesity	Bariatric Surgery	• During 2008–2012, while bariatric surgery admissions were lower for Māori than for European/Other young people, these differences did not reach statistical significance. A total of 4 Māori young people underwent bariatric surgery during this period.
Breastfeeding and Solids	Breastfeeding in Plunket Babies	• In the year ending June 2013, 58.2% of Māori Plunket babies were exclusively or fully breastfed at <6 weeks, with the proportion falling to 43.5% at 3 months and 15.4% at 6 months • During June 2006–2013, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were consistently lower for Māori than for non-Māori non-Pacific babies, with rates being relatively static for both ethnic groups during this period. • In the year ending June 2013, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were higher for Māori babies from the least deprived (NZDep decile 1) areas than for Māori babies from the most deprived (NZDep decile 10) areas.
	Babies Given Solids at <4 Months of Age	• The proportion of Māori children aged 4 months to 4 years given solid food before four months of age declined *significantly* (p=0.04) between NZ Health Surveys, with rates falling from 21.7% (95% CI 17.8–26.0) in 2006/07 to 15.6% (95% CI 11.7–20.2) in 2011/12. • 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, once rates were adjusted for age and gender.
Other Nutritional Indicators	NZ Health Surveys	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. • 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. • 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.
	Youth'12 Survey	In the Youth'12 Survey: • 45.2% (95% CI 42.2–48.3) of Māori secondary school students said they always ate breakfast, with rates for males being *significantly* higher than for females. While there were no age or urban/rural differences, a *significantly* higher proportion of Māori students from the least deprived (NZDep06 deciles 1–3) areas always ate breakfast, than from the most deprived (deciles 8–10) areas. • 59.8% (95% CI 57.3–62.4) of Māori students said they ate a meal with family 5+ times in the last 7 days, with rates for males being *significantly* higher than for females. While there were no NZDep06 or geographic (urban vs. rural) differences, a *significantly* higher proportion of younger (≤13 years) than older Māori students (17+ years) ate meals with family 5+ times in the last 7 days. • 31.9% (95% CI 29.9–33.9) of Māori students said that they ate 2+fruit and 3+ vegetables per day. There were no significant gender, age, NZDep06 or rural/urban differences in the proportion of Māori students who ate 2+ fruit and 3+ vegetables per day.
Physical Activity	Youth'12 Survey	• In the Youth'12 survey, while 65.9% (95% CI 63.1–68.7) of Māori students had participated in more than 20 minutes vigorous physical activity on three or more occasions in the past seven days, only 10.7% (95% CI 8.9–12.6) reported achieving the recommended 60+ minutes of physical activity daily.

Stream	Indicator	Distribution in Māori Children and Young People
Physical Activity	NZ Health Surveys	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. • 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.

CONDITIONS ARISING IN THE PERINATAL PERIOD

PERINATAL CONDITIONS

FETAL DEATHS

Introduction

The following section reviews the distribution of fetal deaths in Māori babies using information from the National Mortality Collection and the Birth Registration Dataset.

Background

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 deaths include stillbirths and termination of pregnancy*" [7]. Fetal deaths are often further subdivided into intermediate fetal deaths (those occurring between 20 and 27 weeks' gestation), and late fetal deaths (those occurring at 28 or more weeks' gestation).

In New Zealand, 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 [8] [9] [10]. Pregnancy-related disorders such as fetal growth restriction, placental abruption and maternal diabetes and hypertension also increase the risk of a fetal death [11].

The risk of late fetal death for Māori women however, is not significantly different from that of European women, with one study finding that once adjusted for other known risk factors (e.g. maternal age, parity, body mass index, smoking and deprivation index) the risk for Māori women was actually lower [10]. The authors noted that while Māori women without lifestyle risk factors had a lower risk of late fetal death, future well designed studies were needed to confirm or refute these findings. They also noted that, while their findings were consistent with the existing literature, other studies had found an increased risk of intermediate fetal deaths amongst Māori babies (which were excluded from their study) [10].

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.

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).

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.

Distribution in Māori Babies

Number of Babies

In New Zealand during 2006–2010, there were on average 71 intermediate fetal deaths per year in Māori babies, with rates being *not significantly* different from those of non-Māori non-Pacific babies (RR 0.90 95% CI 0.80–1.02). In addition, there were on average 67 late fetal deaths per year in Māori babies, with rates being *significantly* higher than for non-Māori non-Pacific babies (RR 1.21 95% CI 1.06–1.39) (**Table 2**).

Table 2. Intermediate and Late Fetal Deaths by Ethnicity, New Zealand 2006–2010

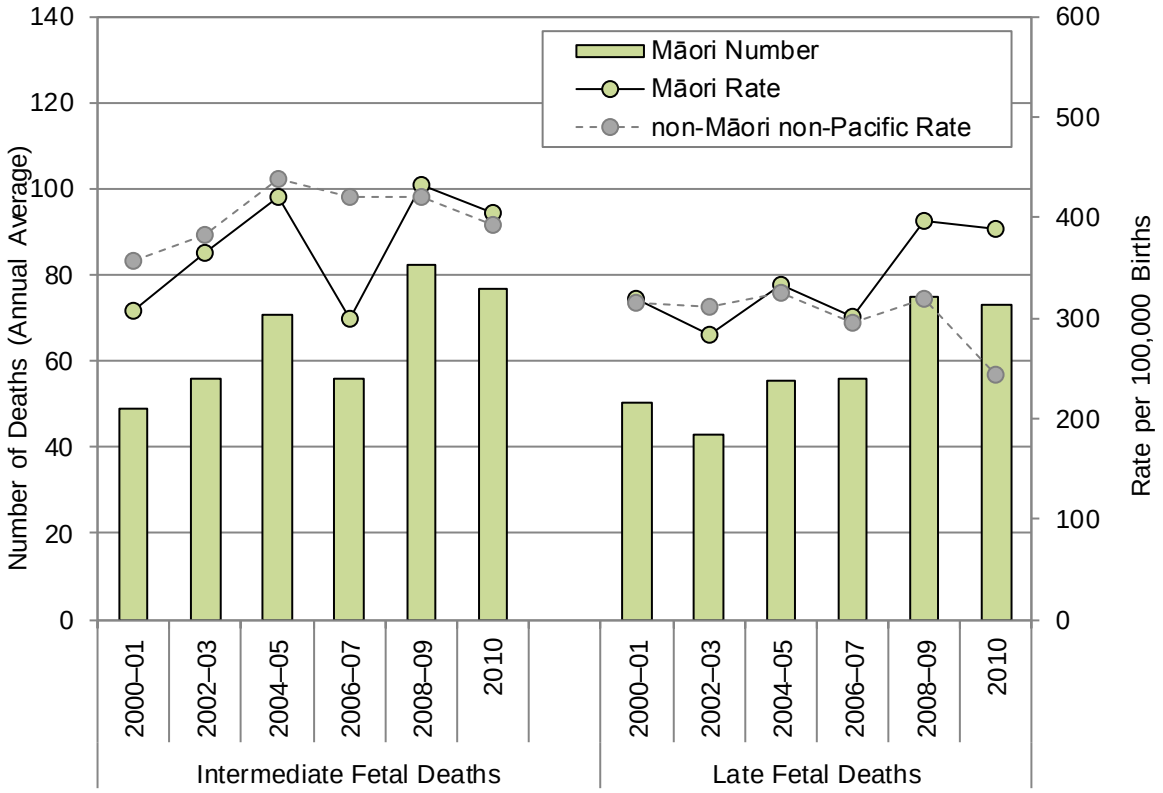
Ethnicity	Number of Deaths: Total 2006–2010	Number of Deaths: Annual Average	Rate per 100,000 Births	Rate Ratio	95% CI
Intermediate Fetal Death					
Māori	354	71	374.27	0.90	0.80–1.02
non-Māori non-Pacific	792	158	415.08	1.00	
Late Fetal Death					
Māori	335	67	357.41	1.21	1.06–1.39
non-Māori non-Pacific	557	111	294.20	1.00	

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

Trends by Ethnicity

In New Zealand during 2000–2010, large year to year variations in rates made trends in intermediate and late fetal deaths for Māori babies difficult to interpret. Late fetal death rates for Māori babies however, were higher than for non-Māori non-Pacific babies during 2008–2010 (**Figure 1**).

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



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

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 Māori babies dying in utero between 20 and 27 weeks of gestation, followed by congenital and chromosomal anomalies and prematurity/low birth weight (**Table 3**).

Late Fetal Deaths: During 2006–2010, unspecified cause was also the most frequently listed main fetal cause of death for Māori babies dying in utero at 28+ weeks' gestation, followed by malnutrition/slow fetal growth, congenital anomalies and intrauterine hypoxia (**Table 3**).

Table 3. Intermediate and Late Fetal Deaths in Māori Babies by Main Fetal Cause of Death, New Zealand 2006–2010

Main Fetal Cause of Death	No. of Deaths: Total 2006–2010	No. of Deaths: Annual Average	Rate per 100,000 Births	% of Fetal Deaths
Māori Babies				
Intermediate Fetal Deaths				
Unspecified Cause	112	22.4	118.41	31.6
Prematurity/Low Birth Weight	66	13.2	69.78	18.6
Chromosomal Anomalies	40	8.0	42.29	11.3
Congenital Anomalies: CNS	28	5.6	29.60	7.9
Congenital Anomalies: CVS	14	2.8	14.80	4.0
Congenital Anomalies: Other	41	8.2	43.35	11.6
Malnutrition/Slow Fetal Growth	18	3.6	19.03	5.1
Fetal Blood Loss	8	1.6	8.46	2.3
Congenital Pneumonia	4	0.8	4.23	1.1
Intrauterine Hypoxia	4	0.8	4.23	1.1
Infections Specific to Perinatal Period	3	0.6	3.17	0.8
Polycythaemia Neonatorum	3	0.6	3.17	0.8
Other Causes	13	2.6	13.75	3.7
Total Intermediate Fetal Deaths	354	70.8	374.27	100.0
Late Fetal Deaths				
Unspecified Cause	187	37.4	199.51	55.8
Malnutrition/Slow Fetal Growth	29	5.8	30.94	8.7
Intrauterine Hypoxia	26	5.2	27.74	7.8
Fetal Blood Loss	11	2.2	11.74	3.3
Congenital Anomalies: CNS	10	2.0	10.67	3.0
Congenital Anomalies: CVS	5	1.0	5.33	1.5
Congenital Anomalies: Other	11	2.2	11.74	3.3
Prematurity/Low Birth Weight	9	1.8	9.60	2.7
Chromosomal Anomalies	8	1.6	8.54	2.4
Aspiration Meconium/Amniotic Fluid/Mucus	8	1.6	8.54	2.4
Infections Specific to Perinatal Period	7	1.4	7.47	2.1
Other Causes	24	4.8	25.61	7.2
Total Late Fetal Deaths	335	67.0	357.41	100.0

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

New Zealand Distribution and Trends

Additional information on the distribution of fetal deaths in New Zealand is available from *the Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand* [12]. In brief, this report found:

Distribution by NZ Deprivation Index Decile, Maternal Age and Gender

During 2006–2010, there were no significant gender or socioeconomic (as measured by NZDep06) differences in intermediate fetal death rates. However, mortality 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.

Late fetal deaths were *significantly* higher for babies from average to more deprived (NZDep deciles 5–10) areas, than for babies from the least deprived (NZDep deciles 1–2) areas. Rates were also *significantly* higher for the babies of teenage women, than for the babies of women 30–34 years.

Distribution by Gestational Age and Cause

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. These rates however, 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.

PRETERM BIRTH

Introduction

The following section reviews preterm birth rates in Māori babies using information from the Birth Registration Dataset.

Background

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 [13].

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) [13]. 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 [13]. 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 [13]. Rates of pre-term birth have risen over recent decades in most developed countries. Factors believed to be responsible include: better monitoring of maternal health problems such as hypertension and diabetes, rising maternal age, increasing rates of multiple pregnancies due to infertility treatments, and increases in elective caesareans before term [14].

In New Zealand, preterm birth rates remain higher for Māori women than for European or Pacific women. However, during 1980–1994 preterm birth rates increased by 30% for European women, as opposed to a non-significant reduction of 7% for Māori women, leading to a significant reduction in ethnic inequalities in preterm birth. It was unclear however, whether these findings arose from increasing obstetric intervention in European women, as opposed to any real reductions in ethnic inequalities in spontaneous preterm birth (as no information on the reasons for preterm delivery is available [15, 16]). In 2010, 8.1% of Māori babies were born before 37 weeks' gestation, compared to 6.5% of Pacific babies, 6.5% of Asian babies and 7.5% of Other babies [17]. (The "Other" ethnic group includes European babies.)

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

1. Numerator: Birth Registration Dataset: All singleton live born babies 20–36 weeks' gestation. Gestational age categories include 20–27 weeks, 28–31 weeks, and 32–36 weeks.

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

2. Numerator: Birth Registration Dataset: All live born babies 20–36 weeks' gestation by plurality (singletons, twins, triplets).

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 3** 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.

Preterm Births in Singleton Pregnancies

Distribution by Ethnicity

In New Zealand during 2008–2012, overall preterm birth rates (20–36 weeks) for Māori babies were *significantly* higher than for non-Māori non-Pacific babies (RR 1.15 95% CI 1.11–1.18). While similar ethnic differences were evident when preterm birth rates were broken down by gestational age, the magnitude of the excess risk seen for Māori babies, was greatest for births at lower gestations (20–27 weeks RR 1.54 95% CI 1.35–1.75 vs. 32–36 weeks RR 1.11 95% CI 1.07–1.15) (**Table 4**).

Table 4. Preterm Birth Rates in Singleton Live Born Babies by Ethnicity and Gestational Age, New Zealand 2008–2012

Ethnicity	Number of Babies: Total 2008–2012	Number of Babies: Annual Average	Rate per 100 Live Births	Rate Ratio	95% CI
All Preterm Singletons (20–36 Weeks)					
Māori	5,990	1,198	6.64	1.15	1.11–1.18
non-Māori non-Pacific	10,613	2,123	5.78	1.00	
Preterm Singletons 20–27 Weeks					
Māori	411	82	0.46	1.54	1.35–1.75
non-Māori non-Pacific	544	109	0.30	1.00	
Preterm Singletons 28–31 Weeks					
Māori	643	129	0.71	1.29	1.17–1.43
non-Māori non-Pacific	1,013	203	0.55	1.00	
Preterm Singletons 32–36 Weeks					
Māori	4,936	987	5.48	1.11	1.07–1.15
non-Māori non-Pacific	9,056	1,811	4.94	1.00	

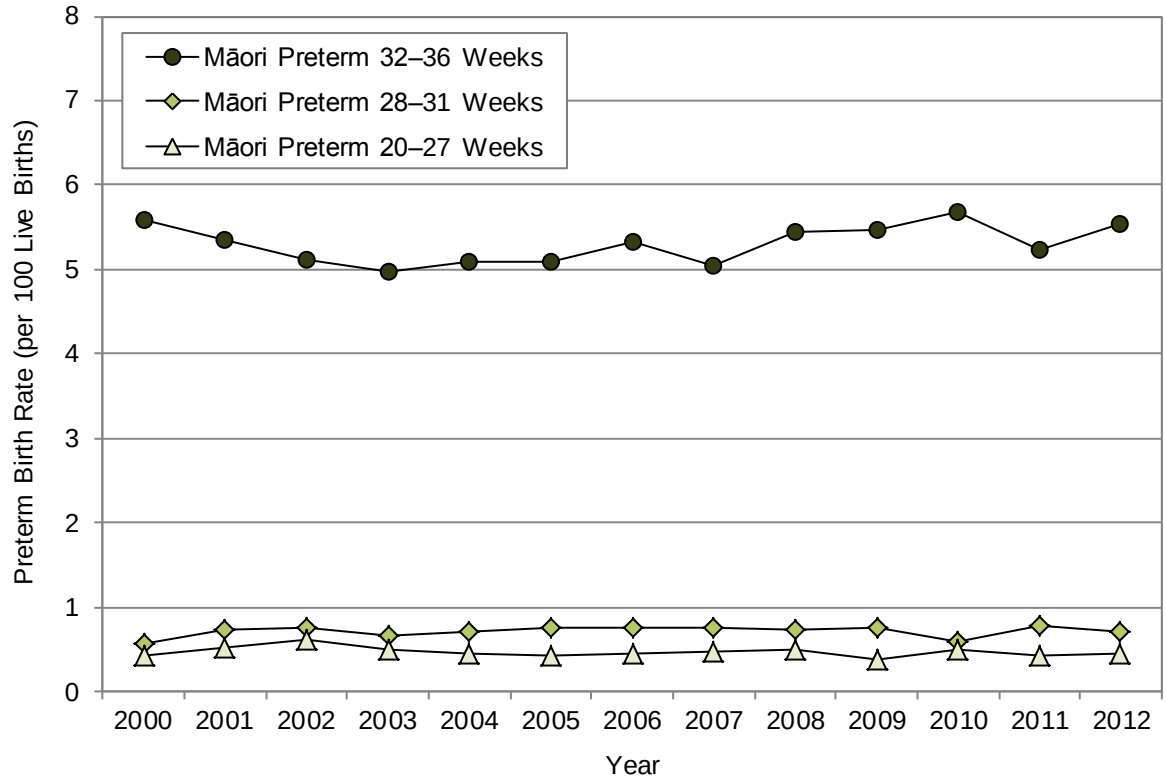
Source: Birth Registration Dataset: Numerator: All singleton live born babies 20–36 weeks' gestation; Denominator: All singleton live born babies 20+ weeks' gestation

Trends for Māori Babies

Preterm Birth Rates: In New Zealand during 2000–2012, preterm birth rates amongst Māori babies were relatively static. Rates (per 100 live births) for those born at 20–27 weeks' gestation were 0.43 per in 2000 and 0.45 in 2012, while rates for those born at 28–31 weeks were 0.56 in 2000 and 0.72 in 2012, and rates for those born at 32–36 weeks were 5.6 in 2000 and 5.5 in 2012 (**Figure 2**).

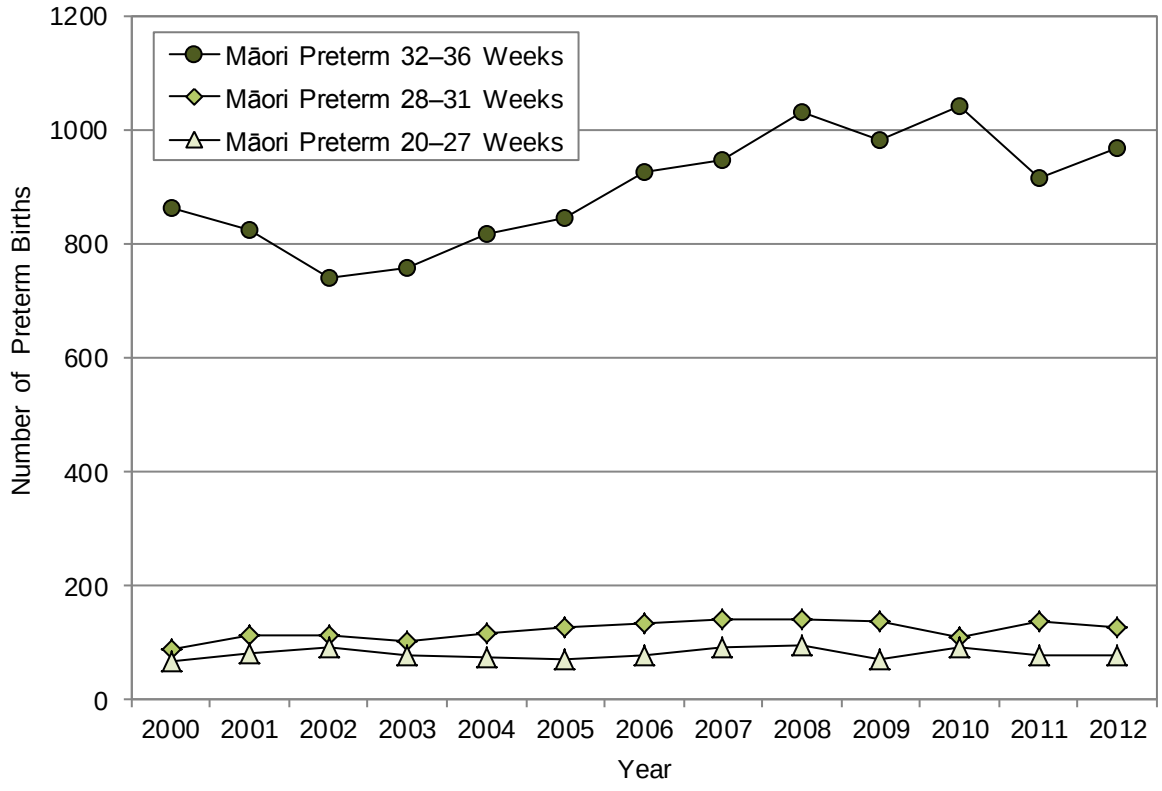
Number of Preterm Births: During the same period however, the number of preterm Māori babies born increased, as the result of a rising birth rate, with the majority of this increase occurring between 2002 and 2008. The largest increases were seen in those born at 32–36 weeks, with numbers in this category rising from 861 in 2000 to 967 in 2012. The number of babies born at 28–31 weeks' gestation also rose slightly, from 86 in 2000 to 125 in 2012, as did the number of babies born at 20–27 weeks' gestation (66 in 2000 and 78 in 2012) (**Figure 3**).

Figure 2. Preterm Birth Rates in Māori Singleton Live Born Babies by Gestational Age, 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 3. Number of Preterm Births in Māori Singleton Live Born Babies by Gestational Age, New Zealand 2000–2012



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

Preterm Births in Multiple Pregnancies

Distribution by Ethnicity

In New Zealand during 2008–2012, preterm birth rates amongst Māori twins were *not significantly* different from those of non-Māori non-Pacific twins (RR 0.97 95% CI 0.93–1.01). On average during this period, 273 Māori preterm babies were born each year as a result of a twin pregnancy (**Table 5**).

Table 5. Preterm Birth Rates in Live Born Twins by Ethnicity, New Zealand 2008–2012

Ethnicity	Number of Babies: Total 2008–2012	Number of Babies: Annual Average	Rate per 100 Live Births	Rate Ratio	95% CI
Preterm Twins 20–36 Weeks					
Māori	1,365	273	54.6	0.97	0.93–1.01
non-Māori non-Pacific	2,997	599	56.2	1.00	

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

Distribution by Gestational Age

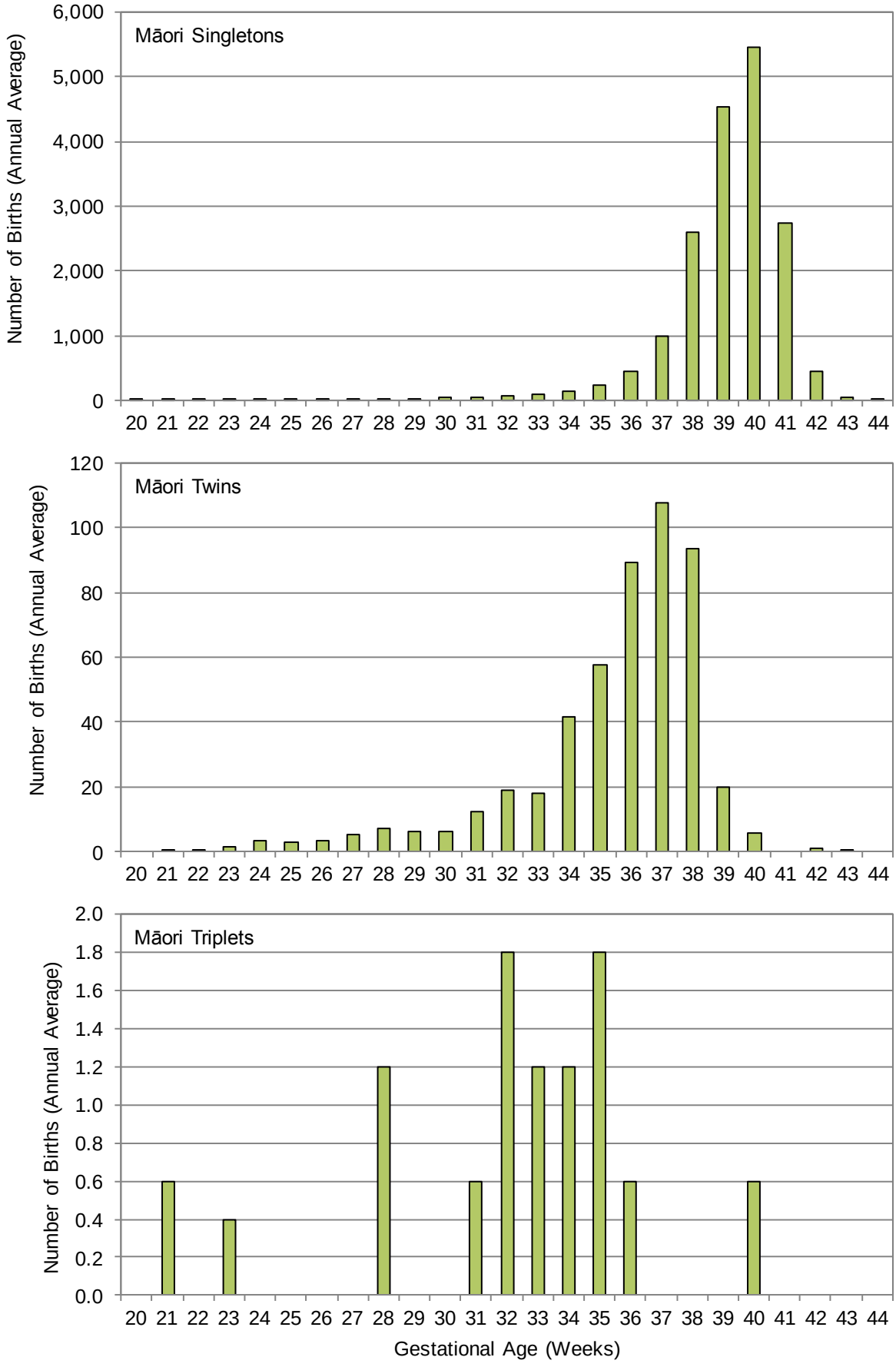
In New Zealand during 2008–2012, 93.4% of singleton Māori babies were born after 36 weeks' gestation, as compared to only 45.4% of twins and 6.0% of triplets, with the gestational age curve shifting increasingly towards the left (i.e. towards younger gestational ages) as the number of babies increased. During this period, the most frequent gestational age for the delivery of a singleton Māori baby was 40 weeks, as compared to 37 weeks for twins (**Figure 4**).

New Zealand Trends

Preterm Birth Rates: In New Zealand during 2000–2012, preterm birth rates were relatively static in Māori singletons and triplets, with rates per 100 live births for Māori singletons being 6.6 in 2000 and 6.7 in 2012. Similarly rates for Māori triplets were 100% for the majority of this period. In contrast, preterm birth rates in Māori twins increased, from 48.7 in 2000 to 56.3 in 2000, with the majority of this increase occurring after 2008 (**Figure 5**).

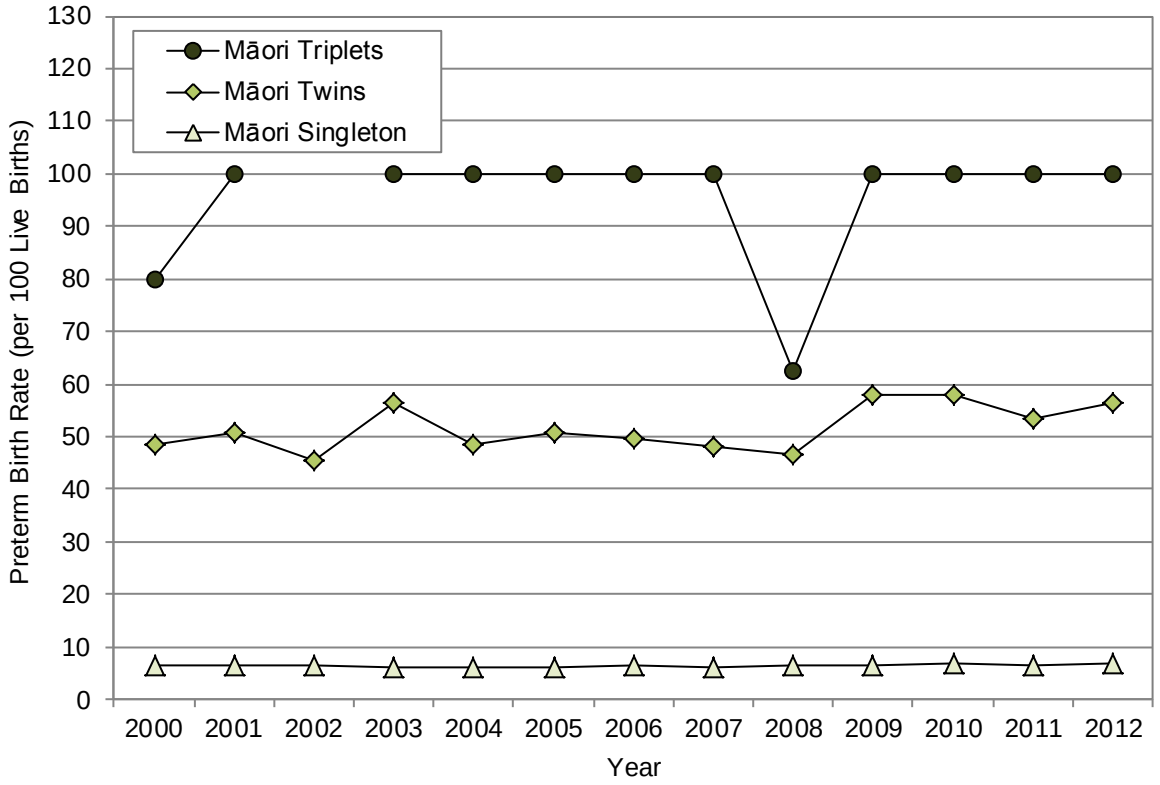
Number of Preterm Births: During the same period, the number of singleton Māori preterm babies born increased (from 1,013 in 2000 to 1,170 in 2012), as the result of a rising birth rate, with all of this increase occurring between 2003 and 2008. Similarly, the number of twin Māori preterm births increased, from 220 in 2000 to 273 in 2012. The number of triplet births however was more static (12 in 2000 and 9 in 2012) (**Figure 6**).

Figure 4. Gestational Age at Delivery by Plurality, Māori Live Births 2008–2012



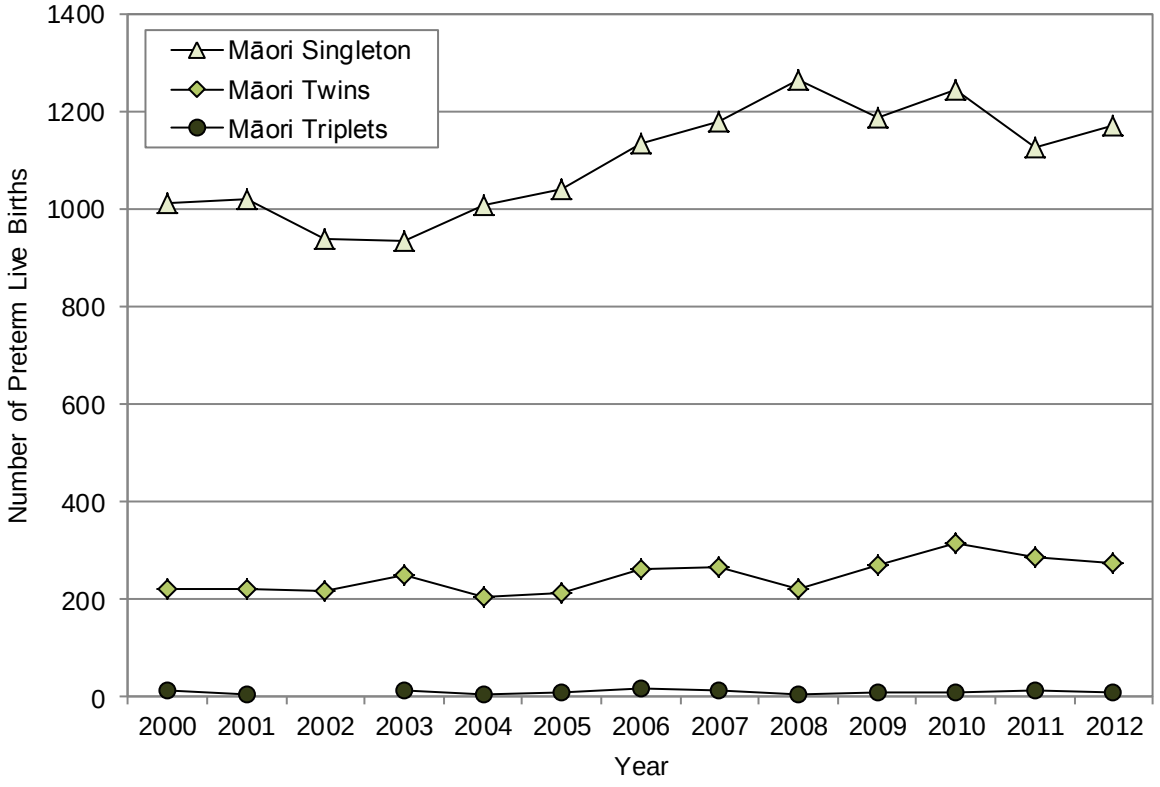
Source: Birth Registration Dataset: All live born babies 20–36 weeks' gestation

Figure 5. Preterm Birth Rates in Māori 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 6. Number of Māori Preterm Live Births by Plurality, New Zealand 2000–2012



Source: Birth Registration Dataset; All live born babies 20–36 weeks' gestation

New Zealand Distribution and Trends

Additional information on the distribution of preterm births in the New Zealand is available from *the Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand* [12]. In brief, this report found:

Singleton Pregnancies

Distribution by NZ Deprivation Index Decile, Gender and Maternal Age

During 2008–2012, total preterm birth rates (20–36 weeks) were *significantly* higher for males, for those from more deprived (NZDep deciles 7–10 vs. 1–2) areas, and for the babies of younger (<25 years) and older (35+ years) mothers, than for the babies of mothers aged 25–29 years.

When broken down by gestational age, a common theme emerged, with the magnitude of the excess risk of preterm birth seen for the babies of teenage mothers, and those from the more deprived areas, being most marked amongst births at lower gestations.

Multiple Pregnancies

Risk of Preterm Birth by Plurality

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.

Distribution by NZ Deprivation Index Decile, Gender and Maternal Age

During 2008–2012, there were no *significant* gender, socioeconomic (as measured by NZDep06), or maternal age-related differences in preterm birth rates amongst twins.

CONGENITAL ANOMALIES EVIDENT AT BIRTH

ANTENATAL AND NEWBORN SCREENING

Overseas research suggests that up to 25% of babies with severe forms of congenital heart disease are discharged from hospital undiagnosed [18]. Similarly, in New Zealand, 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 [19]. 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 [20]).

The early detection of these conditions thus confers significant advantages, with antenatal diagnosis also providing 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 are required [21]. 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 such 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 [22]. However, concerns during the mid-2000s that the existing screening processes were ad-hoc [23], led the National Screening Unit to release a set of guidelines for maternity providers in 2009 [22]. 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 that assesses nuchal translucency (a marker that measures the fluid filled space in the tissue at the back of a fetus' neck and 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 [22].
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), with the optimal time for serum screening being 14–18 weeks [22].

In its updated 2012 Guidelines for Health Practitioners [24], the National Screening Unit also 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 [24].

In addition, while not being part of a formal screening programme, ultrasounds are frequently undertaken between 18–20 weeks of gestation to screen for obvious structural anomalies, although such scans are thought not to be as effective for Down Syndrome screening as the screening modalities listed above [22].

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 (initial examination 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) [25]. At the initial (newborn) examination the Schedule recommends that clinicians undertake a thorough assessment that 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 [26].

Newborn Metabolic Screening Programme

When New Zealand first commenced newborn metabolic screening in 1969, screening was initially only undertaken for phenylketonuria (PKU) [19]. The current Newborn Metabolic Screening Programme (NMSP) however, screens for 28 metabolic disorders [19], with these conditions being outlined in **Table 6**.

Table 6. Conditions Included in New Zealand's Newborn Metabolic Screening Programme

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

Source: National Screening Unit http://www.nsu.govt.nz/current-nsu-programmes/2097.aspx

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 eliminating abnormal markers, while samples taken later may result in a lost window for early intervention, as severe forms of some metabolic disorders may be fatal within 7–10 days, but may not show any signs or symptoms until irreversible damage has occurred [19]. Blood samples are usually taken by heel prick, with blood being 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 [19]. At the time the sample is taken, parents are asked whether they wish the card to be stored for possible future use, or returned to them.

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 [27]. In response to concerns regarding the late age of diagnosis of congenital hearing losses (average age 35.1 months when screening was based on the presence of risk factors [20]), the Government in its 2006 Budget, announced a funding package ($16 million over four years) to establish a National Newborn Hearing Screening Programme. The Programme was rolled out progressively across the country during 2007–2010, with screening now underway in all 20 DHBs [28].

Conditions Detectable by Antenatal and Newborn Screening

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

CONGENITAL ANOMALIES EVIDENT AT BIRTH

Introduction

The following section uses the National Minimum Dataset to review the number of congenital anomalies evident at birth in Māori babies, as well as the number of Māori babies born with one or more congenital anomalies. Subsequent chapters consider cardiovascular anomalies, Down syndrome and neural tube defects in more detail.

Note: This 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).

Background

In 2006, the Household Disability Survey found that of an estimated 28,200 Māori children aged 0–14 years who were disabled, 14,100 (50%) had a disability that had been present since birth [29]. However, little other information is available on the prevalence of congenital conditions in Māori babies, as while New Zealand has a Birth Defects Registry (NZBDR) which has collected data on babies with birth defects born or treated in public hospitals since 1977, no ethnic specific analyses are contained in NZBDR reports [30]. The NZBDR however, does contribute New Zealand data on 39 categories of birth defects to the International Clearinghouse for Birth Defects Surveillance Research (ICBDSR) in Rome [31]. New Zealand data published in the 2010 ICBDSR report indicated an overall rate of congenital anomalies in 2004–2008 of around two anomalies per 100 births [31].

A number of research reports, however, have suggested that Māori babies may have higher rates for some congenital anomalies. For example, the prevalence of talipes equinovarus or club foot is higher for Māori (6–7 per 1,000) than for Europeans (1–3 per 1,000), with genetic factors being thought to play a role [32]. Similarly, a study of cleft lip and palate in the Canterbury/West Coast region during 2000–2009 found that while the prevalence of cleft lip (+/- palate) was similar for Māori and non-Māori babies, Māori babies had higher rates of isolated cleft palate than European babies (1.35 per 1,000 vs. 0.88 per 1,000 live births) [33].

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 7**.

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 that 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 time series 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.

Distribution in Māori Babies

Number and Proportion of Babies

In New Zealand during 2008–2012, on average 559 Māori babies per year (4.2% of all Māori births), had one or more congenital anomalies identified at birth, with rates for Māori babies being *significantly* lower than for non-Māori non-Pacific babies (RR 0.89 95% CI 0.85–0.92) (**Table 7**).

Table 7. Babies with Congenital Anomalies Evident at Birth by Ethnicity, New Zealand Hospital Births 2008–2012

Variable	Number of Babies: Total 2008–2012	Number of Babies: Annual Average	Rate per 100,000 Births	Rate Ratio	95% CI
Babies with Congenital Anomalies					
Māori	2,795	559	4,190	0.89	0.85–0.92
non-Māori non-Pacific	9,542	1,908	4,731	1.00	

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 Anomaly Type

Amongst Māori babies 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. non-neoplastic nevus), through to anomalies that were incompatible with life (e.g. anencephaly/encephalocele). When interpreting the information in **Table 8** and **Table 9**, it must be remembered that the figures presented relate to the number of anomalies identified, rather than the number of babies, with many babies having more than one anomaly.

Table 8. Congenital Anomalies Evident at Birth, Māori Hospital Births 2008–2012 (1 of 2)

Congenital Anomaly	Number: Total 2008–2012	Number: Annual Average	Anomalies per 100,000 Births*
Māori Babies			
Anencephaly/Encephalocele	6	1.2	9.0
Microcephaly	8	1.6	12.0
Congenital Hydrocephalus	12	2.4	18.0
Other Brain Malformations	52	10.4	78.0
Spina Bifida	15	3.0	22.5
Other Spinal Cord Malformations	5	1.0	7.5
Other CNS Malformations	8	1.6	12.0
Total Malformations of the Nervous System	106	21.2	158.9
Eyelid/Lacrimal/Eye/Orbit Malformations	23	4.6	34.5
Ear Malformations Impairing Hearing	5	1.0	7.5
Accessory Auricle	66	13.2	98.9
Other Ear Malformations	39	7.8	58.5
Other Face/Neck Malformations	15	3.0	22.5
Total Malformations of Eye, Ear, Face and Neck	148	29.6	221.9
Malformations Cardiac Chambers/Connections	56	11.2	84.0
Ventricular Septal Defect	107	21.4	160.4
Atrial Septal Defect	127	25.4	190.4
Atrioventricular Septal Defect	10	2.0	15.0
Tetralogy of Fallot	31	6.2	46.5
Pulmonary/Tricuspid Valve Malformations	37	7.4	55.5
Aortic/Mitral Valve Malformations	27	5.4	40.5
Other Heart Malformations	63	12.6	94.5
Patent Ductus Arteriosus	355	71.0	532.2
Malformations Great Arteries (Excluding PDA)	55	11.0	82.5
Malformations Great Veins	10	2.0	15.0
Other Peripheral Vascular Malformations	38	7.6	57.0
Other Circulatory Malformations	4	0.8	6.0
Total Malformations of the Circulatory System	920	184.0	1,379.2
Nose Malformations	12	2.4	18.0
Trachea/Bronchus Malformations	6	1.2	9.0
Lung Malformations	27	5.4	40.5
Larynx Malformations	8	1.6	12.0
Other Respiratory Malformations	<3	s	s
Total Malformations of the Respiratory System	54	10.8	81.0
Cleft Palate	64	12.8	95.9
Cleft Lip	11	2.2	16.5
Cleft Palate and Lip	27	5.4	40.5
Total Cleft Lip and Palate	102	20.4	152.9

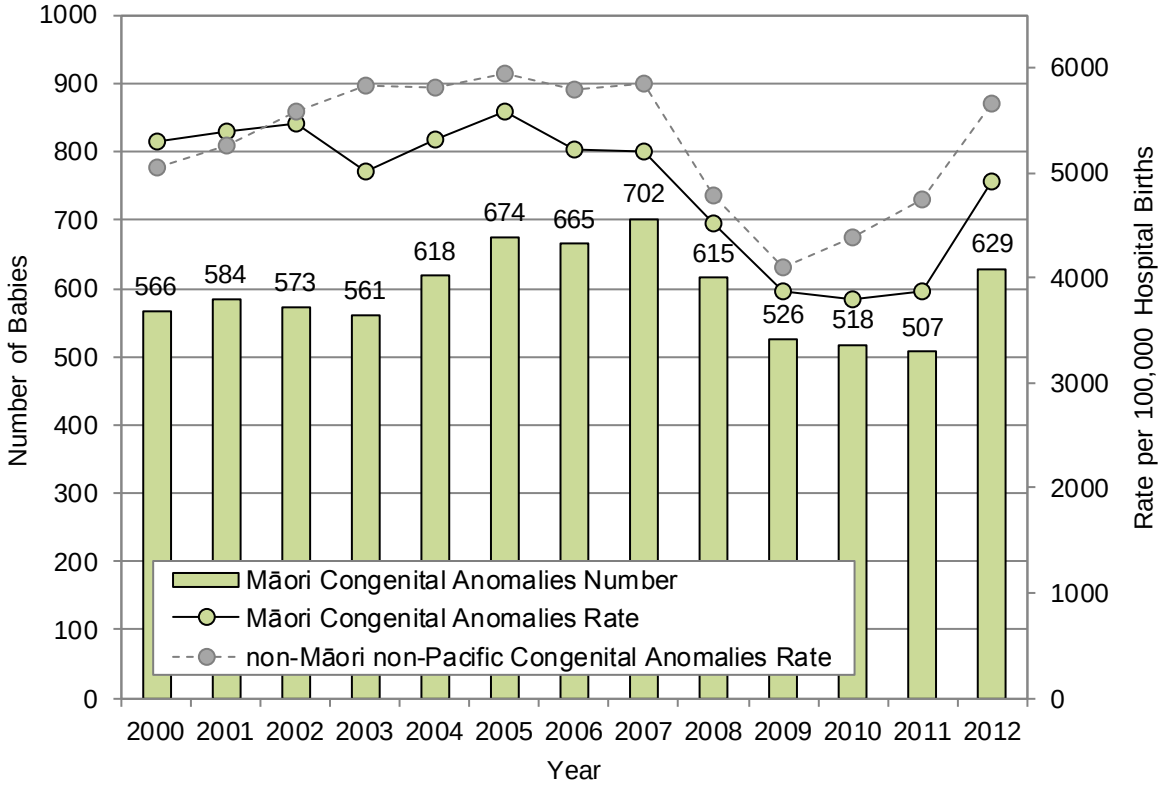
Source: National Minimum Dataset; Numerator: Hospitalisations with event type = birth and a congenital anomaly in any of first 15 diagnoses; Denominator: Hospitalisations with event type = birth; Note: *Anomalies per 100,000 births = number of anomalies rather than number of babies, as some babies have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

Table 9. Congenital Anomalies Evident at Birth, Māori Hospital Births 2008–2012 (2 of 2)

Congenital Anomaly	Number: Total 2008–2012	Number: Annual Average	Anomalies per 100,000 Births*
Māori Babies			
Ankyloglossia (Tongue Tie)	430	86.0	644.6
Tongue/Mouth/Pharynx Malformations	12	2.4	18.0
Oesophagus/Upper Alimentary Malformations	15	3.0	22.5
Intestinal Malformations	49	9.8	73.5
Other Digestive Malformations	9	1.8	13.5
Total Malformations of the Digestive System	515	103.0	772.1
Female Genital Malformations	14	2.8	21.0
Undescended Testicle	222	44.4	332.8
Hypospadias	86	17.2	128.9
Other Male Genital Malformations	26	5.2	39.0
Indeterminate Sex/Pseudohermaphrodism	7	1.4	10.5
Total Malformations of the Genital Organs	355	71.0	532.2
Renal Agenesis/Reduction Defects	20	4.0	30.0
Cystic Kidney Disease	28	5.6	42.0
Renal Pelvis Obstruction/Ureter Malformations	51	10.2	76.5
Other Kidney/Urinary Malformations	65	13.0	97.4
Total Malformations of the Urinary System	164	32.8	245.9
Congenital Dislocation Hip	6	1.2	9.0
Other Deformities Hip	66	13.2	98.9
Foot Deformities	482	96.4	722.6
Other Musculoskeletal Malformations	129	25.8	193.4
Polydactyly	74	14.8	110.9
Syndactyly	26	5.2	39.0
Reduction Defects/Other Limb Malformations	38	7.6	57.0
Osteochondrodysplasia	6	1.2	9.0
Skull/Facial Bones/Spine/Thorax Malformations	43	8.6	64.5
Total Malformations of the Musculoskeletal System	870	174.0	1,304.2
Non-Neoplastic Naevus	106	21.2	158.9
Other Skin Malformations	118	23.6	176.9
Breast Malformations	4	0.8	6.0
Other Integument Malformations	92	18.4	137.9
Other Malformations	84	16.8	125.9
Total Other Congenital Malformations	404	80.8	605.7
Down Syndrome	47	9.4	70.5
Edwards and Patau Syndromes	16	3.2	24.0
Monosomies and Autosomal Deletions/Other Rearrange	6	1.2	9.0
Other Chromosome Anomalies	13	2.6	19.5
Total Chromosomal Anomalies	82	16.4	122.9

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 7. Babies with Congenital Anomalies Evident at Birth by Ethnicity, 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; Rate per 100,000 refers to number of babies with one or more anomalies, rather than number of anomalies

Trends by Ethnicity

In New Zealand, the number of Māori babies with one or more congenital anomalies identified at birth increased gradually during the mid 2000s, reached a peak in 2007 and then declined, with the most rapid declines occurring between 2007 and 2011. Numbers then increased again in 2012 (**Figure 7**). It remains unclear however, whether the large declines seen in both numbers and rates between 2007 and 2011, and their subsequent rebound, reflect real changes in the number of Māori babies born with congenital anomalies (less likely), 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

Additional information on the distribution of congenital anomalies at the time of birth in New Zealand is available from *the Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand* [12]. In brief, this report found:

Distribution by Maternal Age

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.

Distribution by NZ Deprivation Index Decile and Gender

During 2008–2012, the proportion of babies with one or more congenital anomalies identified at birth was *significantly* higher for males, and for those from the least deprived (NZDep06 deciles 1–2 vs. 5–10) areas.

CONGENITAL HEART DISEASE

Introduction

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

Background

While there is little information on the prevalence of congenital heart disease (CHD) in Māori babies, some overseas estimates give a birth prevalence of severe CHD (e.g. transposition of the great arteries, Tetralogy of Fallot) of 2.5–3.0 per 1,000 live births [34], with moderately severe forms of CHD (e.g. large atrial septal defects, complex ventricular septal defects) accounting for another 3 per 1,000 live births. The overall prevalence may increase to 75 per 1,000 if minor anomalies (e.g. small ventricular septal defects, atrial septal defects, or patent ductus arteriosus) are included. Other studies however, have yielded much lower prevalence estimates, ranging from 3–6 per 1,000 live births [35].

The causes of CHD remain largely unknown, with only around 15% of cases being traced to a known cause. Such causes include chromosomal anomalies (e.g. Down syndrome), 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 risk factors such as maternal diabetes, obesity and alcohol use, and specific drugs [35]. Genetic factors may also play a role, with the risk of recurrence being 1–6% if one sibling is affected, and up to 10% if two siblings are [35].

While early detection and timely management are crucial, research suggests that up to 25% of babies with severe forms of CHD may be discharged from hospital undiagnosed. Pulse oximetry, if used in conjunction with a clinical examination prior to discharge, may improve the detection rate of some forms of CHD [18]. However a number of babies with serious cardiovascular anomalies are still missed in the neonatal period and antenatal screening has thus become an established practice in many centres [18].

Data Source and Methods

Definition

1. Number of cardiovascular anomalies evident at birth (by anomaly type)

2. Number of babies with one or more cardiovascular anomalies evident at birth (by anomaly type)

Data Source

1. National Minimum Dataset

Numerator: Hospital Admissions with event type = birth and a cardiovascular anomaly (ICD-10 Q20-Q28) listed in any of the first 15 diagnoses.

Denominator: All hospital admissions with event type = birth

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 (CVS) 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 that are difficult to detect on routine newborn examination.

Note 2: In New Zealand, 64.2% of patent ductus arteriosus (PDA) identified during 2008–2012 were in preterm babies (<37 weeks' gestation) who had no other CVS anomalies. Prematurity is known to increase the risk of PDA as a result of increased exposure to hypoxia and underdeveloped heart and lungs. During 2008–2012, 23.8% of all CVS anomalies were isolated PDAs in preterm infants, many of which would not have had a PDA had they been born at term. As the possibility that any analysis of risk factors for CVS 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 CVS 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 7**.

Distribution in Māori Babies

Cardiovascular Anomalies Evident at Birth

In New Zealand during 2008–2012, patent ductus arteriosus (PDA) was the most frequent cardiovascular anomaly identified at birth in Māori babies, with 65.6% of PDAs being in preterm babies (<37 weeks' gestation) who had no other cardiovascular anomalies (see Methods section for rationale for exclusion of these cases from subsequent analyses). Atrial septal and ventricular septal defects were the next most frequent cardiovascular anomalies identified (**Table 10**).

Table 10. Cardiovascular Anomalies Evident at Birth, Māori Hospital Births 2008–2012

Cardiovascular Anomaly	Number: Total 2008–2012	Number: Annual Average	Anomalies per 100,000 Births*
Māori Babies			
Malformations Cardiac Chambers/Connections	56	11.2	84.0
Ventricular Septal Defect	107	21.4	160.4
Atrial Septal Defect	127	25.4	190.4
Atrioventricular Septal Defect	10	2.0	15.0
Tetralogy of Fallot	31	6.2	46.5
Pulmonary/Tricuspid Valve Malformations	37	7.4	55.5
Aortic/Mitral Valve Malformations	27	5.4	40.5
Other Heart Malformations	63	12.6	94.5
Patent Ductus Arteriosus*	355	71.0	532.2
Malformations Great Arteries (Excluding PDA)	55	11.0	82.5
Malformations Great Veins	10	2.0	15.0
Other Peripheral Vascular Malformations	38	7.6	57.0
Other Circulatory Malformations	4	0.8	6.0
Total Malformations of the Circulatory System	920	184.0	1,379.2

Source: National Minimum Dataset; Numerator: Hospital Admissions with event type = birth and a CVS 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 233 cases of Isolated PDA in preterm infants (<37 weeks), which have been excluded in subsequent analyses

Table 11. Babies with Cardiovascular Anomalies Evident at Birth by Ethnicity, New Zealand Hospital Births 2008–2012

Ethnicity	Number of Babies: Total 2008–2012	Number of Babies: Annual Average	Rate per 100,000 Births	Rate Ratio	95% CI
Cardiovascular Anomalies					
Māori	359	72	538.2	0.96	0.85–1.08
non-Māori non-Pacific	1,129	226	559.7	1.00	

Source: National Minimum Dataset; Numerator: Hospital Admissions with event type = birth and a CVS anomaly in any of the first 15 diagnoses; Denominator: All hospital admissions with event type = birth; 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

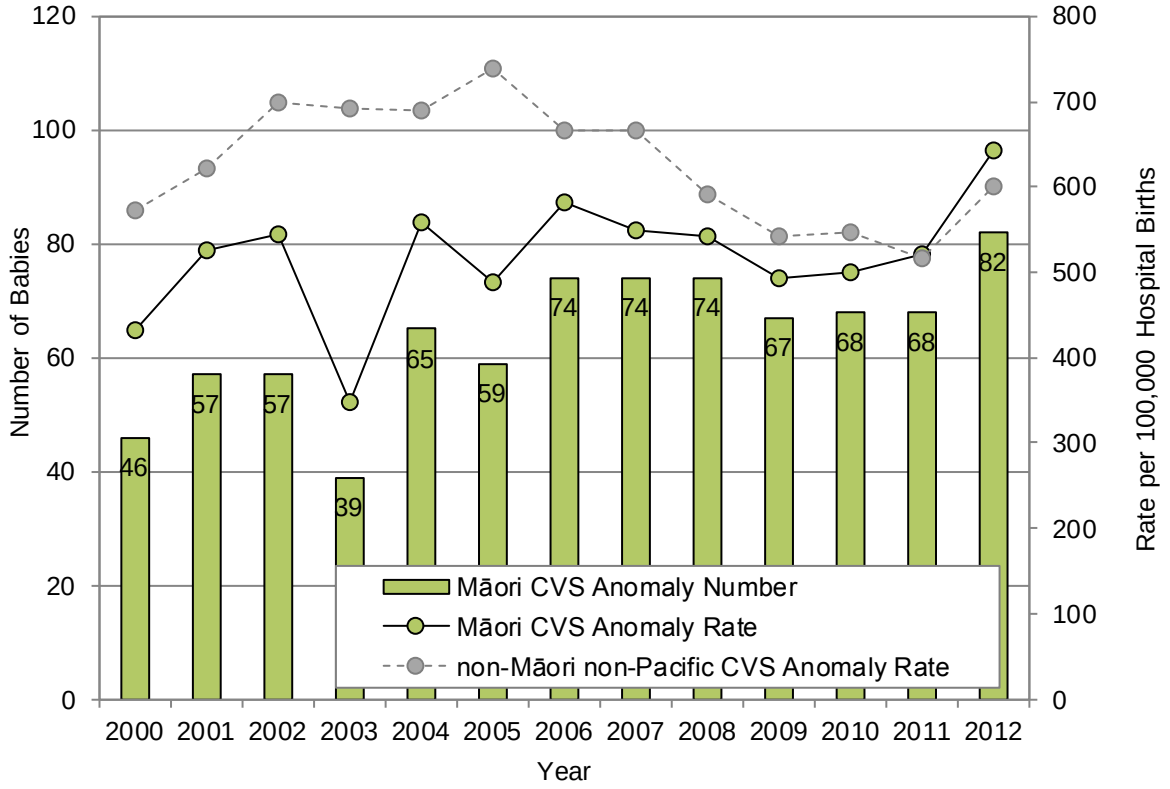
Number of Babies

In New Zealand during 2008–2012, on average 72 Māori babies per year had one or more cardiovascular anomalies identified at birth, with rates not being *significantly* different from those of non-Māori non-Pacific babies (RR 0.96 95% CI 0.85–1.08) (**Table 11**).

Trends by Ethnicity

In New Zealand during 2000–2012 the proportion of Māori babies born with one or more cardiovascular anomalies fluctuated. On average 64 Māori babies each year were born with one or more cardiovascular anomalies during this period (**Figure 8**).

Figure 8. Babies with Cardiovascular Anomalies Evident at Birth by Ethnicity, New Zealand Hospital Births 2000–2012



Source: National Minimum Dataset; Numerator: Hospital Admissions with event type = birth and a CVS 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

New Zealand Distribution and Trends

Additional information on the distribution of cardiovascular anomalies in New Zealand is available from *the Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand* [12]. In brief, this report found:

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 that were 1.81 (95% CI 1.42–2.32) times higher than those whose mothers gave birth in their teens.

Distribution by NZ Deprivation Index Decile and Gender

During 2008–2012, the proportion of babies born with cardiovascular anomalies was *significantly* higher for males and for babies from the least deprived (NZDep06 deciles 1–2 vs. deciles 5–10) areas.

DOWN SYNDROME

Introduction

The following section uses the National Minimum Dataset to review the number of Māori babies born with Down syndrome.

Background

Down syndrome is the most common chromosomal anomaly in newborn babies and results from extra genetic material from chromosome 21 [36]. 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, vision problems, obstructive sleep apnoea, congenital heart defects, and seizures [37].

While there is little information on the prevalence of Down Syndrome in Māori children, in New Zealand, between 50 and 80 babies with Down syndrome are born each year [38], with the risk rising steeply with increasing maternal age [36]. 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 [39].

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 [24]. The National Screening Unit recommends 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 [24]. Women whose test results indicate an increased risk (> 1 in 300) are offered referral for more definitive testing [24].

There are also a number of guidelines on the clinical care of children with Down syndrome [37, 39-41], including one from the Ministry of Health [42]. 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.

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

Numerator: 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 Syndrome (Q96), Other Sex Chromosome Anomalies Female Phenotype (Q97), Sex Chromosome Anomalies Male Phenotype (Q98), Other Chromosome Anomalies (Q99)

2. National Minimum Dataset

Numerator: Hospital admissions with event type = birth and Down syndrome (Q90) listed in any of the first 15 diagnoses.

For this indicator, the unit of analysis is the number of babies with Down syndrome identified at birth.

Denominator: All hospital admissions with event type = birth

Notes on Interpretation

Note: This analysis includes all admissions 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 Down syndrome was overlooked at the time of discharge, but who re-presented shortly thereafter.

Distribution in Māori Babies

Chromosomal Anomalies Evident at Birth

Amongst Māori babies during 2008–2012, Down syndrome was the most frequent chromosomal anomaly identified at birth, accounting for 70.5% of the chromosomal anomalies identified during this period. Such 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 12**).

Table 12. Chromosomal Anomalies Evident at Birth, Māori Hospital Births 2008–2012

Chromosomal Anomaly	Number: Total 2008–2012	Number: Annual Average	Anomalies per 100,000 Births*
Māori Babies			
Down Syndrome	47	9.4	70.5
Edwards and Patau Syndromes	16	3.2	24.0
Monosomies and Autosomal Deletions/Other Rearrange	6	1.2	9.0
Other Chromosome Anomalies	13	2.6	19.5
Total Chromosomal Anomalies	82	16.4	122.9

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

Number of Babies

In New Zealand during 2008–2012, on average 9 Māori babies per year had Down syndrome identified at birth, with rates, while lower, not being *significantly* different from those of non-Māori non-Pacific babies (RR 0.79 95% CI 0.58–1.09) (**Table 13**).

Table 13. Babies with Down Syndrome Evident at Birth by Ethnicity, New Zealand Hospital Births 2008–2012

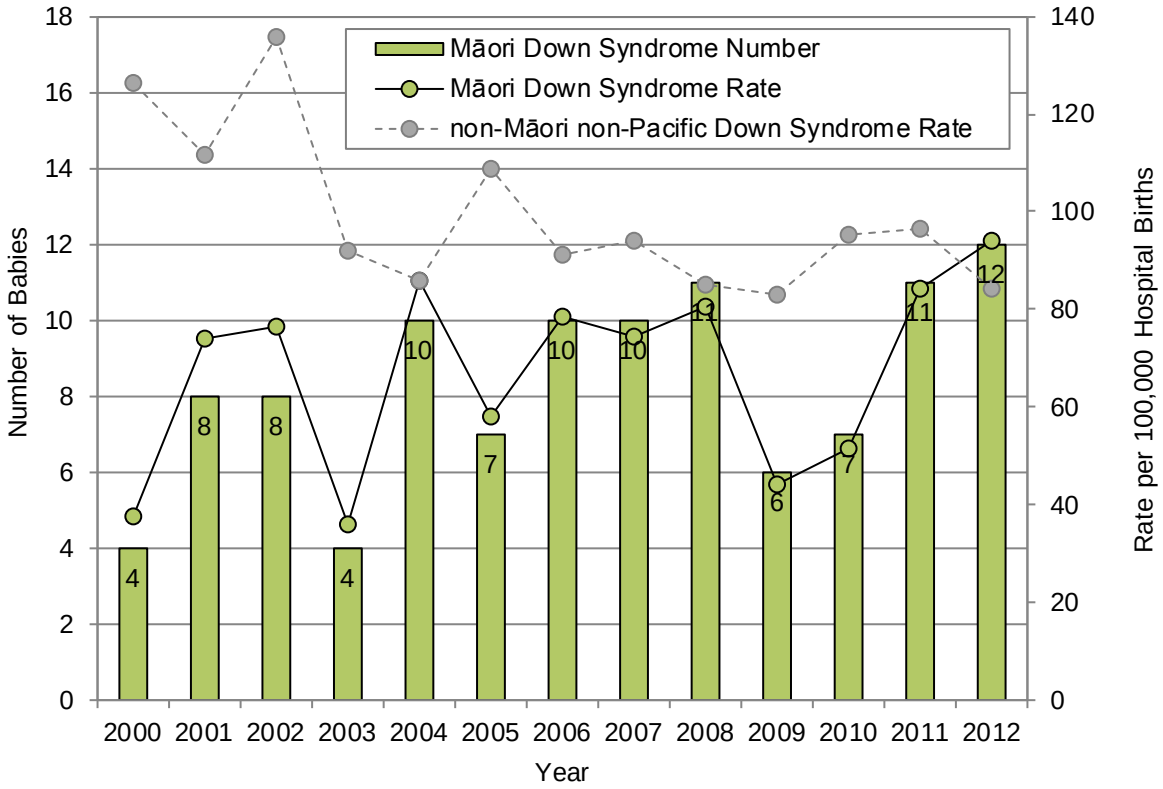
Ethnicity	Number of Babies: Total 2008–2012	Number of Babies: Annual Average	Rate per 100,000 Births	Rate Ratio	95% CI
Down Syndrome					
Māori	47	9	70.5	0.79	0.58–1.09
non-Māori non-Pacific	179	36	88.7	1.00	

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

Trends by Ethnicity

In New Zealand during 2000–2012, on average 8.3 Māori babies per year were identified as having Down syndrome at the time of birth, with numbers fluctuating during this period (**Figure 9**).

Figure 9. 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

New Zealand Distribution and Trends

Additional information on the distribution of Down syndrome at the time of birth in New Zealand is available from *the Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand* [12]. In brief, this report found:

Distribution by Maternal Age, NZ Deprivation Index Decile and Gender

During 2008–2012, there were no statistically significant socioeconomic (as measured by NZDep06 quintile) 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.

NEURAL TUBE DEFECTS

Introduction

The following section uses the National Minimum Dataset to review the number of Māori babies born with neural tube defects.

Background

Neural tube defects (NTDs) have their origin very early in pregnancy. A localised thickening of embryonic cells known as the neural plate emerges around 18 days after conception. This elongates, develops a central groove, and then folds to produce the neural tube, which closes at around 25–27 days [43]. The walls of this neural tube then thicken to produce the brain and spinal cord, with the closed neural tube stimulating the development of the overlying skull and spine. When the neural tube fails to close, the overlying bony structures fail to form, resulting in a NTD [44]. NTDs may occur anywhere along the neural axis, with cranial defects resulting in anencephaly (absence of a major portion of the brain, skull and scalp) and encephalocele (the sac-like protrusion of the brain and meninges through openings in the skull). Defects further down the neural tube result in spina bifida.

In New Zealand there is little recent information on the prevalence of NTDs in Māori babies, although older studies suggest that rates were lower for Māori than for non-Māori parents in the late 1970s and early 1980s [45, 46]. The reason for these ethnic differences are unclear however, with the aetiology of NTDs being complex, and generally thought to arise from a combination of genetic and environmental factors.

A number of studies have also shown that folic acid supplementation prior to or at conception can reduce the risk of NTDs by up to two thirds [43] [47]. In many countries, including the U.S., Canada and Australia, there is thus mandatory fortification of staple foods (flours) with folic acid [48]. In New Zealand, voluntary fortification of certain foods (cereal products, bread and fruit juice) with folic acid is permitted. The Ministry of Health also recommends that women take folic acid supplements for at least four weeks prior to and for 12 weeks after conception, with a view to decreasing their risk of having a baby with a NTD [49, 50].

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 NTD 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.

Distribution in Māori Babies

Distribution by Anomaly Type

Amongst Māori babies during 2008–2012, 21 neural tube defects (NTDs) were identified at the time of birth (anencephaly/encephalocele (n=6), spina bifida (n=15)), with on average 4.2 NTDs being identified per year. NTDs accounted for 19.8% of all nervous system anomalies 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 14**).

Table 14. Nervous System Anomalies Evident at Birth, Māori Hospital Births 2008–2012

Nervous System Anomaly	Number: Total 2008–2012	Number: Annual Average	Anomalies per 100,000 Births*
Māori Babies			
Anencephaly/Encephalocele	6	1.2	9.0
Microcephaly	8	1.6	12.0
Congenital Hydrocephalus	12	2.4	18.0
Other Brain Malformations	52	10.4	78.0
Spina Bifida	15	3.0	22.5
Other Spinal Cord Malformations	5	1.0	7.5
Other CNS Malformations	8	1.6	12.0
Total Malformations of the Nervous System	106	21.2	158.9

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

Number of Babies

In New Zealand during 2008–2012, on average 4.2 Māori babies per year had a neural tube defect identified at birth, with rates, while higher, not being *significantly* different from those of non-Māori non-Pacific babies (RR 1.41 95% CI 0.84–2.37) (**Table 15**).

Table 15. Babies with Neural Tube Defects Evident at Birth by Ethnicity, New Zealand Hospital Births 2008–2012

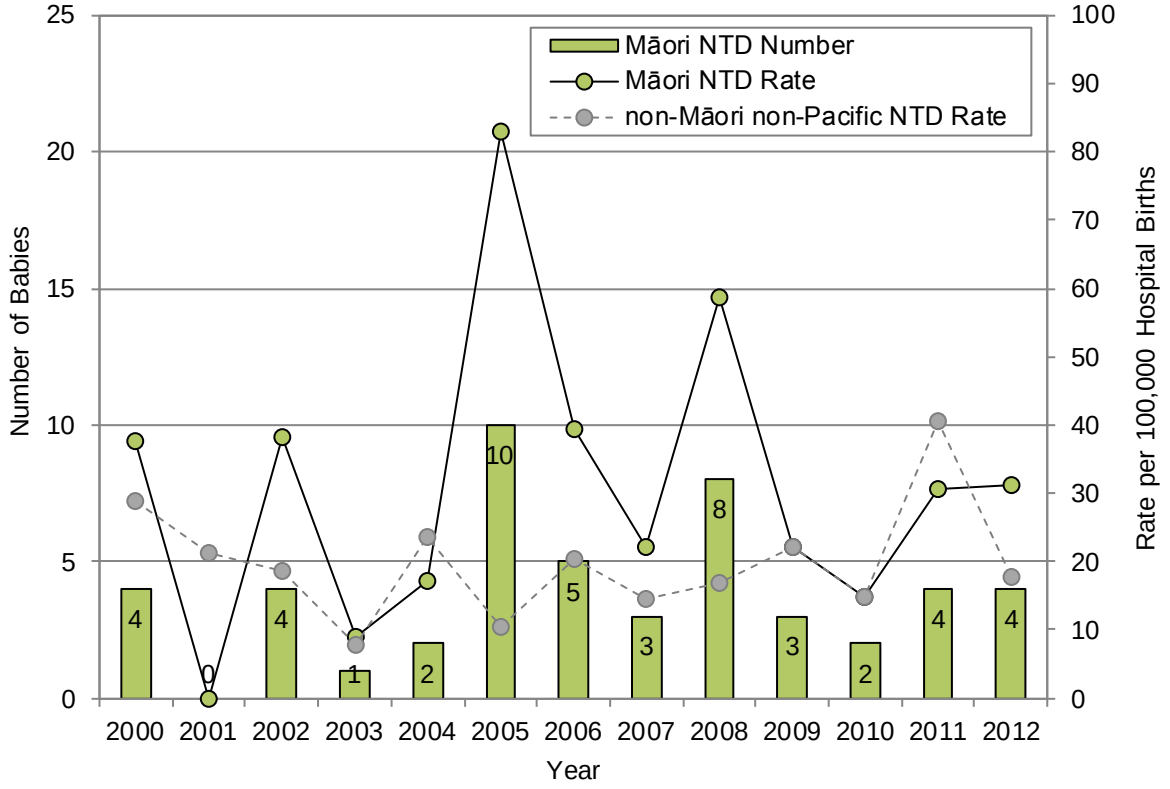
Ethnicity	Number of Babies: Total 2008–2012	Number of Babies: Annual Average	Rate per 100,000 Births	Rate Ratio	95% CI
Neural Tube Defects					
Māori	21	4.2	31.5	1.41	0.84–2.37
non-Māori non-Pacific	45	9.0	22.3	1.00	

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

Trends by Ethnicity

In New Zealand during 2000–2012, on average 3.8 Māori babies per year had one or more neural tube defects identified at birth. Large year to year variations (likely as a result of small numbers) made trends difficult to interpret, although rates for Māori babies were generally higher than for non-Māori non-Pacific babies during the mid-2000s (**Figure 10**).

Figure 10. Babies with Neural Tube Defects Evident at Birth by Ethnicity, 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

New Zealand Distribution and Trends

Additional information on the distribution of neural tube defects at the time of birth in New Zealand is available from *the Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand* [12]. In brief, this report found:

Distribution by Maternal Age

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).

Distribution by NZ Deprivation Index Decile and Gender

During 2008–2012, there were no *significant* 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 babies born into the most deprived (NZDep06 deciles 9–10) areas

OTHER DISABILITIES

PERMANENT HEARING LOSS

Introduction

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, with the limited information by ethnicity that is available being presented part way through the section. This is followed by a review of information from the most recent Universal Newborn Hearing Screening and Early Intervention Programme (NZHSEIP) monitoring reports, on the number of Māori babies diagnosed with permanent congenital hearing losses in recent years.

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, with Ministry of Health funding resuming 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 [51].

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 [52], or from the 2010–2012 Deafness Notification Reports produced by Digby et al. [51]. 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 [52]:

- 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 audiological guidance also suggested that while hearing losses arising from atresia, congenital ossicular fixation, meningitis and other acquired hearing losses should be included, hearing losses that could be fixed by the use of grommets (e.g. hearing losses associated with otitis media) should be excluded [51].

Additional Notes on Interpretation

DND data are reported by year of notification, rather than year of identification. 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 16**). 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 [51].

Table 16. Notifications to the Deafness Notification Database by Degree of Hearing Loss, Using the Old Notification Criteria, New Zealand 2001–2004 and 2010–2012

Degree of Hearing Loss	Proportion of Cases Notified (%)							
	2001	2002	2003	2004		2010	2011	2012
Mild (26–40 dBHL)	47	47	56	43		59	60	54
Moderate (41–65 dBHL)	35	39	33	34		33	28	42
Severe (66–95 dBHL)	10	9	6	15		4	5	1
Profound (96+ dBHL)	8	5	5	7		5	3	3

Source: Deafness Notification Database via Digby et al. [51]; 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, 73 children notified to the DND identified as Māori, 103 as European, 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.

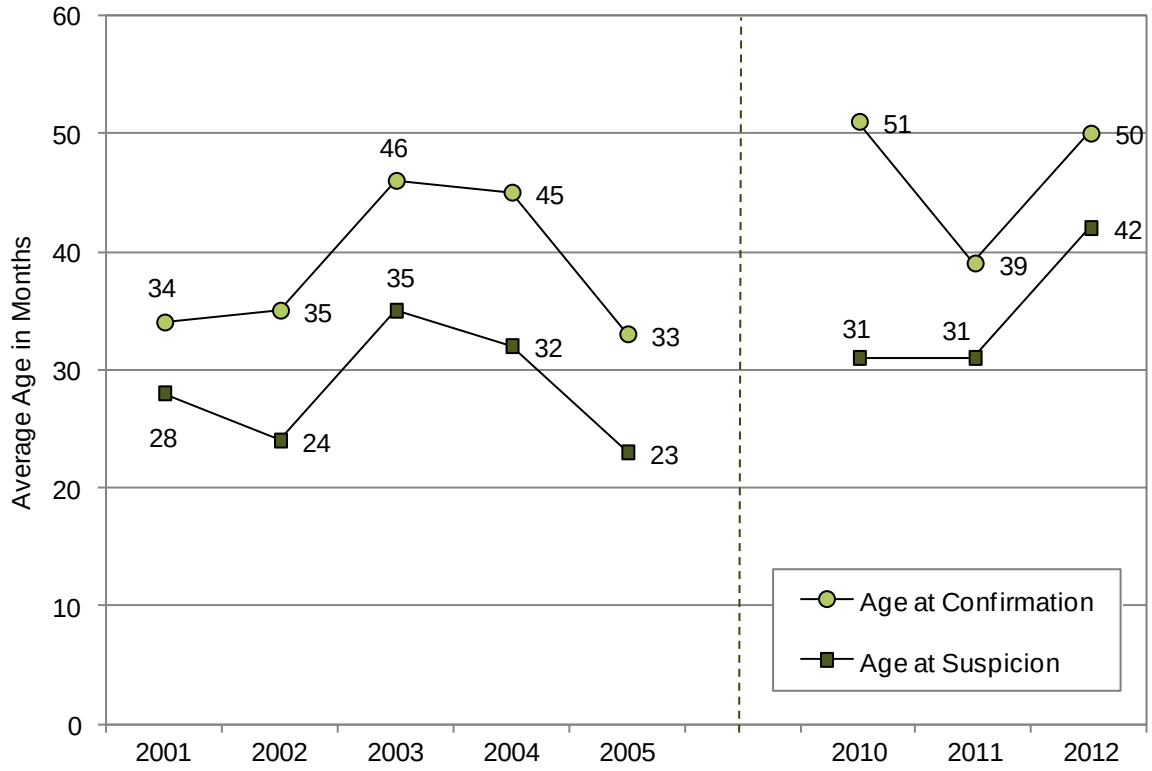
Average Age at Suspicion and Confirmation of Hearing Loss

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 11**).

Number of Notifications by Age

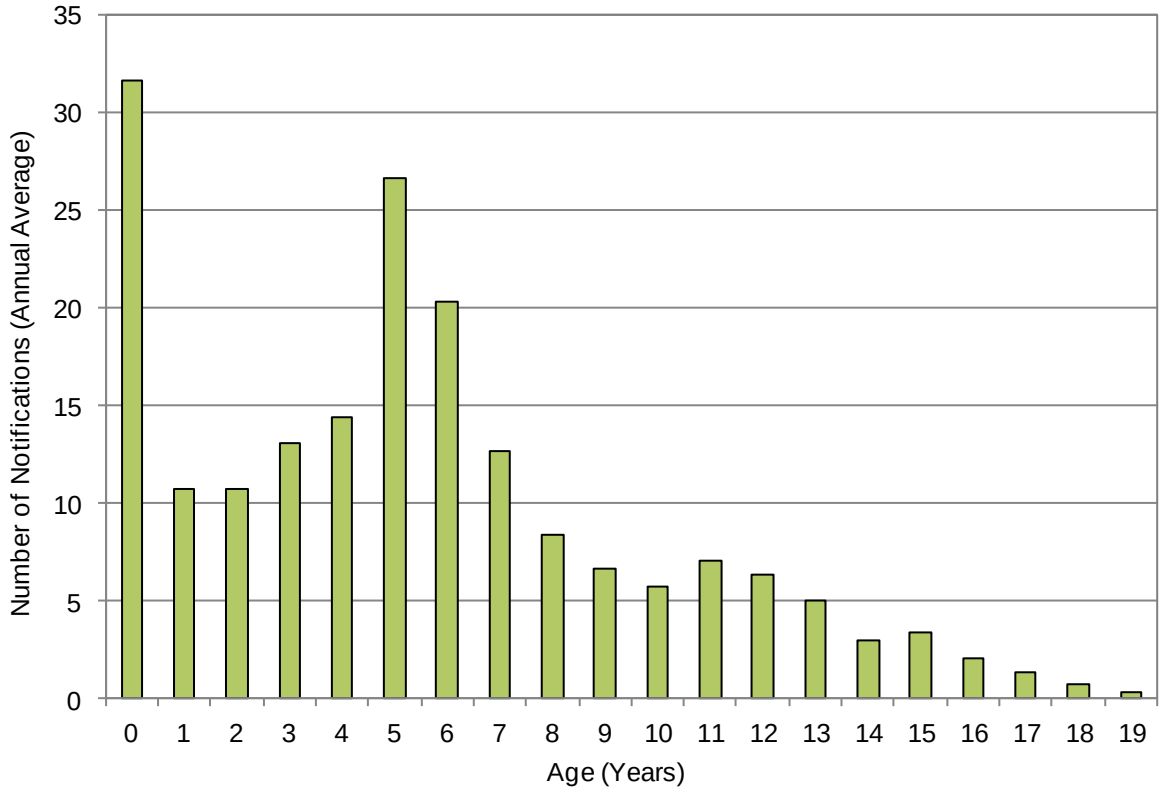
During 2010–2012, the largest numbers of notifications to the DND were for babies under one year of age, likely as a result of newborn hearing screening. Numbers then dropped away during the preschool years. A second peak was evident at five years of age, likely as a result of the B4 School Check, with numbers then falling away again 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 lead to a later age of diagnosis of permanent hearing loss (**Figure 12**). Further, in the 2012 DND Report [51] the peak in notifications in babies under one 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 [51].

Figure 11. 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. [51]; Note: In order to ensure comparability with previous DND criteria, mild, acquired, unilateral, and overseas cases have been excluded

Figure 12. Number of Notifications to the Deafness Notification Database by Age, New Zealand 2010–2012



Source: Deafness Notification Database via Digby et al. [51]

Distribution by Region

Table 17 reviews the number of notifications received by the Deafness Notification Database by region using its old criteria during 1998–2004, while **Table 18** reviews the number of notifications received by DHB using the new criteria during 2010–2012.

Table 17. Number of Notifications Meeting the Old Criteria for Inclusion in the Deafness Notification Database by Region of Residence, New Zealand 1998–2004

Region of Residence	Notification Year						
	1998	1999	2000	2001*	2002	2003	2004+
Northland	10	8	11	10	5	7	10
Auckland Region	21	35	40	74	36	52	37
Waikato	7	13	9	19	10	9	15
Lakeland	3	<3	0	3	3	3	6
Bay of Plenty	10	6	4	21	6	12	9
Tairawhiti	3	0	<3	3	<3	<3	5
Taranaki	<3	<3	<3	<3	3	3	8
Hawke's Bay	<3	<3	<3	31	5	4	5
Manawatu	3	3	0	12	7	12	24
Wellington	3	10	5	8	12	17	5
Nelson Marlborough	<3	3	<3	<3	3	4	4
West Coast	0	0	0	0	<3	<3	<3
Canterbury	0	3	7	10	12	9	10
South Canterbury	0	<3	<3	4	<3	3	3
Otago	0	<3	8	5	5	3	7
Southland	<3	3	<3	0	3	4	6
New Zealand Total	65	90	92	202	113	144	155

Source: National Audiology Centre [52]; 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

Table 18. Number of Notifications Meeting New Criteria for Deafness Notification Database by District Health Board, New Zealand 2010–2012

DHB	2010	2011	2012
Notifications to Deafness Notification Database			
Northland	12	5	15
Waitemata	4	9	6
Auckland	10	15	7
Counties Manukau	25	20	26
Waikato	15	13	15
Bay of Plenty	13	12	3
Lakes	<3	9	4
Tairawhiti	<3	<3	<3
Taranaki	6	5	8
Hawke's Bay	9	11	13
MidCentral	4	<3	6
Whanganui	0	<3	3
Hutt Valley	5	4	10
Capital & Coast	24	24	8
Wairarapa	0	5	<3
Nelson Marlborough	<3	4	10
South Canterbury	<3	3	7
Canterbury	44	27	23
West Coast	<3	<3	0
Southern	<3	15	23
New Zealand	180	187	191

Source: Deafness Notification Database via Digby et al. [51].

Newborn Hearing Screening

In response to concerns regarding the late age of diagnosis of congenital hearing losses in New Zealand (average age 35.1 months when screening was based on the presence of risk factors [52]), the Government in its 2006 Budget, announced a funding package to establish a Universal Newborn Hearing Screening and Early Intervention Programme. The UNHSEIP was rolled out during 2007–2010, with all DHBs now offering hearing screening to the families of newborn babies [28].

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, 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 [28].

The following section reviews data from the UNHSEIP's monitoring reports [28], with the most recent including data for the 6-month period from 1st October 2011–31st March 2012.

Data Sources and Methods

Indicators

1. *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

2. 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

3. 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.

4. *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

5. Number of newborns with a permanent congenital hearing loss

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

6. Number of newborns with a conductive hearing loss

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

Universal Newborn Hearing Screening and Early Intervention Programme Monitoring Reports, available for download at http://www.nsu.govt.nz/health-professionals/4627.aspx

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: 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.

Distribution by Ethnicity

During 1st October 2011–31st March 2012, of those Māori babies who completed newborn hearing screening, 89.8% did so within one month, with 2.3% of those completing screening receiving an audiology referral. Of the Māori babies who passed screening, a further 6.8% were deemed to have risk factors for delayed onset/progressive hearing loss which warranted follow up (**Table 19**).

Audiology Referrals and Outcomes

New Zealand Distribution

In New Zealand during 1st October 2011–31st 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, and 73 as having a conductive hearing loss.

Distribution by Ethnicity

During 1st October 2011–31st March 2012, 100 Māori babies commenced audiology assessment, with 74.5% of those who completed their audiology assessment doing so by 3 months of age. During this period, 12 Māori babies were identified as having a permanent congenital hearing loss, and 32 a conductive hearing loss (**Table 19**).

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. Of these, 32 were Māori babies (**Table 20**).

Table 19. Newborn Hearing Screening and Audiology Indicators by Ethnicity, New Zealand 1st October 2011–31st March 2012

Ethnicity	Completed Screening ≤1 Month*(%)	Referrals to Audiology* (%)	Targeted for Follow Up* (%)	Commenced Audiology (Number)	Completed Audiology ≤3 Months (%)	Permanent Congenital Hearing Loss (Number)	Conductive Hearing Loss (Number)
	Newborn Hearing Screening			Newborn Audiology			
Māori	89.8	2.3	6.8	100	74.5	12	32
Pacific	89.4	1.7	4.5	16	92.9	1	2
Asian	94.6	1.2	3.0	22	100.0	1	7
European	94.3	1.2	4.8	109	91.7	16	30
Unspecified	89.6	3.3	4.1	4	100.0	0	2
Other Ethnic Groups	94.0	0.9	2.7	3	100.0	0	0
Total	92.8	1.5	4.9	254	85.9	30	73

Source: National Screening Unit 2012 [28]; Note: *See Methods for Indicator Definitions

Table 20. Number of Babies Identified by Newborn Hearing Screening as Having Permanent Congenital Hearing Losses by Ethnicity and Monitoring Period, New Zealand 1st April 2010–31st March 2012

Ethnicity	6 Month Monitoring Period			
	1st April 2010–30th Sept 2010	1st October 2010–31st March 2011	1st April 2011–30th Sept 2011	1st October 2011–31st March 2012
Permanent Congenital Hearing Losses Identified by Newborn Screening				
Māori	4	7	9	12
Pacific	1	0	5	1
Asian	1	2	3	1
European	5	6	7	16
Unspecified	0	0	0	0
Other Ethnic Groups	0	1	0	0
Total	11	16	24	30

Source: National Screening Unit 2012 [28];

CEREBRAL PALSY

Introduction

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 Māori children and young people. In the absence of such information, the following section reviews hospital admissions for Māori children and young people with any mention of cerebral palsy in any of their first 15 diagnoses.

Background

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 non-progressive, its physical consequences can evolve over time [53].

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) [54] [55]. In addition, while cerebral palsy refers to the motor impairment, features such as seizures, intellectual impairment and learning disabilities are common [55].

In New Zealand, there is little information on the prevalence or health needs of Māori children and young people with cerebral palsy, although the authors of Hauora IV noted that mortality from cerebral palsy during 2000–2004 was significantly higher (79%) for Māori than for non-Māori [6]. Other local research also suggests that children with cerebral palsy may experience a range of barriers to participation, including those arising from attitudes at school and in the community, difficulties accessing personal equipment, and barriers in the natural and built environments [56]. In one Auckland study (n=32), the parents of 94% of children with cerebral palsy indicated that their child experienced one or more such barriers to participation [56].

Data Source and Methods

Definition

Hospital admissions for children and young people aged 0–24 years with cerebral palsy listed in any of the first 15 diagnoses

Data Source

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.

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, in New Zealand 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.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 384 individual Māori children and young people were hospitalised with a diagnosis of cerebral palsy, with admission rates per 100,000 population not being *significantly* different from those of non-Māori non-Pacific children and young people (RR 0.97 95% CI 0.91–1.04) (**Table 21**).

Table 21. Hospital Admissions for Children and Young People Aged 0–24 Years with Cerebral Palsy by Ethnicity, New Zealand 2008–2012

Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Cerebral Palsy						
Māori	384	1,105	0.58	63.1	0.97	0.91–1.04
non-Māori non-Pacific	1,109	3,352	0.60	64.7	1.00	

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)

Distribution by Primary Diagnosis and Procedure

Acute and Arranged Admissions by Primary Diagnosis: During 2008–2012, only 7.5% of acute and arranged hospitalisations in Māori children and young people with cerebral palsy listed in their first 15 diagnoses, had cerebral palsy listed as the primary reason for admission. Instead 13.9% of acute and arranged admissions were for epilepsy or convulsions and 15.8% were for influenza, pneumonia or unspecified lower respiratory infections. Acute and arranged admissions made up 53.3% of admissions for Māori children and young people with cerebral palsy during this period (**Table 22**).

Waiting List Admissions by Primary Procedure: During the same period, 46.7% of admissions in Māori children and young people with cerebral palsy were from the waiting list, with injections into ligaments, tendons, or soft tissue accounting for 35.9% of waiting list admissions. Orthopaedic procedures collectively were the leading reasons for waiting list admissions in Māori children and young people with cerebral palsy, followed by dental procedures (**Table 23**).

Table 22. Acute and Arranged Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Cerebral Palsy by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Acute and Arranged Admissions	% of All Admissions in those with Cerebral Palsy
Māori Children and Young People 0–24 Years					
Acute and Arranged Admissions by Primary Diagnosis					
Epilepsy, Status Epilepticus, Convulsions	82	16.4	4.68	13.9	7.4
Influenza and Pneumonia	51	10.2	2.91	8.7	4.6
Unspecified Acute Lower Respiratory Infection	42	8.4	2.40	7.1	3.8
Pneumonitis due to Food and Vomit	30	6.0	1.71	5.1	2.7
Acute Upper Respiratory Infections	26	5.2	1.48	4.4	2.4
Other Respiratory Infections and Diseases	23	4.6	1.31	3.9	2.1
Constipation	15	3.0	0.86	2.5	1.4
Other Diseases Digestive System	45	9.0	2.57	7.6	4.1
Cerebral Palsy	44	8.8	2.51	7.5	4.0
Infectious and Parasitic Diseases	31	6.2	1.77	5.3	2.8
Respite Care	14	2.8	0.80	2.4	1.3
Other Factors Influencing Health Service Contact	22	4.4	1.26	3.7	2.0
Complications of Surgical and Medical Care	21	4.2	1.20	3.6	1.9
Other Diagnoses	143	28.6	8.17	24.3	12.9
Total Acute and Arranged Admissions	589	117.8	33.64	100.0	53.3
Total Waiting List Admissions	516	103.2	29.47		46.7
Total Admissions in those with Cerebral Palsy	1,105	221.0	63.10		100.0

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 23. Waiting List Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Cerebral Palsy by Primary Procedure, New Zealand 2008–2012

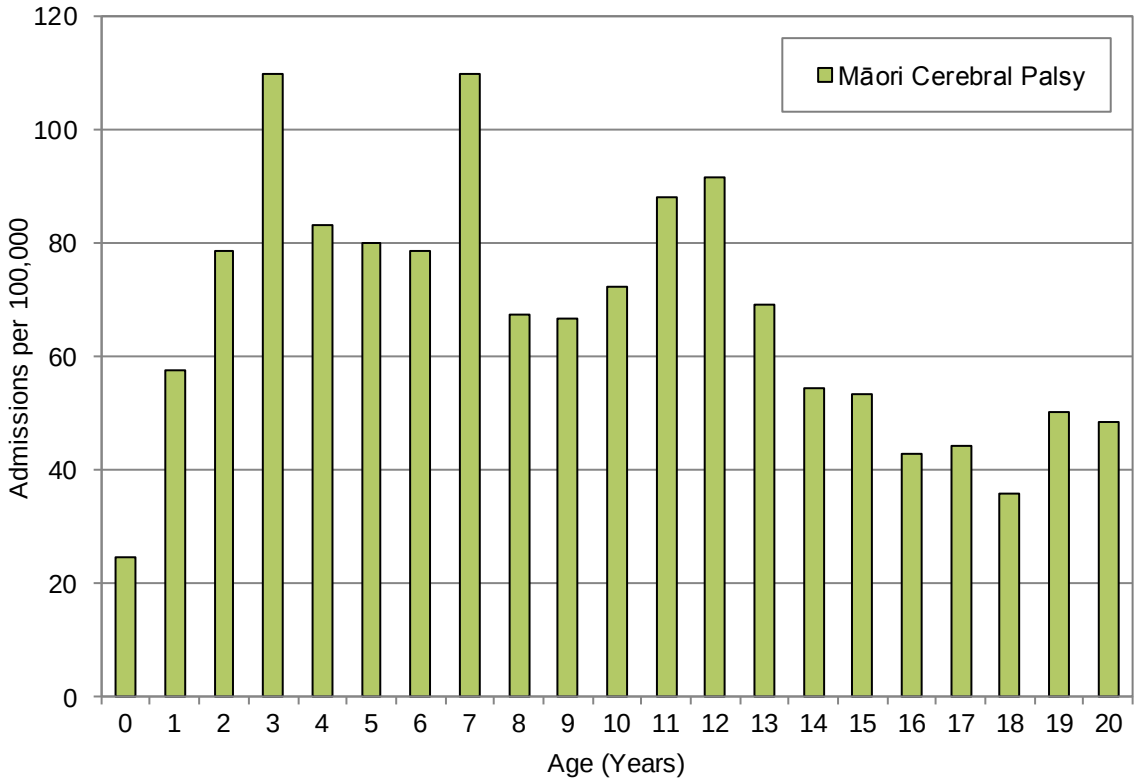
Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Waiting List Admissions	% of All Admissions in those with Cerebral Palsy
Māori Children and Young People 0–24 Years					
Waiting List Admissions by Primary Procedure					
Injection into ligament, tendon or soft tissue	185	37.0	10.56	35.9	16.7
Osteotomy of proximal femur	25	5.0	1.43	4.8	2.3
Release of Hip Contracture	22	4.4	1.26	4.3	2.0
Forage of neck and/or head of femur	18	3.6	1.03	3.5	1.6
Lengthening/Repair of Achilles Tendon	15	3.0	0.86	2.9	1.4
Lengthening of tendon, unspecified	13	2.6	0.74	2.5	1.2
Other Orthopedic Procedures	78	15.6	4.45	15.1	7.1
Dental Procedures	60	12.0	3.43	11.6	5.4
Injection or infusion of other therapeutic or prophylactic substance	12	2.4	0.69	2.3	1.1
Other Procedures	77	15.4	4.40	14.9	7.0
No Procedure Listed	11	2.2	0.63	2.1	1.0
Total Waiting List Admissions	516	103.2	29.47	100.0	46.7
Total Acute and Arranged Admissions	589	117.8	33.64		53.3
Total Admissions in those with Cerebral Palsy	1,105	221.0	63.10		100.0

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 Māori children and young people with cerebral palsy increased during the first few years, reached a peak at three years of age, and then fluctuated. A gradual decline in rates was evident during the late teens (**Figure 13**).

Figure 13. Hospital Admissions for Māori Children and Young People with Cerebral Palsy by Age, New Zealand 2008–2012

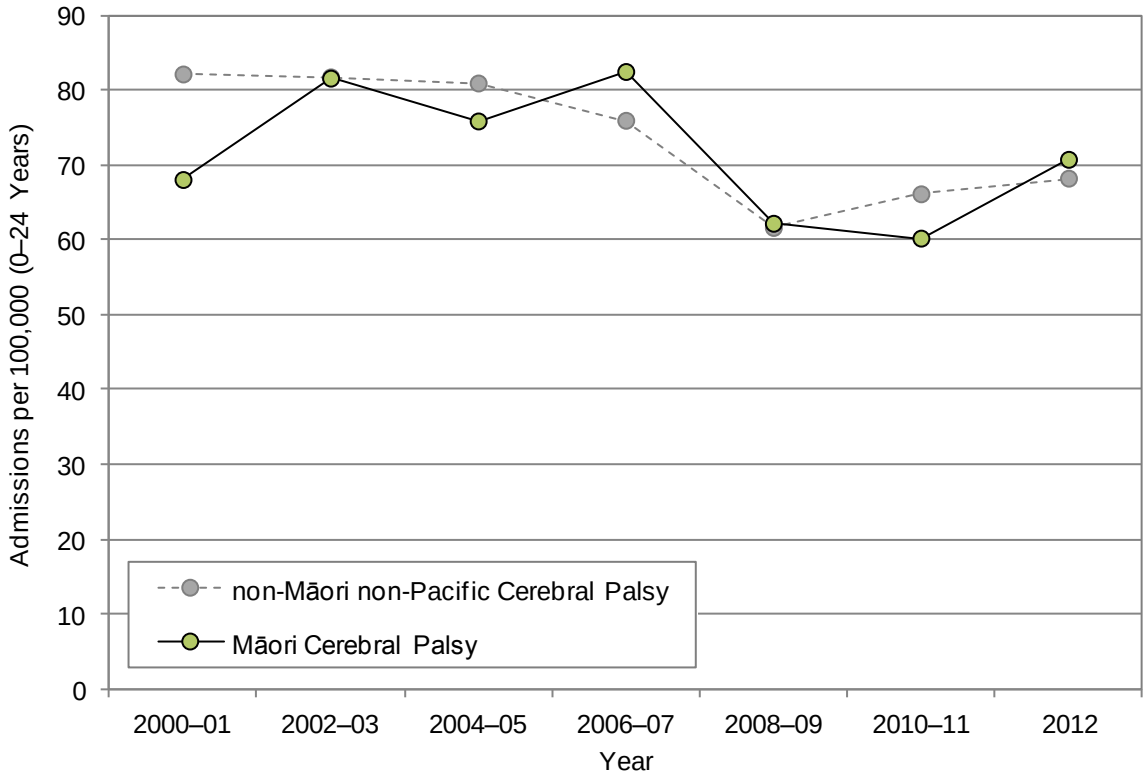


Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with cerebral palsy listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Trends by Ethnicity

In New Zealand, hospital admissions for Māori children and young people with cerebral palsy were relatively static during the early-mid 2000s, but decreased between 2006–07 and 2010–11. Similar trends were seen for non-Māori non-Pacific children and young people, with rates for both ethnic groups being similar during this period (**Figure 14**).

Figure 14. 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 (projected from 2007)

AUTISM SPECTRUM DISORDER

Introduction

The following section reviews hospital admissions for Māori children and young people with any mention of autism or other pervasive developmental disorders in any of their first 15 diagnoses.

Background

The diagnostic criteria for autism have recently changed with the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) moving, in May 2013, to an overarching Autism Spectrum Disorder (ASD) diagnosis [57]. This new classification uses two symptom dimensions to define ASD [57]. 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) [58]. 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 [58].

In New Zealand there is no routinely collected information on the prevalence of ASD in Māori children, although the 2006/07 NZ Health Survey estimated a prevalence of 40 per 10,000 in the total population [59]. Despite this paucity of data, research involving the parents and whānau of Māori children with ASD suggests a range of health and disability support services may be required including: staff to explain to parents what ASD is and the services and entitlements available, and to assist them to access these services; the need to increase ASD expertise amongst Māori medium education and service providers, and to increase bicultural and bilingual expertise in mainstream services; the need for more culturally appropriate assessment measures and procedures; and the need to increase ASD-related financial assistance to parents. The same research also found that ASD-related impairments may have limited children's involvement in culturally valued activities such as kapa haka, learning te reo and staying on the marae [60].

Note: Given that DSM-V was only released in May 2013, the analysis which follows uses ICD-10-AM codes based on the earlier definitions of autism and pervasive developmental disorders (see Methods box below).

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.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 284 individual Māori children and young people were hospitalised with a diagnosis of autism or other pervasive developmental disorders, with admission rates per 100,000 population being *significantly* lower than for non-Māori non-Pacific children and young people (RR 0.65 95% CI 0.58–0.72) (**Table 24**).

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

Ethnicity	Total No. Individuals 2008–2012	Total No. Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Autism or Other Pervasive Developmental Disorders						
Māori	284	415	0.29	23.7	0.65	0.58–0.72
non-Māori non-Pacific	1,178	1,897	0.32	36.6	1.00	

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 Primary Diagnosis

In New Zealand during 2008–2012, autism and other pervasive developmental disorders were listed as the primary diagnosis in only 10.8% of hospitalisations for Māori children and young people with a pervasive developmental disorder in any of the first 15 diagnoses. Overall, 27.0% of admissions were for dental caries or other oral health conditions, while a further 9.6% were for epilepsy or convulsions (**Table 25**).

Distribution by Age

In New Zealand during 2008–2012, no hospital admissions for autism or other pervasive developmental disorders occurred in Māori infants. Admissions however, increased during the pre-school years, to reach a peak at six years of age. Rates then declined during late childhood and again amongst those in their late teens (**Figure 15**).

Trends by Ethnicity

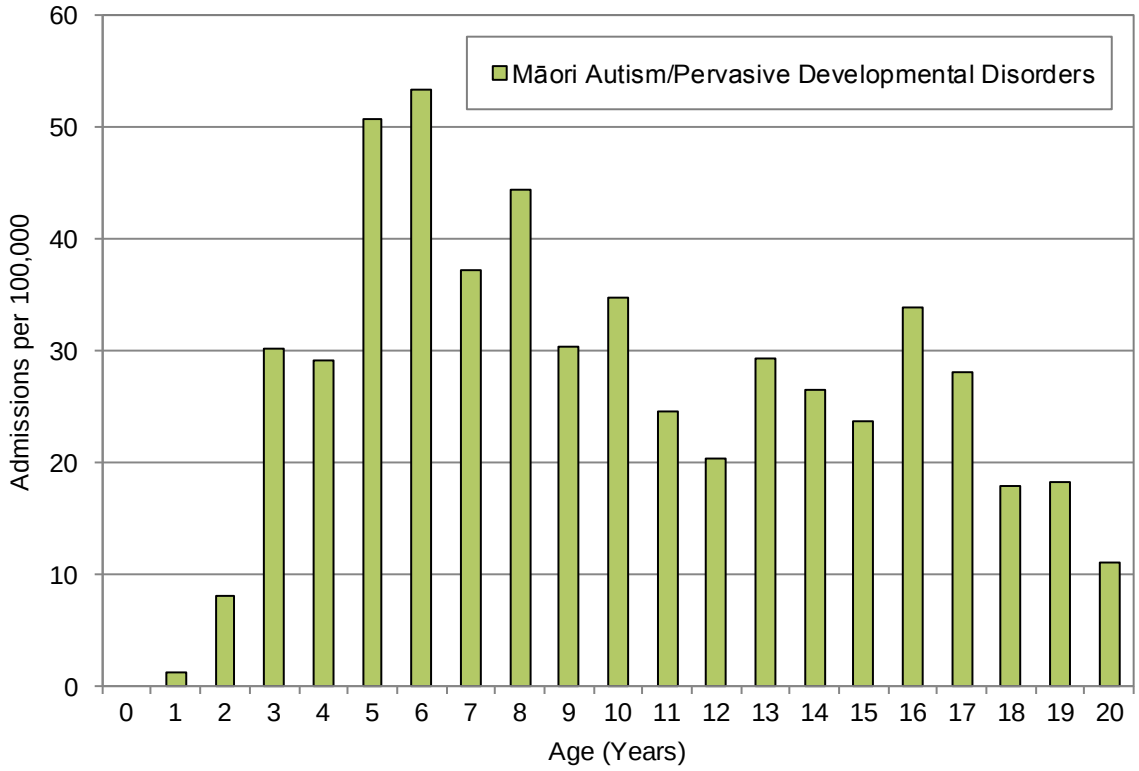
In New Zealand during 2000–2012, hospital admissions for Māori children and young people with autism or other pervasive developmental disorders were consistently lower than for non-Māori non-Pacific children and young people, although rates increased for both ethnic groups during this period (**Figure 16**).

Table 25. Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Autism or Other Pervasive Developmental Disorders by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Admissions in those with Pervasive Developmental Disorders
Māori Children and Young People 0–24 Years				
Pervasive Developmental Disorders				
Childhood Autism	31	6.2	1.77	7.5
Asperger's Syndrome	6	1.2	0.34	1.4
Other Autism and Pervasive Developmental Disorders	8	1.6	0.46	1.9
Total Autism and Other Pervasive Developmental Disorders	45	9.0	2.57	10.8
Other Diagnoses				
Dental Caries	91	18.2	5.20	21.9
Other Dental and Oral Health Issues	21	4.2	1.20	5.1
Epilepsy and Status Epilepticus	32	6.4	1.83	7.7
Unspecified Convulsions	8	1.6	0.46	1.9
Respiratory Infections and Diseases	21	4.2	1.20	5.1
Schizophrenia, Schizotypal and Delusional Disorders	13	2.6	0.74	3.1
Mood Disorders	6	1.2	0.34	1.4
Other Mental and Behavioral Disorders	12	2.4	0.69	2.9
Other Diagnoses	166	33.2	9.48	40.0
Total Other Diagnoses	370	74.0	21.13	89.2
Total	415	83.0	23.70	100.0

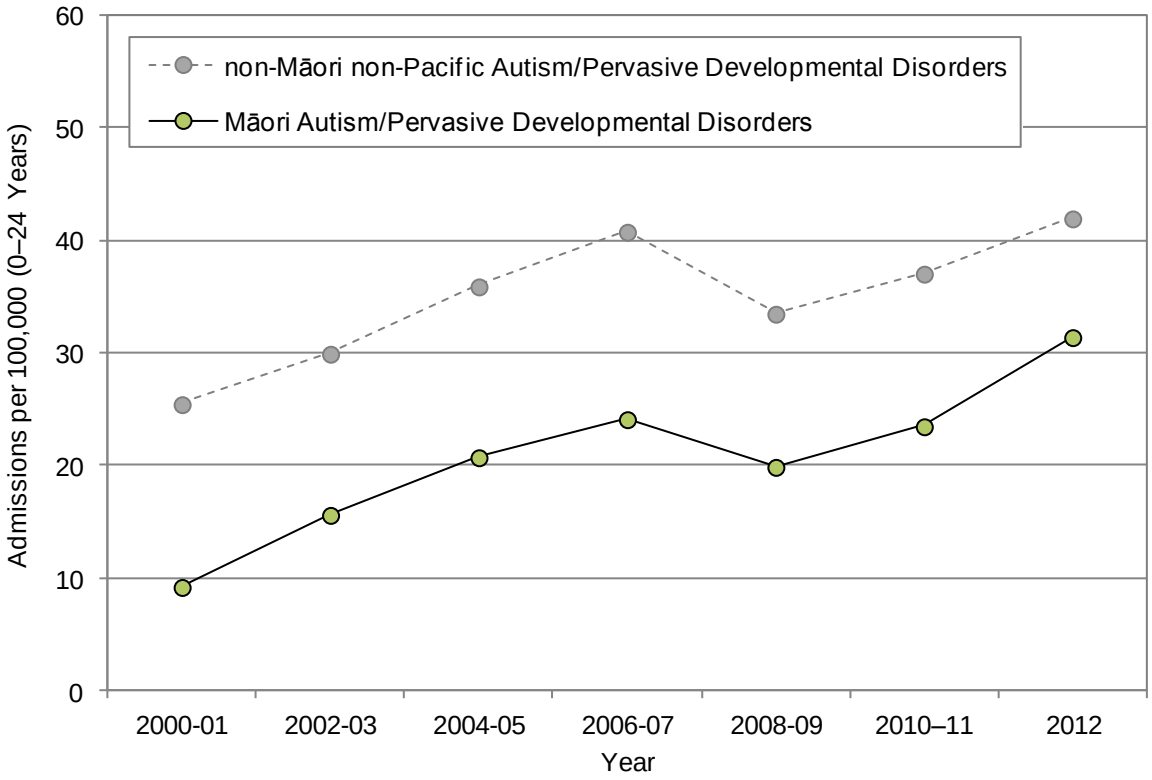
Source: Numerator: National Minimum Dataset: Hospital Admissions by primary diagnosis for children and young people with autism or other pervasive developmental disorders listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Figure 15. Hospital Admissions for Māori 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)

Figure 16. 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)

CHRONIC MEDICAL CONDITIONS

ECZEMA AND DERMATITIS

Introduction

The following section briefly reviews hospitalizations for Māori children aged 0–14 years with eczema or dermatitis listed in any of their 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 Māori 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.

Background

Eczema and dermatitis are terms that are often used interchangeably. Eczema is a common chronic skin condition characterised by an itchy rash, often located on the folds of the elbows and knees. It frequently occurs at a young age (e.g. less than two years) and often follows a chronic relapsing course [61].

New Zealand is among countries with the highest prevalence (15-20%) of eczema and dermatitis [62]. In one New Zealand study undertaken during 2001–2003, the estimated prevalence of current eczema (symptoms in the past 12 months) was 15.0% for children (6–7 years) and 8.8% for adolescents (13–14 years), with 1.8% of children and 1.3% of adolescents having severe eczema (kept awake at night by eczema symptoms). However, 31.5% of children and 26.1% of adolescents were reported as ever having had eczema symptoms [61].

In the same study, Māori children (17.2%) and adolescents (10.4%) had a higher reported prevalence of current eczema than European children (13.8%) and adolescents (7.3%). Similarly Māori children (3.0%) and adolescents (2.1%) had a higher reported prevalence of severe eczema than European children (0.8%) and adolescents (0.8%). However, the proportion of Māori and European children and adolescents who were reported as ever having had eczema was similar [61].

Data Source and Methods

Definition

1. *Hospital admissions for children 0–14 years with eczema or dermatitis listed in any of their first 15 diagnoses*

2. *Hospital admissions for children 0–14 years with eczema or dermatitis listed as a primary diagnosis*

Data Source

1. National Minimum Dataset

Numerator: Hospital admissions for children aged 0–14 years with eczema or dermatitis (ICD-10-AM L01.1, L20–L26, L272, L28–L30, B000, H01.1) listed in any of their first 15 diagnoses

Numerator: Hospital admissions for children aged 0–14 years with eczema or dermatitis (ICD-10-AM L01.1, L20–L26, L272, L28–L30, B000, H01.1) listed as a primary diagnosis.

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

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)

Broader diagnostic groupings include: Infective Dermatitis (L01.1, L303); and Other Eczema and Dermatitis (L20L–26, L28–L29, B00.0, H01.1, L27.2, L30.0–L30.2, L30.4–L30.5, L30.8–L30.9).

Notes on Interpretation

Apart from the first table, 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.

Distribution in Māori Children

Admissions with Eczema or Dermatitis in First 15 Diagnoses

Distribution by Primary Diagnosis

In New Zealand during 2008–2012, only 32.4% of hospitalisations in Māori 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. Infective dermatitis (14.5%) and atopic and other dermatitis (14.3%) were the most frequent primary diagnoses assigned to those with eczema or dermatitis, while bronchiolitis, asthma and wheeze and skin infections were the most frequent non-eczema related reasons for admission (**Table 26**).

Table 26. Hospital Admissions for Māori Children Aged 0–14 Years with Eczema or Dermatitis by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Rate per 100,000 Population	% of Admissions in those with Eczema or Dermatitis
Māori Children Aged 0–14 Years				
Eczema and Dermatitis				
Infective Dermatitis	746	149.2	66.24	14.5
Atopic and Other Dermatitis	734	146.8	65.18	14.3
Eczema Herpeticum	83	16.6	7.37	1.6
Contact Dermatitis	31	6.2	2.75	0.6
Seborrhoeic Dermatitis	31	6.2	2.75	0.6
Eczema Due to Food Ingestions	21	4.2	1.87	0.4
Diaper Dermatitis	20	4.0	1.78	0.4
Total Eczema or Dermatitis	1,666	333.2	147.93	32.4
Total Other Diagnoses*	3,472	694.4	308.30	67.6
Total	5,138	1,027.6	456.23	100.0
*Other Primary Diagnoses				
Bronchiolitis	663	132.6	58.87	12.9
Asthma and Wheeze	453	90.6	40.22	8.8
Skin Infections	373	74.6	33.12	7.3
Pneumonia and Unspecified LRTI	222	44.4	19.71	4.3
Gastroenteritis	197	39.4	17.49	3.8
Prematurity and Low Birth Weight	178	35.6	15.81	3.5
Acute Upper Respiratory Infections	170	34.0	15.10	3.3
Injury and Poisoning	155	31.0	13.76	3.0
Other Infectious and Parasitic Diseases	139	27.8	12.34	2.7
Viral Infections Unspecified	96	19.2	8.52	1.9
Other Perinatal Conditions	85	17.0	7.55	1.7
Epilepsy and Convulsions	40	8.0	3.55	0.8
Cancer Neoplasms (C00-D48)	28	5.6	2.49	0.5
Urinary Tract Infection	17	3.4	1.51	0.3
Other Diagnoses	656	131.2	58.25	12.8
Total Other Primary Diagnoses	3,472	694.4	308.30	67.6

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

Admissions with Eczema or Dermatitis as a Primary Diagnosis

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 1,155 individual Māori children were hospitalised with a primary diagnosis of eczema or dermatitis, with admission rates per 100,000 population being *significantly* higher than for non-Māori non-Pacific children (RR 3.16 95% CI 2.94–3.39) (**Table 27**).

Table 27. Hospital Admissions for Children Aged 0–14 Years with a Primary Diagnosis of Eczema or Dermatitis by Ethnicity, New Zealand 2008–2012

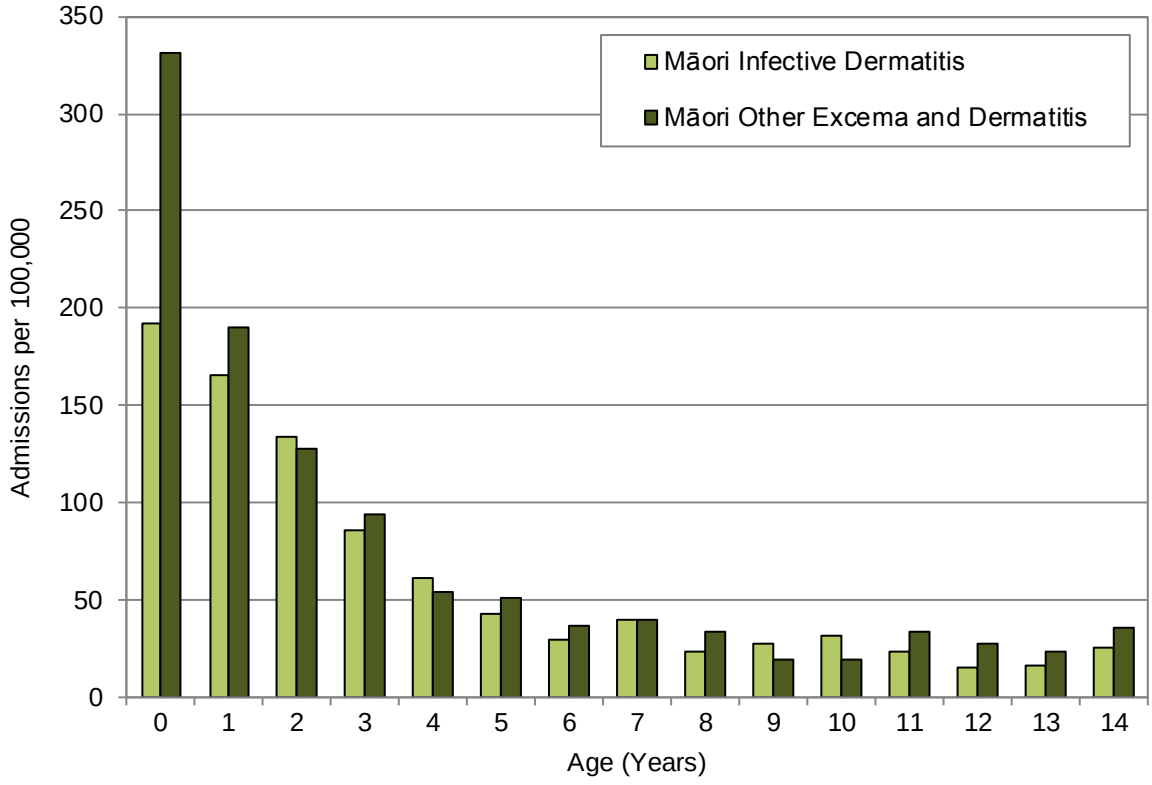
Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
All Eczema and Dermatitis						
Māori	1,155	1,666	0.29	147.9	3.16	2.94–3.39
non-Māori non-Pacific	1,097	1,355	0.25	46.9	1.00	

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)

Distribution by Age

In New Zealand during 2008–2012, hospital admissions for infective dermatitis and other forms of eczema and dermatitis were highest in Māori infants aged less than one year, with rates then tapering off during the preschool years. Admission rates were lowest amongst Māori children over five years of age (**Figure 17**).

Figure 17. Hospital Admissions for Māori Children with a Primary Diagnosis of Eczema or Dermatitis by Age, New Zealand 2008–2012

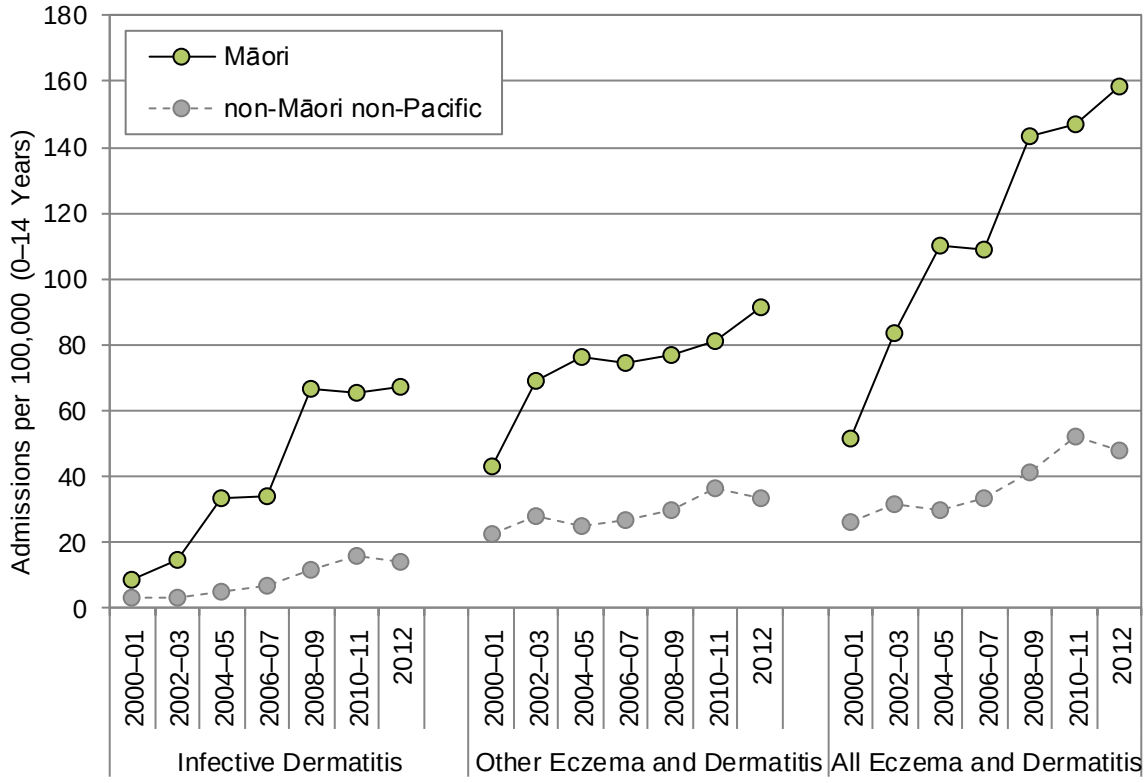


Source: Numerator: National Minimum Dataset, hospital admissions for children with eczema or dermatitis listed as a primary diagnosis; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Trends by Ethnicity

In New Zealand during 2000–2012, hospitalisations for infective eczema and other eczema and dermatitis were consistently higher for Māori children, than for non-Māori non-Pacific children. While rates increased for both ethnic groups during this period, the largest absolute increases were seen in Māori children, resulting in a widening of ethic differences during this period (**Figure 18**).

Figure 18. 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)

INFLAMMATORY BOWEL DISEASE

Introduction

The following section reviews hospital admissions for Māori children and young people with Crohn's disease or ulcerative colitis listed in any of their first 15 diagnoses.

Background

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 intestine [63]. The peak age of onset is between 15 and 30 years [64], with those first presenting with ulcerative colitis typically having obvious symptoms (e.g. 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 [65].

In New Zealand, there has been a large rise in the incidence of IBD over the past 50 years [65]. However, Māori children have been shown to have a lower incidence of inflammatory bowel disease than European children. In one NZ Paediatric Surveillance Unit study, which surveyed clinicians working with children monthly during 2002–2003 for cases of paediatric inflammatory bowel disease, no Māori children were identified as having Crohn's disease or ulcerative colitis [66]. The study authors noted that Māori have been shown to have a low frequency of NOD2/CARD15, a genetic mutation that confers susceptibility to Crohn's disease, and thus genetic factors may be partly responsible for the lower observed prevalence of inflammatory bowel disease in Māori children [66].

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.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, 41 individual Māori children and young people were hospitalised with Crohn's disease, while 21 were hospitalised with ulcerative colitis. Admission rates per 100,000 population for both outcomes were *significantly* lower than for non-Māori non-Pacific children and young people (Crohn's disease RR 0.11 95% CI 0.09–0.13; ulcerative colitis RR 0.11 95% CI 0.07–0.16) (**Table 28**).

Table 28. Hospital Admissions for Children and Young People Aged 0–24 Years with Crohn's Disease or Ulcerative Colitis by Ethnicity, New Zealand 2008–2012

Ethnicity	Total No. Individuals 2008–2012	Total No. Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Crohn's Disease						
Māori	41	132	0.64	7.54	0.11	0.09–0.13
non-Māori non-Pacific	892	3,676	0.82	70.98	1.00	
Ulcerative Colitis						
Māori	21	30	0.29	1.71	0.11	0.07–0.16
non-Māori non-Pacific	371	823	0.44	15.89	1.00	

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)

Distribution by Primary Diagnosis and Procedure

Crohn's Disease: In New Zealand during 2008–2012, 84.0% of acute and arranged hospitalisations in Māori 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 (**Table 29**). Of those admitted from the waiting list with a diagnosis of Crohn's disease, injections or infusions of therapeutic substances (58.8%) and fibreoptic colonoscopies +/− biopsies (15.7%) were the most frequent primary procedures listed (**Table 29**).

Ulcerative Colitis: During the same period, 60.0% of acute and arranged hospitalisations in Māori 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 (**Table 30**). Of those admitted from the waiting list with a diagnosis of ulcerative colitis, fibreoptic colonoscopies +/− biopsies (80.0%) were the most frequent primary procedures listed (**Table 30**).

Distribution by Age

In New Zealand during 2008–2012, hospital admissions for Māori children and young people with Crohn's disease and ulcerative colitis were infrequent during childhood, but increased during adolescence, with the highest rates being seen amongst those in their late teens (Figure 19).

Trends by Ethnicity

In New Zealand during 2000–2012, hospital admissions for Māori children and young people with Crohn's disease were consistently lower than for non-Māori non-Pacific children and young people, although admissions for both ethnic groups increased during this period. While similar patterns were seen for ulcerative colitis, the increases in rates seen in both ethnic groups were not as marked as for Crohn's disease during this period (**Figure 20**).

Table 29. Hospital Admissions in Māori Children and Young People Aged 0–24 Years with Crohn's Disease by Admission Type and Primary Diagnosis or Procedure, New Zealand 2008–2012

Primary Diagnosis or Procedure	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Admissions in Category	% of Admissions in those with Crohn's Disease
Māori Children and Young People Aged 0–24 Years					
Acute and Arranged Admissions by Primary Diagnosis					
Crohn's Disease: Large Intestine	13	2.6	0.74	16.0	9.8
Crohn's Disease: Small Intestine	12	2.4	0.69	14.8	9.1
Crohn's Disease: Other	22	4.4	1.26	27.2	16.7
Crohn's Disease: Unspecified	21	4.2	1.20	25.9	15.9
Other Diseased Digestive System	4	0.8	0.23	4.9	3.0
Other Diagnoses	9	1.8	0.51	11.1	6.8
Total Acute and Arranged Admissions	81	16.2	4.63	100.0	61.4
Waiting List Admissions by Primary Procedure					
Injection or Infusion of Substance	30	6.0	1.71	58.8	22.7
Fibreoptic Colonoscopy + Biopsy	8	1.6	0.46	15.7	6.1
Other Procedures	13	2.6	0.74	25.5	9.8
Total Waiting List Admissions	51	10.2	2.91	100.0	38.6
Total Crohn's Disease Admissions	132	26.4	7.54		100.0

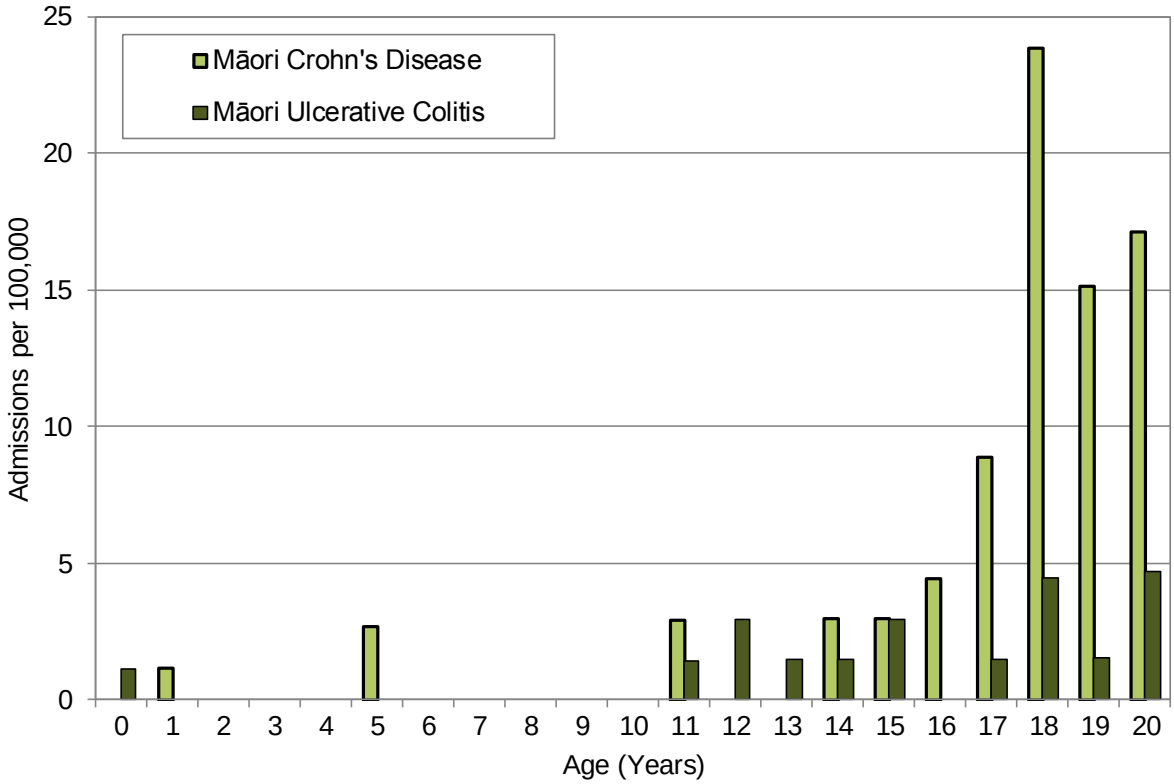
Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with Crohn's disease listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Table 30. Hospital Admissions in Māori Children and Young People Aged 0–24 Years with Ulcerative Colitis by Admission Type and Primary Diagnosis or Procedure, New Zealand 2008–2012

Primary Diagnosis or Procedure	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Admissions in Category	% of Admissions in those with Ulcerative Colitis
Māori Children and Young People Aged 0–24 Years					
Acute and Arranged Admissions by Primary Diagnosis					
Ulcerative Colitis	12	2.4	0.69	60.0	40.0
Other Diagnoses	8	1.6	0.46	40.0	26.7
Total Acute and Arranged Admissions	20	4.0	1.14	100.0	66.7
Waiting List Admissions by Primary Procedure					
Fibreoptic Colonoscopy + Biopsy	8	1.6	0.46	80.0	26.7
Other Procedures	<3	s	s	s	s
Total Waiting List Admissions	10	2.0	0.57	100.0	33.3
Total Ulcerative Colitis Admissions	30	6.0	1.71		100.0

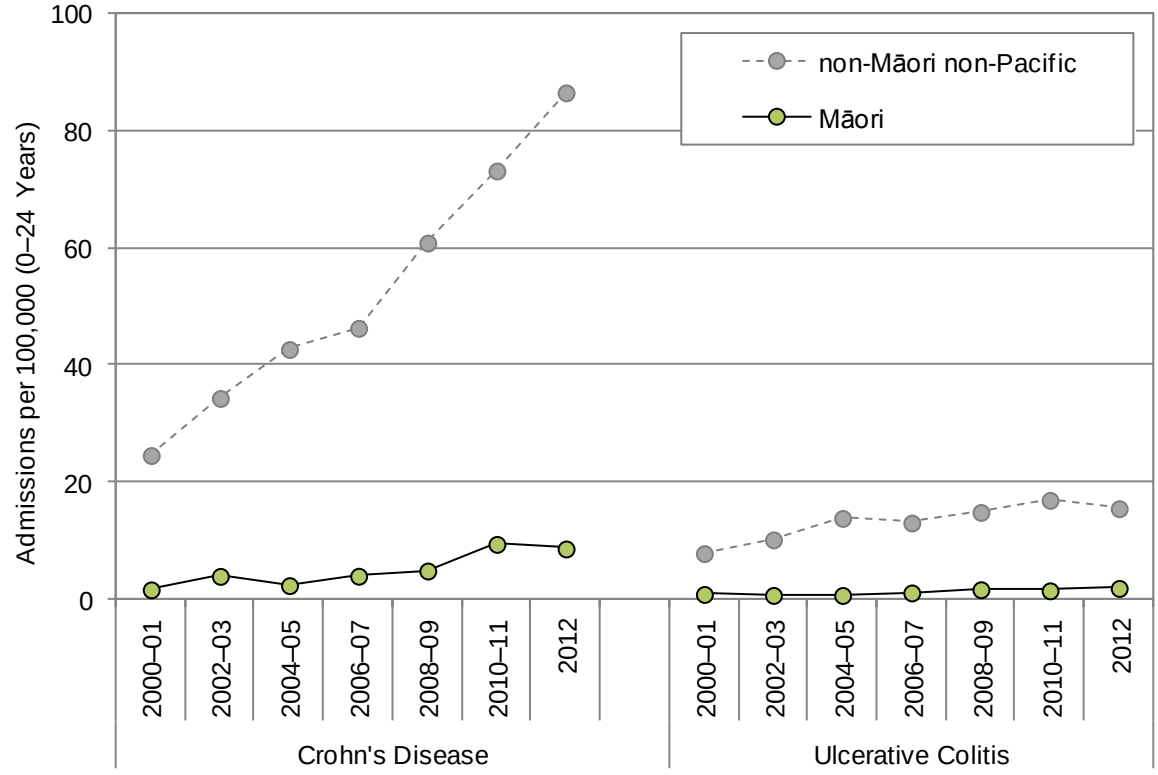
Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with ulcerative colitis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007); Note: s = cells suppressed due to small numbers

Figure 19. Hospital Admissions for Māori Children and Young People with Crohn's Disease or Ulcerative Colitis by Age, New Zealand 2008–2012



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)

Figure 20. 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)

CYSTIC FIBROSIS

Introduction

The following section reviews hospitalisations for Māori children and young people with any mention of cystic fibrosis in any of their first 15 diagnoses.

Background

Cystic fibrosis (CF) is a genetic disease resulting from mutations affecting a gene that controls a chloride channel called the cystic fibrosis transmembrane conductance regulator (CFTR). CFTR is essential for the regulation of salt and water movements across cell membranes [67]. 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. The ducts in the pancreas may also become blocked, leading to digestive problems and malabsorption. Other complications include male infertility, diabetes, liver disease, joint problems, and psychological problems arising from having to cope with a severe long-term medical condition [67]

In New Zealand, there is little research on the health needs of Māori children and young people with cystic fibrosis, although all babies are routinely screened for CF as part of the Newborn Metabolic Screening Programme [19]. However, the available evidence suggests that the prevalence of cystic fibrosis is likely to be lower for Māori than for European children and young people, because of the genetic origins of the condition and the fact that the highest rates internationally are seen in northern European populations [68].

In terms of outcomes, 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 [68]. 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 now being directly correlated with the decade of birth [69]. Thus those born with CF today are now expected to live into their sixth decade [69] [68].

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*

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.

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 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, in New Zealand 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.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 37 individual Māori children and young people were hospitalised with a diagnosis of cystic fibrosis, with admission rates per 100,000 population being *significantly* lower than for non-Māori non-Pacific children and young people (RR 0.46 95% CI 0.41–0.51) (**Table 31**).

Table 31. Hospital Admissions for Children and Young People Aged 0–24 Years with Cystic Fibrosis by Ethnicity, New Zealand 2008–2012

Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Cystic Fibrosis						
Māori	37	370	2.00	21.1	0.46	0.41–0.51
non-Māori non-Pacific	319	2,388	1.50	46.1	1.00	

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)

Distribution by Primary and Secondary Diagnosis

In New Zealand during 2008–2012, 83.0% of hospitalisations in Māori 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 (**Table 32**). Of those with cystic fibrosis listed as the primary diagnosis, the majority (92.5%) also had a secondary diagnosis. In 25.1% of such cases, the secondary diagnosis was influenza, pneumonia or an unspecified lower respiratory infection, while a further 14.0% had bronchiectasis listed as their secondary diagnosis (**Table 33**).

Table 32. Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Cystic Fibrosis by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Admissions in those with Cystic Fibrosis
Māori Children and Young People 0–24 Years				
Cystic Fibrosis				
Cystic Fibrosis, Pulmonary Manifestation	164	32.8	9.37	44.3
Cystic Fibrosis, Intestinal Manifestations	7	1.4	0.40	1.9
Cystic Fibrosis, Other Manifestations	110	22.0	6.28	29.7
Cystic Fibrosis Unspecified	26	5.2	1.48	7.0
Total Cystic Fibrosis-Related Diagnoses	307	61.4	17.53	83.0
Complications Surgical Medical Care	16	3.2	0.91	4.3
Respiratory System Diseases	14	2.8	0.80	3.8
Other Diagnoses	33	6.6	1.88	8.9
Total Other Diagnoses	63	12.6	3.60	17.0
Total	370	74.0	21.13	100.0

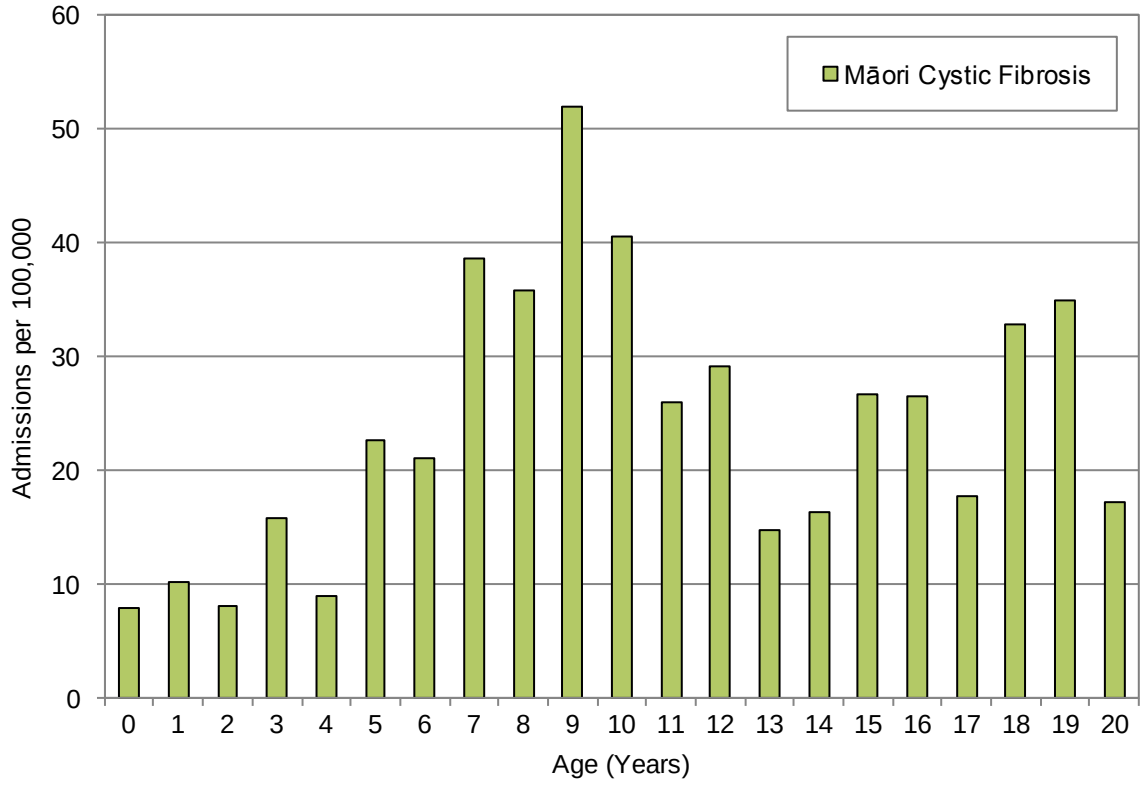
Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with cystic fibrosis listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Table 33. Secondary Diagnoses in Māori Children and Young People Aged 0–24 Years Hospitalised with Cystic Fibrosis as a Primary Diagnosis, New Zealand 2008–2012

Secondary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	% of Admissions with CF as a Primary Diagnosis
Māori Children and Young People 0–24 Years			
Secondary Diagnoses in Admissions with Cystic Fibrosis as a Primary Diagnosis			
Influenza, Pneumonia and Unspecified LRTI*	77	15.4	25.1
Bronchiectasis	43	8.6	14.0
Other Respiratory System Diseases	22	4.4	7.2
Staphylococcus aureus Infection	26	5.2	8.5
Pseudomonas Infection	25	5.0	8.1
Aspergillosis	17	3.4	5.5
Other Infectious and Parasitic Diseases	17	3.4	5.5
Specific Diseases of Pancreas	14	2.8	4.6
Other Diseases Digestive System	13	2.6	4.2
Other Diagnoses	30	6.0	9.8
No Secondary Diagnosis	23	4.6	7.5
Total	307	61.4	100.0

Source: Numerator: National Minimum Dataset, hospital admissions by secondary diagnosis for children and young people with cystic fibrosis as their first diagnosis; *LRTI: lower respiratory tract infection

Figure 21. Hospital Admissions for Māori Children and Young People with Cystic Fibrosis by Age, New Zealand 2008–2012



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)

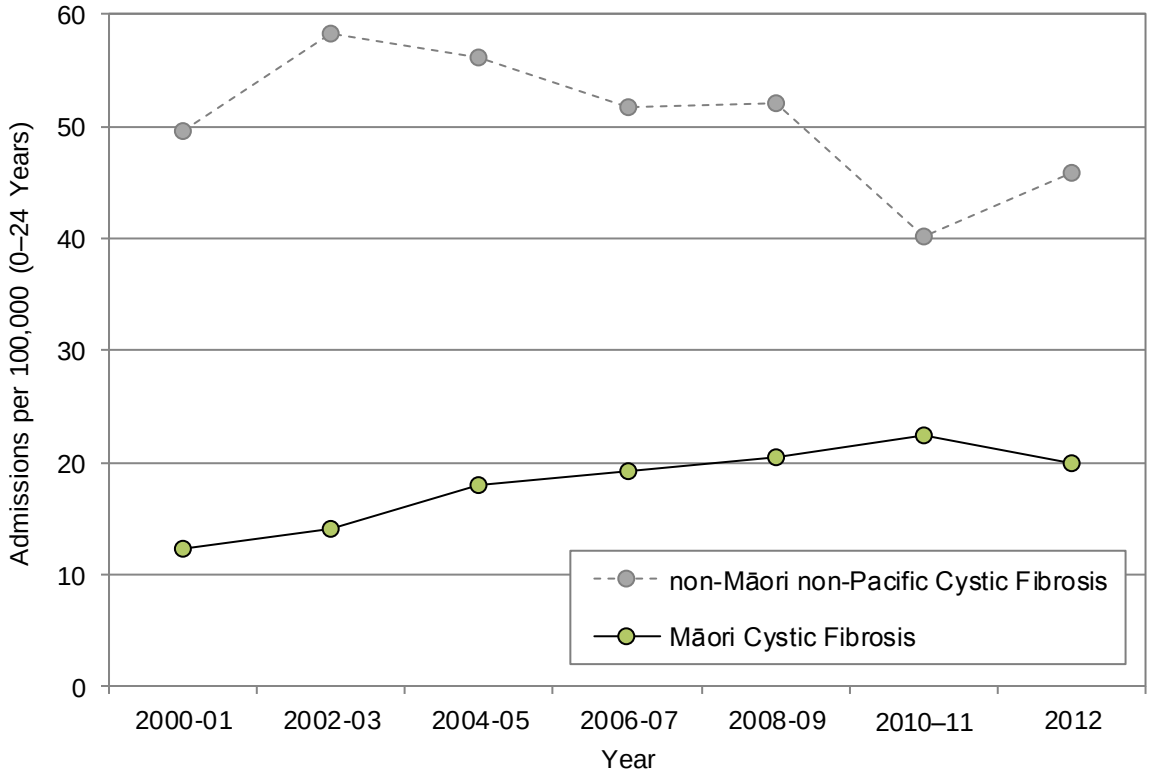
Distribution by Age

In New Zealand during 2008–2012, hospital admissions for Māori children and young people with cystic fibrosis increased during childhood, to reach a peak at nine years of age. Rates then fluctuated (at a lower baseline) during the teenage years (**Figure 21**).

Trends by Ethnicity

In New Zealand during 2000–2012, hospital admissions for Māori children and young people with cystic fibrosis gradually increased, while admissions for non-Māori non-Pacific children and young people declined. Thus ethnic differences were less marked at the end of this period than at the beginning (**Figure 22**).

Figure 22. Hospital Admissions for Children and Young People Aged 0–24 Years with Cystic Fibrosis by Ethnicity, 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)

TYPE 1 DIABETES

Introduction

The following section reviews hospital admissions for Māori children and young people with Type 1 Diabetes listed in any of their first 15 diagnoses.

Background

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, in a small number of cases the reasons for beta cell failure are unknown [70]. The incidence of Type 1 Diabetes increases from birth, peaking 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, with an increasing and on-going need for exogenous insulin [70].

In New Zealand the incidence of Type 1 Diabetes is increasing, with one Auckland study finding the incidence had increased from 10.9 per 100,000 children aged 0–14 years in 1990, to 22.5 per 100,000 in 2009 [71]. In this study, the annual incidence of Type 1 Diabetes was lower for Māori (13.9 per 100,000 95% CI 5.2–29.7) than for European (32.5 per 100,000 95% CI 23.8–43.3) children, although the rate of increase for Māori and European children was similar [71]. However another Auckland study during 1995–2005 [72] found that Māori 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 [72].

Data Source and Methods

Definition

Hospital admissions for children and young people aged 0–24 years with Type 1 Diabetes 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 Type 1 Diabetes (ICD-10-AM E10) 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 Type 1 Diabetes listed in any of the first 15 diagnoses. 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, in New Zealand 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 that 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 1 diabetes 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 that occurred in 2008–09, immediately after the introduction of ICD-10-AM V6.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 429 individual Māori children and young people were hospitalised with a diagnosis of Type 1 diabetes, with admission rates per 100,000 population being *significantly* lower than for non-Māori non-Pacific children and young people (RR 0.76 95% CI 0.72–0.80) (**Table 34**).

Table 34. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 1 Diabetes by Ethnicity, New Zealand 2008–2012

Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Type 1 Diabetes						
Māori	429	1,571	0.73	89.7	0.76	0.72–0.80
non-Māori non-Pacific	2,330	6,140	0.53	118.6	1.00	

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)

Distribution by Primary Diagnosis

Primary Diagnosis: In New Zealand during 2008–2012, 72.8% of hospital admissions for Māori 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 38.3% and Type 1 diabetes without complications for 13.0% of admissions. A further 27.2% of hospitalisations were for diagnoses other than diabetes, with pregnancy and childbirth, gastroenteritis, and respiratory diseases being the most common reasons for non-diabetes related admissions (**Table 35**).

Distribution by Age

In New Zealand during 2008–2012, hospitalisations for Māori children and young people with Type 1 diabetes increased during childhood, reached a peak at 14 years of age, and then declined slightly (**Figure 23**).

Trends by Ethnicity

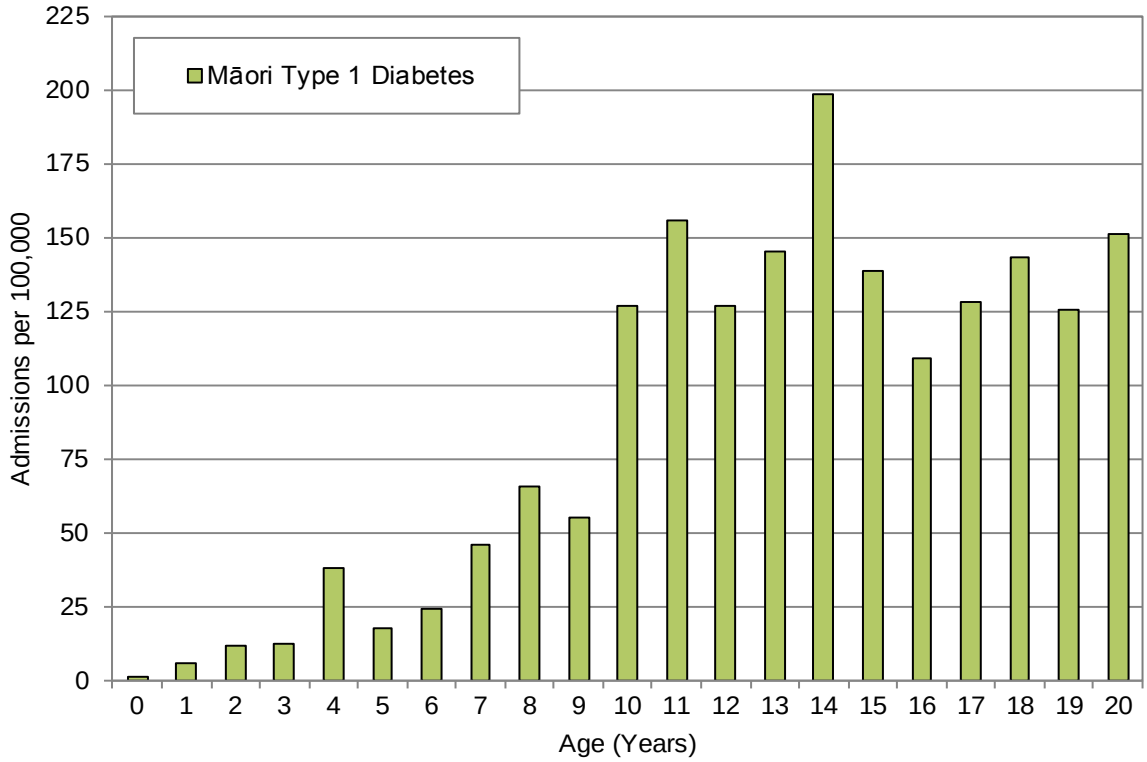
In New Zealand during 2000–2012, hospital admissions for Māori children and young people with Type 1 diabetes were consistently lower than for non-Māori non-Pacific children and young people. While rates increased for both ethnic groups during this period, the rate of increase was faster for Māori than for non-Māori non-Pacific children and young people. Thus ethnic differences were less marked at the end of this period than at the beginning (**Figure 24**).

Table 35. Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Type 1 Diabetes by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	No. of Admissions: Total 2008–2012	No. of Admissions: Annual Average	Rate per 100,000 Population	% of Admissions in those with Type 1 Diabetes
Māori Children and Young People 0–24 Years				
Type 1 Diabetes				
Diagnosis other than Type 1 Diabetes*	428	85.6	24.44	27.2
Type 1 Diabetes with Ketoacidosis/Lactic Acidosis +/− Coma	601	120.2	34.32	38.3
Type 1 Diabetes without Complications	205	41.0	11.71	13.0
Type 1 Diabetes with Renal Complications	6	1.2	0.34	0.4
Type 1 Diabetes with Ophthalmic Complications	5	1.0	0.29	0.3
Type 1 Diabetes with Neurological Complications	4	0.8	0.23	0.3
Type 1 Diabetes with Multiple Complications	3	0.6	0.17	0.2
Type 1 Diabetes with Other Specified Complications	319	63.8	18.22	20.3
Total	1,571	314.2	89.71	100.0
*Diagnoses other than Type 1 Diabetes				
Pregnancy Childbirth Post Partum	61	12.2	3.48	3.9
Gastroenteritis	59	11.8	3.37	3.8
Diseases of Respiratory System	43	8.6	2.46	2.7
Injury and Poisoning	36	7.2	2.06	2.3
Skin Infections	26	5.2	1.48	1.7
Viral Infection Unspecified Site	22	4.4	1.26	1.4
Abdominal and Pelvic Pain	18	3.6	1.03	1.1
Other Infectious Diseases	11	2.2	0.63	0.7
Complications Medical Surgical Care	10	2.0	0.57	0.6
Other Diagnoses	142	28.4	8.11	9.0
Total Diagnoses other than Type 1 Diabetes	428	85.6	24.44	27.2

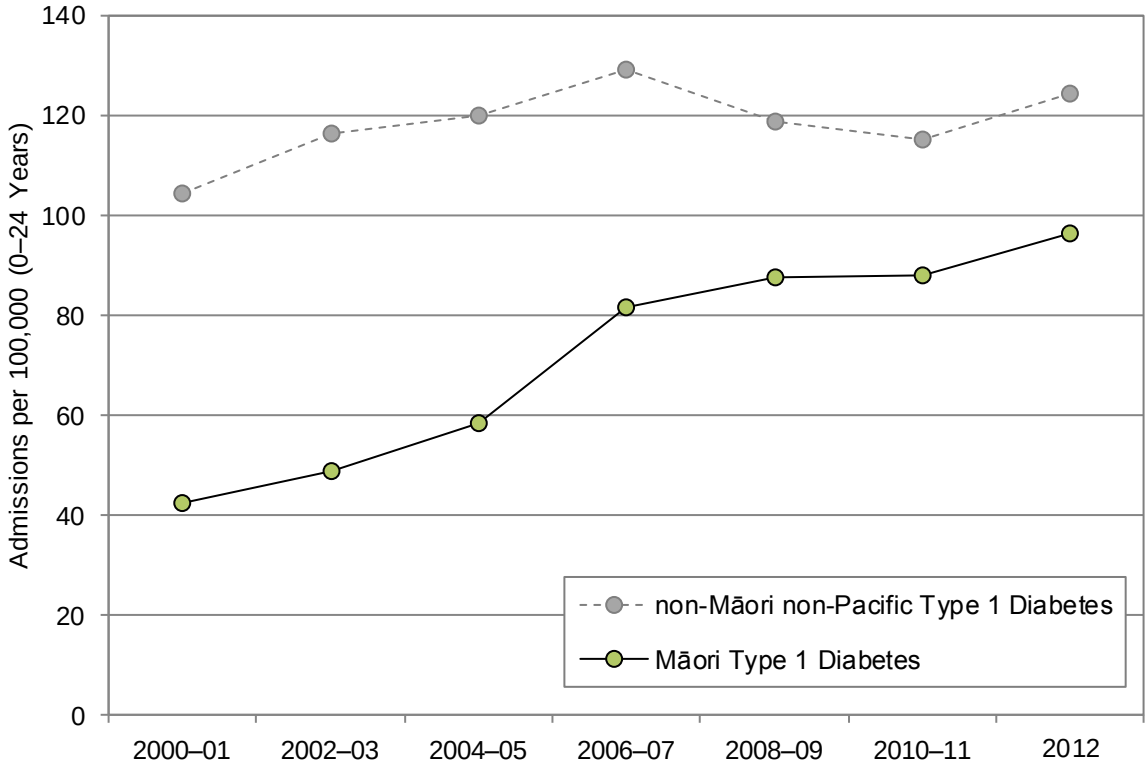
Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with Type 1 Diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Figure 23. Hospital Admissions for Māori Children and Young People with Type 1 Diabetes by Age, New Zealand 2008–2012



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)

Figure 24. 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); 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

EPILEPSY

Introduction

The following section reviews hospital admissions for Māori children and young people with any mention of epilepsy or status epilepticus in any of the first 15 diagnoses.

Background

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, with febrile seizures usually being excluded from these definitions [73].

For most children, the cause of their epilepsy is unknown. Classification systems are useful however, as they serve to guide management for different groups of children. Many group epilepsy into three categories: 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. Epilepsy of unknown origin however is the most common category in childhood [73].

In New Zealand, while there is little routinely collected information on the health needs of Māori children and young people with epilepsy, the 2006/07 Health Survey estimated a total population prevalence of 5 per 1,000 children aged 0–14 years [59]. In addition, Hauora IV [6] reviewed hospital admissions for epilepsy during 2003–2005 and found that while admissions for Māori and non-Māori children (aged 0–4, 5–9 and 10–14 years) were similar, admissions were higher for Māori than for non-Māori young people aged 15–24 years. In contrast, the authors found no differences between Māori and non-Māori in (all age) epilepsy mortality during 2000–2004 [6].

Data Source and Methods

Definition

Hospital admissions for children and young people aged 0–24 years with epilepsy or status epilepticus listed in any of the first 15 diagnoses

Data Source

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.

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 of 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 that 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.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 1,161 individual Māori children and young people were hospitalised with a diagnosis of epilepsy or status epilepticus, with admission rates per 100,000 population being *significantly* higher than for non-Māori non-Pacific children and young people (RR 1.42 95% CI 1.36–1.49) (**Table 36**).

Table 36. Hospital Admissions for Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Ethnicity, New Zealand 2008–2012

Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Epilepsy or Status Epilepticus						
Māori	1,161	2,688	0.46	153.5	1.42	1.36–1.49
non-Māori non-Pacific	2,582	5,582	0.43	107.8	1.00	

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)

Distribution by Primary Diagnosis

Primary Diagnosis: In New Zealand during 2008–2012, 74.3% of all hospital admissions in Māori children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses, had an epilepsy-related primary diagnosis. Generalised idiopathic epilepsy (24.2%) and unspecified epilepsy (21.9%) were the most frequent epilepsy-related diagnoses. A further 25.7% of admissions were for conditions unrelated to epilepsy, with respiratory conditions, injury and poisoning, and pregnancy and childbirth being the most frequent non epilepsy-related diagnoses (**Table 37**).

Distribution by Age

In New Zealand during 2008–2012, hospital admissions for Māori children with epilepsy or status epilepticus were highest in infants aged less than one year. Rates then decreased during childhood to reach their lowest point at 13 years of age, before increasing again amongst those in their mid-late teens (**Figure 25**).

Trends by Ethnicity

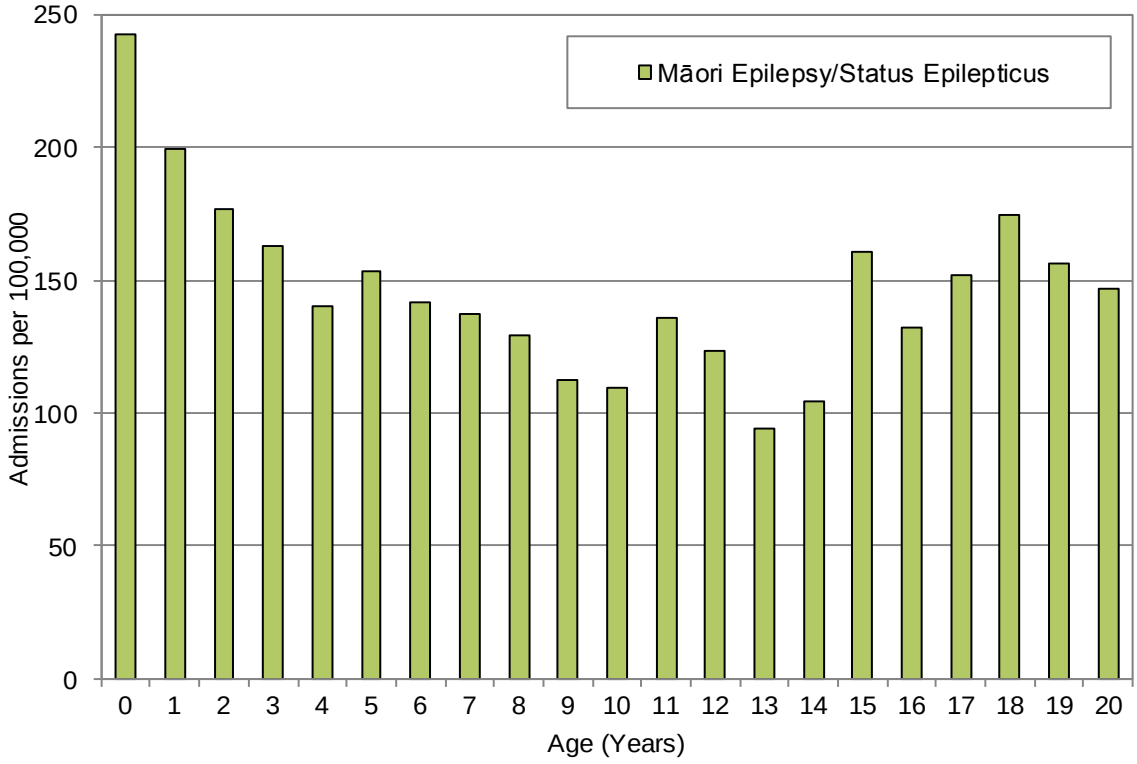
In New Zealand during 2000–01, hospitalisations for Māori children and young people with epilepsy or status epilepticus were similar to those of non-Māori non-Pacific children and young people. However, rates for Māori children and young people declined during the early 2000s, were static during the mid-2000s, and then increased again between 2008–09 and 2012. In contrast, rates for non-Māori non-Pacific children and young people declined during the early-mid 2000s and then became static. Thus ethnic differences were much greater in the late 2000s than they were in 2000–01 (**Figure 26**).

Table 37. Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Epilepsy or Status Epilepticus by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Admission Rate per 100,000 Population	% of Admissions in those with Epilepsy or Status Epilepticus
Māori Children and Young People 0–24 Years				
Epilepsy or Status Epilepticus				
Diagnoses other than Epilepsy*	691	138.2	39.46	25.7
Generalised: Idiopathic	651	130.2	37.18	24.2
Unspecified Epilepsy	589	117.8	33.64	21.9
Status Epilepticus	261	52.2	14.90	9.7
Grand Mal Seizures NOS	161	32.2	9.19	6.0
Focal: Symptomatic with Simple Partial Seizures	106	21.2	6.05	3.9
Focal: Symptomatic with Complex Partial Seizures	98	19.6	5.60	3.6
Generalised: Other	71	14.2	4.05	2.6
Other Epilepsy	42	8.4	2.40	1.6
Petit Mal NOS	10	2.0	0.57	0.4
Focal: Idiopathic with Localised Onset Seizures	8	1.6	0.46	0.3
Total Epilepsy-Related Diagnoses	1,997	399.4	114.04	74.3
Total Admissions	2,688	537.6	153.50	100.0
*Other Diagnoses				
Diseases of Respiratory System	143	28.6	8.17	5.3
Injury and Poisoning	83	16.6	4.74	3.1
Pregnancy, Childbirth, Puerperium	62	12.4	3.54	2.3
Other Diseases Nervous System	45	9.0	2.57	1.7
Other Infectious and Parasitic Diseases	29	5.8	1.66	1.1
Gastroenteritis	21	4.2	1.20	0.8
Congenital Anomalies	21	4.2	1.20	0.8
Dental and Oral Health	21	4.2	1.20	0.8
Other Diagnoses	266	53.2	15.19	9.9
Total Other Diagnoses	691	138.2	39.46	25.7

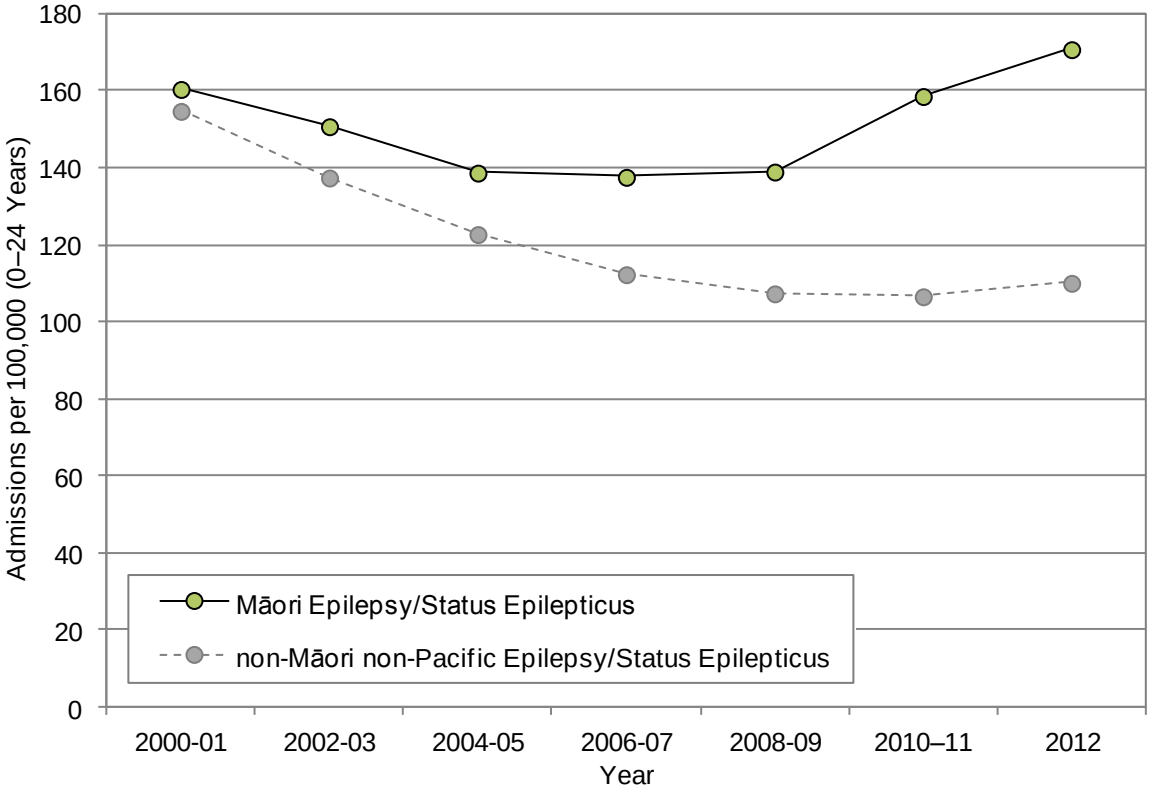
Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Figure 25. Hospital Admissions for Māori Children and Young People with Epilepsy or Status Epilepticus by Age, New Zealand 2008–2012



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)

Figure 26. 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)

CANCER

Introduction

The following section uses data from the NZ Cancer Registry and National Mortality Collection to review cancer incidence and mortality in Māori children and young people.

Background

In New Zealand, there have been a number of reviews of Māori cancer statistics [74] [75], [76]. However, most have focused on adult outcomes, with little information being available on Māori children and young people. One review however, which considered cancer incidence in New Zealand adolescents, found that lymphoid leukaemias, non-Hodgkin lymphomas, astrocytomas, and osteosarcomas were the most frequently diagnosed cancers in Māori children 10–14 years. For Māori young people 15–19 years, malignant gonadal germ cell tumours, lymphoid leukaemias, Hodgkin lymphomas and malignant melanomas were the most frequent cancers, while for Māori young people 20–24 years, malignant gonadal germ cell tumours, thyroid carcinomas and Hodgkin and non-Hodgkin lymphomas made the greatest contribution [77]. Overall, the review found that cancer incidence was lower for Māori than for non-Māori, although this difference was reduced in older young people once the lower rate of melanoma in Māori was taken into account [77].

In terms of mortality, the five year survival ratio for Māori children remains relatively high, with no significant changes in survival ratios between 1998–1999 (0.65) and 2008–2009 (0.77). During this period, there were also no significant differences in survival ratios between Māori and non-Māori children [78]. Over the longer term however, there have been significant improvements in child cancer mortality, with gains largely being achieved through the intensification of therapy using combinations of chemotherapy, radiotherapy, surgery and haematopoietic stem cell transplantation [79]. 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 life [80].

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 0–24 years. Cancer site assigned using ICD-10-AM: 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 (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.

Denominator: Statistics NZ Estimated Resident Population (projected from 2007).

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, gender specific denominators have been used (neoplasms of testis, rates per 100,000 males 0–24 years, malignant neoplasms of ovaries and cervix, rates 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 most notifications are in this age group.

Distribution in Māori Children and Young People

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 Māori children and young people aged 0–24 years, followed by cancers of the testis. Carcinoma in situ of the cervix however, was the most frequent reason for a NZ Cancer Registry notification, accounting for 60.8% of notifications in Māori children and young people during this period (**Table 38**).

Table 38. NZ Cancer Registry Notifications for Māori Children and Young People Aged 0–24 Years by Cancer Type, New Zealand 2002–2011

Cancer Registry Notifications	Total No. 2002–2011	Annual Average	Rate per 100,000	% of Malignant Neoplasms	% of All Notifi-cations
Māori Children and Young People 0–24 Years					
Cancers of Lymphoid and Haematopoietic Tissues					
Acute Lymphoblastic Leukaemia	85	8.5	2.53	14.7	5.5
Non-Hodgkin's Lymphomas	38	3.8	1.13	6.6	2.4
Hodgkin Disease	28	2.8	0.83	4.8	1.8
Acute Myeloblastic Leukaemia	24	2.4	0.71	4.1	1.5
Other Lymphoid/Haematopoietic Neoplasms	19	1.9	0.56	3.3	1.2
Other Myeloid Leukaemias	18	1.8	0.54	3.1	1.2
Cancers of Reproductive Organs					
Malignant Neoplasm of Testis	67	6.7	3.92	11.6	4.3
Malignant Neoplasm of Ovary	10	1.0	0.60	1.7	0.6
Malignant Neoplasm of Cervix	9	0.9	0.54	1.6	0.6
Other Malignant Neoplasms					
Malignant Neoplasm of Brain	50	5.0	1.49	8.6	3.2
Malignant Neoplasms Bone and Cartilage	42	4.2	1.25	7.3	2.7
Malignant Neoplasm Connective/Soft Tissue	30	3.0	0.89	5.2	1.9
Malignant Neoplasm of Thyroid	15	1.5	0.45	2.6	1.0
Malignant Neoplasm of Kidney	14	1.4	0.42	2.4	0.9
Malignant Neoplasm of Adrenal Gland	13	1.3	0.39	2.2	0.8
Malignant Neoplasm of Retina	12	1.2	0.36	2.1	0.8
Malignant Melanoma of Skin	8	0.8	0.24	1.4	0.5
Other Malignant Neoplasms	97	9.7	2.88	16.8	6.2
Total Malignant Neoplasms	579	57.9		100.0	37.2
In Situ Neoplasms or Neoplasms of Uncertain Behavior					
Carcinoma in Situ of Cervix	946	94.6	57.12		60.8
Melanoma in Situ	7	0.7	0.21		0.5
Other In-Situ Neoplasms	17	1.7	0.51		1.1
Neoplasm Uncertain/Unknown Behavior	6	0.6	0.18		0.4
Total In situ or Uncertain Behavior	976	97.6			62.8
Total: All Cancer Registry Notifications	1,555	155.5	46.23		100.0

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)

Cancer Deaths

In New Zealand during 2001–2010, cancers of the brain were the leading cause of cancer mortality in Māori children and young people aged 0–24 years, followed by cancers of the bone and cartilage (**Table 39**).

Table 39. Cancer Deaths in Māori Children and Young People Aged 0–24 Years by Cancer Type, New Zealand 2001–2010

Cancer Deaths	Total No. 2001–2010	Annual Average	Rate per 100,000	% of Cancer Deaths
Māori Children and Young People 0–24 Years				
Cancers of Lymphoid and Haematopoietic Tissues				
Acute Lymphoblastic Leukaemia	17	1.7	0.51	11.8
Acute Myeloblastic Leukaemia	13	1.3	0.39	9.0
Non-Hodgkin's Lymphomas	6	0.6	0.18	4.2
Other Lymphoid/Haematopoietic Neoplasms	7	0.7	0.21	4.9
Cancers of Reproductive Organs				
Malignant Neoplasm of Testis	5	0.5	0.30	3.5
Other Malignant Neoplasms				
Malignant Neoplasm of Brain	24	2.4	0.72	16.7
Malignant Neoplasms Bone and Cartilage	20	2.0	0.60	13.9
Malignant Neoplasm Connective/Soft Tissue	17	1.7	0.51	11.8
Malignant Neoplasm of Adrenal Gland	6	0.6	0.18	4.2
Other Malignant Neoplasms	28	2.8	0.84	19.4
Benign Neoplasms or Neoplasms of Uncertain Behavior				
Neoplasm Uncertain/Unknown Behavior	<3	s	s	s
Total: All Cancer Deaths	144	14.4	4.33	100.0

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 = cells suppressed due to small numbers

Distribution by Ethnicity

In New Zealand during 2002–2011, there were no *significant* differences in NZ Cancer Registry notifications for acute lymphoblastic leukaemia and cancers of the brain between Māori and non-Māori non-Pacific children and young people. In contrast, notifications for Hodgkin Disease and malignant melanoma/melanoma in situ were *significantly* lower for Māori than for non-Māori non-Pacific children and young people (**Table 40**).

Table 40. NZ Cancer Registry Notifications for Selected Cancers in Children and Young People Aged 0–24 Years by Ethnicity, New Zealand 2002–2011

Ethnicity	Notifications: Total Number 2002–2011	Notifications: Annual Average	Notifications per 100,000	Rate Ratio	95% CI
Acute Lymphoblastic Leukaemia 0–24 Years					
Māori	85	8.5	2.53	0.89	0.70–1.13
non-Māori non-Pacific	291	29.1	2.85	1.00	
Hodgkin Disease 0–24 Years					
Māori	28	2.8	0.83	0.49	0.33–0.72
non-Māori non-Pacific	175	17.5	1.71	1.00	
Malignant Neoplasm of Brain 0–24 Years					
Māori	50	5.0	1.49	0.86	0.63–1.18
non-Māori non-Pacific	176	17.6	1.72	1.00	
Melanoma or Melanoma in Situ 0–24 Years					
Māori	15	1.5	0.45	0.10	0.06–0.18
non-Māori non-Pacific	435	43.5	4.26	1.00	

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Carcinoma in Situ of the Cervix

Distribution by Ethnicity

In New Zealand during 2007–2011, NZ Cancer Registry notifications for carcinoma in situ of the cervix in young Māori women were *not significantly* different from those of young non-Māori non-Pacific women (**Table 41**).

Table 41. NZ Cancer Registry Notifications for Carcinoma in Situ of the Cervix in Young Women Aged 15–24 Years by Ethnicity, New Zealand 2007–2011

Ethnicity	Notifications: Total Number 2007–2011	Notifications: Annual Average	Notifications per 100,000	Rate Ratio	95% CI
Carcinoma in Situ of Cervix Females 15–24 Years					
Māori	512	102.4	166.50	0.92	0.84–1.01
non-Māori non-Pacific	1,997	399.4	180.93	1.00	

Source: Numerator: NZ Cancer Registry; Denominator: Statistics NZ Estimated Resident Female Population (projected from 2007)

OBESITY, NUTRITION AND PHYSICAL ACTIVITY

OVERWEIGHT AND OBESITY

THE DISTRIBUTION OF OVERWEIGHT AND OBESITY IN CHILDREN AND YOUNG PEOPLE

Introduction

The following section begins with a brief review of the measurement of overweight and obesity, before exploring their prevalence in Māori children and young people using information from two data sources: The 2011/12 NZ Health Survey, and the Youth'12 Survey of secondary school students.

Background

Increasing rates of childhood obesity are a cause for concern [81, 82] as childhood obesity has significant adverse effects on children's health [83, 84] including an increased risk of adverse psychological effects, lower educational achievement [84, 85], asthma, and slipped upper femoral epiphysis [86-88]. Childhood obesity is also strongly associated with the clustering of a range of risk factors that increase the risk of cardiovascular disease in later life [84]. For those whose obesity persists into adulthood, longer term consequences include an increased risk of cardio-metabolic diseases (diabetes, hypertension, ischaemic heart disease and stroke), premature death, and polycystic ovary syndrome [83].

In New Zealand, Māori children have higher rates of overweight and obesity than non-Māori children, with the 2011/12 NZ Health Survey (NZHS) finding that 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, and 1.40 (95% CI 1.18–1.67) times more likely to be overweight. In addition, the NZHS found that the proportion of Māori children aged 2–14 years who were obese had increased *significantly* between surveys, 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 [81].

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 [89].

Of these, the most popular is the Body Mass Index (BMI) which is calculated using the formula

$$\text{BMI} = \text{weight (kg)} / \text{height (m)}^2$$

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/m^2 being traditionally classified as overweight and those with a BMI of 30 kg/m^2 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 [90]. 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 [90]. 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/m^2 and ≥30 kg/m^2, 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 [91]. 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 [91] [92]) 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 [93] [94] [95], 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 [95]. 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 [96]. Similarly, ethnic differences in the onset of puberty may also make utilisation of a common BMI cut-off difficult, with puberty on average occurring earlier amongst Māori and Pacific groups [97]. Such differences need to be kept in mind when interpreting ethnic specific obesity rates calculated using overseas percentile charts, as they 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) [81] 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 Māori children aged 2–14 years between the 2006/07 and 2011/12 Surveys, before exploring the distribution of overweight and obesity in the most recent 2011/12 NZHS.

Data Sources and Methods

Definitions

The proportion of children aged 2–14 years who are overweight or obese by gender and ethnicity

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

Notes on Interpretation

Sample Size and Weighting: The 2011/12 NZHS [81] 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 that 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) [81].

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 [81]. 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 [81]. 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.

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/m^2). 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 [81].

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 [98].

Trends in Overweight and Obesity

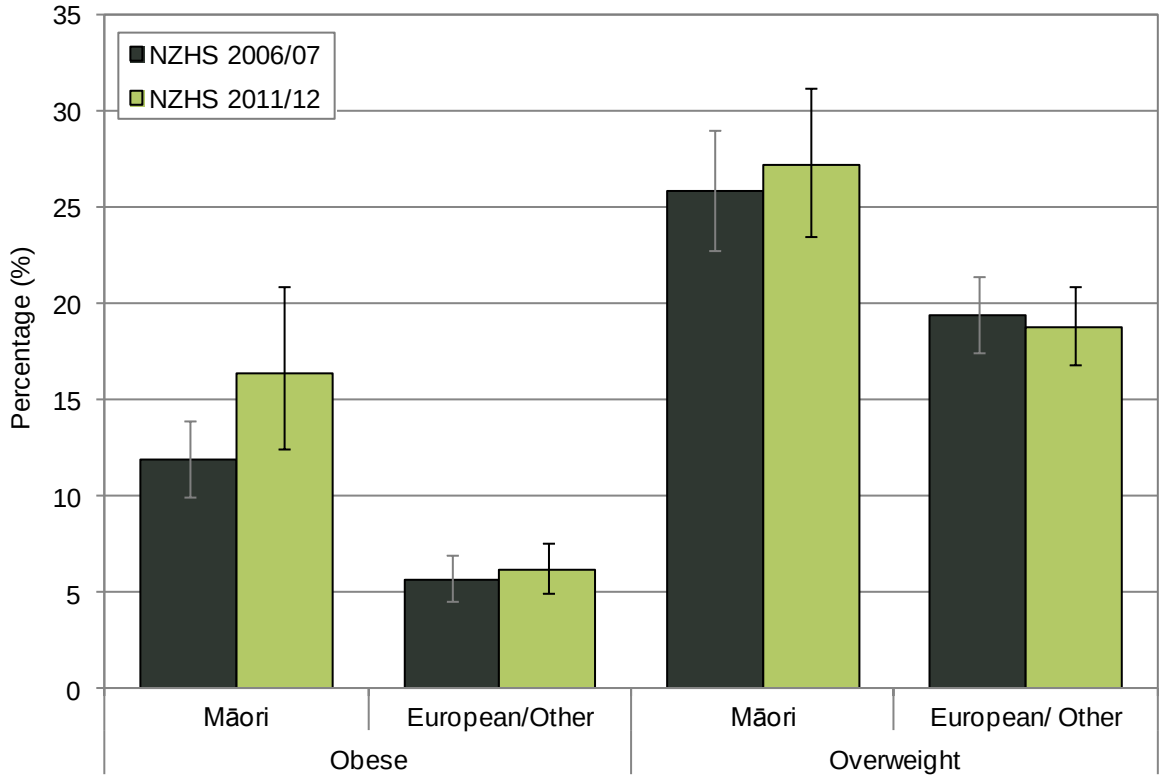
Obesity

The proportion of Māori children aged 2–14 years who were obese increased *significantly* (p=0.03) between NZ Health Surveys, 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. Rates for European/Other children however did not increase *significantly,* being 5.6% (95% CI 4.5–6.9) in 2006/07 and 6.2% (95% CI 5.0–7.6) in 2011/12 (**Figure 27**).

Overweight

The proportion of Māori children aged 2–14 years who were overweight (but not obese) did not change *significantly* between NZ Health Surveys, with rates being 25.8% (95% CI 22.8–29.0) in 2006/07 and 27.2% (95% CI 23.5–31.2) in 2011/12. For European/Other children, rates 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 27**).

Figure 27. Proportion of Children Aged 2–14 Years who were either Overweight or Obese by 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

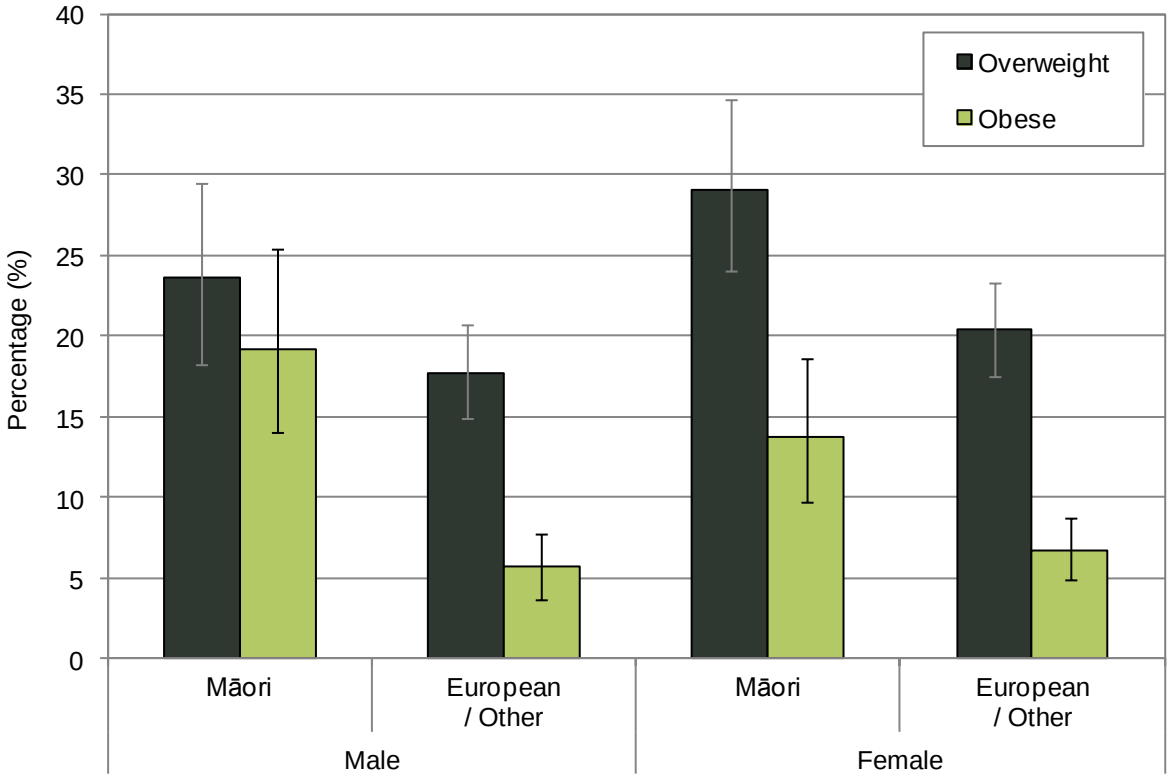
Current Distribution of Overweight and Obesity

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, and 1.40 (95% CI 1.18–1.67) times more likely to be overweight (but not obese) once rates were adjusted for age and gender.

There were however, no *significant* gender differences in the proportion of Māori males and females that were overweight or obese in the 2011/12 NZHS.

Figure 28 reviews the proportion of children aged 2–14 years who were either overweight or obese by gender and ethnicity in the 2011/12 NZHS.

Figure 28. 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

The Youth'12 Survey

Youth'12 was the third national survey of Year 9–15 students (Year 14 and 15 are those students who are repeating Year 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 [99]. The analysis that follows is based on the 1,701 students who identified as being of Māori ethnicity in the Youth'12 Survey [100].

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* [101], and *The Health and Wellbeing of Māori New Zealand Secondary School Students in 2012* [100], a report that focused solely on those 1,701 students who identified as being of Māori ethnicity.

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 [99].

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 [102].

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".

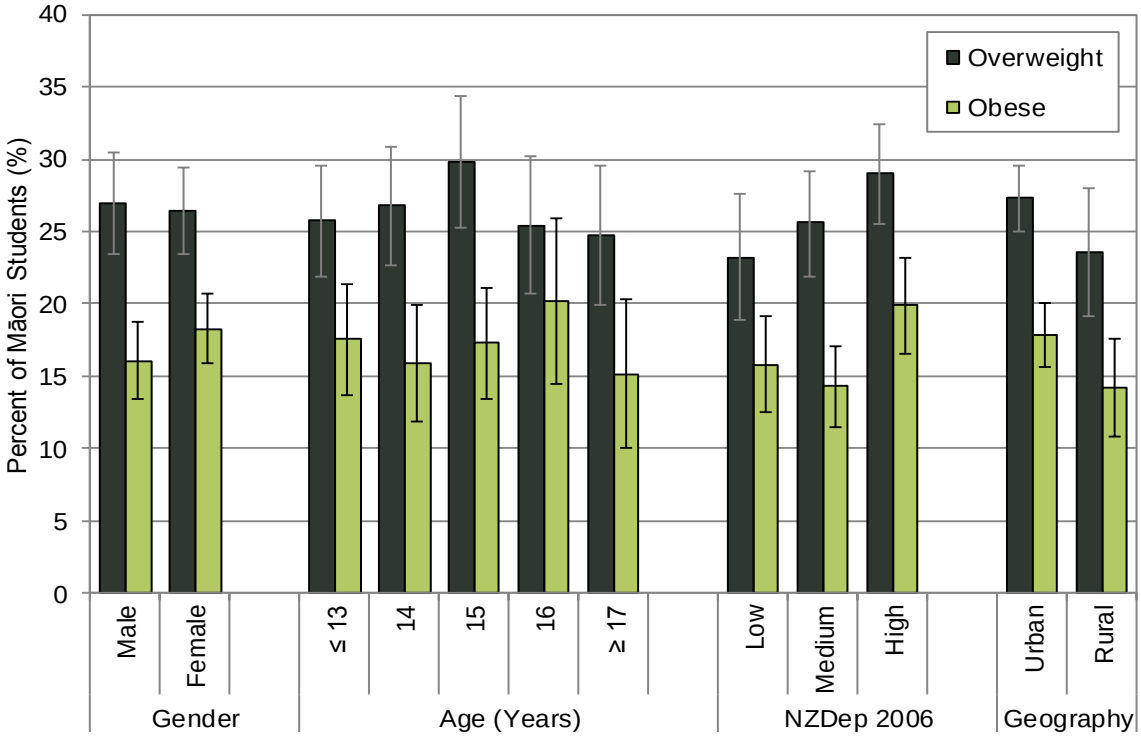
Comparison between Youth'07 and Youth'12 Surveys

There were no *significant* changes in the proportion of Māori students who were overweight or obese between Youth surveys, with rates being 46.5% in Youth'07 and 43.9% in the Youth'12 [100]. In the Youth'12 Survey, 54.5% of Māori students were in a healthy weight range, while 26.7% were overweight and 17.2% were obese [100].

Distribution by Gender, Age, NZDep Index Decile and Geography

In the Youth'12 survey, there were no *significant* gender differences in the proportion of Māori students that were overweight or obese. In the survey, 27.0% (95% CI 23.5–30.5) of Māori males were overweight, and 16.1% (95% CI 13.4–18.7) were obese, while 26.4% (95% CI 23.4–29.4) of Māori females were overweight, and 18.3% (95% CI 15.9–20.7) were obese (**Figure 29**). Similarly there were no *significant* age (by single year), socioeconomic (high deprivation=NZDep deciles 8–10 vs. low deprivation= deciles 1–3) and geographic (rural vs. urban) differences in the proportion of Māori students who were overweight or obese (when each outcome was assessed individually) (**Figure 29**).

Figure 29. Proportion of Māori Secondary School Students 13–17+ Years who were Overweight or Obese by Gender, Age and NZDep06 Index, New Zealand Youth'12 Survey



Source: Youth'12 Survey; Note: NZDep2006: Low = Deciles 1–3; Medium = Deciles 4–7; High = Deciles 8–10; Analysis is for Māori students only

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 [103]. In addition, a number of early markers for coronary artery disease are measureable in obese children including elevated blood pressure and cholesterol [104]. 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 [105].

There is no routinely collected information on the consequences of obesity in Māori children and young people. However, a small number of Māori children and young people do come into contact with the hospital system for obesity related conditions including Type 2 diabetes, slipped upper femoral epiphyses and bariatric surgery. The following section reviews hospitalisations in Māori children and young people for each of these conditions.

TYPE 2 DIABETES

Introduction

The following section reviews hospital admissions for Māori children and young people with Type 2 diabetes listed in any of their first 15 diagnoses.

Background

Type 2 diabetes is a metabolic disorder that leads to high blood glucose levels, as a result of insulin resistance, impaired insulin secretion and increased glucose production. Although more typically developing in adults, it is being diagnosed more frequently in children and adolescents [106] and is considered one of the most serious complications of childhood obesity [107]. The long term consequences of Type 2 diabetes include an increased risk of cardiovascular disease, end stage kidney disease, and blindness [106].

In New Zealand, the incidence of Type 2 diabetes is increasing in children and adolescents, with one Auckland study finding a five-fold increase in children aged 0–14 years between 1995 and 2007. In this study, the annual incidence of Type 2 diabetes was significantly higher for Māori (3.4 per 100,000 95% CI 2.0–5.3) than for European (0.1 per 100,000 95% CI 0.0–0.4) children, with 90% of new cases being of Māori or Pacific Island ethnicity. The authors also noted a more than doubling of the annual incidence of Type 2 diabetes in Māori children between 2000 and 2007 [108].

Data Source and Methods

Definition

Hospital admissions for children and young people aged 0–24 years with Type 2 diabetes 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 Type 2 diabetes (ICD-10-AM E11) 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 Type 2 Diabetes listed in any of the first 15 diagnoses. 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.

Note 2: As the majority of those with Type 2 Diabetes 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 Type 2 Diabetes. This is supported by the finding that during 2008–2012, over three-quarter of hospitalisations for children and young people with Type 2 Diabetes were for reasons other than their diabetes. Thus if they had not been admitted for another reason, their Type 2 diabetes would have gone unrecorded in the hospital admission dataset. 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 Type 2 Diabetes in the community.

Note 2: The admission rates presented here differ to 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. In this analysis, this resulted in the loss of a number of cases where the primary diagnosis was cystic fibrosis, but Type 2 diabetes was recorded as a secondary code. While these changes have been taken into account in back mapping (which is why overall rates are lower in trend analysis than previously), it is likely that the changes were also responsible for the drop off in Type 2 diabetes admissions that occurred in 2008–09, immediately after the introduction of ICD-10-AM V6.

Note 3: 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 2 diabetes with coma and Type 2 diabetes with ketoacidosis, whereas in this report, these have been combined into the category *Type 2 Diabetes with Ketoacidosis/Lactic Acidosis +/−* Coma.

Note 4: If no mention of Type 2 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.

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 157 individual Māori children and young people were hospitalised with a diagnosis of Type 2 diabetes, with admission rates per 100,000 population being *significantly* higher than for non-Māori non-Pacific children and young people (RR 4.36 95% CI 3.66–5.19) (**Table 42**).

Table 42. Hospital Admissions for Children and Young People Aged 0–24 Years with Type 2 Diabetes by Ethnicity, New Zealand 2008–2012

Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Type 2 Diabetes						
Māori	157	311	0.40	17.8	4.36	3.66–5.19
non-Māori non-Pacific	146	211	0.29	4.1	1.00	

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)

Distribution by Primary Diagnosis

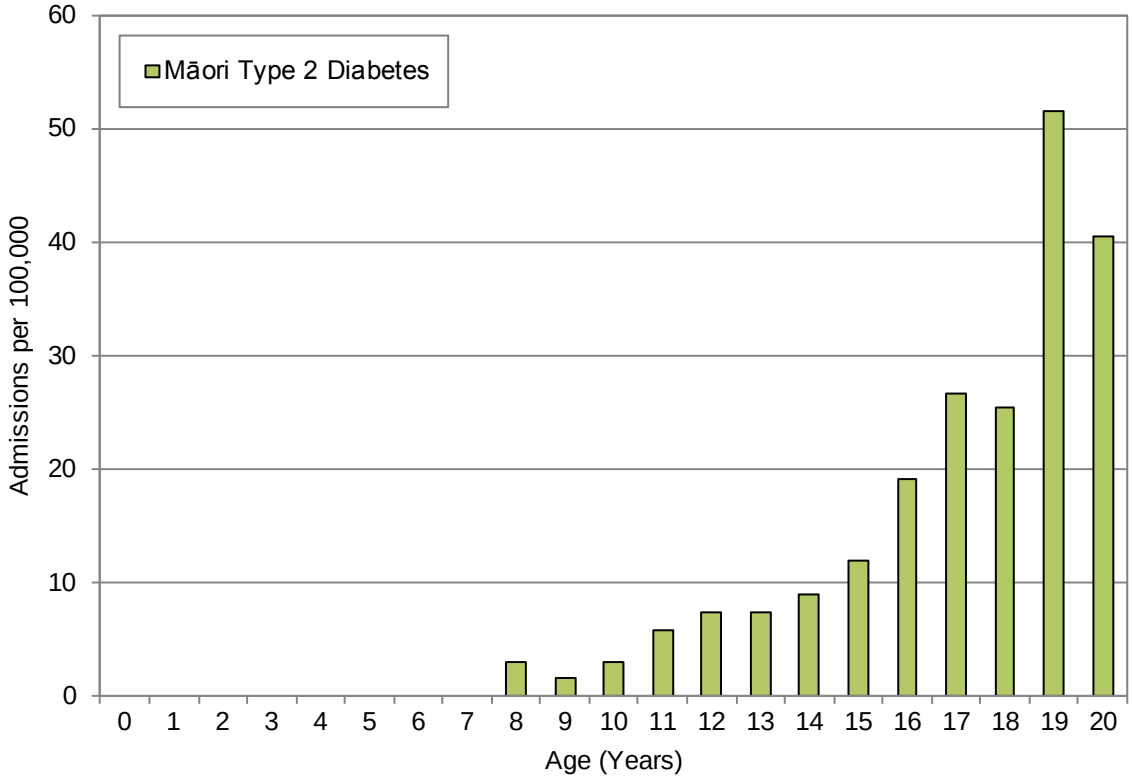
In New Zealand during 2008–2012, 19.0% of hospital admissions for Māori children and young people with Type 2 diabetes in any of their first 15 diagnoses had a diabetes related primary diagnosis. The remaining 81.0% of admissions had non-diabetes related primary diagnoses, with pregnancy and childbirth (14.8%), skin infections (7.7%) and diseases of the respiratory system (5.8%) being the leading non-diabetes related reasons for admission (**Table 43**).

Table 43. Hospital Admissions for Māori Children and Young People Aged 0–24 Years with Type 2 Diabetes by Primary Diagnosis, New Zealand 2008–2012

Primary Diagnosis	Number of Admissions: Total 2008–2012	Number of Admissions: Annual Average	Rate per 100,000 Population	% of Admissions in those with Type 2 Diabetes
Māori Children and Young People 0–24 Years				
Type 2 Diabetes				
Diagnoses other than Type 2 Diabetes*	252	50.4	14.39	81.0
Type 2 Diabetes without Complications	9	1.8	0.51	2.9
Type 2 Diabetes with Multiple Complications	7	1.4	0.40	2.3
Type 2 Diabetes with Ketoacidosis/Lactic Acidosis +/- Coma	6	1.2	0.34	1.9
Type 2 Diabetes with Renal Complications	5	1.0	0.29	1.6
Type 2 Diabetes with Other Specified Complications	32	6.4	1.83	10.3
Total Type 2 Diabetes Admissions	311	62.2	17.76	100.0
*Conditions Contributing to Diagnoses other than Type 2 Diabetes				
Pregnancy Childbirth Post Partum	46	9.2	2.63	14.8
Skin Infections	24	4.8	1.37	7.7
Diseases of Respiratory System	18	3.6	1.03	5.8
Injury and Poisoning	16	3.2	0.91	5.1
Schizophrenia	11	2.2	0.63	3.5
Other Mental Health Issues	12	2.4	0.69	3.9
Urinary Tract Infection	11	2.2	0.63	3.5
Complications Medical Surgical Care	9	1.8	0.51	2.9
Cardiovascular Diseases	9	1.8	0.51	2.9
Gastroenteritis	7	1.4	0.40	2.3
Other Infectious Diseases	7	1.4	0.40	2.3
Acute Pancreatitis	6	1.2	0.34	1.9
Other Diagnoses	76	15.2	4.34	24.4
Total Other Diagnoses	252	50.4	14.39	81.0

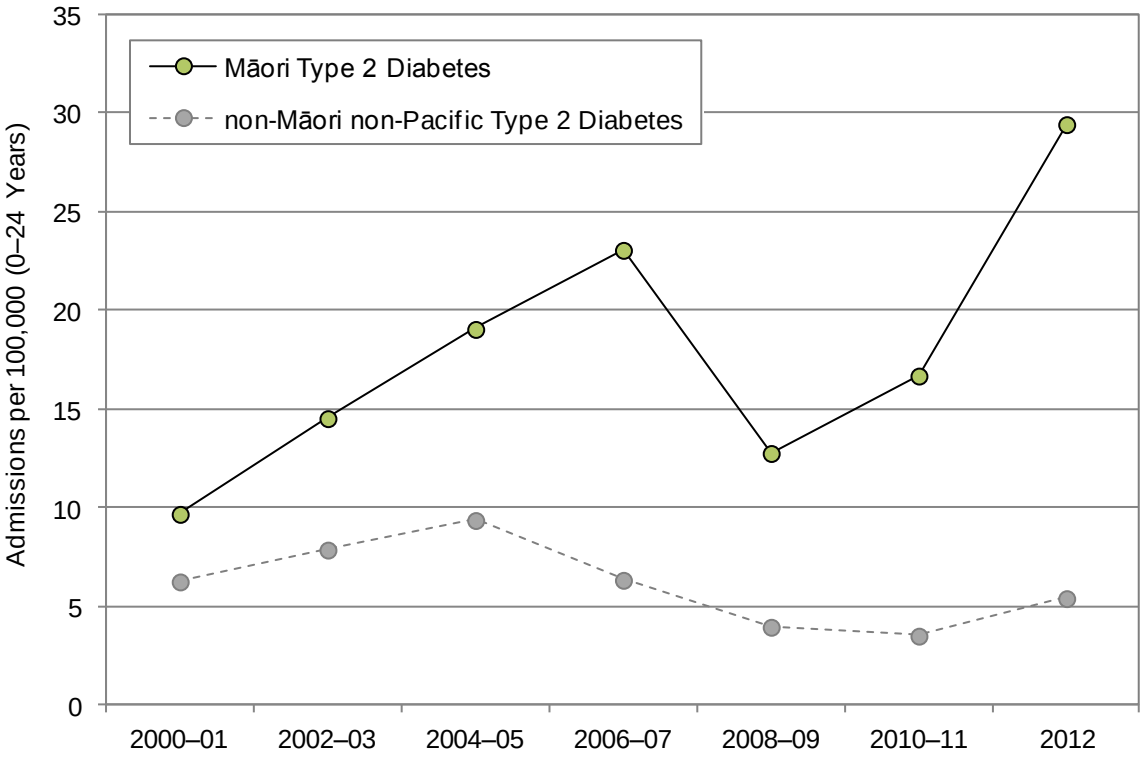
Source: Numerator: National Minimum Dataset, hospital admissions by primary diagnosis for children and young people with Type 2 Diabetes listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Figure 30. Hospital Admissions for Māori Children and Young People with Type 2 Diabetes by Age, New Zealand 2008–2012



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

Figure 31. 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: 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 Age

In New Zealand during 2008–2012, hospitalisations for Māori 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 late teens (**Figure 30**).

Trends by Ethnicity

In New Zealand during 2000–2012, hospital admissions for Māori children and young people with Type 2 diabetes exhibited a general upward trend (although it is likely that changes in the way diabetes was coded (see Data Sources and Methods box) were responsible for the large drop off in rates that occurred in 2008–09). In contrast, admissions for non-Māori non-Pacific children and young people declined after 2004–05. As a result, ethnic differences were larger at the end of this period than at the beginning (**Figure 31**).

SLIPPED UPPER FEMORAL EPIPHYSIS

Introduction

The following section reviews hospital admissions for Māori children and young people with a slipped upper femoral epiphysis listed in any of their first 15 diagnoses.

Background

Children's long bones grow from areas of cartilage near their ends, known as growth plates or physes. The upper femoral epiphysis is the rounded upper end of the thigh bone, with the growth plate being situated between the upper femoral epiphysis and the femoral shaft. A slipped upper femoral epiphysis (SUFE) occurs when the femoral head is displaced from the shaft at the growth plate [109]. The term SUFE is a little misleading since the head of the femur remains in the hip socket, while the rest of the femur has moved upwards, forwards and sideways from its normal position.

SUFE is one of the most common hip disorders in adolescents and affects 10–60 per 100,000 children and adolescents per year [110, 111]. The peak age of incidence is around 13 years for boys and 11 years for girls, and while its precise cause is unclear, obesity is a significant risk factor, especially for bilateral SUFEs [112]. In one New York study, 81.1% of 106 children with a SUFE on x-ray had a BMI above the 95th percentile, compared to 41.1% of 46 children who had a hip x-ray for hip pain but did not have a SUFE [113].

In New Zealand, a study of 211 children admitted to Starship Children's Hospital with a SUFE between 1988 and 2000 estimated that SUFE admission rates for Māori children were 4.2 times higher than for European children, with a much higher proportion of Māori children having bilateral SUFE at first presentation. In contrast, European children presented with higher rates of sequential SUFE, leading to similar bilateral rates over a two year period [114]. While the authors speculated that the ethnic differences seen may have been due to ethnic differences in body mass index (BMI) and physical development, information on weight, height and bone age was not collected in this study [114].

Data Source and Methods

Definition

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

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.

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

Distribution in Māori Children and Young People

Numbers of Individuals and Hospital Admissions

In New Zealand during 2008–2012, a total of 289 individual Māori children and young people were hospitalised with a slipped upper femoral epiphysis, with admission rates per 100,000 population being *significantly* higher than for non-Māori non-Pacific children and young people (RR 5.09 95% CI 4.29–6.04) (**Table 44**).

Table 44. Hospital Admissions for Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis by Ethnicity, New Zealand 2008–2012

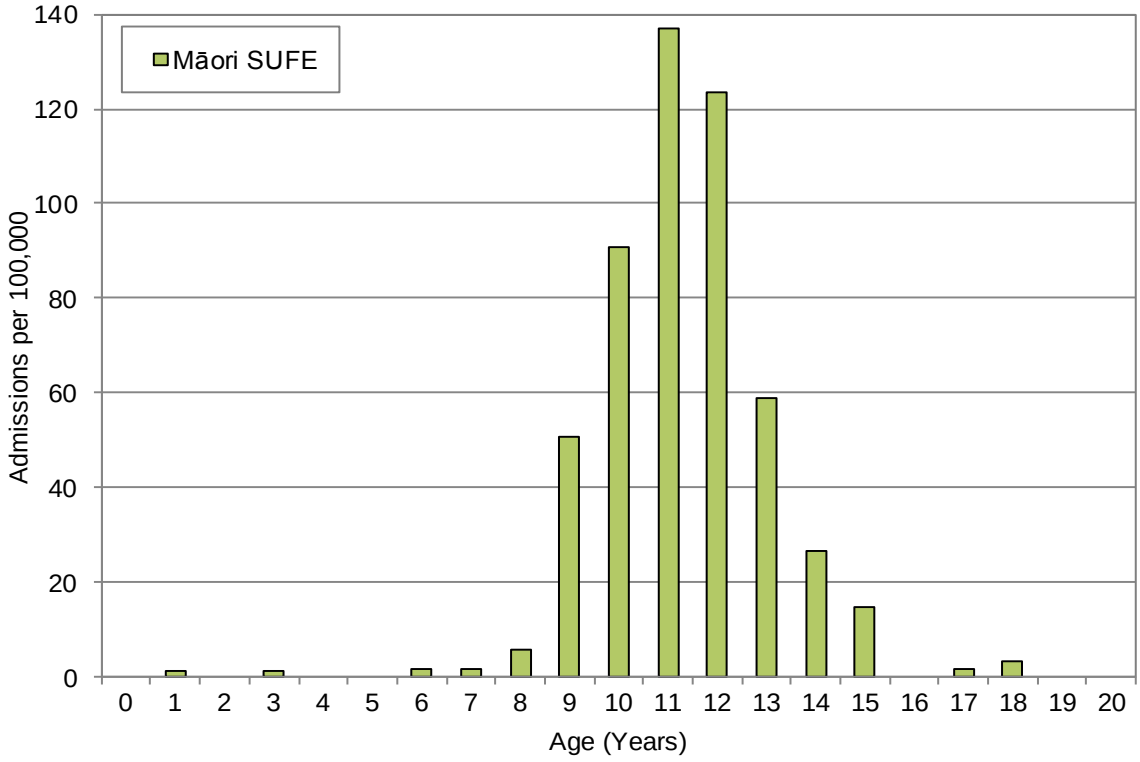
Ethnicity	Total Number Individuals 2008–2012	Total Number of Admissions 2008–2012	Average No. Admissions per Individual per Year	Admission Rate per 100,000 Population	Rate Ratio	95% CI
Slipped Upper Femoral Epiphysis						
Māori	289	358	0.25	20.4	5.09	4.29–6.04
non-Māori non-Pacific	173	208	0.24	4.0	1.00	

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)

Distribution by Age

In New Zealand during 2008–2012, hospitalisations for Māori 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, before declining again amongst those in their early teens (**Figure 32**).

Figure 32. Hospital Admissions for Māori Children and Young People with a Slipped Upper Femoral Epiphysis by Age, New Zealand 2008–2012

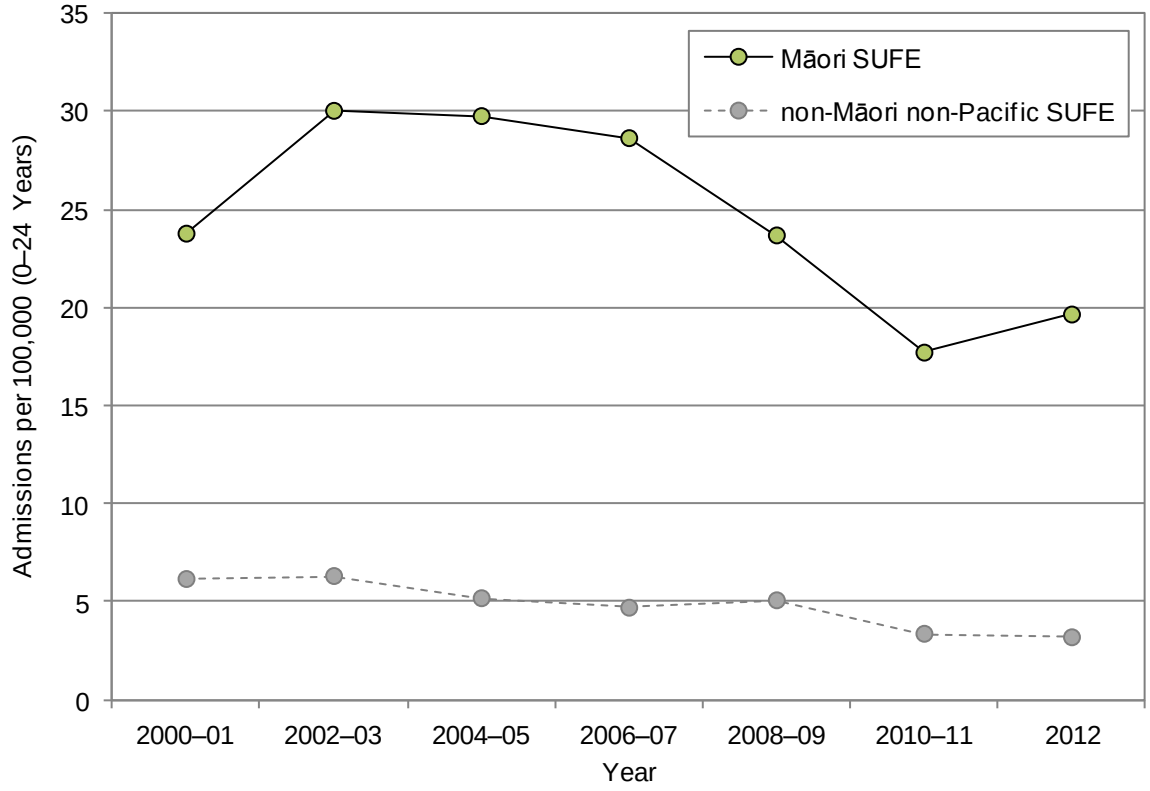


Source: Numerator: National Minimum Dataset, hospital admissions for children and young people with SUFE listed in any of the first 15 diagnoses; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Trends by Ethnicity

In New Zealand during 2000–2012, hospital admissions for Māori children and young people with SUFE were consistently higher than for non-Māori non-Pacific children and young people. Rates for Māori children and young people declined between 2006–07 and 2010–11 however, although a small upswing in rates was evident in 2012 (**Figure 33**).

Figure 33. Hospital Admissions for Children and Young People Aged 0–24 Years with a Slipped Upper Femoral Epiphysis by Ethnicity, 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

The following section reviews hospital admission for bariatric surgery in young people aged 15–24 years using data from the National Minimum Dataset. Note: Because of the small number of cases involved, the analysis initially focuses on the total population, with rates for Māori young people being presented at the end of the section.

Background

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 [115] suggest that a post-pubertal adolescent with a BMI of >40 kg/m^2, or >35 kg/m^2 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.

Data Source and Methods

Definition

1. *Hospital admissions for young people aged 15–24 years with bariatric surgery listed in any of their first 15 procedures*

Data Source

1. National Minimum Dataset

Numerator: Hospital admissions for young people aged 15–24 years with bariatric surgery listed in any of their first 15 procedures

Specific procedures (ACHI codes) included: Gastric reduction (3051100), Laparoscopic gastric reduction (3051101), Gastric bypass (3051200), Laparoscopic biliopancreatic diversion (3051201), Biliopancreatic diversion (3051202), Surgical reversal of procedure for morbid obesity (3051400), Insertion of gastric bubble (balloon) (9095000), Adjustment of gastric band (9095300), Revision of gastric band (1421500).

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

Notes on Interpretation

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.

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 45**).

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 46**).

Table 45. Hospital Admissions for Bariatric Surgery by Primary Diagnosis in Young People Aged 15–24 Years, New Zealand 2008–2012

Primary Diagnosis	Number: 2008–2012	Percent of Admissions (%)
Bariatric Surgery		
Obesity	27	65.9
Type 2 Diabetes	4	9.8
Mechanical Complications Gastrointestinal Prosthetic Devices	4	9.8
Other Diagnoses	6	14.6
Total	41	100.0

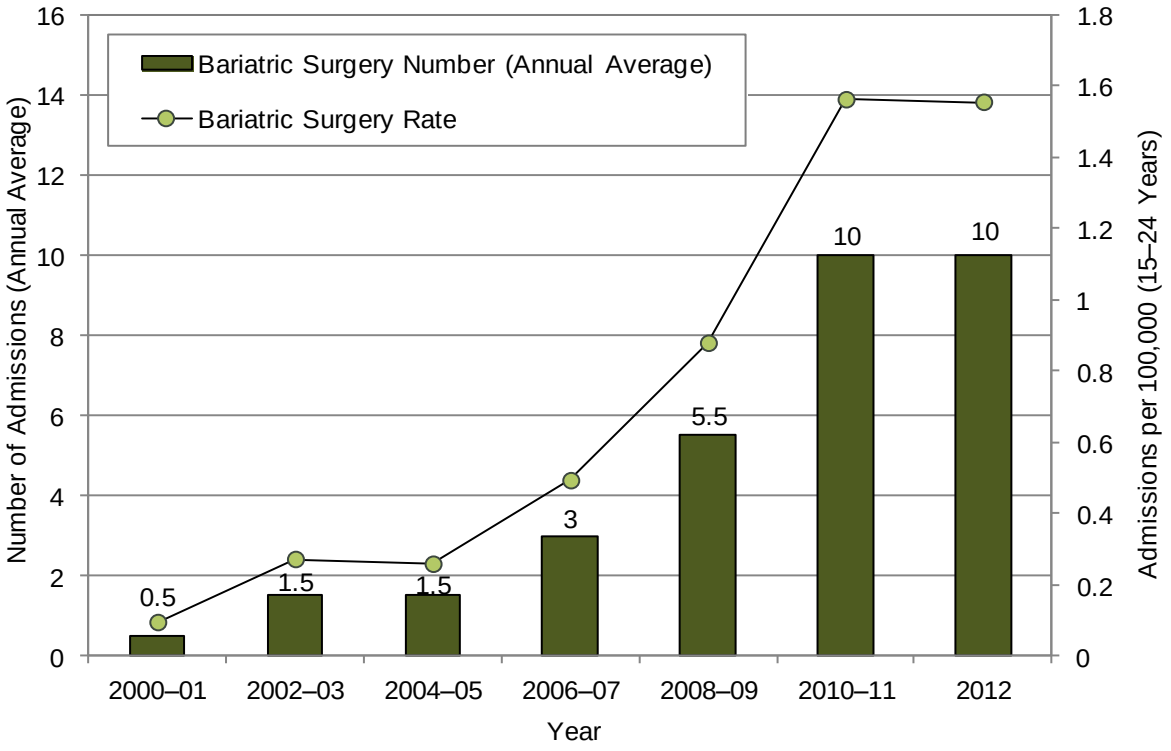
Source: Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population (projected from 2007)

Table 46. Hospital Admissions for Bariatric Surgery by Primary Procedure in Young People Aged 15–24 Years, New Zealand 2008–2012

Primary Procedure	Number: 2008–2012	Percent of Admissions (%)
Bariatric Surgery		
Laparoscopic Gastric Reduction	17	41.5
Gastric Bypass for Morbid Obesity	12	29.3
Revision of Gastric Band	4	9.8
Biliopancreatic Diversion	3	7.3
Other Procedures	5	12.2
Total	41	100.0

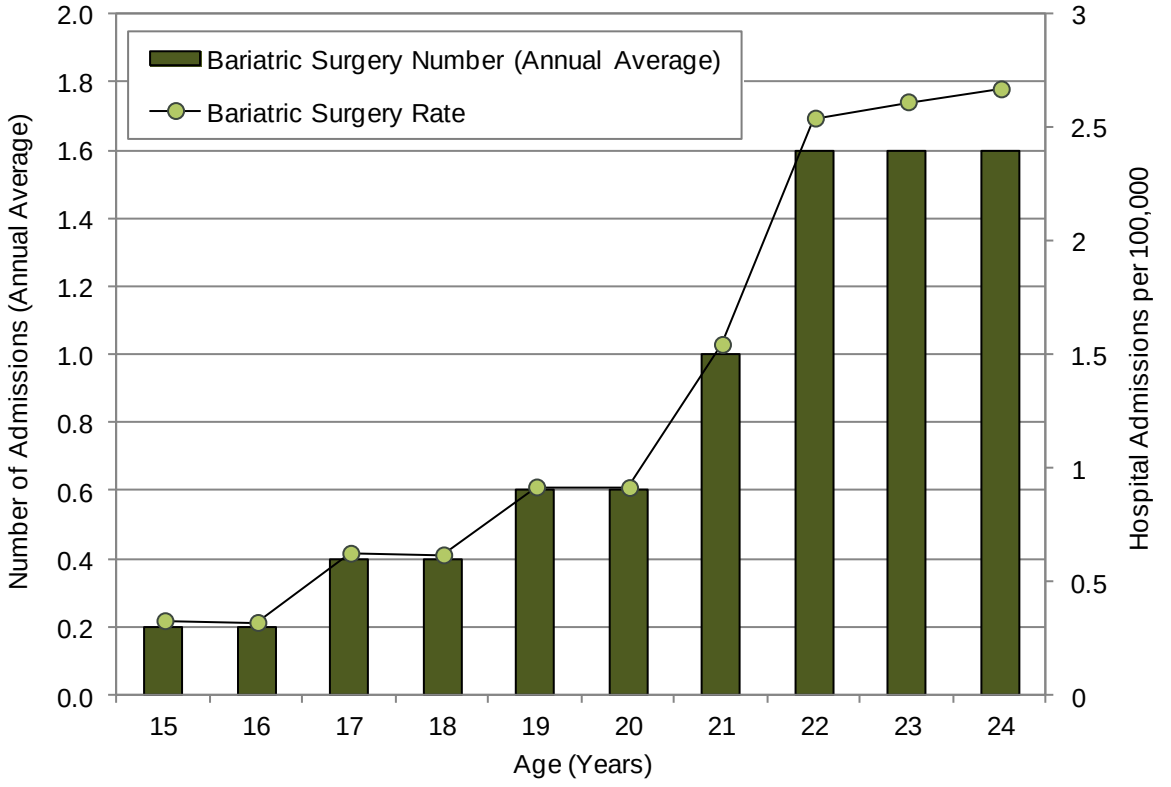
Source: Numerator: Hospital Admissions Dataset; Denominator: Statistics New Zealand Estimated Resident Population (projected from 2007)

Figure 34. Hospital Admissions for Bariatric Surgery in Young People Aged 15–24 Years, New Zealand 2000–2012



Source: Numerator: Hospital Admissions Dataset; Denominator: Statistics New Zealand Estimated Resident Population (Projected from 2007)

Figure 35. Hospital Admissions for Bariatric Surgery in Young People Aged 15–24 Years by Age, New Zealand 2008–2012



Source: Numerator: Hospital Admissions Dataset; Denominator: Statistics New Zealand Estimated Resident Population (projected from 2007)

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 34**).

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 35**).

Distribution by Ethnicity and Gender

In New Zealand during 2008–2012, while bariatric surgery admissions were lower for Māori than for European/Other young people, these differences did not reach statistical significance. Admission rates however, were *significantly* higher for females than for males (**Table 47**).

Table 47. Hospital Admissions for Young People Aged 15–24 Years for Bariatric Surgery by Ethnicity and Gender, New Zealand 2008–2012

Variable	Number: Total 2008–2012	Rate	Rate Ratio	95% CI
Bariatric Surgery				
Prioritised Ethnicity				
Asian/Indian	<3	s	s	s
European/Other	28	1.52	1.00	
Māori	4	0.64	0.42	0.15–1.20
Pacific	6	2.29	1.51	0.62–3.64
Gender				
Female	34	2.20	1.00	
Male	7	0.43	0.20	0.09–0.44

Source: Numerator: Hospital Admissions Dataset; Denominator: Statistics New Zealand Estimated Resident Population (projected from 2007); Note: Rate is per 100,000 15–24 years; Note: s = cells suppressed due to small numbers; Ethnicity is Level 1 Prioritised

NUTRITION AND PHYSICAL ACTIVITY

BREASTFEEDING AND SOLIDS

Introduction

The following section reviews breastfeeding rates for Māori babies at <6 weeks, 3 months and 6 months using Plunket data. The proportion of Māori babies who were given solid food prior to four months is also reviewed, using data from the 2011/12 NZ Health Survey.

Background

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 [81]. 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 [116]. Similarly, 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 [116-118].

In New Zealand, research suggests that most Māori women are aware of the benefits of breastfeeding and wish to breastfeed. However a number of barriers exist including difficulty establishing breastfeeding, lack of professional support, perceptions of inadequate milk supply, the need to return to work, and whānau members who have either not breastfed, or who have had trouble breastfeeding. Other barriers include not attending antenatal classes, the false belief that you shouldn't breastfeed if you smoke and the knowledge that bed-sharing is frowned upon (although it makes breastfeeding at night easier) [119]. Glover et al. [119] note that such research suggests that "*there are opportunities for maternity services to improve, monitor and maintain the effectiveness and delivery of antenatal education, breastfeeding resources and postnatal care to Māori. There is also a need for recognition and valuing of Māori infant care practices and health beliefs and adaptations to increase partner involvement and other whānau support*".

Exclusive/Full Breastfeeding Rates in Plunket Babies

Data Sources and Methods

Indicator

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

Distribution in Māori Babies

In New Zealand during the year ending June 2013, 58.2% of Māori Plunket babies were exclusively or fully breastfed at <6 weeks, with the proportion falling to 43.5% at 3 months and 15.4% at 6 months (**Table 48**).

Table 48. Breastfeeding Status of Plunket Babies by Ethnicity, New Zealand Year Ending June 2013

Ethnicity	Number of Babies: Year Ending June 2013				
	Exclusive	Full	Artificial	Partial	Total
<6 Weeks					
Māori	3,746	705	1,690	1,510	7,651
non-Māori non-Pacific	16,925	3,098	3,522	5,693	29,238
3 Months					
Māori	3,191	1,116	3,648	1,954	9,909
non-Māori non-Pacific	16,564	4,703	7,949	6,819	36,035
6 Months					
Māori	903	636	4,796	3,631	9,967
non-Māori non-Pacific	6,797	3,283	11,359	14,470	35,909
Ethnicity	Percent of Babies: Year Ending June 2013				
	Exclusive	Full	Artificial	Partial	Exclusive or Full
<6 Weeks					
Māori	49.0	9.2	22.1	19.7	58.2
non-Māori non-Pacific	57.9	10.6	12.0	19.5	68.5
3 Months					
Māori	32.2	11.3	36.8	19.7	43.5
non-Māori non-Pacific	46.0	13.1	22.1	18.9	59.0
6 Months					
Māori	9.1	6.4	48.1	36.4	15.4
non-Māori non-Pacific	18.9	9.1	31.6	40.3	28.1

Source: Plunket Client Information System

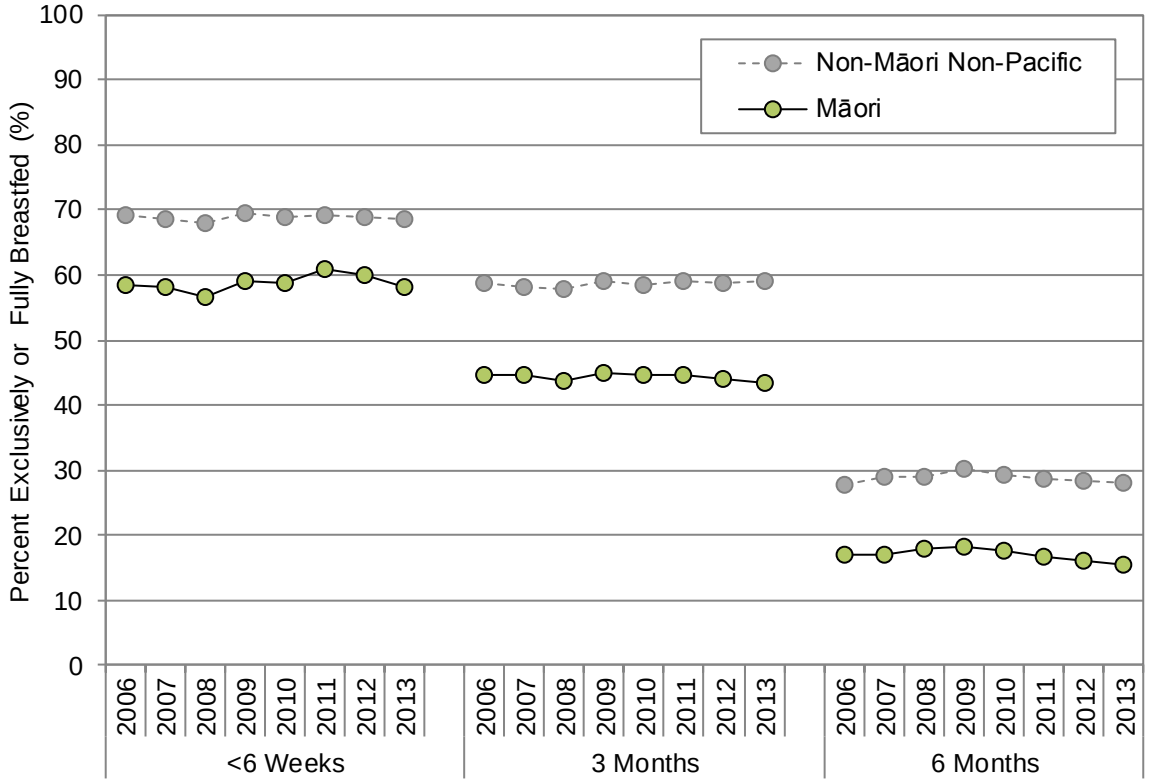
Trends by Age and Ethnicity

In New Zealand during the years ending June 2006–2013, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were consistently lower for Māori than for non-Māori non-Pacific babies, with rates being relatively static for both ethnic groups during this period (**Figure 36**).

Distribution by Ethnicity and NZ Deprivation Index Decile

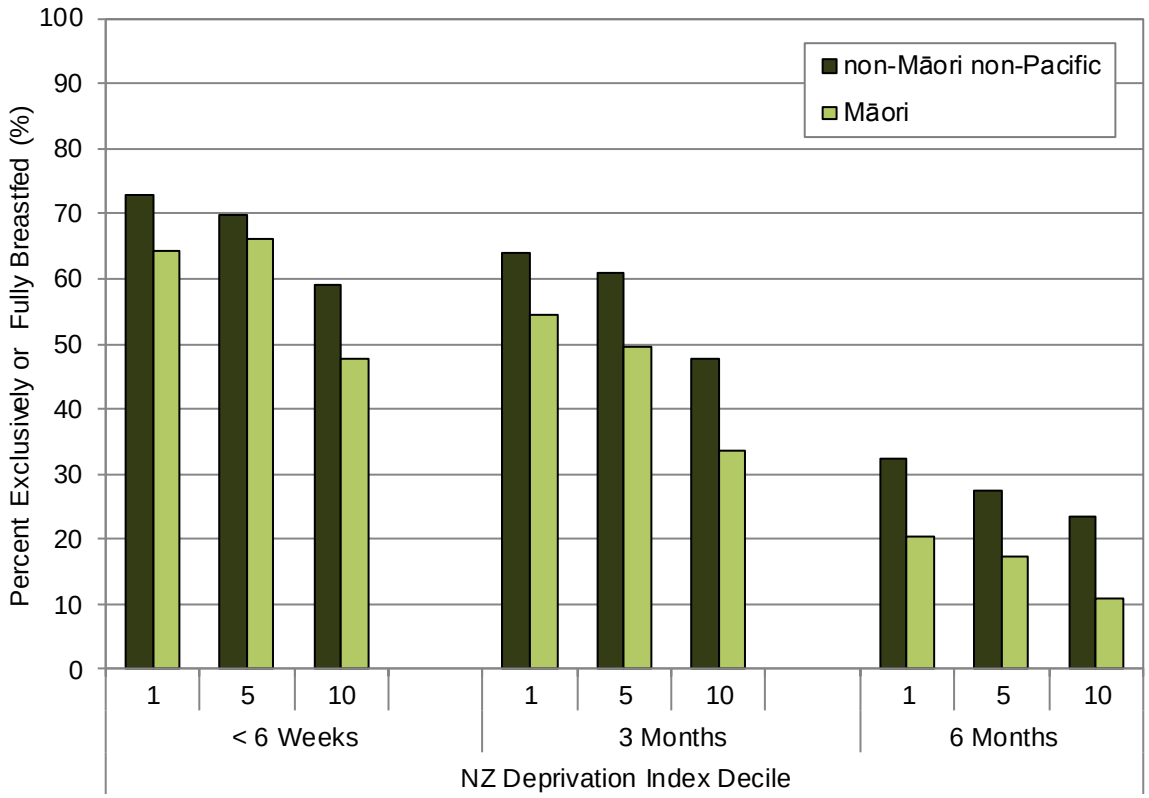
In New Zealand during the year ending June 2013, exclusive/full breastfeeding rates at <6 weeks, 3 months and 6 months were higher for Māori babies from the least deprived (NZDep decile 1) areas than for Māori babies from the most deprived (NZDep decile 10) areas. At each level of NZDep deprivation however, breastfeeding rates were lower for Māori than for non-Māori non-Pacific babies (**Figure 37**).

Figure 36. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age and Ethnicity, New Zealand, Years Ending June 2006–2013



Source: Plunket Client Information System

Figure 37. Proportion of Plunket Babies who were Exclusively or Fully Breastfed by Age Ethnicity and NZ Deprivation Index Decile, New Zealand, Year Ending June 2013



Source: Plunket Client Information System

Babies Given Solid Food before Four Months of Age

In the 2011/12 NZ Health Survey [81], 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 briefly reviews changes in the proportion of Māori 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 in Māori children, in the most recent 2011/12 NZHS.

Data Sources and Methods

Indicator

The proportion of children aged 4 months to 4 years who were given solid food before four months of age

Data Source

The 2011/12 New Zealand Health Survey (NZHS)

The data 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

Notes on Interpretation

The 2011/12 NZ Health Survey [81] 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 115**.

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 that 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) [81].

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 [98].

Trends in Giving Solids before Four Months

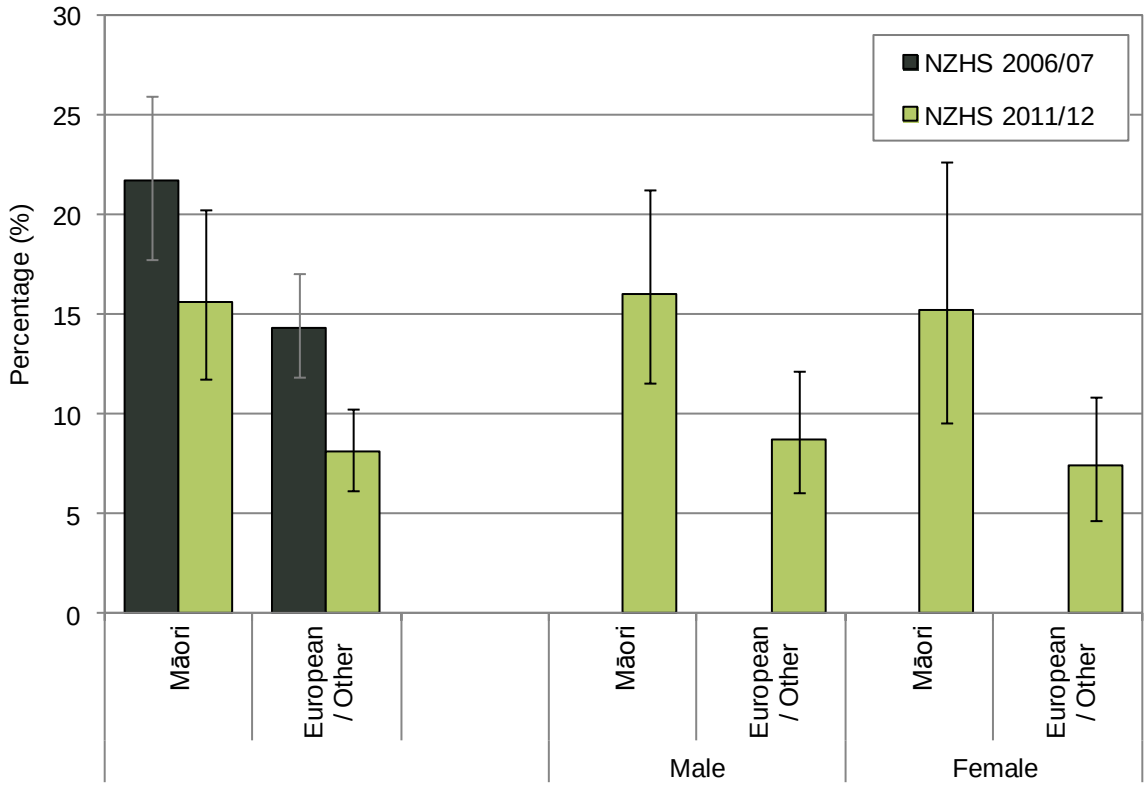
The proportion of Māori children aged 4 months to 4 years given solid food before four months of age declined *significantly* (p=0.04) between NZ Health Surveys, with rates falling from 21.7% (95% CI 17.8–26.0) in 2006/07 to 15.6% (95% CI 11.7–20.2) in 2011/12 (**Figure 38**).

Current Distribution of Giving Solids before Four Months

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, once rates were adjusted for age and gender.

There were however, no *significant* gender differences in the proportion of Māori children given solid foods before four months of age in the 2011/12 NZ Health Survey (**Figure 38**).

Figure 38. 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

OTHER NUTRITIONAL INDICATORS

Introduction

The following section reviews a range of nutritional indicators of relevance to Māori 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.

Background

Optimal nutrition during childhood is vital for good health, growth and development, and the prevention of obesity [120]. Children's dietary patterns and food choices are strongly influenced by their parents and caregivers however. 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 diet [121-123]. Children's diets are also influenced by a complex interplay of personal, social, cultural, and economic factors [120, 123]. The wider food environment has also 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 [120]. Understanding the role such 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, can also influence nutritional intake, resulting in both under and over nutrition. Food insecurity is influenced by family income, the number of people living in a household and the location of households in relation to food sources, [120, 124, 125] with research suggesting that food security may be an issue for some Māori families. For example the longitudinal Survey of Families, Income and Employment found that Māori were 2.8 (95% CI 2.5–3.1) times more likely to be food insecure than European peoples, although this difference fell to 1.2 (95% CI 1.1–1.4) times more likely once other factors such as family composition, educational qualifications and household income were taken into account [124].

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 [81] 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 that 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) [81].

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 [81]. 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 [81]. 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 [98].

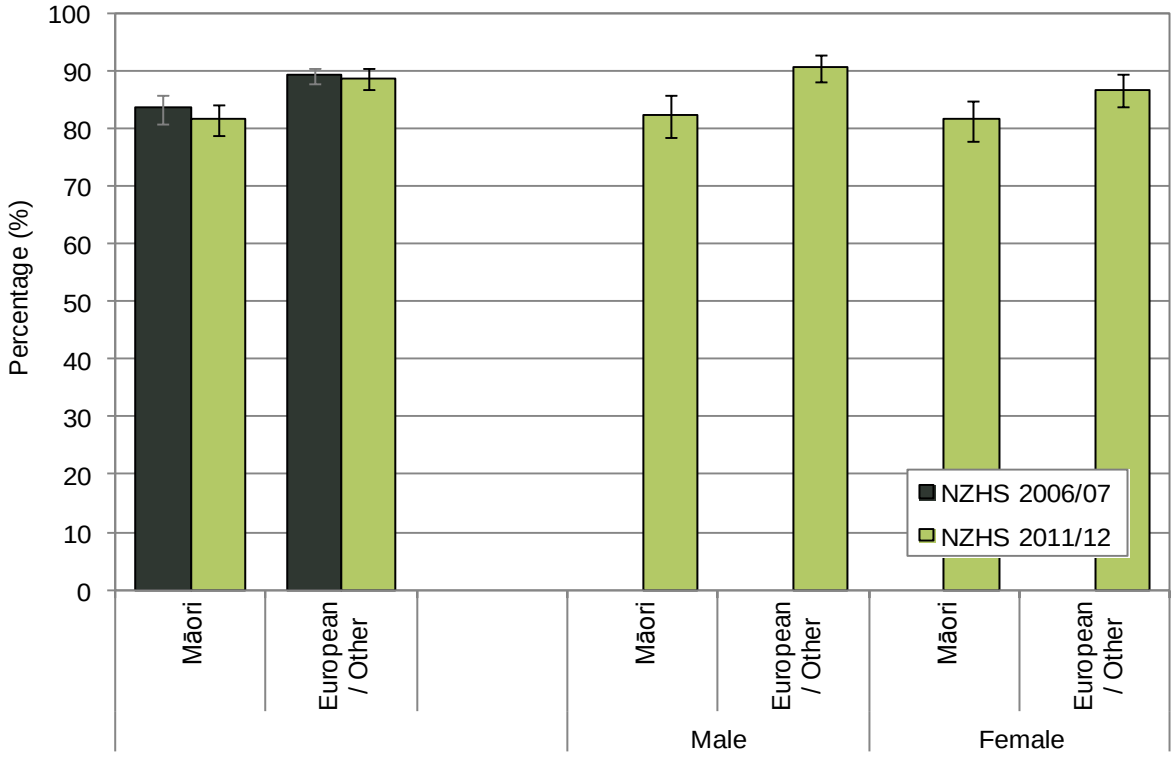
Breakfast Eaten at Home

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 [81]. Further, the 2002 National Children's Nutrition Survey [97] found that children who usually eat breakfast at home have, on average, a lower BMI, even once other potentially confounding risk factors are taken into account.

Trends in Proportion of Children Who Ate Breakfast at Home

There were no *significant* changes in the proportion of Māori 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 children ate breakfast at home every day in the last week (**Figure 39**).

Figure 39. Proportion of Children 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

Current Distribution of Children Who Ate Breakfast at Home

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.

There were however, no *significant* gender differences in the proportion of Māori children who ate breakfast at home every day in the last week in the 2011/12 NZ Health Survey (**Figure 39**).

Fast Food Consumption

Trends in Proportion of Children Who Ate Fast Food

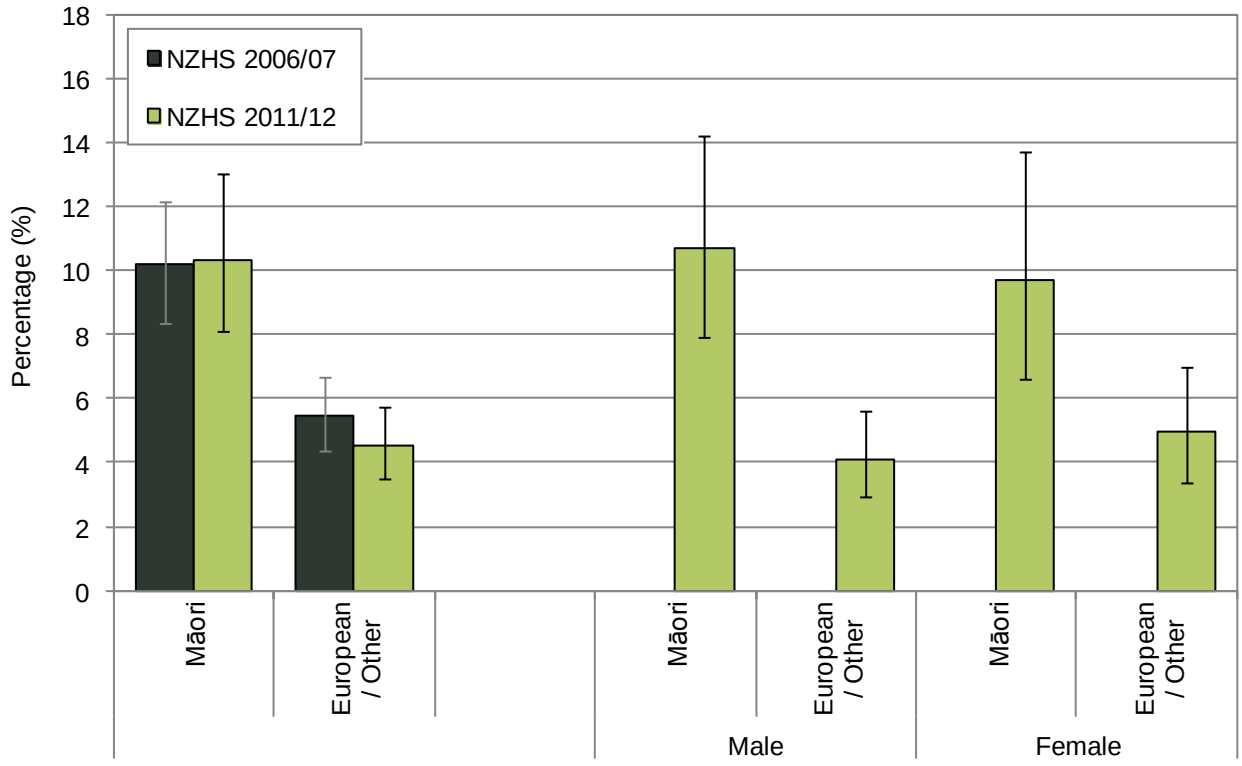
There were no *significant* changes in the proportion of Māori children who had eaten fast food three or more times in the past week between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 10.3% (95% CI 8.1–13.0) of Māori children had eaten fast food three or more times in the past week (**Figure 40**).

Current Distribution of Children Who Ate Fast Food

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.

There were however, no *significant* gender differences in the proportion of Māori children who had eaten fast food three or more times in the past week in the 2011/12 NZ Health Survey week (**Figure 40**).

Figure 40. 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

Fizzy Drink Consumption

Trends in the Proportion of Children Who Consumed Fizzy Drinks

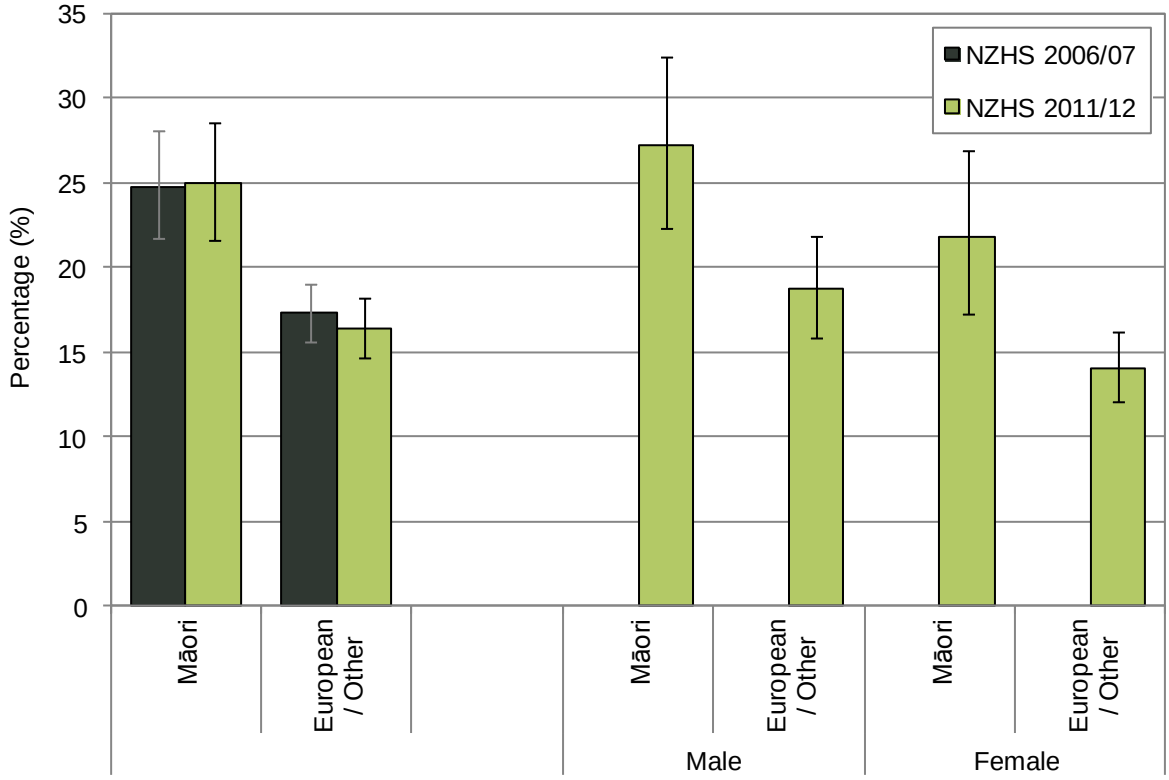
There were no *significant* changes in the proportion of Māori children who had consumed fizzy drinks three or more times in the past week between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 25.0% (95% CI 21.6–28.6) of Māori children had consumed fizzy drinks three or more times in the past week (**Figure 41**).

Current Distribution of Children Who Consumed Fizzy Drinks

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.

There were however, no *significant* gender differences in the proportion of Māori children who had consumed fizzy drinks three or more times in the past week in the 2011/12 NZ Health Survey (**Figure 41**).

Figure 41. Proportion of Children Aged 2–14 Years Who Had Fizzy Drinks 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

Youth'12 Survey

Youth'12 was the third national survey of Year 9–15 students (Year 14 and 15 are those students who are repeating Year 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 [99]. The analysis that follows is based on the 1,701 students who identified as being of Māori ethnicity in the Youth'12 Survey [100].

Data Sources and Methods

Definitions

1. Secondary school students aged 13–17+ years who always ate breakfast

2. Secondary school students aged 13–17+ years who ate meals with family 5+ times in the last 7 days

3. 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* [101], and *The Health and Wellbeing of Māori New Zealand Secondary School Students in 2012* [100], a report that focused solely on those 1,701 students who identified as being of Māori ethnicity.

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) [99].

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 [126], which reported five ethnic groups: Māori, Pacific, Asian, European and Other.

Eating Breakfast

In the Youth'12 Survey, 45.2% (95% CI 42.2–48.3) of Māori secondary school students said they always ate breakfast, with the proportion of males (53.3% 95% CI 49.7–56.9) being *significantly* higher than for females (38.0% 95% CI 33.7–42.4).

While there were no age (by single year) or geographic (urban vs. rural) differences in the proportion of Māori students who always ate breakfast, a *significantly* higher proportion of Māori students from the least deprived (NZDep deciles 1–3) areas (52.8% 95% CI 46.4–59.2) said they always ate breakfast, than students from the most deprived (NZDep06 deciles 8–10) areas (38.0% 95% CI 34.2–41.7) (**Figure 42**).

Family Meals

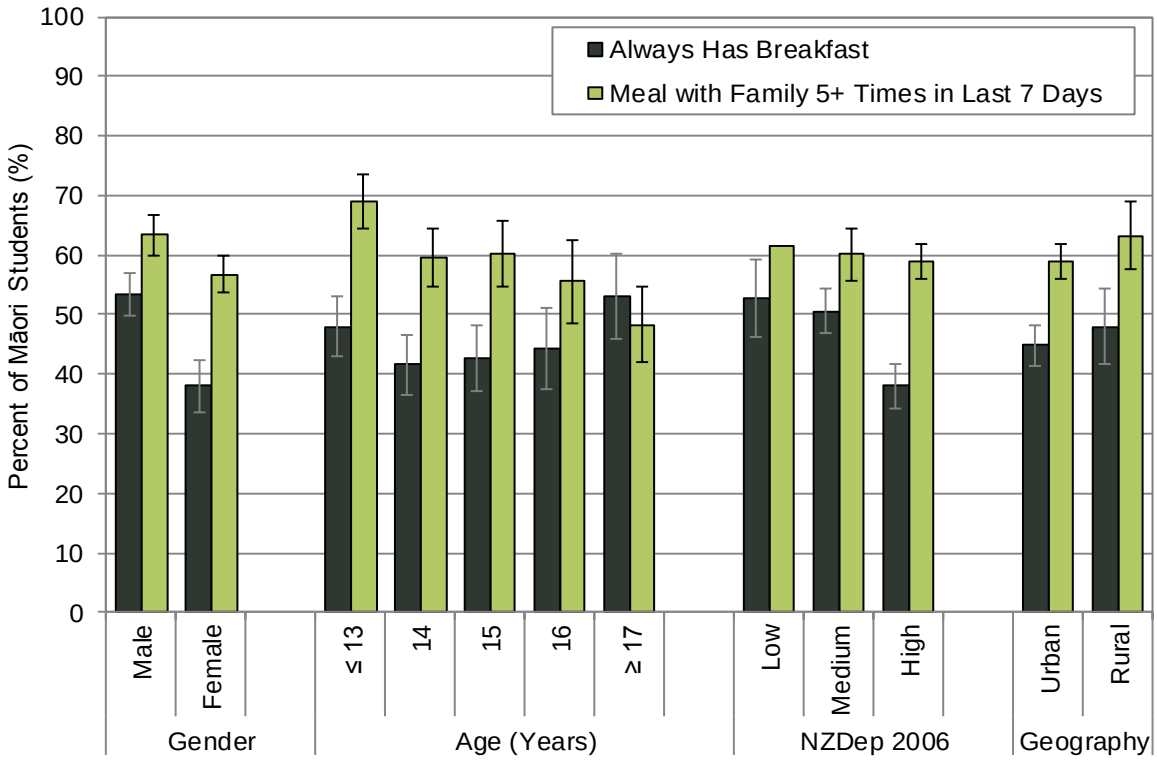
In the Youth'12 Survey, 59.8% (95% CI 57.3–62.4) of Māori secondary school students said they ate a meal with family five or more times in the last seven days, with the proportion of males (63.4% 95% CI 59.9–66.8) being *significantly* higher than for females (56.7% 95% CI 53.6–59.8).

While there were no NZDep06 or geographic (urban vs. rural) differences, a *significantly* higher proportion of younger Māori students (13 and under, 69.0% 95% CI 64.5–73.4) than older students (17 and over, 48.3% 95% CI 42.0–54.6) said they ate meals with family five or more times in the last seven days (**Figure 42**).

Fruit and Vegetable Consumption

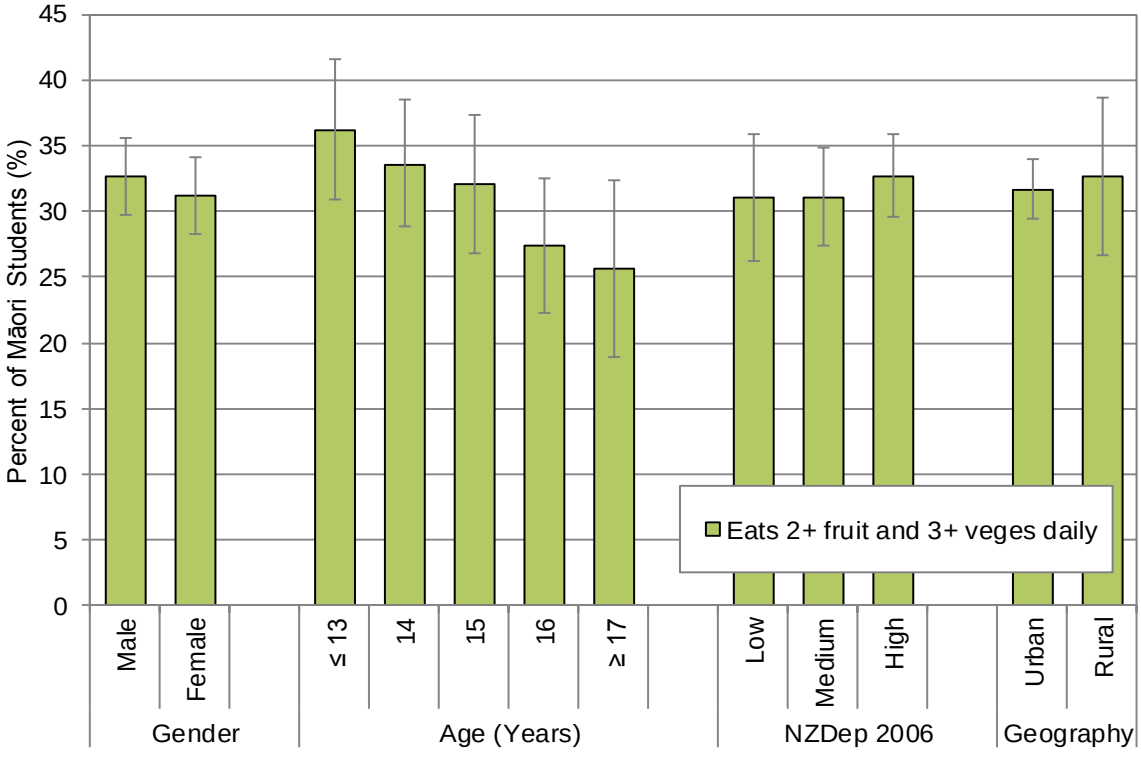
In the Youth'12 survey, 31.9% (95% CI 29.9–33.9) of Māori secondary school students said that they ate 2+fruit and 3+ vegetables per day. There were no *significant* gender or age differences in the proportion of Māori students who ate 2+ fruit and 3+ vegetables per day. Rates were also *not significantly* different between those living in the most and least deprived NZDep06 areas, or in urban and rural areas (**Figure 43**).

Figure 42. Breakfast and Family Meals for Māori 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; Analysis is for Māori students only

Figure 43. Fruit and Vegetable Consumption in Māori 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; Analysis is for Māori students only

PHYSICAL ACTIVITY

Introduction

The following section reviews a range of physical and sedentary activities in Māori children and young people using data from two sources: The Youth'12 Survey of secondary school students and the 2011/12 NZ Health Survey.

Background

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 [127]. Physical activity also plays an important role in preventing overweight and obesity, with a decline in physical activity in recent decades being thought to have contributed to rising obesity levels [128].

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 [128-130]. Physical activity also appears to decline as children transition through adolescence [131].

In New Zealand, the available evidence presents a mixed picture for the engagement of Māori children in physical activity, with the 2011/12 NZ Health Survey finding that 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 (e.g. walk, bike). However, in the same survey, Māori children were *significantly* more likely (aRR 1.24 95% CI 1.15–1.34) to watch two or more hours of television per day [81].

Youth'12 Survey

Youth'12 was the third national survey of Year 9–15 students (Year 14 and 15 are those students who are repeating Year 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 [101]. The analysis that follows is based on the 1,701 students who identified as being of Māori ethnicity in the Youth'12 Survey [100].

Data Sources and Methods

Definitions

1. Proportion of Māori secondary school students 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 Māori secondary school students 13–17+ years who did 60+ minutes physical activity daily

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* [101], and *The Health and Wellbeing of Māori New Zealand Secondary School Students in 2012* [100], a report that focused solely on those 1,701 students who identified as being of Māori ethnicity.

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) [101].

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. 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 also included the option "I can't choose only one ethnic group". For the purposes of comparing ethnic groups, Statistics NZ's ethnicity prioritisation methods were used [126], which reported five ethnic groups: Māori, Pacific, Asian, European and Other.

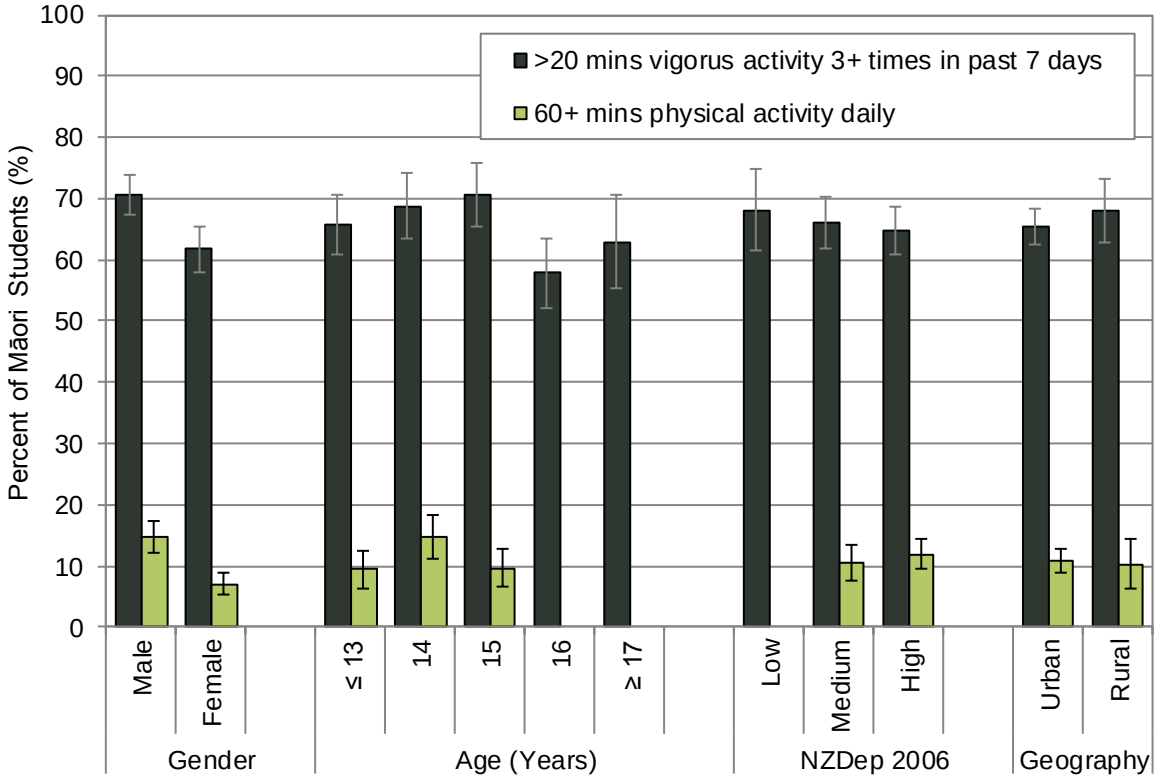
Students Participation in Physical Activity

In the Youth'12 survey, while 65.9% (95% CI 63.1–68.7) of Māori students had participated in more than 20 minutes vigorous physical activity on three or more occasions in the past seven days, only 10.7% (95% CI 8.9–12.6) reported achieving the recommended 60+ minutes of physical activity daily.

The proportion of Māori males (70.6% 95% CI 67.4–73.9) undertaking more than 20 minutes vigorous physical activity on three or more occasions in the past seven days was *significantly* higher than for Māori females (61.7% 95% CI 58.0–65.4), although there were no consistent differences by (single year of) age, NZDep2006 decile or geography (rural vs. urban) (**Figure 44**).

The proportion of Māori males (14.7% 95% CI 12.0–17.5) achieving the recommended 60+ minutes of physical activity a day was also *significantly* higher than for Māori females (7.1% 95% CI 5.3–8.9). Small numbers however precluded a valid analysis by (single year of) age and NZDep2006 decile No *significant* differences were evident by geography (rural vs. urban) (**Figure 44**).

Figure 44. Proportion of Māori 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



Source: Youth'12 Survey; Note: NZDep2006: Low = Deciles 1–3; Medium = Deciles 4–7; High = Deciles 8–10; Analysis is for Māori students only.

The 2011/12 NZ Health Survey

In the 2011/12 NZ Health Survey [81], 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 Māori children travelling to school by active means between the 2006/07 and 2011/12 NZ Health Surveys, as well as the proportion of Māori 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 [81] 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 that 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) [81].

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 [81].

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 [81]. 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 [98].

Travel to School by Active Means

Trends in Proportion of Children Travelling to School by Active Means

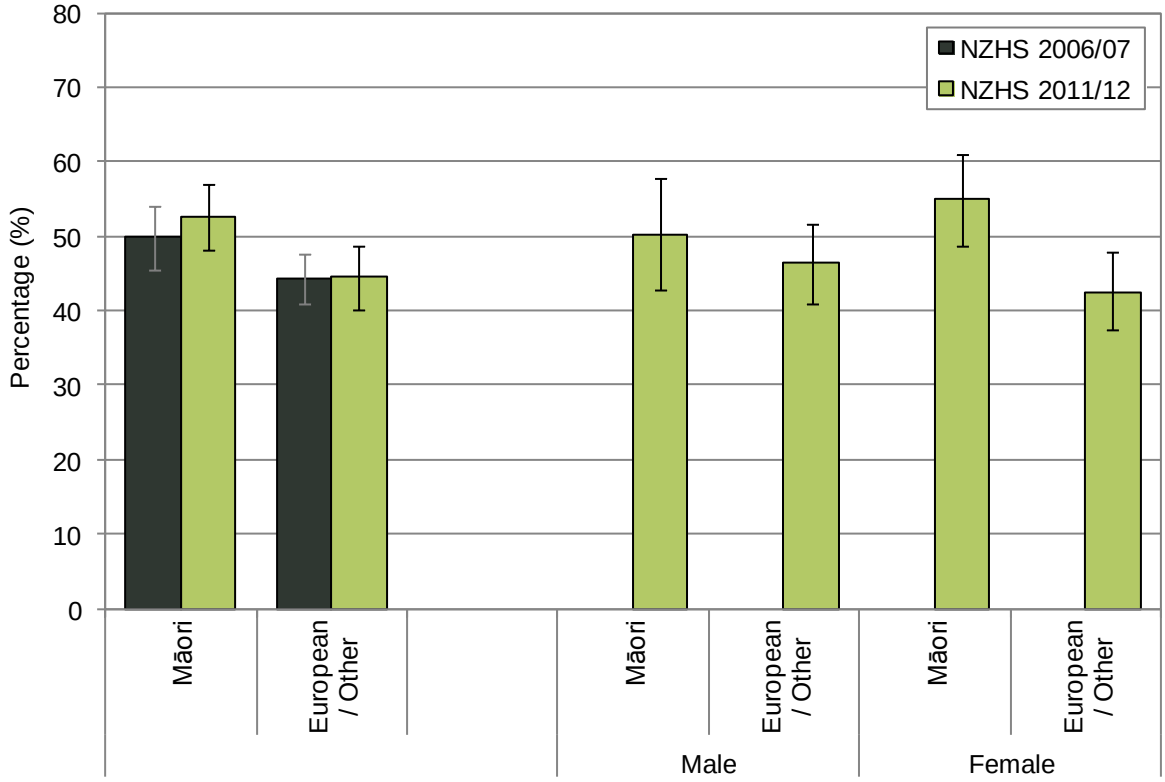
There were no *significant* changes in the proportion of Māori children who usually travelled to school by active means, between the 2006/07 and 2011/12 Surveys. In the 2011/12 NZHS, 52.6% (95% CI 48.1–57.0) of Māori children usually travelled to school by active means (**Figure 45**).

Current Distribution of Children Travelling to School by Active Means

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.

There were however, no *significant* gender differences in the proportion of Māori children who travelled to school by active means in the 2011/12 NZ Health Survey (**Figure 45**).

Figure 45. 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



Source: 2011/12 New Zealand Health Survey

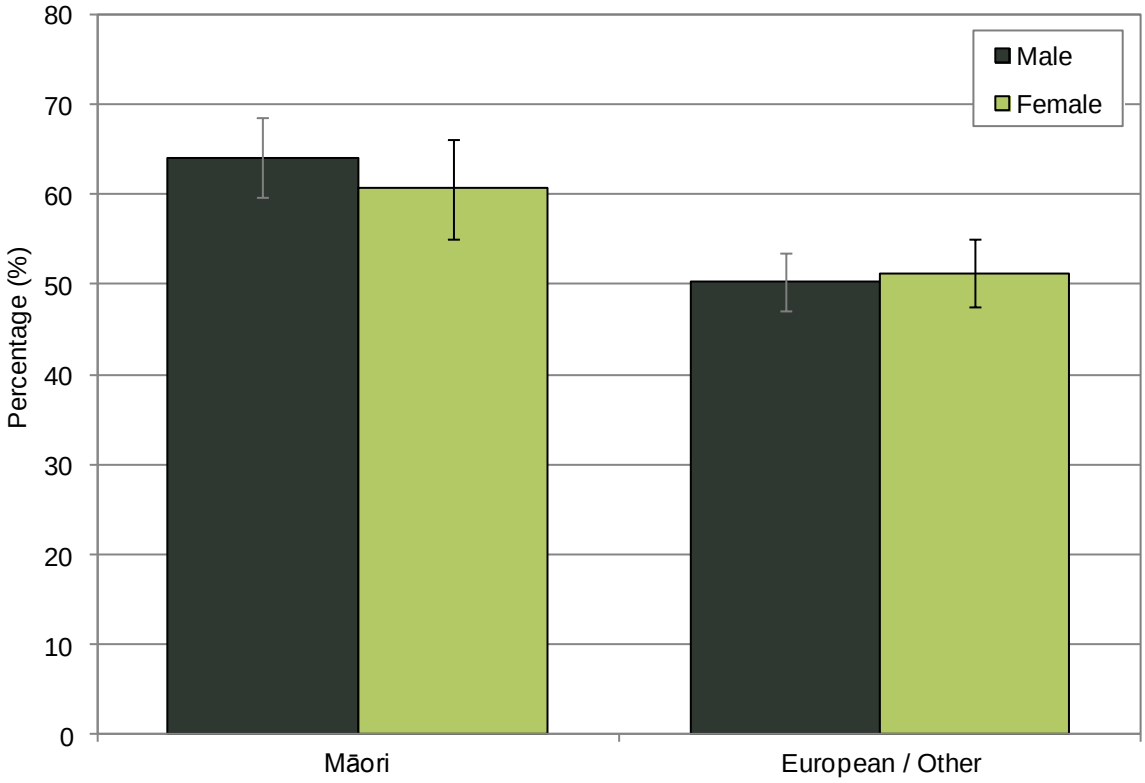
Television Watching

Current Distribution of Children Watching 2+ Hours of Television per Day

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.

There were however, no *significant* gender differences in the proportion of Māori children who usually watched two or more hours of television per day in the 2011/12 NZ Health Survey (**Figure 46**).

Figure 46. Proportion of Children Aged 2–14 Years Who Usually Watch 2+ Hours of Television per Day by Gender and Ethnicity, 2011/12 New Zealand Health Survey



Source: 2011/12 New Zealand Health Survey

APPENDICES AND REFERENCES

APPENDIX 1: 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%) [132].

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) [132].

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 [133] 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 [133].

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 2: 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 [5]. 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 that 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 5**).

The following sections discuss the first two of these issues, while the third is discussed in **Appendix 5**, 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 [134].

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 [134]. NZHIS provided feedback that 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 that 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 [135].

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 [135]. 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 [5].

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 [136]. 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 [137]. 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 5** 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 3: 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 [138].

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 ethnicity 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 5** 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 4: NATIONAL MORTALITY COLLECTION

Mode of Data Collection

The National Mortality Collection is a dataset managed by the Ministry of Health that 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 [139].

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 [140].

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, LSTA, Police, Water Safety NZ and ESR [140]. 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 5** of this report.

APPENDIX 5: 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 [141]. 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 [142].

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 [141]. 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 [142].

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 [142].

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

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 [141]. 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 [142]. 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 [142].

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 [141]. 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 that 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* [143] 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 [144]. 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 [143].

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* [145], 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 [146]. In 2009 Statistics NZ amended the Standard to reflect these issues [147] 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 [147].

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 [5]. In this Classification System increasing detail is provided at each level. For example [143]:

- 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 [143]. This ensures that each individual is counted only once and that the sum of the ethnic group sub-populations equals the total NZ population [142]. 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 [143]. 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 [143].

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 [6] 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 that 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 [148].

2. Research also suggests that ethnic misclassification may vary significantly by DHB [148], 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 6: 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 that may be independent of their own social position within a community [149]. 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 49**). 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 approximately 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 [150].

Table 49. Variables used in the NZDep2006 Index of Deprivation [151]

No	Factor	Variables in Order of Decreasing Weight in the Index
1	Income	People aged 18–64 receiving means tested benefit
2	Employment	People aged 18–64 unemployed
3	Income	People living in households with income below an income threshold
4	Communication	People with no access to a telephone
5	Transport	People with no access to a car
6	Support	People aged <65 living in a single parent family
7	Qualifications	People aged 18–64 without any qualifications
8	Owned Home	People not living in own home
9	Living Space	People living in households below a bedroom occupancy threshold

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 [149]. Despite these limitations, the NZDep has been shown to be predictive of mortality and morbidity from a number of diseases in New Zealand.

APPENDIX 7: CONGENITAL ANOMALY CODES

Table 50. ICD-10-AM Congenital Anomaly Coding Used in this Report (Table 1 of 2)

Q00–Q07 Malformations of the Nervous System	
Q00	Anencephaly
Q01	Encephalocele
Q02	Microcephaly
Q03	Congenital Hydrocephalus
Q04	Other Brain Malformations
Q05	Spina Bifida
Q06	Other Spinal Cord Malformations
Q07	Other CNS Malformations
Q10–Q18 Malformations of Eye, Ear, Face and Neck	
Q10–Q15	Eyelid/Lacrimal/Eye/Orbit Malformations
Q16	Ear Malformations Impairing Hearing
Q170	Accessory Auricle
Q171–Q175,Q178–Q179	Other Ear Malformations
Q18	Other Face/Neck Malformations
Q20–28 Malformations of the Circulatory System	
Q20	Malformations Cardiac Chambers/Connections
Q210	Ventricular Septal Defect
Q211	Atrial Septal Defect
Q212	Atrioventricular Septal Defect
Q213	Tetralogy of Fallot
Q214, Q218–Q219	Other Cardiac Septal Malformations
Q22	Pulmonary/Tricuspid Valve Malformations
Q23	Aortic/Mitral Valve Malformations
Q24	Other Heart Malformations
Q250	Patent Ductus Arteriosus
Q251–Q259	Malformations Great Arteries Excluding PDA
Q26	Malformations Great Veins
Q27	Other Peripheral Vascular Malformations
Q28	Other Circulatory Malformations
Q30–34 Malformations of the Respiratory System	
Q30	Nose Malformations
Q31	Larynx Malformations
Q32	Trachea/Bronchus Malformations
Q33	Lung Malformations
Q34	Other Respiratory Malformations
Q35–37 Cleft Lip and Cleft Palate	
Q35	Cleft Palate
Q36	Cleft Lip
Q37	Cleft Palate and Lip

Table 51. ICD-10-AM Congenital Anomaly Coding Used in this Report (Table 2 of 2)

Q38–45 Other Congenital Malformations of the Digestive System	
Q381	Ankyloglossia Tongue Tie
Q380,Q382–Q388	Tongue/Mouth/Pharynx Malformations
Q39–Q40	Oesophagus/Upper Alimentary Malformations
Q41–Q43	Intestinal Malformations
Q44–Q45	Other Digestive Malformations
Q50–56 Malformations of Genital Organs	
Q50–Q52	Female Genital Malformations
Q53	Undescended Testicle
Q54	Hypospadias
Q55	Other Male Genital Malformations
Q56	Indeterminate Sex/Pseudohermaphrodism
Q60–64 Malformations of Urinary System	
Q60	Renal Agenesis/Reduction Defects
Q61	Cystic Kidney Disease
Q62	Renal Pelvis Obstruction/Ureter Malformations
Q63–Q64	Other Kidney/Urinary Malformations
Q65–79 Malformations of Musculoskeletal System	
Q650–Q652	Congenital Dislocation Hip
Q653–Q655	Congenital Subluxation Hip
Q656,Q658–Q659	Other Deformities Hip
Q66	Foot Deformities
Q67–Q68, Q79	Other Musculoskeletal Malformations
Q69	Polydactyly
Q70	Syndactyly
Q71–Q74	Reduction Defects/Other Limb Malformations
Q75–Q76	Skull/Facial Bones/Spine/Thorax Malformations
Q77–Q78	Osteochondrodysplasia
Q80–89 Other Congenital Malformations	
Q80	Ichthyosis
Q81	Epidermolysis Bullosa
Q825	Non-Neoplastic Naevus
Q820–Q824, Q828–Q829	Other Skin Malformations
Q83	Breast Malformations
Q84	Other Integument Malformations
Q85–Q87, Q89	Other Malformations
Q90–99 Chromosomal Abnormalities	
Q90	Down Syndrome
Q91	Edwards and Patau Syndromes
Q92	Other Autosomal Trisomies
Q93,Q95	Monosomies and Autosomal Deletions/Other Rearrangements
Q96	Turners Syndrome
Q97	Other Sex Chromosome Anomalies Female Phenotype
Q98	Sex Chromosome Anomalies Male Phenotype
Q99	Other Chromosome Anomalies

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