THE HEALTH OF
CHILDREN AND YOUNG PEOPLE

WITH CHRONIC CONDITIONS AND DISABLITIES

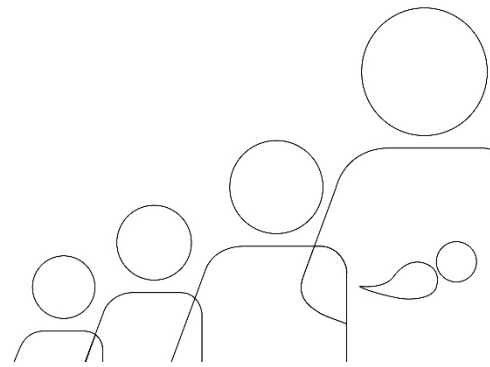## IN NEW ZEALAND 2016

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The Health of Children and Young People with Chronic Conditions and Disabilities in New Zealand 2016

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Cover Artwork: New Zealand Sea Lions - by Karen Davis

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INTRODUCTION

Reporting cycle

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

Year 1 (2014): The determinants of health for children and young people

Year 2 (2015): The health status of children and young people

Year 3 (2016): The health of children and young people with chronic conditions and disabilities

Chronic conditions and disabilities

This report aims to assist district health boards to plan to meet current and future demands in order to improve the quality of life for children with disabilities and chronic conditions by providing:

1. Information from a range of routinely collected data on children and young people's disability and chronic conditions, including prevalence of conditions arising in the perinatal period

2. Information about children's and young people's use of secondary health services

3. Evidence for good practice derived from current policies, guidelines and evidence-based interventions for each of the indicators presented

The choice of indicators included in this report was informed by an indicator framework developed by the NZ Child and Youth Epidemiology Service and by recent peer-reviewed literature about chronic conditions in children and young people.[1-3] Chronic conditions and disabilities often affect people for life. Having a good quality of life and flourishing to your best ability is dependent, at least in part, on what happened as you were growing up. Understanding the dimensions of chronic conditions and disabilities among children and young people is essential to planning and developing good quality health services for New Zealand's children and young people.

About 11% of children aged 0–14 years have a disability according to the 2013 New Zealand Disability Survey.[4] Of these children, the survey identified that 49% of children with impairments that limited daily activity were affected by conditions that existed at birth, while for a third of the children, the reason was one of a range of 'other causes', not usually diagnosed at birth, which include autism spectrum disorder, attention deficit hyperactivity disorder, developmental delay, dyslexia and dyspraxia. Learning difficulty was the most common impairment with 6% of children surveyed, and 52% of disabled children having difficulty learning. Boys had a disability rate of 13% compared with the girls' rate of 8%, with boys having higher rates in psychiatric/psychological, speaking and learning impairments. Māori children had a disability rate of 15% compared with 9% for non-Māori children. Twenty five percent of disabled children had impairments that were caused by a disease or illness, and 3% by an injury.[4]

The New Zealand Health Survey (NZHS) also indicates chronic conditions and disabilities that affect children and young people in New Zealand. The majority of 2–24 year olds have a BMI category of healthy weight, however, an estimated 11% prevalence of obesity was found among 2–14 year olds in the 2014/15 NZHS. This is of concern because there are implications for future health, given the association with type 2 diabetes and the risk of high blood pressure, coronary heart disease and stroke later in life.[5] The unadjusted prevalence rate of childhood obesity in the 2014/15 NZHS was significantly higher than that in the 2006/07 survey. The rate for type 2 diabetes among 15–24 year olds is less than 1% and has been much the same in recent years.

The prevalence of children being diagnosed with autism spectrum disorder varies with age, with the 2014/15 NZHS rate being about 0.5% among those aged 2–4 years and 1.5% among the 10–14 year olds. Rates have increased over the years, reflecting similar trends overseas not fully understood but possibly influenced by increased recognition of the condition.

The NZHS 2014/15 indicated that prevalence rates for eczema have been increasing, especially among those aged 0–4 years. Hospitalisations of 0–24 year olds has risen from below 100 to over 200 per 100,000 population for eczema and dermatitis as the primary diagnosis, while the rates rose from about 540 to 790 per 100,000 population for cases where eczema and dermatitis were reported but were not the primary reason for the hospitalisation. These are often for the 0–4 year olds. The presence of such conditions for children and young people in hospital has implications for health services.

New indicators and new thinking have been taken into consideration in presenting data in this report. While obesity has traditionally been viewed as separate from eating disorders, increasingly they are being considered as parts of a continuum with common risk factors and scope for integrated prevention. Musculoskeletal conditions have been included reflecting an increasing rate of hospitalisations for the suite of conditions that includes juvenile arthritis, juvenile osteochondrosis, and scoliosis.

A major limitation of this report is that it cannot address questions that require outpatient data. These data are not available at a national level. While the inclusion of the New Zealand Health Survey data provides a little more detail about estimated population prevalence for a few indicators, the full picture of outpatient and primary health care data is required for planning nationally and by district health boards.

A further limitation in this report is the lack of data on the transition of children and young people into adult services, particularly in health, a process that those with chronic conditions and disabilities almost inevitably face. A transition into adults able to reach their potential by achieving in education, gaining meaningful employment, and participating in their community is an aim that many children and young people with chronic conditions and disabilities aspire to.

Review topics

Two issues were selected by participating DHBs for review and inclusion in this report: Fetal alcohol spectrum disorder (FASD) by Judith Adams, and The health needs of children and young people in State care by Mavis Duncanson.

Fetal alcohol spectrum disorder (FASD) contributes to many poor outcomes for New Zealand's young people including early mortality, abuse and neglect, poor educational achievement, engagement with the criminal justice system, benefit dependence, and mental health and alcohol and drug problems. This chapter provides information on the features of FASD, the life course consequences of FASD and the comorbidities of FASD. It discusses international studies on the epidemiology of FASD. Diagnosis of FASD is not straightforward so methods of diagnosis are reviewed, along with the findings from a review of the Hawke's Bay DHB's FASD assessment pathway. Subsequent sections look at the evidence base for interventions to address the difficulties faced by people with FASD and their families. The final sections reviews what is known about New Zealand women's drinking habits, the patterns of drinking that are associated with the greatest risk of FASD and the evidence regarding the effectiveness of interventions to prevent the harm resulting from prenatal exposure to alcohol.

In New Zealand there have consistently been around 5,000 children and young people in the custody of the Chief Executive of Child, Youth and Family (in CYF care) each year. Many children and young people referred to Child, Youth and Family have high levels of long-term need and disadvantage. Health and educational assessments (Gateway Assessments) of children and young people in CYF care have identified multiple health needs for most children. Internationally it is well recognised that children and young people in State care often present with complex and serious health problems. This chapter uses published reports to describe the group of children in CYF care in New Zealand and to outline key characteristics of the current and proposed new care and protection systems, particularly those relevant to health services. The chapter also provides a rapid review of New Zealand and international literature regarding the health needs of children and young people in State care and guidelines for best practice in providing care to them.

Appendices

The content of the appendices may be helpful when interpreting information in this report. **Appendix** 1 provides an overview of the methods used to develop the reviews of evidence for good practice, which appear at the conclusion of most indicator chapters.

Appendix 2 describes the statistical methods used, including a description of rates calculated within the data. **Appendix 3** contains information on the data sources used to develop each indicator and discusses data limitations. **Appendix 4** deals with the measurement of ethnicity and also provides an overview of the NZ Deprivation Index; NZDep 2013 is used where data relate to the 2013 Census. **Appendix 5** displays the tables of clinical codes referred to in this report.

Conclusion

This report reviews the prevalence of a range of disabilities and chronic conditions experienced by children and young people living in New Zealand. These conditions place demands on health and disability support services. This report provides information on the secondary health service utilisation patterns of children and young people with chronic conditions and disabilities. It is unable to provide data on all health service use as these data are not collated nationally. It does, however, aim to provide some insights into two quite different perspectives of disability and chronic conditions: the consequences and management of children with fetal alcohol syndrome, and a review of the health needs of children in care.

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

Increasingly high quality evidence is becoming available to direct future initiatives for a number of the conditions included here. The number of systematic reviews has risen exponentially in many of these fields. Some of these will inform the development of integrated services to benefit the children and young people with chronic conditions and disabilities, and those who care for them.

Table 1 presents an overview of children and young people hospitalised in New Zealand with chronic conditions, babies born with congenital anomalies and babies who died as a result of congenital anomalies, and cancer notifications of children and young people.

Table 1. Summary of health service data for 0–24 year olds with chronic conditions

Indicator	New Zealand		
	Unique individuals*	Hospitalisations	
		Primary diagnosis	All cases
Cystic fibrosis	336	2,122	2,625
Diabetes	4,137	6,466	12,308
Eating disorders	1,012	1,509	2,301
Autism spectrum disorder	1,853	485	3,015
Cerebral palsy	1,678	2,458	5,852
Epilepsy	4,336	7,440	9,876
Chronic lower respiratory diseases	29,184	37,909	48,433
Inflammatory bowel disease	1,447	5,168	5,999
Constipation	14,578	7,805	18,723
Eczema and dermatitis	15,331	5,022	18,950
Musculoskeletal disorders	1,506	2,431	3,016

	n	NZ rate	95% CI
Babies with one or more congenital anomalies	21,219	67.56	66.66–68.48
Infant mortality with congenital anomalies	439	1.41	1.28–1.55
Cancer notifications 0–14 year olds	699	14.67	10.53–19.91
Cancer notifications 15–24 year olds	821	26.34	24.57–28.21

Unique individuals* were identified as being hospitalised at least once from 2011–2015; Hospitalisations are all relevant hospitalisations 2011–2015; Congenital anomaly data are 2009–2013; Congenital anomaly rates are per 1,000 total births; Cancer notifications are 2010–2014; Cancer notification rates are age standardised rates per 100,000 population

References

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CONGENITAL ANOMALIES

Introduction

Congenital anomalies, also known as birth defects, congenital disorders or congenital malformations, are structural or functional anomalies that exist at or before birth (although they may not be detected until later in life).[1] They are major causes of fetal, infant and child deaths, and chronic illness and disability.[1] Congenital anomalies may occur in isolation or they may occur together in a pattern corresponding to a named syndrome, such as Down syndrome or achondroplasia (dwarfism).[2]

Congenital anomalies vary in severity from the inevitably lethal, such as absent kidneys, to minor abnormalities of cosmetic significance only. Major anomalies, those anomalies that have medical and/or social implications and often require surgical repair, occur in approximately three to four percent of all live births.[3] Common major anomalies include heart defects, cleft lip and palate, neural tube defects, and chromosomal abnormalities (the most common of which is Down syndrome).[3]

In at least half of all occurrences of congenital anomaly, no cause can be identified.[1,4] Genetic factors are important in many congenital anomalies, particularly syndromic anomalies.[1] Genetic factors cause around one third of all congenital anomalies and about 85% of those with known causes.[5] Environmental factors of various kinds, including vitamin deficiencies, can cause congenital anomalies, for example: insufficient maternal intake of folic acid increases the risk of neutral tube defects; maternal infection with Zika virus seems to cause microcephaly; and poorly controlled pregestational maternal diabetes increases the risk of major anomalies in the cardiovascular and central nervous systems and in craniofacial structures.[1,6]

The following section uses data from the National Minimum Dataset to describe congenital anomalies in babies from 2000–2015 and concludes with a brief overview of some of the evidence relating to early diagnosis of these conditions.

Data sources and methods

Indicators

> Prevalence of congenital anomalies
> Infant mortality associated with congenital anomalies

Definition

Anomalies diagnosed in stillbirths and live born babies up to one year of age are included, and cases identified based on codes adapted from those used by BINOCAR and by registers in Australia[7]

Stillbirths are of at least 400g birthweight or 20 weeks gestation (as defined by the Ministry of Health)

Numerators: Live born infants hospitalised: National Minimum Dataset

Stillbirths: National Mortality Collection

Infant deaths associated with congenital anomalies: National Mortality Collection

Denominators: Total births
Live births: Birth Registration Dataset
Stillbirths: National Mortality Collection

Prevalence per 1,000 live births

Additional information

Anomalies are year of birth; births are registration year.
Codes used for identifying cases are documented in **Appendix 5**
Maternal age was only available for 72% of cases.
Prevalence rates presented for sex-specific anomalies are sex-specific rates.
The confidence intervals are calculated using the Poisson distribution.

National trends and distribution

The number of babies with at least one congenital anomaly (including minor defects) gradually increased from 2000 to 2007. There was a noticeable dip between 2008 and 2011, which corresponded with a decrease in diagnoses of minor skin anomalies, such as tags and birthmarks. From 2012 onwards, numbers have been similar to those in 2007 **(Figure 1)**. The proportion of babies born with one or more congenital anomalies has fluctuated slightly from year to year but, on average, has been around seven per cent **(Table 2)**.

Figure 1. Number of babies born and those with one or more congenital anomalies, by year, New Zealand 2000-2015
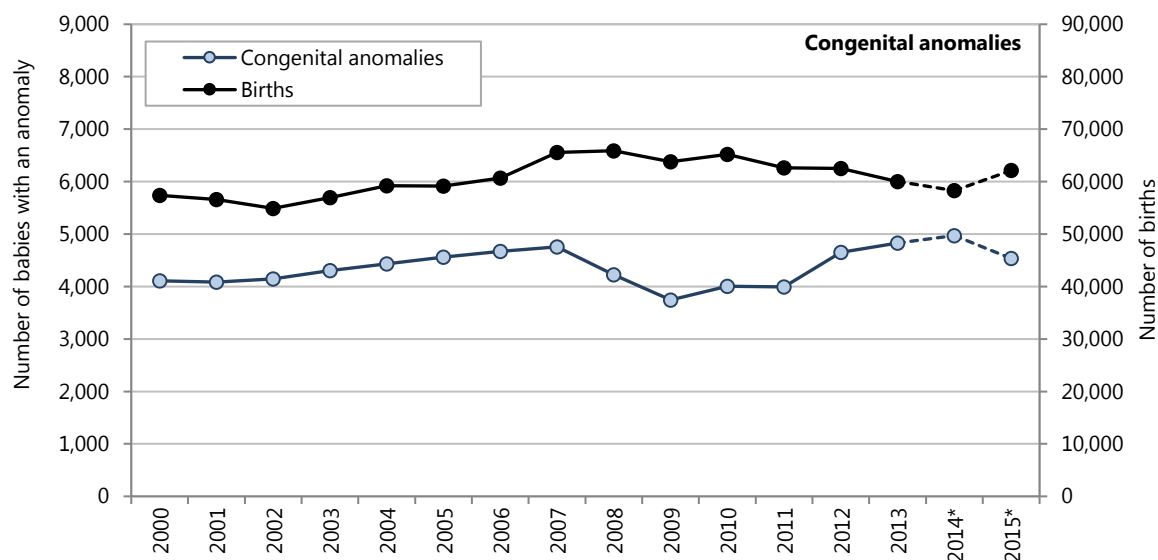
Source: National Minimum Dataset, National Mortality Collection, Birth registration dataset. (Total) births corresponds to live births and fetal deaths, * 2014 and 2015 are live births only

Table 2. Proportion of babies with one or more congenital anomalies, by year, New Zealand 2000–2015

Year	Babies with an anomaly (*n*)	Total births (*n*)	% of total births
New Zealand			
Congenital anomalies			
2000–2008	39,291	536,368	7.3
2009	3,744	63,767	5.9
2010	4,004	65,168	6.1
2011	3,991	62,624	6.4
2012	4,650	62,483	7.4
2013	4,830	60,026	8.0
2014*	4,971	58,285	8.5
2015*	4,535	62,122	7.3
Total	70,016	970,843	7.2

Source: National Minimum Dataset, National Mortality Collection, Birth registration dataset. Total births corresponds to live births and fetal deaths, * 2014 and 2015 are live births only

Diagnosis

Table 3 presents the number of cases and prevalence for each congenital anomaly subgroup. The group of anomalies with the highest prevalence between 2009 and 2013 was digestive system anomalies, followed by congenital heart defects, and then limb anomalies. Refer to **Appendix 5** for details on the specific congenital anomalies included in each category.

Table 3. Babies with one or more congenital anomalies, by anomaly subgroup, New Zealand 2009–2013

Babies with an anomaly	(n)	Rate per 1,000 births	95% CI
Congenital anomalies			
New Zealand			
All cases*	21,219	67.56	66.66–68.48
Digestive system	7,075	22.53	22.01–23.06
Congenital heart defects	3,902	12.42	12.04–12.82
Limb	3,536	11.26	10.89–11.64
Genital	2,140	6.81	6.53–7.11
Urinary	1,322	4.21	3.99–4.44
Respiratory	1,186	3.78	3.56–4.00
Nervous system	748	2.38	2.21–2.56
Chromosomal anomalies	701	2.23	2.07–2.40
Ear, Face and Neck	689	2.19	2.03–2.36
Orofacial clefts	524	1.67	1.53–1.82
Eye	233	0.74	0.65–0.84
Abdominal wall defects	228	0.73	0.63–0.83
Neural tube defects	150	0.48	0.40–0.56

Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Birth registration dataset and National Mortality Collection. Babies with at least one diagnosed anomaly. *Babies are counted once overall, once for each sub-group and for each anomaly documented

Demographic distribution

Table 4 presents the demographic distribution of congenital anomalies in New Zealand between 2009 and 2013. There were no significant differences by New Zealand Deprivation Index quintile. The prevalence of anomalies was significantly higher for male babies. Māori, Pacific, Asian/Indian and MELAA babies all had statistically significantly lower congenital anomaly prevalence rates than babies of European/Other ethnicities but only the Māori rate was markedly lower than the European/Other rate. Compared to babies born to mothers aged 30–34 years, babies born to mothers aged 20–29 years had slightly but significantly lower rates while babies born to mothers aged over 35 years had significantly higher rates. Over the period 2000–2015, rates for the four largest ethnic groups were reasonably steady although there was a dip in rates between 2006 and 2012 (**Figure 2**). Rates for Māori babies were consistently considerably lower than rates for other babies (**Figure 2**).

Table 4. Babies with one or more congenital anomalies, by demographic factor, New Zealand 2009–2013

Variable	2009–2013 (n)	Prevalence per 1,000 births	Rate ratio	95% CI
Congenital anomalies				
NZ Deprivation Index quintile				
Deciles 1–2	3,088	67.77	1.00	
Deciles 3–4	3,430	68.06	1.00	0.96–1.05
Deciles 5–6	3,731	64.79	0.96	0.91–1.00
Deciles 7–8	4,737	68.61	1.01	0.97–1.06
Deciles 9–10	6,168	68.35	1.01	0.97–1.05
Prioritised ethnicity				
Māori	4,353	47.76	0.61	0.59–0.63
Pacific	2,445	69.47	0.89	0.85–0.93
Asian/Indian	2,865	72.82	0.93	0.90–0.97
MELAA	343	64.29	0.82	0.74–0.91
European/Other	11,144	77.97	1.00	
Gender				
Female	8,442	55.20	1.00	
Male	12,773	79.28	1.44	1.40–1.47
Maternal age				
<20 years	996	48.55	1.00	0.93–1.06
20–24 years	2,526	43.52	0.89	0.85–0.94
25–29 years	3,554	45.02	0.92	0.88–0.96
30–34 years	4,291	48.79	1.00	
35+ years	3,860	56.26	1.15	1.11–1.20

Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Birth registration dataset and National Mortality Collection. Babies with at least one diagnosed anomaly, Rate ratios are unadjusted, Ethnicity is level 1 prioritised, Decile is NZDep2013, Maternal age reported where available

Figure 2. Babies with one or more congenital anomalies, by ethnicity, New Zealand 2000–2015
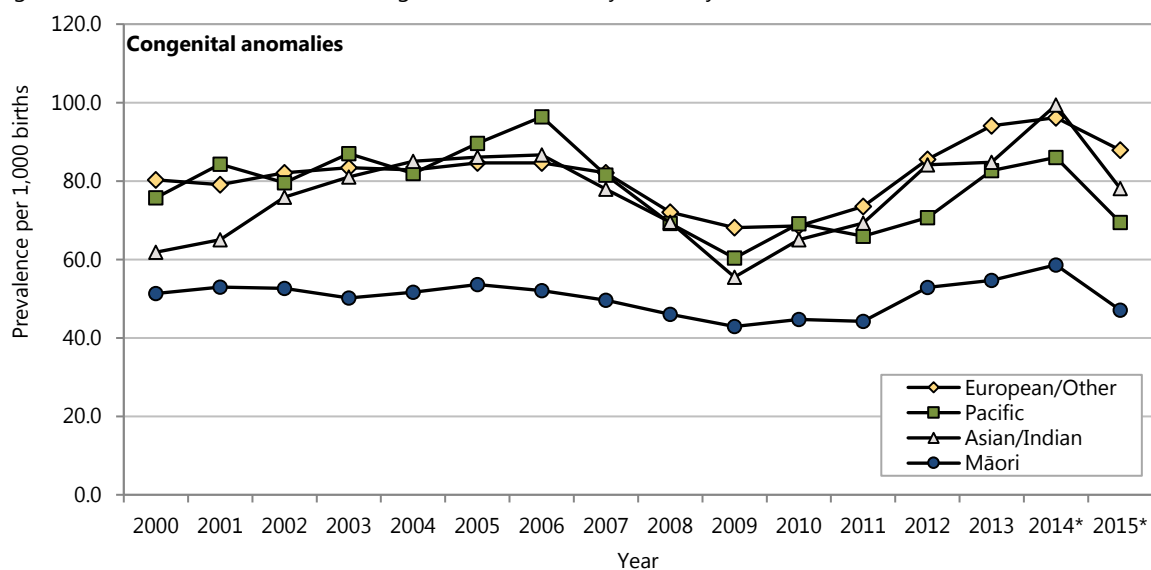
Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Birth registration dataset and National Mortality Collection. (Total) births corresponds to live births and fetal deaths, 2014 and 2015 are live births only. Ethnicity is level 1 prioritised

Maternal age

The lowest prevalence rate of congenital anomalies between 2009 and 2013 was for babies born to mothers aged 20–24 years at delivery. Prevalence was significantly lower for babies born to mothers aged 20–29 years and significantly higher for babies born to mothers 35 years and over (compared to mothers aged 30–34 years; **Table 4**, **Table 5**). The prevalence of chromosomal anomalies increased with increasing maternal age with the highest prevalence being for babies born to mothers aged 35 years and over. Prevalence of non-chromosomal anomalies was highest for babies born to mothers aged under 20 years and 35 years and over (U-shaped distribution across the maternal age groups). Most of the non-chromosomal anomaly subgroups had a U-shaped prevalence distribution except for abdominal defects, for which the prevalence was highest for the under 20 year maternal age group and decreased as maternal age increased (**Table 5**).

Table 5. Babies with one or more congenital anomalies, by maternal age and anomaly type, New Zealand 2009– 2013

2009–2013	Maternal age					
	Total	<20 years	20–24 years	25–29 years	30–34 years	35+ years
Congenital anomalies						
New Zealand						
Total births (*n*)	314,068	20,513	58,042	78,941	87,956	68,616
Babies with an anomaly (*n*)	21,219	996	2,526	3,554	4,291	3,860
Prevalence per 1,000 births (95% CI)						
All cases*	67.56 (66.66–68.48)	48.55 (45.59–51.67)	43.52 (41.84–45.25)	45.02 (43.55–46.53)	48.79 (47.34–50.27)	56.26 (54.49–58.06)
Non-chromosomal**	65.33 (64.44–66.23)	47.77 (44.83–50.86)	42.47 (40.81–44.18)	43.74 (42.29–45.23)	47.17 (45.75–48.63)	51.98 (50.29–53.72)
NS	2.23 (2.07–2.40)	2.73 (2.06–3.55)	1.91 (1.57–2.30)	1.24 (1.01–1.51)	1.49 (1.25–1.77)	1.92 (1.61–2.28)
CHD	11.45 (11.08–11.83)	10.38 (9.04–11.88)	7.87 (7.17–8.63)	7.64 (7.04–8.27)	8.07 (7.49–8.69)	9.71 (8.98–10.47)
Respiratory	3.56 (3.36–3.78)	1.71 (1.19–2.37)	1.19 (0.92–1.50)	1.33 (1.09–1.61)	1.36 (1.13–1.63)	1.85 (1.54–2.20)
OFC	1.58 (1.44–1.72)	1.85 (1.31–2.54)	1.12 (0.86–1.43)	1.05 (0.84–1.30)	1.08 (0.87–1.32)	1.50 (1.23–1.82)
Digestive	22.27 (21.75–22.79)	13.50 (11.96–15.19)	13.11 (12.20–14.08)	15.48 (14.62–16.37)	18.67 (17.78–19.59)	19.03 (18.02–20.09)
Abdominal	0.71 (0.62–0.81)	3.02 (2.32–3.87)	1.22 (0.96–1.54)	0.53 (0.38–0.72)	0.30 (0.19–0.43)	0.16 (0.08–0.29)
Urinary	4.13 (3.90–4.36)	2.83 (2.15–3.66)	2.52 (2.12–2.96)	2.85 (2.49–3.25)	3.39 (3.01–3.80)	3.86 (3.41–4.36)
Genital	6.66 (6.38–6.95)	5.26 (4.32–6.36)	4.67 (4.13–5.26)	4.24 (3.80–4.72)	4.63 (4.19–5.10)	4.74 (4.24–5.28)
Limb	11.10 (10.73–11.47)	8.48 (7.27–9.84)	8.82 (8.07–9.62)	8.36 (7.73–9.02)	7.84 (7.27–8.45)	8.80 (8.11–9.53)
Chromosomal	2.23 (2.07–2.40)	0.78 (0.45–1.27)	1.05 (0.80–1.35)	1.28 (1.04–1.55)	1.61 (1.36–1.90)	4.27 (3.80–4.79)

Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Birth registration dataset and National Mortality Collection. * Babies are counted once overall, some babies will have multiple anomalies and appear in more than one row, Maternal age reported where available. ** Non-chromosomal anomalies exclude cases with chromosomal anomalies present, NS = Nervous system, CHD = Congenital heart defects, OFC = Oro-facial clefts, Abdominal = Abdominal wall defects

The overall prevalence of babies with one or more congenital anomalies was significantly higher than the national rate between 2009 and 2013 in the four Northern DHBs, and in Hutt Valley and Capital & Coast DHBs. The prevalence was significantly lower than the national rate in Waikato, Bay of Plenty, Lakes, Taranaki, Hawke's Bay, MidCentral, Whanganui and Nelson Marlborough DHBs. There was no significant difference from the national rate in the remaining district health boards (**Figure 3**, **Table 6**).

Table 6. Babies with one or more congenital anomalies, by district health board, New Zealand 2009–2013

Babies with an anomaly	2009–2013 (n)	Rate per 1,000 births	Rate ratio	95% CI
Congenital anomalies				
Northland	847	72.90	1.08	1.01–1.15
Waitemata	3,321	83.90	1.24	1.20–1.29
Auckland DHB	2,394	72.59	1.07	1.03–1.12
Counties Manukau	3,175	73.01	1.08	1.04–1.12
Waikato	1,563	56.61	0.84	0.80–0.88
Bay of Plenty	898	61.03	0.90	0.85–0.96
Lakes DHB	465	58.78	0.87	0.80–0.95
Tairawhiti	226	60.07	0.89	0.78–1.01
Taranaki	312	39.49	0.58	0.52–0.65
Hawke's Bay	534	46.23	0.68	0.63–0.74
MidCentral	544	47.91	0.71	0.65–0.77
Whanganui	257	58.21	0.86	0.76–0.97
Hutt Valley	870	83.47	1.24	1.16–1.32
Capital & Coast	1,498	77.03	1.14	1.08–1.20
Wairarapa	183	68.49	1.01	0.88–1.17
Nelson Marlborough	450	55.07	0.82	0.74–0.89
South Canterbury	215	68.45	1.01	0.89–1.15
Canterbury	2,035	64.49	0.95	0.91–1.00
West Coast	134	63.12	0.93	0.79–1.10
Southern DHB	1,242	67.41	1.00	0.94–1.05
New Zealand	21,219	67.56	1.00	

Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Birth registration dataset and National Mortality Collection. Prevalence per 1,000 births. (Total) births corresponds to live births and fetal death. Babies with one or more diagnosed anomalies

Figure 3. Babies with one or more congenital anomalies, by district health board, New Zealand, 2009–2013
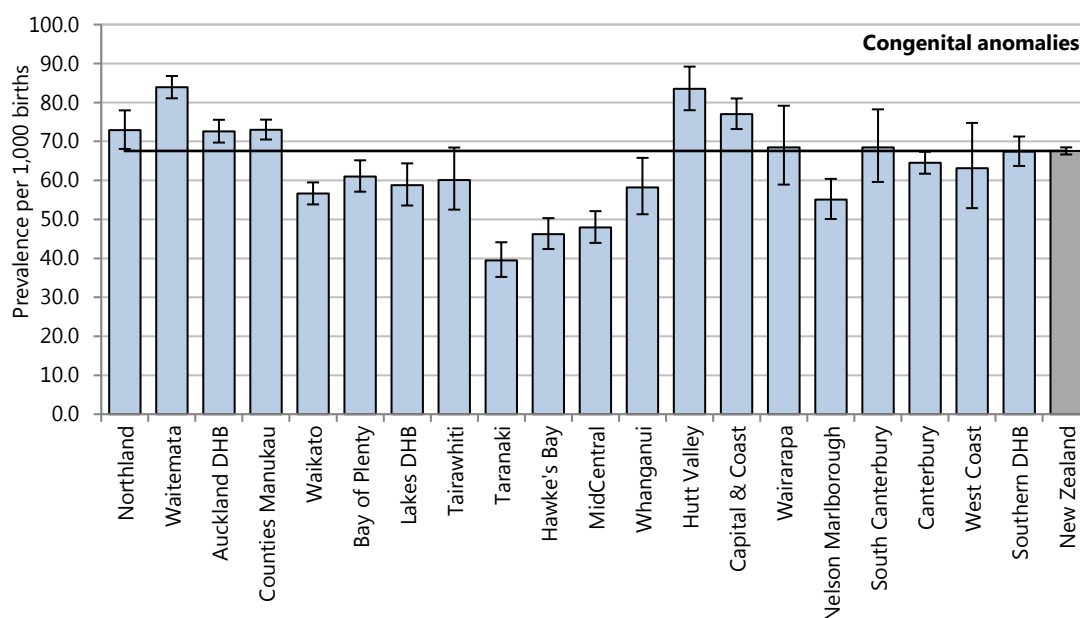
Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Birth registration dataset and National Mortality Collection. (Total) births corresponds to live births and fetal death. Babies with one or more diagnosed anomalies

Congenital anomaly-related infant mortality

The infant mortality rate in New Zealand between 2009 and 2013 was 5.14 per 1,000 live births. Over 27% of the infants who died had at least one congenital anomaly (**Table 7**). The infant mortality rate associated with congenital anomalies gradually decreased from 1990–91 to 2006–07 and from then on increased slightly from year to year (**Figure 4**). Congenital heart defects, nervous system defects, and chromosomal anomalies were the predominant congenital anomaly subgroups contributing to infant mortality (**Table 7**).

Table 7. Infant mortality with one or more congenital anomalies, by anomaly subgroup, New Zealand 2009–2013

2009–2013	*n*	Rate per 1,000 live births	95% CI
Congenital anomaly infant mortality			
New Zealand			
Infant mortality	1,603	5.14	4.89–5.40
Infant mortality with a congenital anomaly*	439	1.41	1.28–1.55
Congenital heart defects	181	0.58	0.50–0.67
Nervous system	82	0.26	0.21–0.33
Chromosomal anomalies	79	0.25	0.20–0.32
Digestive system	64	0.21	0.16–0.26
Urinary	54	0.17	0.13–0.23
Respiratory	48	0.15	0.11–0.20
Neural tube defects	33	0.11	0.07–0.15
Limb	20	0.06	0.04–0.10
Abdominal wall defects	11	0.04	0.02–0.06
Eye	<10	s	s
Orofacial clefts	<10	s	s
Genital	<10	s	s
Ear, Face and Neck	0	..	..

Numerator: National Mortality Collection, Denominator: Birth registration data. Congenital anomaly infant mortality per 1,000 live births. *Infant mortality with one or more anomalies. One infant excluded for presence of single anomaly of minor severity; Some Infant mortality will have multiple anomalies and appear in more than one subgroup

Figure 4. Infant mortality with one or more congenital anomalies, by year, New Zealand 1990–2013
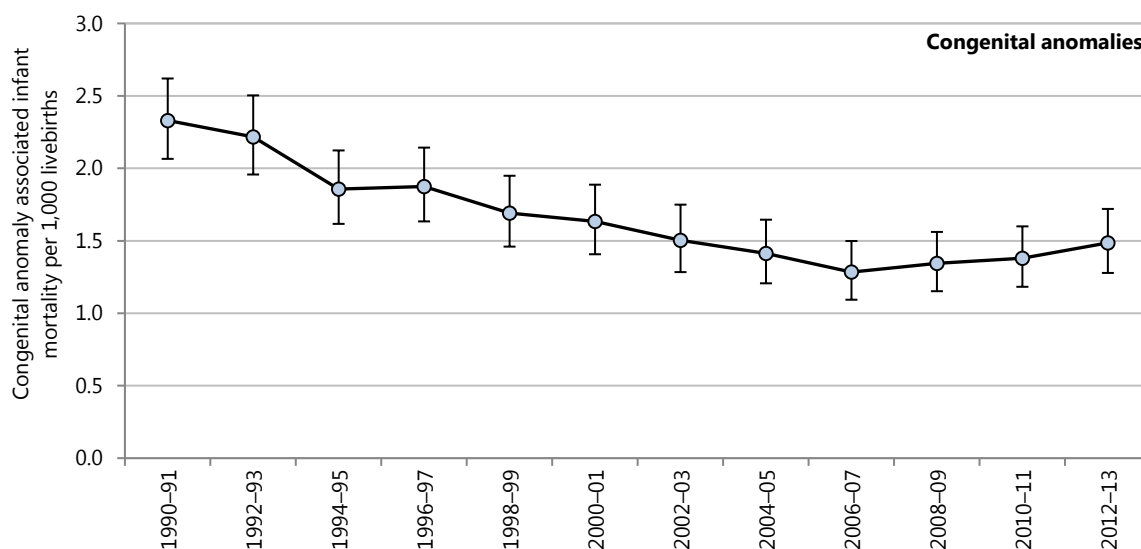
Numerator: National Mortality Collection, Denominator: Birth registration dataset. Babies with one or more diagnosed congenital anomalies

The congenital anomaly infant mortality rate was significantly higher than the national rate between 2009 and 2013 in Counties Manukau DHB, and was significantly lower in Waitemata and Canterbury DHBs. There was no significant difference from the national rate in the remaining district health boards (**Figure 5, Table 8**).

Figure 5. Infant mortality with one or more congenital anomalies, by district health board, New Zealand, 2009– 2013
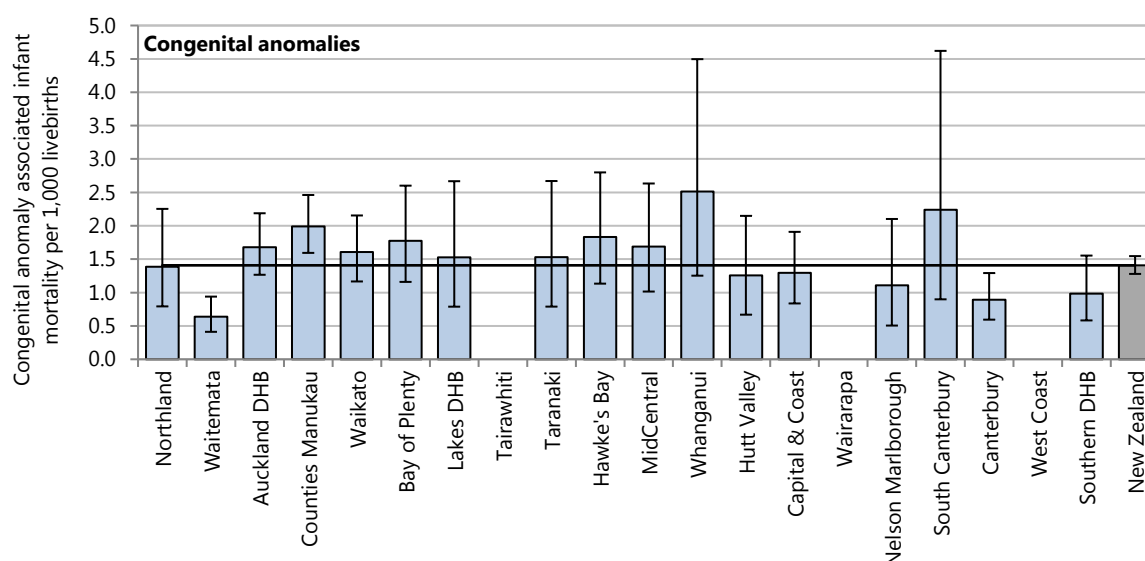
Numerator: National Mortality Collection, Denominator: Birth registration dataset. Infant mortality with one or more diagnosed congenital anomalies; Congenital anomaly infant mortality per 1,000 live births

Table 8. Infant mortality with one or more congenital anomalies, by district health board, New Zealand 2009–2013

DHB	Infant mortality with an anomaly (n)	Rate*	Rate ratio	95% CI	% of infant deaths
Congenital anomaly associated infant mortality					
Northland	16	1.39	0.99	0.60–1.62	19.8
Waitemata	25	0.64	0.45	0.30–0.68	21.4
Auckland	55	1.68	1.19	0.90–1.58	38.5
Counties Manukau	86	1.99	1.42	1.12–1.78	27.4
Waikato	44	1.60	1.14	0.84–1.55	25.7
Bay of Plenty	26	1.78	1.26	0.85–1.87	29.2
Lakes	12	1.53	1.08	0.61–1.92	25.0
Tairawhiti	<5	s	s	s	s
Taranaki	12	1.53	1.09	0.61–1.93	29.3
Hawke's Bay	21	1.83	1.30	0.84–2.02	30.4
MidCentral	19	1.69	1.20	0.76–1.90	33.9
Whanganui	11	2.51	1.79	0.98–3.24	33.3
Hutt Valley	13	1.26	0.89	0.51–1.55	24.5
Capital & Coast	25	1.29	0.92	0.61–1.37	34.2
Wairarapa	<5	s	s	s	s
Nelson Marlborough	9	1.11	0.79	0.41–1.52	32.1
South Canterbury	7	2.24	1.59	0.76–3.36	31.8
Canterbury	28	0.89	0.63	0.43–0.93	19.9
West Coast	<5	s	s	s	s
Southern	18	0.98	0.70	0.44–1.12	25.4
New Zealand	439	1.41	1.00		27.4

Numerator: National Mortality Collection, Denominator: Birth registration dataset. Infant mortality with one or more diagnosed anomalies; * Congenital anomaly infant mortality per 1,000 live births

Evidence for good practice

Possibilities for prevention

The majority of congenital malformation have no known cause. For this reason, the avenues for prevention are limited and the effects of interventions modest. Some major structural malformations can be detected during pregnancy via ultrasound examination and most chromosomal and some genetic disorders can be detected via amniocentesis, chorionic villus sampling or non-invasive prenatal testing. Prenatal diagnosis allows parents to choose termination when their fetus has a condition likely to be fatal or severely disabling, or delivery in a tertiary centre with appropriate surgical expertise when their baby will require surgery soon after birth.

Table 9 indicates where prenatal detection is possible (to a variable degree), by ultrasound examination and/or genetic testing, where optimal health status before and during pregnancy and good antenatal care may reduce the incidence of the condition, and where early postnatal detection improves outcomes. For genetic conditions (including cystic fibrosis), pre-natal genetic testing is generally offered only in cases where there is a family history of the condition or where a genetic condition is suspected, for example, as a result of findings from maternal blood tests and/or prenatal ultrasound examination. The prenatal detection rate varies from condition to condition. The overall detection rate for structural anomalies via ultrasound in the first trimester is around 50%,[8] and for lethal anomalies the ultrasound detection rate in the second semester is over 80%.[9,10]

Table 9. Possibilities for prevention and early detection of genetic and congenital conditions

Condition	Potentially detectable prenatally	Incidence reduced by optimal health status before and during pregnancy and/or good antenatal care	Outcomes improved by early post-natal detection	References
Down syndrome and other chromosomal disorders	✓	x[*]	x	11,12
Cystic fibrosis	✓		✓	13-15
Neural tube defects	✓	✓	✓	16
Cardiovascular anomalies	✓	✓	✓	17-20
Other structural congenital anomalies	✓	✓	✓	8-10
Genetic metabolic disorders	✓	x	✓	21,22
Congenital hearing loss		✓	✓	23-25

*Incidence is lower in younger women

Brief notes relevant to the prevention of congenital anomalies

Some neural tube defects can be prevented by peri-conceptional folic acid supplementation: The Ministry of Health recommends that women wishing to become pregnant who are at low risk of having a pregnancy affected by a neural tube defect should take 800 µg of folic acid daily for at least four weeks prior to conception and for 12 weeks after.[26] The 2015 Cochrane review on this topic found high quality evidence that daily folic acid supplementation prevents some neural tube defects (risk ratio 0.31, 95% CI 0.17 to 0.58; five studies; 6708 births).[16] Since this review, some evidence has been published for a possible protective effect of folic acid in relation to congenital heart defects and cleft lip and palate.[27,28]

Maternal smoking is associated with an increased risk of non-chromosomal birth defects: Odds ratios in the range 1.25–1.50 for limb reduction defects, clubfoot, oral clefts and defects of the eyes and gastrointestinal system (especially gastroschisis and abdominal hernias), and odds ratios in the range 1.09–1.19 for digit anomalies, cryptorchidism and defects of the heart and musculoskeletal system.[29]

Heavy drinking in pregnancy, especially binge drinking, can have severe effects on the developing fetus by disrupting brain development leading to cognitive, motor and behavioural disability with life-long consequences.[30,31] Fetal alcohol syndrome also produces distinctive facial anomalies if fetal exposure occurs in mid-gestation, and growth retardation, and is associated with a greatly increased risk of vision and hearing impairments, and a wide variety of congenital malformations.[30,32]

Maternal obesity seems to be associated with a small increase in the rates of some congenital anomalies, including heart defects and neural tube defects, and the risk may increase with greater degree of obesity.[33-35] In addition, maternal obesity is associated with at least 20% lower rates of detection of fetal anomalies via ultrasound, in comparison to women with normal body mass index.[36,37]

Diabetic women who become pregnant have a risk of having a baby with a major congenital anomaly around that is twice that of other women[38] and a risk having a baby with a congenital heart defect that is almost four times higher.[17] These risks can be reduced by optimising maternal health in the peri-conception period especially by maintaining good control of blood glucose levels.[20]

Maternal infections known to cause birth defects include toxoplasmosis, rubella, cytomegalovirus, herpes, and syphilis (the TORCH infections), as well as varicella, influenza, Zika virus and Lymphocytic Choriomeningitis (the last two are not known to occur in New Zealand).[39-42]

Common medications causing birth defects include: angiotensin converting enzyme inhibitors (for hypertension), anticonvulsants (for epilepsy), anti-neoplastic agents (for cancer), and systemic retinoids (for acne and other skin conditions).[39,43,44] In general, medicines should be prescribed to pregnant women only when absolutely necessary, when the benefits outweigh the risks. Risks associated with some medications can be mitigated, for example folic acid supplementation can reduce risks associated with anti-convulsant medication.

Key points for achieving optimal pre-pregnancy health status to reduce congenital anomaly risk:[45]

- Take folic acid supplements (to prevent neural tube defects)
- Seek medical advice before becoming pregnant if you have a chronic condition such as diabetes, hypothyroidism, epilepsy or hypertension where the condition itself, or the medication used to treat it, may increase the risk of congenital anomalies in your baby
- Avoid smoking, alcohol and recreational drugs
- Lose weight if obese
- Seek medical advice before becoming pregnant if there is a family history of a genetic disorder
- Make sure you are immune to rubella, consider varicella vaccination if not already immune

Key points for achieving optimal health status during pregnancy to reduce congenital anomaly risk[45-47]

- Avoid smoking, alcohol and recreational drugs
- Take folic acid and iodine
- Do not take therapeutic drugs except on medical advice that the benefits outweigh the risks
- Register with a Lead Maternity Carer early in pregnancy (before 10 weeks' gestation)
- Make a decision about screening tests
- Take care with personal and food hygiene, wash hands before eating (especially if you have contact with young children), and avoid contact with cat faeces

Although listeria infection does not cause congenital anomalies, pregnant women should avoid eating soft cheeses, delicatessen meats, pâtés, hummus-based spreads, refrigerated smoked seafood and salad bar cold salads to prevent miscarriage, preterm birth and stillbirth due to listeria infection.[48,49]

The following section uses data from the National Minimum Dataset to describe congenital anomalies in babies from 2000–2015 and concludes with a brief overview of some of the evidence relating to early diagnosis of these conditions.

Evidence-based health care for children, young people and parents affected by congenital anomalies

There are many thousands of different congenital anomalies so it is not practical to provide information here on the specific care each one requires. Instead, this section offers information on some new developments in prenatal and postnatal detection of congenital anomalies and highlights some of the findings from a review of the maternity care received by women who experienced perinatal deaths due to congenital anomalies in New Zealand in 2010.

In New Zealand, pregnant women are offered screening tests for Down syndrome and a fetal anatomy scan at 18–20 weeks' gestation.[50] The aim of fetal anomaly screening is to identify potential problems so that parents can make an informed choice about whether to continue the pregnancy if an anomaly is identified, and have time to prepare for what is to come, whether it is a termination, a baby who will need postnatal treatment or palliative care, or a child who will have long term disability.[51] Prenatal detection also allows planning for delivery in a specialist centre and, for a few conditions, intra-uterine therapy.[51] Ultrasound scanning to detect fetal malformations reduces perinatal mortality rates if there is a high level of diagnostic expertise and termination of pregnancy for fetal abnormality is widely accepted in the population.[52]

In New Zealand in 2010 there were 211 perinatal deaths due to congenital anomalies (30% of the 704 perinatal deaths in that year).[53] A review project based on the Perinatal and Maternal Mortality Review Committee dataset assessed the quality of the maternity care received by the women with one of the 137 perinatal deaths that were due to a congenital cardiovascular, central nervous system or chromosomal abnormality in that year.[53]

The review found that first contact with a health practitioner (most often a GP) occurred within 10 weeks of gestation in 74% of the women and within 14 weeks in 85% but there was often a significant delay in registering with a lead maternity carer (LMC). This meant that some women presented to a LMC too late for first trimester screening for Down syndrome. Of the 129 women who presented to a health professional at less than 20 weeks' gestation, 97 (71% of the 137) were offered first and/or second trimester screening and 82 (60% of the 137) had first and or second trimester screening. Fifteen women (11% of the 137) declined screening.

The review also found that only 7% of the women were documented as having taken folate supplements (to prevent neural tube defects) prior to pregnancy although 54% had taken them during pregnancy. On review of the women's ultrasound images it was found that some anomalies could have been detected earlier. The review made a number of recommendations, including those following. All women should receive preconception counselling to optimise their health and identify any risks for congenital anomalies resulting from previous obstetric history or family history. There should be a media campaign to promote peri-conceptional folate and the evidence on fortification of bread with folate should be further investigated. All women should be educated about the importance of booking before 10 weeks. GPs should be effective at offering first trimester screening since they are often a woman's first point of contact with maternity care and they should expedite booking with a LMC. If screening has not already been arranged then LMCs should offer all women first and second trimester screening, as required by the Ministry of Health, as this will enable the early diagnosis of a proportion of congenital anomalies.

It is now possible to test for Down, Edwards and Patau syndromes early in pregnancy using cell-free fetal DNA obtained from a sample of the mother's blood (non-invasive prenatal testing, NIPT).[54] A recent systematic review commissioned by the UK National Screening Committee, which included 41 studies relevant to Down syndrome, found that NPIT has very high sensitivity (99.3%) and specificity (99.9%) for Down syndrome.[55] Nevertheless, in the general obstetric population where the prevalence of Down syndrome is low, it could be expected that for every four Down syndrome cases detected there would be one false positive result so it is essential that, if a woman is considering a termination following a positive NIPT result, she has the diagnosis confirmed with an invasive diagnostic test (amniocentesis or chorionic villus sampling).[55] Non-invasive prenatal testing is available to New Zealand women on a user pays basis but is not yet part of publicly funded ante-natal screening.[56]

Congenital heart disease (CHD) causes more early neonatal deaths that any other type of congenital anomaly, accounting for around 30% of all early neonatal deaths associated with congenital anomalies in EUROCAT (a European network of population-based registries) in 2008–2012.[57] In cases of major or critical CHD (defined as cases requiring intervention or resulting in death within one year or within four weeks, respectively) delayed diagnosis is associated with increased mortality and morbidity.[58]

In New Zealand during 2006–2010 antenatal ultrasound picked up only 46% of critical CHD.[59] Postnatal physical examination cannot detect all babies with CHD as some do not display any physical symptoms until after hospital discharge.[58] Almost 20% of New Zealand infants with critical CHD are not diagnosed until after initial hospital discharge.[59]

Newborn pulse oximetry will detect hypoxaemic infants and is a simple and non-invasive method for screening for CHD which increases detection rates for CHD when used as an adjunct to physical examination.[58] A number of developed countries have such screening and a pulse oximetry pilot programme is currently underway in Auckland and Wellington.[60,61]

These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of congenital anomalies are provided for further reading.

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CYSTIC FIBROSIS

Introduction

Cystic fibrosis (CF) is a multi-organ disease with an autosomal recessive pattern of inheritance. For a child to have CF both parents need to be carriers of a CF gene. It is most common in populations of predominantly Northern European descent where around one in 3,000 babies are born with the condition.[1] Most developed countries where CF is common, including New Zealand, have national newborn screening programmes that identify most babies with CF soon after birth.[1,2]

Cystic fibrosis is caused by a number of mutations in the gene encoding the cystic fibrosis transmembrane conductance regulator (CFTR) protein.[3] The CFTR regulates anion transport across cell membranes. When CFTR activity is reduced or absent the mucus secreting functions of the epithelial cells lining the airways, pancreatic ducts and other tissues are impaired.[3] The most significant result of this dysfunction is obstruction of the small airways by thick mucus leading to frequent infections, bronchiectasis and progressively worsening lung function. Other effects include pancreatic insufficiency leading to malabsorption of nutrients (and diabetes in some cases), and cirrhosis of the liver. Most males with CF have congenital absence of the vas deferens which makes them infertile.[4]

Life expectancy for people with CF is improving due to better treatment and it is now around forty years.[1,5] Recently, new drugs have been developed that correct the basic defect in one genetic mutation in CFTR.[6] These drugs hold the promise of effective disease-modifying treatment and could potentially prevent lung disease if they were started as soon as the disease was identified by newborn screening.[1]

The following section reviews cystic fibrosis in children and young people using information from the newborn metabolic screening programme, New Zealand Cystic Fibrosis Registry, National Mortality Collection and National Minimum Dataset. The section concludes with a brief overview of possibilities for prevention and evidence-based health care for children and young people with CF.

Data sources and methods

Indicator

> *Rates of cystic fibrosis (CF) among 0–24 year olds*

Definition

Hospitalisations of 0–24 year olds with cystic fibrosis per 100,000 population

Data sources

Numerator: National Minimum Dataset

Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

Cystic fibrosis was the primary diagnosis or was documented as one of the first 15 diagnoses
Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

There were 14 deaths of 0–24 year olds with cystic fibrosis (CF) as the underlying cause of death in New Zealand during 2009 to 2013, as documented within the National Mortality Collection.

CF is one of the 20 congenital metabolic disorders that babies are screened for within the New Zealand Newborn Metabolic Screening Programme (NMSP).[2] Screening tests are performed utilising blood samples obtained from the babies' heels during the first 48–72 hours of life. The NMSP screened 58,673 babies in 2014, of which 15 had cystic fibrosis detected. The 2014 incidence rate of CF was 27.1 per 100,000 live births, including one case diagnosed outside of the NMSP.

The National Cystic Fibrosis Data Registry includes over 95% of people with CF in New Zealand. Around two-thirds (*n*=293) of the 443 individuals registered in 2014 were registered before 24 years. Of those registered, 33 (7.4%) were aged 0–3 years at registration.[7]

The number of 0–24 year olds hospitalised with CF during 2011 to 2015 is presented in **Table 10**. It also presents the number of hospital discharges in which CF was documented as the primary diagnosis or as any diagnosis.

While there has been year-on-year variability in hospitalisations for CF since 2000, the hospitalisation rate has remained relatively stable over the last five years (**Figure 6**).

Table 10. Individuals aged 0–24 years hospitalised with cystic fibrosis using primary diagnosis compared to all cases, New Zealand 2011–2015

Age group	Unique individuals *(n)*	Hospitalisations *(n)*		Ratio All:Primary
		Primary diagnosis	All cases	
Cystic fibrosis				
Hospitalisation				
0–24 years	336	2,122	2,625	1.24
0–14 years	226	1,199	1,533	1.28
15–24 years	147	923	1,092	1.18

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with cystic fibrosis listed in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Figure 6. Hospitalisations for cystic fibrosis in 0–24 year olds, New Zealand 2000–2015
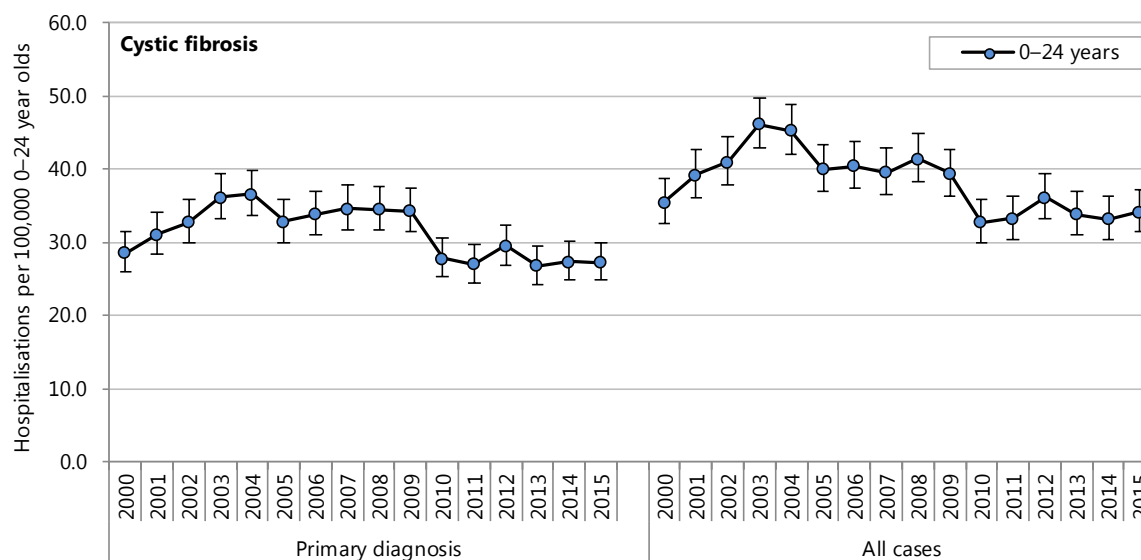
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with cystic fibrosis listed in any of the first 15 diagnoses

Diagnosis

The majority of hospitalisations of 0–24 year olds involving cystic fibrosis had CF as the primary reason for hospitalisation. The diagnoses with the highest hospitalisation rate were CF with pulmonary or other manifestations (**Table 11**).

Table 11. Hospitalisations involving cystic fibrosis in 0–24 year olds, by primary diagnosis, New Zealand 2011– 2015

Primary diagnosis	*n*	Annual average	Rate	95% CI	%
Cystic fibrosis* in 0–24 year olds					
New Zealand					
Cystic fibrosis with pulmonary manifestations	955	191	12.43	11.67–13.25	36.4
Cystic fibrosis with intestinal manifestations	76	15	0.99	0.79–1.24	2.9
Cystic fibrosis with other manifestations	968	194	12.60	11.83–13.42	36.9
Cystic fibrosis, unspecified	123	25	1.60	1.34–1.91	4.7
Cystic fibrosis total	2,122	424	27.63	26.48–28.83	80.8
Other endocrine, nutritional and metabolic diseases	12	2	0.16	0.09–0.27	0.5
Diseases of the respiratory system	106	21	1.38	1.14–1.67	4.0
Diseases of the musculoskeletal system and connective tissue	76	15	0.99	0.79–1.24	2.9
Factors influencing health service contact	75	15	0.98	0.78–1.22	2.9
Symptoms and/or abnormal clinical findings NEC	69	14	0.90	0.71–1.14	2.6
Injury and/or poisoning	58	12	0.76	0.58–0.98	2.2
Infectious and parasitic diseases	38	8	0.49	0.36–0.68	1.4
Other diagnoses	69	14	0.90	0.71–1.14	2.6
Total	2,625	525	34.18	32.89–35.51	100.0

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Cystic fibrosis in any of the first 15 diagnoses; Rate per 100,000 0–24 year olds; NEC = not elsewhere classified

Demographic distribution

Table 12 presents the demographic distribution of individuals with CF in New Zealand between 2011 and 2015. CF was significantly lower among individuals residing in areas with high deprivation scores (NZDep2013 deciles 9–10), and significantly higher among 0–4 year olds compared to 15–24 year olds. The majority of individuals with CF were of European/Other ethnicities.

Although the age specific rate of 0–4 year olds with CF is higher than those for the other age groups, hospitalisations have generally been lower for 0–4 year olds (**Figure 7**). The hospitalisation rate for Māori has gradually increased since 2000, although is still consistently lower than the hospitalisation rate for European/Other (**Figure 8**).

Table 12. Individuals aged 0–24 years hospitalised with cystic fibrosis, by demographic factor, New Zealand 2011– 2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Cystic fibrosis* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	97	6.84	1.00	
Deciles 3–4	105	7.85	1.15	0.87–1.51
Deciles 5–6	100	6.94	1.01	0.77–1.34
Deciles 7–8	97	5.97	0.87	0.66–1.16
Deciles 9–10	74	3.98	0.58	0.43–0.79
Prioritised ethnicity				
Māori	38	2.11	0.30	0.22–0.43
Pacific	5	0.71	0.10	0.04–0.25
Asian/Indian	6	0.63	0.09	0.04–0.20
MELAA	5	4.96	0.71	0.30–1.73
European/Other	285	6.94	1.00	
Gender				
Female	154	4.10	1.00	
Male	183	4.66	1.14	0.92–1.41
Age group (years)				
0–4	106	6.80	1.45	1.13–1.86
5–14	153	5.13	1.09	0.87–1.37
15–24	147	4.69	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. Cystic fibrosis* in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is Level 1 prioritised; Decile is NZDep2013

Figure 7. Hospitalisations for cystic fibrosis in 0–24 year olds, by age group, New Zealand 2000–2015
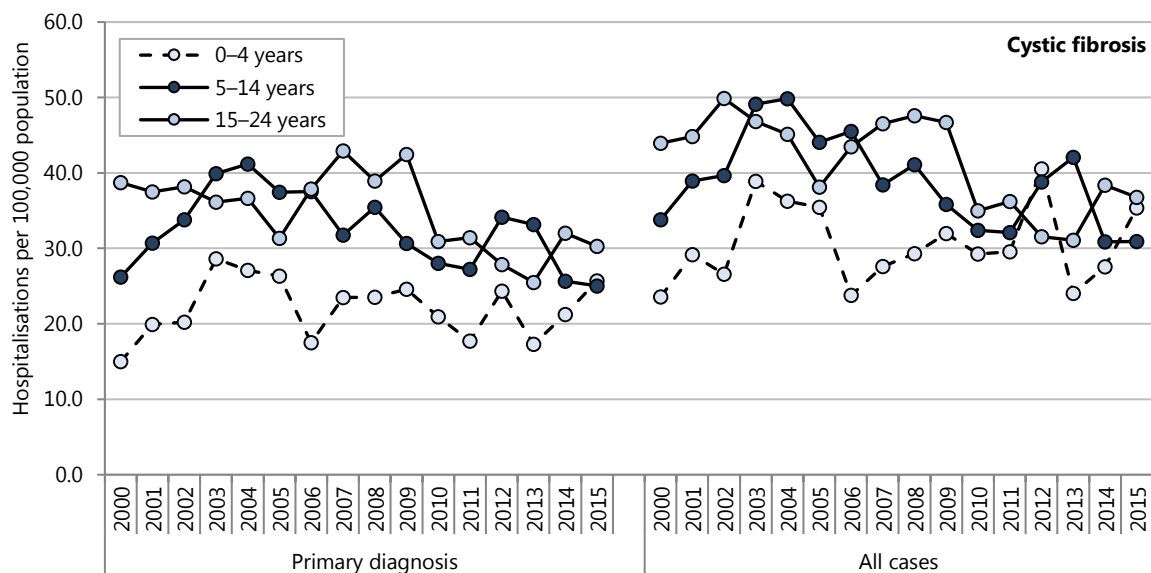
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with cystic fibrosis listed in any of the first 15 diagnoses; Hospitalisations per 100,000 population

Figure 8. Hospitalisations involving cystic fibrosis in 0–24 year olds, by ethnicity, New Zealand 2000–2015
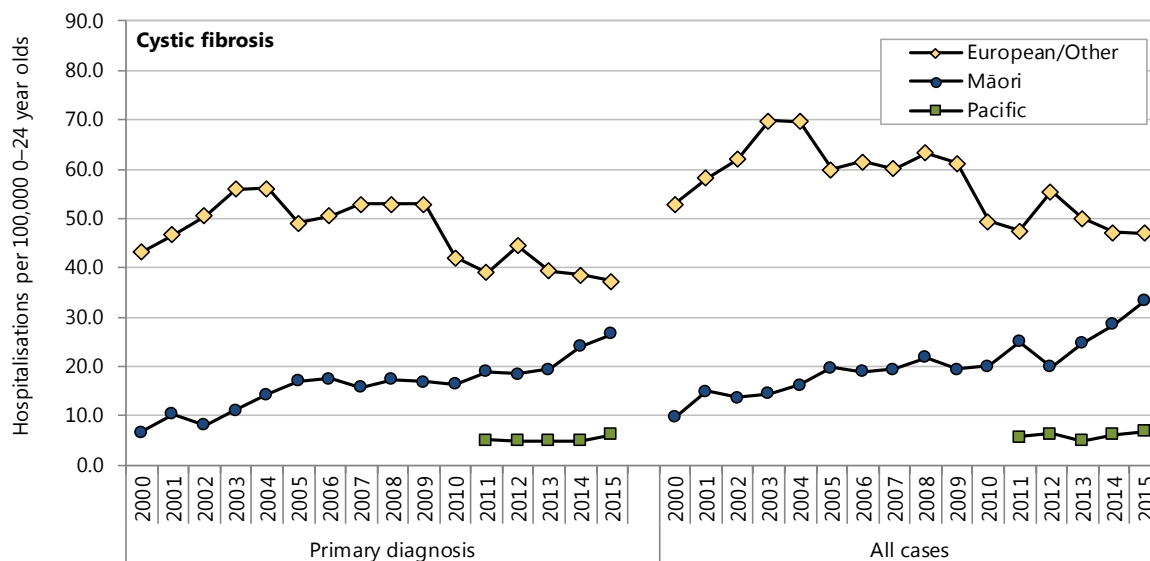
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with cystic fibrosis listed in any of the first 15 diagnoses

Regional distribution

Table 13 presents the number of individuals resident in each district health board that had a CF diagnosis during 2011 to 2015. It also presents the number of hospital discharges in which CF was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and avoid undercounting that would occur if only primary diagnoses are included in discharge data. For CF the All:Primary diagnosis ratio was higher than the national ratio in Counties Manukau, Waikato, Bay of Plenty, Whanganui, Hutt Valley, and Canterbury DHBs and lower in Northland, Waitemata, Auckland, Lakes, Taranaki, Hawke's Bay, MidCentral, Nelson Marlborough, and Southern DHBs. Ratios were similar to the national ratio in the remaining district health boards (**Table 13**).

Table 13. Hospitalisations for cystic fibrosis in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Cystic fibrosis in 0–24 year olds				
Northland	8	45	48	1.07
Waitemata	27	138	159	1.15
Auckland	35	170	203	1.19
Counties Manukau	26	70	90	1.29
Waikato	32	209	280	1.34
Bay of Plenty	28	167	228	1.37
Lakes	9	81	92	1.14
Tairawhiti	<5	s	s	s
Taranaki	11	50	60	1.20
Hawke's Bay	15	79	87	1.10
MidCentral	18	167	178	1.07
Whanganui	8	47	62	1.32
Hutt Valley	10	65	90	1.38
Capital & Coast	27	115	143	1.24
Wairarapa	<5	s	s	s
Nelson Marlborough	18	142	157	1.11
South Canterbury	<5	s	s	s
Canterbury	59	310	397	1.28
West Coast	<5	s	s	s
Southern	27	167	200	1.20
New Zealand	336	2,122	2,625	1.24

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with cystic fibrosis listed in any of the first 15 diagnoses

Evidence for good practice

Possibilities for prevention

Preconception carrier screening of couples planning a pregnancy and prenatal testing early in pregnancy are both possible and recommended by the American College of Medical Genetics,[8] the American College of Obstetricians and Gynecologists,[9] the National Institutes of Health,[10] and the Human Genetics Society of Australasia.[11] The sensitivity of carrier screening varies between ethnic groups and ranges (in the US population) from almost 90% in non-Hispanic whites to around 50% in Asian Americans.[9] In places where carrier screening has been carried out, there has been a decrease in the incidence of cystic fibrosis.[12,13]

In New Zealand, carrier screening and prenatal testing is free only for family members and partners of people with CF and relatives and partners of known carriers of CF.[11,14] In Australia, since 2006, CF carrier screening has been available to individuals and couples in Victoria as a fee-for-service programme at a cost of $150 per patient.[15] There has been some resistance to the uptake of CF screening from both the public (who lack awareness of CF) and health professionals. Some health professionals have concerns about the time needed to counsel patients about genetic testing, lack knowledge about carrier frequency and the risks of CF and are unaware that 95% of cases of CF occur without a family history of the condition.[15]

It is difficult to assess the cost-effectiveness of carrier screening and published economic evaluations of CF screening have had considerable variation in methods and results.[16] Estimating cost-effectiveness involves weighing the cost of screening against the cost savings that result from the lifetime healthcare costs averted because there are fewer people with CF (because couples who are both carriers can choose to terminate their affected foetuses or to use IVF with preimplantation genetic testing). There are new developments in genetic testing, including next-generation DNA sequencing, that make it possible to screen for many disorders at once and are likely to bring down the cost of genetic testing.[17] Therapies for CF are advancing, and it is possible that in the future there may be treatments that can be given from birth to correct the defect in CFTR function and prevent some or all of the complications of CF.[18] The availability of such therapies could have several consequences: people with CF would be more likely to live longer with a good quality of life (although new drugs will probably be very expensive), and couples might be less likely to terminate a CF pregnancy if they were more hopeful of their child's prognosis, (providing they did not have to pay for their child's healthcare themselves).[18]

Evidence-based health care for children and young people with cystic fibrosis

Newborn screening leads to better nutritional outcomes for children with CF and has the potential to improve pulmonary outcomes.[19] When a couple have a baby with CF, there is a 25% risk that any future pregnancies may also be affected by CF, so genetic counselling is indicated. The international consensus is that a person newly diagnosed with CF should have immediate and on-going access to a CF specialist centre staffed by a multidisciplinary team.[20] New Zealand does not have the population to support the types of specialist CF centres found overseas. Care for New Zealand children with CF should be provided using a shared care model. The majority of care should be based in a clinic at a hospital near their home, supplemented with at least annual reviews at a regional CF centre.[21]

To maintain the best possible lung function, people with CF (PWCF) need meticulous daily management of their lung disease. This may involve the use of airway clearance techniques taught by physiotherapists and inhaled aerosol medications together with prompt and aggressive treatment of infective exacerbations.[20] There is no good evidence to indicate which is the best way of educating PWCF to manage their disease.[22] People with CF need monitoring of their nutritional status as they are at risk of CF-related malnutrition due to pancreatic insufficiency and they (and their families) need psychosocial support to deal with the demoralisation that results from having multiple health problems.[20]

These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of CF are provided for further reading.

New Zealand guidelines
- Standards of Care for Cystic Fibrosis in New Zealand Group, Medical Advisory Committee of Cystic Fibrosis Association of New Zealand. 2011. Standards of Care for Cystic Fibrosis in New Zealand Cystic Fibrosis Association of New Zealand.
http://www.cfnz.org.nz/wp-content/uploads/Standard-of-Care-NZ-2011.pdf

International guidelines
- Lahiri T, Hempstead SE, Brady C, et al. 2016. Clinical Practice Guidelines from the Cystic Fibrosis Foundation for preschoolers with cystic fibrosis. *Pediatrics*.
http://pediatrics.aappublications.org/content/137/4/e20151784.long

- Smyth AR, Bell SC, Bojcin S, et al. 2014. European Cystic Fibrosis Society Standards of Care: Best Practice guidelines. *Journal of Cystic Fibrosis*, 13 Suppl 1, S23–42.
http://www.sciencedirect.com/science/article/pii/S156919931400085X

- Conway S, Balfour-Lynn IM, De Rijcke K, et al. 2014. European Cystic Fibrosis Society Standards of Care: Framework for the Cystic Fibrosis Centre. *Journal of Cystic Fibrosis*, 13, S3-S22.
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http://www.sciencedirect.com/science/article/pii/S1569199314000861

- Cystic Fibrosis Trust. 2011. Standards for the clinical care of children and adults with cystic fibrosis in the UK (second edition). London: Cystic Fibrosis Trust.
https://www.cysticfibrosis.org.uk/~/media/documents/the-work-we-do/care/consensus-documents/standards-for-the-clinical-care-of-children-and-adults-with-cf-dec-11.ashx?la=en

Evidence-based medicine reviews
- The Cochrane Library Reviews relating to Cystic Fibrosis
http://www.cochranelibrary.com/topic/Lungs%20%26%20airways/Fibrosis%3A%20cystic%20fibrosis/?per-page=100&stage=review

- Edmondson C, Davies JC. 2016. Current and future treatment options for cystic fibrosis lung disease: latest evidence and clinical implications. *Therapeutic Advances in Chronic Disease*, 7(3), 170-83.
http://www.ncbi.nlm.nih.gov/pmc/articles/pmid/27347364/

- Saiman L, Siegel JD, LiPuma JJ, et al. 2014. Infection prevention and control guideline for cystic fibrosis: 2013 update. *Infection Control & Hospital Epidemiology*, 35 Suppl 1, S1-S67.
(summary at http://www.guideline.gov/content.aspx?id=48772&search=cystic+fibrosis)

- Mogayzel PJ, Jr., Naureckas ET, Robinson KA, et al. 2013. Cystic fibrosis pulmonary guidelines. Chronic medications for maintenance of lung health. *American Journal of Respiratory and Critical Care Medicine*, 187(7), 680-9. (summary at https://www.guideline.gov/content.aspx?id=45307)

Other relevant publications

- Quon BS, Rowe SM. 2016. New and emerging targeted therapies for cystic fibrosis. *BMJ*, 352, i859. http://www.bmj.com/content/352/bmj.i859.long

- Massie J, Ioannou L, Delatycki M. 2014. Prenatal and preconception population carrier screening for cystic fibrosis in Australia: where are we up to? *The Australian & New Zealand Journal of Obstetrics & Gynaecology*, 54(6), 503-9

- PORT CFNZ National Data Registry. 2014 Registry Report. Cystic Fibrosis New Zealand. http://cfnz.org.nz/wp-content/uploads/2015/12/2014-PORT-CFNZ-Registry-Report.pdf

Websites

- National Institute for Health and Care Excellence Cystic Fibrosis https://www.nice.org.uk/guidance/conditions-and-diseases/genetic-conditions/cystic-fibrosis

- Cystic Fibrosis Association Of New Zealand Publications http://www.cfnz.org.nz/our-services/library/downloads/

- Cystic Fibrosis Trust (UK) Consensus Documents https://www.cysticfibrosis.org.uk/the-work-we-do/clinical-care/consensus-documents

- European Cystic Fibrosis Society Standards of care available in open access https://www.ecfs.eu/content/ecfs-standards-care-available-open-access

- Cystic Fibrosis Foundation (US) CF Clinical care guidelines https://www.cff.org/For-Caregivers/CF-Clinical-Care-Guidelines

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7. PORT CFNZ National Data Registry. 2014 Registry Report. Cystic Fibrosis New Zealand. http://cfnz.org.nz/wp-content/uploads/2015/12/2014-PORT-CFNZ-Registry-Report.pdf
8. Grody WW, Cutting GR, Klinger KW, et al. 2001. Laboratory standards and guidelines for population-based cystic fibrosis carrier screening. *Genetics in Medicine,* 3(2) 149-54. http://dx.doi.org/10.109700125817-200103000-00010
9. The American College of Obstetricians and Gynaecologists. 2011. ACOG Committee Opinion No. 486: Update on carrier screening for cystic fibrosis. *Obstetrics and Gynecology,* 117(4) 1028-31. http://dx.doi.org/10.1097/AOG.0b013e31821922c2
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14. Cystic Fibrosis Association of New Zealand. Genetics of cystic fibrosis. http://cfnz.org.nz/wp-content/uploads/2015/10/Genetics-of-CF-Laypersons-Guide.pdf
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21. Standards of Care for Cystic Fibrosis in New Zealand Group, Medical Advisory Committee of Cystic Fibrosis Association of New Zealand,. 2011. Standards of Care for Cystic Fibrosis in New Zealand Cystic Fibrosis Association of New Zealand. http://www.cfnz.org.nz/wp-content/uploads/Standard-of-Care-NZ-2011.pdf

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DIABETES

Introduction

Type 1 diabetes mellitus (T1DM) is an autoimmune disease in which there is progressive destruction of the β cells in the pancreas.[1] The disease is believed to develop as a result of interaction between susceptibility genes and as yet unidentified environmental factors.[2] The pancreas no longer produces the hormone insulin, which promotes the absorption of glucose from the blood into cells and is essential for life, and so people with T1DM must take exogenous insulin for the rest of their lives to keep their blood sugar levels under control.[1]

Excess blood glucose (hyperglycaemia) leads to diabetic ketoacidosis, the leading cause of morbidity and mortality in children with T1DM. In the long-term high blood glucose levels can damage blood vessels and nerves resulting in atherosclerosis, loss of vision, kidney disease and nerve damage. Intensive insulin therapy, which aims to keep blood glucose as close to normal as possible, prevents nerve damage,[3] kidney damage and macrovascular complications such as heart disease and stroke but is associated with an increased risk of severe hypoglycaemia (low blood glucose).[4] Mild hypoglycaemia can produce irritability and inattention while severe hypoglycaemia can produce loss of consciousness and seizures and there is some suggestion that episodes of severe hypoglycaemia in early childhood can have long lasting (although mild) effects on cognitive function.[5]

Type 1 diabetes mellitus is much the most common type of diabetes in children and young people.[6] Most people with T1DM (around 75%) were diagnosed in childhood or young adulthood.[6] The incidence of T1DM has been rising over recent decades, both globally[6] and in New Zealand.[7]

The following section reviews diabetes in children and young people using information from the National Minimum Dataset and New Zealand Health Survey. The section concludes with a brief overview of evidence-based health care for children and young people with diabetes.

Data sources and methods

Indicators

> *Prevalence of diabetes*
> *Hospitalisations for diabetes*

Definitions

Prevalence of diabetes

> Diabetes (diagnosed, excluding diabetes during pregnancy) among adults aged 15+ years

Hospitalisations for diabetes

> Hospitalisations of 0–24 year olds with a diagnosis of diabetes per 100,000 population

Data sources

Prevalence of diabetes

New Zealand Health Survey (2006/07–2014/15), see **Appendix 3**

Hospitalisations for diabetes

Numerator:	National Minimum Dataset
Denominator:	Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

Prevalence of diabetes

Adult respondents (aged 15+ years) are defined as having diabetes if they had ever been told by a doctor that they have diabetes. This does not include diabetes during pregnancy (gestational diabetes).
Note that NZHS definition is likely to underestimate the true number of people with diabetes, as some people may not be aware that they have diabetes.

Hospitalisations for diabetes

Diabetes was the primary diagnosis or was documented as one of the first 15 diagnoses.
Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

The percentage of those aged 15–24 years diagnosed with diabetes in the New Zealand Health Surveys was similar for the years 2006/07 to 2014/15, with the exception of 2012/13, although this year was not significantly different. The percentage of males diagnosed with diabetes was slightly higher than for females, but not significantly so (**Figure 9**).

There was a total of 19 deaths of 0–24 year olds where diabetes was the underlying cause of death in New Zealand between 2000 and 2013, as documented within the National Mortality Collection. The majority of these deaths were due to type 1 diabetes.

The number of 0–24 year olds hospitalised with diabetes during 2011 to 2015 is presented in **Table 14**. It also presents the number of hospital discharges in which diabetes was documented as the primary diagnosis or as any diagnosis. The majority of hospitalisations were for type 1 diabetes.

The rate of hospitalisations for diabetes has increased since 2000, particularly where diabetes was documented within the first 15 diagnoses (**Figure 10**).

Figure 9. Diabetes (diagnosed), by demographic factor, NZ Health Survey 2006/07–2014/15
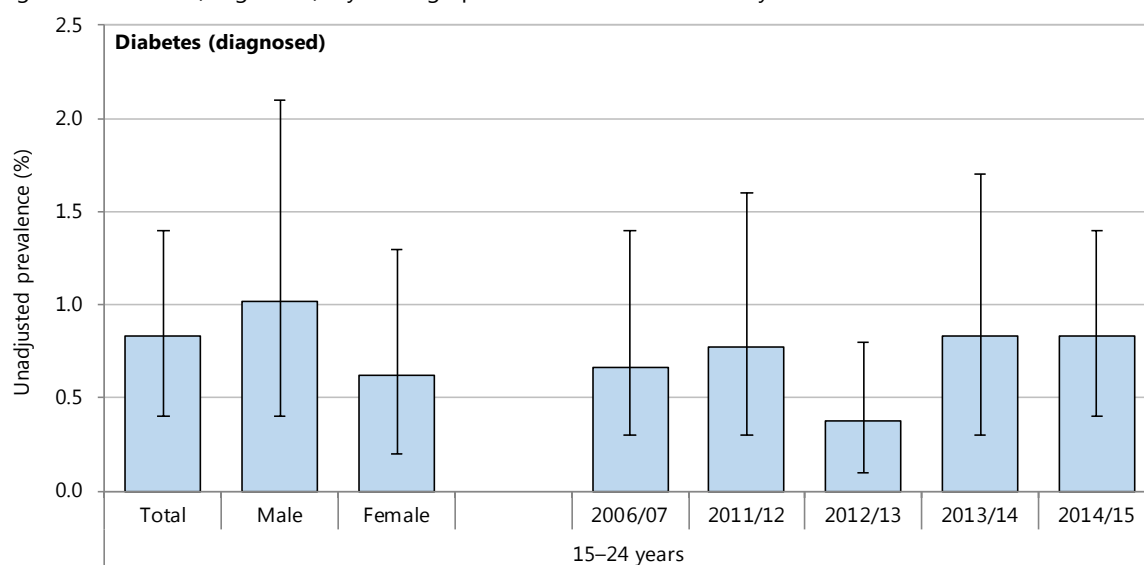
Source: NZ Health Survey

Table 14. Individuals aged 0–24 years hospitalised with diabetes using primary diagnosis compared to all cases, New Zealand 2011–2015

	Unique individuals (n)	Hospitalisations (n)		Ratio All : Primary
		Primary diagnosis	All cases	
Diabetes				
Hospitalisation				
0–24 years	4,137	6,466	12,308	1.90
0–14 years	1,772	2,771	4,252	1.53
15–24 years	2,614	3,695	8,056	2.18
Diabetes in 0–24 year olds				
Diabetes: Type 1	3,242	6,098	9,796	1.61
Diabetes: Type 2	718	257	1,462	5.69
Diabetes: other	350	111	1,056	9.51

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with diabetes listed in any of the first 15 diagnoses; The sum of the age groups or of the diagnoses may total to more than the 0–24 year old total

Figure 10. Hospitalisations for diabetes in 0–24 year olds, New Zealand 2000–2015
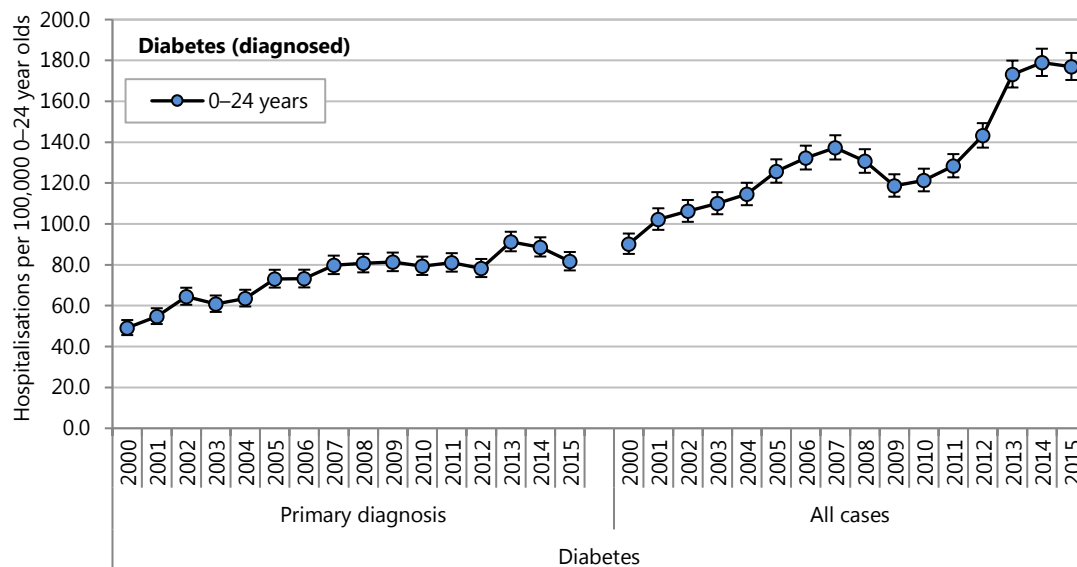
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with diabetes listed in any of the first 15 diagnoses

Diagnosis

The majority of hospitalisations of 0–24 year olds involving type 1 diabetes had diabetes as the primary reason for hospitalisation within which type 1 diabetes mellitus with ketoacidosis was the diagnosis with the highest hospitalisation rate (**Table 15**).

Table 15. Hospitalisations involving type 1 diabetes in 0–24 year olds in 0–24 year olds, by primary diagnosis, New Zealand 2011–2015

Primary diagnosis	2011–2015 (n)	Annual average	Rate	95% CI	%
Type 1 diabetes* in 0–24 year olds					
New Zealand					
Type 1 diabetes mellitus with ketoacidosis†	3,109	622	40.48	39.08–41.92	31.7
Type 1 diabetes mellitus with poor control	816	163	10.62	9.92–11.38	8.3
Type 1 diabetes mellitus with hypoglycaemia	598	120	7.79	7.19–8.44	6.1
Type 1 diabetes mellitus with ophthalmic complications‡	85	17	1.11	0.90–1.37	0.9
Type 1 diabetes mellitus with other complications	107	21	1.39	1.15–1.68	1.1
Type 1 diabetes mellitus without complication	1,383	277	18.01	17.08–18.98	14.1
Type 1 diabetes mellitus total	6,098	1,220	79.39	77.42–81.41	62.2
Other endocrine, nutritional and metabolic diseases	60	12	0.78	0.61–1.01	0.6
Symptoms and/or abnormal clinical findings NEC	580	116	7.55	6.96–8.19	5.9
Infectious and parasitic diseases	553	111	7.20	6.62–7.83	5.6
Injury and/or poisoning	458	92	5.96	5.44–6.53	4.7
Other diagnoses	2,047	409	26.65	25.52–27.83	20.9
Total	9,796	1,959	127.54	125.04–130.09	100.0

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Type 1 diabetes in any of the first 15 diagnoses; Rate per 100,000 0–24 year olds; NEC = not elsewhere classified; † Type 1 diabetes mellitus with ketoacidosis includes those hospitalised with/without coma and/or lactic acidosis or where stated as uncontrolled; ‡ Type 1 diabetes mellitus with ophthalmic complications includes those hospitalised with ophthalmic complications stated as uncontrolled, with advanced ophthalmic disease, with other specified ophthalmic complication, with proliferative or other retinopathy, or with diabetic cataract

Demographic distribution

Table 16 presents the demographic distribution of individuals with diabetes in New Zealand between 2011 and 2015. There was a social gradient among these individuals with statistically significant increases in prevalence in each deprivation quintile (NZDep deciles 3–4 to 9–10) compared with those living in least deprived areas (deciles 1–2). Diabetes was significantly lower among males, and among 0–4 and 5–14 year olds (compared to 15–24 year olds), and significantly lower for Māori and Asian/Indian than for European/Other ethnic groups.

Table 16. Individuals aged 0–24 years hospitalised with diabetes, by demographic factor, New Zealand 2011– 2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Diabetes* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	754	53.13	1.00	
Deciles 3–4	792	59.23	1.11	1.01–1.23
Deciles 5–6	891	61.81	1.16	1.06–1.28
Deciles 7–8	1,103	67.90	1.28	1.16–1.40
Deciles 9–10	1,371	73.79	1.39	1.27–1.52
Prioritised ethnicity				
Māori	857	47.51	0.74	0.69–0.80
Pacific	455	64.21	1.00	0.91–1.11
Asian/Indian	185	19.30	0.30	0.26–0.35
MELAA	50	49.58	0.78	0.59–1.03
European/Other	2,626	63.91	1.00	
Gender				
Female	2,185	58.20	1.00	
Male	1,953	49.74	0.85	0.80–0.91
Age group (years)				
0–4	260	16.67	0.20	0.18–0.23
5–14	1,590	53.28	0.64	0.60–0.68
15–24	2,614	83.33	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Diabetes in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is Level 1 prioritised; Decile is NZDep2013

Since 2000, hospitalisations for diabetes had generally increased for each age group (**Figure 11**). The increase was most notable for the 0–4 year olds, for whom rates were consistently higher despite the age specific diabetes rate being lower than those for the other age groups. Over the same period, the primary diagnosis hospitalisation rate had gradually increased for all ethnic groups with notable increases for Māori and Pacific ethnic groups (**Figure 12**).

Figure 11. Hospitalisations involving diabetes in 0–24 year olds, by age group, New Zealand 2000–2015
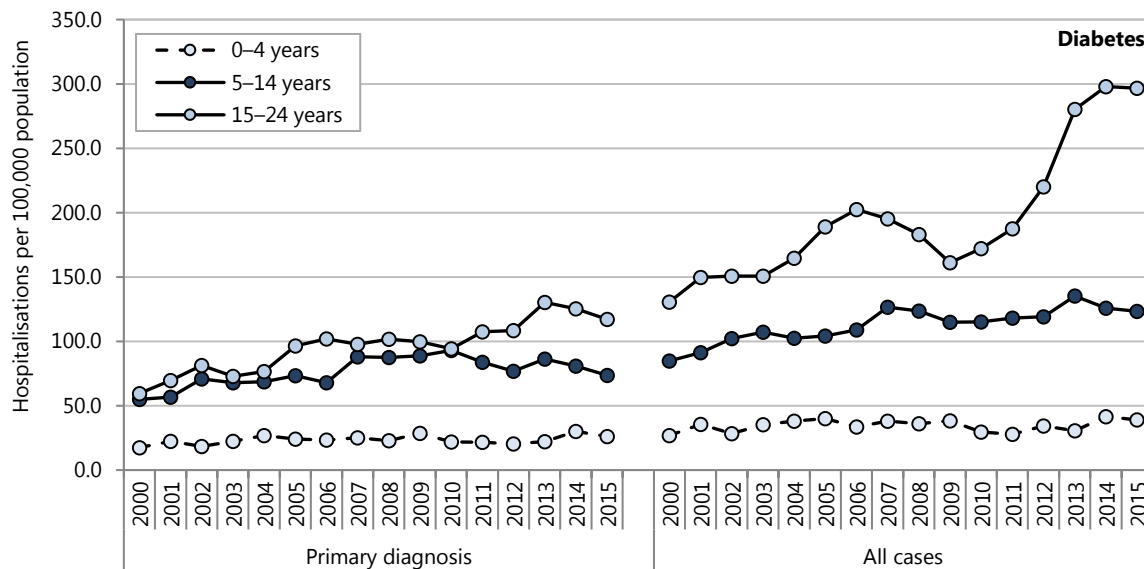
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with diabetes listed in any of the first 15 diagnoses; Caution rates for under 5 year olds are based on small numbers

Figure 12. Hospitalisations involving diabetes in 0–24 year olds, by ethnicity, New Zealand 2000–2015
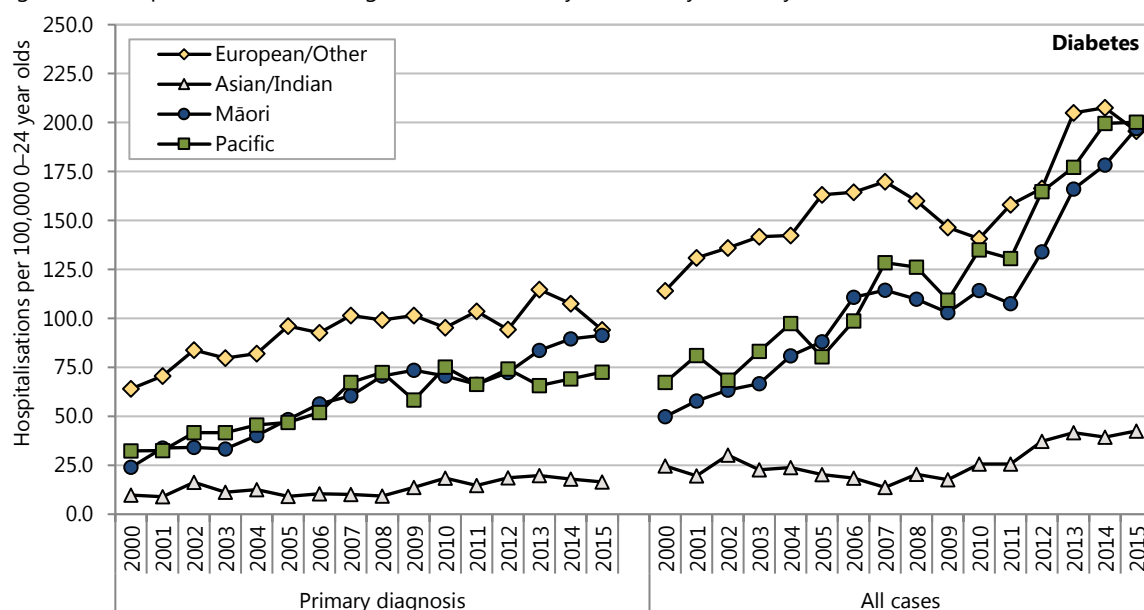
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with diabetes listed in any of the first 15 diagnoses

Regional distribution

Figure 13 shows the prevalence of diagnosed diabetes for the district health boards from the 2011/12 to 2013/14 New Zealand Health Surveys. None of the district health boards were significantly different to the New Zealand rate.

Table 17 presents the number of individuals resident in each district health board that had a diabetes diagnosis during 2011 to 2015. It also presents the number of hospital discharges in which diabetes was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and avoid undercounting that would occur if only primary diagnoses are included in discharge data. For diabetes the All:Primary diagnosis ratio was higher than the national ratio in Northland,

Waitemata, Auckland, Counties Manukau, Waikato, Tairawhiti, Hutt Valley, and Canterbury DHBs. Ratios were lower than the national ratio in the remaining district health boards (**Table 17**).

Figure 13. Diabetes (diagnosed) in 15–24 year olds, by district health board, NZ Health Survey 2011/12 to 2013/14
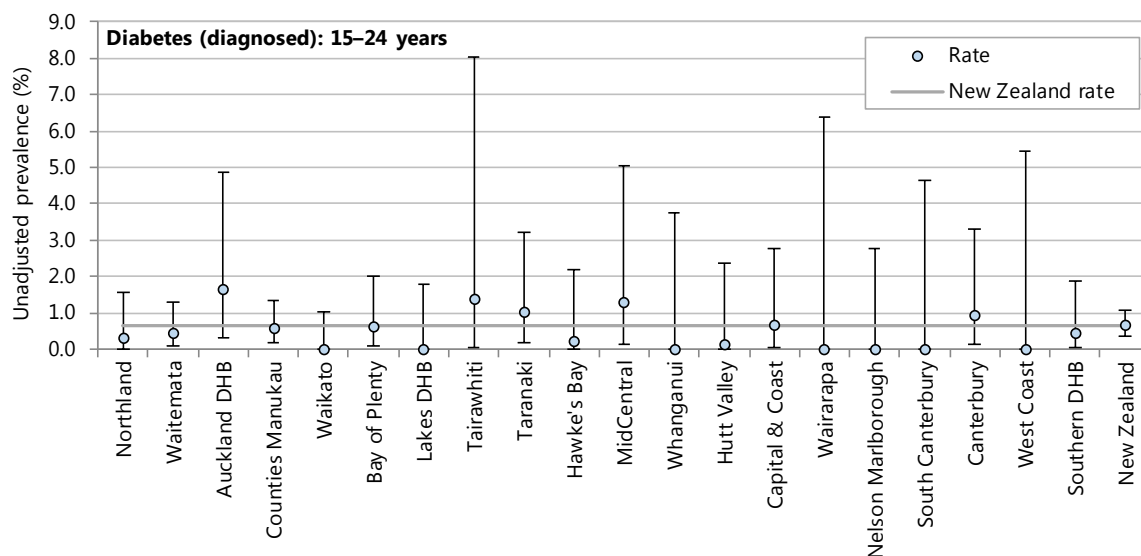
Source: NZ Health Survey

Table 17. Hospitalisations for diabetes in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All : Primary
		Primary diagnosis	All cases	
Diabetes in 0–24 year olds				
Northland	147	192	452	2.35
Waitemata	524	644	1,321	2.05
Auckland	433	423	975	2.30
Counties Manukau	540	557	1,275	2.29
Waikato	409	574	1,126	1.96
Bay of Plenty	215	346	642	1.86
Lakes	102	225	402	1.79
Tairawhiti	34	70	149	2.13
Taranaki	131	194	348	1.79
Hawke's Bay	169	354	579	1.64
MidCentral	175	290	529	1.82
Whanganui	66	92	162	1.76
Hutt Valley	142	180	346	1.92
Capital & Coast	271	345	625	1.81
Wairarapa	35	179	263	1.47
Nelson Marlborough	124	234	427	1.82
South Canterbury	64	167	226	1.35
Canterbury	457	577	1,121	1.94
West Coast	35	49	90	1.84
Southern	338	743	1,187	1.60
New Zealand	4,137	6,466	12,308	1.90

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with diabetes listed in any of the first 15 diagnoses

Evidence for good practice

Possibilities for prevention

There are currently no interventions proven to prevent or delay the onset of clinically apparent type 1 diabetes.[1] Primary prevention trials to date have been dietary interventions for infants at increased genetic risk designed to interrupt putative environmental risk factors for T1DM (such as infant formulas free of either cow's milk or bovine insulin, delayed introduction of gluten-containing foods, and vitamin D supplementation) and none of the dietary factors investigated have been shown to be unequivocal risk factors.[1] The European Nicotinamide Diabetes Intervention Trial found no effect of nicotinamide on the prevention or delay of T1DM development in at-risk relatives.[8]

Evidence-based health care for children and young people with diabetes

Insulin

From the time of diagnosis children and young people with type 1 diabetes require either multiple daily injections of insulin or continuous subcutaneous insulin infusion (via a pump) to control their blood glucose levels.[9] They (or their parents) also need to plan and monitor their carbohydrate intake so they can adjust their insulin dosage accordingly, and to monitor their blood glucose levels at least five times per day.[9] Intensive glucose control, which requires frequent blood glucose monitoring, has become standard therapy for T1DM because there is evidence that it lowers the risk of developing microvascular complications (such as eye and kidney disease), particularly in younger patients in the early stages of disease.[10]

Care by a multidisciplinary team

Children and young people with T1DM and their families need ongoing access to a multidisciplinary paediatric diabetes team.[9] Regular clinic attendance (four times per year) is associated with better blood glucose control.[11]

The Ministry of Health's quality standards for diabetes care[12] state that: "young people with diabetes should have access to an experienced multidisciplinary team including developmental expertise, youth health, health psychology and dietetics". However, a 2012 survey which enquired about resourcing for services for children and young people with diabetes in New Zealand secondary care services found that, by international standards, New Zealand services were significantly under-resourced.[13] All centres were below the international recommendations for medical staff, there was wide variation in nurse educator numbers, and most centres did not have dedicated psychologists or dieticians.

Diabetes education

Children and young people with T1DM and their families need on-going education covering insulin therapy, blood glucose monitoring, the effects of diet, physical activity and intercurrent illness on blood glucose control, managing sick days, detecting and managing hypoglycaemia, hyperglycaemia and ketosis, and for adolescents, the effects of alcohol on blood glucose and the particular hazards of smoking, recreational drugs and pregnancy for people with diabetes.[9] The recent (2015) NICE guideline did not identify any RCTs that had evaluated the content of education programmes but noted that there are many discussion papers suggesting appropriate content for such programmes.[9]

Hospital admission

It is common for children and young people to be admitted to hospital soon after diagnosis of T1DM.[14] A significant proportion of children with diabetes have ketoacidosis at diagnosis and moderate to severe ketoacidosis necessitates hospitalisation for intravenous therapy.[14,15] There is some evidence suggesting that it is possible to receive initial management as an outpatient which does not lead to any disadvantages in terms of metabolic control, acute diabetic complications and hospitalisations, psychosocial variables and behaviour, or total costs.[14] Outpatient initial management is unlikely to be suitable for young children, children who live a long way from a hospital or children from families affected by complex social and/or emotional problems.[9]

Management of diabetes is complex. It can be expected that children with T1DM will experience complications of inadequate blood glucose control, such as hypoglycaemia and ketoacidosis, and may then require admission to hospital.[14,16] A recently published Welsh study looking at all-cause hospitalisation rates in a cohort of 1,577 children with newly-diagnosed T1DM (who were followed up for a total of 12,102 person years) found that these children had a rate of hospital admission almost five times higher than control children.[16] Ketoacidosis in children with established T1DM has been reported to be associated with insulin omission or treatment error in around 75% of cases and with inadequate insulin therapy during intercurrent illness in the remainder.[17] It has

been suggested that hospital admissions for ketoacidosis could be reduced by better supporting families of children with T1DM, especially those who find diabetes management challenging due to chaotic and dysfunctional family situations[18,19] but there is no good quality evidence to indicate which particular types of support are most effective.[9,20]

Evidence-based health care for children and young people with diabetes

These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of diabetes are provided for further reading.

Ministry of Health Publications and webpages

- Ministry of Health. 2016. Diabetic Retinal Screening, Grading, Monitoring and Referral Guidance. Wellington. http://www.health.govt.nz/system/files/documents/publications/diabetic-retinal-screening-grading-monitoring-referral-guidance-mar16.pdf
- Ministry of Health. 2015. Living well with diabetes: A plan for people at high risk off diabetes 2015−2020. Wellington: Ministry of Health. http://www.health.govt.nz/system/files/documents/publications/living-well-with-diabetes-oct15.pdf
- Ministry of Health. 2014. Quality standards for diabetes care. http://www.health.govt.nz/our-work/diseases-and-conditions/diabetes/quality-standards-diabetes-care
- Ministry of Health. 2014. Quality Standards for Diabetes Care Toolkit 2014. Wellington: Ministry of Health. http://www.health.govt.nz/publication/quality-standards-diabetes-care-toolkit-2014
- Ministry of Health. 2014. National Diabetes Work Programme 2014/15. Wellington: Ministry of Health. http://www.health.govt.nz/publication/national-diabetes-work-programme

International Guidelines

- American Diabetes Association. 2016. Standards of medical care in diabetes—2016. http://professional.diabetes.org/content/clinical-practice-recommendations/?loc=rp-slabnav
- National Institute for Health and Care Excellence. 2015. Diabetes (type 1 and type 2) in children and young people: diagnosis and management. https://www.nice.org.uk/Guidance/NG18
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- International Society for Pediatric and Adolescent Diabetes. 2014. ISPAD Clinical Practice Consensus Guidelines 2014. http://www.ispad.org/?page=ISPADClinicalPract
- Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. 2013. Canadian Diabetes Association 2013 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada. *Canadian Journal of Diabetes*, 37(suppl 1):S1-S212. http://guidelines.diabetes.ca/fullguidelines
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- Working Group of the Clinical Practice Guideline on Diabetes mellitus type 1. 2012. Clinical practice guideline for diabetes mellitus type 1. Vitoria-Gasteiz, Spain: Basque Office for Health Technology Assessment (Osteba). http://www.guiasalud.es/GPC/GPC_513_Diabetes_1_Osteba_compl_en.pdf
- Craig ME, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. 2011. National evidence-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing. https://diabetessociety.com.au/documents/Type1guidelines14Nov2011.pdf
- Klonoff DC, et al. 2011. Continuous glucose monitoring: an Endocrine Society Clinical Practice Guideline. *Journal of Clinical Endocrinology and Metabolism*, 96(10), 2968-79. http://jcem.endojournals.org/content/96/10/2968.long
- Scottish Intercollegiate Guidelines Network. 2010. Management of Diabetes: A national clinical guideline. Edinburgh: Scottish Intercollegiate Guidelines Network. http://www.sign.ac.uk/guidelines/fulltext/116/index.html

Recent Evidence-Based Medicine Reviews

- The Cochrane Library reviews relevant to diabetes:
  http://www.cochranelibrary.com/topic/Endocrine%20%26%20metabolic/Diabetes/?per-page=100&stage=review

- Edwards D, Noyes J, Lowes L, et al. 2014. An ongoing struggle: a mixed-method systematic review of interventions, barriers and facilitators to achieving optimal self-care by children and young people with type 1 diabetes in educational settings. *BMC Pediatrics*, 14 228.
  http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4263204/?report=classic

- Hynes L, Byrne M, Dinneen SF, et al. 2014. Barriers and facilitators associated with attendance at hospital diabetes clinics among young adults (15-30 years) with type 1 diabetes mellitus: a systematic review. *Pediatric Diabetes*, 17(7) 509–518. http://dx.doi.org/10.1111/pedi.12198

- Sheehan AM, While AE, Coyne I. 2015. The experiences and impact of transition from child to adult healthcare services for young people with Type 1 diabetes: a systematic review. *Diabetic Medicine*, 32(4), 440-58. http://onlinelibrary.wiley.com/doi/10.1111/dme.12639/abstract

Other Relevant Publications

- Jefferies C, Cutfield SW, Derraik JG, et al. 2015. 15-year incidence of diabetic ketoacidosis at onset of type 1 diabetes in children from a regional setting (Auckland, New Zealand). *Scientific Reports*, 5, 10358

- Jefferies C, Owens N, Wiltshire E. 2015. Care for children and adolescents with diabetes in New Zealand District Health Boards: Is the clinical resourcing ready for the challenge? *New Zealand Medical Journal*, 128(1424). https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2015/vol-128-no-1424-30-october-2015/6707

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- Obaid B, Britt E, Wallace-Bell M, et al. 2012. The demographics and prevalence of youth (15–24 year olds) with type 1 diabetes in the Canterbury District Health Board catchment area in 2010: has the prevalence changed since 2003? *New Zealand Medical Journal*, 125(1363), 22-8.
  https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2012/vol-125-no-1363/article-obaid

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- American Diabetes Association. 2012. Diabetes management at camps for children with diabetes. *Diabetes Care*, 35 Suppl 1, S72-5. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3632173/

Websites

- Health Quality and Safety Commission New Zealand. Diabetes. http://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/diabetes/

- Diabetes New Zealand http://www.diabetes.org.nz/home

- Diabetes Youth New Zealand http://www.diabetesyouth.org.nz/

- Australian Diabetes Society https://diabetessociety.com.au/

- British Society for Paediatric Endocrinology and Diabetes https://www.bsped.org.uk/

- Clinical Network for Children and Young People's Diabetes Services https://www.starship.org.nz/for-health-professionals/new-zealand-child-and-youth-clinical-networks/clinical-network-for-children-and-young-peoples-diabetes-services/

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3.   Callaghan BC, Little AA, Feldman EL, et al. 2012. Enhanced glucose control for preventing and treating diabetic neuropathy. *Cochrane Database of Systematic Reviews,*(6). http://dx.doi.org/10.1002/14651858.CD007543.pub2

4.   Kähler P, Grevstad B, Almdal T, et al. 2014. Targeting intensive versus conventional glycaemic control for type 1 diabetes mellitus: a systematic review with meta-analyses and trial sequential analyses of randomised clinical trials. *BMJ Open,* 4(8). http://dx.doi.org/10.1136/bmjopen-2014-004806

5.   Blasetti A, Chiuri RM, Tocco AM, et al. 2011. The effect of recurrent severe hypoglycemia on cognitive performance in children with type 1 diabetes: a meta-analysis. *Journal of Child Neurology,* 26(11) 1383-91. http://dx.doi.org/10.1177/0883073811406730

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16.   Sayers A, Thayer D, Harvey JN, et al. 2015. Evidence for a persistent, major excess in all cause admissions to hospital in children with type-1 diabetes: results from a large Welsh national matched community cohort study. *BMJ Open,* 5(4). http://dx.doi.org/10.1136/bmjopen-2014-005644

17.   Dunger DB, Sperling MA, Acerini CL, et al. 2004. European Society for Paediatric Endocrinology/Lawson Wilkins Pediatric Endocrine Society consensus statement on diabetic ketoacidosis in children and adolescents. *Pediatrics,* 113(2) e133-40. http://pediatrics.aappublications.org/content/113/2/e133.long

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WEIGHT AND EATING DISORDERS

Introduction

This section covers a broad spectrum of eating- and weight-related problems. Although traditionally viewed as separate entities, they are increasingly considered as parts of a continuum with common risk factors and scope for integrated prevention.[1-4]

Obesity is a condition of excess body fat in relation to lean body mass to the extent that it may have a negative effect on health.[5] Body mass index (BMI) is the measure commonly used to classify body weight, with sex and age-specific BMI cut-off points used to define thinness, overweight and obesity in children.[6] In contrast with adult health, there is currently a lack of scientific evidence on the relationship between specific BMI thresholds and any potential short- and long-term health risks in childhood.[5] Factors associated with an increased risk of developing childhood obesity include maternal over-nutrition, pre-term birth and infants being small or large for gestational age.[7,8] Children with developmental disabilities and autism also have higher prevalence of obesity than other children.[9,10] Obese children are at greater risk than other children of short- and long-term health problems including musculoskeletal problems, asthma, and psychological problems, and may develop abnormal lipid profiles, impaired glucose tolerance and high blood pressure at a younger age than non-obese peers.[6]

Eating disorders comprise a range of syndromes encompassing physical, psychological and social features, including anorexia nervosa and bulimia nervosa. Eating disorders can be chronic conditions with substantial long-term physical and social sequelae.[11] Onset is usually in adolescence, although is increasingly recognised at much younger ages.[1] Alongside asthma and obesity, eating disorders are among the most prevalent chronic conditions for adolescent girls.[2] Children and younger adolescents with eating disorders have a higher risk of rapid medical deterioration compared with older adolescents and adults. Young people are also at risk of potentially irreversible effects of physical and emotional development.[12] Major depression is commonly co-morbid with eating disorders and is associated with poorer prognosis.[13]

In the current obesogenic environment, unhealthy weight loss and muscle gaining behaviours have been observed in children as young as ten years.[3] Risk factors for both obesity and eating disorders include dieting (caloric restriction with the goal of weight loss), weight talk, weight teasing and body dissatisfaction.[1]

The following section uses data from the New Zealand Health Survey to describe the weight of 0–24 year olds and presents data on eating disorders from the New Zealand Health Survey, the National Minimum Dataset and the National Mortality Collection. The section concludes with brief overviews of evidence for good practice for these conditions and references to relevant literature including obesity-related in-depth topics published in NZCYES 2013 reports.[14]

Data sources and methods

Indicators

> Prevalence of underweight and overweight/obese individuals among 2–24 year olds
>
> Rates of eating disorders among 0–24 year olds

Definition

Prevalence of underweight and overweight/obese individuals among 2–24 year olds
BMI was calculated using measured height and weight. The extended international IOTF BMI cut-offs for underweight, healthy weight, overweight and obese categories are age and sex-specific.
Underweight: BMI less than 18.5
Healthy weight: BMI of 18.5–24.9
Overweight: BMI of 25.0–29.9

Obese: BMI of 30 or greater

Rates of eating disorders among 0–24 year olds

Hospitalisations of 0–24 year olds with an eating disorder per 100,000 population

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National trends and distribution

From 2009 to 2013 there were seven deaths of 0–24 year olds with obesity as an underlying cause, as documented within the National Mortality Collection.

Using the IOTF standard, the majority of 2–17 year olds had a BMI category of healthy weight (**Figure 14**). Among 16–24 year olds the proportion with healthy weight was significantly lower than the proportion with healthy weight in younger age groups, and the proportion who was obese was significantly higher than for those in the younger age groups (**Figure 15**). There was no significant difference by gender in either age group (**Figure 16**). Since 2006 there has been year to year variability in prevalence of thinness, overweight and obesity within the age groups. The greatest variability has been amongst 2–4 year olds (**Figure 17, Figure 18**).

Prevalence of underweight for 2–14 year olds was significantly lower for Māori compared with non-Māori and significantly higher for Asian compared with non-Asian (**Figure 19**; **Figure 20**).

The prevalence of overweight or obesity was higher for 2–14 year olds living in areas with the highest deprivation scores (NZDep2013 deciles 9–10) compared with those living in areas with the lowest NZDep2013 scores areas (deciles 1–2). There was no significant difference by gender in this age group. Prevalence of obesity was significantly higher among Pacific 2–14 year olds compared with non-Pacific and for Māori compared with non-Māori, and significantly lower for Asian compared with non-Asian (**Figure 21**, **Figure 22**).

Figure 14. Body mass index (BMI) category in 2–24 year olds, by age group and BMI category, NZ Health Survey 2014/15
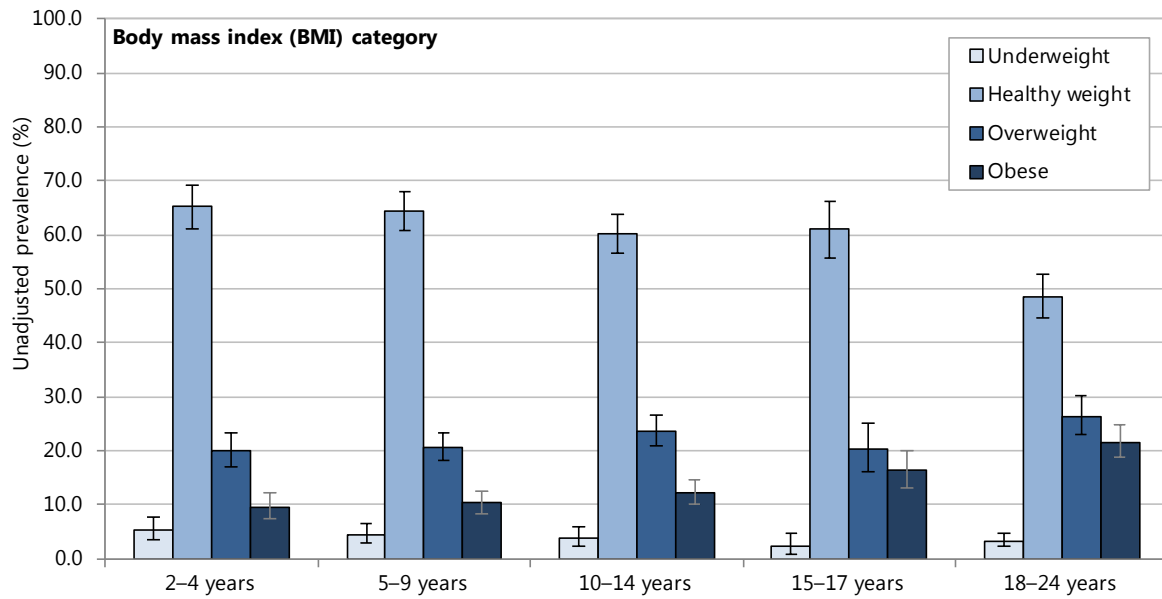
Source: NZ Health Survey

Figure 15. Body mass index (BMI) category in 2–24 year olds, by age group, NZ Health Survey 2014/15

Source: NZ Health Survey

Weight and eating disorders

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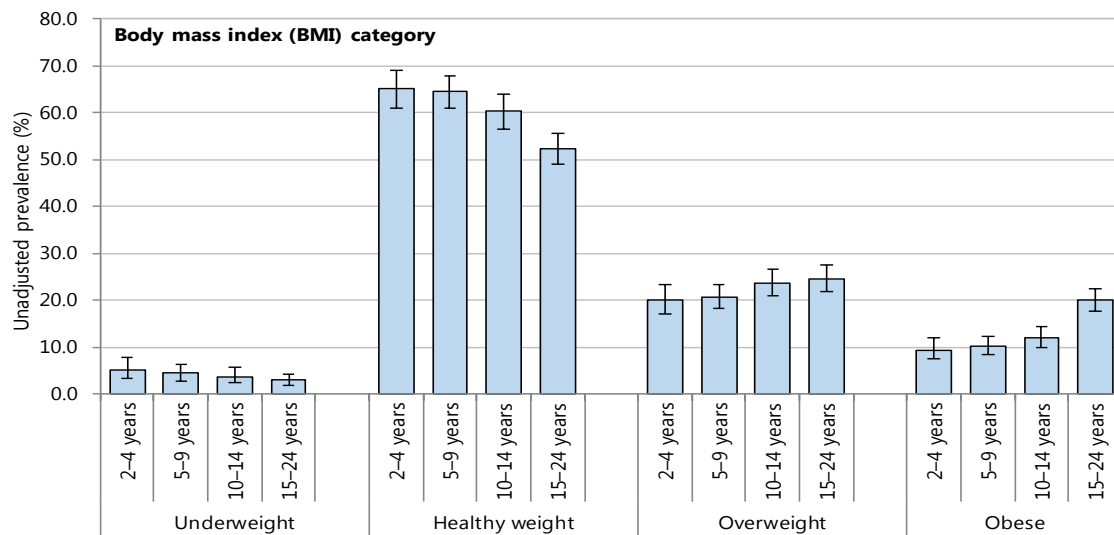
Figure 16. Body mass index (BMI) category in 2–24 year olds, by age group and sex, NZ Health Survey 2014/15
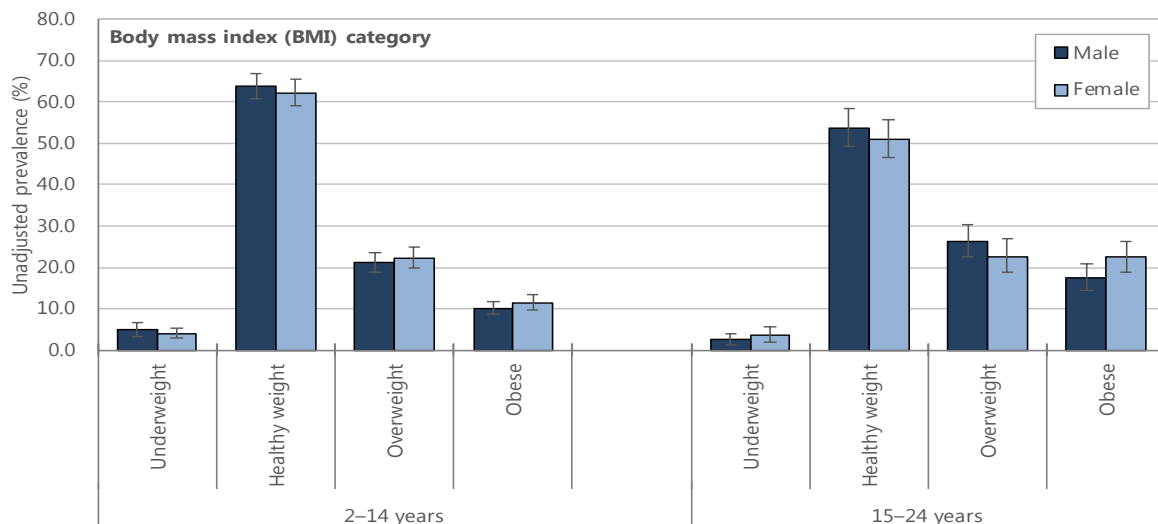
Source: NZ Health Survey

Figure 17. BMI: Underweight among 2–24 year olds, by age group and survey year, NZ Health Surveys 2006/07–2014/15
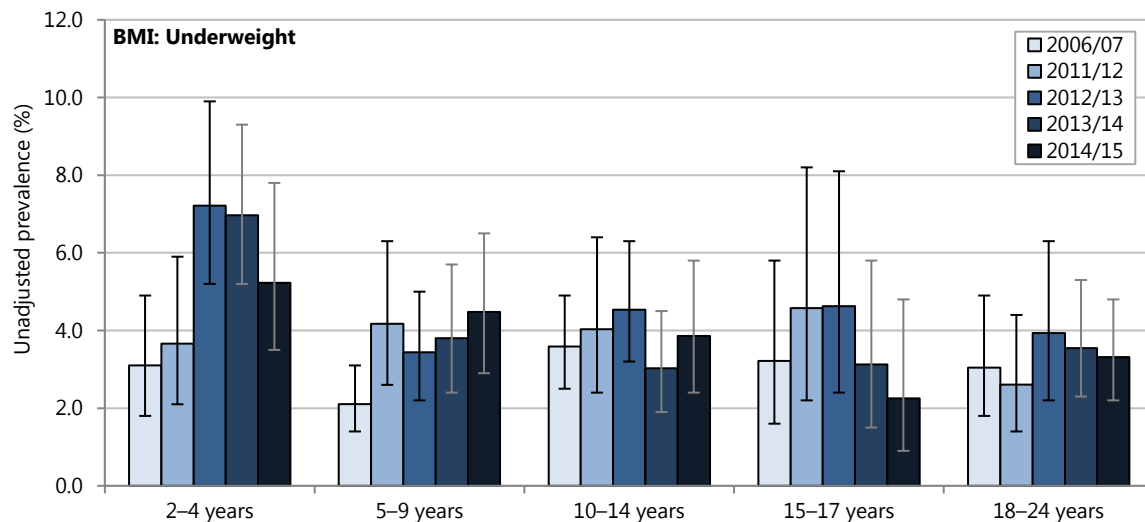
Source: NZ Health Survey

Figure 18. BMI: Overweight or obese among 2–24 year olds, by age group and survey year, NZ Health Surveys 2006/07–2014/15
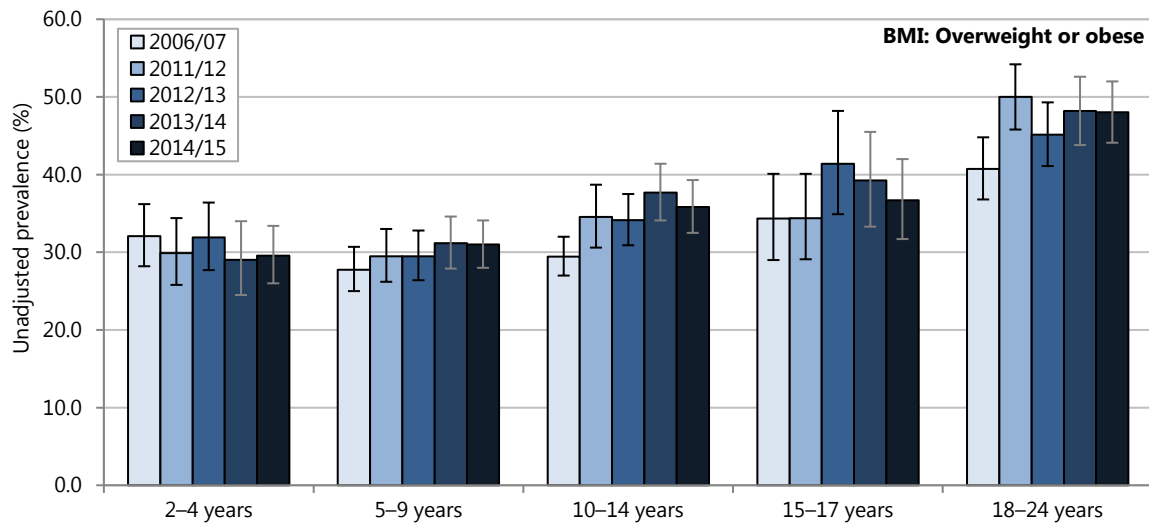
Source: NZ Health Survey

Figure 19. BMI: Underweight in 2–14 year olds, by ethnicity and sex, NZ Health Survey 2014/15
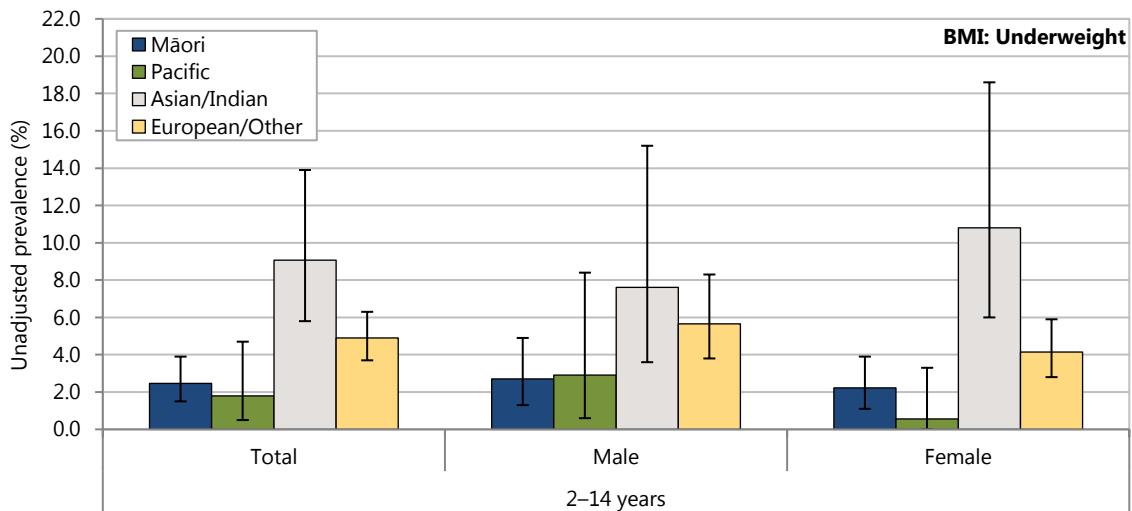
Source: NZ Health Survey. Ethnicity is total response

Figure 20. BMI: Underweight among 2–14 year olds, by demographic factor, NZ Health Survey 2014/15
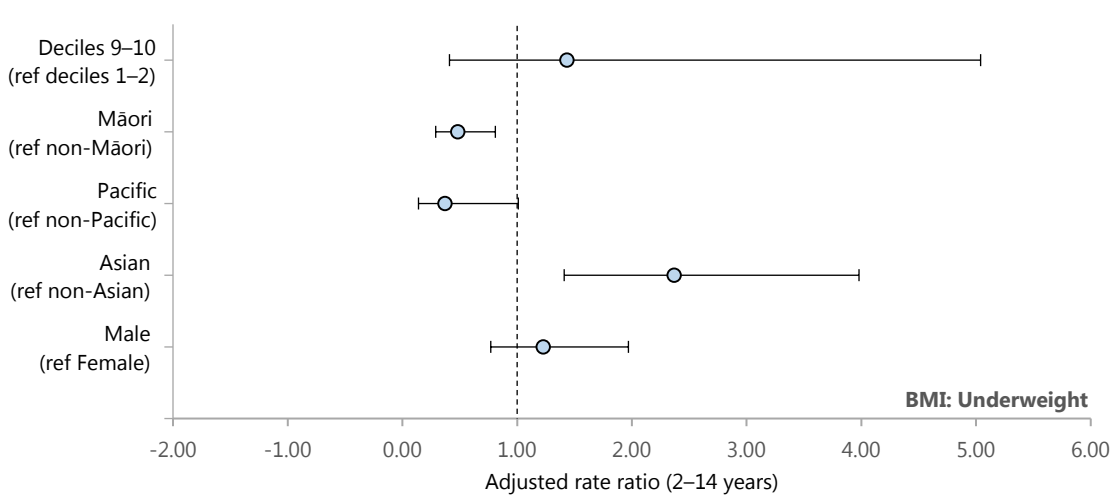
Source: NZ Health Survey. Underweight=children who are thin, with a BMI equivalent to an adult BMI of 18.5 or lower. Ethnicity is total response

Figure 21. BMI: Overweight or obese in 2–14 year olds, by ethnicity and sex, NZ Health Survey 2014/15
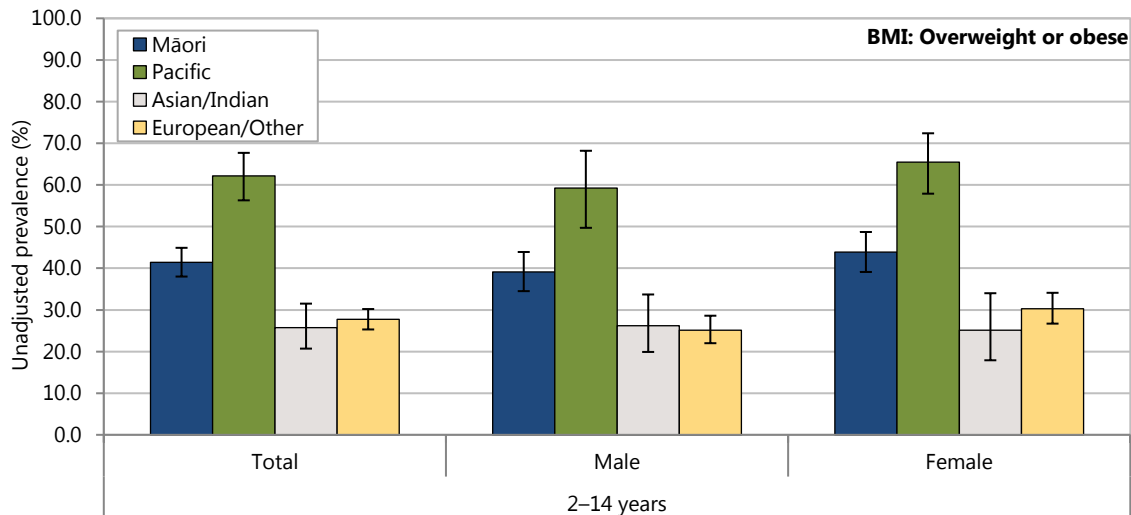
Source: NZ Health Survey. Ethnicity is total response

Figure 22. BMI Overweight or obese among 2–14 year olds, by demographic factor, 2014/2015 NZ Health Survey
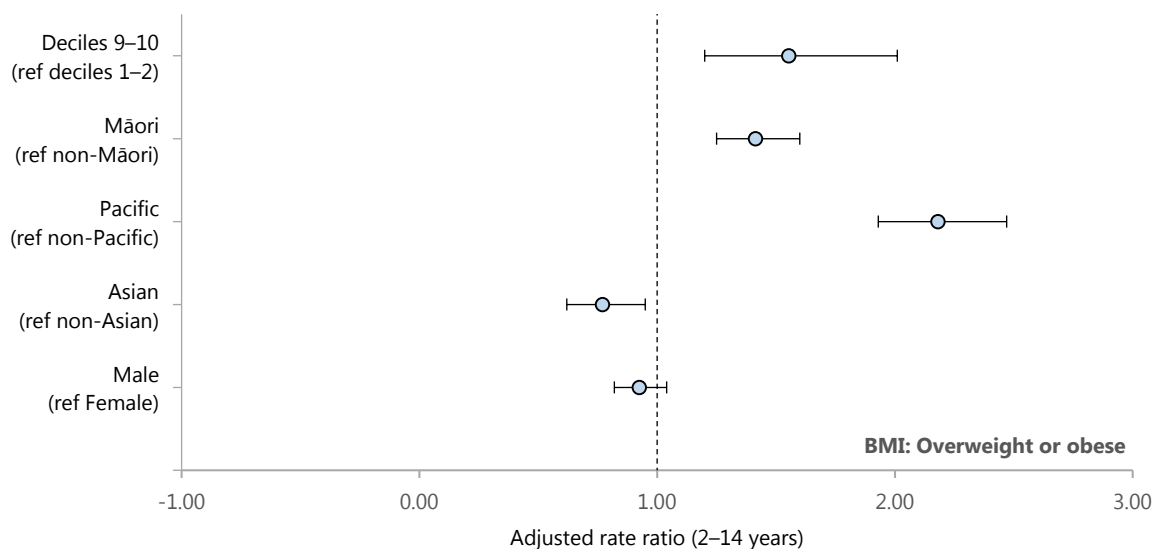
Source: NZ Health Survey. Children who are overweight or obese have a BMI equivalent to an adult BMI of 25.0 or greater. Ethnicity is total response

From 2009 to 2013 there were fewer than five deaths of 0–24 year olds with an eating disorder as an underlying cause, as documented within the National Mortality Collection.

Hospitalisation rates were higher for 15–24 year olds than 0–14 year olds (**Table 18**). Since 2000 hospitalisation rates for eating disorders have risen **(Figure 23)**.

Table 18. 0–24 year olds hospitalised with eating disorders using primary diagnosis compared to all cases, New Zealand 2011–2015

	Unique individuals (n)	Hospitalisations (n)		Ratio All : Primary
		Primary diagnosis	All cases	
Eating disorders				
Hospitalisation				
0–24 years	1,012	1,509	2,301	1.52
0–14 years	266	363	417	1.15
15–24 years	781	1,146	1,884	1.64
Eating disorders in 0–24 year olds				
Anorexia nervosa	632	1,174	1,467	1.25
Bulimia nervosa	145	90	264	2.93
Other eating disorders†	382	245	598	2.44

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with an eating disorder in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Figure 23. Hospitalisations for eating disorders in 0–24 year olds, New Zealand 2000–2015
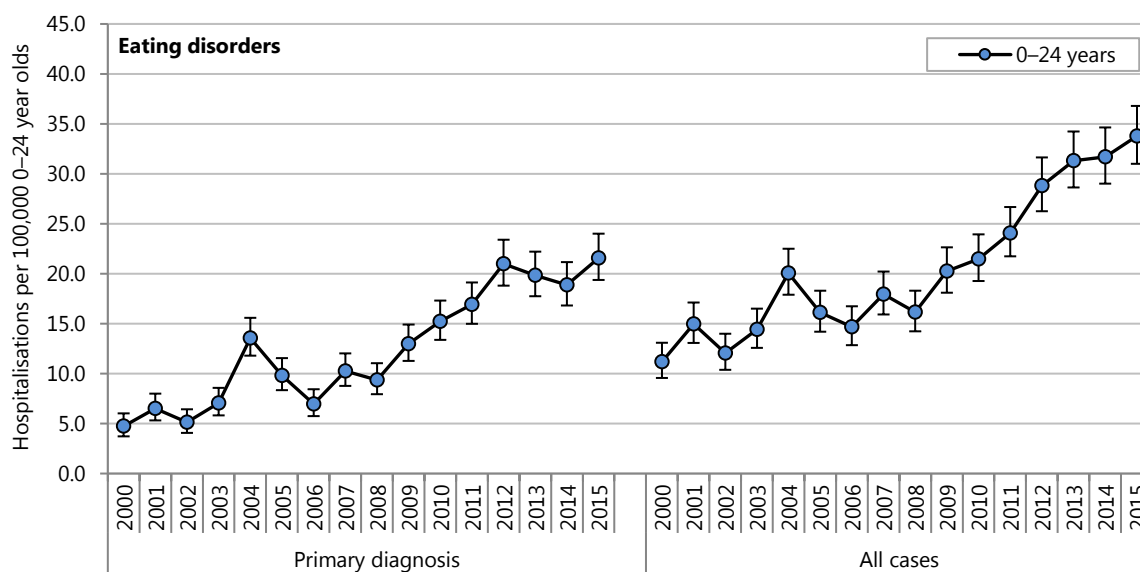
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. ''All cases' corresponds to hospitalisations with an eating disorder in any of the first 15 diagnoses

Diagnosis

Most hospitalisations of 0–24 year olds with eating disorders had Anorexia nervosa as the primary diagnosis, though other eating disorders and other mental and behavioural disorders also feature highly as primary diagnoses (**Table 19**).

Table 19. Hospitalisations involving eating disorders in 0–24 year olds, by primary diagnosis, New Zealand 2011– 2015

Primary diagnosis	2011–2015 (n)	Annual average	Rate	95% CI	%
Eating disorders* in 0–24 year olds					
New Zealand					
Anorexia nervosa	1,174	235	15.28	14.44–16.18	51.0
Bulimia nervosa	90	18	1.17	0.95–1.44	3.9
Other eating disorders	245	49	3.19	2.81–3.61	10.6
Eating disorders total	1,509	302	19.65	18.68–20.66	65.6
Other mental and behavioural disorders	389	78	5.06	4.59–5.59	16.9
Injury and/or poisoning	126	25	1.64	1.38–1.95	5.5
Endocrine, nutritional and metabolic diseases	105	21	1.37	1.13–1.65	4.6
Symptoms and/or abnormal clinical findings NEC	71	14	0.92	0.73–1.17	3.1
Other diagnoses	101	20	1.31	1.08–1.60	4.4
Total	2,301	460	29.96	28.76–31.21	100.0

Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population; * Eating disorder in any of the first 15 diagnoses; Rate per 100,000 0–24 year olds; NEC = not elsewhere classified

Table 20 presents the demographic distribution of individuals with eating disorders in New Zealand between 2011 and 2015. The prevalence of eating disorders was significantly lower among individuals residing in areas with higher deprivation scores (NZDep2013 deciles 3–10) compared to the lowest deprivation scores (deciles 1– 2), and significantly higher among 15–24 year olds compared to 0–14 year olds. The majority of 0– 24 year olds with eating disorders were of European/Other ethnicity.

Table 20. 0–24 year olds hospitalised with eating disorders by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Eating disorders* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	323	22.76	1.00	
Deciles 3–4	217	16.23	0.71	0.60–0.85
Deciles 5–6	217	15.05	0.66	0.56–0.79
Deciles 7–8	222	13.67	0.60	0.51–0.71
Deciles 9–10	148	7.97	0.35	0.29–0.43
Prioritised ethnicity				
Māori	76	4.21	0.20	0.16–0.25
Pacific	6	0.85	0.04	0.02–0.09
Asian/Indian	53	5.53	0.26	0.20–0.35
MELAA	10	9.92	0.47	0.25–0.88
European/Other	866	21.08	1.00	
Gender				
Female	918	24.45	1.00	
Male	94	2.39	0.10	0.08–0.12
Age group (years)				
0–4	9	0.58	0.02	0.01–0.04
5–14	257	8.61	0.35	0.30–0.40
15–24	781	24.90	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Eating disorders in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is level 1 prioritised; Decile is NZDep2013; Summation of components may equal more than the 0–24 year old unique total

Regional distribution

Figure 24 shows the percentage of 2–14 and 15–24 year olds in each DHB who were assessed as overweight, and **Figure 25** shows the percentage who were assessed as obese, in the 2011/12 to 2013/14 New Zealand Health Survey. Prevalence of overweight and of obesity were similar to the national prevalence in all DHBs. Prevalence of being assessed as overweight was significantly higher than the national prevalence for 2–14 year olds in Northland, Waikato, and Tairawhiti and for 15–24 year olds in Wairarapa. Prevalence of being assessed

as obese was significantly higher than the national prevalence for 2–14 year olds in Counties Manukau, Tairawhiti, Taranaki and Whanganui and for 15–24 year olds in Hawkes Bay.

Figure 24. BMI: Overweight in 2–24 year olds, by age group and district health board, NZ Health Survey 2011– 2014
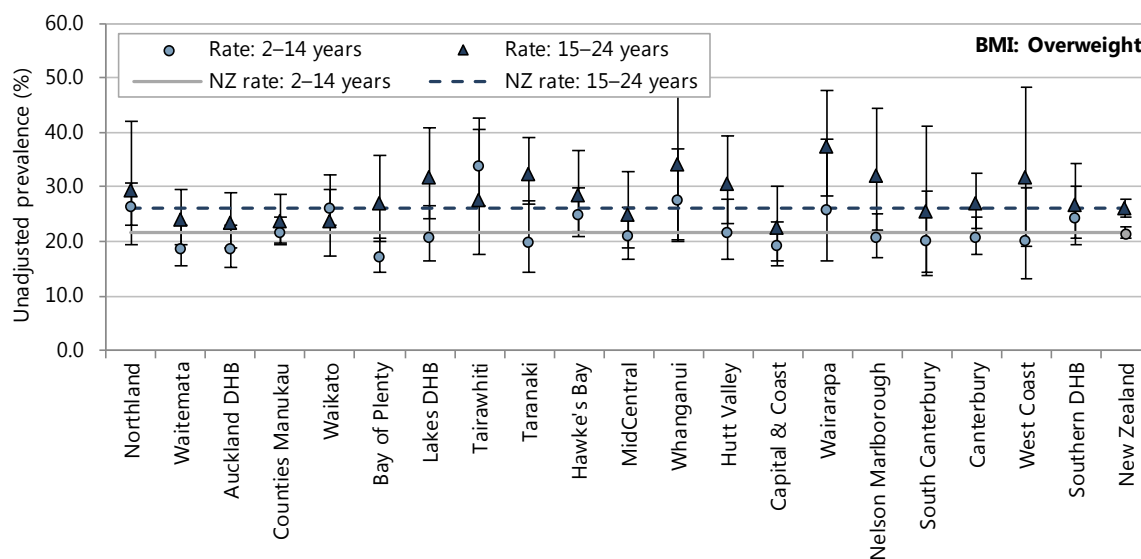
Source: NZ Health Survey

Figure 25. BMI: Obese in 2–24 year olds, by age group and district health board, NZ Health Survey 2011–2014
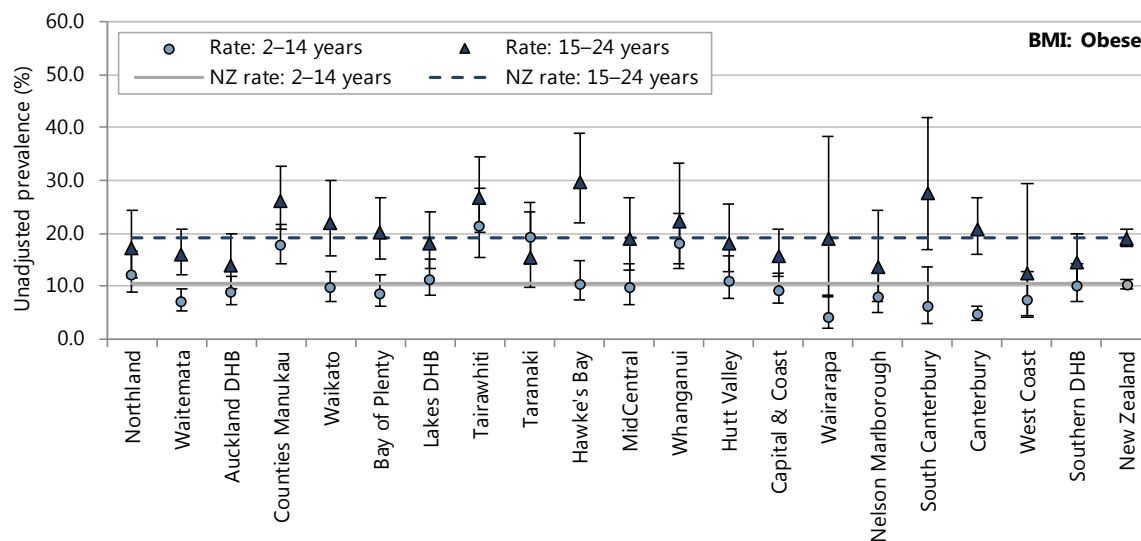
Source: NZ Health Survey

Table 21 presents the number of individuals resident in each district health board area that had an eating disorder diagnosis between 2011 and 2015. It also presents the number of hospital discharges in which an eating disorder was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and result in less undercounting than would occur if only the primary diagnosis is included in discharge data. For eating disorders, the All:Primary diagnosis ratio was particularly high in Taiarawhiti, and higher than the national ratio in Taranaki, Capital & Coast, Nelson Marlborough, Canterbury and Southland DHBs and lower in Northland, Waitemata, Auckland, Counties Manukau, Waikato, Bay of Plenty, Lakes, Hawke's Bay, Mid Central, Whanganui, Wairarapa and South Canterbury DHBs. Ratios were similar to the national ratio in the remaining district health boards (**Table 21**).

Table 21. Hospitalisations for eating disorders in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Eating disorders in 0–24 year olds				
Northland	30	49	71	1.45
Waitemata	119	216	311	1.44
Auckland	114	161	203	1.26
Counties Manukau	46	56	80	1.43
Waikato	48	82	119	1.45
Bay of Plenty	39	81	109	1.35
Lakes	14	22	26	1.18
Tairawhiti	8	5	15	3.00
Taranaki	17	15	27	1.80
Hawke's Bay	21	29	37	1.28
MidCentral	40	53	72	1.36
Whanganui	9	16	20	1.25
Hutt Valley	39	52	77	1.48
Capital & Coast	146	198	312	1.58
Wairarapa	11	19	27	1.42
Nelson Marlborough	29	49	77	1.57
South Canterbury	17	25	32	1.28
Canterbury	188	222	443	2.00
West Coast	9	14	21	1.50
Southern	105	144	221	1.53
New Zealand	1,012	1,509	2,301	1.52

Numerator: National Minimum Dataset. 'All cases' corresponds to hospitalisations with eating disorders listed in any of the first 15 diagnoses; Rate per 100,000 0–24 year olds; Rate ratios are unadjusted; Note that individuals may appear in multiple DHBs

Evidence for good practice

Possibilities for prevention

The increase in obesity worldwide over the past few decades suggests a key role for environmental determinants rather than changes in humans' basic genetic code.[8] Obesity prevention and treatment requires a whole-of-government approach in which policies across all sectors systematically take health into account, avoid harmful health impacts, and thus improve population health and health equity.[15] It is important that interventions to prevent obesity do not result inadvertently in disordered eating or increase weight stigmatisation.[1,2] From a social justice perspective BMI screening does not address underlying issues that lead to obesity, such as genetic predisposition and economic inequality, and nor does it ensure access to healthy food.[3] There is moderate evidence that health promoting schools (HPS) interventions seeking to reduce BMI and increase physical activity or fitness and fruit and vegetable intake have positive effects at an individual level with potential to produce public health benefits at the population level.[16] Conversely, the provision of sugary drinks in schools and in reward packets can be considered direct-to-consumer marketing of unhealthy, empty calories.[7] An integrated approach to prevention of both eating disorders and obesity will focus on sustainable, healthy, family-based lifestyle modification rather than on weight, promote a positive body image, encourage more frequent family meals, facilitate healthy eating and physical activity especially within the family, and address any history of maltreatment or bullying.[1] Different BMI thresholds and descriptors may be relevant for population surveillance compared with individual level clinical management.[5]

Evidence-based health care for children and young people with obesity or eating disorders

For children and young people identified as obese or overweight, family-based lifestyle interventions that include dietary, physical activity and behavioural components produce significant and clinically meaningful weight reductions in the short and the long term. Parental involvement is important, particularly for pre-adolescent children. It is not possible to say whether any one lifestyle intervention is better than any other.[14] Pharmacological treatment or bariatric surgery may be indicated, as part of a multidisciplinary treatment programme, for post-pubertal adolescents with severe obesity and associated severe co-morbidities.[17,18] Family members, including siblings, should also be included in the treatment of children and adolescents with eating

disorders.[11] Outpatient family-based treatment is the treatment of choice for children and adolescents with eating disorders, although some will need a period of management as an inpatient. Medical and nutritional stabilisation is the first and most important goal of inpatient treatment and this is usually necessary before psychological therapy can be effective.[12] Mindfulness training is associated with a decrease in disordered eating patterns.[19,20] When assessing outcomes of treatment it is important to use an appropriate rating scale that includes the perceptions of the child or young person. Qualitative studies of parents of children with health conditions have shown that the process of answering many personal and negative questions found in some instruments can have a negative impact.[21]

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AUTISM SPECTRUM DISORDER (ASD)

Introduction

Autism spectrum disorder (ASD) comprises a cluster of childhood onset neurodevelopmental conditions characterised by delays or difficulties in social communication and social interactions and restricted, repetitive patterns of behaviour, interests or activities.[1] The diagnostic criteria in the DSM-5 for ASD[1] list two dimensions which must be present. The first is a persistent impairment in reciprocal social communication and interaction, for example, the failure to engage in reciprocal conversations, lack of eye contact, and not understanding social context such as nonverbal communication. The second is inflexibility in thinking and behaviour, characterised by repetitive or stereotyped movements and ritualised patterns of behaviour.

Prevalence studies across developed countries have identified individuals with ASD with an average prevalence of between 1% and 2% but there is considerable variation between countries and studies.[2] There has been much less research on the prevalence of autism in adults but it appears to be similar to that in children.[3] The ratio of males to females is around 3:1 among those with the most severe forms of ASD and around 8:1 among those with less severe forms of ASD[4] According to the World Health Organization (WHO), the prevalence of ASD is increasing.[5] Changes in the diagnostic criteria, development in services and greater awareness of the condition may explain the increase that is being seen worldwide, although other factors, as yet unknown, may contribute.[6]

The manifestations of ASD vary considerably, in severity, and by developmental stage and age.[1,7] Among young children aged 1–3 years, a lack of development in language and play can become more obvious with increasing age and there can be a gradual or rapid deterioration of social behaviours or language.[8] Social and educational demands can increase difficulties for children aged 5–8 years and feeling socially isolated or having relationship difficulties is likely to be experienced by adolescents and adults with ASD.[7]

There are a number of genetic conditions associated with autism including Down syndrome, fragile X, muscular dystrophy, neurofibromatosis, and tuberous sclerosis.[9] Other conditions associated with autism include birth defects associated with central nervous system malformation and/or dysfunction, such as cerebral palsy, and premature birth.[9,10] Research has indicated that around 70% of people with ASD met the criteria for one or more other psychiatric disorders, for example ADHD or anxiety, although they may not have received a formal diagnosis of such a disorder.[9] About half of the children with autism have an intellectual disability with an IQ below 70.[9] Epilepsy is substantially more common in people with autism than in the general population, especially in those who also have intellectual disability.[11]

Experiencing discrimination and stigmatisation, including unjust deprivation of health, education and opportunities to participate in community, is common for people with ASD.[9] People with ASD have the same general health problems as the rest of the population and they may also have specific health care needs relating to their ASD or other co-occurring conditions.[9] Compared to the general population they have higher unmet need for health care.[9] One reason may be that health care providers lack sufficient knowledge of ASD.[9] Increased diagnosis is putting greater demands on diagnostic services and on services providing care and support. Caring for people with ASD can be a very heavy emotional and economic burden for their family, particularly for families caring for people with severe ASD where access to services and support are inadequate.[9]

The following section reviews ASD in children and young people using information from the New Zealand Health Survey and National Minimum Dataset. The section concludes with a brief overview of evidence for good practice in caring for children and young people with ASD.

Data sources and methods

Indicators

Prevalence of autism spectrum disorder (ASD)

Hospitalisations for ASD

Definition

Prevalence of autism spectrum disorder (ASD)

Diagnosed Autism Spectrum Disorder (including Asperger's Syndrome) (2–14 years) [12] Child respondents (aged 2–14 years) are defined as having autism spectrum disorder if the child's parents or caregivers had ever been told by a doctor that the child has autism spectrum disorder*

National trends and distribution

There were fewer than five deaths of 0–24 year olds with autism as the underlying cause of death in New Zealand from 2000 to 2013, as documented within the National Mortality Collection.

About one in a hundred children aged 2–14 years were reported to have received a diagnoses of ASD in the NZ Health Survey 2014/15.[13] **Figure 26** shows the percentage of children reported as having ever been diagnosed with ASD over the year of the NZ Health Surveys from 2006/07 to 2014/15. A greater percentage of children aged 5–9 years and 10–14 years were reported than those aged 2–4 years. The percentage for males was significantly higher than that for females (**Figure 27**).

Children aged 5–9 years and 10–14 years had higher rate of diagnosis than those aged 2–4 years, and European/Other children had higher rates than Māori, and both had higher rates than other ethnicities. There was little difference between the NZDep 2013 index quintile scores (**Figure 28**).

Males were more likely to be diagnosed with ASD than females (**Figure 29**) and of all the demographic factors collected, sex was the only factor that was statistically significantly different (**Figure 30**).

Figure 26. Autism Spectrum Disorder (diagnosed) in 2–14 year olds, by age group and survey year, NZ Health Surveys 2006/07–2014/15
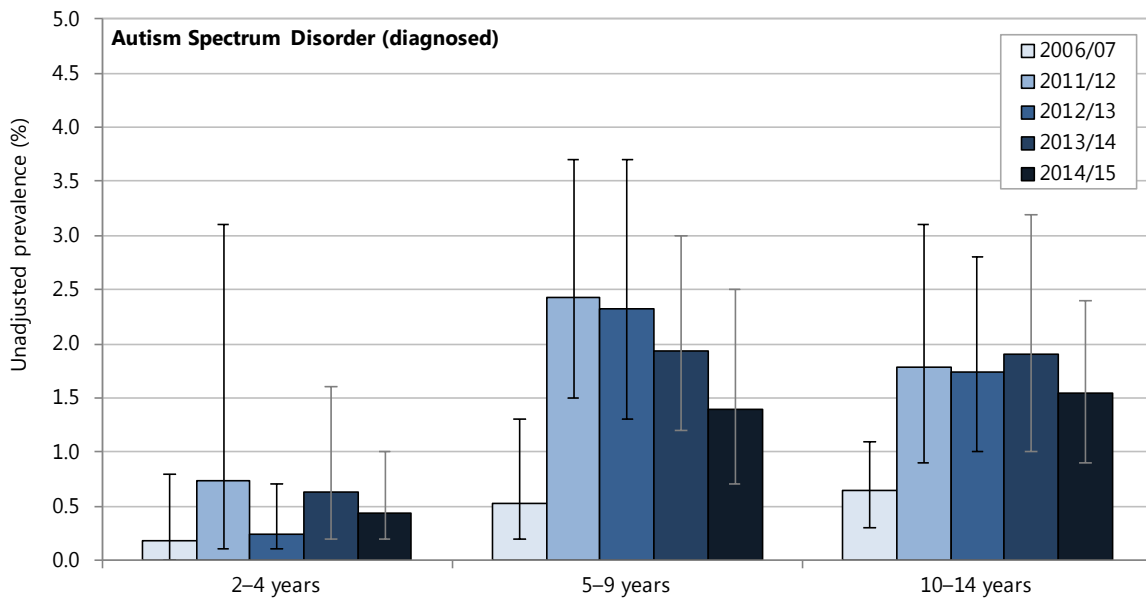
Source: NZ Health Survey; Diagnosed Autism Spectrum Disorder (including Asperger's Syndrome) (2–14 years); Percent of children (among children aged 2–14 years, by sex (unadjusted prevalence, 95% confidence intervals)

Figure 27. Autism Spectrum Disorder (diagnosed) in 2–14 year olds, by sex and survey year, NZ Health Surveys 2006/07–2014/15
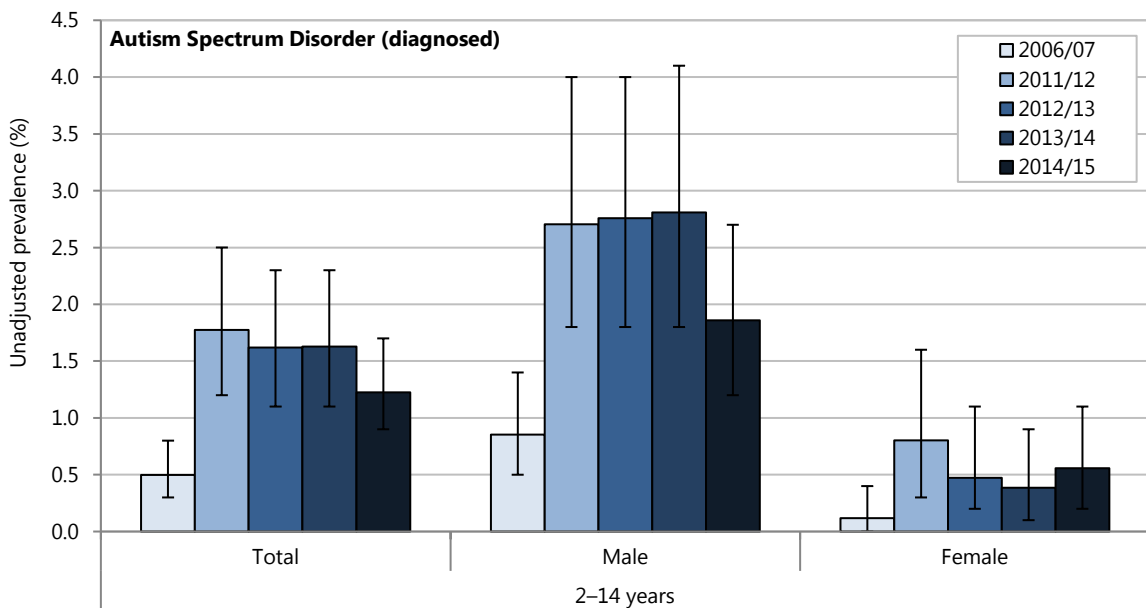
Source: NZ Health Survey; Diagnosed Autism Spectrum Disorder (including Asperger's Syndrome) (2–14 years); Percent of children (among children aged 2–14 years, by sex; (unadjusted prevalence, 95% confidence intervals)

Figure 28. Autism Spectrum Disorder (diagnosed), in 2–14 year olds, by demographic factor, NZ Health Survey 2014/15
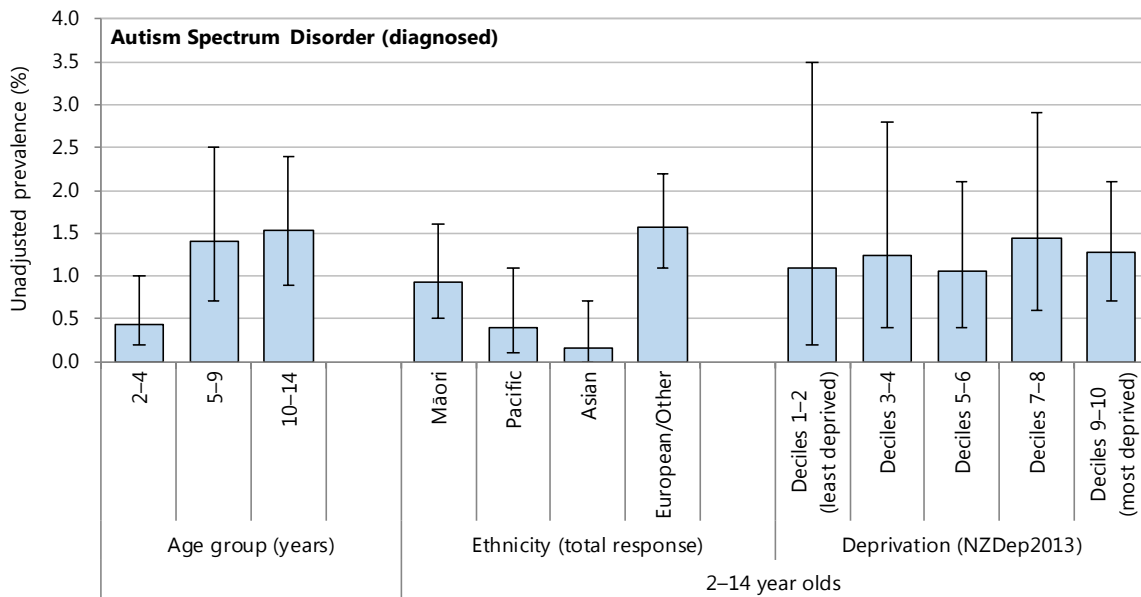
Source: NZ Health Survey; Diagnosed Autism Spectrum Disorder (including Asperger's Syndrome) (2–14 years); Percent of children (among children aged 2–14 years, by sex (unadjusted prevalence, 95% confidence intervals)

Figure 29. Autism Spectrum Disorder (diagnosed) in 2–14 year olds, by ethnicity and sex, NZ Health Survey 2014/15
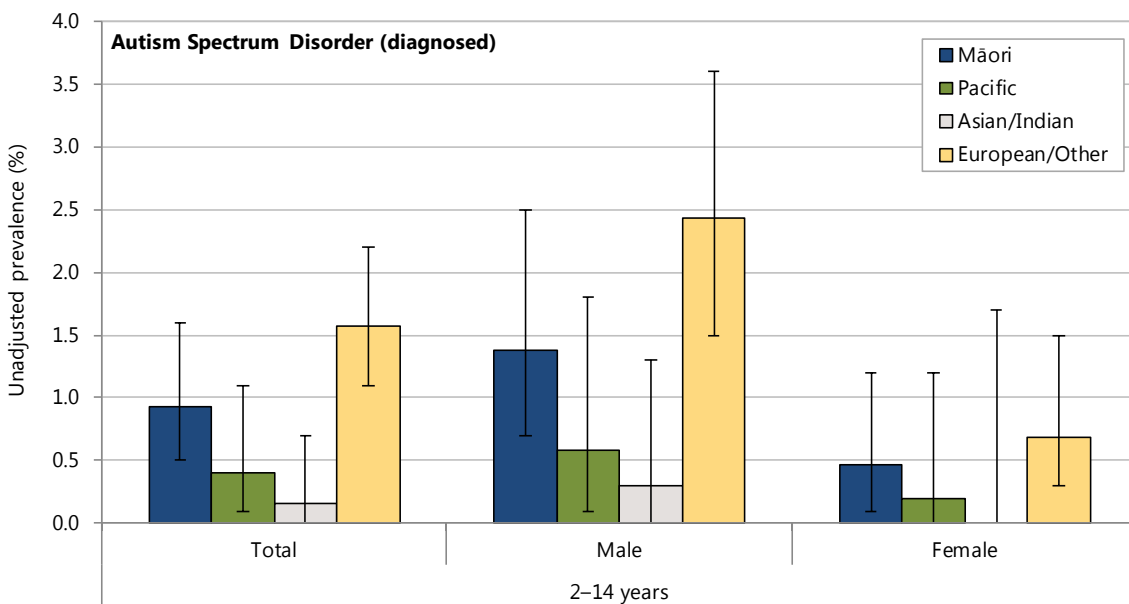
Source: NZ Health Survey; Diagnosed Autism Spectrum Disorder (including Asperger's Syndrome) (2–14 years); Ethnicity is total response

Autism spectrum disorder (ASD)

Figure 30. Comparisons for 2–14 year olds diagnosed with ASD, by demographic factor, NZ Health Survey 2014/15
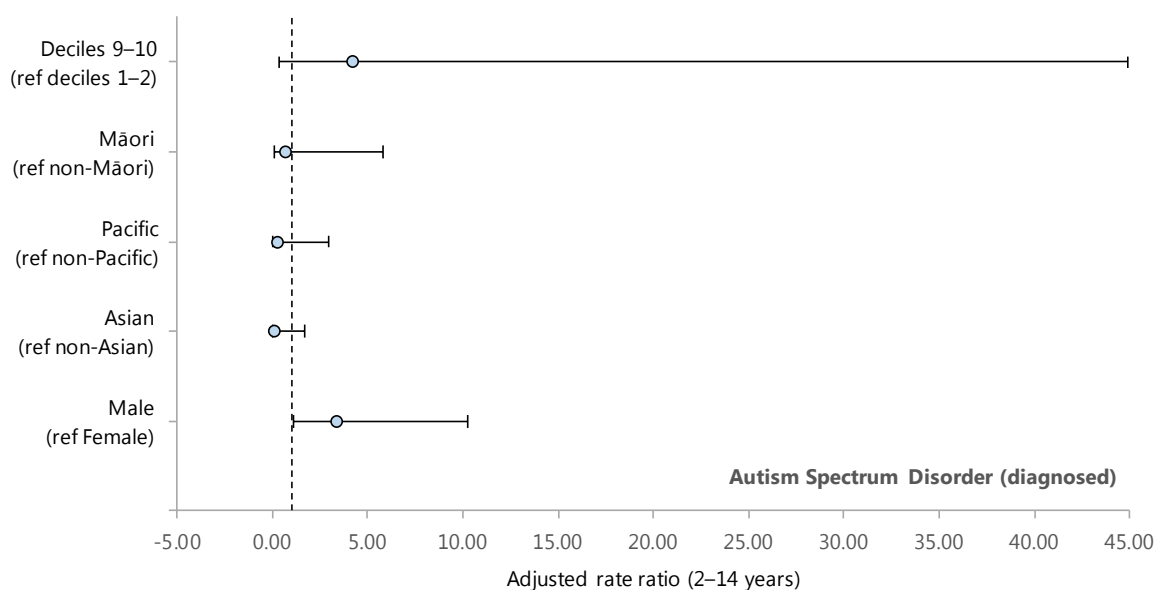
Source: NZ Health Survey; Diagnosed Autism Spectrum Disorder (including Asperger's Syndrome) (2–14 years); Comparisons for children (among children aged 2–14 years) by sex, ethnic group, neighbourhood deprivation, 2014/15 (adjusted rate ratios, 95% confidence intervals)

The number of 0–24 year olds hospitalised with autism or other pervasive developmental disorders (autism) between 2011 and 2015 is presented in **Table 22** together with the number of hospital discharges in which autism was documented as the primary diagnosis or as any diagnosis.

Table 22. Individuals hospitalised with autism, 0–24 year olds, New Zealand 2011–2015

Age group	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Autism				
Hospitalisation				
0–24 years	1,853	485	3,015	6.22
0–14 years	1,228	250	1,816	7.26
15–24 years	674	235	1,199	5.10

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with autism or other pervasive developmental disorder listed in any of the first 15 diagnoses; Note: The sum of the age groups may total to more than the 0–24 year old total

The rate of hospitalisations for autism has increased overall since 2000, particularly for 5–14 and 15–24 year olds. In all age groups the hospitalisation rate was consistently much higher where autism was documented within the first 15 diagnoses than for autism as the primary diagnosis (**Figure 31**).

Figure 31. Hospitalisations for autism in 0–24 year olds, by age group, New Zealand 2000–2015
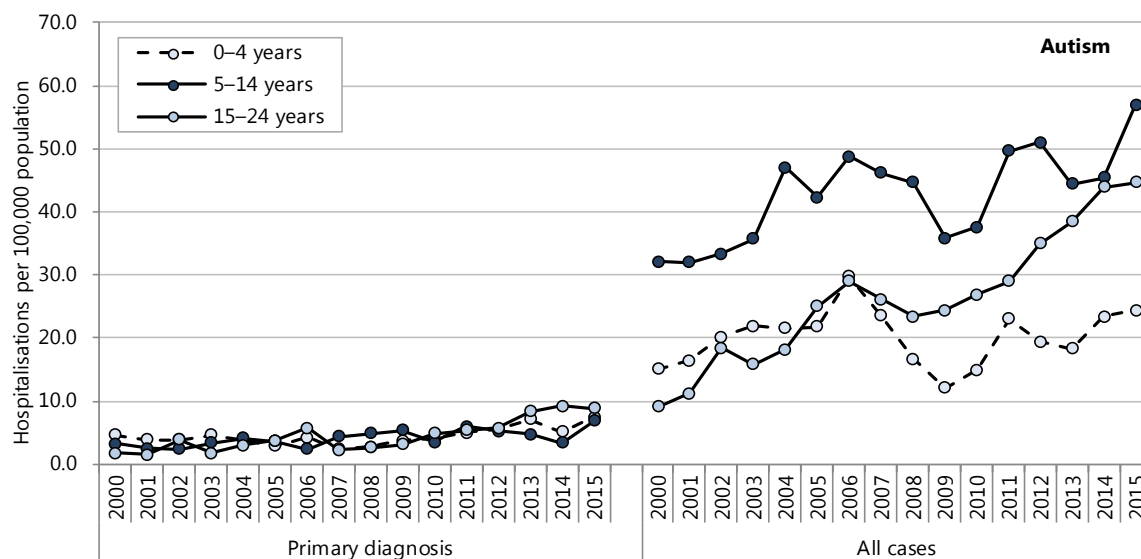
Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with autism or other pervasive developmental disorder listed in any of the first 15 diagnoses

Diagnosis

The most frequent primary diagnosis for 0–24 year olds hospitalised with any diagnosis of autism was dental caries. Only 16.1% of hospitalisations involving autism had autism or other pervasive developmental disorders as the primary diagnosis (**Table 23**).

Table 23. Hospitalisations involving autism in 0–24 year olds, by primary diagnosis, New Zealand 2011–2015

Primary diagnosis	2011–2015 (n)	Annual average	Rate per 100,000 0–24 year olds	95% CI	%
Autism* in 0–24 year olds					
New Zealand					
Childhood autism	340	68	4.43	3.98–4.92	11.3
Atypical autism	17	3	0.22	0.14–0.35	0.6
Rett syndrome	16	3	0.21	0.13–0.34	0.5
Asperger syndrome	83	17	1.08	0.87–1.34	2.8
Pervasive developmental disorders, other or unspecified	29	6	0.38	0.26–0.54	1.0
Total autism or other pervasive developmental disorders	485	97	6.31	5.78–6.90	16.1
Other mental and behavioural disorders	387	77	5.04	4.56–5.57	12.8
Dental caries	586	117	7.63	7.04–8.27	19.4
Other diseases of the digestive system	258	52	3.36	2.97–3.79	8.6
Other diagnoses	1,299	260	16.91	16.02–17.86	43.1
Total	3,015	603	39.25	37.88–40.68	100.0

Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population. * Autism or other pervasive developmental disorder in any of the first 15 diagnoses

Demographic distribution

Table 24 presents the demographic distribution of individuals with autism hospitalised in New Zealand between 2011 and 2015. There was a social gradient among these individuals with significantly higher hospitalisation rates in areas with higher NZDep2013 scores (NZDep deciles 3–4 to 9–10) compared with those living in areas with the lowest scores (deciles 1–2). Hospitalisation rates were significantly higher among males compared with females, and significantly lower for Māori, Pacific and Asian/Indian than for European/Other ethnic groups. Compared with 15–24 years olds, hospitalisation prevalence rates were significantly higher for 5–14 year olds and lower for 0–4 year olds.

Table 24. Individuals aged 0–24 years hospitalised with autism, by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (*n*)	Rate per 100,000 population	Rate ratio	95% CI
Autism* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	278	19.59	1.00	
Deciles 3–4	335	25.05	1.28	1.09–1.50
Deciles 5–6	355	24.63	1.26	1.07–1.47
Deciles 7–8	448	27.58	1.41	1.21–1.63
Deciles 9–10	574	30.89	1.58	1.37–1.82
Prioritised ethnicity				
Māori	391	21.68	0.82	0.73–0.92
Pacific	131	18.49	0.70	0.58–0.84
Asian/Indian	220	22.95	0.87	0.75–1.00
MELAA	35	34.71	1.31	0.94–1.84
European/Other	1,086	26.43	1.00	
Gender				
Female	465	12.38	1.00	
Male	1,388	35.35	2.85	2.57–3.17
Age group (years)				
0–4	275	17.63	0.82	0.71–0.94
5–14	998	33.44	1.56	1.41–1.72
15–24	674	21.49	1.00	

Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population. * Autism or other pervasive developmental disorder in any of the first 15 diagnoses; Rate ratios are unadjusted; Ethnicity is Level 1 prioritised; Decile is NZDep2013; Summation of components may equal more than the 0–24 year old unique total

Regional distribution

The 2011/12 to 2013/14 data for the district health boards shows that only Waitemata had a significantly higher percentage of ASD diagnosis than the New Zealand rate while Nelson Marlborough had a significantly lower percentage. Apart from Taranaki which had the highest percentage of all DHBs, most had very wide confidence intervals (**Figure 32**).

Figure 32. Autism Spectrum Disorder (diagnosed) in 2–14 year olds, by district health board, NZ Health Survey 2011/12 to 2013/14
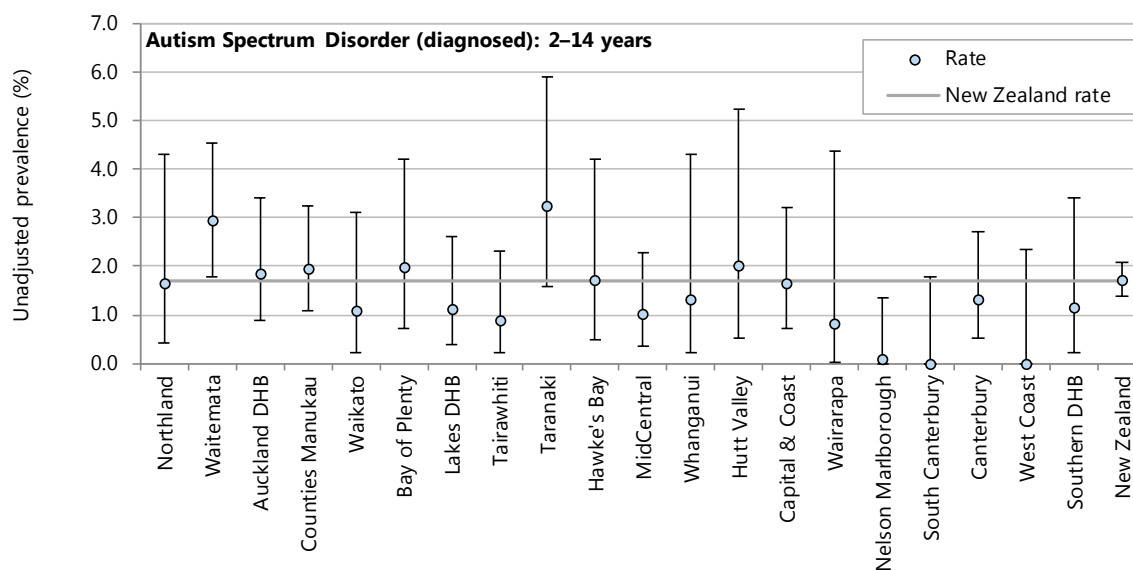
Source: NZ Health Survey

Table 25 presents the number of individuals resident in each district health board that had any diagnosis of autism or other pervasive developmental disorder (autism) between 2011 and 2015. It also presents the number of hospital discharges in which autism was documented as the primary diagnosis or as any diagnosis. The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and avoid undercounting that would occur if only primary diagnoses are included in discharge data. For autism the All:Primary diagnosis ratio was six nationally, and close to four or higher in all DHBs. Counting only hospitalisations with autism as the primary diagnosis will underestimate considerably the number and rate of hospitalisations of children and young people with this condition.

Table 25. Hospitalisations for autism in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Autism* spectrum disorder in 0–24 year olds				
Northland	52	19	73	3.84
Waitemata	267	61	395	6.48
Auckland	216	72	302	4.19
Counties Manukau	251	40	352	8.80
Waikato	140	25	191	7.64
Bay of Plenty	147	26	270	10.38
Lakes	40	<5	64	s
Tairawhiti	27	5	44	8.80
Taranaki	28	11	37	3.36
Hawke's Bay	59	9	136	15.11
MidCentral	105	11	149	13.55
Whanganui	63	9	103	11.44
Hutt Valley	52	24	87	3.63
Capital & Coast	99	39	147	3.77
Wairarapa	13	<5	17	s
Nelson Marlborough	71	18	137	7.61
South Canterbury	14	<5	29	s
Canterbury	140	69	277	4.01
West Coast	12	<5	15	s
Southern	91	34	180	5.29
New Zealand	1,853	485	3,015	6.22

Source: National Minimum Dataset. All cases' corresponds to hospitalisations with autism* listed in any of the first 15 diagnoses; * Autism or other pervasive developmental disorder; Note that individuals may appear in multiple DHBs; s = data suppressed due to small numbers

Evidence for good practice

Possibilities for prevention

Currently there is no evidence that any intervention can prevent ASD in the general population. It is thought that interaction between complex genetic and environmental factors is the cause of ASD as parents have a greater likelihood of having a subsequent child with ASD if a previous child has this condition and it is common for identical twins to both develop ASD.[10] Increasing maternal and paternal age is associated with increased autism risk.[14,15]

Maternal use of sodium valproate for the treatment of epilepsy and other neuropsychological disorders is associated with a significantly increased rate of autism in offspring, even after adjusting for the increased risk associated with maternal epilepsy.[16] The absolute risk is still small, however, so women need to weigh the benefits of treatment to control their epilepsy against the potential risks for their unborn child.[16] Folic acid supplementation may mitigate the risks associated with this medication.[17]

There is also an association between antidepressant use, particularly selective serotonin reuptake inhibitors (SSRI) in the second and/or third trimester of pregnancy, and increased risk of ASD in children, even after

considering risks associated with maternal depression. More research is needed to assess the risks associated with different type of antidepressant.[18]

Current research is exploring factors that may potentially increase the risk of ASD in offspring, such as maternal infection,[19] mitochondrial dysfunction,[20] and possible overlap between risk genes for ASD, schizophrenia and bipolar disorder.[21]

Evidence-based care for children and young people with ASD

While there is no cure for ASD, interventions can help improve the quality of life for children with ASD in relation to some of the features, symptoms, behaviours and problems commonly associated with the condition.[22] Due to the heterogeneous nature of ASD no single intervention can be expected to work for all people with ASD.[5] Programmes that may be effective include behavioural therapy, educational interventions, speech therapy, occupational therapy, social skills therapy, and medication (for problems like attention, hyperactivity and sleep).[5]

Early diagnosis is important for children with ASD because early intervention may improve prognosis and because families can then be linked to information and support services.[23] Although the clinical diagnosis of ASD is based on behavioural criteria, a thorough diagnostic evaluation may detect comorbidities that have implications for the diagnosis, treatment and prognosis not only of the child himself or herself, but also, in the event a genetic disorder such as fragile X is identified, for other family members including future siblings.[23]

Early intervention for communication, care and support

The quality of life for children with ASD is improved by early interventions to promote optimal development and wellbeing.[5] Efficacious interventions that address communication, social behaviour and behaviour inflexibility through psycho-educational, developmental, and behavioural methods are very labour intensive and therefore costly.[24]

Interventions should support both the individual with ASD and their family and carers.[5] A recent Cochrane review reported sufficient evidence of the effectiveness of parent-mediated interventions in treatment of ASD in young children. Child outcomes such as language improved when individual or groups of parents or carers were trained by professionals to be more observant and responsive during interactions with their child and improved communication skills decreased some of their other ASD related difficulties.[25]

It is good practice for local service providers to use approaches that facilitate parent participation in therapies.[25] Combining parent-mediated interventions with other locally available services can reduce the burden on parents. Non-specialists in school, family and community settings should task-share to deliver psychosocial interventions as this can increase access to care in low-resource settings. Changes that make the physical, social, and attitudinal environments more accessible, inclusive and enabling complement interventions for individuals with ASD.[24]

Music therapy

Music therapy has been shown to be better than a placebo, or standard care, for social interaction, non-verbal and verbal communication skills, initiating behaviour and social emotional reciprocity.[26] It is also better for social adaptation, joy, and quality of parent-child relationships.[26] There were no negative side effects. It is best delivered by specialists with academic and clinical training.[26]

Early intensive behavioural intervention (EIBI)

Early intensive behavioural intervention (EIBI) is widely used for increasing functional behaviours and skills in young children with ASD. It is based on the principles of applied behaviour analysis and delivered over multiple years at an intensity of 20 to 40 hours per week. There have been very few RCTs of EIBI but limited low-quality evidence suggests that children who received EIBI performed better than control children after 1-3 years of treatment on tests of adaptive behaviour, intelligence, social skills, communication and language, autism symptoms and quality of life.[27]

Assessment of ASD

A systematic review of tools used for measuring outcomes in anxiety interventions studies for children with ASD examined studies in which at least half the participants were aged 8–14 years.[28] Most studies were with children with high functioning ASD. The studies had small sample sizes but the review authors concluded that there is encouraging evidence that cognitive behavioural therapy (CBT) can be efficacious for children with ASD and anxiety disorder. Three questionnaires were considered to be robust: Spence Children's Anxiety Scale

(revised), the Revised Children's Anxiety and Depression Scale and the Screen for Child Anxiety Related Emotional Disorders.

Assessment tools for anxiety are designed for typically developing children and young people, and there has been little discussion about whether these are appropriate for young people with ASD.[29] Based on available research, and a clinical consensus process where data were lacking, a set of recommendations has been developed to assist primary care providers with the assessment and treatment of anxiety in children with ASD.[30] This research has resulted in two sets of recommendations, the first for the assessment of anxiety, as a systematic approach is needed to evaluate symptoms and factors such as the stage of development of the child. The second set of recommendations address the treatment of ASD associated anxiety, including coordination of care, education, modified cognitive behavioural therapy, and with care, possibly medication.

Childhood IQ is a reliable predictor of cognitive functioning in mid to later adulthood.[31,32] In people with higher IQ childhood scores, there appears to be greater IQ stability over time, however, even with an IQ that is above average, social outcomes in later life are generally poor.[31] A review of tools to measure outcomes for young children with ASD has recently been published.[33] It found that it is not yet possible to recommend fully robust tools and that there are gaps in outcome measurement tools for assessing the results of intervention studies, wellbeing and participation outcomes, and family quality of life outcomes, which are domains particularly valued by the review's informants (young people with ASD and parents).

Treatment for anxiety

It has been estimated that about 50% of children with ASD meet the criteria for at least one anxiety disorder.[34] A number of systematic reviews of treatments for anxiety in children and young people with ASD have been undertaken in recent years[29,34,35] yet there is a paucity of evidence for effective short and long term treatments.[29] The lack of large RCTs examining psychopharmacological treatment is of concern particularly given the concerns regarding adverse effects associated with certain selective serotonin reuptake inhibitors (a class of anti-depressant commonly used to treat anxiety). These include agitation, impulsivity, insomnia and disinhibition without manic symptoms.[29] There is potentially a problem with over prescribing, given the level of adverse effects.[29]

There is evidence that CBT is efficacious in achieving moderate improvements in a range of outcome measures in youth with high functioning ASD and anxiety.[34,35] In the absence of manuals specific to anxiety in ASD, the standard CBT treatment manuals for typically developing young people may be used if adapted according to the recommendations for ASD-specific content modifications that have been developed by the UK's National Institute for Clinical Excellence (NICE).[22,35] Cognitive behavioural therapy can be delivered in individual or group sessions, with or without parents.[34] Most studies of CBT have found it to be to be at least promising.[29] Although around 70% of youth with ASD and anxiety responded to CBT in research studies, the same success rate may not be achieved in clinical practice where compliance may be lower and individuals miss sessions thereby interrupting skill acquisition.[29] It is important that CBT is delivered by trained and experienced practitioners. There are limitations to the evidence base, especially related to small sample sizes and heterogeneity and there is a need for further research on a range of issues relating to the use of CBT in people with ASD.[36]

Interventions to reduce problem behaviours such as irritability and aggression

Mental health and behavioural problems are more prevalent in children with ASD than typically developing children.[37] Tantrums and rages may become chronic and disabling and limit opportunities for education and recreation.[37] They may also result in inpatient psychiatric care or residential placement.[37] Early intervention to reduce disruptive, aggressive and self-injurious behaviour is likely improve cognitive functioning as an adult.[32] CBT does not appear to be an effective intervention for outwardly-directed aggression in children with intellectual disabilities.[38]

A multidisciplinary team sponsored by the Autism Intervention Research Network on Physical Health and Autism Speaks Autism Treatment Network have developed a practice irritability and aggression pathway for primary care practitioners caring for children with ASD.[37] It has not yet been tested in primary health care settings.

The atypical antipsychotics, particularly risperidone and aripiprazole, are effective in reducing irritability, stereotypical behaviours and hyperactivity.[39,40] They are the only two medications approved by the US FDA for treating aggression, self-injury and tantrums in children with ASD.[40] They are commonly associated with metabolic adverse events, including weight gain and dyslipidaemia.[40] Methylphenidate is effective in reducing attention-deficit hyperactivity disorder (ADHD) symptoms in children with ASD and ADHD.[40] Atomoxetine

and alpha-2 agonists appear effective in reducing ADHD symptoms.[40] Selective serotonin reuptake inhibitors do not reduce repetitive behaviours in children with ASD, and often cause adverse events.[40] The efficacy of antiepileptic drugs is inconclusive.[40] The efficacy and tolerability of pharmacotherapy in children with ASD are generally less favourable than in typically developing children with similar symptoms. Newer agents, including glutamatergic agents and oxytocin, appear promising but results from trials have been mixed. [40]

Behavioural interventions combined with anti-psychotic medication may be more effective in treating aggression in people with ASD than either intervention alone.[41,42]

ASD and sleep
The prevalence of sleep difficulties among children with ASD has been estimated to be from 50% to 80%. Medications for sleep problems that are commonly used in children with ASD include melatonin, α-agonists, anticonvulsants, antidepressants, atypical antipsychotics, and benzodiazepines.[43] Although medication may improve sleep in the short term this can be at the cost of worsening daytime behaviour.[43] Further research is needed to develop evidence-based interventions for promoting night time sleep in children with ASD.[43]

Evidence-based health care for children and young people with autism
These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of autism are provided for further reading.

New Zealand guidelines
- Ministries of Health and Education 2016 New Zealand Autism Spectrum Disorder Guideline (2nd edn) Wellington: Ministry of Health. http://www.health.govt.nz/publication/new-zealand-autism-spectrum-disorder-guideline

International guidelines
The National Institute for Health and Care Excellence (NICE)

- NICE Pathway – Autism spectrum disorder: https://pathways.nice.org.uk/pathways/autism-spectrum-disorder

- NICE: Recognition, referral and diagnosis of autism in children and young people from birth to 19 years (clinical guideline 128): https://www.nice.org.uk/guidance/cg128

- NICE: Autism spectrum disorder in under 19s: support and management (clinical guideline 170). https://www.nice.org.uk/guidance/cg170

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Evidence-based medicine reviews

Cochrane reviews
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Other reviews
- Anagnostou E, Zwaigenbaum L, Szatmari P, et al. 2014. Autism spectrum disorder: advances in evidence-based practice. *CMAJ: Canadian Medical Association Journal,* 186(7) 509-19. http://dx.doi.org/10.1503/cmaj.121756

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Websites
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- Kidshealth (New Zealand site) http://kidshealth.org/en/teens/autism.html#

- Ministry of Health. How is ASD diagnosed? http://www.health.govt.nz/publication/how-asd-diagnosed

- Ministry of Education: ASD information supporting children and young people with ASD and resources for working with students who have ASD http://www.education.govt.nz/school/student-support/special-education/supporting-children-and-young-people-with-autism-spectrum-disorder-asd/

- Altogether Autism (A free, nationwide ASD information and advisory service in New Zealand) http://www.altogetherautism.org.nz/

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EMOTIONAL OR BEHAVIOURAL PROBLEMS

Introduction

Good mental health, as indicated by social and emotional well-being, is fundamental for healthy child development and success in school and in later life.[1] Mental health problems, which in children are usually manifested as emotional and behavioural problems, are estimated to affect 10–20% of children.[2-6] Common mental health conditions in children include anxiety, depression, attention deficit hyperactivity disorder (ADHD), behaviour disorders such as conduct disorder and oppositional defiant disorder, and substance use disorders.[3,4]

Mental health problems that arise in childhood can have consequences throughout the rest of life that are burdens not only for the individuals affected but also for families, communities, and the health, justice and welfare systems.[7] Without treatment, only half of preschool children grow out of behaviour problems.[8] Half of all lifetime mental disorders are apparent by age 14 and three-quarters by age 24.[9] It is both more effective and less costly to address mental health problems early in life, than to attempt to fix long standing problems later in life that have resulted from complex interactions between mental health difficulties, family breakdown, employment problems and drug and alcohol abuse.[10]

The following section provides data from the New Zealand Health Survey for children with emotional or behaviour problems and children with ADD/ADHD (Attention Deficit Disorder/Attention Deficit and Hyperactivity Disorder) using data from the New Zealand Health Surveys.

Data sources and methods

Indicators

Prevalence of emotional or behavioural problems
Prevalence of ADD/ADHD

Definition

Diagnosed emotional or behavioural problems (depression, anxiety disorder, ADD and/or ADHD) (2–14 years)
Child respondents (aged 2–14 years) are defined as having emotional or behavioural problems if the child's parents or caregivers had ever been told by a doctor that the child has depression, anxiety disorder (this includes panic attack, phobia, post-traumatic stress disorder, and obsessive-compulsive disorder), attention deficit disorder (ADD) or attention deficit hyperactivity disorder (ADHD).

Data sources

New Zealand Health Survey (2006/07–2014/15)

Additional information

This definition is likely to underestimate the true number of children with emotional or behavioural problems, as some people may not be aware that their child has a mood or anxiety disorder, and also to underestimate the true number of children with ADD or ADHD, as some people may not be aware that their child has ADD or ADHD.
Not all of the respondents who have ever had depression, anxiety disorder, ADD or ADHD would meet the criteria for depression, anxiety disorder, ADD or ADHD at the time they were surveyed.

National trends and distribution

Over the five years of the NZHS 2006/07 to 2014/15, the percentage of children aged 2–14 years who were ever diagnosed with an emotional or behaviour problem is lower for 2–4 year olds than for those older, with percentages for 5–9 year olds slightly lower than those of 10–14 year olds (**Figure 33**). Over the five years of the NZHS 2006/07 to 2014/15, the percentage of children aged 2–14 years who were ever diagnosed with ADD/ADHD (Attention Deficit Disorder/Attention Deficit and Hyperactivity Disorder) is lower for 2–4 year olds, with percentages for 5–9 year olds slightly lower than those of 10–14 year olds (**Figure 34**).

Data from NZHS for one year, 2014/15 shown in **Figure 35**, indicates that the percentages of children aged 5–9 and 10–14 years with emotional or behavioural problems were significantly higher than for 0–4 year olds. The percentages for Māori and European are higher than for Pacific and Asian.

Figure 33. Emotional or behavioural problems (diagnosed) in 2–14 year olds, by age group and survey year, NZ Health Survey 2006/07–2014/15
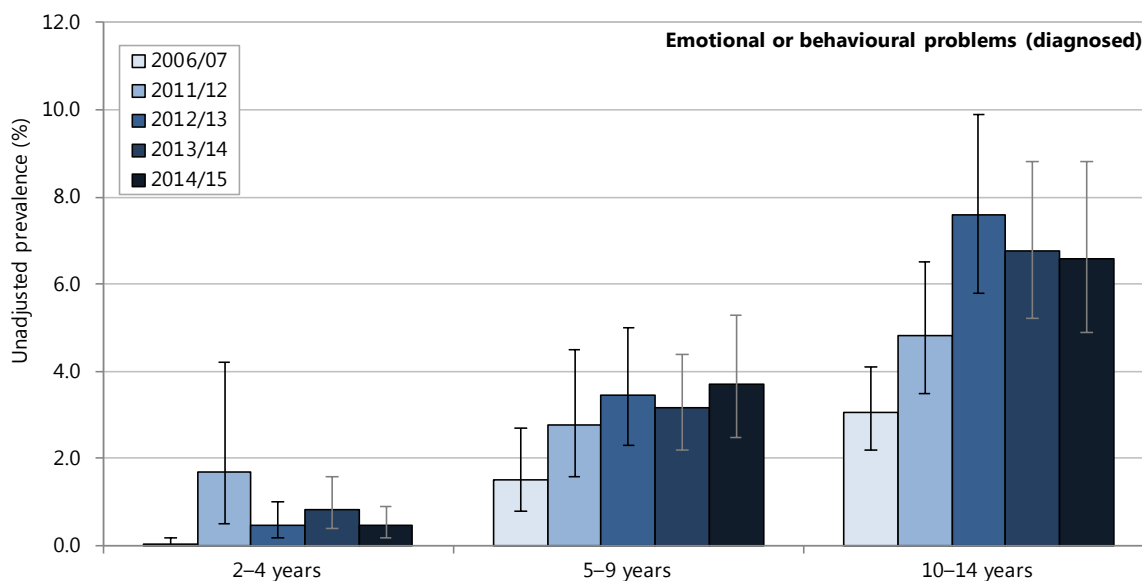
Source: NZ Health Survey

Figure 34. ADD or ADHD (ever diagnosed) in 2–14 year olds, by age group and survey year, NZ Health Survey 2006/07–2014/15
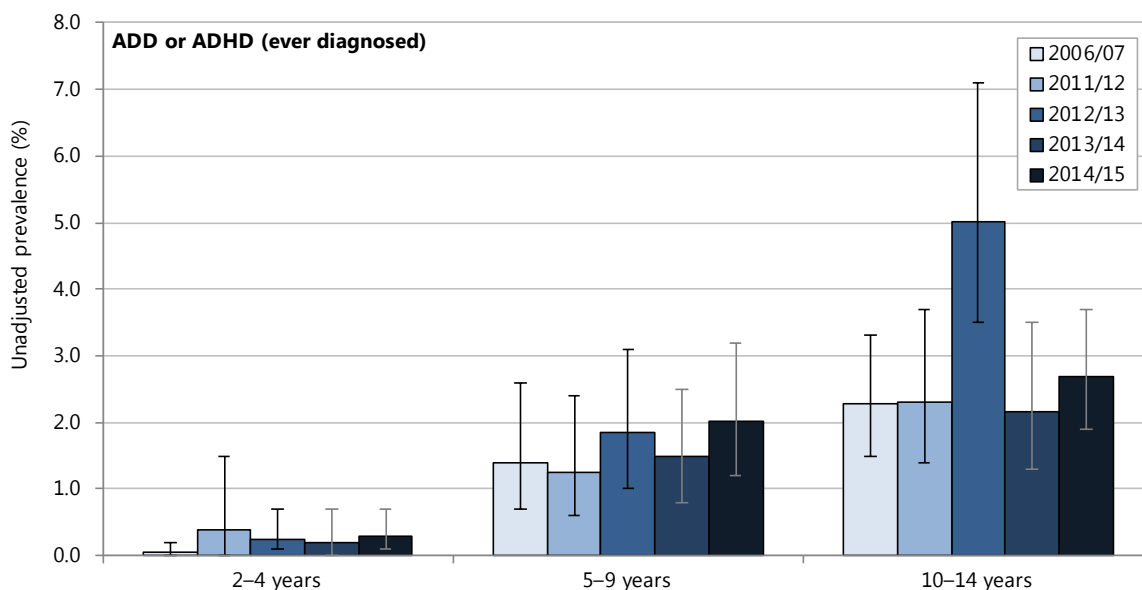
Source: NZ Health Survey

Data from NZHS for one year, 2014/15 shown in **Figure 36**, indicates that the percentages for 5–9 year olds and 10–14 year olds with ADD/ADHD (Attention Deficit Disorder/Attention Deficit and Hyperactivity Disorder) are significantly higher than for 0–4 year olds. The percentages for Māori and European are higher than for Pacific and Asian, but not significantly so. The percentages are higher for children in areas with higher deprivation scores, but the differences between deprivation quintiles are not statistically significant.

Figure 35. Emotional or behavioural problems (diagnosed), by demographic factor, NZ Health Survey 2014/15
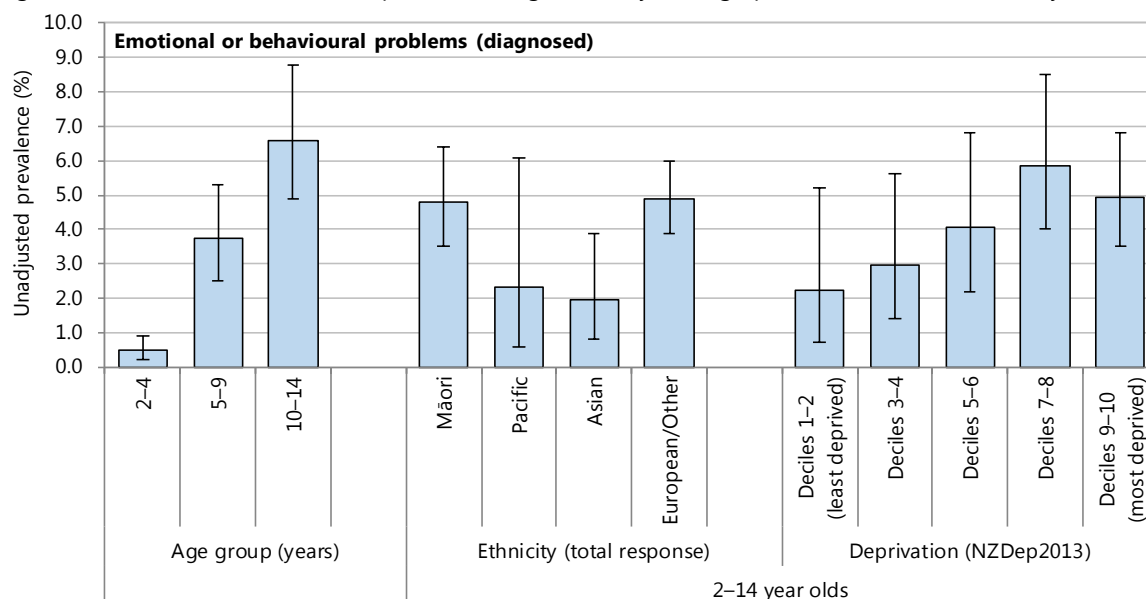
Source: NZ Health Survey

Figure 36. ADD or ADHD (ever diagnosed), by demographic factor, NZ Health Survey 2014/15
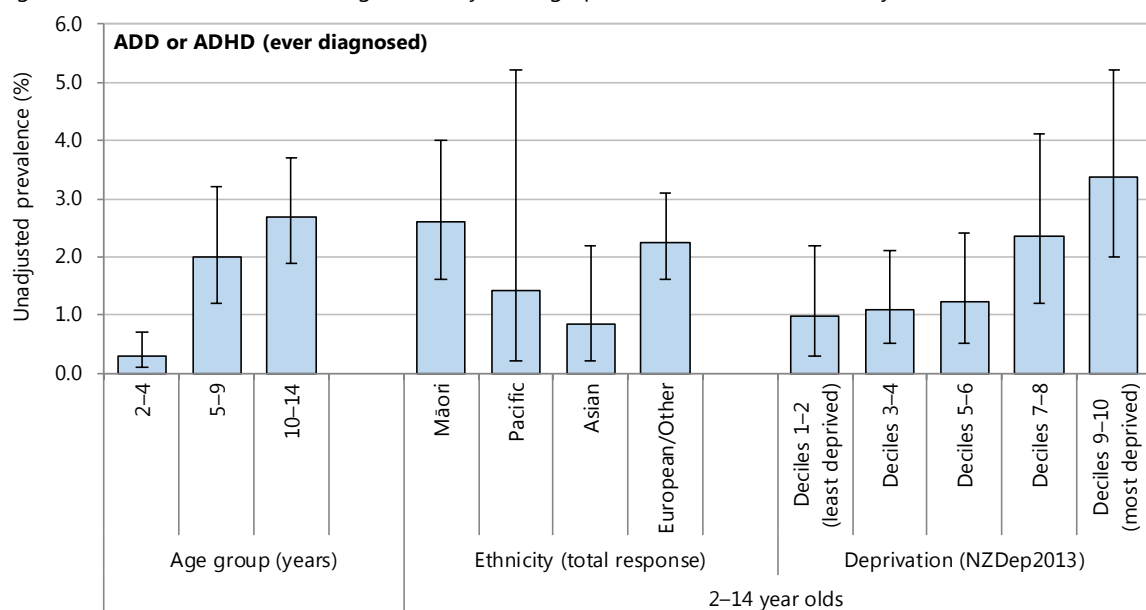
Source: NZ Health Survey

The percentage of emotional or behavioural problems was significantly higher for males and significantly lower for Asian children (**Figure 37**). The percentage of ADD/ADHD (Attention Deficit Disorder/Attention Deficit and Hyperactivity Disorder) was significantly higher for males (**Figure 38**).

The percentages of emotional or behavioural problems for males were higher for Māori and for European/Other (**Figure 39**). The percentage of ADD/ADHD (Attention Deficit Disorder/Attention Deficit and Hyperactivity Disorder) for males was higher for all ethnicities reported (**Figure 40**).

Figure 37. Comparisons for 2–14 year olds diagnosed with emotional or behavioural problems (diagnosed), by sex, ethnic group, deprivation, NZ Health Survey 2014/15
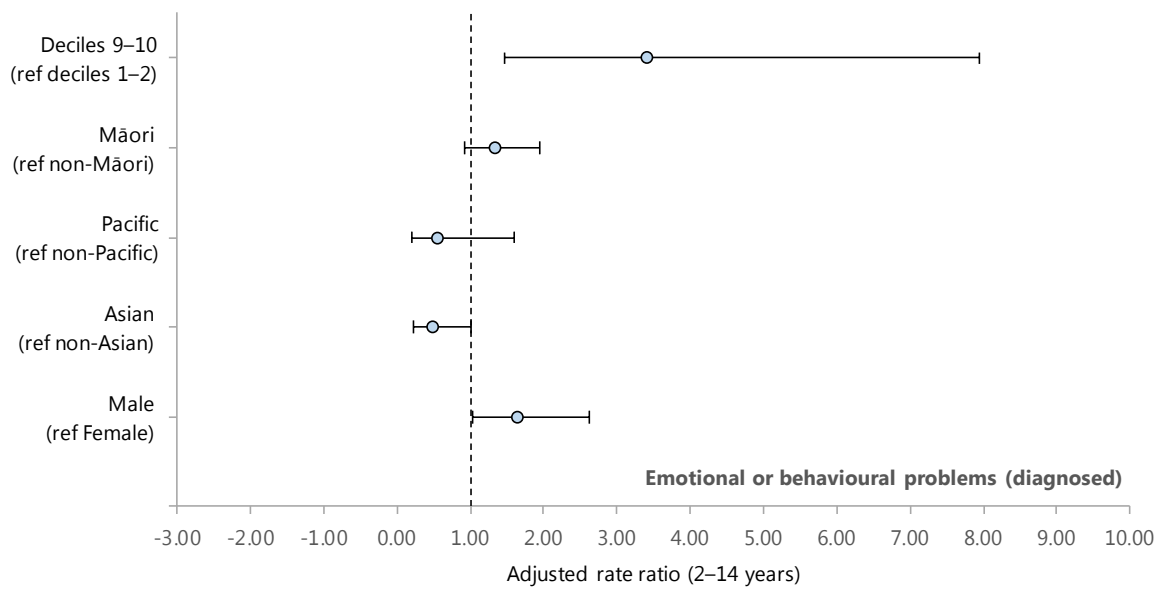
Source: NZ Health Survey. Ethnicity is total response

Figure 38. Comparisons for 2–14 year olds diagnosed with ADD/ADHD, by sex, ethnic group, deprivation, NZ Health Survey 2014/15
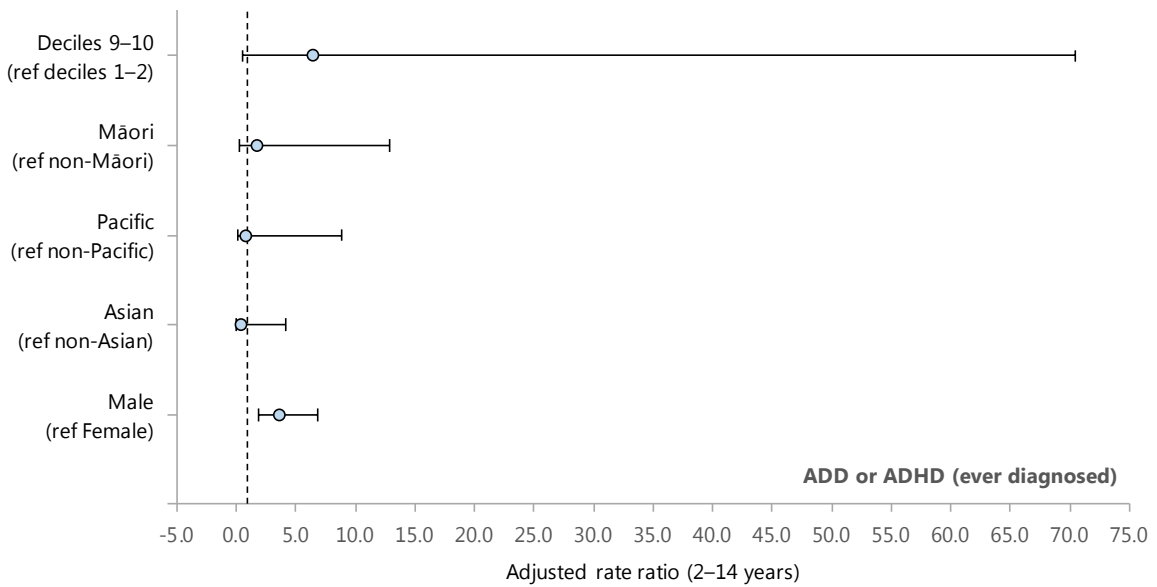
Source: NZ Health Survey. Ethnicity is total response

Figure 39. Emotional or behavioural problems (diagnosed) in 2–14 year olds, by ethnicity and sex, NZ Health Survey 2014/15
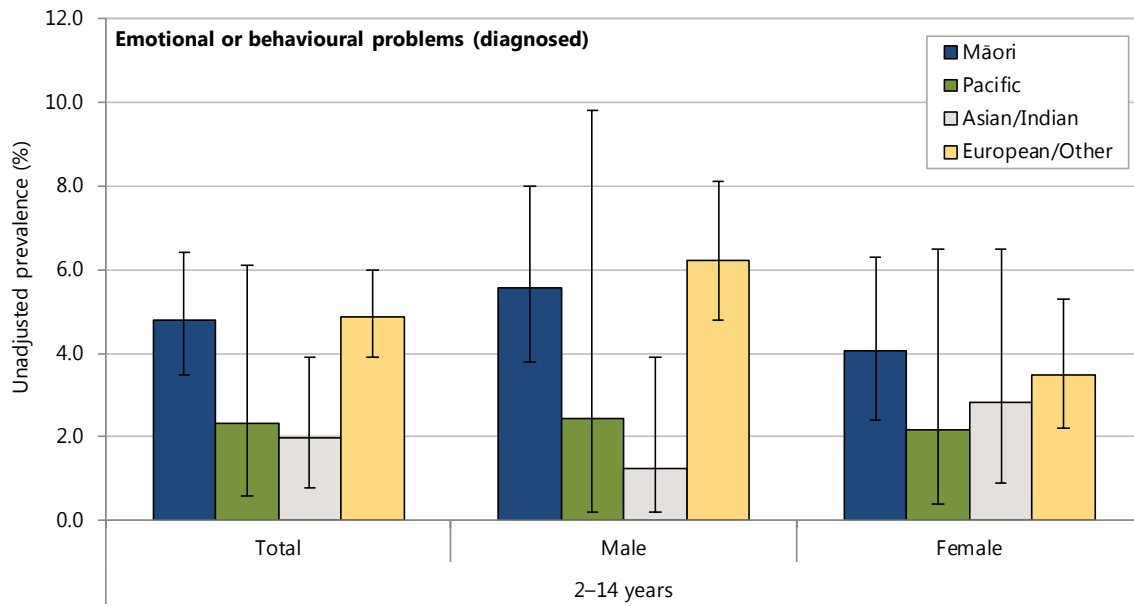
Source: NZ Health Survey. Ethnicity is total response

Figure 40. ADD or ADHD (ever diagnosed) in 2–14 year olds, by ethnicity, and sex NZ Health Survey 2014/15

Source: NZ Health Survey. Ethnicity is total response

Emotional or behavioural problems
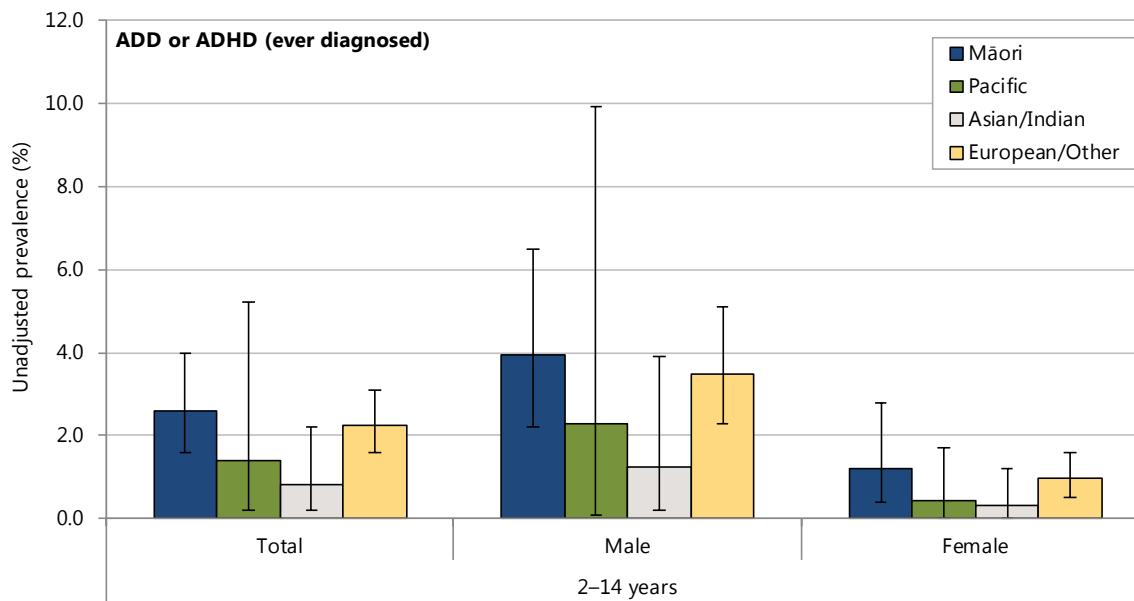
Regional distribution

Figure 41 shows the distribution across the district health boards was varied. Rates in Taranaki, MidCentral and Whanganui DHBs were significantly higher than the New Zealand rate.

Figure 41. Emotional or behavioural problems (diagnosed) in 2–14 year olds, by district health board, NZ Health Survey 2011–2014
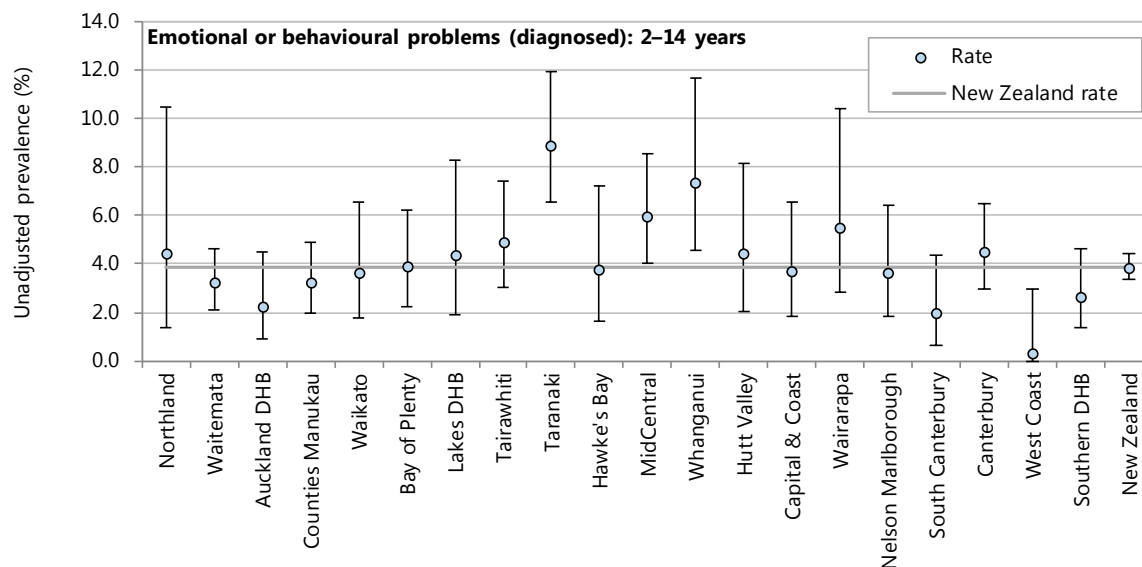
Source: NZ Health Survey

Evidence for good practice

Possibilities for prevention

Children's emotional and behavioural problems have both genetic and environmental causes. Since little can be done about the genetic factors that may predispose a person to mental health problems, preventive interventions primarily focus on optimising children's environments to prevent or manage emotional and behavioural problems.[10] The early years are especially important for a child's healthy mental, social and emotional development.[11]

Parenting style has been found to be the single most important environmental influence on children's behaviour and therefore most preventive interventions aim to improve parenting skills.[10] Parenting programmes are designed to improve parents' capabilities to identify, define, observe and respond to child behaviours in new ways. They help parents to identify problem behaviours and to use non-punitive discipline techniques, such as ignoring and time out, to discourage undesirable behaviour. They teach parents to use positive reinforcement to encourage desirable behaviour, to understand the ways children think and their motives for their behaviour, and to adopt strategies that strengthen parent-child relationships such as active listening, and responding warmly and sensitively when their child is distressed or seeking interaction.[10]

Preventive strategies can be either universal (aimed at the whole population) or targeted (aimed at children who have identified risk factors such as young, low-income parents).[10] A 2009 systematic review that aimed to identify evidence-based preventive interventions for emotional and behavioural problems in children aged 0–8 years found that over fifty such interventions, most targeting behavioural rather than emotional problems had been evaluated through randomised controlled trials.[10] The review authors identified a number of interventions that were both effective and showed promise in the Australian context. Three US programmes well supported by evidence were the individual *Nurse Home Visitation Programme* (for infants), the individual *Family Check-up* (for pre-schoolers), and the class programme the *Good Behaviour Game* (for children of school age).[10] Three parenting programmes in England and Australia were also considered especially worthwhile: the *Incredible Years* (group format), *Triple P* (individual format), and *Parent Education Programme* (group format).[10]

A 2007 Canadian systematic review identified and reviewed 15 RCTs on programmes intended to prevent conduct disorder, anxiety and depression in children aged 0–18 years.[1] Four especially noteworthy programmes for preventing conduct disorder targeted at-risk children in the early years using parent training or child social

skills training (or a combination of both), typically delivered over one to two years in homes, preschools or schools by clinicians or teachers.[1] All of these four: Fast Track, Johns Hopkins, Nurse Visitation and Perry Pre-school, significantly reduced two or more symptom measures. One universal group cognitive-behavioural training (CBT) programme (the Australian Friends programme) was effective in reducing anxiety in 10–13 year olds, with a greater magnitude of effect in at-risk children. Two of the four RCTs of programmes to prevent depression, both assessing the targeted US CBT group programme Coping With Stress, showed significant reductions in both diagnostic and symptomatic measures of depression.[1] Few studies had evaluated costs but the review authors pointed out that the lifetime costs of a single case of conduct disorder may be over a million dollars therefore the costs of not implementing preventive programmes is high.

A 2013 review sponsored by the US Agency for Healthcare Research and Quality assessed the effectiveness of interventions for preschoolers at risk for attention-deficit/hyperactivity disorder (ADHD).[12] The interventions evaluated were parent behavior training (PBT), combined home and school/day care interventions, and methylphenidate use. Data from eight good-quality studies provided high quality evidence that PBT improved child behaviour. Only one relatively small good quality study evaluated use of methylphenidate as a preventive strategy and evidence for its effectiveness in asymptomatic children was low. Some adverse effects were reported for methylphenidate but not for PBT. Studies of combined home and school/daycare interventions showed inconsistent results.

A recent Cochrane review (2016) assessed cognitive behavioural therapy and other psychological interventions for preventing depression in children and adolescents.[13] It included 83 trials, most of which (67) were carried out in school settings. It found that, overall, there was low to moderate quality evidence of small positive effects in depression prevention in the short- to medium-term (up to 12 months). In universal populations, prevention programmes showed a lack of effects in comparison with an attention placebo control. Interventions delivered to targeted populations, especially where these were selected because of having depression symptoms, had larger effects but few of these trials had used an attention placebo control. (An attention placebo control is a control intervention that attempts to control for non-specific factors associated with receiving a therapeutic intervention, such as being part of a trial and receiving attention from researchers.) The review authors concluded that there is still not sufficient evidence to support the implementation of depression prevention programmes.

Mindfulness-based interventions to improve children's social and emotional wellbeing have become popular in recent years.[14,15] Mindfulness is a practice derived from the Buddhist contemplative tradition. Mindfulness exercises teach practitioners to continually bring their attention back to present moment experience, noticing current thoughts, emotions or bodily sensations.[14] There have been a number of studies of mindfulness interventions for young people age less than 18 years, including more than ten RCTs.[14] The research evidence to date suggests that mindfulness is not harmful and that is has small mental health benefits for young people in general and moderate benefits for young people with symptoms of psychopathology.[14,15] New Zealand's Mental Health Foundation has developed the *Pause, Breathe, Smile* mindfulness programme for schools.[14,16]

Evidence-based healthcare for children with mental health problems

It is important that children's mental health difficulties are recognised and treated early since early intervention can prevent problems worsening. It is important that all those working with young children and their parents, whether they work in in education, health, child welfare or other services, are aware of the importance of children having nurturing and responsive relationships with their caregivers. They need to able to recognise and respond to emotional and behavioural problems in children and maternal mental health issues that may make it difficult for a mother to provide the best care for her child. When a parent seeks help, there should be no wrong door: all professionals need to be able to link parents with appropriate services.

Interventions for emotional and behavioural problems in young children always need to involve parents. There is low quality evidence that group-based parenting programmes reduce parent-reported emotional and behavioural problems in children under four years old.[17]

Parent-infant psychotherapy (PIP) aims to improve parent-infant relationships.[18] It involve a parent-child psychotherapist working with a parent and infant at home or in a clinic to identify unconscious patterns of relating and behaving, and influences from the parent's past, that are negatively affecting the parent-child relationship.[18] A recent Cochrane review concluded that, although PIP seems to be a promising way of improving the security of infant attachment, there is no evidence that it improves other outcomes, and no evidence to indicate whether it is any more effective than other types of treatment for parents and infants.[18]

In children with conduct disorder (up to 11 years old), parent-focused interventions are effective for reducing anti-social behaviour.[19] Limited evidence suggests that group interventions for parents, interventions based on cognitive behavioural principles, and interventions using the Triple P or Incredible Years programmes are

especially effective.[19] Child-focused interventions for children and young people with conduct disorder also seem to reduce anti-social behaviour and limited evidence suggests that they may be more effective if delivered in a school rather than a clinic setting.[19] It is uncertain whether interventions delivered separately to both the parent and the child are any more or less effective than those delivered to parents alone.[19] Multi-modal interventions may reduce anti-social and offending behaviour in young people with conduct disorder.[19]

In children with disruptive behaviour associated with conditions such as conduct disorder, oppositional defiant disorder or ADHD, the atypical antipsychotic drug risperidone reduces aggression and conduct problems to some degree after six weeks of treatment but is associated with significant weight gain.[20]

Young children and school-aged children (6–12 years) with ADHD are likely to benefit from psychological interventions, particularly those that train parents in behaviour management techniques.[21] Behavioural classroom management and behavioural peer interventions are also well-established treatments.[21] There is little evidence that parent-based interventions are effective in adolescents with ADHD.[21]

Pharmacological treatment of ADHD is highly effective in most children with diagnosed ADHD and the psychostimulant methylphenidate is the most commonly prescribed drug.[22] In patients who cannot tolerate psychostimulant therapy or have comorbid conditions, atomoxetine or apha$_2$-adrenergic agonists (such as clonidine and guanfacine) may be effective.[22]

In children and young people with depression of any severity, psychological therapy, such as cognitive behavioural therapy should be offered initially.[23] There is little clear evidence to favour one psychological therapy over another.[23] In young people (12–18 years) with severe depression, antidepressants and psychological therapy may be started concurrently as an alternative to trying psychological therapy first and starting antidepressants only if this trial is unsuccessful.[23] The evidence regarding the relative effectiveness of psychological interventions, antidepressant medication and a combination of these interventions in children and adolescents with depression is very limited.[24]

There is evidence that cognitive behavioural therapy is an effective treatment for anxiety disorders in children and adolescents but the evidence suggesting that CBT is more effective than active controls or treatment as usual or medication at follow-up, is limited and inconclusive.[25]

Evidence-based care for children and young people with ADHD and other mental health conditions

International guidelines relevant to the treatment of ADHD
- Canadian Agency for Drugs and Technologies in Health. 2016. Pharmacologic Management of Patients with ADHD: A Review of Guidelines. Ottawa (ON): Canadian Agency for Drugs and Technologies in Health. https://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0086535/
- National Institute for Health and Care Excellence. 2008 (updated February 2016). Attention deficit hyperactivity disorder: diagnosis and management. https://www.nice.org.uk/guidance/cg72?unlid=30803879420163291834448
- Gorman DA, Gardner DM, Murphy AL, et al. 2015. Canadian guidelines on pharmacotherapy for disruptive and aggressive behaviour in children and adolescents with attention-deficit hyperactivity disorder, oppositional defiant disorder, or conduct disorder. *Canadian Journal of Psychiatry* 60(2) 62-76. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4344948/
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- Seixas M, Weiss M, Muller U. 2012. Systematic review of national and international guidelines on attention-deficit hyperactivity disorder. *Journal of Psychopharmacology* 26(6) 753-65. http://dx.doi.org/10.1177/0269881111412095
- National Health and Medical Research Council. 2012. Clinical Practice Points on the diagnosis, assessment and management of Attention Deficit Hyperactivity Disorder in children and adolescents. Canberra: Commonwealth of Australia. https://www.nhmrc.gov.au/guidelines-publications/mh26
- Subcommittee on Attention-Deficit/Hyperactivity Disorder, Steering Committee on Quality Improvement and Management. 2011. ADHD: Clinical Practice Guideline for the Diagnosis, Evaluation, and Treatment of Attention-Deficit/Hyperactivity Disorder in Children and Adolescents. *Pediatrics* 128(5) 1007-22.

- Canadian Attention Deficit Hyperactivity Disorder Resource Alliance (CADDRA). 2011. Canadian ADHD Practice Guidelines, Third edition. Toronto, ON: CADDRA. http://www.caddra.ca/pdfs/caddraGuidelines2011.pdf

International guidelines relevant to the treatment of other mental health conditions

- Siu AL. 2016. Screening for Depression in Children and Adolescents: U.S. Preventive Services Task Force Recommendation Statement. *Annals of Internal Medicine* 164(5) 360-6 http://dx.doi.org/10.7326/m15-2957

- National Collaborating Centre for Mental Health. 2015. Children's attachment: attachment in children and young people who are adopted from care, in care or at high risk of going into care. London: National Institute for Health and Care Excellence. https://www.nice.org.uk/guidance/ng26

- National Collaborating Centre for Mental Health. 2014. Bipolar disorder: the assessment and management of bipolar disorder in adults, children and young people in primary and secondary care. London: National Institute for Health and Care Excellence. https://www.nice.org.uk/guidance/cg185

- National Collaborating Centre for Mental Health, Social Care Institute for Excellence. 2013. Antisocial Behaviour and Conduct Disorders in Children and Young People: Recognition, Intervention and Management. Leicester (UK): The British Psychological Society & The Royal College of Psychiatrists. https://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0078142

- American Academy of Child and Adolescent Psychiatry. 2012. Practice parameter for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry* 51(1) 98-113 http://dx.doi.org/10.1016/j.jaac.2011.09.019

- McClellan J, Stock S. 2013. Practice parameter for the assessment and treatment of children and adolescents with schizophrenia. *Journal of the American Academy of Child and Adolescent Psychiatry* 52(9) 976-90 http://dx.doi.org/10.1016/j.jaac.2013.02.008

Evidence-based medicine reviews relevant to the treatment of ADHD

- Punja S, Shamseer L, Hartling L, et al. 2016. Amphetamines for attention deficit hyperactivity disorder (ADHD) in children and adolescents. *Cochrane Database of Systematic Reviews* (2) http://dx.doi.org/10.1002/14651858.CD009996.pub2

- Briars L, Todd T. 2016. A Review of Pharmacological Management of Attention-Deficit/Hyperactivity Disorder. *Journal of Pediatric Pharmacology and Therapeutics* 21(3) 192-206 http://dx.doi.org/10.5863/1551-6776-21.3.192

- Canadian Agency for Drugs and Technologies in Health. 2016. Combination Therapy for Attention Deficit Hyperactivity Disorder: A Review of the Clinical Effectiveness. Ottawa: Canadian Agency for Drugs and Technologies in Health. https://www.cadth.ca/combination-therapy-attention-deficit-hyperactivity-disorder-review-clinical-effectiveness

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- Tsai MH, Hsu JF, Huang YS. 2016. Sleep Problems in Children with Attention Deficit/Hyperactivity Disorder: Current Status of Knowledge and Appropriate Management. *Current Psychiatry Reports* 18(8) 76. http://dx.doi.org/10.1007/s11920-016-0711-4

- Hoza B, Martin CP, Pirog A, et al. 2016. Using Physical Activity to Manage ADHD Symptoms: The State of the Evidence. *Current Psychiatry Reports* 18(12) 113. http://dx.doi.org/10.1007/s11920-016-0749-3

- Dvorsky MR, Langberg JM. 2016. A Review of Factors that Promote Resilience in Youth with ADHD and ADHD Symptoms. *Clinical Child and Family Psychology Review* 19(4) 368-91. http://dx.doi.org/10.1007/s10567-016-0216-z

- Den Heijer AE, Groen Y, Tucha L, et al. 2016. Sweat it out? The effects of physical exercise on cognition and behavior in children and adults with ADHD: a systematic literature review. *Journal of Neural Transmission (Vienna)* http://dx.doi.org/10.1007/s00702-016-1593-7

- Kidwell KM, Van Dyk TR, Lundahl A, et al. 2015. Stimulant Medications and Sleep for Youth With ADHD: A Meta-analysis. *Pediatrics* 136(6) 1144-53. http://dx.doi.org/10.1542/peds.2015-1708

- Schatz NK, Fabiano GA, Cunningham CE, et al. 2015. Systematic Review of Patients' and Parents' Preferences for ADHD Treatment Options and Processes of Care. *Patient* 8(6) 483-97. http://dx.doi.org/10.1007/s40271-015-0112-5

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- Barkla XM, McArdle PA, Newbury-Birch D. 2015. Are there any potentially dangerous pharmacological effects of combining ADHD medication with alcohol and drugs of abuse? A systematic review of the literature. *BMC Psychiatry* 15 270. http://dx.doi.org/10.1186/s12888-015-0657-9

- Antshel KM. 2015. Psychosocial interventions in attention-deficit/hyperactivity disorder: update. *Child and Adolescent Psychiatric Clinics of North America* 24(1) 79-97. http://dx.doi.org/10.1016/j.chc.2014.08.002

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FETAL ALCOHOL SPECTRUM DISORDER

Author: Judith Adams

Introduction

There is substantial evidence from both observational studies of humans, and experimental studies in animals, that fetal alcohol exposure can cause physical malformations, growth retardation and brain damage.[1-5] Alcohol exposure in pregnancy is commonly cited as the leading preventable cause of intellectual disability.[6,7]

Fetal Alcohol Spectrum Disorder (FASD) is the umbrella term used for the range of physical, cognitive, and developmental disabilities caused by exposure to alcohol in utero.[8]

The Ministry of Health has recently published an Action Plan *Taking Action on Fetal Alcohol Spectrum Disorder 2016 to 2019*[7] which is New Zealand's first attempt at taking a coordinated and strategic national approach to FASD. The plan recognises that FASD contributes to many poor outcomes for New Zealand's young people including early mortality, abuse and neglect, poor educational achievement, engagement with the criminal justice system, benefit dependence, and mental health and alcohol and drug problems.

Features of FASD

The effects of prenatal alcohol exposure include malformations, growth retardation and central nervous system dysfunction of varying severity.[9] For a child's long term future the most devastating effects of prenatal alcohol exposure are the cognitive and behavioural problems caused by alcohol-induced brain damage.[9]

Facial features

Facial anomalies are characteristic of fetal alcohol syndrome (FAS) and a diagnosis of FAS requires (in addition to other criteria) that the following three facial features are present: short palpebral fissures (the distance from the inner corner to the outer corner of the eye), a smooth philtrum (absent or shallow vertical groove between the upper lip and the nose), and a thin upper lip.[6] Assessment of these facial features requires specialised training and these features can become less noticeable as children grow up.[10,11] Only a small proportion of children with FASD have the three facial features, but their presence is highly specific to FAS.[12]

Studies of the effects of alcohol on mouse and chick embryos have indicated that the period of vulnerability for these anomalies corresponds to the human gestational stage between three and six weeks after fertilisation and that there is a general trend for the facial anomalies to become more pronounced as alcohol exposure increases.[13]

It is often stated that FAS is the most severe form of FASD but cognitive and behavioural impairments can be of similar severity in people with FAS and people with FASD who lack the facial features of FAS and it is the cognitive and behavioural deficits rather than the facial abnormalities that have the greatest impacts on life outcomes.[14,15]

Neurodevelopmental problems

Children exposed to alcohol in utero at any stage of gestation can have impairments of variable severity in many areas of central nervous system function.[16] Their cognitive and behavioural problems can seem similar to those of other neurodevelopmental conditions such as ADHD but specialised assessment can distinguish FASD from other conditions.[17] Cognitive problems that children with FASD may have include:[16,17]

- Lower than normal intellectual ability. The average IQ of children with FASD is in the cognitively impaired to low normal range but there is a wide variation[18]
- Difficulty with sustaining attention
- Difficulty with executive function in both cognition-based and emotion-based tasks (problems with coordinated planning and execution of tasks)
- Difficulty with language
- Difficulty with mathematics
- Difficulty with memory
- Deficits in visual-spatial perception and construction

- Slower than normal information processing

Overall, these problems can be summarised as a generalised deficit in processing complex information.[17] Mathematics is a particular area of weakness in children with FASD, and deficits have been found even in studies that control for IQ and other potentially confounding variables. It is the cognitive weakness that is most highly correlated with the amount of prenatal alcohol exposure.[19]

The effects that these problems have on everyday life can be profound. Memory problems and difficulties with information processing make it hard to follow instructions and to learn new skills. Teachers may report that children with FASD are disorganised, tactless, distractible, lack persistence, and have poor processing and reasoning abilities.[17,20]

People with FASD commonly have problems with adaptive and social behaviour and their deficits in these areas are more severe than those of people with similar verbal IQs who were not exposed to alcohol.[21-24] Social problems include impairments in social competence and relationships, being inappropriately friendly as children, and difficulties in perceiving and responding to social cues, exhibiting consideration for others, following social norms, and forming reciprocal friendships.[25] These problems often result in social rejection, which contributes to poor self-esteem, aggression, anxiety, depression, trouble at school, mental health problems and anti-social behaviour.[25]

Standardised tests of adaptive functioning, such as the Vineland Adaptive Behavior Scales, may not fully capture the extent of the difficulties experienced by a person with FASD since they include only a few questions relating to poor decision making and dangerously impulsive behaviours that pose risks both for the person themselves and those around them.[26] Functioning as an independent adult requires not only being capable of shopping, cooking, cleaning and laundry but also being able to deal with problem associates who may enter your home, steal your belongings or cause property damage that results in you being evicted.[26] The Vineland scales have questions covering the former but not the latter skills.[26]

Social impairments in people with FASD are life-long and tend to become more pronounced with age. Some adults with FASD have been reported to have the social functioning of a six-year-old child.[27]

Growth retardation

Prenatal alcohol exposure is associated with intrauterine growth retardation with smaller weight, length and head circumference at birth.[28] A number of cohort studies have found growth deficits to persist through childhood into adulthood.[29-32] Most of the international diagnostic guidelines include growth retardation in the diagnostic criteria for FAS and other FASDs,[10,33-36] although the latest Canadian[37] and Australian[38] guidelines have adopted the term FASD as a diagnostic term and deleted growth impairment as a diagnostic criterion due to the fact that many children with prenatal alcohol exposure and central nervous system dysfunction do not have growth impairment. A population-based cohort study done in Western Australia found that, although infants of mothers with moderate to heavy alcohol consumption in pregnancy had increased odds of being born small for gestational age, this effect was eliminated after adjustment for smoking status.[39]

Comorbidities of FASD

A recent systematic review aimed to estimate the pooled prevalence of co-morbid conditions in people with FASD (using data from 33 studies with 1728 participants) and to highlight conditions that were much more common in people with FASD than in the general US population.[40] There were 18 disorders that were found to be present in more than half of those with FASD, including conduct disorder, visual problems and refractive errors, speech and language deficits, premature birth, hearing loss, alcohol or drug dependence, and ADHD.[40] The prevalence of hearing impairments was estimated to be 100 times higher in individuals with FASD than in the general population, the prevalence of visual impairments at least thirty times higher, and the prevalence of mental and behavioural disorders due to multiple drug use around twenty times higher.

Life course consequences of FASD

Over the lifespan, the primary impairments of FASD interact with environmental risks to produce secondary problems. Infants exposed to alcohol in utero tend to be more irritable than other infants and often have disturbed sleep patterns and feeding problems.[21] These problems can disrupt mother-infant attachment, which is believed to be fundamental to later social interactions, especially interactions between two people.[21]

As pre-schoolers, children with FASD may be hyperactive, excessively friendly and fearless, and have poor motor coordination and delayed speech, language and other developmental milestones.[41] They may be prone to

temper tantrums and non-compliant.[42] When they go to school, children with FASD struggle with cognitive, academic and social, emotional, and behavioural challenges that have negative effects on their ability to learn and function in the school environment.[43]

The University of Washington's Secondary Disabilities Study conducted life history interviews with all available caretakers or informants of 415 individuals with FAS or fetal alcohol effects, whose ages ranged from six to 51 years.[44] The most important findings from this study were:

- 90% of all study participants had experienced mental health problems, most commonly attention deficit and behaviour problems in young children, and depression in adults
- 60% of those aged 12 or more years had been suspended or expelled from school, or dropped out of school
- 60% of those aged 12 and over had been in trouble with the law
- 50% of those aged 12 and over had experienced loss of liberty (23% because of psychiatric disorders, 15% via compulsory admission for drug or alcohol dependency, and 35% because of imprisonment by the criminal justice system)
- 50% of those aged 12 and over were reported to have a history of inappropriate sexual behaviour
- 30% of those aged 12 and over had drug and alcohol problems
- 80% of the 90 individuals aged 21 years or more were not living independently
- 80% of those aged over 21 had problems with employment

The University of Washington study noted that many of the people with FASD had come from dysfunctional, transient and abusive living situations which may have contributed to their secondary disabilities.[44] It found that 72% of those aged 12 years and older had experienced violence (physical or sexual abuse, or domestic violence) and that having experienced violence was by far the strongest risk factor for inappropriate sexual behaviour. It also found that half of those aged 12 years or more had not stayed in each living situation for an average of more than two years, and half had spent at least 30% of their lives living with a person who had an alcohol or drug problem.

There have been few longitudinal studies of people with FASD.[45-49]

A study done in Finland followed up children born to eighty-two pregnant women who had attended a special clinic aimed at reducing heavy drinking in pregnancy.[47] Of the 69 surviving children who could be followed up at 12 years, 41 had FASD (FAS, fetal alcohol effects or alcohol related neurodevelopmental disorder), 10 had growth retardation but no other signs of FASD, and 18 had normal development. Of the 41 children with FASD, 21 were receiving mainstream education, seven were in a mainstream class with either a personal assistant or adjusted requirements in some subjects, and 13 were in a special education class.

In this study there was a high rate of children being taken into care because of parental alcohol abuse and inability to provide adequate daily care for their child, often in combination with psychosocial problems.[47] Only 11 of the 42 children with FASD and 18 of the 28 children without FASD had lived only with a biological parent by the age of 12 years. Behavioural problems were common: families of 24 of the 41 children with FASD, and nine of the 28 children without FASD had sought help for behavioural problems. According to the examining psychologist/child psychiatrist behavioural problems were associated with one or more of the following factors: poor quality of early attachment and daily care; repeated experience of neglect; physical and/or sexual abuse; having observed a frightening experience (abuse of mother or sibling); not being liked by peers – loneliness and poor understanding of social rules; and difficulties in learning capacity, attention and impulse inhibition. Eleven biological parents of study children (eight mothers and three fathers) had died. Their deaths were due to alcohol abuse and/or psychological problems. Eight biological parents (four mothers and four fathers) had been in prison.

Another prospective longitudinal study[50] which assessed 24 children of alcoholic mothers in Sweden at the age of 12–14 years had similar findings. A later Swedish study,[51] which included some of the same participants as the previously mentioned study, used national registers to follow-up a group a 79 Swedish adults with FAS at a mean age of 32 years and compare their educational, social adjustment and mental health outcomes with those of a comparison group of 3,160 individuals matched on age, gender and place of birth. Compared to the comparison group, the FAS group were much more likely to have received special education (25% vs 2%), be unemployed (51% vs 15%), and receive a disability pension (31% vs 3%), but they had similar levels of criminal offending. They had higher rates of hospital admission for alcohol abuse (9% vs. 2%) and psychiatric disorders (33% vs. 5%) and were more likely to have been prescribed psychotropic drugs (57% vs. 27%).

The Swedish register-based study[51] noted that 81% of the adults with FAS had been placed in state care in their youth (compared to 4% of the general population) so the authors attempted to account for the effects of this difference. When the outcomes for the 79 adults with FAS were compared to those for a matched group of 122 adults who had been placed in state care before the age of 18 years it was found that although the FAS group were more likely to have attended special education (25% vs. 3%), there were no significant differences between the two groups in terms of completed education, income, self-support, or hospital care. Compared to the state care group, the FAS group were less likely to have received a criminal conviction (28% vs. 55%) and much less likely to have been convicted of a severe crime (6% vs. 30%). The study authors noted that the study subjects had full-blown FAS and had received their diagnosis early in life and had therefore received financial support and had close contact with social workers during childhood. They suggested that these factors might have contributed to the relatively high rates of completed secondary education and employment of the individuals with FAS.

A German study which was a 20-year follow-up study of 37 adults with FASD (out of a cohort of 52 eligible subjects originally diagnosed as having FAS or FAE in infancy and childhood) found that, although the facial features of FAS were less marked in adulthood, only a very small proportion of the study population were living a normal adult life.[11] Eighteen (49%) had received special education only. Only five (13%) had ever held an "ordinary" job, despite 25 (69%) having received at least some preparatory job training and 21 (58%) having either started or progressed to formal occupational training. Assessment of living situations revealed that 27% lived in institutions, 35% were in a dependent-living situation, 14% lived independently alone, 8% lived with a partner, 8% had their own family and 8% lived with their father plus a mother surrogate. Study subjects had higher than normal rates of emotional and behavioural problems, especially attention difficulties and aggressive, intrusive and delinquent behaviour. Their mental and behavioural problems were independent of intellectual impairment and whether they had been diagnosed with FAS or with fetal alcohol effects.

Epidemiology of FASD

There is no New Zealand data on the prevalence of FASD but it has been conservatively estimated to be at least one percent in the general population and around 50% in children and youth in Child, Youth and Family (CYF) care.[7] Ospina and Dennett's 2013 systematic review of 54 FASD prevalence studies, mostly conducted in North America and Europe, found that FASD rates have been examined in a variety of settings including the community, schools, foster care settings, prisons and correctional settings.[52] The review found considerable variation in prevalence estimates. Some of this was due to factors such as differences in methods of case ascertainment, diagnostic criteria and study participants' ages, but variation in prevalence estimates is also likely to reflect genuine variation between different geographic and other population groups.

Ospina and Dennett's review found the reported prevalence of FASD in the community ranged from 0.2 to 5 per 1,000 population across five studies.[52] The prevalence of FAS in non-South African school settings ranged from 0.2% to 0.8% giving a pooled estimate of 0.4% (4 per 1,000). The prevalence of partial FAS in schools, based on a pooled estimate from the four studies that assessed prevalence in either random samples or the whole population, was 2.9%. The prevalence of FASD in special education settings ranged from 2% to 9%.

The prevalence of FASD was found to be much higher in certain population groups. In foster care settings the prevalence of FASD ranged from 30 to 50%.[52] Studies in the US and Canada estimated the prevalence in prisons and correctional facilities as being between 10% and 23%. Prevalence estimates for FASD in aboriginal populations in Canada, the US and Australia were highly variable, but a pooled estimate of FAS prevalence in Aboriginal peoples was calculated from six studies as 0.2%, not substantially higher than that found in community samples from the general population.

Diagnosis of FASD

It is important that children with FASD are recognised early in life because they can then receive the understanding and support that will increase their chances of having the best possible outcomes in later life and help them avoid some of the secondary problems that can result from their primary neuropsychological impairments, such as exclusion from school, criminal offending and mental health problems.[23,53-55]

Making a diagnosis of FASD can also enable a child's mother to get help to address her alcohol problem and associated difficulties, and avoid having more children with FASD.[53,55] It may lead to the identification of siblings with FASD.[56] Early diagnosis is helpful for parents because it gives them an explanation for their child's behavioural problems and their parenting skills can increase with greater understanding of their child's disabilities and impairments.[57] Professionals often fear that mothers will feel judged and shamed when their

child is diagnosed with FASD[58] but the New Zealand birth mothers interviewed by Jenny Salmon reported that they were relieved when they finally received what they perceived to be the correct diagnosis for their child because, as well as knowing what was actually wrong with the child, they could make sense of the confusing behaviours that they had observed and had thought were due to their poor mothering skills.[59]

Without the capacity to diagnose FASD it is impossible to determine how many people in a community have FASD and whether prevention or mitigation efforts are having any effect.[60]

Diagnostic criteria

There a number of international guidelines on the diagnosis of FAS and FASD and there are well-established diagnostic criteria for FAS.[8,10,33,35-38] There is less international consensus on how to diagnose FASD but there is a consensus that a comprehensive physical and neurodevelopmental assessment is needed to understand the full impact of fetal alcohol on a child's development.[38,55] Such an assessment requires input from educators, social workers and a multidisciplinary team of health professionals.[37,55]

According to the Australian diagnostic guidelines[38] for a child to receive diagnosis of FASD he or she must have:

- severe impairment in at least three of the following ten neurodevelopmental domains: brain structure; motor skills; cognition; language; academic achievement; memory; attention; executive function, including impulse control and hyperactivity; affect regulation; and adaptive behaviour, social skills or social communication

AND either

- All three of the sentinel facial features OR

- 0,1 or 2 facial features plus confirmed prenatal alcohol exposure

Confirmed prenatal alcohol exposure is not required if all three facial features are present (small palpebral fissures, smooth philtrum and thin upper lip) because it is then unlikely that some other condition is the cause of the child's examination findings.

Because there is no pattern or type of CNS dysfunction that is specific to FASD, it is never possible to say for certain that a child without the facial features of FAS has an alcohol-related neurodevelopmental disorder, only to say that, on the balance of probabilities, given known prenatal alcohol exposure and observed CNS dysfunction consistent with the diagnostic criteria for FASD, it is likely that he or she does.

The diagnosis Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE), has been proposed for inclusion in DSM-5, The Diagnostic and Statistical Manual, 5th edition, from the American Psychiatric Association, to encompass the range of neurodevelopmental disabilities that can be associated with prenatal alcohol exposure.[61] According to the proposed definition of ND-PAE, diagnosis requires confirmed prenatal alcohol exposure plus impairment in all three of the following domains:[61]

- neurocognitive (as indicated by impairment in one or more of: intellectual function/global development; executive functioning; learning; memory; visual spatial reasoning)

- self-regulation (as indicated by one or more of: impairment in mood or behavioural regulation; attention deficit; impairment in impulse control)

- adaptive functioning (as indicated by two or more of the following, with at least one of the first two symptoms: communication deficits; impairment in social communication and interaction; impairment in daily living; motor impairment)

Practical difficulties in diagnostic assessment

While guidelines recommend that children suspected of having FASD be assessed by multidisciplinary teams who have specific training in assessing children prenatally exposed to alcohol, there are few specialised FASD diagnostic services. An international survey conducted in 2006 identified 34 FASD diagnostic clinics: 29 in North America, two in Africa, two in Europe and one in South America.[62] The UK currently has only one specialist FASD clinic.[63] A 2011 Canadian survey of clinical capacity for diagnosing FASD found that, based on an estimated FASD prevalence of one percent, diagnostic capacity would need to increase seventeen-fold to be able to diagnose all cases of FASD.[64] A US expert estimated that the US would need another 200 multidisciplinary teams and the UK would need 39 fulltime teams to identify all new cases of FASD[65] and he cited the view that the current diagnostic criteria for FASD are so complex that even expert clinicians have difficulty applying them.[66]

Another Canadian study estimated that a multidisciplinary diagnostic FASD assessment used between 23 and 33 hours of staff time (costing between $2,650 to $3,750) and that the total cost for one individual to be screened, referred, admitted, and diagnosed ranged from $3,110 to $4,570 (32 to 47 hours per person).[57]

Many clinicians lack the training and experience to confidently diagnose FASD.[67-69] The Ministry of Health's 2015 survey of a wide range of New Zealand clinicians found that those who felt extremely confident about this diagnosis were definitely in the minority.[7] A 2004 survey of paediatricians in Western Australia found that, while 81% knew that abnormal facial appearance was a feature of FAS, only 19% knew all of the essential diagnostic features of FAS, and only 23% routinely asked about alcohol use in pregnancy when taking a pregnancy history.[58] Most admitted having suspected but not diagnosed FAS and almost 70% thought that diagnosis could stigmatise a child or family.[58] The provision of educational resources did little to change these paediatricians' knowledge of the essential diagnostic features of FAS or their confidence in making the diagnosis.[70]

Most children with FASD do not have the characteristic facial features of FAS. Of 1,270 patients seen at clinics belonging to the Washington State Fetal Alcohol Syndrome Diagnostic & Prevention Network during 1993–2005 who had confirmed prenatal alcohol exposure (or all of the features of FAS) and CNS dysfunction, and would therefore meet the criteria for FASD, only 59 (4.6%) could be diagnosed with FAS.[71]

Making a diagnosis of FASD in the absence of the facial features characteristic of FAS requires confirmation of maternal alcohol intake during pregnancy.[53] This can be difficult to obtain. Midwives and other clinicians may be reluctant to ask a pregnant woman about the details of her alcohol intake for fear of alienating or stigmatizing her, because they lack knowledge about the amounts of alcohol and the patterns of alcohol consumption that are especially harmful in pregnancy, or because there is a lack of services to refer a woman to if an alcohol problem is identified.[72] Women may be reluctant to disclose their alcohol intake because of embarrassment, guilt or fear that they may lose custody of their children.[73] A woman may not be able to recall the details of her drinking in pregnancy if she is asked only years later when her child is being evaluated for developmental and/or behavioural problems suggestive of FASD, and she is likely to be especially vulnerable to feeling judged if she did drink during pregnancy and knows that there is a possibility that her drinking has contributed to her child's problems.[73] For children in foster care or international adoptees it may be impossible to obtain information on their mothers' alcohol consumption.[74]

Children exposed to alcohol in utero often have other adversities in their backgrounds so it can be hard to determine the whether a child's emotional and behavioural problems are primarily due to prenatal alcohol or to other environmental or possibly genetic factors.[74] Children of alcohol abusing parents have been found to be more likely than other children to experience emotional, physical and sexual abuse; emotional and physical neglect; exposure to domestic violence; substance abuse or mental illness within their household; parental separation or divorce; and incarceration of a household member.[75,76] Parental substance use disorders have been reported to be a contributing factor for between one-third and two-thirds of children involved with child protection services in the US.[77]

Given their often difficult family environments it is not surprising that children with prenatal alcohol exposure often meet the diagnostic criteria for one or more mental health conditions including reactive attachment disorder, posttraumatic stress disorder, learning disabilities, depression and anxiety, conduct disorder, and attention deficit hyperactivity disorder (ADHD).[74,78,79] It can be hard to decide whether these disorders are independent of, secondary to, or comorbid with any effects of prenatal alcohol exposure on neurodevelopment.[74,80] This may be why some clinicians are reluctant to make a formal diagnosis of FASD.[74] New research may develop more specialised assessment tools that will help with discriminating the impairments of FASD from those due to other conditions.[74,81]

It is important that mental health professionals consider the possibility of FASD in someone with a mental health problem because it may be the reason they are not responding to medications and psychosocial therapies, and an indication that they need on-going psychosocial support.[78]

New Zealand experience with FASD diagnosis: The FASD assessment pathway in the Hawke's Bay DHB

The Hawke's Bay District Health Board (DHB) has a Developmental Assessment Programme (DAP) that provides multi-disciplinary diagnostic assessments of children with complex developmental and behavioural concerns.[54] It also provides training in developmental and behavioural conditions for people working in health, education and social service settings. One component of the DAP is a FASD Assessment Pathway for children whose developmental and behavioural issues are possibly related to pre-natal alcohol exposure. The Health Promotion Agency (HPA) commissioned an independent process evaluation of this FASD Assessment Pathway

to learn more about this service model and explore considerations related to replication of this programme in other regions. A brief summary of the findings from this evaluation, which took place during April to June 2015, is presented below.[54]

The FASD Assessment Pathway is provided by a multidisciplinary team. Paediatric, speech language, psychological and social work assessments are combined in a diagnostic assessment based on Canadian guidelines. Key aspects of the pathway are: assessment at home, school and clinic; collaboration with families, caregivers, teachers, referring paediatricians and others; presentation of synthesised assessment findings and recommendations in a single report, and provision of feedback sessions to the family and to school personnel involved with the child.

The evaluation found that the FASD Assessment Pathway is successfully identifying children with FASD, engaging families living in low decile areas, and engaging many Māori whānau. It is reaching into homes and schools. Factors identified as being linked to the success of the pathway were categorised as relating either to the service model or to the workforce, infrastructure and service context.

Essential aspects of the service model were:

- Shared vision and values
- A best practice multi-disciplinary approach
- A synthesised report and feedback sessions
- A team approach
- Close working relationships with paediatricians
- Strong and effective relationships with other services
- Building capacity through training

Regarding the workforce, infrastructure and service context, the evaluation found that the DAP FASD Assessment Pathway is provided by a highly skilled and experienced professional team supported by strong leadership. The Pathway is part of a broader assessment programme that is located within a child development service. This was viewed as contributing to the programme being sustainable and of high quality. The evaluation noted that locating the specialised FASD assessment pathway within a child development service is widely viewed as a way of providing an effective continuum of service for children, enabling a flexible and efficient use of resources, and helping to ensure programme sustainability. It also noted that the people delivering such a programme need specialist training in FASD assessment. (Three of the Hawke's Bay team members travelled to Canada for this.)

The evaluation highlighted a number of important factors relevant to the replication of the FASD Assessment Pathway. The most significant was a general consensus that the effectiveness of the pathway is limited by the lack of post-assessment support for children identified as having FASD, and their families. Effectiveness would be improved if support could be provided, similar to that provided for children diagnosed with Autism Spectrum Disorder (who are also diagnosed within the DAP). Extra funding would be needed for this. Additional on-going challenges were:

- Waiting times
- A need to keep refining assessment reports so they are both as comprehensive and as user-friendly as possible
- Ensuring cultural responsiveness. Stakeholders suggested that Māori and Pasifika input should be obtained in the programme set-up phase to ensure that cultural responsiveness is maximised from the outset
- A mismatch between the perspectives of the health and education sectors: health focuses on diagnosis but education is more concerned with functional impact in the learning environment
- The need to develop and maintain a team culture characterised by strong professionalism, collaboration, collegial respect and openness. This requires ensuring that team members have opportunities for on-going professional development

The DAP team aims to provide 40 assessments per year (one a week during school terms), and data indicates that approximately one third of these are FASD assessments. The average number of professional hours used per FASD assessment (excluding paediatrician, administration and management hours) was 34.5. The FASD Assessment Pathway does not accept children until they reach the age of eight years so any child younger than eight must wait under the care of a paediatrician until they are old enough to be assessed. Most children assessed for FASD receive general health oversight from a paediatrician both before and after assessment. During

2010-2015, 74 children accessed the FASD Assessment Pathway. Seventy-four percent were boys and 26% girls. Most were Māori (73%). The ages of children (at referral) ranged from three to fifteen years and around 61% were aged eight or older. Most children lived in high deprivation areas and only 26% lived with two parents (birth parents, adoptive parents or whangai parents).

Of the 42 children with data on FASD assessment outcomes, all but one child met the criteria for at least one disorder and 38 (90%) met the criteria for FASD. Most children had more than one disorder: 29% had two disorders and 64% had three or more disorders. The other disorders included attention deficit hyperactivity disorder, intellectual disability, language disorder, attachment disorder, adaptive behaviour limitations and cerebral palsy.

Interventions for children and families living with FASD

There is little point in investing in improving capacity to diagnose FASD without also being able to provide services that will make a meaningful difference to the lives of children with FASD and their families, both in the short term and the long term.

There is a small but growing evidence base for interventions for children with FASD, children at risk of FASD because of known prenatal alcohol exposure, and parents who are themselves affected by FASD.[82-84]

Intervention studies in humans have mostly aimed to improve specific cognitive or adaptive skills such as language, literacy, mathematics, self-regulation, working memory and social skills. Kodituwakku's 2011 review identified only 12 papers reporting on scientific evaluations (RCTS or quasi-RCTS) of interventions for children pre-natally exposed to alcohol.[83] While some of these studies found positive effects, only two interventions had been evaluated more than once, and none were considered to be well established. Because each person with FASD (or prenatal alcohol exposure) is not affected in the same way or to the same degree, a thorough assessment of a person's deficits and strengths is necessary to determine what kind of specific intervention might be beneficial for that person.[84] Cognitive and behavioural interventions often require time and effort from families and therefore they may be ineffective for children in chaotic families.[83] Such interventions are more likely to be successful when they include both direct child intervention and parent support.[82,83]

The 2015 review by Reid et al.[85] had broader inclusion criteria and included studies if they reported quantitative measures of functioning so that comparisons could be made about potential gains, regardless of the study design or type of outcomes. It identified 32 studies, most of which targeted aspects of neurocognitive functioning in early to middle childhood although there were two studies which aimed to improve developmental outcomes in infants born to substance-abusing mothers, four studies of education and advocacy for parents and caregivers (two studies), and teachers or child welfare workers (one each), and two studies of support for parents who were themselves affected by FASD. The studies were generally small, at some risk of selection bias, and of variable methodological quality. Nineteen were considered to have a "strong" study design (randomised controlled trial or controlled clinical trial).

The only studies with long term follow up (six months or more) were two studies of home visiting programmes for substance abusing mothers, neither of which found clear benefits for children's development at two to three years of age.[85] The review authors suggested that standard developmental measures may not be the best tools for measuring intervention effects in infants and toddlers with prenatal alcohol exposure. Other findings from the review were: a number of small studies have found positive effects from interventions to improve self-regulation or attentional control; interventions aiming to improve some specific areas of cognitive difficulty in children are promising; there is strong evidence for the utility of structured programmes that include both parents and children in improving social skills; there is promising evidence that parents and caregivers benefit from support in managing their children's behaviour and that improvements in children's behaviour are associated with improvements in parents' wellbeing; and support, education and advocacy services for parents, caregivers, and child welfare caseworkers can be beneficial both for these adults (increasing confidence, decreasing stress) and for the children they support (decreasing school problems, reducing number of changes in foster care placement).[85] The two small cohort studies of support for parents with FASD (all mothers except one), found benefits including decreased drug and alcohol use, and improvements in mental health, finances, housing stability, parenting, use of contraception, and use of medical and mental health services.[85]

A modification of the Alert Program®[86], which was used in three of the studies in the Reid et al. review, and found to have positive effects, is being trialled with Aboriginal children attending schools in remote communities in Western Australia.[87,88] The estimated prevalence of alcohol-related neurodevelopmental disorders among these children is almost 20%.[89] The Alert Program®, which was designed by occupational

therapists, teaches children about self-regulation (the ability to change the arousal state of your nervous system so it is appropriate to the task or situation).[90]

There has been very little published research on interventions for adolescents or young adults with FASD but a recent review identified two recently completed trials in the US.[82] One of these, Project Step-Up, found that a six-week intervention aimed at reducing or preventing alcohol and substance abuse that was delivered to adolescents with FASD and their parents (separately and concurrently) led to reductions in drinking, risky drinking, and negative consequences from drinking in the adolescents.[91] The other trial, of a multi-component intervention for 13–25 year olds with FASD and their families called Partners for Success, did not find any positive effects on youth outcomes, but did find changes in parent positive coping and self-controlling behaviours.[92] This trial's capability for finding positive effects may have been compromised by problems with recruitment and implementation.[82]

Animal studies have found a number of substances that, when given to alcohol-exposed pregnant mothers, mitigate the effects of alcohol on their offspring. These substances include 5-HT$_{1A}$ agonists, neuroprotective peptides, anti-oxidants, and choline and other nutrients.[83,93] Some studies of rats with prenatal alcohol exposure have found benefits from post-natal nutritional supplementation[94,95] and social and environmental enrichment[96] suggesting that these approaches might be of benefit to children with, or at risk of, FASD. Investigating the efficacy of these approaches in humans could be difficult because women who drink heavily in pregnancy are probably unlikely to reveal their drinking habits to health professionals.[83]

Maternal undernutrition may exacerbate the effects of prenatal alcohol exposure on the developing fetus.[93] A case-control study done in South Africa found that the mothers of children with FAS were significantly smaller than control mothers as indicated by height, weight, head circumference, and BMI.[97] There has been very little research on nutritional supplements for pregnant women to reduce the impact of prenatal alcohol exposure on their children but one study of micronutrient supplementation in pregnant Ukrainian women suggested that choline supplementation, administered together with routinely recommended multivitamins and minerals, may improve basic learning mechanisms involved in encoding and memory of environmental events for infants from both alcohol-exposed pregnancies and non or low alcohol-exposed pregnancies.[98]

Children with FASD often have ADHD.[80] There have been only two very small randomised controlled trials of pharmacological treatment of ADHD symptoms in children with FASD.[80,99] It appears that, while there is considerable individual variability in response to medication, psychostimulant medication, such as methylphenidate or dextroamphetamine, improves hyperactivity but not inattentiveness.[80,83] A recently published consensus guideline from the UK on the identification and treatment of individuals with ADHD and associated FASD[99], the first such guideline, warns that side effects from medication may be more marked (though similar) in individuals with ADHD and FASD than in those with ADHD alone and that the use of short-acting stimulants in adolescents with FASD and ADHD may potentially contribute to the development of addictive disorders during the teenage years.

The long term outcome for a child with FASD depends on the interaction of the child's individual characteristics with the broader ecological context, including the family, social systems and culture to which the child belongs.[82] Being raised in a stable and nurturing family who are aware of the child's diagnosis and can adapt to the child's limitations and advocate for his or her needs reduces the risk of adverse life outcomes.[23] Many children with FASD, however, experience difficult caregiving environments with adversities such as maternal alcohol and drug abuse and depression (resulting in less responsive and less stimulating parenting), abuse and neglect, poverty, family violence and multiple foster care placements.[100]

There is very little systematic research on the impact of raising a child with FASD on the family, on how parent and family variables influence outcomes for people with FASD, or on interventions for families affected by FASD.[100] For this reason, Olson et al. drew on the findings from both systematic research, and the informal literature based on parents' accounts on raising a child with FASD, to highlight important childrearing and family issues.[100]

The issues identified from the informal literature included guilt and shame (for birth mothers), financial stress, the lack of knowledgeable professionals, the time needed to advocate for the child in the education and justice systems, the need for respite care, difficulties in determining the causes of a child's misbehaviour, the value of parent support and self-hep materials from parent support organisations, and the need for formal intervention services.[100]

Issues identified from research studies included the impact of maternal substance abuse and/or depression on infant attachment and maternal responsiveness, child externalising behaviour as the most significant cause of parental stress, the need for foster parents who can provide structure and a high level of organisation and are

well supported by a care team knowledgeable about FASD, parents' need for respite care and support in dealing with negative emotions regarding their child, the risk of caregiver burnout and depression, and the difficulties of dealing with multiple service providers, such as mental health, education, medical and social services, who may not agree on the best way of helping the child.[100]

Given the lack of research on interventions specifically for families affected by FASD, Olson et al. suggest that useful insights can be gained from the more general developmental disabilities literature, and the literature on traumatic brain injury, as families of children with these conditions face many problems that are similar to those facing families of children with FASD.[100]

Petrenko[82] suggests that the lived experiences of people with FASD, their parents and caregivers, and service providers are a valuable source of information for those developing interventions for people with FASD. A qualitative study done in up-state New York asked 25 parents of children with FASD (only one of whom was a biological parent) and 18 service providers for their views on desirable characteristics for intervention programmes aimed at preventing secondary conditions, such as mental health problems or trouble with the law.[101] Study participants reported that their children did not qualify for special education or disability support services because their IQ scores were too high although some were able to obtain such services after multiple appeals and advocacy. The study identified five key characteristics for FASD intervention programmes: availability across the lifespan; having a prevention focus; being individualised; being comprehensive; and being co-ordinated across systems and developmental stages. The study authors stated that the five key characteristics are consistent with the positive behaviour support framework which focuses primarily on adapting the environment to improve an individual's quality of life with reduction of problem behaviours as a secondary goal.[102]

Drinking in pregnancy as a public health issue in New Zealand

Alcohol is pervasive in New Zealand society. The 2012/2013 New Zealand Health Survey (NZHS) found that 76% of New Zealand women aged over 15 years had drunk alcohol in the past 12 months and that one quarter of women drinkers drank alcohol regularly, at least three to four times per week.[103] Almost half of all women drinkers had drunk to intoxication at least once in the past year and around five percent reported drinking to intoxication at least weekly.[103]

The NZHS included 565 women aged 15–54 years who had been pregnant within the past 12 months and 19% of them reported having drunk alcohol at some time during their most recent pregnancy.[103] Younger women were more likely to have drunk alcohol during pregnancy: 28% of 15–24 year olds compared to 17% of 25–34 year olds and 13% of 35–54 year olds.[103]

Of the women who had been pregnant in the last year, 31% reported that they had stopped drinking before becoming pregnant, 55% that they stopped drinking as soon as they learned they were pregnant, and 15% that they continued to drink while pregnant.[103] Of those who continued to drink, the majority reported reducing their drinking while pregnant.[103]

Binge drinking or heavy episodic drinking is the pattern of drinking recognised as the most harmful to the developing fetus.[5] For this reason, the NZHS enquired about past year risky drinking, defined as drinking four or more standard drinks on one drinking occasion, in women who had been pregnant in the last year and who reported drinking alcohol in their most recent pregnancy. It needs to be understood that rates for past-year risky drinking among women who drank during pregnancy are not necessarily the same as the rates of risky drinking during pregnancy. Of the women who reported drinking during pregnancy, 78% also reported risky drinking during the past year, including 11% who reported risky drinking at least weekly and 16% who reported risky drinking at least monthly.[103]

Another New Zealand survey, which was a retrospective survey of 723 post-partum women across New Zealand, found that overall, 34% of women reported drinking in pregnancy and that 12% of pregnancies were at high risk of heavy alcohol exposure in early gestation.[104] The percentage of pregnancies with heavy alcohol exposure in early gestation was almost five times higher in Māori women and 3.4 times higher in Pacific women, than in New Zealand European women.[104]

This survey found that 44% of pregnancies were unplanned and that pregnancy confirmation occurred later for women with unplanned pregnancies (7.3 vs. 5.3 weeks gestation).[104] Three quarters of pregnancies in Māori and Pacific women were unplanned.[104] The authors of this study suggested that this indicates that alcohol policies that facilitate the reduction of heavy episodic drinking throughout Māori and Pacific communities—not just among women planning a pregnancy and pregnant women—need to be adopted.[104]

Keriata Stuart conducted in-depth interviews with ten Māori women with the aim of understanding how Māori women negotiate decisions about alcohol in pregnancy.[105] She used a grounded theory methodology to analyse the information gathered and she proposed that Māori women make decisions about drinking during pregnancy using a process of *Trading off*. This process was considered to be individually based but to exist within a complex social context; a fluid process that continued throughout pregnancy. Māori women learned social rules about alcohol consumption within their whānau and social circles; their beliefs about alcohol consumption in pregnancy were acquired in much the same way. Māori women regarded health professionals as a less trustworthy source of information. Reasons why women continued drinking while pregnant included fitting in (it is hard to be the only non-drinker in a social group), releasing pressure (drinking to relax) and carrying on as normal. Supportive partners, friends and whānau surrounding a pregnant woman could make a major contribution to a Māori woman's resource bank making it easier for her to refuse offered drinks and change her drinking habits, and also helping her to deal with stresses in her life, maintain a positive self-image, take control, and act with self-determination.

The Growing Up in New Zealand study interviewed 6,822 expectant mothers in either their last trimester of pregnancy or soon after the birth of their child and asked them to recall their alcohol consumption in three periods: before becoming pregnant or before being aware they were pregnant; in the first three months of pregnancy; and after the first three months of pregnancy.[106]

Data from this study suggested that 71% of women drank alcohol before becoming aware of their pregnancy, 23% drank during their first trimester after becoming aware of their pregnancy, and 13% drank after their first trimester.[106] The proportions of women drinking four or more drinks per week were 29% before becoming aware of pregnancy, 7% in the first trimester and less than one percent after the first trimester.[106] The women who reported drinking in pregnancy were very heterogeneous in regard to their socio-demographic characteristics but the heavy drinkers were more likely to be younger women, Māori women, women with no secondary qualifications, smokers and women whose pregnancy was unplanned whereas the women who reported light drinking (three or fewer drinks per week) were more likely to be older women, European women and women from socio-economically advantaged backgrounds.[106]

A 2005 survey of a random sample of 1,109 non-pregnant New Zealand women aged 16–40 years aimed to assess New Zealand women's opinions on the safety of alcohol consumption in pregnancy and the sociodemographic and lifestyle factors associated with these opinions.[107] It found that 44% of women in the study were of the opinion that no alcohol is safe in pregnancy and 45% that one standard drink or less on any one day is safe. Only 10% thought that more than one standard drink was safe.[107] The moderate and heavy drinkers, who made up 55% of the study sample, were more likely than abstainers or light drinkers to be of the opinion that more than one standard drink on a typical drinking day during pregnancy is safe.[107]

In summary:

- Most New Zealand women drink alcohol (as do most New Zealand men)
- A sizeable minority engage in risky (binge) drinking
- Most women stop or reduce their drinking once they are aware they are pregnant
- Nearly half of all pregnancies are unplanned
- There is heavy alcohol exposure in early gestation in at least 10% of pregnancies
- Binge drinking and unplanned pregnancies are more common in younger women and Māori women
- Women have mostly got the public health message to stop drinking when they are pregnant
- Women have not got the message not to drink if they are planning to get pregnant or could get pregnant

Patterns of drinking associated with FASD

The earliest studies of the effects of pre-natal alcohol exposure, done in the late 1970s and early 1980s, involved identifying children with the facial features of FAS whose mothers who were chronic alcoholics.[2,108-112] The amounts of alcohol consumed by the mothers in these studies were considerable: for example, a study done in the west of Scotland reported alcohol intakes during pregnancy in the range from six measures of vodka daily to one bottle of vodka daily.[112] An early US study of 23 children of alcoholic mothers found that four infants died in the perinatal period (a mortality rate of 17%) and that 44% of the surviving children had borderline-to-moderate mental deficiency and 32% had enough features on physical examination to suggest fetal alcohol syndrome.[113]

A prospective study which began in Boston in 1974 questioned 633 women on their alcohol consumption at their first prenatal visit and classified women into three groups: abstinent and rare drinkers (326 women), moderate drinkers (249 women) and heavy drinkers (58 women, 9%).[114] The heavy drinkers were women who consumed five or more drinks on occasion and they also had a consistent daily intake of more than 45ml of absolute alcohol daily: the average daily intake in this group was 174 ml. A standard drink is defined in New Zealand as 12.7 ml (10g) of alcohol and is equivalent to one 330 ml can of beer or one 100ml glass of wine[115] so these women were drinking an average of around 14 standard drinks per day. Moderate drinkers were women who drank more than once per month but did not meet the criteria for heavy drinking. The babies were given detailed paediatric, neurological and developmental examinations two or three days after delivery by a physician with no prior knowledge of the mothers' or infants' histories. The infants of the heavy drinkers had significantly higher rates of congenital anomalies than the infants of mothers in the other two groups. Thirty-two percent of infants of heavy drinkers had congenital anomalies of any kind and 17% had major anomalies (compared to 3% and 2% in the other groups). Ten of the fifteen (67%) heavy drinkers who significantly reduced their alcohol intake during pregnancy had apparently normal babies but only two of the 27 (7%) women who drank heavily throughout pregnancy did.

While it soon became clear that daily heavy drinking by a pregnant woman was very hazardous to the fetus, attempting to determine exactly what level of alcohol consumption was dangerous was more difficult. Studies done in humans usually rely on mothers honestly and accurately recalling their alcohol consumption during a specific period and they often do not ask sufficiently detailed questions to establish the amounts and timing of drinking in relation to gestation or drinking patterns.[5]

In animal studies researchers can control the size, number, pattern and timing of alcohol doses given to the mother and so examine the effects of variations in alcohol exposure on the developing fetal brain. Animal studies have established that peak blood alcohol concentration is highly correlated with the degree of brain damage (as indicated by reduced brain weight). High blood alcohol concentrations are achieved by ingesting a large quantity of alcohol over a short period of time, a pattern of drinking known as binge drinking.[5]

Populations that have the highest rates of binge drinking tend to have the highest rates of FASD, and the highest ratio of FAS rates to partial FAS rates.[116] May and Gossage[116] compared rates of FAS and partial FAS reported from seven community studies and found the highest the highest ratio of FAS to partial FAS rates was in in South Africa, where mothers of children with FAS and partial FAS drank heavily (an average of 6.6 standard drinks per night) almost every Friday and Saturday evening.

The timing of maternal drinking determines which anatomical features of the fetus are affected. The facial features characteristic of FAS are the result of heavy drinking between the sixth and ninth weeks of gestation.[117] The central nervous system is developing for the whole nine months of pregnancy therefore brain damage can result from drinking at any time during pregnancy. It is possible that the specific pattern of behavioural and cognitive deficits seen in a child with FASD is related to the timing of the mother's drinking episodes and future research may determine which regions of the brain are linked to particular deficits and behaviours.[4,117]

Factors that modify the risk of FASD

Even if two pregnant women, or one woman during two different pregnancies, have a similar quantity, frequency and timing of drinking, the resulting children may not be affected to the same degree.[117] The incidence of FASD among the children of women who drank heavily in pregnancy is far from 100%. A 1995 review of 29 prospective studies estimated the incidence of FAS among heavy drinking women (defined as those who had average of 2 or more drinks per day, or 5 to 6 drinks per occasion, or a positive score on the Michigan Alcoholism Screening Test, or clinical diagnosis for alcohol abuse) as 4.3%.[118]

Maternal factors that have been found to increase the risk and severity of FASD are higher maternal age, higher number of previous pregnancies, higher number of previous births, smaller body size, undernutrition, low socio-economic status, smoking, other drug abuse and depression.[117]

Genetic factors are probably involved in susceptibility to FASD and there is continuing research in this area.[119] There has been found to be greater concordance of FASD diagnosis and IQ in identical than non-identical twins of alcoholic mothers.[120]

Women who have children with FASD frequently come from heavy drinking families, have heavy drinking partners, move in social circles where heavy drinking is common, and are socially isolated from the mainstream economy and society.[117]

Is light or moderate drinking in pregnancy hazardous?

Government agencies in developed countries, including New Zealand's Ministry of Health,[121] commonly advise that even low levels of alcohol intake may be harmful to unborn babies and that pregnant women should abstain from alcohol.[122-124] There is conflicting evidence about whether occasional or light drinking in pregnancy is a risk factor for later mental health or learning problems in exposed children but most studies have not found any significant detrimental effects.[125-132]

A 2007 systematic review of 46 studies looking at the effects of low to moderate alcohol intake on miscarriage, stillbirth, intrauterine growth restriction, prematurity, birthweight, small for gestational age at birth and birth defects including fetal alcohol syndrome found no convincing evidence for any of these outcomes. This review's authors stated that many of the studies had methodological weaknesses therefore the evidence precludes concluding that low to moderate drinking in pregnancy is safe.[133]

All studies of prenatal alcohol in humans rely on maternal self-report and most have not taken into account the amount, timing and pattern of maternal drinking.[134] If studies classify women's drinking according to average alcohol intake per week then many women who are heavy or binge drinkers on the occasions when they do drink may be classified as light drinkers because their average intake is less than one standard drink per day.[134] This may have contributed to some studies failing to find differences between different groups of drinkers. An Australian study examining the association between prenatal alcohol exposure and fetal effects compared the results of using three different published methods of quantifying maternal alcohol consumption with a composite method of categorisation that combined total quantity, dose per occasion and frequency.[134] It found that effects of moderate and binge drinking were only evident when the composite method was used but effects from heavy drinking were evident and similar with all methods.

A 2012 review of animal studies of moderate alcohol intake found that it was associated with long lasting neuro-behavioural deficits including alterations in learning, memory, motor coordination, social behaviour, and stress responses.[135] It was also associated with CNS changes that could account for these behavioural effects including alterations in neuronal migration, adult neurogenesis, neurotransmitter receptor function, synaptic plasticity, and intracellular signalling pathway. The review defined moderate alcohol intake as that producing a blood alcohol level of ≤ 0.17 g/dl, which has been assumed to model human moderate alcohol intake. (New Zealand's drink drive legal alcohol limit is 0.05 g/dl.[136])

Prevention of FASD

Prenatal brain damage caused by alcohol is irreversible but totally preventable. Heavy drinking and drinking to get drunk are a significant part of New Zealand's drinking culture.[137] Compared to previous generations young women are drinking from an earlier age and drinking greater quantities when they drink.[138] Fetal damage is only one of the many individual and community harms produced by alcohol abuse.[137] There is not space here to discuss in detail strategies to reduce alcohol-related harm in the whole population. The Law Commission's 2010 report *Alcohol in our lives: Curbing the harm*[139] states that the international literature indicates that there are seven major policy levers for regulating alcohol-related harm:

- Regulating the physical availability of alcohol through restrictions on time, place and density of outlets
- Regulating conduct in commercial drinking establishments
- Taxing alcohol and imposing controls on price
- Regulating advertising, promotions and marketing
- Imposing penalties for alcohol-related anti-social behaviour such as drink driving
- Education and persuasion with the provision of information
- Increased availability of treatment programmes with screening and brief interventions in health care.

Only the last two of these are within the ambit of the health system and will be considered here.

Communication and education strategies

A 2014 literature review for the Health Promotion Agency, entitled *Drinking alcohol in pregnancy,*[140] considered, among other things, research on primary prevention communication strategies. These strategies aim to influence knowledge, beliefs and attitudes about alcohol use in pregnancy and they can support clinical strategies, such as brief interventions, that may be used in maternity or other healthcare settings.[140] Communication strategies include media campaigns, social marketing approaches, educational materials, messages for healthcare providers and health warning labels on alcohol containers.[140]

Although there have been many communication campaigns addressing alcohol use in pregnancy, these have rarely been based on theory or on formative research that could help identify the target audience, the target behaviours, and the factors that influence the behaviours of the target audience.[141] Campaign evaluations have tended to be of only fair to poor quality and have often not drawn any meaningful conclusions.[141] There is therefore a lack of evidence to inform the design of campaign strategies and messages.[141] Despite this, some best practice approaches have been identified from reviews of Canadian campaigns,[142-144] as follows:

- Campaigns should be carefully planned with objectives that are specific, realistic, attainable, measurable and time-specific

- Campaigns should be one component of a broader strategy and should involve a wide range of partners

- Campaigns should be carefully designed for a specific group or groups. Campaigns are most likely to be effective in larger, well-defined lower-risk groups

- Campaigns should assess the current levels of awareness in the target audience and focus messages on areas where awareness is low

- Campaigns should have good exposure and reach to make messages more likely to be heard and remembered.

There is limited evidence on the specific elements that make a campaign message effective.[140] It has been suggested that messages need to take a positive and supportive approach and avoid the use of blame, shame and fear-based strategies.[143] They can link people to further information, services and support.[143] The Ministry of Health's Action Plan notes that women are currently receiving mixed messages about the risks of drinking during pregnancy and it emphasises the need to disseminate a clear, consistent and unambiguous message that women should stop drinking alcohol if they could be pregnant, are pregnant or are trying to get pregnant as there is no known safe level of alcohol consumption during pregnancy.[7]

The Health Promotion Agency's review describes a number of international communication campaigns aimed at reducing or preventing alcohol use among pregnant women that have been evaluated through assessing campaign recall, changes in awareness and knowledge, and intended changes in behaviour in the target audience. It also provides brief details on some recent non-evaluated campaigns including the *Babies and Booze* social media campaign developed in Auckland.[145]

There is a lack of evidence for the efficacy of public health campaigns in changing behaviour (as opposed to knowledge or intentions) regarding consumption of alcohol in pregnancy.[146-149] There is also little evidence that mass media campaigns have been effective in modifying alcohol use in the general population, except in relation to drink driving.[150-152]

Identifying at-risk women

Interventions to prevent FASD need to be directed both at the general population of women of childbearing age (universal prevention) and at the women at highest risk of having a child with FASD such as women with alcohol addiction, women who themselves have FASD, and women who already have a child with FASD (targeted and indicated prevention). It is necessary to identify women with risky drinking patterns and women who are alcohol-addicted so that they may be helped to reduce or stop their drinking, to deal with the factors that underlie their drinking, and to use effective contraception. Research studies have consistently identified high levels of pre-pregnancy drinking as being predictive of drinking during pregnancy.[153]

Women may not disclose their alcohol abuse to health practitioners for various reasons including embarrassment and denial. The Ministry of Health recommends that primary care health professionals ask women who are planning a pregnancy or are pregnant about whether they drink alcohol; provide brief advice about not drinking alcohol when planning a pregnancy or when pregnant and explain why; and assist women who are having difficulty stopping, or whose drinking is problematic, by directing them to addiction treatment services.[154] A *Pregnancy and Alcohol Cessation Toolkit*[155] has been developed to support health professionals in this.

There are a number of standardised screening questionnaires that can be used to identify problem drinking.[156,157] A 2010 systematic review[157] looked at cohort or cross-sectional studies that had compared that compared one or more brief alcohol screening questionnaire(s) with reference criteria obtained via structured interviews to detect 'at-risk' drinking, alcohol abuse or dependency in pregnant women receiving prenatal care. The review authors identified five studies that evaluated the sensitivity, specificity and positive predictive value of seven brief screening questionnaires. They concluded that three questionnaires showed promise for screening for risky drinking in pregnant women: T-ACE (Take[number of drinks], Annoyed, Cut down, Eye-opener), TWEAK (Tolerance, Worried, Eye-opener, Amnesia, Kut down), and AUDIT-C (Alcohol Use Disorder Identification

Test – Consumption). They stated that AUDIT-C might also be useful for identifying alcohol dependency or abuse.

Once a woman has been identified as having an alcohol problem, depending on the severity of the problem, she can be offered advice, a brief intervention, or referral to an alcohol treatment service.[157]

Interventions to reduce alcohol-exposed pregnancies

Brief interventions for pregnant women typically involve from one to four short counselling sessions with a professional trained in motivational interviewing, such as a midwife, GP or social worker, followed by personalised feedback.[157] There have been a number of randomised controlled trials of brief interventions for alcohol use in pregnant women[158-162] and the results of these suggest that brief interventions may be useful in reducing pregnant women's alcohol consumption. The Cochrane review that assessed these trials found that the evidence is insufficient to determine the type of brief intervention that is likely to be most effective.[163]

O'Connor and Whaley examined the effectiveness of a brief intervention in helping low-income minority women in Southern California achieve abstinence from alcohol during pregnancy. The intervention consisted of 10–15 minute counselling sessions guided by a scripted manual. It was delivered in accessible community-based settings by nutritionists from the Public Health Foundation Enterprises Management Solutions Special Supplemental Nutrition Program for Women, Infants, and Children (PHFE-WIC). Twelve 12 PHFE-WIC centres were randomised into assessment only or assessment plus brief intervention and this yielded 255 pregnant drinkers as study participants. Women who received the brief intervention were five times more likely to report abstinence at third trimester follow-up than the women who received assessment only and their infants had higher birthweights and birth lengths, and a lower mortality rate (0.9% vs. 2.9%).

Since almost half of all pregnancies are unplanned,[104] there is a need for preconception interventions that help drinking women to use effective contraception and/or help women who might become pregnant to reduce or stop their drinking. In the US, randomised controlled trials have indicated that several motivational interviewing-based interventions targeting risky drinking and ineffective contraception are effective in reducing alcohol-exposed pregnancies (AEPs).[164]

CHOICES[165,166] is a motivational interviewing plus feedback counselling intervention designed to reduce AEPs among community women.[164] It involves four 30–75 minute counselling sessions and a medical contraception counselling appointment. A multi-site RCT that compared CHOICES to provision of an informational brochure found a significant 18% reduction in AEP risk (due to reductions in risky drinking, increases in contraception effectiveness, or both) in participants who received CHOICES.[165]

BALANCE was a modification of CHOICES that contained the same components as the original intervention condensed into a single session pre-conception motivational interview plus assessment feedback counselling intervention. It was tested in a RCT among college women aged 18–25 at risk of AEP.[167] The intervention recipients were encouraged to have a contraception counselling visit with a medical provider through student health services but this was not a formal part of the intervention as it had been in CHOICES.[167] At four-month follow-up 80% of BALANCE recipients, vs. 65% of participants who received the information brochure, reported no AEP risk, a 15% absolute risk difference.[167,168]

EARLY was an adaptation of CHOICES and BALANCE for community women. It was recognised that the college women involved in the BALANCE trial were typically younger and less likely to have already given birth than community women, and also less likely to have other psychosocial risks associated with increased AEP risk including a history of tobacco smoking, recent drug use, a history of inpatient treatment for addiction or mental illness, multiple sexual partners or recent physical abuse.[169] While CHOICES had been shown to be efficacious in community women it was resource-intensive which made it challenging to implement on a large scale.[164] EARLY aimed to address both these issues. EARLY was a 60-minute, face-to-face, individual, one-session, motivational interview plus assessment feedback counselling intervention.[164]

During the intervention counsellors built rapport, discussed reactions to the baseline assessment, elicited views on drinking and use of contraception, provided personalised feedback, showed that participant a video about AEP and FASD an elicited the participant's response, discussed drug use and other mental health issues pertinent to prevention of AEP if indicated, selected and performed at least one intervention activity, encouraged a contraception visit with a reproductive healthcare practitioner, and summarised the intervention.[164] The intervention activity addressed the issue (contraception or drinking) that appeared to be most relevant or of greater interest to the participant through addressing ambivalence, readiness, tempting situations or goal setting.[164]

A three-arm RCT involving 217 participants compared EARLY, an informational video, and an informational brochure.[164] All three interventions were associated with significant decreases in ineffective contraception rates at six months and small decreases in drinks per drinking day. Compared to the other two interventions in this trial, EARLY led to greater reductions in ineffective contraception and AEP rates (although the differences were not significant) but not in drinks per drinking day. Compared to previous RCTs of CHOICES and BALANCE, EARLY produced smaller risk reductions, and, unlike CHOICES and BALANCE, it did not perform better than the comparison interventions in its RCT. The study authors suggested that raising women's awareness of their drinking habits through assessment seems to reduce women's drinking even if no further intervention is provided. Other studies have also found this to be the case.[159,170-172] This phenomenon may lead to RCTs that rely on assessment underestimating the effects of interventions.[171]

A program of screening and alcohol brief interventions (ABIs) has recently been implemented in antenatal care settings in Scotland.[173] A qualitative study was undertaken to explore midwives attitudes and practices regarding alcohol screening and ABI.[173] The study involved semi-structured interviews with 15 midwives and a focus group of six midwifery team leaders. Interview transcripts were analysed in a thematic analysis. Midwives were positive about being involved in the screening and ABI programme but they were not completely convinced of the programme's value in antenatal care. They felt that, at the first antenatal appointment, they had not established sufficient rapport with a pregnant woman to be able to discuss alcohol issues with ease although they recognised that early in pregnancy was the best time to do this. They pointed out that many women were already not drinking or drinking very little prior to the first antenatal appointment. Midwives were concerned that the women who had not reduced their drinking were the group most likely to be alienated by discussion of alcohol issues. They thought that pre-pregnancy preventive measures would have a greater impact on reducing alcohol-exposed pregnancies.

Conclusion

Fetal alcohol spectrum disorder is a significant threat to the health and well-being of New Zealand's children. Prenatal exposure to alcohol can have devastating life-long consequences both for the individual child and for those he or she comes into contact with. Fetal alcohol spectrum disorder is exceedingly costly for the health, education, child welfare, justice, mental health and social welfare systems.

Although early identification and support is helpful for children with FASD and their families the evidence base for interventions to ameliorate the cognitive and social deficits resulting from alcohol-induced brain damage is limited. Even with the best care during childhood and adolescence, many of those with FASD will still need continuing support to manage their daily lives in adulthood. It is vital that we change New Zealand's drinking culture from one that encourages and tolerates alcohol abuse to one that supports moderation as normal drinking behaviour.

Health services need to ensure that women of reproductive age do not face barriers to seeking help with alcohol and drug problems or associated mental health issues, and that women who have difficulty moderating their alcohol consumption have access to effective contraception.

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HEALTH NEEDS OF CHILDREN AND YOUNG PEOPLE IN STATE CARE

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Introduction

This chapter reviews health needs of children and young people in State care and outlines what is required of health services to meet these needs. The aim of this chapter is to inform health service providers of some of the key issues to be addressed in developing and expanding services for children and young people in State care. The voices of children and young people who have experienced State care are included where possible, highlighting the importance of their participation in service development and provision. The chapter is not an itemised catalogue of evidence for best practice in relation to specific health conditions; this information is provided elsewhere through clinical networks and guidelines referred in NZCYES reports. This chapter presents some key aspects that will be important to deliver high quality child-centred services to some of the most vulnerable children in New Zealand.

Children and young people in State care have experienced a lack of nurturing care, and this lack can have a serious impact on their health. In 2016 it was reported that 4,394 (83%) of the 5,312 children and young people in State care lived in out-of-home care either with members of their whānau or extended family, or in foster care with people to whom they are unrelated.[1] Children in out-of-home care live very complex lives. They experience divided loyalties to the people they live with, who provide the day-to-day care and protection they need, and also to their birth families for whom they may have strong but ambivalent feelings.[2] These children need intensive support so that they have the chance to develop a sense of stability and to enjoy the same opportunities and outcomes as other New Zealand children.[3]

The complex experience of children and young people in State care has important implications for health services. For health service systems to enable continuity of care, they need to take account of changes in caregivers and in domicile which can occur multiple times for some of those children in State care.[4] Strong co-ordination between District Health Boards (DHBs) is important so that when children or young people move they do not have to start again from the beginning of a health assessment process. Within DHBs it is important for child health services and child and adolescent mental health services (CAMHS) to work together to meet the needs of vulnerable children and their whānau. Effective services for children and young people in State care involve multiple government agencies, including health, education and justice, as well as non-government organisations. Information sharing between agencies and organisations is an essential part of ensuring that the needs of children and young people in State care are prioritised and met.[3]

More than half of the children and young people in State care have Māori recorded as their primary ethnicity.[5] Processes to embed a high degree of cultural competence and confidence are required to meet the needs of all children and young people in State care.[3] In this chapter, the principles of the Meihana model have been used as a framework to present findings from the international scholarly literature about the health needs of children and young people in State care. The Meihana model acknowledges physical, spiritual, psychological and family dimension of health and wellbeing, as well as the role of the services and systems and the physical environment in contributing to the health outcomes.[6] International experience has been drawn upon to identify possible ways forward from the perspective of the health sector.

The chapter concludes with a description of the child protection process in New Zealand and why and how children and young people are taken into State care. In any one year, about 5000 children and young people in New Zealand are in the custody of the Chief Executive of Child, Youth and Family. They have been through a formal assessment and investigative process and found to need statutory care and protection.[1] Further detail from recent reviews is also included with an overview of the proposed changes to the child protection system.

Experiences of children and young people in State care

Children and young people who enter State care have been exposed to a lack of appropriate and consistent parenting and in particular, a lack of nurturing care, which impacts significantly on their wellbeing. The report of the Modernising Child, Youth and Family Expert Panel described a marked difference between the intentions of New Zealand's child protection system and the nature of the actual experience described by young people.

Although the experiences of the young people differed greatly, themes of chaos, disruption, a sense of loss and abandonment came through very strongly during the interviews with them. Their subjective experience was dominated by negative emotions such as anxiety, powerlessness and grief. To them, the system lacks humanity and struggles to keep children and young people safe, let alone helps them recover from the impact of abuse and neglect.[7]

Transition into State care often meant that there were abrupt changes in young people's lives, for example, separation from their siblings, living in a different geographical location and having to change school. Few young people reported any support to help them manage the trauma and emotional impact of being removed from their family or being moved from placement to placement.[7] Finding a sense of family in an environment where they feel accepted and loved is critical for young people. Caregiving arrangements need to be well matched to the young person's age, gender and stage of development. A key mark of effective caregivers is that they identified a strength area or a source of happiness and supported the young person to excel at something.[7]

The expert panel heard from 19 young people about their experiences in the New Zealand care and protection system. The key messages from these young people are shown in **Box 1**.[7]

Box 1. Main things told by young people to the Modernising Child, Youth and Family Expert Panel

> ***We need more nurturing and love***
> The young people told us the system often did not provide them with the nurturing and love they required. They felt their caregivers should provide more than food, shelter and supervision – they should also provide an environment where children and young people could learn, grow and heal.
>
> ***We want a say in what happens to us***
> The young people felt they did not have a voice in important decisions being made about their futures. They felt people involved in the process were not being honest and transparent about the decisions that were being made. This left them confused, anxious and disempowered.
>
> ***We have experienced trauma and need help to make sense of what has happened to us***
> Young people described being expected to transition into new environments frequently with little support. Young people commented that they need to be empowered to make sense of what they have been through and the reasons why things have happened to them.
>
> ***We crave belonging and being part of a family who bring out the best in us***
> Finding a sense of family is critical for young people. Young people were concerned that if others knew they were in care then they would also know that they did not belong to anyone. Many reported the life-changing impacts of finding 'the one' adult who understood and supported them.
>
> ***We want to strengthen our cultural identity and connection***
> Young people talked of the value of cultural connections, especially in relation to building their sense of identity and wellbeing. They felt this was not well recognised or supported, and adults did not understand the importance of connection and under-valued it.
>
> ***We do not stop needing help, support and nurturing just because we turn 17***
> Some young people felt very unprepared, stressed and vulnerable when they age out of the care system at the age of 17. For some young people leaving care, the only option is going back to the unsafe environments they were initially removed from.

Source: Modernising Child, Youth and Family Expert Panel. 2015. Interim report.[7] (pp. 8–9)

The messages from young people who met with the Expert Panel were consistent with what the Children's Commissioner heard from children and young people in the custody of the Chief Executive of Child, Youth and Family (in CYF care) through surveys and focus groups. Those in CYF residences were generally positive about their experience but experiences were more variable for those in-kin and non-kin out-of-home care. Some described overwhelmingly negative experiences including abuse in foster homes, separation from siblings, constant change, "Bible bashing", depression, alcohol and drug use.[5] The messages by children and young people in the 2016 *State of Care* report can be summarised as follows:[8] (p. 7)

- **We need to feel like we belong.** Children and young people wanted help to manage relationships with family, opportunities to learn about and connect with their culture, and to be able to enjoy their childhoods with a range of activities, a positive school life, and no stigma attached to being a "CYF kid."

- **Involve us, listen to us, and communicate with us.** Children and young people wanted to be given a voice in decisions that affect them, involved in care plans and transition planning, and communicated with clearly and respectfully.

- **Social workers have a big impact on our lives.** Children and young people spoke in detail about their interactions with social workers. Their comments highlighted that, as the chief interface between the child or young person and CYF, the social worker plays a critical role in determining whether the child has a positive or negative experience in the care and protection and youth justice systems. A child in non-kin foster care stated "[My social worker is] kind of like family now; like a friend now."

Health needs of children and young people in State care

There has been limited research on factors associated with wellbeing of children in care in New Zealand, with a tendency to rely on international research to inform policy and practice.[9] Recent reviews have found that current systems in CYF and in the Ministry of Social Development (MSD) are not set up to measure and record the most important information about children in care. Reviews have also found that there is poor integration between MSD and other government agencies, including health.[5] Nevertheless it is recognised that families of children who are referred to CYF have often experienced high levels of long-term need and disadvantage, often including combined effects of long-term unemployment, low income, unaddressed physical and mental health needs, addiction disorders and family violence.[7]

Gateway assessments have been provided since 2011 for children and young people who may be at risk of coming into CYF care, entering care or already in care.[10] Gateway assessments are managed by DHB co-ordinators, after referral of children by CYF, and include assessment by health professionals. The Expert Panel observed that health needs were identified frequently among the children and young people who had completed a gateway assessment. For every ten children or young people who completed a gateway assessment:[7]

- Three had a mental health need
- Five had emotional needs
- Three had developmental needs
- Six had learning needs
- Three had dental needs
- Four had needs as a result of parent or caregiver alcohol or drug addiction.

Research in other countries has shown that children and young people in State care are a very vulnerable group with relatively high levels of unrecognised or unmet health needs.[11] Children in State care have been classified as a population of children with special health care needs in the USA and in the UK.[12] Understanding and recognition of the health needs of children and young people in State care is important so that plans are in place for them to receive health care wherever they are placed, and to appropriately review the services in place for them.[11] In addition to their important health care needs, children and young people in State care experience barriers in accessing appropriate and continuous care that can worsen their prognoses.[13] In this chapter the concept of te waka hourua, or the double-hulled waka, is used as a framework to present findings from research about the health needs of children and young people in State care.[6]

To date there has been inadequate research about the long-term outcomes of foster care. By long-term outcomes researchers are referring to what happens among young adults who were in State care as children. This group is referred to as having 'aged out' of care. Prevalence studies on specific populations of young people who have aged out of foster care show high rates of clinically significant mental health problems (54%), chronic medical illness (30%), unemployment (19%–37%), poverty (33% live at or below the US poverty level), lack of health insurance (33%–50%), and homelessness within one year of leaving care (22%–36%). High school completion rates vary and completion may occur years after leaving care. One study of young adults who lived in foster care during adolescence found a prevalence of post-traumatic stress disorder (PTSD) twice that of combat veterans. Although these prevalence studies of high-risk adult populations are not representative and therefore cannot be used to indicate 'cause and effect', they do provide a measure of the ongoing health problems affecting young people who leave State care as they transition to adulthood.[12]

It is difficult to compare information about children and young people in State care in different countries, mainly due to different approaches, organisational structures and definitions of formal out-of-home care.[7,13]. In many countries the categories are broader than that used in New Zealand for children and young people in CYF care, for example many jurisdictions include children and young people in voluntary placements with family or friends and not only those formally in statutory care. Also, in many jurisdictions monitoring and surveillance systems continue to follow young people after they have left State care. Despite these limitations, the literature suggests that all children who have contact with child welfare services share similar risk factors associated with a higher likelihood of poor long-term outcomes whether in out-of-home care, kin care or remaining with parent(s).[12] The following terms can be considered as synonyms in this part of the report, bearing in mind that they may describe slightly different population groups dependent on national definitions: looked-after children and young people; looked-after and accommodated children; children in care; and children and adolescents involved in the child welfare system (CWS). Children in foster care refers to out-of-home care in a family with kin or non-kin carers, and children in kinship care refers to out-of-home care within a child or young person's extended family.

Te waka hourua

The concept of te waka hourua is a key component of the Meihana model, which has been developed to enable health practitioners to develop a broader understanding of Māori patients and their needs.[6] The principles of the model are not unique to Māori, and in this section are applied more widely to all children and young people in State care. Importantly, statistics show that over 50% of children and young people in CYF care identified with Māori as their primary ethnicity and this model is therefore directly relevant to them.[5]

Te waka hourua was the traditional mode of transport used in the migration of Māori from Hawaiki to Aotearoa (New Zealand). The two hiwi (hulls) represent the child or young person and their family or whānau on a journey to health and wellbeing. The hiwi are joined by five crossbeams:[6 (p. 108)]

- Tinana: Physical health and functioning
- Hinengaro: Psychological and emotional wellbeing
- Wairua: Beliefs regarding connectedness and spirituality
- Taiao: Physical environment including home environment and neighbourhood
- Iwi katoa: Services and systems that provide support within the health environment

The journey toward health and wellbeing of the child or young person and their whānau or family is also impacted by nga hau e wha, the four winds, that represent wider societal and historical influences. The forces of colonisation, racism, internal migration from traditional iwi land, and marginalisation have significant influence on the journey and are important components of clinical assessment.[6]

The concept of nga roma moana, or ocean currents, reflects the way in which each specific child or young person, and their whānau, relate to te ao Māori (the Māori worldview), including use of te reo Māori, tikanga Māori including karakia and prayer, whānau roles and responsibilities, and specific geographical and genealogical connections, or whenua.[6] When health practitioners identify aspects of Te Ao Māori that are important to a child or young person and their whānau it can help all involved to share more freely in the clinical setting and thus facilitate better care.[6] Health practitioners can also provide more culturally appropriate services when they become familiar with specific cultural principles and integrate these within clinical practice.[6]

Whānau

Whānau may refer to biological family and/or other key support people.[6] Occurrence of child abuse strengthens rather than diminishes the importance of the whānau: "Parental rights often tend to be seen as secondary to the interests of whānau … to ensure that future generations are protected."[14 (p. 73)] Many young people with experience of the New Zealand care and protection system indicated to the Expert Panel that they "wanted to remain in the very environment they were removed from. They could not understand why the offending adult(s) remained at the home while the child was the one taken away."[7 (p. 41)]

> *I just thought it took only one call for me and my sister to be uplifted without a question, without even coming in to meet my dad or anything. I mean I understand now he drank a lot and my mum and dad used to fight but we were never hit, we were never mistreated, we got our food, we went to school sometimes, and I can understand why they took us away now, but being a young kid and being taken away, you think oh my gosh my parents are like awesome, they're like the best.* Female, 19, about experience in CYF care[7 (p. 41)]

It is of critical importance that children and young people in out-of-home care find a sense of family in an environment where they feel accepted and loved. A key mark of effective caregivers is that they identified a strength area or a source of happiness and supported the young person to excel at something (such as cheerleading, music, sport, dance, bike repairs).

> *… But if you put them in something they enjoy and something they're good at, they will be able to see the potential in themselves. I loved it because I got involved in sport. I was so involved in sport that I didn't have time to rebel and then with that family I started to see the importance of school and education because the oldest daughter she was actually my best friend and because she was in my class I was always hanging out with her. I would always do my work, and she was just like a really good role model even though she was my age. I graduated high school because of them.* Female, 17, about experience in CYF care[7 (p. 43)]

A competent, caring, nurturing, stable foster or kinship placement can be very important in supporting and advocating for the health and wellbeing of a child or young person. For children who have suffered severe neglect and abuse, placement in non-kin foster care or kinship care can be an important opportunity for intervention and healing.[12]

Tinana

Childhood trauma and adversity contribute to development of a number of health issues, and the ongoing loss and uncertainty in out-of-home care may exacerbate rather than ameliorate problems.[12]

Children and young people in State care have at least the same health needs as other children. The New Zealand Health Survey estimates that 17% of all 0–14 year olds and 24% of Māori 0–14 year olds have medicated asthma.[15] It is reasonable to expect similar prevalence amongst children and young people in State care. It is important to identify allergies, especially for those children who have anaphylaxis, as the carers need to be aware of substances/foods to avoid and to receive advice on management.[11]

There are, however, other health conditions more prevalent among children and young people in State care than among other children, including incomplete immunisation status and developmental disorders, as well as substance use, early sexual initiation and teen pregnancy among young people.[11,12] In the US, studies suggest that 30–80% of children who enter foster care have at least one physical health problem and at least one-third have a chronic health condition. These health problems are commonly undiagnosed and untreated before the child enters State care.[12] The authors also noted particular health issues for adolescents in foster care, who were at high risk of having untreated mental health conditions as well as educational, physical and reproductive health and psychosocial needs. Around 50% of young people in foster care had chronic medical problems unrelated to behavioural concerns.[12] Young people in care are a diverse group who may have come into care as a result of behavioural issues such as school truancy or petty criminal activity, or may have grown up in State care. This research identified that whatever the circumstances, most young people in State care have experienced neglect or abuse at some point in their lives. Those entering foster care as adolescents report high rates of exposure to violence, substance abuse and school failure with low literacy levels. Those who have positive peer relationships and adult mentors seem to fare better than those without. By age 18 years, 93% of youth in foster care reported being sexually active and almost half of these youth reported sexual initiation before the age of 16 years. Compared with peers not in foster care, foster care youth were approximately 2.5 times more likely to experience a pregnancy by age 19 years. Youth in foster care are more likely than youth not in foster care to report unintended pregnancy and less likely to use regular contraception. Repeat pregnancies among youth in foster care occur in almost half of all young women, compared with less than one-third of their peers not in foster care. Young adults in foster care are almost twice as likely as their peers to have experienced "forced sex".[12 (p. e1148)] Youth in foster care not only experienced substantial health issues, but also experienced difficulties transitioning from adolescence to adulthood with higher rates of unemployment, poverty, and homelessness than youth not in foster care.[12]

A comprehensive audit of a representative sample of 237 looked-after children in the London Borough of Hillingdon (LBH) in the 2014–2015 year found that 79.5% of the children and young people had at least one physical, mental or behavioural health need, 17% had a physical or mental impairment which had a substantial and long-term effect on the ability to undertake normal daily activities (recognised disability), and 3% had extremely complex needs. Prevalence of allergy and of overweight/obesity were comparable to prevalence in the general population. While it is encouraging that looked-after children were not more overweight than the local population the authors note that healthy diet and exercise continues to be important. Health needs of looked-after children and young people in LBH differed by age group, with higher prevalence of physical health needs, including developmental needs, at younger age groups and higher prevalence of mental health needs in older age groups (See **Box 2** for further details).[11]

Box 2. Physical health needs by age group of children and young people in State care, representative sample of looked-after children, London Borough of Hillingdon (United Kingdom) 2014–2015

> **< 1 year**: 87% of looked-after children in this age group had one or more health needs. 56% had experienced antenatal substance misuse/neonatal abstinence syndrome; 32% had neurodevelopmental problems; 19% had congenital abnormalities.
>
> **1–4 years**: 78% of looked-after children in this age group had one or more health needs, with 10% having complex needs. 14% had suffered antenatal substance misuse/neonatal abstinence syndrome; 53% had neurodevelopmental problems, 21% had some toileting difficulties; 21% had both physical and emotional problems.
>
> **5–9 years**: 61% of looked-after children in this age group had one or more health needs. 42% had neurodevelopmental problems or speech and language difficulties.
>
> **10–15 years**: 62% of looked-after children in this age group had one or more physical health needs. 1.5% had congenital abnormalities; 7% had dental problems, such as caries/orthodontics/enamel loss; 11% had gastrointestinal problems; 12% had skin problems, mostly acne; 4% presented with various injuries.
>
> **16–17** years: 66% of looked-after children in this age group had health needs, with half of these having only one health need. Symptoms included: skin conditions (17%); musculoskeletal (16%); gastrointestinal, including liver disease (13%); ear nose throat (12%); respiratory (10%); dental issues (4%); neurodevelopmental difficulties (9%); gynaecology, including pregnancy and female genital mutilation (7%).

Source: Health needs audit for looked after children and young people in the care of the London Borough of Hillingdon[11]

Hinengaro

To provide appropriate treatment for children, the importance of the role of childhood trauma in the emergence of child mental health problems needs to be understood. Children who have experienced trauma, especially those who have lacked appropriate caregiving and treatment, may develop depression over time or may have comorbid mental health problems.[12] Bronsard et al.'s systematic review and meta-analysis of eight epidemiological studies across different countries demonstrates the complexity of mental health screening and care in children and young people in the child welfare system (CWS). For these children and young people externalised and internalised mental health and emotional disorders are associated and complexly entangled.[13] The authors found that almost half (49% pooled prevalence) of children and adolescents in child welfare systems met criteria for a current mental disorder, a prevalence almost four times higher than in the general child and adolescent population.[13] They also cite the clear association between attachment insecurities and vulnerability to mental disorders and posit that the central role of child-to-parent attachment in a child's development may explain the high prevalence of mental disorder observed in their work. Adverse experiences of children and adolescents in the CWS, including maltreatment and serious neglect, reduce the likelihood of creating secure attachments that are crucial for developmental health. Multiple placements and temporary or disrupted relationships with caregivers can also potentially prevent the formation of secure attachments by these children and adolescents.[13] Multiple placements and maltreatment during the time of placement may contribute to worsening of pre-existent externalised mental disorders or promote emergence of such disorders. Externalised disorders were the most prevalent mental disorders in the meta-analysis: 27% of children and adolescents met the diagnostic criteria for a disruptive disorder including 20% with conduct disorder (10 times higher than child and adolescent population prevalence) and 12% with oppositional defiant disorder (three times higher than child and adolescent population prevalence). The estimated 11% prevalence of attention deficit hyperactivity disorder (ADHD) among children and adolescents in the CWS was three times higher than that of their peers. The prevalence of internalised disorders such as anxiety disorder (18%) and depressive disorder (11%) was also three to four times higher than the prevalence of these disorders in the general child and adolescent population. Limitations to this systematic review included heterogeneity of results, possibly influenced by factors such as differences between countries in interpretation of symptoms, and patterns of child welfare provision, and the small number of epidemiological studies describing children and young people in State care.

Szilagyi et al.'s data suggest that children in foster care are prescribed psychotropic medications at a rate three times that of other Medicaid-enrolled children in the US and they have higher rates of polypharmacy. Once psychotropic medications are prescribed, children in foster care are likely to be kept on them longer than children who are not in foster care. Treatment with psychotropic medications may not address the underlying trauma and attachment issues at the root of challenging behaviours. Factors that may contribute to the apparent overtreatment with psychotropic medications include caregiver demand for medication to manage disruptive behaviours, lack of understanding of childhood trauma, lack of paediatric mental health resources, and misdiagnosis of trauma symptoms as other mental health conditions, such as attention deficit hyperactivity disorder.[12] Szilagyi et al. note that psychotropic medications should be prescribed for children and young people in State care only after a mental health evaluation and trauma assessment by a child mental health expert and only for a specific mental health diagnosis. The medication should be appropriate to the diagnosis, initiated at

the lowest appropriate dose, and increased slowly while monitoring for efficacy and adverse effects. Polypharmacy should be avoided whenever possible.[12]

Child health professionals may need assistance from mental health professionals trained in trauma-informed care to correctly assess and diagnose mental health problems in children and young people in State care. The American Academy of Pediatrics recommends a mental health evaluation within 30 days of out-of-home placement, ideally by a child mental health professional trained in trauma-informed care. Periodic reassessment of mental health should occur whether or not a child is receiving mental health services because of the many uncertainties and transitions that can occur in their lives. Trauma-informed, evidence-based therapies include parent-child interaction therapy, child-parent psychotherapy, and trauma-focused cognitive behavioural therapy. A shortage of appropriately trained and experienced mental health professionals is a barrier to all children who might benefit from these interventions being able to access them.[12] Within New Zealand this highlights the importance of child health services and child and adolescent mental health services (CAMHS) working closely together and facilitating cross-referrals when indicated. Adequate CAMHS service provision is a prerequisite to such collaborative working.

Wairua

Atwool noted the following with respect to carers:

> Recognition of their vital role in supporting children in short-term and long-term placements and ensuring they are in a position to answer questions and contribute to a child's search for meaning is an essential prerequisite for improved practice. All caregivers (including those offering short-term care) need to be equipped to support children in keeping an ongoing record of their time in care through the use of memory boxes, life story books and digital records. Provision of back-up storage in case records are lost or destroyed is also needed.[16 (p. 10)]

Children and young people in State care expressed a clear need for help to manage relationships with family as well as opportunities to learn about and connect with their culture.[8] Young people reported a wide range in the help they needed to build or maintain cultural connections. One young woman (age 17) reported that her caregiver "didn't understand and she wouldn't let me go to kapa haka practices and she couldn't see why it was important to me."[7 (p. 44)] Such connection is essential for their wellbeing, is an integral part of clinical assessment and of providing child-centred care. Life story work is an inclusive term that emphasises the process of providing children with access to a coherent story of their life.[16] Life story work, adapted to suit a child's interests and needs, can contribute to bolstering self-esteem and a sense of identity. Children and young people find that a coherent life story helps them to learn about themselves, their families and their past, and to manage emotions, particularly emotions associated with negative experiences. There is currently a rather haphazard approach to such work in Aotearoa New Zealand which may contribute to poor outcomes.[16] Understanding their own 'health history' is an essential part of growing up securely. Inconsistent record keeping can lead to wrong decisions by professionals and adversely affect the child or young person.[4] Ensuring that a comprehensive health record is available to the child, young person and their caregivers, as part of the life story record is a positive contribution that health services can make to children and young people in State care. Health practitioners can ask to see records of the child's lived experience to date, and also ensure that important medical information is included and available to the child or young person as well as to any future caregivers.

Taiao

The underlying model on which New Zealand approaches care and protection is reflected in the direct questions asked of the child or young person and their caregivers about their home environment, neighbourhood and safety.[6] In the UK, Bilson (2016) suggests an alternative approach to the forensic child protection model that is common in the UK, USA and New Zealand. Such an alternative approach would aim to improve the conditions of families and communities, enabling them to enhance the wellbeing of children, rather than relying on agents of child protection to seek out harm. A community orientation would embrace approaches that focus on increasing neighbourhood cohesion to promote the wellbeing of children and families, evidence-based delivery of early childhood education that increases social capital, and also advocate for increased incomes for poor families that, in itself, has been shown to increase the wellbeing of children.[17] In New Zealand, many of the families of children who are referred to CYF live in areas with high deprivation scores.[7] Reducing social inequalities overall, and breaking the link between deprivation and extreme interventions by promoting good child development, could become a central goal of child protection policy and practice.[18,19]

Although it is rare, children may experience further abuse or neglect in foster homes.[12] Biehal et al.'s review found that it was difficult to come to clear conclusions about the extent of maltreatment in foster care.[20] Some

studies reported the total number of substantiated or unsubstantiated allegations of maltreatment per foster carer or foster family, whereas others reported the number of children experiencing maltreatment or incidents of maltreatment, with no indication of the proportion of children or foster carers involved in multiple incidents. There were also differences in the data sources used and reporting bias may result from children being unwilling or unable to report abuse. These factors need to be borne in mind when considering the following results on the extent of maltreatment in foster care. Five US studies of incidence found that between 0.27% and 2% of fostered children per year were known to have experienced maltreatment. Some of the variability in reported incidence may be due to local variations in thresholds for investigation or in recording policies. Separate studies from Baltimore, England and Scotland found that allegations of maltreatment were made in relation to 3–4% of foster homes per year, and substantiated in less than 1% of all foster homes. Biehal (2014) further notes that it is important to distinguish maltreatment in foster care from maltreatment by foster carers. Although foster carers are responsible in the majority of cases of maltreatment, perpetrators also include other children (including other fostered children, siblings of the child experiencing maltreatment, foster carers' own children or other unrelated children) and partners of foster 'mothers' (mothers may be unaware of the abuse). Fostered children may also be reabused during contact with their parents.

Iwi katoa: Health service implications

An integral part of the assessment process is to identify whether children, young people and whānau have had appropriate access to health services and systems.[6] The Expert Panel heard that health services

> "… do not prioritise work with vulnerable children ahead of their general accountability for universal services, despite the fact that vulnerable children are harder to reach and have more complex needs. If we are to have the same high level of aspiration for vulnerable children as we do for all other New Zealand children then we need to establish specific targets for children in the care and protection and youth justice system and hold social sector agencies to account for the achievement of these targets."[7 (p. 11)]

Health services are key actors in the planned legislative and operational changes to the New Zealand care and protection system as children and young people in State care will need access to appropriate services to address health concerns. Health services will need to ensure that health needs of children and young people in State care are actively prioritised, and respond appropriately to the full range of children's and young people's needs.[5,8] In addition to evidence-based best practice, children in State care have health service needs related to the context in which they live and grow.

Foundational principles and values

The National Institute for Health Care and Excellence (NICE) in the UK has articulated a group of principles that apply to all services for children in State care, including health services:[4,21]

- Put the voices of children, young people and their families at the heart of service design and delivery
- Deliver services that are tailored to the individual and diverse needs of children and young people by ensuring effective joint commissioning and integrated professional working
- Develop services that address health and wellbeing and promote high-quality care
- Encourage warm and caring relationships between child and carer that nurture attachment and create a sense of belonging so that the child or young person feels safe, valued and protected
- Help children and young people to develop a strong sense of personal identity and maintain the cultural and religious beliefs they choose
- Ensure young people are prepared for and supported in their transition to adulthood
- Support the child or young person to participate in the wider network of peer, school and community activities to help build resilience and a sense of belonging
- Ensure children and young people have a stable experience of education that encourages high aspiration and supports them in achieving their potential

Participation in health care decision-making by children and young people

The Children, Young Persons, and Their Families (Oranga Tamariki) Legislation Bill has a renewed emphasis on the participation rights of children and young people.[22] The participation rights of individuals in their health care is well established, including the importance of securing the participation of children in decisions that affect them.[23,24] Being child-centred is a way of elevating the interests, wellbeing and views of children. The overarching reason to be child-centred is to make sure decisions by health services and health professionals do not harm children and, in fact, support them to thrive. Services seeking to become more child-centred can seek the views of children and young people and ensure that their opinions and views are taken into account when

decisions are made.[25] The Children's Commissioner's guidance for agencies to become child centred describes the following core principles of child-centred thinking necessary in both top-down leadership and bottom-up practice: [25 (p. 3)]

- The best interests of the child should be an important consideration in all decisions
- Children should have the opportunity to have a say in decisions that affect them
- Decisions should ensure that children are not discriminated against
- Decisions should support, and not prevent, children to live, grow and achieve their full development
- Instituting child-centred thinking requires adoption at all levels of the organisation, including leadership commitment, training of staff and embedding in operations
- Any assessment of how children may be affected needs to be made at the beginning of a decision making process so issues can be addressed in the ultimate decision

Identifying children in need of care and protection

In practice, it is very difficult for CYF to restrict its role to a narrow statutory scope when health, education and welfare services are either insufficient or poorly aligned to the needs of vulnerable children and their families.[7] Within current legislation as well as the proposed amendments, health services have a major role in identifying child abuse and neglect and lodging appropriate reports of concern. The New Zealand Clinical Network for Child Protection (the Network) provides a source of expertise on issues of child abuse and neglect across the country and acts to assist in the co-ordination of services to abused and neglected children and young people.[26] New Zealand DHBs have a memorandum of understanding (MoU) with Child, Youth and Family that recognises the expertise of DHB staff in the recognition and management of child abuse and neglect to guide effective practice. This MoU can be accessed from the Network webpage.[26]

The National Institute for Health and Care Excellence (NICE) in the UK has developed a pathway that provides an integrated view of guidance, standards and indicators relevant to looked-after babies, children and young people.[4] There is also a specific pathway with information and resources about attachment difficulties in children and young people; although attachment difficulties are not specific to children in State care they do affect almost all children and young people in State care. It is essential that all professionals working with children in State care understand the complex issues affecting the lives of these children and young people, including discrimination and its impact, and recognise the importance of culture, identity and education in achieving a state of wellbeing.[4]

Information sharing and collaboration

It is generally accepted that effective child protection requires responses from health, education, justice, police and non-government organisations. For example, in New South Wales, the 'Keep them Safe' initiative involved expanding responsibility for responding to children at risk to increase the involvement of health, education, Police and other government agencies, as well as the non-government sector.[7] New Zealand is one of the few jurisdictions that does not have information-sharing settings in relation to vulnerable children in its care, protection and youth justice legislation.[3] The Expert Panel noted that New South Wales and Scotland have recently introduced major changes to information-sharing settings with some common features:

- Changing the threshold for information exchange towards promoting safety, welfare and wellbeing of children and young people and away from averting threats of serious harm
- Enabling greater information exchange between a much broader range of people involved in the lives of children and young people and their families than just those directly employed by the care and protection service
- Accompanying information-sharing duties with duties to collaborate with other professionals

The Children, Young Persons and Their Families (Oranga Tamariki) Legislation Bill includes provision for an information sharing framework that will include health services. This may be in response to the Expert Panel's observation that health services and other agencies are not prioritising services for vulnerable children ahead of a general accountability for delivery of universal services. Although there is clear government policy for every child in State care to receive a comprehensive health and education assessment, CYF data indicate that only five to six out of every 10 children in State care have a completed assessment, and there is little record of how many of these children have accessed required services to meet assessed needs.[7]

Healthcare staff knowledge skill and competence

In England, providers of health services are expected to identify a named doctor and nurse for looked-after children. These named professionals coordinate the provision of services for individual children and also provide advice and expertise for fellow professionals.[27] To fulfil their role and responsibilities in respect of looked-after children, all health staff need access to appropriate training, learning opportunities, and support to

facilitate their understanding of the clinical aspects of child welfare and information sharing.[4] Those in specific child health service roles also need the skills and competences to undertake health assessments, contribute to healthcare planning, ensure clinical governance arrangements to assure the quality of services for looked-after children, and coordinate care for each young person. The high prevalence of mental health conditions among children in State care, and the shortage of mental health professionals, calls for strong collaboration between paediatricians and mental health partners, including psychiatrists, psychologists, and developmental and behavioural paediatricians, among others. One promising model for the care of children in foster care is integrated paediatric and mental health care services in the paediatric medical home setting.[12]

The concept of a medical home was first mooted in the US in the 1960s among paediatricians who cared for children with chronic health conditions. At that time a medical home was a primary care practice for children that offered central coordination of specialists involved and a way to keep all information and data about the child in one place.[28] A medical home may contribute to mitigating the impact of adverse events on the wellbeing of children, and provide support which enhances family and child resilience.[29]

Primary prevention of child abuse and neglect

Bilson's[17] suggested alternative approach to the forensic child protection model common in the UK, USA and New Zealand aims to improve the conditions of families and communities, enabling them to enhance the wellbeing of children, rather than relying on agents of child protection to seek out harm. A community orientation would encompass approaches that focus on increasing neighbourhood cohesion to promote the wellbeing of children and families, evidence-based approaches to early years education that increase social capital, and advocacy to increase incomes of poor families that on its own has been shown to increase the wellbeing of children.[17] Reducing social inequalities overall and breaking the link between deprivation and extreme interventions by promoting good child development are policy goals that foster good practice for health as well as for education and other services.[18,19] Neighbourhood-based strategies for child maltreatment prevention can be effective in low- and high-resource communities leading to a reduction in child maltreatment for children aged under six years.[30] Although the authors observed that mechanisms related to successful interventions differed in different communities, they noted that the final outcomes always included improved child safety.

Parenting interventions and support of caregivers

Akin et al.[31] reviewed the implementation of evidence-based parenting interventions with substance-affected families involved with the child welfare system in five United States (US) counties using the following framework:

- Process factors including staff selection, training, coaching, and performance assessment
- Provider factors such as health service providers' attitudes toward evidence-based practice, which may in turn be affected by individuals' education level and professional status
- Innovation factors including logistical issues such as transport availability and the congruence between the planned intervention and the culture, values, and methods of the organisation implementing it
- Client factors which include the complexity of clients' presenting concerns as well as environmental barriers they may encounter
- Organisational factors ranging from the culture, climate, attitudes, and values of an agency to its policies and procedures
- Structural factors in the broader operating environment of organisations, including workforce issues, court systems, and interagency collaboration

Successful uptake of an evidence-based parenting intervention was associated with interactive and supportive staff training, well-balanced staff composition, programme flexibility that allowed for adaptation to local conditions and client circumstances, a focus on family skill development rather than clinical therapy and peer support among participants. Strong leadership at executive and management levels, particularly where there was a person dedicated to the coordination and implementation of the programme, was critically important for successful uptake. Community partners' donations of space, volunteers, food, and prizes helped successful programmes but varied between sites. Lack of adequate funding was a key barrier to effective implementation, and was associated with reliance on volunteer input and minimal capacity to make allowance for staff absences or staff turnover. Another barrier was difficulty in finding a time that worked well for families. Barriers experienced in rural communities included lack of transport, maintaining a minimum number of participants, and securing community partners.[31]

Many children in foster care have emotional and behavioural difficulties and some carers may respond poorly to these challenging behaviours.[20] Training in childhood trauma for caseworkers and foster parents has improved in recent years, and ongoing support for foster parents by well-educated professionals is important so that they have the opportunity to develop specific skills in parenting the traumatised child.[12]

High quality data

Accurate and up-to-date personal health information has significant implications for the immediate and future wellbeing of children and young people during their time in care and afterwards.[4] Deficiencies in available data have been noted in New Zealand (see **Monitoring State care in New Zealand** on **page 121**).[5] There is an urgent need to improve the range and quality of information on the needs of children, family/whānau and caregivers. Reliable information on the range of indicators is required to measure whether we are making a difference in the lives of children. Better and more accessible information is required on child and family risk factors, and evidence-based practice needs to be implemented by front-line health workforce and other agencies. This is likely to require some investment in the data infrastructure, information technology and workforce skills and training.[7]

Child protection processes in New Zealand

Since first introduced in 1925, child welfare legislation in New Zealand has demonstrated a strong preference for non-institutional foster care over institutional care.[9] The current legislative framework is the Children, Young Persons, and Their Families Act 1989.[32] Family preservation is a key principle underlying this Act, and the Act sets forth the family group conference (FGC) as a mechanism to provide for the participation of family, whānau, hapu, iwi, and family group in decision-making about the care of a child or young person.[9] Under this Act, the welfare and interests of the relevant child or young person shall be the first and paramount consideration[32[Section 6; Section 13]]. The guiding principle for determining the welfare and interests of a child or young person is that children and young people must be protected from harm and have their rights upheld.[32[Section 13]] Under the current Act, a child is a boy or girl under the age of 14 years. A young person is defined as a boy or girl of or over the age of 14 years but under 17 years, but does not include any person who is or has been married or in a civil union.[32[Section 2]] From 1 April 2017 the upper age limit of the statutory definition of young person will be increased to 18 years.[33] In the 2015/2016 year, ahead of this amendment to the Children, Young Persons, and Their Families Act, 1989, the Ministry of Social Development began providing increased advice and assistance to support young people aged 15–20 years who have been in statutory care.[33,34]

Child, Youth and Family (CYF) is a service arm of the Ministry of Social Development with legal powers to intervene to protect and help children who are in need of care and protection.[5] The full definition of a child or young person in need of care and protection, as contained in the Children, Young Persons, and Their Families Act, 1989, is provided in **Box 3**.

Box 3. Definition of a child or young person in need of care and protection, Children, Young Persons, and Their Families Act, 1989 Section 14.

(a) the child or young person is being, or is likely to be, harmed (whether physically or emotionally or sexually), ill-treated, abused, or seriously deprived; or

(b) the child's or young person's development or physical or mental or emotional wellbeing is being, or is likely to be, impaired or neglected, and that impairment or neglect is, or is likely to be, serious and avoidable; or

(ba) the child is a subsequent child of a parent to whom section 18A applies, and the parent has not demonstrated to the satisfaction of a social worker (under section 18A) or the court (under section 18C) that he or she meets the requirements of section 18A(3); or

(c) serious differences exist between the child or young person and the parents or guardians or other persons having the care of the child or young person to such an extent that the physical or mental or emotional wellbeing of the child or young person is being seriously impaired; or

(d) the child or young person has behaved, or is behaving, in a manner that—
  (i) is, or is likely to be, harmful to the physical or mental or emotional wellbeing of the child or young person or to others; and
  (ii) the child's or young person's parents or guardians, or the persons having the care of the child or young person, are unable or unwilling to control; or

(e) in the case of a child of or over the age of 10 years and under 14 years, the child has committed an offence or offences the number, nature, or magnitude of which is such as to give serious concern for the wellbeing of the child; or

(f) the parents or guardians or other persons having the care of the child or young person are unwilling or unable to care for the child or young person; or

(g) the parents or guardians or other persons having the care of the child or young person have abandoned the child or young person; or

(h) serious differences exist between a parent, guardian, or other person having the care of the child or young person and any other parent, guardian, or other person having the care of the child or young person to such an extent that the physical or mental or emotional wellbeing of the child or young person is being seriously impaired; or

(i) the ability of the child or young person to form a significant psychological attachment to the person or persons having the care of the child or young person is being, or is likely to be, seriously impaired because of the number of occasions on which the child or young person has been in the care or charge of a person ... for the purposes of maintaining the child or young person apart from the child's or young person's parents or guardians.

Families become known to CYF when someone who is worried about the safety and wellbeing of a child and their family makes a report of concern to CYF. Reports of concern come from a variety of sources including the Police, health, education and social service providers, family members and friends, and concerned members of

the public.[5] Notifications about a child are first received by a National Contact Centre social worker who decides whether the concerns meet the threshold for CYF action. If so, the notification is recorded as a report of concern. If not, the notification remains a contact record only.[7] The following process for managing reports of concern is described by the Office of the Children's Commissioner[5 (p. 8)]

> When CYF receives a report of concern, they undertake an initial safety and risk screen about the child and family's situation, and decide whether any further action is required to make sure the child is safe. In many cases, no statutory intervention is required; the family may simply need some advice, or to be connected with the right support services.

> In more serious cases, CYF care and protection teams work with the family to identify issues and find a solution, which could include a formal investigation with Police. When it is established that a child is in need of care and protection, a family group conference may be held where the child's family/whānau and other key people agree on a plan to keep the child safe and identify the support they need.

A child or young person comes into statutory CYF care through a formal declaration by the Family Court that the child or young person is in need of care and protection, which can occur only after a family group conference (FGC) has been held. An interim Custody Order can be put in place without delay if a child or young person is in immediate danger.[35] The child or young person is then in the custody of the Chief Executive of CYF, and a CYF social worker files a report and plan for their care and protection with the Family Court.[35] The law allows that the child or young person may be placed with:[32]

- a parent or guardian of the child or young person; or
- any other person who previously had the care of the child or young person; or
- any member of the child's or young person's family, whānau, or family group; or
- any person approved by a social worker.

If it is not practicable or appropriate to place the child or young person with any of these specified persons the child or young person may be placed in a residence.[32]

Children and young people in Child, Youth and Family care

Child, Youth and Family (CYF) data show that as at 30 June 2016 there were 5,312 children and young people in New Zealand who were in in CYF care. Of these 5,312 children and young people, 578 (11%) were aged under two years; 869 (18%) were 2–4 year olds; 1,538 (29%) were 5–9 year olds; 1,193 (22%) were 10–13 year olds and 1,067 (20%) were aged 14 or older.[36] The approach to ethnicity classification within CYF is that children and young people (or their families) are asked to self-identify their ethnicity. For the purposes of reporting, only the client's self-identified primary ethnic group is extracted. If a client does not nominate a primary ethnic group, their ethnicity is reported as "Other / Multiple Ethnicities".[36] As at 30 June 2016 3,208 (60%) of children and young people in CYF care had Māori recorded as their primary ethnic group; New Zealand Pākeha was recorded as the primary ethnic group for 1,478 (28%), Pacific Peoples for 407 (8%), Asian for 79 (1%), Other European for 55 (1%) and other or multiple ethnicities for 85 (2%).[1] Most (83%) of the children and young people in CYF care were in out-of-home placements, with the remainder remaining in their own homes, returned to their homes after a period of out-of-home care or in independent living situations.[36] **Table 26** presents the numbers of New Zealand children and young people in out-of-home State care by placement type, as well as the total numbers in CYF care, in each of the past five years. Numbers of children in care have been fairly stable at around 5000 as of 30 June each year and their distribution between placement types has also been similar from year to year.

Table 26. Individual children and young people in out-of-home State care, by placement type, and total number of children and young people in CYF care New Zealand 2012–2016

Placement Type	June 2012	June 2013	June 2014	June 2015	June 2016
Non Family / Whānau Placement	1,427	1,298	1,269	1,182	1,281
Family / Whānau Placement	1,639	1,698	1,999	2,193	2,303
Child and Family Support Services*	518	521	536	502	507
CYF Family Home Placement	114	103	114	133	154
Residential Placement	47	47	34	29	35
Other Supported Accommodation	139	177	177	124	114
Total children and young people in out-of-home placements	**3,884**	**3,844**	**4,129**	**4,163**	**4,394**
Total children and young people in CYF care	**4,979**	**4,960**	**5,188**	**5,026**	**5,312**

Source: Child, Youth and Family.[36] The number of children and young people in out of home placements is as at 30 June each year. Out of home placements exclude placement types: 'Independent Living', 'Remain Home' and 'Return Home'; *The placement type 'Child and Family Support Services' is approved under s396 CYPF Act, and provided by non-government organisations (NGOs); In CYF care = children and young people in the custody of the Chief Executive of Child, Youth and Family as at 30 June each year; Each child or young person in CYF care at the point in time shown is counted once in this table

Children and young people in CYF care represent only around 10% of all children and young people who come to the attention of CYF.[7] A review of data for every child and young person who was a CYF client showed that for every ten 0–17 year olds who came to CYF attention in 2013:[7]

- Two were assessed at intake as not requiring a statutory intervention; the case was closed or, in a minority of cases, children were referred to a non-government organisation for a partnered response
- Five were subject to a formal assessment or investigation but no further statutory action was taken; the case was closed or, in a minority of cases, children were referred to a non-government organisation for a partnered response
- An additional two were subject to a formal assessment or investigation and as a result received support from CYF in the form of family group conferences (FGC) or family whānau agreements (FWA)
- One was brought into CYF care as a result of care and protection concerns, after a formal assessment or investigation, most often when those concerns were not resolved through a FGC process.

The *Interim Report* notes that about half of the notified children and young people who were assessed as not requiring a statutory intervention were re-referred to CYF within a year because of community or professional concern for their safety and care. A change in patterns of repeat referrals has been noticed over time. In 2004 most notifications to CYF concerned children who were not previously known to the agency. In contrast, 6 out of every 10 children referred to CYF in 2014 were already known to the agency and, on average, CYF had engaged with these children three times previously.[7]

Atwool (2013) notes that children in all children and young people in foster or kin care are members of two or more families. They must manage divided loyalties: to the people with whom they live who are their current source of care and protection, and also to their birth families toward whom they may have strong but ambivalent feelings. Research has generally shown that children want to continue contact with birth families, and some never give up hope that they can return to live with them. Contact with birth families puts the child or young person into a situation where the two worlds of birth and foster family overlap. The impact of such contact will vary depending on the pattern of belonging to each family as experienced by the child or young person.[2]

Monitoring State care in New Zealand

The statutory functions of the New Zealand Children's Commissioner include monitoring and assessing the policies and practices of CYF.[5,8,37] In 2015 the Children's Commissioner found that CYF had strong intake and assessment practices with well-developed processes for investigating and making decisions about cases of potential abuse and neglect. Once such decisions had been made, however, oversight and case management of children in all types of CYF placement was poor. There was little available data about the outcomes for children and young people in CYF care, and not enough information to draw any conclusion about whether or not children and young people are better off as a result of state intervention.[5] The aggregated and summarised recommendations from the 2015 *State of Care* report were:[5]

- Set clear expectations about CYF's core purpose and the outcomes it needs to achieve
- Ensure CYF is fully child-centred in all its activities
- Invest more in on-going support for children in all types of care placements

- Address capacity and capability issues across the CYF workforce
- Improve cultural capability across the organisation
- Collect and analyse relevant data to drive improved outcomes for children
- Set clear expectations for other state agencies responsible for improving the outcomes of children in care

The 2016 *State of Care* report focussed on the quality of CYF case management and found some examples of positive practice.[8] Children and young people were considered to be safe in all of the six CYF residences visited by the Office of the Children's Commissioner; most were receiving care and services that met daily needs. However, the review also found that CYF was not sufficiently child centred and was of variable quality. Barriers to achieving high quality case management included inadequate resources, high caseloads, and a lack of cultural capacity and other skills to work in child-centred ways. While welcoming the planned changes resulting from the Modernising Child Youth & Family Expert Panel reports, the Children's Commissioner also expressed concern at potential risks associated with transition between operating models. There is increased risk of a dip in performance at times of operational change. The overarching recommendations from the 2016 *State of Care* report were:[8]

- Plan to reduce the risk to children and young people of a dip in performance during the transition period
- Clarify what child-centred practice means in the New Zealand care and protection and youth justice systems
- Empower and support staff now to strengthen their child-centred practice

Child Youth and Family to Oranga Tamariki

The Children, Young Persons and Their Families (Oranga Tamariki) Legislation Act received Royal assent in July 2017.[38] The Act changed the name of the principal Act (previously Children, Young Persons and Their Families Act 1989) to Oranga Tamariki Act 1989 or Children's and Young People's Well-being Act 1989. The Act established a statutory framework required to create a more child-centred operating model for the Ministry for Children, Oranga Tamariki to meet the needs of children and young people in Aotearoa New Zealand. The changes included:

- Updating the definition of young person to include young persons who are (or have been) married or in civil unions
- Updating the general principles of the principal Act to take a more child-centred approach placing all children and young persons (including children with disabilities) at the centre of decision-making, considering them within the context of their families, whānau, hapū, iwi, and broader networks and communities, with specific recognition and respect for a child's or young person's mana tamaiti (tamariki) and the whakapapa and whanaungatanga responsibilities of whānau, hapū, and iwi
- Introducing a new and separate principle of child and young person participation that clearly recognises the importance of the voice of the child, elevates its status, and more firmly embeds this aspect of child-centred practice
- Development of National Care Standards and provision of financial support for caregivers that is more responsive to the changing needs of children in care. These will set out the rights of children and young persons in care, the standard of care that they can expect, and standards for caregiver training, monitoring, and support
- Amendments to extend the youth justice jurisdiction to include 17-year-olds
- A new entitlement for young persons transitioning out of care to remain or return to living with a caregiver up to age 21 and for CYF, where required, to provide transition advice and assistance to young persons leaving care or a youth justice facility up to age 25
- A bespoke information sharing framework within the Act
- Accountability arrangements to ensure the co-ordination of prevention activity across government and to address the needs of children and young persons in need of care or protection.

The *Investing in New Zealand's children and their families* report notes that the scale of change required will take many years to rollout and embed. The essential issue is that the change occurs in a way that ensures better outcomes for vulnerable children.[3] The Expert Panel observed a growing appetite for meaningful and lasting change to achieve better outcomes for the children and young people at the heart of the care and protection system. The aim of the proposed changes is to achieve better outcomes for vulnerable children. The aspirational goals for vulnerable children are that they will enjoy positive childhoods and have the opportunity to fully realise their potential.[3]

> When children are unable to live with their birth parents at home, they require intensive
> support to develop a sense of stability and to build new relationships with a family who will

be there for them now and in the future. The State has a role in supporting families to build loving and stable relationships with children in their care and to help them ensure that those children enjoy the same opportunities and outcomes as other New Zealand children.[3]

The operating model envisages a wider range of professional domains (such as health, education, and psychology) working with children and families, both within the department and across agencies. This change will require transformational leadership at all levels that engages people, communities and all of New Zealand to build the momentum to deliver the scale of change required. Services, including health services, would work with young people who have been in care to proactively identify and meet their needs up to the age of 25.[3]

A key component of the operating model proposed in the *Investing in New Zealand's children and their families* report is that the future department (Oranga Tamariki) will commission and directly purchase services for vulnerable children, including therapeutic interventions and health related services that support healing and recovery for children, young people and families. All practitioners will need a robust understanding of child development and trauma-informed approaches.[3] The Expert Panel recommended legislative mechanisms to support more effective interagency and multidisciplinary working at an individual client level, including provisions around information exchange, and support governance and collaboration at a system level. This might include introducing a duty on agencies such as District Health Boards to collaborate and coordinate services to children and families, extending the current obligation under the Vulnerable Children Act. Strong leadership will be required to establish, manage and govern a change programme of this size.[3]

Conclusion

Children and young people in State care are a New Zealand population group with high needs across all health domains, including secure whānau and family attachment as well as timely assessment and effective management of physical, spiritual, and mental health needs. Health services have a major role in identifying children and young people in need of care and protection, and in working collaboratively with other agencies to address the health needs of children and young people in State care. The proposed direct purchasing of services for vulnerable children will mean that DHBs and other health services need to be well prepared to deliver services that support healing and recovery for children, young people and families. Key infrastructure elements to achieve positive outcomes will include gathering and use of good data, development of staff knowledge, skills and understanding, and effective community programmes to improve child safety.

Within health services it is important to identify children and young people in State care, so that particular attention can be paid to ensuring that vulnerable children, young people and their caregivers have a sound understanding of existing health concerns and the management of them. This may include specific support for caregivers particularly where the children and young people in care have challenging behaviour. Keeping accurate health records are important not only at this individual level but also at a service and aggregate national level, to add to the sparse evidence base about effective interventions that enable children and young people in State care to enjoy the same health outcomes as their peers.

Health professionals involved with children and young people in State care, and with their whānau, wider families and caregivers, need appropriate skills and competencies to assure high quality services. Guidelines for staff skill development are available from other jurisdictions and could be adapted for New Zealand context.[19,23] There is a high level of co-occurrence of physical and mental health conditions as well as emotional and sleep disorders for many children and young people in State care. Such complex health histories mean that there needs to be excellent co-ordination between paediatric and youth health services and mental health services within and between DHBs.

Making our communities and households safe environments for children and young people to develop and grow will require strong collaborative efforts from many sectors of society. Health promotion and community development initiatives to strengthen parenting skills, provide early additional support when required, reduce social inequalities overall and promote good child development have a valid place within health services' mandate. Health professionals are also in a privileged position meeting with children and young people in State care and can include assessment of placement safety in clinical encounters.

Proposed changes in New Zealand signal a commitment to and effective child-centred approach that will enable all children, including the most vulnerable, to enjoy positive childhoods and have the opportunity to fully realise their potential.

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CEREBRAL PALSY

Introduction

Cerebral Palsy (CP) is the name given to a group of disorders characterised by impairments in the development of movement and posture due to non-progressive damage or malformation of the fetal or infant brain.[1] Although the brain damage is non-progressive, its manifestations may change over time as the brain develops.[1] Cerebral palsy is the most common physical disability in children, affecting around two in every 1,000.[2-4]

In addition to motor disorders, children with CP often have other comorbidities including intellectual disability (30–65% of cases), epilepsy (30–50%), speech and language deficits (40%), visual impairments (40%), hearing problems (5–15%), psychosocial and behavioural problems (20%) and autism spectrum disorder (9%).[5] Medical complications of CP can involve multiple bodily systems including the genitourinary (incontinence, urinary infections, and voiding dysfunction), gastrointestinal (dysphagia, gastroesophageal reflux disease, constipation), respiratory (recurrent pneumonia, atelectasis, bronchiectasis, restrictive lung disease), and endocrine (reduced growth and osteopenia).[5]

In clinical practice, diagnosis of CP is based on observation and parental reports of delayed attainment of motor milestones (such as achieving head control, sitting and standing), and evaluation of posture, deep tendon reflexes, and muscle tone.[6] There is wide variation in the degree of impairment associated with CP. The least severely affected children have only minor limitations in speed, balance and coordination, while the most severely affected are unable to maintain anti-gravity head and trunk postures, or control leg and arm movements, and need wheelchairs and assistance with all aspects of daily life.[7,8]

Data sources and methods

Indicators

    Rates of cerebral palsy among 0–24 year olds

Definition

Hospitalisations of 0–24 year olds with cerebral palsy per 100,000 population

Data sources

Numerator: National Minimum Dataset

Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

This section presents analyses where the condition was the primary diagnosis or was documented within any of the first 15 diagnoses (all cases). The rationale for presenting all cases is to highlight the full spectrum of health issues experienced by those with this condition, and their consequent requirement for acute health services. Analyses are per hospital discharge event therefore events are only included if the condition is documented within either the primary diagnosis or within any of the first 15 diagnoses.

Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

There were 58 deaths of 0–24 year olds with cerebral palsy as the underlying cause of death in New Zealand during 2009 to 2013, as documented within the National Mortality Collection.

The number of 0–24 year olds hospitalised between 2011 and 2015 with any diagnosis of cerebral palsy is presented in **Table 27**, together with the total number of hospitalisations with cerebral palsy as a primary or any diagnosis**.**

Since 2000, hospitalisation rates for cerebral palsy as the primary diagnosis have risen steadily for 0–14 year olds, peaking in 2012 for 0–4 year olds and 2014 for 5–14 year olds. Hospitalisation rates were lowest for 15–24 year olds. Overall, there is a steady increase in hospitalisations of children hospitalised with cerebral palsy as the primary diagnosis (**Figure 42**). Similar patterns over time were seen in all ethnic groups (**Figure 43**).

The majority of hospitalisations of 0–24 year olds with a primary diagnosis of cerebral palsy had spastic cerebral palsy as the primary diagnosis. Other associated primary diagnoses included in respiratory, digestive or musculoskeletal conditions (**Table 28**).

Table 27. Individuals hospitalised with cerebral palsy, 0–24 year olds, New Zealand 2011–2015

| Age group | Unique individuals (n) | Hospitalisations (n) | | Ratio All:Primary |
		Primary diagnosis	All cases	
Cerebral palsy				
Hospitalisation				
0–24 years	1,678	2,458	5,852	2.38
0–14 years	1,198	2,221	4,469	2.01
15–24 years	581	237	1,383	5.84

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with cerebral palsy in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Figure 42. Hospitalisations for cerebral palsy in 0–24 year olds, by age group, New Zealand 2000–2015
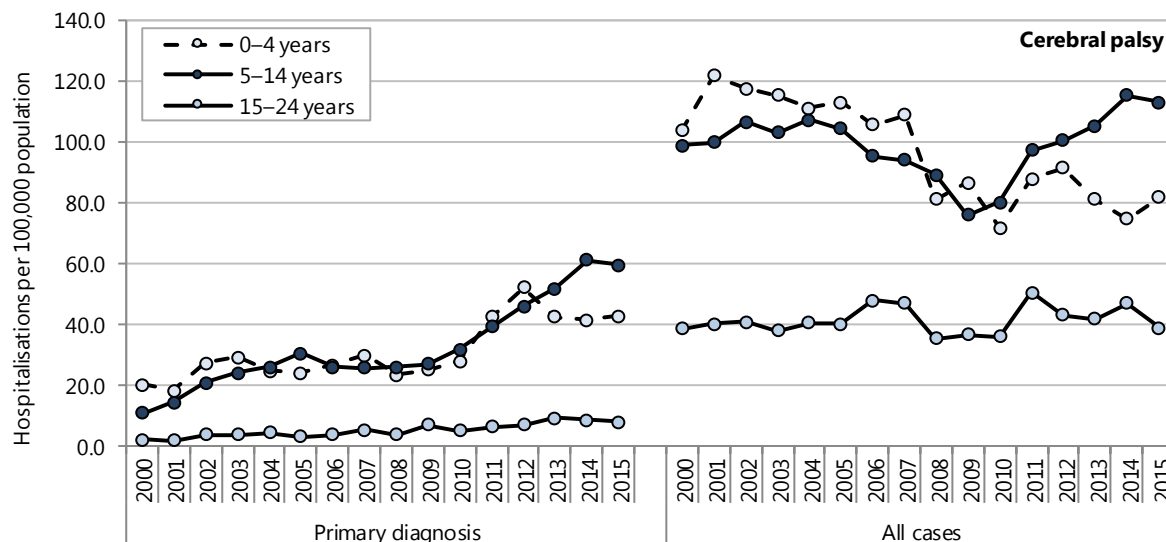
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population

Figure 43. Hospitalisations involving cerebral palsy in 0–24 year olds, by ethnicity, New Zealand 2000–2015
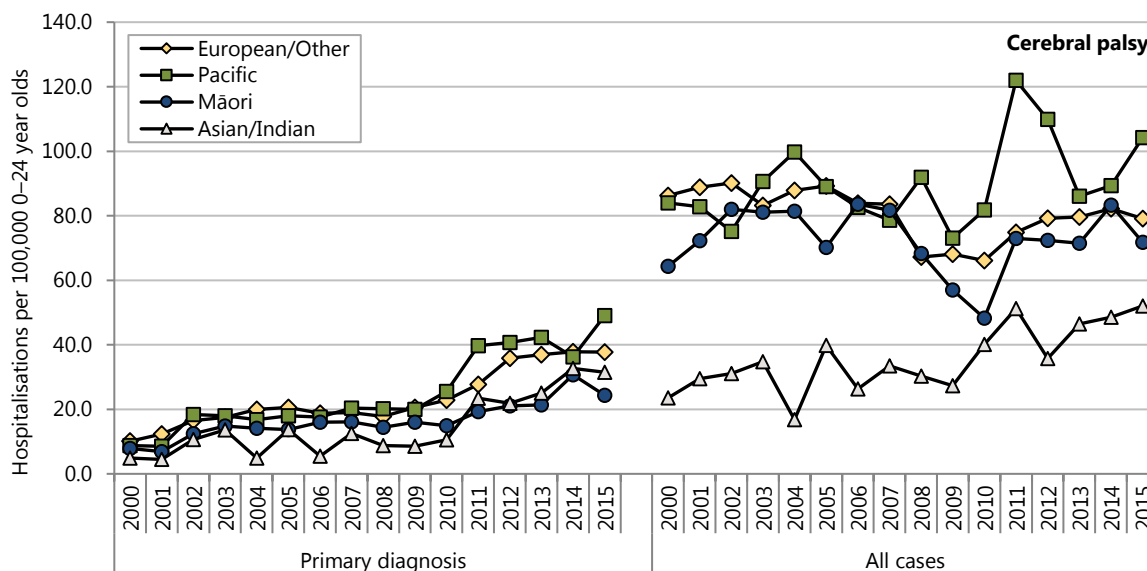
Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population

Table 28. Hospitalisations involving cerebral palsy in 0–24 year olds, by primary diagnosis, New Zealand 2011– 2015

Primary diagnosis	2011–2015 (n)	Annual average	Rate per 100,000 0–24 year olds	95% CI	%
Cerebral palsy* in 0–24 year olds					
New Zealand					
Spastic cerebral palsy	1,958	392	25.49	24.39–26.65	33.5
Dyskinetic cerebral palsy	85	17	1.11	0.90–1.37	1.5
Ataxic cerebral palsy	5	1	0.07	0.03–0.15	0.1
Cerebral palsy, other or unspecified	410	82	5.34	4.85–5.88	7.0
Cerebral palsy total	2,458	492	32.00	30.76–33.29	42.0
Other diseases of the nervous system	375	75	4.88	4.41–5.40	6.4
Diseases of the respiratory system	662	132	8.62	7.99–9.30	11.3
Diseases of the digestive system	547	109	7.12	6.55–7.74	9.3
Diseases of the musculoskeletal system and connective tissue	482	96	6.28	5.74–6.86	8.2
Symptoms and/or abnormal clinical findings NEC	347	69	4.52	4.07–5.02	5.9
Other diagnoses	981	196	12.77	12.00–13.60	16.8
Total	5,852	1,170	76.19	74.26–78.17	100.0

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Cerebral palsy in any of the first 15 diagnoses; NEC = not elsewhere classified

Demographic distribution

Table 29 presents the demographic distribution of individuals with cerebral palsy in New Zealand between 2011 and 2015. Cerebral palsy rates were significantly higher in higher deprivation areas (NZDep2013 deciles7–10) compared to low deprivation areas (NZDep2013 deciles 1–2), and significantly higher among 0–4 year olds and 5–14 year olds compared to 15–24 year olds. Hospitalisation rates were significantly lower for Asian/Indian 0–24 year olds than other ethnic groups. The majority of 0–24 year olds with cerebral palsy were of European/Other ethnicity.

Table 29. 0–24 year olds hospitalised with cerebral palsy, by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Cerebral palsy* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	305	21.49	1.00	
Deciles 3–4	328	24.53	1.14	0.98–1.33
Deciles 5–6	317	21.99	1.02	0.87–1.20
Deciles 7–8	458	28.19	1.31	1.13–1.52
Deciles 9–10	574	30.89	1.44	1.25–1.65
Prioritised ethnicity				
Māori	405	22.45	0.99	0.88–1.11
Pacific	191	26.95	1.18	1.01–1.38
Asian/Indian	126	13.14	0.58	0.48–0.69
MELAA	38	37.68	1.65	1.20–2.29
European/Other	936	22.78	1.00	
Gender				
Female	723	19.26	1.00	
Male	955	24.32	1.26	1.15–1.39
Age group (years)				
0–4	439	28.15	1.52	1.34–1.72
5–14	943	31.60	1.71	1.54–1.89
15–24	581	18.52	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Cerebral palsy in any of the first 15 diagnoses; Rate ratios are unadjusted; Ethnicity is level 1 prioritised; Decile is NZDep2013

Regional distribution

Table 30 presents the number of individuals resident in each district health board area that had a diagnosis of cerebral palsy between 2011 and 2015. It also presents the number of hospital discharges in which cerebral palsy was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and result in less undercounting than would occur if only the primary diagnosis is included in discharge data. For cerebral palsy the All:Primary diagnosis ratio was higher than the national ratio in Northland, Waikato, Bay of Plenty, Lakes, Tairawhiti, Whanganui, Hawkes Bay and Southern DHBs and lower in Waitemata, Auckland, Counties Manukau, Taranaki and Wairarapa DHBs. Ratios were similar to the national ratio in the remaining district health boards (**Table 30**).

Table 30. Hospitalisations for cerebral palsy in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Cerebral palsy* in 0–24 year olds				
Northland	55	28	132	4.71
Waitemata	226	611	985	1.61
Auckland	165	431	668	1.55
Counties Manukau	267	402	760	1.89
Waikato	168	158	577	3.65
Bay of Plenty	99	86	349	4.06
Lakes	56	35	167	4.77
Tairawhiti	16	16	76	4.75
Taranaki	32	47	96	2.04
Hawke's Bay	61	34	194	5.71
MidCentral	51	52	140	2.69
Whanganui	23	14	78	5.57
Hutt Valley	52	46	113	2.46
Capital & Coast	86	100	222	2.22
Wairarapa	11	14	25	1.79
Nelson Marlborough	51	44	162	3.68
South Canterbury	11	10	29	2.90
Canterbury	214	253	705	2.79
West Coast	12	13	32	2.46
Southern	103	62	340	5.48
New Zealand	1,678	2,458	5,852	2.38

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Cerebral palsy is listed in any of the first 15 diagnoses; Rate ratios are unadjusted; Note that individuals may appear in multiple DHBs

Evidence for good practice

Possibilities for prevention

The causes of the brain damage that produces cerebral palsy are not well understood therefore the possibilities for prevention are limited.[9,10] While it was formerly believed that lack of oxygen during birth was the cause of CP, it is now thought that, in the majority of cases (at least 80%), the brain injury or malformation occurs before birth and that no more than 10% of cases are the result of perinatal factors.[4] A small proportion of cases (5–10%) are due to postnatal brain injury in the early months and years of life, for example from cerebrovascular accident (stroke); head trauma due to motor vehicle crashes, falls or child abuse; near drowning and other forms of asphyxia; and infection such as meningitis.[4,11]

Few studies investigate more than one risk factor yet it seems probable that, in many cases, CP results from the interaction of multiple risk factors.[12]

Prenatal

There is a genetic component to cerebral palsy risk as the recurrence rate in families of people with CP is greater the closer the degree of genetic relationship.[13] Nevertheless, the absolute risks for family members are low because CP is not a very common condition. Current guidelines do not recommend routine genetic testing.[14]

In developed countries, increasing maternal age and the use of assisted reproduction therapies have both increased the rate of multiple births, which have a higher risk of CP than singleton births.[15]

Among the few preventable prenatal causes of CP are severe maternal iodine deficiency,[16] rhesus isoimmunisation,[17] consanguineous marriages,[9] and maternal methyl mercury poisoning.[18] These factors cause almost no cases of CP in developed countries.[19]

Babies who are born small for their gestational age or well above the normal weight for their gestational age are at increased risk of CP but it is uncertain whether the abnormal growth patterns are a cause or a consequence of CP.[20]

Perinatal

High quality maternity care can prevent or mitigate complications of labour and delivery that increase the risks of adverse health outcomes for babies, including brain damage leading to CP.[21]

Preterm birth is an important risk factor for CP and the risk increases markedly with decreasing gestational age, nevertheless more than half of all children with CP were born at term.[4] Prevention of preterm birth is challenging as its causes are multifactorial and poorly understood.[22]

In women threatening to or likely to give birth preterm, there is evidence that antenatal magnesium sulphate therapy substantially reduces the risk of their child having CP,[23] although mothers can experience unpleasant side effects such as tachycardia, flushing and nausea/vomiting.[24]

A recent review of risk factors for CP in children born at term in developed countries identified 10 statistically significant risk factors, three of which were considered possibly preventable.[25] These three were: birth asphyxia, low birthweight and meconium aspiration.[25]

In infants born at 35+ weeks gestation with evidence of peripartum asphyxia (such as needing mechanical ventilation or resuscitation at 10 minutes after birth) and encephalopathy (such as seizures), induced hypothermia (cooling) reduces mortality and neurodevelopmental disability in survivors.[26]

Fetal heart rate monitoring using continuous cardiotocography (CTG) has not been shown in RCTS to reduce cerebral palsy rates but it does increase rates of caesarean sections and instrumental vaginal births.[27]

The passage of fetal bowel movement (meconium) in the amniotic fluid is common, and it can (rarely) result in meconium aspiration syndrome if the baby inhales meconium during the birth process.[28] According to a 2012 Cochrane review, curtailment of post-term pregnancies by induction of labour reduces the occurrence of meconium stained amniotic fluid and meconium aspiration syndrome.[29] There is little research evidence regarding the benefits or otherwise of obstetric interventions such as expedited delivery when there is meconium-stained amniotic fluid without other evidence of fetal distress.[28]

Postnatal

Because infants born preterm have a greatly increased risk of developing CP, early intervention programmes have been used in the hope of preventing cerebral palsy and promoting normal brain development in preterm infants. The evidence indicates that general development programmes for preterm infants have no effect on CP rates in survivors of preterm birth.[30] There is some evidence that such programmes improve cognitive outcomes in the preschool years although not at school age. There is little evidence for positive effects on motor outcomes beyond infancy.[30,31] It has been argued that early intervention research to date does not provide sufficient evidence to exclude the possibility that early intervention could have long lasting benefits for infants at high risk of developing CP or with early signs of CP.[32]

Interventions to prevent head injuries in infants and toddlers, such as promoting car seat use and preventing shaken baby syndrome,[33,34] may help prevent post-natally acquired CP.

Evidence-based health care for children and young people with cerebral palsy

Children with CP often have multiple medical issues and they typically need services from multiple healthcare professionals including paediatricians, orthopaedic surgeons, neurologists, ophthalmologists, optometrists, audiologists, physiotherapists, dieticians, occupational therapists, speech language therapists and dentists.[35] They also may need special education services, in-home care and respite care.[35]

The evidence base for CP therapies is limited largely to systematic reviews, meta-analyses, and large multicentre prospective cohort studies because of the lack of well conducted prospective randomised controlled trials on this topic.[11,36] The interventions that are best supported by evidence are: anticonvulsants, bimanual training, botulinum toxin, bisphosphonates to prevent osteoporosis, casting, constraint-induced movement therapy, context-focused therapy, diazepam, fitness training, goal-directed training, hip surveillance, home programmes, occupational therapy after botulinum toxin, pressure care, and selective dorsal rhizotomy.[36]

Coordination of care

In English-speaking developed countries, and increasingly in other developed countries, family-centred service provision for children with special needs is considered to be best practice.[37,38] This model of service provision recognises that each family is unique, that the family is the constant in the child's life, and that they are the experts on the child's abilities and needs. It involves the family working together with service providers to make informed decisions about the services and supports the child will receive and it considers the strengths and needs of all family members.[39,40]

There has been very little research on family-centred care specifically for families affected by CP but a review of 24 studies (including seven RCTs) of family-centred care for US children with conditions associated with having special health care needs found positive associations between family-centred care and improvements in efficient use of services, health status, satisfaction, access to care, communication, systems of care, family functioning, and family financial impact and cost.[41] It did not identify any negative outcomes.

While multidisciplinary teams are an integral part of child health services, they are often lacking in adult healthcare services and young adults with CP (or their families) can be left to coordinate their own healthcare at a time when they are dealing with many transitions in other areas of life including education and employment, finances and benefits, housing, transportation, leisure activities and relationships.[42-44]

Caring for a child with cerebral palsy is often challenging for families and primary caregivers of children with CP have been found to have lower incomes than other parents despite similar levels of education, to be less likely to be working for pay and to have a greater likelihood of physical and psychological health problems.[45,46] In contrast to parents of normal children, parents of children with CP can find that the demands of caregiving become greater as their child ages.[47] Research has not consistently found that the severity of a child's motor impairment determines the impact of CP on families' wellbeing but it has consistently found that CP that is accompanied by intellectual disability and behaviour problems is associated with a more severe impact.[48]

There is research indicating that respite care is an appropriate and effective intervention for decreasing parental stress and giving parents the chance to spend time with other family members.[49] Respite care can also be considered a child abuse prevention intervention, particularly for children with challenging behaviours.[49] There seems to have been little research on effective models for respite care for children with developmental disability and severe behaviour problems.[49]

Physiotherapy

Physiotherapy is an established component of the management of CP. There is an increasing evidence base to support a number of physiotherapy interventions including constraint-induced movement therapy (where a child's less-affected hand is restrained in a mitt or cast and the more-affected hand is given intensive training)[50,51], strengthening interventions for individual muscle groups[52] and functional training (training focussed on motor activities similar to those involved in daily living such as stair climbing, walking and moving from sitting to standing) but research studies have generally been small (< 30 participants).[52-54]

Exercise interventions may improve postural control in children with CP.[55] There is a moderate level of evidence to support gross motor task training, therapeutic horseback riding, treadmill training with no body weight support, trunk-targeted training, and reactive balance training.[55]

Orthoses, especially ankle-foot orthoses which aim to facilitate standing upright and walking, are commonly prescribed for children with CP although the evidence for their efficacy is limited to low level evidence that they improve gait in the short term.[56]

To achieve their functional potential children with CP need to be motivated to persist with their rehabilitation therapy. There has been very little research on the effects of motivational interventions for children with CP.[57]

Treatments for spasticity

Spasticity (tight muscles as a result of damage to the parts of the brain that control movement) can be a significant source of functional disability in many children with CP. Spasticity inhibits movement and causes

pain both directly through producing cramp and indirectly through producing extreme joint positions. Interventions to manage spasticity include physiotherapy, casting and splinting, orthopaedic and neurosurgery, botulinum toxin injections and oral medications.

Oral medications to treat spasticity include benzodiazepines, dantrolen, baclofen and tizanidine. The evidence base for the use of oral medications for spasticity in CP is limited because most studies were conducted many years ago and, by modern standards, had methodological limitations.[58] The choice of medication is therefore largely based on personal experience or trial and error.

There is a growing body of evidence for the effectiveness of botulinum toxin A (Botox) in reducing spasticity and improving motor function in children with CP when it is used in combination with other treatment measures.[59-61]

There is a small amount of evidence that intrathecal baclofen (baclofen infused by a pump connected to a catheter directly into the subarachnoid space around the spinal cord) is an effective treatment for treatment of spasticity in children with CP in the short term.[62]

Selective dorsal rhizotomy (SDR) is an irreversible neurosurgical procedure involving cutting selected sensory nerve roots in the lumbar spine under intraoperative neurophysiological guidance. Intensive post-operative physiotherapy is necessary. Selective dorsal rhizotomy is effective for reducing spasticity in certain carefully selected young patients with bilateral spastic CP and can reduce the need for further surgical interventions and improve quality of life and independence with activities of daily living.[63] Further research is needed regarding the long term outcomes of SDR, especially with regard to functional activity and participation.[36,64]

Orthopaedic surgery

Musculoskeletal pathology develops in the limbs of most children with CP and orthopaedic surgery procedures such as tendon lengthenings, tendon transfers, rotational osteotomies, and joint stabilisation procedures have been developed to address the various components of this pathology.[65]

Children with severe CP commonly develop hip problems involving displacement of the femoral head resulting in pain, caregiving problems, seating problems, pressure sores, fractures and contractures. Regular hip surveillance programmes for children with CP allow earlier identification of subluxation and reduces the need for surgery on dislocated hips.[66] There is on-going debate about the best surgical intervention and the best timing of surgical intervention to deal with this problem.[67]

Equinus foot deformity is the most common musculoskeletal deformity in CP. There is low quality evidence supporting surgical intervention to correct this problem but no evidence to indicate which particular surgical technique is best.[68]

Surgical correction of thumb-in-palm deformity in children with spastic CP seems to improve hand function, to facilitate hygiene, and to improve the appearance and quality of life but the evidence is limited to prospective studies comparing pre- and post-operative outcomes.[69]

To avoid having to subject a child with CP to multiple hospital stays and rehabilitation periods it has become common to do single-event multilevel surgery (SEMLS) in which multiple levels of musculoskeletal pathology in both lower limbs are addressed by multiple surgical teams during a single surgical operation.[65] The evidence base for this approach is limited due to a lack of RCTs.[65] Most studies to date have been retrospective studies or prospective cohort studies and most have had fewer than 30 participants.[65]

Feeding interventions

Children with severe CP often have problems with sucking, chewing and swallowing their food. This puts them at risk of under nutrition and of recurrent aspiration pneumonia (due to inhaling food into their lungs).[70] Both behavioural and surgical techniques may be used to deal with feeding difficulties. The available research literature provides little evidence on the effectiveness of behavioural therapies (such as positioning, altering food consistency, or use of feeding devices) due to a lack of RCTs and generally small sample sizes.[70,71] There is no RCT evidence regarding feeding tubes.[72] Evidence from six case series indicates that surgically placed feeding tubes (gastrostomy or jejeunostomy tubes) improve weight gain[73] but there is insufficient evidence regarding their effects on respiratory outcomes, parent and child quality of life, or long term morbidity and mortality. There is some low quality evidence that they increase the potential for over feeding and gastro-oesophageal reflux.[73]

Evidence-based health care for children and young people with cerebral palsy

These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of CP are suggestions for further reading.

New Zealand guidelines
- Child Development Centre Therapy Team, Waikato District Health Board. 2014. Cerebral Palsy Clinical Practice Guideline. Hamilton: Waikato District Health Board. http://www.waikatodhb.health.nz/assets/for-health-professionals/Primary-care-management-guidelines/Cerebral-Palsy-Clinical-Practice-Guideline.pdf

International guidelines
- National Institute for Health and Care Excellence. 2017. Cerebral palsy in under 25s: assessment and management https://www.nice.org.uk/guidance/ng62
- National Institute for Health and Care Excellence. 2015. Preterm labour and birth. https://www.nice.org.uk/guidance/ng25
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Evidence-based medicine reviews
The following review provides an excellent overview of the evidence for interventions for children with cerebral palsy:
- Novak I, McIntyre S, Morgan C, et al. 2013. A systematic review of interventions for children with cerebral palsy: state of the evidence. *Developmental Medicine & Child Neurology,* 55(10) 885-910. http://dx.doi.org/10.1111/dmcn.12246
- The Cochrane Library reviews related to cerebral palsy. http://www.cochranelibrary.com/topic/Neurology/Neurodevelopmental%20disorders/Cerebral%20palsy/?stage=review
- Ozel S, Switzer L, Macintosh A, et al. 2016. Informing evidence-based clinical practice guidelines for children with cerebral palsy at risk of osteoporosis: an update. *Developmental Medicine & Child Neurology,* 58(9) 918-923 http://dx.doi.org/10.1111/dmcn.13196
- Lindsay S. 2016. Child and youth experiences and perspectives of cerebral palsy: a qualitative systematic review. *Child: Care, Health & Development,* 42(2) 153-75. http://dx.doi.org/10.1111/cch.12309
- Kruijsen-Terpstra AJ, Ketelaar M, Boeije H, et al. 2014. Parents' experiences with physical and occupational therapy for their young child with cerebral palsy: a mixed studies review. *Child: Care, Health & Development,* 40(6) 787-96. http://dx.doi.org/10.1111/cch.12097

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- Andersen JC, Majnemer A, O'Grady K, et al. 2013. Intensive upper extremity training for children with hemiplegia: from science to practice. *Seminars Pediatric Neurology,* 20(2) 100-5. http://dx.doi.org/10.1016/j.spen.2013.06.001

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Other relevant publications

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Websites
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- CanChild. Cerebral palsy related resources. https://www.canchild.ca/en/diagnoses/cerebral-palsy/related_resources
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EPILEPSY

Introduction

An epileptic seizure is the manifestation of abnormal or excessive discharge of neurons in the brain.[1] Epilepsy is defined as recurrent seizures, specifically two or more seizures separated by at least 24 hours but within 18 months of one another.[1] There are many different types of epileptic seizure. Seizures may involve abnormal movements such as jerking, twitching, repetitive movements or sudden loss of muscle tone, loss of consciousness, abnormal sensory perceptions such as strange tastes or smells or a feeling of déjà vu, or intense emotions such as joy or fear.[2] Prolonged or recurrent seizures (without a return to normal function between seizures) lasting 30 minutes or more constitute status epilepticus.[3]

Epilepsy is relatively common in children, affecting around four in every 1,000.[4,5] Epilepsy in children often occurs in association with mental health disorders or disabilities including developmental delay, attention deficit hyperactivity disorder, autism, anxiety and depression.[6,7] Epilepsy can result from genetic conditions (such as Angelman and Rett syndromes) and metabolic or structural conditions (such as head trauma and central nervous system malformations, infections or tumours) but in the majority of cases of epilepsy in children no cause can be identified.[2,5]

Children with epilepsy and their families need on-going specialist medical care.[8] While there are effective drugs for treating epilepsy, they are not effective for all children, and some children have difficulty adapting to drug treatment.[2,8] Living with epilepsy affects many aspects of quality of life. Studies have generally found that the emotional impacts are more significant than the physical ones.[9] Negative impacts of epilepsy include fatigue, medication side effects, anxiety and depression, social isolation due to being unwilling to disclose epilepsy to peers, restriction of leisure activities because of fear of having a seizure, and, for young people, being unable to get a driver licence, stay up late or drink alcohol.[9,10]

The majority of people with childhood-onset epilepsy do not need to take anti-epileptic drugs for life.[2] Patients followed long term have remission rates of around 65%.[11]

Data sources and methods

Indicators

> *Rates of epilepsy or status epilepticus among 0–24 year olds*

Definition

Hospitalisations of 0–24 year olds with epilepsy or status epilepticus per 100,000 population

Data sources

Numerator: National Minimum Dataset

Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

This section presents analyses where the condition was the primary diagnosis or was documented within any of the first 15 diagnoses (all cases). The rationale for presenting all cases is to highlight the full spectrum of health issues experienced by those with this condition, and their consequent requirement for acute health services.

Analyses are per hospital discharge event therefore events are only included if the condition is documented within either the primary diagnosis or within any of the first 15 diagnoses

Appendix 3 outlines the limitations of the data utilised from the National Minimum Dataset. The reader is advised to review this appendix before interpreting any trends.

Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

There were 58 deaths of 0–24 year olds with epilepsy or status epilepticus as the underlying cause of death in New Zealand during 2009 to 2013, as documented within the National Mortality Collection.

The number of 0–24 year olds hospitalised between 2011 and 2015 with any diagnosis of epilepsy or status epilepticus is presented together with the total number of hospitalisations with epilepsy or status epilepticus as a primary or any diagnosis. Most hospitalisations of 0–24 year olds with epilepsy or status epilepticus had this condition as the primary diagnosis (**Table 31**).There has been a small increase in hospitalisation rates for

epilepsy or status epilepticus since 2000 with a drop in 2015 (**Figure 44**). Hospitalisation rates were highest for 0–4 year olds (**Figure 45**). Similar patterns over time were seen in Māori and Pacific ethnic groups while rates for European/Other are trending down, and Asian ethnicities are trending up (**Figure 46**).

Table 31. Individuals aged 0–24 years hospitalised with epilepsy or status epilepticus using primary diagnosis compared to all cases, New Zealand 2011–2015

Age group	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Epilepsy or status epilepticus				
Hospitalisation				
0–24 years	4,336	7,440	9,876	1.33
0–14 years	2,542	4,809	6,237	1.30
15–24 years	1,884	2,631	3,639	1.38

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations where epilepsy or status epilepticus was in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0– 24 year old total

Figure 44. Hospitalisations for epilepsy or status epilepticus in 0–24 year olds, New Zealand, 2000–2015
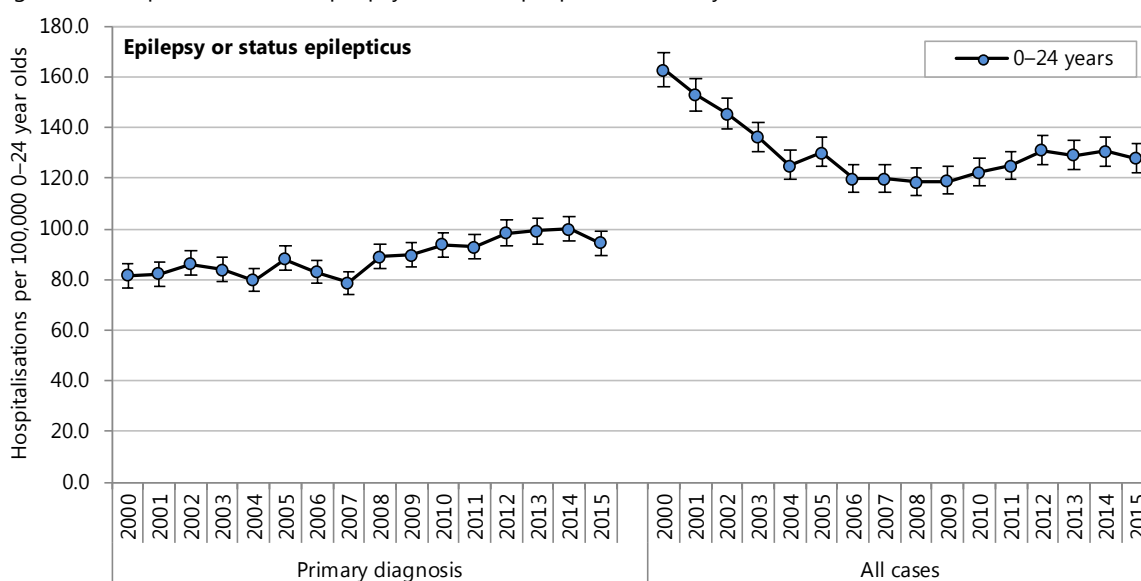
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with epilepsy or status epilepticus in any of the first 15 diagnoses

Figure 45. Hospitalisations for epilepsy or status epilepticus in 0–24 year olds, by age group, New Zealand, 2000– 2015
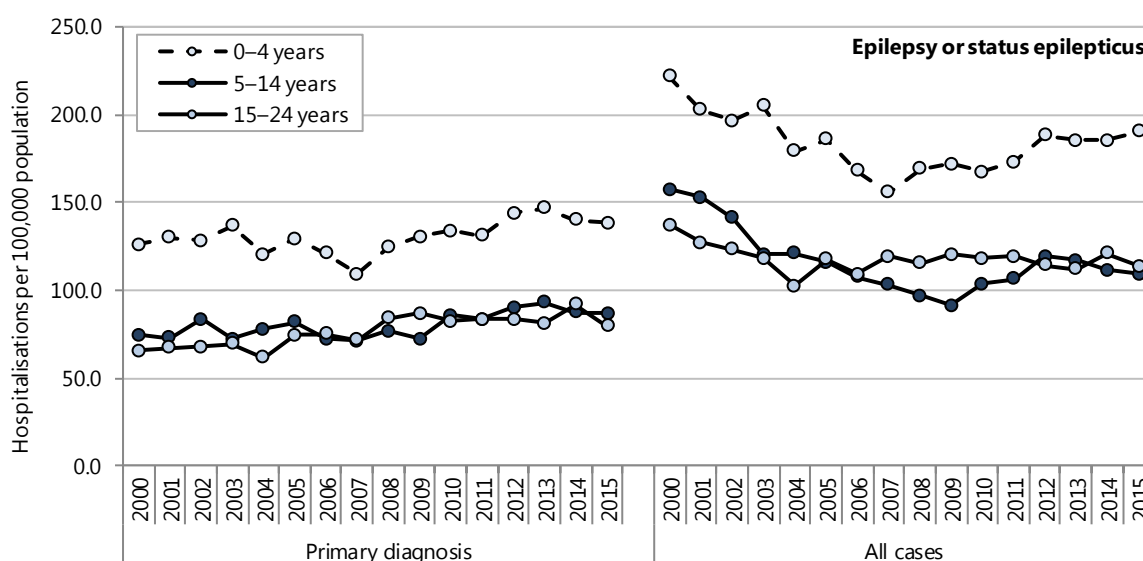
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with epilepsy or status epilepticus in any of the first 15 diagnoses

Figure 46. Hospitalisations involving epilepsy or status epilepticus in 0–24 year olds, by ethnicity, New Zealand, 2000–2015
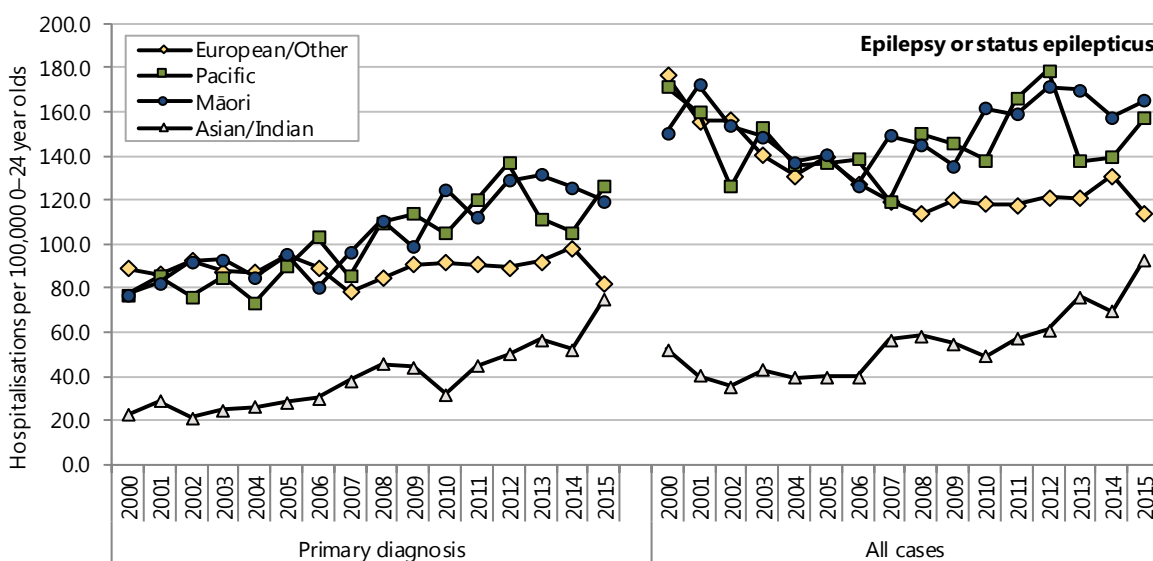
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. Rate per 100,000 age-specific population; 'All cases' corresponds to hospitalisations with epilepsy or status epilepticus listed in any of the first 15 diagnoses

Demographic distribution

Table 32 presents the demographic distribution of individuals hospitalised with epilepsy or status epilepticus in New Zealand between 2011 and 2015. The prevalence of epilepsy or status epilepticus was significantly higher among individuals residing in areas with high deprivation scores (NZDep2013 deciles 9–10). Compared to 15–24 year olds, the prevalence was significantly higher among 0–4 year olds and significantly lower among 5–14 year olds. The prevalence in Māori and Pacific 0–24 year olds was significantly higher than in European/Other 0–24 year olds. The majority of 0–24 year olds with epilepsy or status epilepticus were of European/Other or Māori ethnicities.

Table 32. Individuals aged 0–24 years hospitalised with epilepsy or status epilepticus, by demographic factor, New Zealand, 2011–2015

Variable	Unique individuals 2011–2015 *(n)*	Rate per 100,000 population	Rate ratio	95% CI
Epilepsy or status epilepticus* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	578	40.73	1.00	
Deciles 3–4	711	53.17	1.31	1.17–1.46
Deciles 5–6	757	52.51	1.29	1.16–1.44
Deciles 7–8	1,062	65.37	1.61	1.45–1.78
Deciles 9–10	1,621	87.24	2.14	1.95–2.36
Prioritised ethnicity				
Māori	1,230	68.19	1.23	1.14–1.31
Pacific	491	69.29	1.25	1.13–1.37
Asian/Indian	289	30.15	0.54	0.48–0.61
MELAA	70	69.41	1.25	0.98–1.58
European/Other	2,286	55.64	1.00	
Gender				
Female	2,060	54.87	1.00	
Male	2,276	57.97	1.06	1.00–1.12
Age group (years)				
0–4	1,195	76.62	1.28	1.19–1.37
5–14	1,466	49.13	0.82	0.76–0.88
15–24	1,884	60.06	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Epilepsy or status epilepticus in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is level 1 prioritised; Decile is NZDep2013

Regional distribution

Table 33 presents the number of individuals resident in each district health board area that had an epilepsy or status epilepticus diagnosis between 2011 and 2015. It also presents the number of hospital discharges in which epilepsy or status epilepticus was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and result in less undercounting than would occur if only the primary diagnosis is included in discharge data. For epilepsy or status epilepticus the All:Primary diagnosis ratio was higher than the national ratio in Northland, Bay of Plenty, Waikato, Taiarawhiti, MidCentral, Nelson Marlborough and Canterbury DHBs and lower in Auckland, Counties Manukau, Lakes, Taranaki, Whanganui, Hutt Valley, Capital & Coast, Wairarapa, and Southland DHBs. Ratios were similar to the national ratio in the remaining district health boards. The pattern of hospitalisations in the Waitemata, Hawke's Bay, South Canterbury, West Coast, Otago, Southern DHBs has been similar to/ different from the NZ pattern from 2000 to 2014 (**Table 33**).

Table 33. Hospitalisations for epilepsy or status epilepticus in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Epilepsy or status epilepticus in 0–24 year olds				
Northland	192	351	486	1.38
Waitemata	527	932	1,221	1.31
Auckland	388	621	789	1.27
Counties Manukau	616	1,057	1,341	1.27
Waikato	469	635	877	1.38
Bay of Plenty	248	359	515	1.43
Lakes	126	225	286	1.27
Tairawhiti	48	81	112	1.38
Taranaki	108	167	212	1.27
Hawke's Bay	192	394	518	1.31
MidCentral	184	269	381	1.42
Whanganui	100	157	198	1.26
Hutt Valley	106	214	274	1.28
Capital & Coast	250	465	576	1.24
Wairarapa	46	80	102	1.28
Nelson Marlborough	94	147	212	1.44
South Canterbury	43	51	69	1.35
Canterbury	427	645	935	1.45
West Coast	29	37	49	1.32
Southern	275	505	662	1.31
New Zealand	4,336	7,440	9,876	1.33

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with epilepsy or status epilepticus listed in any of the first 15 diagnoses; Note that individuals may appear in multiple DHBs

Evidence for good practice

Possibilities for prevention

Epilepsy in children is generally not preventable. Interventions to prevent traumatic brain injuries could reduce the number of children with epilepsy due to brain injury but this would have only a very small impact on the total number of children with epilepsy.

Evidence-based health care for children and young people with epilepsy

All children suspected of having epilepsy should be seen by a specialist paediatrician with expertise in epilepsy and children diagnosed with epilepsy need on-going regular specialist care.[12] Drug therapy is generally recommended after a person has had a second epileptic seizure and around two thirds of people with epilepsy achieve satisfactory control of their epilepsy with anti-epileptic drugs.[12] The use of sodium valproate is contraindicated in young women of childbearing age because it poses risks to unborn children (fetuses).[13] These include increased risks of congenital anomalies, developmental delay and autism.[13]

When seizures are not controlled by anti-epileptic drugs other treatments that may be effective include the ketogenic diet, vagus nerve stimulation and brain surgery to remove the area of the brain causing the seizures.[12]

Most childhood seizures last for less than five minutes. Prolonged or recurrent seizures (without a return to normal function between seizures) lasting 30 minutes or more constitute status epilepticus, a potentially fatal medical emergency requiring prompt treatment (usually in hospital) to prevent brain damage.[3]

Children and families affected by epilepsy need to be given, and have access to, high quality information about epilepsy and its management and they should be able to contact a named member of the healthcare team when they require information.[12] There is insufficient evidence to determine how best to facilitate self-management of epilepsy.[14]

These national and international guidelines, systematic reviews, other publications and websites relevant to the management of epilepsy are provided for further reading.

International guidelines

- Sauro KM, Wiebe S, Dunkley C, et al. 2016. The current state of epilepsy guidelines: A systematic review. *Epilepsia*, 57(1), 13-23. http://dx.doi.org/10.1111/epi.13273
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- National Clinical Guideline Centre. 2012. The epilepsies: The diagnosis and management of the epilepsies in adults and children in primary and secondary care. London: National Clinical Guideline Centre. https://www.nice.org.uk/guidance/cg137/evidence/full-guideline-6664855034 (This is the full guideline with details of the evidence.) The appendices to the guideline, including a 2014 evidence update, can be found here: https://www.nice.org.uk/guidance/cg137/evidence accessed, July 2016
- American Epilepsy Society. Guidelines https://www.aesnet.org/clinical_resources/guidelines accessed, July 2016
- American Academy of Neurology. Practice guidelines Epilepsy. https://www.aan.com/Guidelines/home/ByTopic?topicId=23 accessed, July 2016

Evidence-Based Medicine Reviews

- The Cochrane Library has many reviews relating to epilepsy management, mostly concerned with drug treatment: http://www.cochranelibrary.com/topic/Neurology/Epilepsy/?per-page=100&stage=review
- Lewis SA, Noyes J, Hastings RP. 2015. Systematic review of epilepsy self-management interventions integrated with a synthesis of children and young people's views and experiences. *Journal of Advanced Nursing*, 71(3), 478-97. http://dx.doi.org/10.1111/jan.12511
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- Geerlings RPJ, Aldenkamp AP, de With PHN, et al. 2015. Transition to adult medical care for adolescents with epilepsy. *Epilepsy & Behavior*, 44, 127-35.

Other relevant publications

- Keenan N, Sadlier LG. 2015. Paediatric EEG provision in New Zealand: a survey of practice. *New Zealand Medical Journal*, 128(1411), 43-50. http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2015/vol-128-no-1411–27-mar-2015/6479
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- Ridsdale L, McCrone P, Morgan M, et al. 2013. Can an epilepsy nurse specialist-led self-management intervention reduce attendance at emergency departments and promote well-being for people with severe epilepsy? A non-randomised trial with a nested qualitative phase. *Health Services and Delivery Research*, 1(9). http://www.journalslibrary.nihr.ac.uk/__data/assets/pdf_file/0011/87086/FullReport-hsdr01090.pdf

Websites

- Epilepsy NZ. http://epilepsy.org.nz/
- Epilepsy Australia. http://www.epilepsyAustralia.net/
- Epilepsy Society. https://www.epilepsysociety.org.uk/
- American Epilepsy Society. https://www.aesnet.org/

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CHRONIC LOWER RESPIRATORY DISEASES

Introduction

Chronic lower respiratory diseases, including asthma, chronic suppurative lung disease and bronchiectasis, affect the airways and other structures of the lung causing symptoms including: difficulty breathing, chronic cough, tiredness and sputum production.[1,2] There is a high degree of inequality across the socioeconomic spectrum and between ethnic groups in rates of respiratory disease.[3]

Chronic lower respiratory diseases are a component of the health status of individuals and also reflect the environments within which children live, work, and play. Common risk factors include poverty, poorly heated homes and household crowding, poor nutrition, frequent or severe lower respiratory infections during childhood, exposure to tobacco smoke and environmental air pollution.[1,2] Bronchiectasis is a chronic lung disease that causes a decline in lung function and is associated with repeated acute lower respiratory infections especially during the first year of life.[4,5] Household crowding is associated with an increased risk of hospitalisation with severe infections with respiratory syncytial virus (RSV) which is the most common cause of lower respiratory tract infections in children worldwide.[6,7] The effects of air pollution may begin before birth increasing the susceptibility of the unborn child to respiratory and other diseases. Children who develop chronic respiratory disease are also more likely than other children to experience adverse effects of air pollution.[8]

The following section reviews the community prevalence of asthma using information from the New Zealand Health Survey. Chronic lower respiratory diseases are reviewed using information from the National Minimum Dataset and the National Mortality Collection. Detailed information about hospitalisations for individual conditions is available in the 2015 NZCYES reports at www.otago.ac.nz/nzcyes. The section concludes with a brief overview of evidence for good practice for these conditions.

Data sources and methods

Indicators

Prevalence of asthma (medicated)
Rates of chronic lower respiratory diseases among 0–24 year olds

Definition

Prevalence of asthma
Asthma diagnosed by doctor and using inhalers, medicine, tablets, pills or other medication. Child respondents (aged 2–14 years) are defined as having asthma if the child's parents or caregivers had ever been told by a doctor that the child has asthma and if they now take treatments for asthma (inhalers, medicine, tablets or pills).[a] Adult respondents (aged 15+ years) are defined as having asthma if they had ever been told by a doctor that they have asthma and if they were taking treatments for asthma (inhalers, medicine, tablets or pills, or any other treatments). Medication can be taken daily to prevent symptoms, or only when needed to relieve symptoms.[a]
Rates
Hospitalisations of 0–24 year olds with a chronic lower respiratory disease per 1,000 population

Data sources

Prevalence of asthma (medicated)
New Zealand Health Survey (2006/07–2014/15, See **Appendix 3**
Rates of chronic lower respiratory diseases among 0–24 year olds

Numerator: National Minimum Dataset
Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

Prevalence of asthma (medicated): A total of 13,497 adults (15 years and over) and 4,754 children (0–14 year olds) participated in the NZ Health Survey 2014/15 during the period 1 July 2014 to 30 June 2015. The coverage rates were 59 percent for adults and 69 percent for children.[b] The NZ Health Survey utilised adjusted rate ratios to account for the potential influence of other demographic factors when undertaking demographic comparisons. Gender comparisons are adjusted for age, ethnic comparisons are adjusted for age and gender, and deprivation comparisons are adjusted for age, sex and ethnicity.[b]
Rates of chronic lower respiratory diseases: A chronic lower respiratory disease was the primary diagnosis or was documented as one of the first 15 diagnoses. Chronic lower respiratory diseases comprises asthma (and wheeze), bronchitis, bronchiectasis, emphysema and other lower respiratory. Codes used for identifying cases are documented in **Appendix 5**.

References

[a.] Ministry of Health. 2015. Indicator Interpretation Guide 2014/15: New Zealand Health Survey. Wellington: Ministry of Health.
[b.] Ministry of Health. 2015. Methodology Report 2014/15: New Zealand Health Survey. Wellington: Ministry of Health.

National trends and distribution

There was a total of 32 deaths of 0–24 year olds where a chronic lower respiratory disease was the underlying cause of death in New Zealand between 2009 and 2013, as documented within the National Mortality Collection. The majority of these deaths (26) were due to asthma (including status asthmaticus and wheeze).

Over the five years of the NZHS 2006/07 to 2014/15, from 14% to 15% of 2–14 year olds and 9% to 12% of 15–24 year olds were reported to be using medication for asthma diagnosed by a doctor. Prevalence has been stable over time with some variation from survey year to survey year (**Figure 47**).

In the 2014/15 NZHS year the prevalence of asthma (medicated) was highest among 5–9 year olds and the prevalence rate for 15–24 year olds was significantly lower than that for 5–9 and 10–14 year olds (**Figure 48**). Among 2–14 year olds, prevalence was significantly higher for Māori compared with other ethnic groups. There was no significant difference in prevalence by NZDep2013 score of home address, nor by gender (**Figure 48**; **Table 34**).

Figure 47. Asthma (medicated) in 2–24 year olds, by age group and survey year, NZ Health Survey 2006/07–2014/15
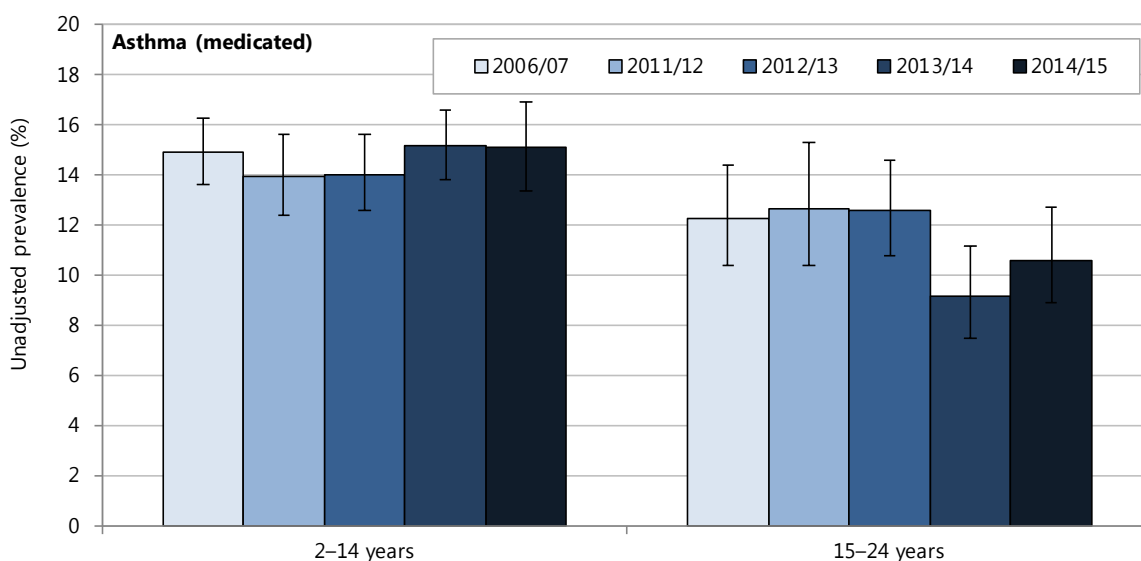
Source: New Zealand Health Survey

Figure 48. Asthma (medicated), by demographic factor, 2014/15 NZ Health Survey
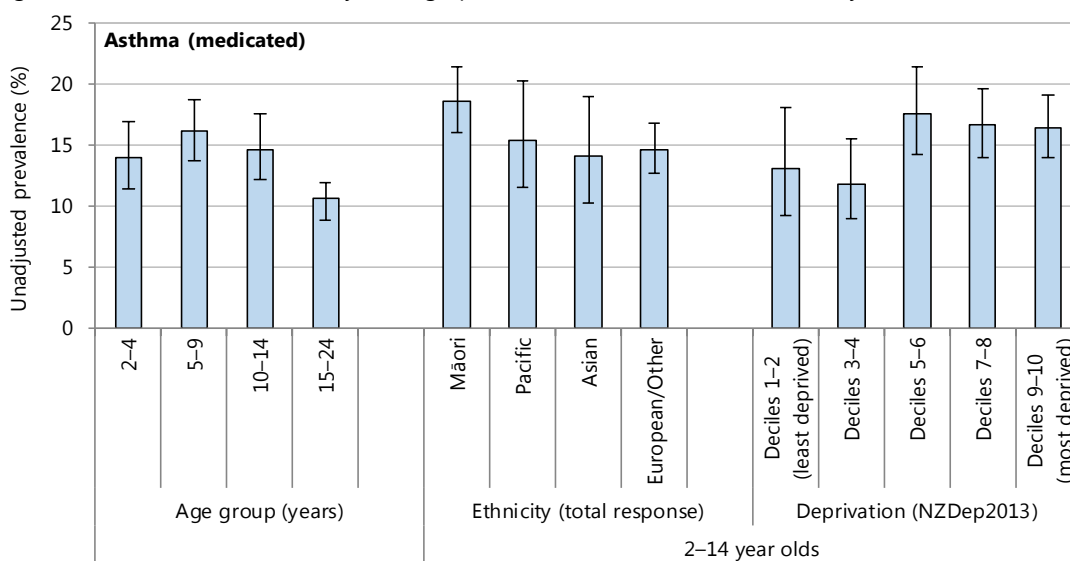
Source: New Zealand Health Survey

Table 34. Comparisons for 2–14 year olds diagnosed with asthma, by demographic factors, NZ Health Survey 2014/15

Variable	Unadjusted prevalence (%)	Adjusted rate ratio	95% CI
Asthma (medicated) in 2–14 year olds			
New Zealand			
NZ Deprivation Index quintile			
Deciles 1–2	13.09	1.00	
Deciles 3–4	11.84	...	
Deciles 5–6	17.54	...	
Deciles 7–8	16.66	...	
Deciles 9–10	16.42	1.39	0.96–2.02
Ethnicity (total response)			
Māori (vs non-Māori)	18.63	1.35	1.14–1.60
Pacific (vs non-Pacific)	15.38	1.03	0.77–1.36
Asian (vs non-Asian)	14.09	0.91	0.66–1.26
European/Other	14.63	...	
Gender			
Female	13.58	1.00	
Male	16.52	1.22	0.99–1.50

Source: New Zealand Health Survey

The number of 0–24 year olds hospitalised with chronic lower respiratory diseases during 2011 to 2015 is presented in **Table 35**. It also presents the number of hospital discharges in which chronic lower respiratory diseases were documented as the primary diagnosis or as any diagnosis.

The rate of hospitalisations where a chronic lower respiratory disease was the primary diagnosis has increased since 2000 (**Figure 49**).

Table 35. Individuals aged 0–24 years hospitalised with chronic lower respiratory diseases using primary diagnosis compared to all cases, New Zealand 2011–2015

	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Chronic lower respiratory diseases				
Hospitalisation				
0–24 years	29,184	37,909	48,433	1.28
0–14 years	25,094	33,314	41,913	1.26
15–24 years	4,236	4,595	6,520	1.42
Chronic lower respiratory diseases in 0–24 years year olds				
Asthma and wheeze	28,195	35,982	45,274	1.26
Bronchiectasis	729	1,362	2,516	1.85
Bronchitis	341	197	384	1.95
Emphysema	124	24	137	5.71
Other	219	344	525	1.53

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with chronic lower respiratory diseases listed in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Figure 49. Hospitalisations for chronic lower respiratory diseases in 0–24 year olds, New Zealand 2000–2015
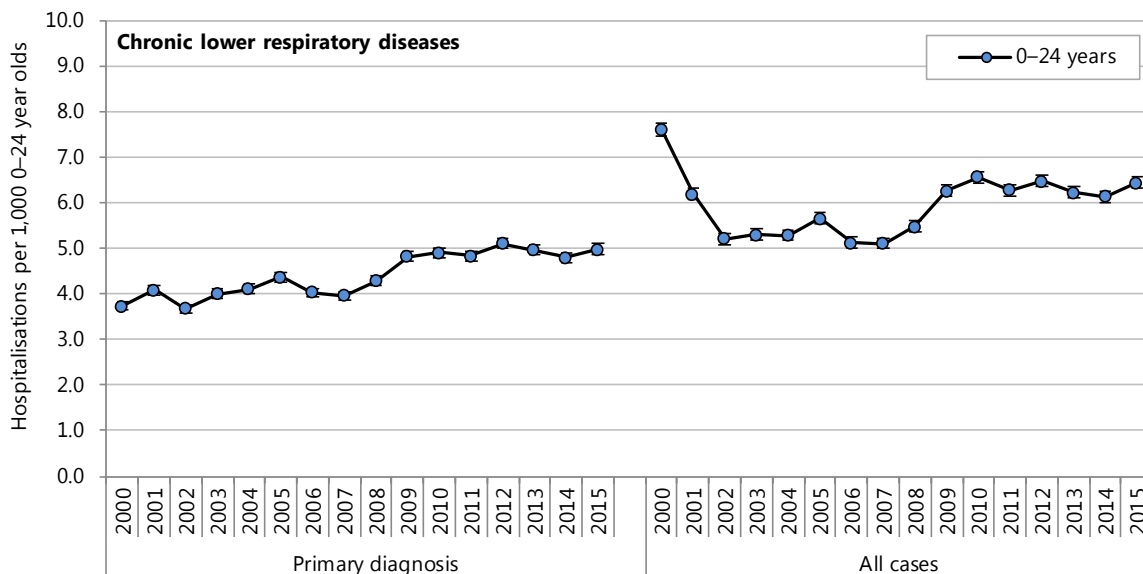
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with chronic lower respiratory diseases listed in any of the first 15 diagnoses

Demographic distribution

Table 36 presents the demographic distribution of individuals with chronic lower respiratory diseases in New Zealand between 2011 and 2015. Chronic lower respiratory diseases were significantly higher among males, and among 0–4 and 5–14 year olds (compared to 15–24 year olds). There was a strong social gradient among these individuals, with statistically significant increases in prevalence in each deprivation quintile (NZDep deciles 3–4 to 9–10) compared with those living in least deprived areas (deciles 1–2). The rate of chronic lower respiratory diseases was significantly lower for European/Other ethnicities than for the other ethnic groups.

Table 36. Individuals aged 0–24 years hospitalised with chronic lower respiratory diseases, by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (n)	Rate per 1,000 population	Rate ratio	95% CI
Chronic lower respiratory diseases* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	3,458	2.44	1.00	
Deciles 3–4	4,005	3.00	1.23	1.17–1.29
Deciles 5–6	4,895	3.40	1.39	1.33–1.46
Deciles 7–8	7,173	4.42	1.81	1.74–1.89
Deciles 9–10	11,351	6.11	2.51	2.41–2.60
Prioritised ethnicity				
Māori	9,815	5.44	2.06	2.00–2.12
Pacific	5,300	7.48	2.83	2.74–2.92
Asian/Indian	2,825	2.95	1.11	1.07–1.16
MELAA	490	4.86	1.84	1.68–2.01
European/Other	10,860	2.64	1.00	
Gender				
Female	12,516	3.33	1.00	
Male	16,670	4.25	1.27	1.24–1.30
Age group (years)				
0–4	18,079	11.59	8.58	8.30–8.88
5–14	8,040	2.69	2.00	1.92–2.07
15–24	4,236	1.35	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Chronic lower respiratory diseases in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is Level 1 prioritised; Decile is NZDep2013

Since 2000, both primary diagnosis and all cases hospitalisations for chronic lower respiratory diseases have increased considerably for 0–4 year olds (**Figure 50**). Over the same period, the primary diagnosis hospitalisation rate gradually increased for Pacific, Māori, and Asian/Indian ethnic groups, and remained relatively constant for European/Other (**Figure 51**).

Figure 50. Hospitalisations involving chronic lower respiratory diseases in 0–24 year olds, by age group, New Zealand 2000–2015
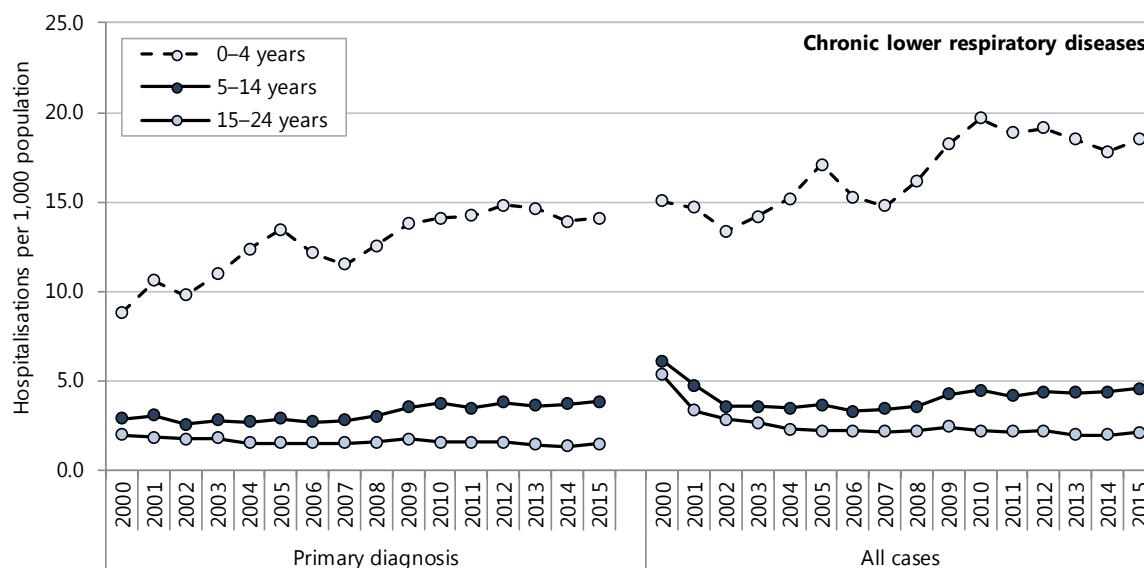
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with chronic lower respiratory diseases listed in any of the first 15 diagnoses

Figure 51. Hospitalisations involving chronic lower respiratory diseases in 0–24 year olds, by ethnicity, New Zealand 2000–2015
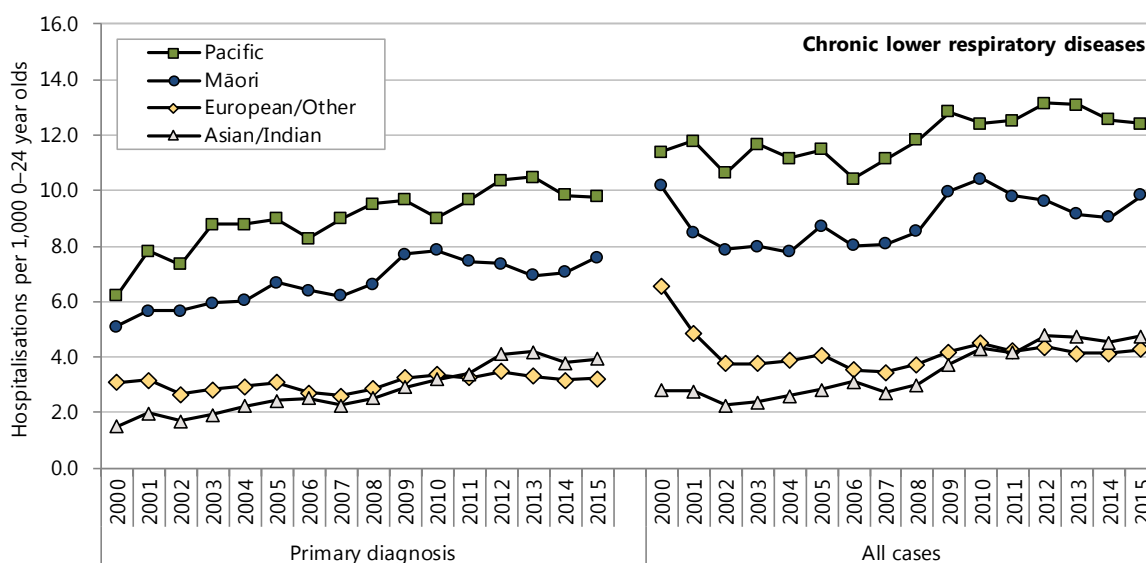
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with chronic lower respiratory diseases listed in any of the first 15 diagnoses

Regional distribution

Figure 52 shows the prevalence of asthma in each DHB, as reported in the NZ Health Survey. Prevalence of asthma in 2–14 year olds was slightly but significantly higher than the national prevalence in Northland, Tairawhiti and Whanganui DHBs, and signicantly lower than the national prevalence in Auckland DHB.

Figure 52. Asthma (medicated) in 2–14 year olds, by district health board, NZ Health Survey 2011–2014
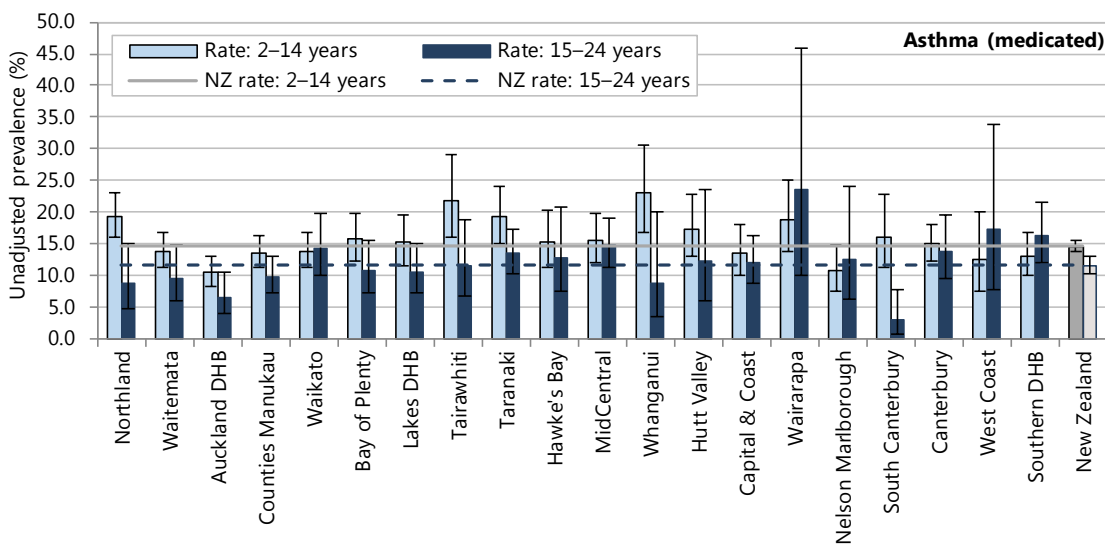
Source: NZ Health Survey

Table 37 presents the number of individuals resident in each district health board that had a chronic lower respiratory disease diagnosis during 2011 to 2015. It also presents the number of hospital discharges in which a chronic lower respiratory disease was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and avoids the undercounting that would occur if only primary diagnoses are included in discharge data. For chronic lower respiratory diseases the All:Primary diagnosis ratio was higher than the national ratio in Northland, Counties Manukau, Bay of Plenty, Tairawhiti, Hawke's Bay, MidCentral,

Whanganui, Hutt Valley, Nelson Marlborough, South Canterbury, and West Coast DHBs. Ratios were lower than the national ratio in the remaining district health boards (**Table 37**).

Table 37. Hospitalisations for chronic lower respiratory diseases in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Chronic lower respiratory diseases in 0–24 year olds				
Northland	1,218	1,564	2,068	1.32
Waitemata	3,561	4,492	5,640	1.26
Auckland	3,815	5,465	6,272	1.15
Counties Manukau	4,477	5,282	7,061	1.34
Waikato	2,592	3,190	4,065	1.27
Bay of Plenty	1,777	2,012	2,860	1.42
Lakes	775	1,066	1,323	1.24
Tairawhiti	426	497	741	1.49
Taranaki	675	878	1,078	1.23
Hawke's Bay	887	1,002	1,443	1.44
MidCentral	960	1,085	1,497	1.38
Whanganui	471	543	780	1.44
Hutt Valley	1,168	1,409	1,938	1.38
Capital & Coast	1,764	2,347	2,738	1.17
Wairarapa	230	298	369	1.24
Nelson Marlborough	539	546	889	1.63
South Canterbury	157	154	214	1.39
Canterbury	2,698	3,721	4,519	1.21
West Coast	139	144	206	1.43
Southern	1,488	2,038	2,503	1.23
New Zealand	29,184	37,909	48,433	1.28

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with chronic lower respiratory diseases listed in any of the first 15 diagnoses; Sum of DHBs may equal more than NZ total due to some individuals changing DHB of domicile during the period

Evidence for good practice

Possibilities for prevention

Childhood respiratory disease can be prevented or ameliorated by several basic measures including: improving childhood nutrition, promoting breastfeeding, complete and timely immunisation, improving living conditions to prevent crowding, avoiding tobacco smoke exposure and reducing indoor air pollution. Avoiding smoking during pregnancy and avoidance of passive smoke exposure after birth can reduce asthma severity in children.[8,9] The emphasis needs to be on smoking cessation, as exposure to environmental tobacco smoke remains high even when smoking parents maintain smoke-free homes and cars.[10] Legislation and political action on clean air makes a difference and can significantly reduce hospitalisations for respiratory disease.[8]

Interventions to effectively reduce disparities in respiratory health are essential and will require change from individuals, health care providers and health policy leaders to create the broad societal change needed to address the wider determinants of health.[11] Addressing social determinants of health and improving health service delivery are both important.[5] Health service providers need appropriate clinical skills to understand patients' beliefs, attitudes, experiences, and behaviours and demonstrate cross-cultural communication and competence in interactions with patients.[11] Measures to prevent premature birth and to reduce acute lower respiratory infections are important equity issues to reduce bronchiectasis in indigenous children.[4,5]

An effective approach to addressing respiratory disease includes ready access to highly skilled health care, early (rather than late) intervention, close links between the various components of the health sector and high levels of health literacy.[2] Asthma severity and hospitalisation rates can be reduced through better treatment, improved access to primary care and educational interventions for parents, children and healthcare providers.[12] Observed disparities in the dispensing of preventive asthma treatment to Māori and Pacific children need to be addressed.[13,14] Technology such as mobile phones, combined with a culturally sensitive approach, can be used to facilitate adherence to treatment.[15] It may be appropriate to trial interventions such as patient education

delivered by health-care professionals and long-term follow-up after acute care visits provided that an appropriate plan is in place to monitor effectiveness.[11]

Vaccination against non-typeable *Haemophilus influenza* can help to prevent infections for children who have chronic suppurative lung diseases.[5] Long term treatment with weekly azithromycin can decrease pulmonary exacerbations in indigenous children with chronic suppurative lung disease but is also associated with carriage of azithromycin-resistant bacteria.[16] Vaccination with palivizumab is safe and effective for preventing RSV hospitalisation in infants at high risk of severe infection, but it is expensive and requires monthly intravenous injection.[7] Randomised controlled trials are being undertaken in Auckland and Christchurch to study the efficacy and safety of maternal RSV vaccination in the third-trimester of pregnancy and particularly whether this can prevent RSV infection in their infants.[17]

Evidence-based health care for children and young people with chronic lower respiratory diseases

Timely diagnosis and effective treatment are important components of secondary and tertiary prevention of chronic lower respiratory disease, including reducing the number and severity of exacerbations for children with the conditions and in particular reducing the need for hospitalisation.[5,8,18] These national and international guidelines, systematic reviews, other publications and websites relevant to the management of chronic lower respiratory diseases are suggestions for further reading.

New Zealand guidelines

- Asthma and Respiratory Foundation of New Zealand. 2015. Te Hā Ora (The Breath of Life): National Respiratory Strategy. Wellington: The Asthma Foundation. https://www.asthmafoundation.org.nz/about-us/advocacy/national-respiratory-strategy

- Chang AB, et al. 2015. Chronic suppurative lung disease and bronchiectasis in children and adults in Australia and New Zealand Thoracic Society of Australia and New Zealand guidelines. http://dx.doi.org/10.5694/mja14.00287

- Edwards E. 2008. Starship Children's Health clinical guideline: Cough - Investigation of chronic cough &/or confirmed bronchiectasis. http://www.adhb.govt.nz/starshipclinicalguidelines/_Documents/Cough-Investigation of Chronic.pdf

International guidelines

- Global Initiative for Asthma. 2016. Global Strategy for Asthma Management and Prevention. http://ginasthma.org/2016-gina-report-global-strategy-for-asthma-management-and-prevention/

- British Thoracic Society, Scottish Intercollegiate Guidelines Network. 2014. British guideline on the management of asthma: A national clinical guideline. London, Edinburgh: British Thoracic Society, Scottish Intercollegiate Guidelines Network https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-2014/

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- Hill AT, et al. 2011. Primary care summary of the British Thoracic Society Guideline on the management of non-cystic fibrosis bronchiectasis. *Primary Care Respiratory Journal*, 20, 135. http://dx.doi.org/10.4104/pcrj.2011.00007

Evidence-based medicine reviews

- Anderson LM, et al. 2015. Community coalition-driven interventions to reduce health disparities among racial and ethnic minority populations. *Cochrane Database of Systematic Reviews, (6).* http://dx.doi.org/10.1002/14651858.CD009905.pub2

- Okelo SO, Butz AM, Sharma R, et al. 2013. Interventions to modify health care provider adherence to asthma guidelines. Comparative effectiveness review No. 95. (Prepared by Johns Hopkins University Evidence-based Practice Center under Contract No. 290-2007-10061-I.) AHRQ Publication No. 13-EHC022-EF. Rockville, MD: Agency for Healthcare Research and Quality. http://www.ncbi.nlm.nih.gov/books/NBK144097/
- Chang AB, et al. 2016. Children with chronic wet or productive cough—Treatment and investigations: A systematic review. *Chest,* 149(1), 120–42. http://dx.doi.org/10.1378/chest.15-2065
- Lee AL, et al. 2015. Airway clearance techniques for bronchiectasis. *Cochrane Database of Systematic Reviews*, (11). http://dx.doi.org/10.1002/14651858.CD008351.pub3
- Welsh Emma J, et al. 2015. Interventions for bronchiectasis: an overview of Cochrane systematic reviews. *Cochrane Database of Systematic Reviews,* http://dx.doi.org/10.1002/14651858.CD010337.pub2
- Frazer K, et al. 2016. Legislative smoking bans for reducing harms from secondhand smoke exposure, smoking prevalence and tobacco consumption. *Cochrane Database of Systematic Reviews,* (2). http://dx.doi.org/10.1002/14651858.CD005992.pub3

Other relevant publications
- Jones B, Ingham TR, Reid S, et al. 2015. He māramatanga huangō: Asthma health literacy for Māori children in New Zealand. Wellington: University of Otago. https://www.asthmafoundation.org.nz/research/he-maramatanga-huango-asthma-health-literacy-for-Māori-children-in-new-zealand
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Websites
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- PHARMAC. 2010. Space to breathe. http://www.spacetobreathe.co.nz/
- Menzies School of Health Research Centre for Research Excellence in respiratory health http://www.menzies.edu.au/page/Research/Centres_for_Research_Excellence/Centre_for_Research_Excellence_CRE_in_Respiratory_Health/
- Ministry of Health. 2015. Bronchiectasis. http://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/bronchiectasis
- Kidshealth. 2014. Bronchiectasis. http://www.kidshealth.org.nz/bronchiectasis-bx
- Health Navigator New Zealand. 2015. Bronchiectasis. http://www.healthnavigator.org.nz/health-topics/bronchiectasis/

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3. Telfar Barnard L, Baker M, Pierse N, et al. 2015. The impact of respiratory disease in New Zealand: 2014 update. https://www.asthmafoundation.org.nz/research/the-impact-of-respiratory-disease-in-new-zealand-2014-update accessed July, 2016
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6. Colosia AD, Masaquel A, Hall CB, et al. 2012. Residential crowding and severe respiratory syncytial virus disease among infants and young children: A systematic literature review. *BMC Infectious Diseases,* 12 95-95. http://dx.doi.org/10.1186/1471-2334-12-95
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8.   Forum of International Respiratory Societies. 2013. Respiratory diseases in the world: realities of today - Opportunities for tomorrow. https://www.thoracic.org/advocacy/global-public-health/firs/resources/firs-report-for-web.pdf accessed July, 2016

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16.   Valery PC, Morris PS, Byrnes CA, et al. 2013. Long-term azithromycin for Indigenous children with non-cystic-fibrosis bronchiectasis or chronic suppurative lung disease (Bronchiectasis Intervention Study): a multicentre, double-blind, randomised controlled trial. *The Lancet. Respiratory Medicine,* 1(8) 610-20. http://dx.doi.org/10.1016/s2213-2600(13)70185-1

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INFLAMMATORY BOWEL DISEASE (IBD)

Introduction

The two main forms of inflammatory bowel disease are ulcerative colitis (UC) and Crohn's Disease (CD), which both involve inflammation of the intestines with symptoms including diarrhoea (which may be bloody particularly in UC), abdominal pain, tiredness and weight loss.[1,2] IBD can develop at any age, with peak incidence between the ages of 15 and 25 years.[2] Over recent decades there has been an increase in IBD around the world, and the disorders are occurring more commonly in younger children.[3,4] These conditions affect multiple body systems including liver, joints, bone, skin and eyes.[4] Some children present with joint pain or rashes before bowel disease is evident.[3] Important comorbidities include impaired growth and pubertal development, anaemia, osteoporosis and increased risk of developing colorectal cancer.[2] Almost all children with CD and at least half of children with UC have poor weight gain or weight loss prior to diagnosis.[3] Treatments for IBD can suppress the immune system making children and young people more susceptible to vaccine-preventable diseases and increasing risk of some cancers.[4] IBD can disrupt normal activities including education, reduce quality of life and affect social and psychological wellbeing.[2]

The following section reviews IBD in children and young people using information from the National Minimum Dataset. The sections conclude with an overview of evidence for good practice for these conditions.

Data sources and methods

Indicators

> Rates of inflammatory bowel disease (IBD) among 0–24 year olds

Definition

Hospitalisations of 0–24 year olds with inflammatory bowel disease (IBD) per 100,000 population

Data sources

Numerator: National Minimum Dataset

Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

This section presents analyses where the condition was the primary diagnosis or was documented within any of the first 15 diagnoses (all cases). The rationale for presenting all cases is to highlight the full spectrum of health issues experienced by those with this condition, and their consequent requirement for acute health services. Analyses are per hospital discharge event therefore events are only included if the condition is documented within either the primary diagnosis or within any of the first 15 diagnoses.
Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

From 2000 to 2013 there were fewer than five deaths of 0–24 year olds with inflammatory bowel disease (IBD) as an underlying cause, as documented within the National Mortality Collection.

The number of 0–24 year olds hospitalised between 2011 and 2015 with any diagnosis of IBD is presented together with the total number of hospitalisations with Crohn disease or ulcerative colitis as the primary diagnosis or within any of the first 15 diagnoses (**Table 38**).

Since 2000 hospitalisation rates for IBD have risen, particularly for 15–24 year olds. Hospitalisation rates were consistently higher for 15–24 year olds compared with 0–14 year olds (**Figure 53**). Hospitalisation rates were consistently highest for European/Other 0–24 year olds and lowest for Pacific and Māori 0–24 year olds. The rise in rates over time was particularly marked for European/Other and Asian/Indian 0–24 year olds (**Figure 54**).

Table 38. 0–24 year olds hospitalised with inflammatory bowel disease, New Zealand 2011–2015

	Unique individuals (n)	Hospitalisations (n)		Ratio All : Primary
		Primary diagnosis	All cases	
Inflammatory bowel disease				
Hospitalisation				
0–24 years	1,447	5,168	5,999	1.16
0–14 years	312	1,092	1,277	1.17
15–24 years	1,205	4,076	4,722	1.16
Inflammatory bowel disease in 0–24 year olds				
Crohn disease	1,050	4,145	4,842	1.17
Ulcerative colitis	471	1,023	1,176	1.15

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations where inflammatory bowel disease was listed in any of the first 15 diagnoses; The sum of the age groups and of the diagnoses may total to more than the 0–24 year old total

Figure 53. Hospitalisations for inflammatory bowel disease, 0–24 year olds, by age group New Zealand 2000–2015
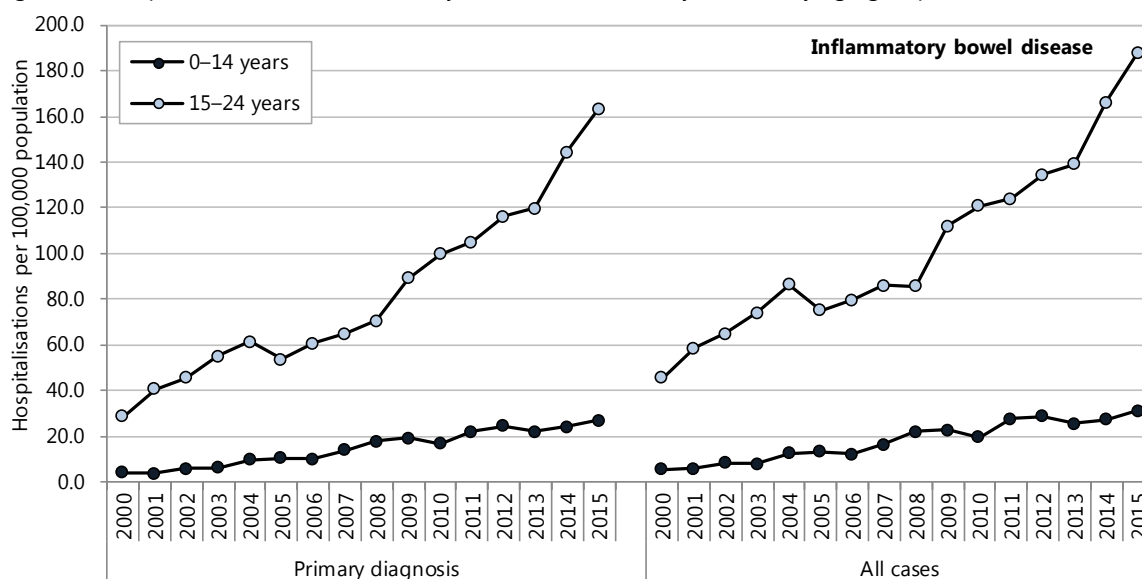
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations where inflammatory bowel disease was listed in any of the first 15 diagnoses

Demographic distribution

Table 39 presents the demographic distribution of individuals with inflammatory bowel disease in New Zealand between 2011 and 2015. Inflammatory bowel disease prevalence was significantly lower among individuals residing in areas with higher deprivation scores (NZDep2013 deciles 5–10) compared with areas with low deprivation scores (NZDep2013 deciles 1–2). Most individuals with IBD were in the 15–24 age group with very few aged under five years. By far the majority of 0–24 year olds with IBD were of European/Other ethnicity.

Figure 54. Hospitalisations involving inflammatory bowel disease in 0–24 year olds, by ethnicity, New Zealand 2000–2015
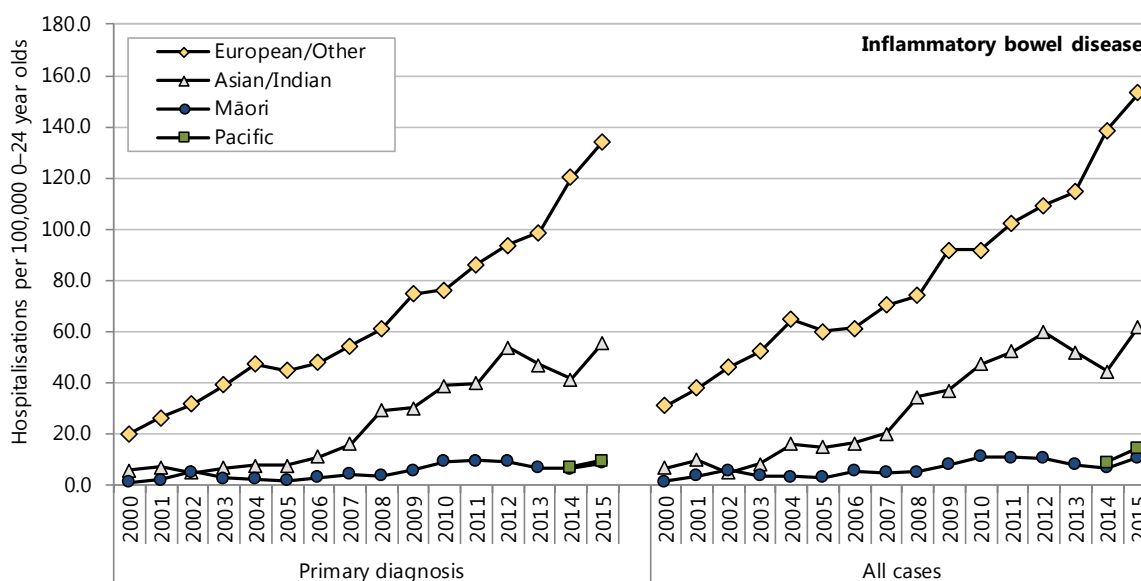
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with inflammatory bowel disease listed in any of the first 15 diagnoses; Rates for Pacific are suppressed (prior to 2014) due to small numbers

Table 39. 0–24 year olds hospitalised for inflammatory bowel disease, by demographic factor, New Zealand 2011– 2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Inflammatory bowel disease* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	374	26.35	1.00	
Deciles 3–4	381	28.49	1.08	0.94–1.25
Deciles 5–6	302	20.95	0.79	0.68–0.93
Deciles 7–8	358	22.04	0.84	0.72–0.97
Deciles 9–10	263	14.15	0.54	0.46–0.63
Prioritised ethnicity				
Māori	71	3.94	0.14	0.11–0.17
Pacific	21	2.96	0.10	0.07–0.16
Asian/Indian	124	12.94	0.45	0.37–0.54
MELAA	34	33.71	1.16	0.83–1.63
European/Other	1,193	29.04	1.00	
Gender				
Female	674	17.95	1.00	
Male	773	19.69	1.10	0.99–1.22
Age group (years)				
0–4	35	2.24	0.06	0.04–0.08
5–14	281	9.42	0.25	0.22–0.28
15–24	1,205	38.41	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Inflammatory bowel disease is listed in any of the first 15 diagnoses; Rate ratios are unadjusted; Ethnicity is level 1 prioritised; Decile is NZDep2013

Regional distribution

Table 40 presents the number of individuals resident in each district health board area that had an IBD diagnosis between 2011 and 2015. It also presents the number of hospital discharges in which inflammatory bowel disease was documented as the primary diagnosis or any diagnosis. The All:Primary diagnosis ratio for inflammatory bowel disease was close to one nationally and in all DHBs, indicating that IBD was the primary diagnosis for most hospitalisations with these conditions.

Table 40. Hospitalisations for inflammatory bowel disease in 0–24 year olds, by district health board, New Zealand 2010–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Inflammatory bowel disease in 0–24 year olds				
Northland	36	158	171	1.08
Waitemata	250	1,059	1,179	1.11
Auckland	154	510	561	1.10
Counties Manukau	124	248	320	1.29
Waikato	127	567	629	1.11
Bay of Plenty	60	176	192	1.09
Lakes	23	43	52	1.21
Tairawhiti	10	27	28	1.04
Taranaki	29	43	61	1.42
Hawke's Bay	47	89	115	1.29
MidCentral	53	296	315	1.06
Whanganui	12	41	43	1.05
Hutt Valley	53	168	209	1.24
Capital & Coast	105	373	436	1.17
Wairarapa	16	100	116	1.16
Nelson Marlborough	49	172	206	1.20
South Canterbury	21	50	59	1.18
Canterbury	204	583	725	1.24
West Coast	11	26	30	1.15
Southern	136	422	534	1.27
New Zealand	1,447	5,168	5,999	1.16

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' means inflammatory bowel disease is listed in any of the first 15 diagnoses; Rate ratios are unadjusted; Note that individuals may appear in multiple DHBs

Evidence for good practice

Possibilities for prevention

The precise cause of inflammatory bowel disease (IBD) is not clear although genes, bacteria and immune system responses in the digestive tract are all important contributing factors.[3] Although there is no strategy for primary prevention, one of the aims of IBD treatment is to prevent flare-ups happening once acute inflammation has healed.[2]

Evidence-based health care for children and young people with inflammatory bowel disease

Treatment of IBD aims to induce remission by healing inflammation and reducing symptoms during a flare-up, or to maintain remission by preventing flare-ups occurring. Various drugs can help with both of these aims and surgery may be an option for some young people.[2] IBD is a systemic condition that affects many body systems, and there are risks associated with the various immunosuppressive treatments used. It is important to be attentive to all aspects of physical and psychological health of children and young people with IBD, including monitoring growth, bone health and vitamin and mineral deficiencies, ensuring vaccinations are up-to-date, screening for depression, monitoring eye and skin health, providing dietary recommendations and making special considerations for international travel.[4] For young people who plan or become pregnant management of

IBD poses particular challenges as there is an increased risk of adverse birth outcomes.[5,6] These complex management requirements mean that children and young people with IBD benefit from engagement with a multidisciplinary team with appropriate specialist experience and expertise. Within New Zealand's dispersed geography this can be achieved by a shared care arrangement involving a tertiary level gastroenterologist in Auckland or Christchurch, regional paediatrician, tertiary and regional paediatric dieticians, nurses and other allied health professionals, and local primary care services. Regional Health Schools can provide continuity of education for children with significant or prolonged disruption to their usual schooling.[3]

Details of treatment options for people with inflammatory bowel disease, including risks associated with treatment, can be found in the guidelines and other evidence-based information sources provided below for further reading.

New Zealand guidelines
- Day A & Paediatric Gastroenterology Clinical Network. 2014. Management of inflammatory bowel disease in children and adolescents in New Zealand: A clinical guideline. Paediatric Society New Zealand & National Child and Youth Clinical Networks.
  https://www.starship.org.nz/media/256562/nz_ibd_clinical_guideline_aug_2015.pdf

International guidelines
- National Institute for Health and Care Excellence. 2016. Ulcerative colitis pathway.
  https://pathways.nice.org.uk/pathways/ulcerative-colitis
- National Institute for Health and Care Excellence. 2016. Crohn's disease pathway.
  http://pathways.nice.org.uk/pathways/crohns-disease
- Nguyen GC, et al. 2016. The Toronto consensus statements for the management of inflammatory bowel disease in pregnancy. *Gastroenterology*, 150(3), 734-57.e1. http://dx.doi.org/10.1053/j.gastro.2015.12.003

Evidence-based medicine reviews
- Ariyaratnam J & Subramanian V. 2014. Association between thiopurine use and nonmelanoma skin cancers in patients with inflammatory bowel disease: a meta-analysis. *American Journal of Gastroenterology*, 109, 163-69 http://dx.doi.org/10.1038/ajg.2013.451
- Bonovas S, et al. 2016. Intravenous versus oral iron for the treatment of anemia in inflammatory bowel disease: A systematic review and meta-analysis of randomized controlled trials. *Medicine*, 95(2), e2308. http://dx.doi.org/10.1097/md.0000000000002308
- Huang V, et al. 2014. Distance management of inflammatory bowel disease: Systematic review and meta-analysis. *World Journal of Gastroenterology*, 20, 829-42 http://dx.doi.org/10.3748/wjg.v20.i3.829
- Melek J & Sakuraba A. 2014. Efficacy and safety of medical therapy for low bone mineral density in patients with inflammatory bowel disease: a meta-analysis and systematic review. *Clinical Gastroenterology and Hepatology*, 12, 32-44.e5 http://dx.doi.org/10.1016/j.cgh.2013.08.024
- Mozaffari S, et al. 2015. Pregnancy outcomes in women with inflammatory bowel disease following exposure to thiopurines and antitumor necrosis factor drugs: A systematic review with meta-analysis. *Human and Experimental Toxicology*, 34(5), 445-59. http://dx.doi.org/10.1177/0960327114550882
- Toussi S, et al. 2013. Infections in children and adolescents with juvenile idiopathic arthritis and inflammatory bowel disease treated with tumor necrosis factor-alpha inhibitors: Systematic review of the literature. *Clinical Infectious Diseases*, 1318-30 http://dx.doi.org/10.1093/cid/cit489
- Wang Y, et al. 2016. Oral 5-aminosalicylic acid for induction of remission in ulcerative colitis. *Cochrane Database of Systematic Reviews*, 4, CD000543. http://dx.doi.org/10.1002/14651858.CD000543.pub4
- Wang Y, et al. 2016. Oral 5-aminosalicylic acid for maintenance of remission in ulcerative colitis. *Cochrane Database of Systematic Reviews*, 5, CD000544. http://dx.doi.org/10.1002/14651858.CD000544.pub4
- Wedlake L, et al. 2014. Fiber in the treatment and maintenance of inflammatory bowel disease: a systematic review of randomized controlled trials. *Inflammatory Bowel Diseases*, 20(3), 576-86 http://dx.doi.org/10.1097/01.MIB.0000437984.92565.31
- Williams C, et al. 2014. Systematic review with meta-analysis: malignancies with anti-tumour necrosis factor-alpha therapy in inflammatory bowel disease. *Alimentary Pharmacology and Therapeutics*, 39, 447-58 http://dx.doi.org/10.1111/apt.12624

Websites

- Ministry of Health. 2015. Inflammatory bowel disease http://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/inflammatory-bowel-disease

- Health Navigator New Zealand. 2015. Inflammatory bowel disease video and information about Crohn's disease http://www.healthnavigator.org.nz/videos/i/inflammatory-bowel-disease/ http://www.healthnavigator.org.nz/health-a-z/c/crohns-disease/

- Crohn's & Colitis New Zealand http://crohnsandcolitis.org.nz/ includes a teenager's guide to living with IBD.

References

1. Ministry of Health. 2015. Inflammatory bowel disease. http://www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/inflammatory-bowel-disease accessed September, 2016
2. National Institute for Health and Care Excellence. 2015. Inflammatory bowel disease NICE quality standard [QS81]. https://www.nice.org.uk/guidance/qs81 accessed September, 2016
3. Day A, Paediatric Gastroenterology Clinical Network. 2014. Management of inflammatory bowel disease in children and adolescents in New Zealand: A clinical guideline. Paediatric Society New Zealand; National Child and Youth Clinical Networks. https://www.starship.org.nz/media/256562/nz_ibd_clinical_guideline_aug_2015.pdf
4. DeFilippis E, Sockolow R, Barfield E. 2016. Health care maintenance for the pediatric patient with inflammatory bowel disease. *Pediatrics,* 138(3) e20151971. http://dx.doi.org/10.1542/peds.2015-1971
5. Mozaffari S, Abdolghaffari A, Nikfar S, et al. 2015. Pregnancy outcomes in women with inflammatory bowel disease following exposure to thiopurines and antitumor necrosis factor drugs: A systematic review with meta-analysis. *Human and Experimental Toxicology,* 34(5) 445-59. http://dx.doi.org/10.1177/0960327114550882
6. Nguyen GC, Seow CH, Maxwell C, et al. 2016. The Toronto consensus statements for the management of inflammatory bowel disease in pregnancy. *Gastroenterology,* 150(3) 734-57.e1. http://dx.doi.org/10.1053/j.gastro.2015.12.003

CONSTIPATION

Introduction

Constipation is common in young children, a frequent cause of referral secondary care, and can become chronic in more than one-third of those affected. The most common form is idiopathic constipation, where there is no anatomical or physiological explanation for the symptoms.[1] Children and young people with Down syndrome, autism or cerebral palsy are particularly prone to idiopathic constipation and there is also a higher prevalence in children and young people in local authority care.[1] Severe constipation can be associated with faecal soiling which can have a significant emotional impact on children and young people and be stressful for parents and carers; prolonged support may be required to address social, psychological and educational consequences.[1,2]

The following section reviews severe constipation in children and young people using information from the National Minimum Dataset. The sections conclude with an overview of evidence for good practice for these conditions.

Data sources and methods

Indicators

> *Rates of constipation among 0–24 year olds*

Definition

Hospitalisations of 0–24 year olds with constipation per 100,000 population

Data sources

Numerator: National Minimum Dataset

Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

This section presents analyses where the condition was the primary diagnosis or was documented within any of the first 15 diagnoses (all cases). The rationale for presenting all cases is to highlight the full spectrum of health issues experienced by those with this condition, and their consequent requirement for acute health services. Analyses are per hospital discharge event therefore events are only included if the condition is documented within either the primary diagnosis or within any of the first 15 diagnoses.
Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

The number of 0–24 year olds hospitalised between 2011 and 2015 with any diagnosis of constipation is presented together with the total number of hospitalisations with constipation as a primary or any diagnosis. Constipation was the primary diagnosis in half of all such hospitalisations for 0–14 year olds and in one-quarter for 15–24 year olds (**Table 41**).

Hospitalisation rates for constipation rose in all age groups from 2000 to 2014, with a fall in rates for 0–4 year olds from 2014 to 2015. The rise in hospitalisation rates was most marked when all cases were included, particularly for 15–24 year olds. Hospitalisation rates were consistently highest for 0–4 year olds until 2014 (**Figure 55**). Similar patterns over time were seen in all ethnic groups, with rates for European/Other tending to have the highest rates and Asian/Indian tending to have the lowest (**Figure 56**).

Table 41. 0–24 year olds hospitalised with constipation New Zealand 2011–2015

Age group	Unique individuals *(n)*	Hospitalisations *(n)*		Ratio All : Primary
		Primary diagnosis	All cases	
Constipation				
Hospitalisation				
0–24 years	14,578	7,805	18,723	2.40
0–14 years	8,541	5,948	11,247	1.89
15–24 years	6,108	1,857	7,476	4.03

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations where constipation was listed in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Figure 55. Hospitalisations for constipation, 0–24 year olds, by age group, New Zealand 2000–2015
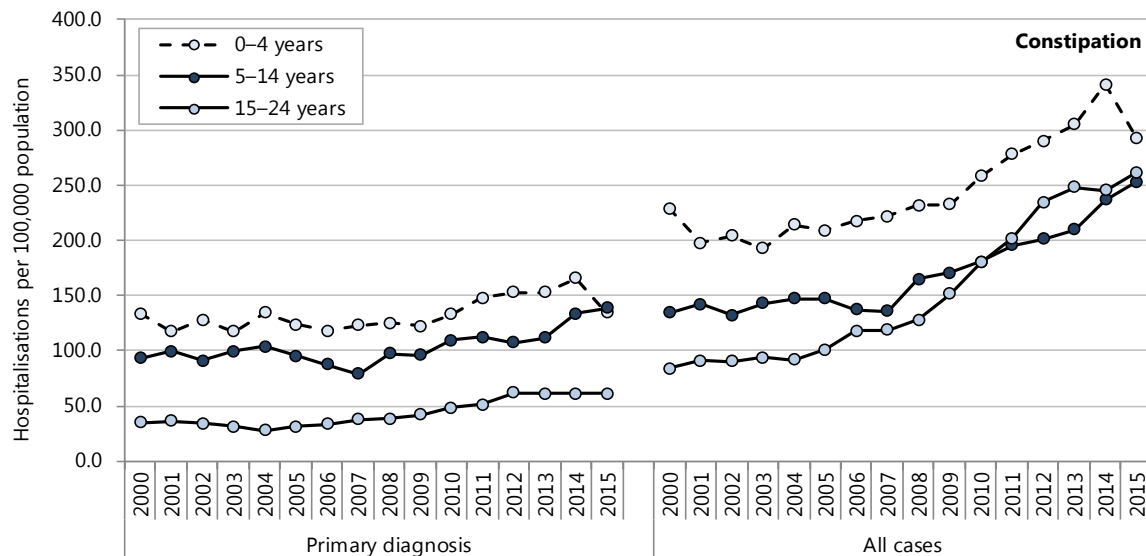
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with constipation listed in any of the first 15 diagnoses; Rate per 100,000 age-specific population

Figure 56. Hospitalisations involving constipation in 0–24 year olds, by ethnicity, New Zealand 2000–2015

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. All cases corresponds to hospitalisations with constipation included in any of the first 15 diagnoses; Rate per 100,000 age-specific population

Fewer than half of the hospitalisations of 0–24 year olds with constipation had this condition as the primary diagnosis (**Table 41**). Constipation was included within the first 15 diagnoses in hospitalisations for a variety of conditions including intestinal and digestive disorders, pregnancy, childbirth and the puerperium, diseases of the genitourinary and respiratory systems and injury and poisoning (**Table 42**).

Table 42. Hospitalisations involving constipation in 0–24 year olds, by primary diagnosis, New Zealand 2011–2015

Primary diagnosis	2011–2015 (n)	Annual average	Rate per 100,000 0–24 year olds	95% CI	%
Constipation* in 0–24 year olds					
New Zealand					
Constipation	7,805	1,561	101.62	99.39–103.89	41.7
Other functional intestinal disorders	22	4	0.29	0.19–0.43	0.1
Paralytic ileus and intestinal obstruction without hernia	75	15	0.98	0.78–1.22	0.4
Fissure and fistula of anal and rectal regions	46	9	0.60	0.45–0.80	0.2
Irritable bowel syndrome	24	5	0.31	0.21–0.46	0.1
Other intestinal diseases	55	11	0.72	0.55–0.93	0.3
Total intestinal diseases	8,027	1,605	104.51	102.25–106.82	42.9
Inflammatory bowel disease	52	10	0.68	0.52–0.89	0.3
Other diseases of the digestive system	573	115	7.46	6.87–8.10	3.1
Symptoms and/or abnormal clinical findings NEC	1,916	383	24.94	23.85–26.09	10.2
Pregnancy, childbirth and the puerperium	1,162	232	15.13	14.28–16.02	6.2
Diseases of the genitourinary system	971	194	12.64	11.87–13.46	5.2
Diseases of the respiratory system	909	182	11.83	11.09–12.63	4.9
Injury and/or poisoning	897	179	11.68	10.94–12.47	4.8
Other diagnoses	4,216	843	54.89	53.26–56.57	22.5
Total	18,723	3,745	243.76	240.30–247.27	100.0

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Constipation in any of the first 15 diagnoses; NEC = not elsewhere classified

Demographic distribution

Table 43 presents the demographic distribution of individuals with constipation in New Zealand between 2011 and 2015. There was a slight but statistically significant social gradient with increasing prevalence of constipation requiring hospitalisation for individuals living at each increasing NZDep2013 quintile, compared with individuals living in areas with lower deprivation scores (NZDep2013 deciles 1–2). Prevalence of constipation requiring hospitalisation was significantly higher for 0–4 year olds and lower for 5–14 year olds, compared to 15–24 year olds. Compared to rates for European/Other 0–24 year olds, Māori and Pacific rates were not significantly different while Asian/Indian rates were significantly lower and MELAA rates significantly higher.

Table 43. 0–24 year olds hospitalised for constipation, by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Constipation* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	2,174	153.19	1.00	
Deciles 3–4	2,220	166.03	1.08	1.02–1.15
Deciles 5–6	2,588	179.54	1.17	1.11–1.24
Deciles 7–8	3,420	210.52	1.37	1.30–1.45
Deciles 9–10	4,537	244.18	1.59	1.51–1.68
Prioritised ethnicity				
Māori	3,455	191.55	0.96	0.92–1.00
Pacific	1,447	204.19	1.02	0.96–1.08
Asian/Indian	1,136	118.51	0.59	0.56–0.63
MELAA	318	315.32	1.58	1.41–1.76
European/Other	8,223	200.14	1.00	
Gender				
Female	8,745	232.92	1.00	
Male	5,833	148.56	0.64	0.62–0.66
Age group (years)				
0–4	3,746	240.17	1.23	1.18–1.28
5–14	4,926	165.07	0.85	0.82–0.88
15–24	6,108	194.71	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Constipation in any of the first 15 diagnoses; Rate ratios are unadjusted; Ethnicity is level 1 prioritised; Decile is NZDep2013

Regional distribution

Table 44 presents the number of individuals resident in each district health board area that had a constipation diagnosis between 2011 and 2015. It also presents the number of hospital discharges in which constipation was documented as the primary diagnosis or any diagnosis. The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and result in less undercounting than would occur if only the primary diagnosis is included in discharge data. For constipation the national All:Primary diagnosis ratio was greater than two, with similar ratios in all DHBs. The undercount of constipation hospitalisations would be two–three fold if only primary diagnoses were included (**Table 44**).

Table 44. Hospitalisations for constipation in 0–24 year olds, by district health board, New Zealand 2010–2014

DHB	Unique individuals *(n)*	Hospitalisations *(n)*		Ratio All : Primary
		Primary diagnosis	All cases	
Constipation in 0–24 year olds				
Northland	627	283	813	2.87
Waitemata	1,649	886	2,073	2.34
Auckland	1,544	745	1,859	2.50
Counties Manukau	1,961	824	2,338	2.84
Waikato	1,424	934	1,940	2.08
Bay of Plenty	644	357	796	2.23
Lakes	461	161	558	3.47
Tairawhiti	209	168	295	1.76
Taranaki	275	174	353	2.03
Hawke's Bay	438	328	598	1.82
MidCentral	489	259	590	2.28
Whanganui	191	139	249	1.79
Hutt Valley	511	288	661	2.30
Capital & Coast	926	381	1,146	3.01
Wairarapa	119	59	162	2.75
Nelson Marlborough	303	208	415	2.00
South Canterbury	142	71	187	2.63
Canterbury	1,574	701	2,010	2.87
West Coast	80	39	98	2.51
Southern	1,109	778	1,504	1.93
New Zealand	14,578	7,805	18,723	2.40

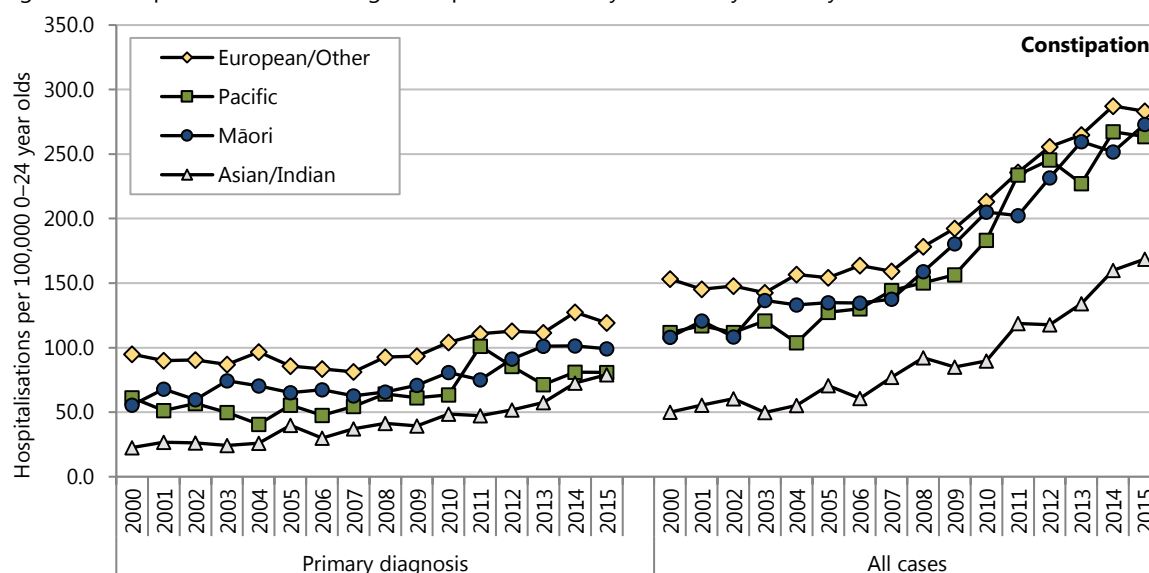Numerator: National Minimum Dataset, 'All cases' corresponds to hospitalisations with constipation listed in any of the first 15 diagnoses; Note that individuals may appear in multiple DHBs

Evidence for good practice

Possibilities for prevention

The exact cause of idiopathic constipation is not fully understood; contributing factors may include pain, fever, dehydration, dietary and fluid intake, psychological issues, toilet training, medicines and family history of constipation.[1] If simple measures such as increasing fluid intake, increasing fruit and vegetables in the child's diet, encouraging a regular toileting habit and encouraging more exercise are not effective then early assessment by a health professional is important.[3]

Evidence-based health care for children and young people with constipation

Conflicting advice and inconsistent practice make treatment of constipation potentially less effective and frustrating for children and their families. Consistent messages from the variety of healthcare professionals from whom children and young people with idiopathic constipation seek help is important. This will help to reduce rates of ED presentation and unplanned hospitalisation for constipation, reduce rates of recurrent constipation and/or impaction in children and young people, increase parent or carer satisfaction with information and advice, and enable children and young people to satisfactorily manage constipation.[1]

The following national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of constipation are provided for further reading.

New Zealand guidelines
- Starship Children's Health. 2012. Starship clinical guidelines: Constipation. https://www.starship.org.nz/for-health-professionals/starship-clinical-guidelines/c/constipation/

Evidence-based medicine reviews
- Gordon M, et al. 2016. Osmotic and stimulant laxatives for the management of childhood constipation. Cochrane Database of Systematic Reviews. http://dx.doi.org/10.1002/14651858.CD009118.pub3

- Lu ML, et al. 2015. Electrical stimulation therapy for slow transit constipation in children: A systematic review. *International Journal of Colorectal Disease*, 30(5), 697-702. http://dx.doi.org/10.1007/s00384-015-2180-3
- Siminas S & Losty PD. 2015. Current surgical management of pediatric idiopathic constipation: A systematic review of published studies. *Annals of Surgery*, 262(6), 925-33. http://dx.doi.org/10.1097/sla.0000000000001191

Other relevant publications
- Sbahi H & Cash BD. 2015. Chronic constipation: A review of current literature. *Current Gastroenterology Reports*, 17(12), 1-13. http://dx.doi.org/10.1007/s11894-015-0471-z
- Vandenplas Y, et al. 2015. Prevalence and health outcomes of functional gastrointestinal symptoms in infants from birth to 12 months of age. *Journal of Pediatric Gastroenterology and Nutrition*, 61(5), 531-37. http://dx.doi.org/10.1097/MPG.0000000000000949
- Wald A. 2016. Constipation: Advances in diagnosis and treatment. *JAMA*, 315(2), 185-91. http://dx.doi.org/10.1001/jama.2015.16994

Websites
- Kidshealth. Constipation. http://www.kidshealth.org.nz/constipation

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2. Duncanson M, Oben G, Wicken A, et al. 2016. Constipation: A commonly costly complex condition. Paediatric Society of New Zealand 68th Annual Scientific Meeting - The science of healing, The art of medicine. Bethlehem College, Tauranga https://ourarchive.otago.ac.nz/handle/10523/6933
3. Kidshealth. Constipation. http://www.kidshealth.org.nz/constipation accessed September, 2016.

ECZEMA AND DERMATITIS

Introduction

Eczema is a chronic inflammatory skin condition, usually beginning in infancy or early childhood, that affects over 20 percent of children in New Zealand. It is characterised by itch, scratching and inflamed, dry, scaling and crusted areas of skin. Eczema is also known as atopic eczema or atopic dermatitis.[1] Eczema is typically an episodic condition with flares and remissions, although in some children it is continuous.[1-3] Eczema and its treatment can negatively affect quality of life for children and their families to an extent similar to that of other chronic conditions such as asthma or diabetes. Children with eczema can experience disturbed sleep and fatigue, restriction of activities, school absenteeism, bullying, poor self-esteem and poor peer relationships. Children with eczema have high rates of psychological problems compared with their peers. Direct medical, hospital and treatment costs and indirect costs such as time off work for caregivers can be substantial.[4]

There is a strong genetic component to eczema, and affected children often go on to develop co-morbidities of asthma and allergic rhinitis. The dry cracked skin that occurs in eczema can provide a portal of entry for bacterial and viral skin infections.[3] Children and adolescents with eczema have an increased risk for attention deficit hyperactivity disorder, possibly affected by sleeping problems, and higher prevalence rates of depression, anxiety, conduct disorder, and autism compared with their peers.[5] There is emerging evidence that eczema may be associated with an increased risk of rheumatoid arthritis and inflammatory bowel disease, and decreased risk of type 1 diabetes.[6]

The following section reviews eczema and other conditions of the skin and subcutaneous tissue in children and young people using information from the New Zealand Health Survey and National Minimum Dataset. The section concludes with a brief overview of evidence for good practice for these conditions.

Data sources and methods

Indicators

Prevalence of eczema

Rates of eczema and dermatitis among 0–24 year olds

Definition

Prevalence of eczema

Eczema diagnosed by doctor and using medicines, tablets, pills, cream or ointment (0–14 years); Child respondents (aged 0–14 years) are defined as having eczema if the child's parents or caregivers had ever been told by a doctor that the child has eczema and if they now have treatments for eczema (cream, ointment, medicine, tablets or pills)

Rates of eczema and dermatitis among 0–24 year olds

Hospitalisations of 0–24 year olds with eczema and dermatitis per 100,000 population

Data sources

Prevalence of eczema

New Zealand Health Survey (2006/07–2014/15), see **Appendix 3**

Rates of eczema and dermatitis among 0–24 year olds

Numerator:	National Minimum Dataset
Denominator:	Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

Prevalence of eczema

The NZ Health Survey utilised adjusted rate ratios to account for the potential influence of other demographic factors when undertaking demographic comparisons. Gender comparisons are adjusted for age, ethnic comparisons are adjusted for age and gender, and deprivation comparisons are adjusted for age, sex and ethnicity.[7]

Rates of eczema and dermatitis among 0–24 year olds

This section presents analyses where the condition was the primary diagnosis or was documented within any of the first 15 diagnoses (all cases). The rationale for presenting all cases is to highlight the full spectrum of health issues experienced by those with this condition, and their consequent requirement for acute health services. Analyses are per hospital discharge event therefore events are only included if the condition is documented within either the primary diagnosis or within any of the first 15 diagnoses. Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

From 2009 to 2013 there were less than five deaths of 0–24 year olds with a skin condition as an underlying cause, as documented within the National Mortality Collection.

Children aged 0–4 had the greatest prevalence of eczema that required medication with male children having a higher prevalence than females. Female children aged 5–9 and 10–14 had a higher incidence of eczema than male children of the same age (**Figure 57**). The prevalence of children aged 0–14 with eczema reduced with age. Children of Pacific ethnicity had the highest prevalence of eczema, followed by children of Māori, Asian or European/Other ethnicities. There was not a marked difference between the NZDep index deciles, however children living in the most deprived areas did have the highest incidence of eczema (**Figure 58; Figure 59**). There has been some year to year variation amongst the sexes (**Figure 60**).

Figure 57. Eczema (medicated) in 0–14 year olds, by age group and sex, NZ Health Survey 2014/15
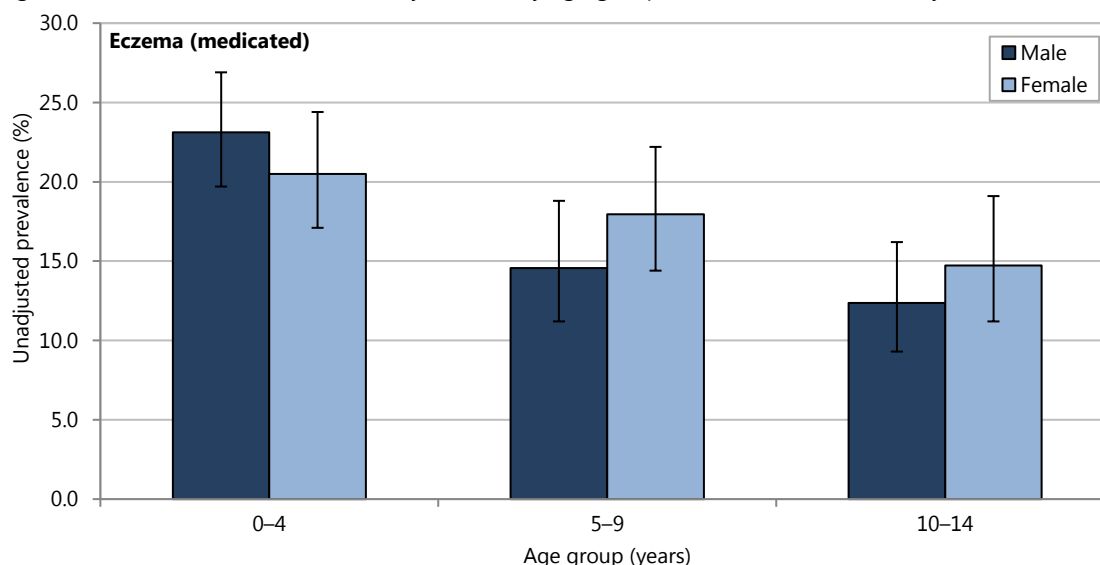
Source: NZ Health Survey

Figure 58. Eczema (medicated) in 0–14 year olds, by demographic factor, NZ Health Survey 2014/15
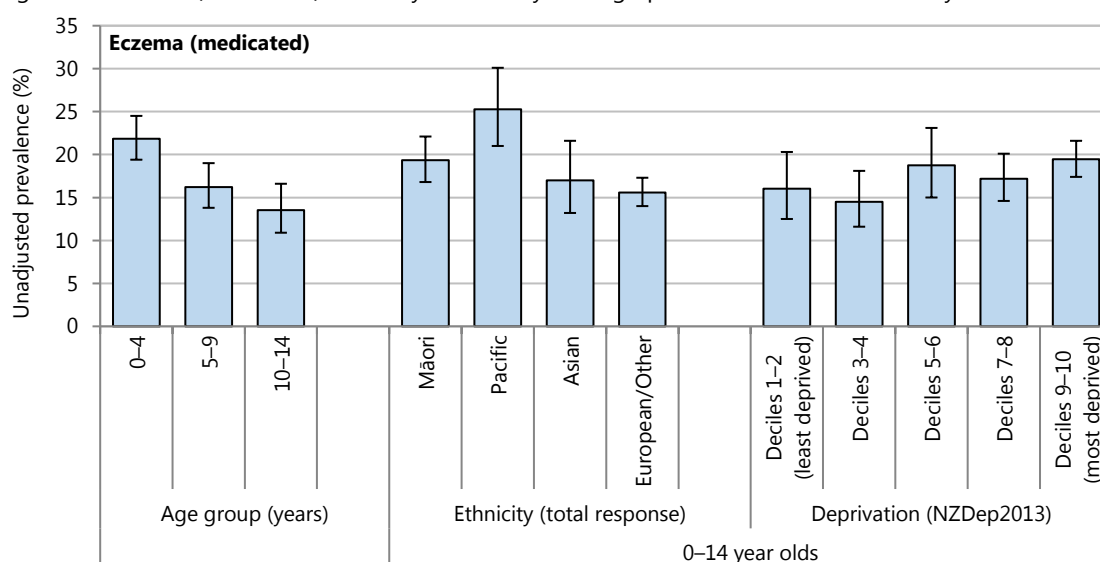
Source: NZ Health Survey

Figure 59. Comparisons for 0–14 year olds diagnosed with eczema, by demographic factor, NZ Health Survey 2014/15
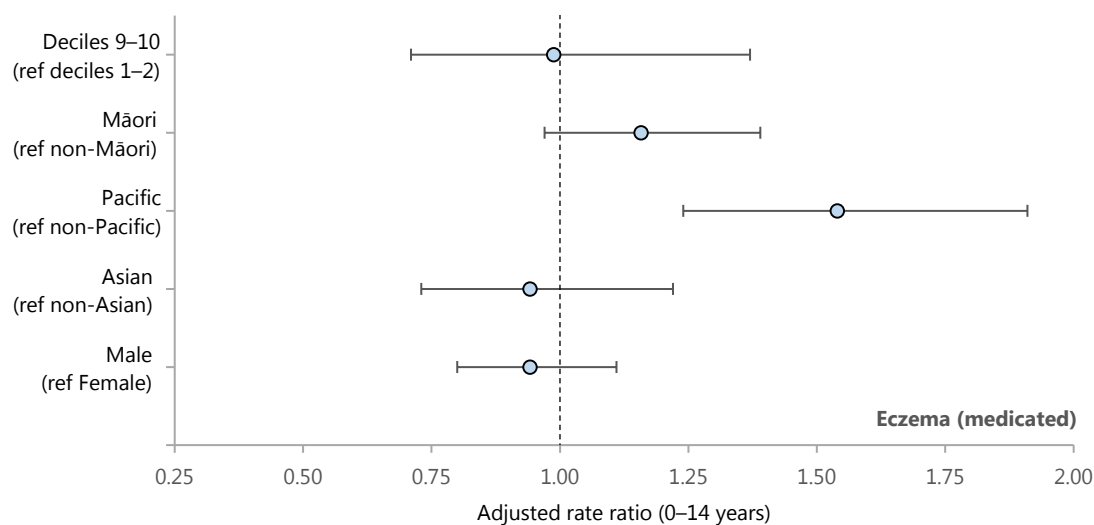
Source: NZ Health Survey

Figure 60. Eczema (medicated) in 0–14 year olds, by sex and survey year, NZ Health Surveys 2006/07–2014/15
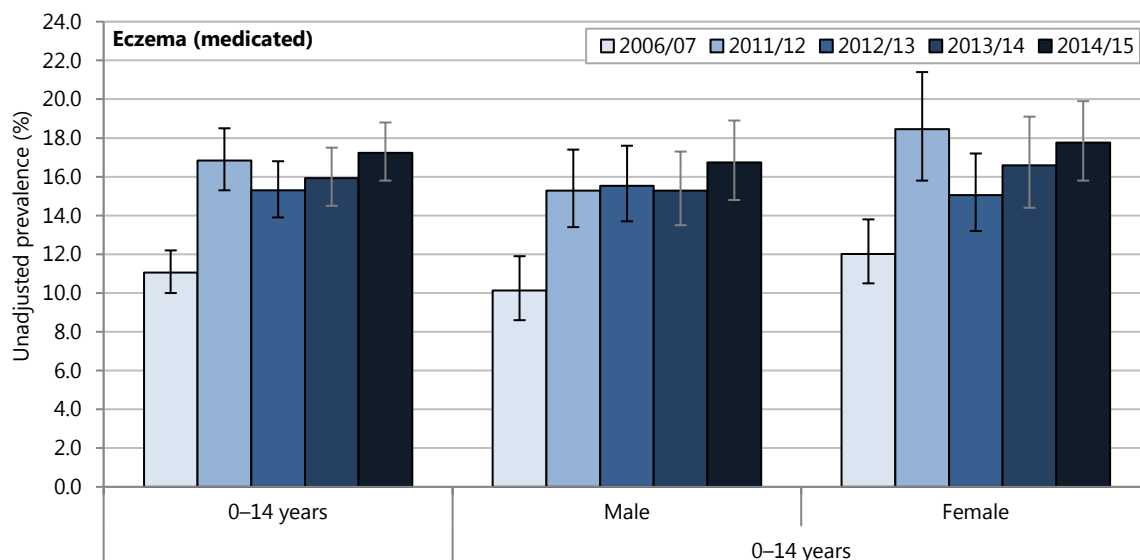
Source: NZ Health Survey

The number of 0–24 year olds hospitalised between 2011 and 2015 with any diagnosis of eczema and dermatitis is presented by age group in **Table 45**, together with the total number of hospitalisations with eczema and dermatitis as a primary or any diagnosis. These diagnoses were characterised by high All:Primary ratios in 0–14 and 15–24 year olds which means that hospitalisations occurred more often when the primary diagnosis was a condition other than eczema and dermatitis. Surveillance using primary diagnoses only for these conditions would lead to a large undercount of hospitalisations.

Only about a quarter of hospitalisations of 0–24 year olds with eczema and dermatitis had these conditions as the primary diagnosis and the specific types of dermatitis are listed in **Table 46**. Other common primary diagnoses included respiratory and infectious diseases (**Table 46**).

Table 45. Individuals hospitalised with eczema and dermatitis by age group, 0–24 year olds New Zealand 2011– 2015

Age group	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Eczema and dermatitis*				
0–24 years	15,331	5,022	18,950	3.77
0–14 years	11,949	4,153	15,065	3.63
15–24 years	3,412	869	3,885	4.47

Source: National Minimum Dataset. All cases' corresponds to hospitalisations with eczema or dermatitis included in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Table 46. Hospitalisations involving eczema and dermatitis in 0–24 year olds, by primary diagnosis, New Zealand 2011–2015

Primary diagnosis	2011–2015 (n)	Annual average	Rate per 100,000 0–24 year olds	95% CI	%
Eczema and dermatitis* in 0–24 year olds					
New Zealand					
Dermatitis due to substances taken internally	480	96	6.25	5.71–6.83	2.5
Atopic dermatitis	279	56	3.63	3.23–4.08	1.5
Diaper [napkin] dermatitis	144	29	1.87	1.59–2.21	0.8
Allergic contact dermatitis	143	29	1.86	1.58–2.19	0.8
Seborrhoeic dermatitis	118	24	1.54	1.28–1.84	0.6
Herpesviral infections†	389	78	5.06	4.59–5.59	2.1
Other dermatitis‡	3,469	694	45.16	43.69–46.69	18.3
Eczema and dermatitis total	5,022	1,004	65.38	63.60–67.22	26.5
Other skin and subcutaneous tissue diseases	1,126	225	14.66	13.83–15.54	5.9
Infectious and parasitic diseases§	1,441	288	18.76	17.82–19.75	7.6
Diseases of the respiratory system	3,623	725	47.17	45.66–48.73	19.1
Certain conditions originating in the perinatal period	1,467	293	19.10	18.15–20.10	7.7
Injury and/or poisoning	1,148	230	14.95	14.11–15.84	6.1
Symptoms and/or abnormal clinical findings NEC	1,082	216	14.09	13.27–14.95	5.7
Other diagnoses	4,041	808	52.61	51.01–54.26	21.3
Total	18,950	3,790	246.72	243.23–250.25	100.0

Numerator: National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population; * Eczema or dermatitis in any of the first 15 diagnoses; † Herpes viral infections encompasses: Eczema herpeticum, Herpesviral vesicular dermatitis; ‡Other dermatitis includes: Irritant contact dermatitis, Unspecified contact dermatitis, Exfoliative dermatitis, Lichen simplex chronicus and prurigo, Pruritus; § Infectious and parasitic diseases excludes herpesviral infections; NEC = not elsewhere classified

Since 2000 hospitalisation rates for eczema and dermatitis have risen markedly for 0–4 year olds and slightly for 5–14 and 15–24 year olds. The highest hospitalisation rates were for 0–4 years olds when all cases were included (**Figure 61**). Similar increases in hospitalisation rates over time were seen in all ethnic groups. Hospitalisation rates for 0–24 year olds were consistently higher for Pacific and Māori 0–24 year olds compared with their European and Asian/Indian peers. This ethnic disparity was most marked when all cases were included in the analysis (**Table 47**).

Figure 61. Hospitalisations for eczema and dermatitis in 0–24 year olds, by age group, New Zealand 2000–2015
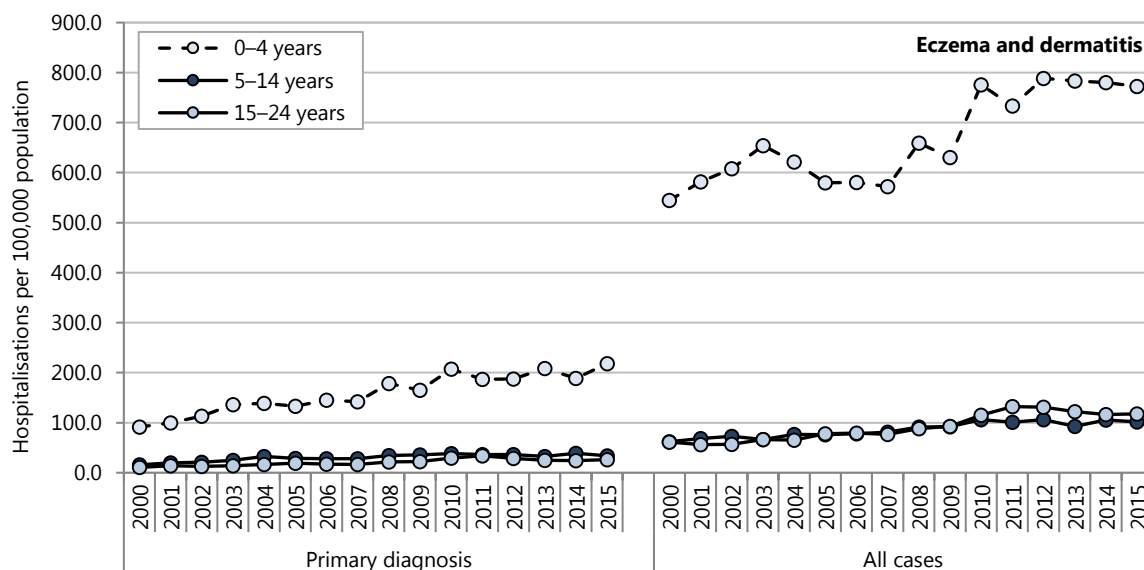
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with eczema or dermatitis in any of the first 15 diagnoses

Table 47 presents the demographic distribution of individuals hospitalised with eczema and dermatitis in New Zealand between 2011 and 2015. There was a strong social gradient among these individuals, with statistically significant increases in prevalence in each NZDep 2013 quintile (NZDep2013 deciles 3–4 to 9–10) compared with those living in areas with the lowest NZDep2013 deprivation scores (deciles 1–2). Prevalence rates were significantly higher for Māori, Pacific, Asian/Indian and MELAA 0–24 year olds than for European/Other. The majority of 0–24 year olds hospitalised with eczema and dermatitis were 0–4 year olds.

Table 47. 0–24 year olds hospitalised for eczema and dermatitis, by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Eczema and dermatitis* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	1,606	113.17	1.00	
Deciles 3–4	1,876	140.30	1.24	1.16–1.33
Deciles 5–6	2,343	162.54	1.44	1.35–1.53
Deciles 7–8	3,617	222.65	1.97	1.86–2.09
Deciles 9–10	6,234	335.51	2.96	2.81–3.13
Prioritised ethnicity				
Māori	5,197	288.12	2.07	2.00–2.15
Pacific	2,800	395.12	2.84	2.72–2.97
Asian/Indian	1,421	148.24	1.07	1.01–1.13
MELAA	199	197.32	1.42	1.23–1.63
European/Other	5,714	139.07	1.00	
Gender				
Female	7,643	203.57	1.00	
Male	7,687	195.78	0.96	0.93–0.99
Age group (years)				
0–4	9,501	609.16	5.60	5.39–5.82
5–14	2,531	84.81	0.78	0.74–0.82
15–24	3,412	108.76	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population; * Eczema or dermatitis in any of the first 15 diagnoses; Rate ratios are unadjusted; Ethnicity is level 1 prioritised; Decile is NZDep2013

Regional distribution

Table 48 presents the number of individuals resident in each district health board area that had one or more hospitalisations with a diagnosis of eczema and dermatitis between 2011 and 2015. The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and result in less undercounting than would occur if only the primary diagnosis is included in discharge data. For eczema and dermatitis the All:Primary diagnosis ratio was higher than two in all DHBs, and particularly high in West Coast, Waikato, Canterbury, Otago and Lakes DHBs. The All:Primary diagnosis ratio was considerably lower than the national ratio in Hawke's Bay, Whanganui, MidCentral and South Canterbury DHBs and similar to the national ratio in the remaining district health boards.

Table 48. Hospitalisations for eczema and dermatitis in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Eczema and dermatitis in 0–24 year olds				
Northland	782	239	977	4.09
Waitemata	1,567	505	1,909	3.78
Auckland	1,613	493	1,926	3.91
Counties Manukau	2,443	747	2,937	3.93
Waikato	1,342	316	1,606	5.08
Bay of Plenty	845	309	1,035	3.35
Lakes	616	177	798	4.51
Tairawhiti	270	120	382	3.18
Taranaki	201	87	266	3.06
Hawke's Bay	351	198	450	2.27
MidCentral	477	200	575	2.88
Whanganui	212	105	272	2.59
Hutt Valley	610	263	789	3.00
Capital & Coast	1,049	409	1,287	3.15
Wairarapa	129	51	161	3.16
Nelson Marlborough	253	81	311	3.84
South Canterbury	115	49	146	2.98
Canterbury	1,598	393	1,865	4.75
West Coast	80	6	92	15.33
Southern	903	264	1,100	4.17
New Zealand	15,331	5,022	18,950	3.77

Source: National Minimum Dataset; 'All cases' corresponds to hospitalisations with eczema or dermatitis listed in any of the first 15 diagnoses; Sum of DHBs may equal more than NZ total due to some individuals changing DHB of domicile during the period

Evidence for good practice

Possibilities for prevention

No primary prevention strategy has been established for eczema and there is no clear evidence for effectiveness of measures such as maternal dietary antigen avoidance during pregnancy and breastfeeding, long-term breastfeeding, hydrolysed protein formulas, soy formulas, omega-3 or omega-6 fatty acid supplementation or delayed introduction of solid foods.[5] There is some emerging but weak evidence about possible preventive effects of probiotics administered in pregnancy where there is a high risk of eczema in the fetus.[8,9] Prevention approaches aiming to enhance skin barrier function, such as daily full-body emollient therapy from birth may reduce the cumulative incidence of eczema in infants.[5] Secondary prevention through prevention and control of flares is a key goal of evidence-based management of eczema.[3]

Evidence-based health care for children and young people with eczema

Topical corticosteroids are the mainstay of the management of active eczema in combination with the regular use of emollients, the management of triggers and the treatment of concurrent infection. It is important for all healthcare professionals to be aware of the robust safety profile of topical corticosteroids so as to facilitate optimal treatment and counteract prevalent misinformation which raises unwarranted concerns about adverse effects.[4] Treatment for eczema should be tailored to the individual child, with treatments stepped up and down according to the recorded severity of symptoms.[2,3]

Eczema can usually be managed in primary care.[10] Specialist referral is recommended if: symptoms are severe and persist despite optimum topical treatment, flares are occurring frequently or eczema is associated with repeated infections, there are serious social or psychological problems for the child or caregiver or if a child has severe food allergy or fails to grow at the expected growth trajectory.[2,3] Engagement with a multidisciplinary team and nurse-led clinics may improve outcomes for children with eczema and their families.[11]

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Other relevant publications
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MUSCULOSKELETAL DISORDERS

Introduction

Musculoskeletal disorders are common in children and often debilitating.[1] This section includes juvenile idiopathic arthritis, juvenile osteochondroses and scoliosis.

Juvenile idiopathic arthritis (JIA) is the most common paediatric rheumatologic disease.[2] The term JIA describes all forms of arthritis that have an onset before age 16 years with joint inflammation that lasts more than 6 weeks and exclusion of other known conditions.[2-4] Children with specific subtypes of JIA can have additional symptoms or conditions including: fever, tiredness, rash, loss of appetite and weight loss. The arthritis that occurs as a manifestation of the skin condition psoriasis is also included as a subtype of JIA.[4] JIA has significant morbidity and can be painful, disable a child, limit quality of life, add to the financial stress of families, and have a substantial societal cost.[1,5] Children with arthritis that involves five or more joints have a particularly refractory disease course and are at higher risk of poorer functional outcomes compared to those with involvement of fewer joints.[2,6] Inadequately controlled disease may lead to abnormalities of growth such as short stature, localised bone overgrowth or premature fusion, and alteration of limb length.[3] Patients with JIA are less likely than healthy peers to engage in physical activity.[7] Important differential diagnoses include rheumatic fever, systemic lupus erythematosus, and orthopaedic problems. Some children with inflammatory bowel disease (IBD) may have a peripheral arthritis before or after the bowel problems become obvious.[1] Fatigue and uveitis are common comorbidities; other comorbidities include allergic rhinitis, migraine and atopic dermatitis and heart problems such as pericarditis.[8-10]

The term juvenile osteochondrosis describes a group of disorders that affect patients with an immature skeleton. The usual presentation is pain and limited mobility of the affected joint.[5] Joints commonly affected are the hip, knee, elbow and back. Osteochondrosis occurs as a result of abnormal growth, injury, or overuse of the developing bone growth plate.[5] Symptoms may be intermittent, and may be associated with athletic activity.[11] The association of symptoms with athletic activity means that osteochondrosis is sometimes classified as sports-related overuse injury.[12] Osteochondrosis at different body sites has traditionally been considered as separate diseases, often with eponymous titles (e.g. osteochondrosis of the knee is known as Osgood-Schlatter disease) although the disease process is similar wherever it occurs in the body.[5,11,12]

Scoliosis is a lateral deviation of the spine and is one of the most common paediatric spinal deformities. Most cases, especially if diagnosed after infancy, are 'idiopathic' or not associated with any known cause. Congenital scoliosis has a high association with other congenital anomalies arising at the same period of fetal development.[13] Juvenile scoliosis also occurs in the setting of neuromuscular disorders such as cerebral palsy and muscular dystrophy, or as part of genetic syndromes and conditions such as Marfan syndrome and neurofibromatosis.[13-15]

Data sources and methods

Indicators

> *Rates of chronic musculoskeletal diseases among 0–24 year olds*

Definition

Hospitalisations of 0–24 year olds with a chronic musculoskeletal disease per 100,000 population

Data sources

Numerator: National Minimum Dataset

Denominator: Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

Additional information

A chronic musculoskeletal disease was the primary diagnosis or was documented as one of the first 15 diagnoses. Chronic musculoskeletal diseases comprises juvenile arthritis, juvenile osteochondrosis, and scoliosis.
Codes used for identifying cases are documented in **Appendix 5**.

National trends and distribution

There were less than five deaths of 0–24 year olds where a chronic musculoskeletal disease was the underlying cause of death in New Zealand between 2009 and 2013, as documented within the National Mortality Collection.

The number of 0–24 year olds hospitalised with chronic musculoskeletal diseases during 2011 to 2015 is presented in **Table 49**. It also presents the number of hospital discharges in which chronic musculoskeletal diseases were documented as the primary diagnosis or as any diagnosis.

The rate of hospitalisations where a chronic musculoskeletal disease was the primary diagnosis has increased since 2000 (**Figure 62**).

Table 49. Individuals aged 0–24 years hospitalised with chronic musculoskeletal diseases using primary diagnosis compared to all cases, New Zealand 2011–2015

	Unique individuals *(n)*	Hospitalisations *(n)*		Ratio All:Primary
		Primary diagnosis	All cases	
Chronic musculoskeletal diseases				
Hospitalisation				
0–24 years	1,506	2,431	3,016	1.24
0–14 years	962	1,595	1,968	1.23
15–24 years	578	836	1,048	1.25
Chronic musculoskeletal diseases in 0–24 year olds				
Juvenile arthritis	377	1,269	1,494	1.18
Juvenile osteochondrosis	415	444	541	1.22
Scoliosis	720	718	982	1.37

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with chronic musculoskeletal diseases listed in any of the first 15 diagnoses; The sum of the age groups may total to more than the 0–24 year old total

Figure 62. Hospitalisations for chronic musculoskeletal diseases in 0–24 year olds, New Zealand 2000–2015
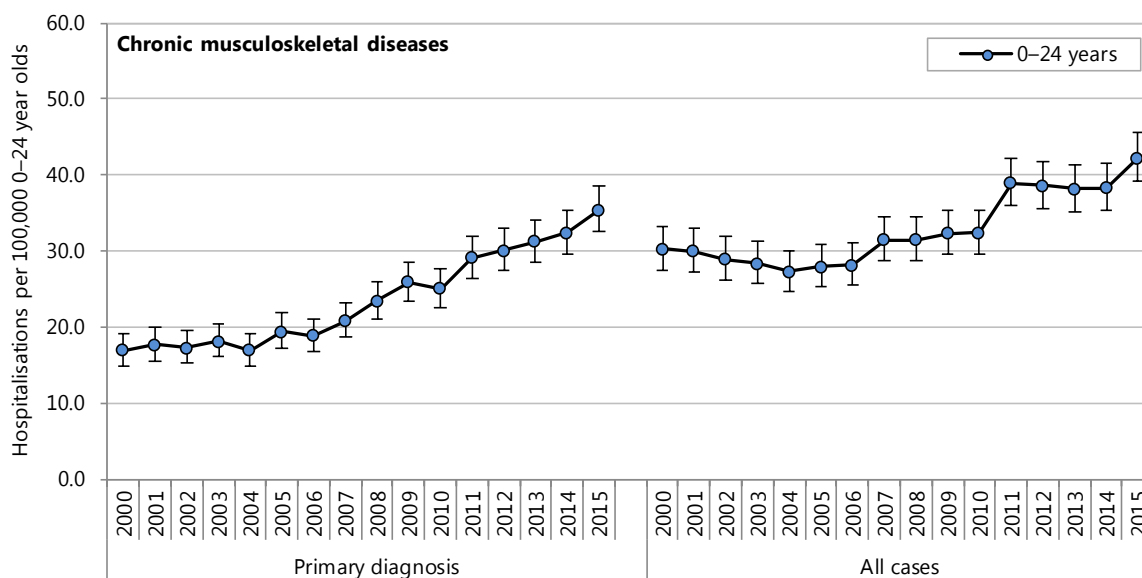
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with chronic musculoskeletal diseases listed in any of the first 15 diagnoses

Demographic distribution

Table 50 presents the demographic distribution of individuals with chronic musculoskeletal diseases in New Zealand between 2011 and 2015. Chronic musculoskeletal diseases were significantly lower among males, and among 0–4 year olds compared to 15–24 year olds, and significantly higher among 5–14 year olds. There was no significant difference by deprivation score. The majority of individuals with chronic musculoskeletal diseases were of European/Other ethnicities.

Table 50. Individuals aged 0–24 years hospitalised with chronic musculoskeletal diseases, by demographic factor, New Zealand 2011–2015

Variable	Unique individuals 2011–2015 (n)	Rate per 100,000 population	Rate ratio	95% CI
Chronic musculoskeletal diseases* in 0–24 year olds				
New Zealand				
NZ Deprivation Index quintile				
Deciles 1–2	308	21.70	1.00	
Deciles 3–4	286	21.39	0.99	0.84–1.16
Deciles 5–6	287	19.91	0.92	0.78–1.08
Deciles 7–8	315	19.39	0.89	0.76–1.05
Deciles 9–10	371	19.97	0.92	0.79–1.07
Prioritised ethnicity				
Māori	248	13.75	0.54	0.47–0.62
Pacific	95	13.41	0.53	0.43–0.65
Asian/Indian	89	9.28	0.36	0.29–0.45
MELAA	19	18.84	0.74	0.47–1.16
European/Other	1,049	25.53	1.00	
Gender				
Female	875	23.31	1.00	
Male	631	16.07	0.69	0.62–0.76
Age group (years)				
0–4	137	8.78	0.48	0.40–0.57
5–14	860	28.82	1.56	1.41–1.74
15–24	578	18.42	1.00	

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. * Chronic musculoskeletal diseases in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is Level 1 prioritised; Decile is NZDep2013; Summation of components may equal more than the 0–24 year old unique total

Hospitalisations for chronic musculoskeletal diseases had increased for the three age groups since 2000, and most notably for the 0–4 year olds (**Figure 63**). Over the same period, the hospitalisation rate had gradually increased for each ethnic group, although European/Other had a consistently higher hospitalisation rate than the other ethnic groups (**Figure 64**).

Figure 63. Hospitalisations involving chronic musculoskeletal diseases in 0–24 year olds, by age group, New Zealand 2000–2015
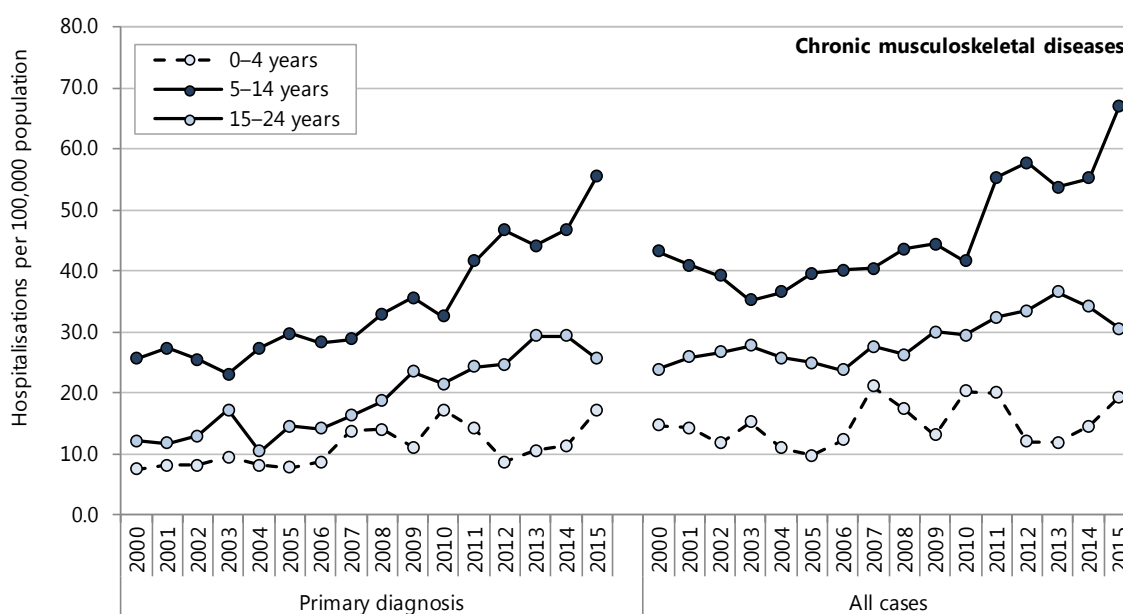
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with chronic musculoskeletal diseases listed in any of the first 15 diagnoses

Figure 64. Hospitalisations involving chronic musculoskeletal diseases in 0–24 year olds, by ethnicity, New Zealand 2000–2015
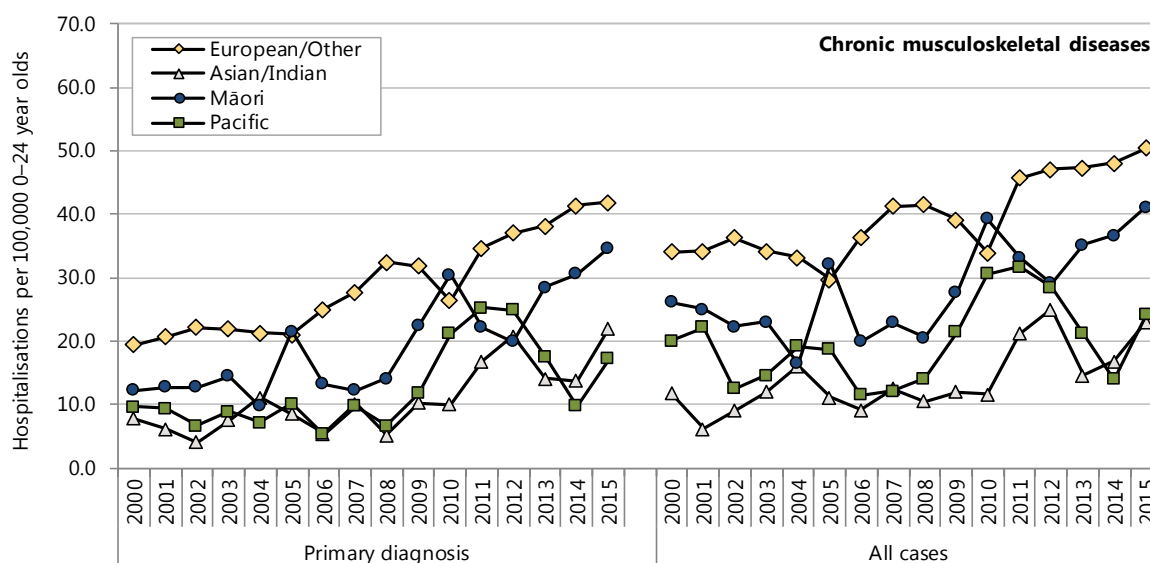
Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. 'All cases' corresponds to hospitalisations with chronic musculoskeletal diseases listed in any of the first 15 diagnoses

Regional distribution

Table 51 presents the number of individuals resident in each district health board that had a chronic musculoskeletal disease diagnosis during 2011 to 2015. It also presents the number of hospital discharges in which a chronic musculoskeletal disease was documented as the primary diagnosis or any diagnosis.

The All:Primary diagnosis ratio reflects the extent to which hospitalisations of 0–24 year olds with chronic conditions occur when this condition is not the primary diagnosis. A high ratio may be associated with more thorough documentation and avoid undercounting that would occur if only primary diagnoses are included in discharge data. For chronic musculoskeletal diseases the All:Primary diagnosis ratio was higher than the national ratio in Waikato, Bay of Plenty, Lakes, Hawke's Bay, MidCentral, Whanganui, Hutt Valley, Nelson Marlborough, and Southern DHBs. Ratios were lower than the national ratio in the remaining district health boards (**Table 51**).

Table 51. Hospitalisations for chronic musculoskeletal diseases in 0–24 year olds, by district health board, New Zealand 2011–2015

DHB	Unique individuals (n)	Hospitalisations (n)		Ratio All:Primary
		Primary diagnosis	All cases	
Chronic musculoskeletal diseases in 0–24 year olds				
Northland	55	109	133	1.22
Waitemata	209	275	336	1.22
Auckland	137	279	342	1.23
Counties Manukau	184	354	429	1.21
Waikato	97	157	209	1.33
Bay of Plenty	97	181	227	1.25
Lakes	43	61	90	1.48
Tairawhiti	15	49	52	1.06
Taranaki	38	54	63	1.17
Hawke's Bay	63	82	116	1.41
MidCentral	54	77	99	1.29
Whanganui	33	32	43	1.34
Hutt Valley	52	67	87	1.30
Capital & Coast	98	150	166	1.11
Wairarapa	18	21	25	1.19
Nelson Marlborough	39	79	101	1.28
South Canterbury	16	19	19	1.00
Canterbury	155	246	294	1.20
West Coast	6	6	7	1.17
Southern	113	123	167	1.36
New Zealand	1,506	2,431	3,016	1.24

Source: National Minimum Dataset. 'All cases' corresponds to hospitalisations with chronic musculoskeletal diseases listed in any of the first 15 diagnoses; Rate per 100,000 0–24 year olds; Sum of DHBs may equal more than NZ total due to some indiviudal's changing DHB of domicile during the period

Evidence for good practice

Possibilities for prevention

The causes of juvenile idiopathic arthritis (JIA) are unknown but include complex interactions between genetic makeup and environmental exposures.[16] The causes of osteochondrosis are also complex and involve vascular, traumatic, microtraumatic factors.[12] Most cases (80%) of scoliosis are also idiopathic.[13] For all of these conditions the focus is on prompt diagnosis and treatment that is consistent with best practice. There is a lack of evidence for or against population screening for scoliosis, although school programmes exist in many US States.[13] Every clinician involved in the care of children and young people ought to competent in the examination of the musculoskeletal system, and musculoskeletal examination results should be documented in all paediatric hospitalisation records.[1]

Evidence-based health care for children and young people with juvenile idiopathic arthritis

The goal of therapy is to target the underlying inflammation and prevent complications associated with the condition. Early accurate diagnosis is important to allow early aggressive treatment of juvenile idiopathic arthritis (JIA) and optimal prognosis.[1,6] Uveitis is a common and often asymptomatic comorbidity of JIA which can lead to blindness if untreated.[4] Initial screening for uveitis is recommended within a month of JIA diagnosis.[17] Commonly used therapies for JIA include nonsteroidal anti-inflammatory drugs (NSAIDs), disease-modifying antirheumatic drugs (DMARDs), and biologic agents such as the tumour necrosis factor (TNF-α) inhibitors.[3] Flares affecting one or more joints are common after the disease becomes inactive. Clinical decision-making needs to balance the risk of flare if treatment is discontinued with the risks and inconveniences of continuing treatment.[18] Markers of early atherosclerosis are present more often in patients with JIA patients than in their healthy peers, and, in relation to later overt cardiovascular disease, this is likely to be part of a slowly-developing, multifaceted process which may be amenable to preventive measures. A broader management approach to JIA will include promotion of a healthy lifestyle so that patients gain full benefit of effective anti-

inflammatory treatment and secure a healthy life in adulthood.[19] Physical activity can both increase and decrease inflammatory effects for patients with JIA depending on duration and intensity of exercise and personal training.[20]

Juvenile osteochondrosis usually comes to attention only when it is symptomatic.[11] Symptoms are usually self-limiting and respond well to activity modification and anti-inflammatory drug treatment. Surgery may be required for older children and young people with mature skeletons who continue to have disabling symptoms.[5]

Surgery for adolescent idiopathic scoliosis can prevent curve progression and lessen deformity, with statistically and clinically significant improvement in self-image.[21] Surgery is also associated with positive outcomes for children with neuromuscular or genetic conditions.[14,15] A plethora of non-surgical interventions is used for scoliosis including bracing, scoliosis-specific exercises, manual therapy and electrical stimulation. The methodological quality of systematic reviews for such interventions is generally low, and findings from higher quality reviews have not found sufficient evidence to make an informed judgment about the effectiveness of non-surgical interventions in adolescents with idiopathic scoliosis.[22]

International guidelines

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Evidence-based medicine reviews

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Other relevant publications

- Launay F. 2015. Sports-related overuse injuries in children. *Orthopaedics and traumatology: Surgery and Research*, 101(1) S139-S47. http://dx.doi.org/10.1016/j.otsr.2014.06.030

Websites

- Kids with arthritis New Zealand http://www.kidswitharthritis.org.nz/
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CANCER

Introduction

Cancer is the name given to a group of related diseases all characterised by having abnormal (cancer) cells which grow and divide in an uncontrolled fashion and can invade and damage nearby tissues and spread to other parts of the body.[1] The types of cancer that commonly affect children are different from those that commonly affect adults.[2]

In New Zealand around 130 children (aged 0–14 years) and 160 young people (aged 15–24 years) are diagnosed with cancer each year[3,4] and around 50 children and young people die from cancer.[5] Cancer is the third most common cause of death in New Zealand children and young people (aged 28 days to 24 years), after transport-related injuries and suicide.[5]

The most common types of cancer diagnosed in New Zealand children during 2000–2009 were leukaemias and central nervous system tumours.[3] In young people, the most common types were melanomas, carcinomas, lymphomas, germ cell and trophoblastic neoplasms, and leukaemias.[4]

Over the last half century there have been great improvements in the treatment of childhood cancer. Overall child cancer five-year survival rates in New Zealand are now around 80%, comparable to those in other developed countries.[2] Five-year cancer survival rates for New Zealand 15−24 year olds (81%) and 15−19 year olds (75%) are somewhat lower than those in other developed countries.[4]

Data sources and methods

Indicators

> *Incidence of cancer as notified to the New Zealand Cancer Registry (NZCR)*

Data sources

<u>Numerator</u>:   New Zealand Cancer Registry (NZCR), unless indicated otherwise

<u>Denominator</u>:   NZ Statistics NZ Estimated Resident Population

Additional information

The NZCR records cancers diagnosed in New Zealand. NZCR registers each cancer once, in the year of first known diagnosis. Registrations cover new cases of primary cancer, or secondary cancers where the primary cancer is unknown. In the few instances where an individual had multiple registrations for the same cancer, only one registration for the same cancer has been kept.

Year is registration year. Age is age at date of diagnosis. Rates are age-standardised, unless stated otherwise.

Included cancers are as defined by the Ministry of Health, and the codes utilised to identify cases are available in **Appendix 5**. Unless otherwise stated, cancer notifications exclude in-situ neoplasms.

National trends and distribution

From 2000 to 2014 a total of 12,309 individual 0–24 year olds were diagnosed with cancer, an average of 880 individuals per year. Most of these individuals had one cancer diagnosis. As shown in **Figure 65** there has been a small but statistically significant decline in cancer registration rates for 0–14 and 15–24 year olds over this 15-year time period. In 15–24 year olds the decline is due to a fall in incidence of in-situ neoplasm registrations and cancer registration rates were stable over time (**Figure 66**).

Figure 65. Notifications to NZ Cancer Registry for 0–24 year olds, by age group and year, New Zealand 2000–2014
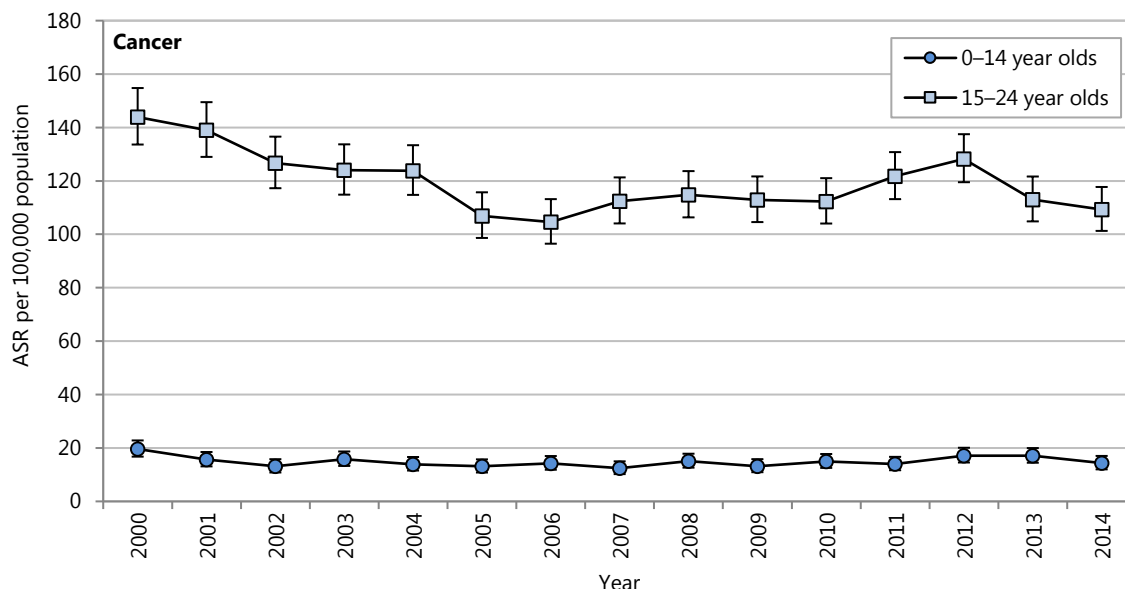
Figure 66. Notifications to NZ Cancer Registry for 15–24 year olds, by type and year, New Zealand 2000–2014
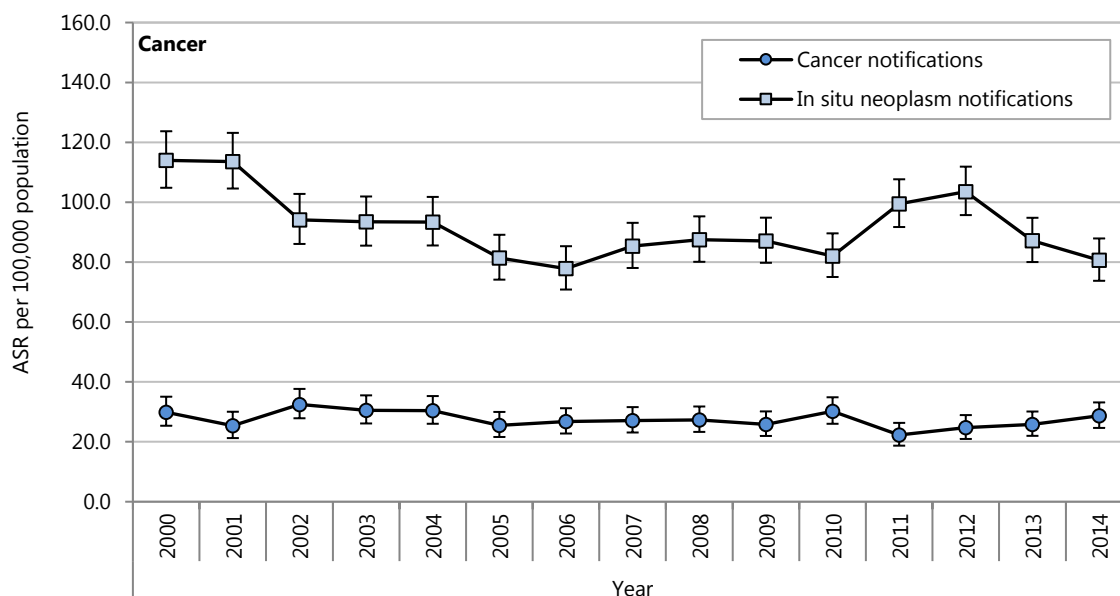
Table 52 presents the rate of cancer diagnosis, deaths and hospitalisations by age group and sex during the most recent five-year period that had data available for each measure. The highest rates of new cancer diagnoses and cancer deaths were among 20–24 year olds, followed by 0–4 year olds. The highest hospitalisation rate was for 0–4 year olds. With the exception of 20–24 year olds, cancer incidence was higher in males than females. The death rate was higher for 0–4 and 15–19-year-old males compared to females, with little difference by gender in other age groups. Hospitalisation rates were higher for males compared with females in all age groups.

Table 52. Cancer in 0–24 year olds, by age group and sex, New Zealand

Age (years)	Incidence per 100,000 population (2010–2014)	Mortality per 100,000 population (2009–2013)	Hospitalisation per 100,000 population (2011–2015)
Cancer			
New Zealand			
Total			
0–4	23.16	3.61	188.37
5–9	10.71	1.48	117.71
10–14	12.19	2.06	112.41
15–19	19.77	4.16	113.67
20–24	32.87	4.80	103.14
Males			
0–4	24.66	4.23	215.03
5–9	12.29	1.18	133.09
10–14	13.70	2.07	126.03
15–19	20.37	5.00	130.87
20–24	31.28	4.76	109.43
Females			
0–4	21.59	2.97	160.23
5–9	9.04	1.79	101.58
10–14	10.59	2.05	98.08
15–19	19.14	3.27	95.58
20–24	34.49	4.84	96.71

Numerator(s): *Incidence:* National Cancer Registry; *Mortality:* National Mortality Collection; *Hospitalisations:* National Minimum Dataset; Denominator: Statistics NZ Estimated Resident Population. Rates are age-specific rates; Cancer includes all malignant tumours and other neoplasms of uncertain/unknown behaviour; Incidence by new registrations, Mortality by underlying cause of death; Hospitalisation by primary diagnosis

Diagnosis

The most common types of cancer in 0–14 year olds between 2010 and 2014 were tumours of the lymphoid, haematopoietic and related tissue, in particular leukaemia, followed by tumours of the central nervous system (including brain and eye) (**Table 53**). Tumours of the lymphoid, haematopoietic and related tissue were also the most common types of cancer for 15–24 year olds, followed by cancers of the skin and male genital organs (**Table 54**).

Table 53. Cancer notifications for 0–14 year olds, by cancer group, New Zealand, 2010–2014

2010–2014	Notifications (n)	ASR per 100,000 population	95% CI
New Zealand			
0–14 year olds			
Cancer notifications			
All cancers	699	15.47	14.34–16.66
Lymphoid, haematopoietic and related tissue	325	7.19	6.43–8.02
Leukaemia	*246*	*5.45*	*4.79–6.17*
Leukaemia: Lymphoid leukaemia	*194*	*4.30*	*3.72–4.95*
Leukaemia: Acute myeloid leukaemia	*20*	*0.44*	*0.27–0.68*
Hodgkin lymphoma	*30*	*0.66*	*0.44–0.94*
Non-Hodgkin lymphoma (except Burkitt lymphoma)	*26*	*0.57*	*0.38–0.84*
Burkitt lymphoma	*12*	*0.27*	*0.14–0.46*
Eye, Brain and other parts of the central nervous system	138	3.06	2.57–3.61
Mesothelial and soft tissue	62	1.38	1.05–1.76
Bones, joints and articular cartilage	47	1.03	0.76–1.37
Thyroid and other endocrine glands	35	0.78	0.54–1.08
Urinary tract	35	0.78	0.54–1.08
Digestive organs	26	0.57	0.38–0.84
Respiratory system and intrathoracic organs	<10	s	s
Lip, oral cavity and pharynx	<10	s	s
Skin	<10	s	s
Female genital organs	<10	s	s
Male genital organs	<10	s	s
Ill-defined, secondary or unspecified sites	<10	s	s

Numerator: National Cancer Registry, Denominator: Statistics NZ Estimated Resident Population. Cancer includes all malignant tumours and other neoplasms of uncertain/unknown behaviour; ASR = Age standardised rate (standardised to 2013 NZ Census population)

Table 54. Cancer notifications and incidence for 15–24 year olds, by cancer group, New Zealand, 2010–2014

2010–2014	Notifications (n)	ASR per 100,000 population	95% CI
New Zealand			
15–24 year olds			
Cancer notifications			
All cancers	821	26.34	24.57–28.21
Lymphoid, haematopoietic and related tissue	233	7.47	6.54–8.49
Leukaemia	*87*	*2.79*	*2.23–3.44*
Leukaemia: Lymphoid leukaemia	*34*	*1.09*	*0.75–1.52*
Leukaemia: Acute myeloid leukaemia	*23*	*0.74*	*0.47–1.10*
Hodgkin lymphoma	*99*	*3.17*	*2.58–3.86*
Non-Hodgkin lymphoma	*38*	*1.22*	*0.86–1.67*
Skin	115	3.70	3.05–4.44
Male genital organs*	109	6.89	5.66–8.31
Thyroid and other endocrine glands	67	2.15	1.67–2.73
Digestive organs	57	1.83	1.39–2.37
Eye, Brain and other parts of the central nervous system	51	1.64	1.22–2.15
Bones, joints and articular cartilage	46	1.47	1.08–1.96
Female genital organs*	55	3.60	2.71–4.68
Mesothelial and soft tissue	36	1.15	0.81–1.60
Lip, oral cavity and pharynx	16	0.51	0.29–0.83
Respiratory system and intrathoracic organs	14	0.45	0.25–0.75
Breast*	14	0.92	0.50–1.54
Urinary tract	<10	s	s

Numerator: National Cancer Registry, Denominator: Statistics NZ Estimated Resident Population. Cancer includes all malignant tumours and other neoplasms of uncertain/unknown behaviour; ASR = Age standardised rate (standardised to 2013 NZ Census population); * = sex-specific rate

Regional distribution

Table 55 and **Table 56** present the number of cancer notifications age-standardised incidence rate (ASR) in each district health board (DHB) between 2010 and 2014 for 0–14 year olds (**Table 55**) and 15–24 year olds (**Table 56**). Five DHBs had fewer than ten notifications; notifications in all remaining DHBs did not differ significantly from the national notification rate.

Table 55. Cancer notifications and incidence for 0–14 year olds, by district health board, New Zealand 2010–2014

2010–2014	Notifications (*n*)	ASR per 100,000 population	Rate ratio	95% CI
0–14 year olds				
Cancer notifications				
Northland	27	14.94	0.97	0.41–2.27
Waitemata	72	12.88	0.83	0.49–1.43
Auckland DHB	68	16.51	1.07	0.61–1.86
Counties Manukau	90	15.18	0.98	0.60–1.60
Waikato	65	15.99	1.03	0.59–1.82
Bay of Plenty	32	14.13	0.91	0.41–2.01
Lakes DHB	22	18.78	1.21	0.47–3.14
Tairawhiti	<10	s	s	s
Taranaki	20	16.83	1.09	0.41–2.92
Hawke's Bay	29	16.76	1.08	0.47–2.48
MidCentral	20	11.64	0.75	0.28–2.04
Whanganui	<10	s	s	s
Hutt Valley	32	21.19	1.37	0.62–3.03
Capital & Coast	53	19.38	1.25	0.67–2.34
Wairarapa	<10	s	s	s
Nelson Marlborough	17	12.69	0.82	0.28–2.40
South Canterbury	<10	s	s	s
Canterbury	81	17.13	1.11	0.66–1.85
West Coast	<10	s	s	s
Southern DHB	41	14.67	0.95	0.47–1.91
New Zealand	699	15.47	1.00	

Numerator: National Cancer Registry, Denominator: Statistics NZ Estimated Resident Population. Cancer includes all malignant tumours (ICD-10-AM codes C00–C97) and other neoplasms of uncertain/unknown behaviour (D45–D47); ASR = Age standardised rate (standardised to 2013 NZ Census population)

Table 56. Cancer notifications and incidence for 15–24 year olds, by district health board, New Zealand 2011–2015

2010–2014	Notifications (*n*)	ASR per 100,000 population	Rate ratio	95% CI
15–24 year olds				
Cancer notifications				
Northland	30	31.16	1.18	0.53–2.66
Waitemata	83	21.71	0.82	0.50–1.36
Auckland DHB	99	26.70	1.01	0.64–1.61
Counties Manukau	91	24.13	0.92	0.57–1.48
Waikato	65	24.11	0.92	0.52–1.61
Bay of Plenty	40	32.01	1.22	0.60–2.46
Lakes DHB	25	37.72	1.43	0.59–3.47
Tairawhiti	<10	s	s	s
Taranaki	19	27.81	1.06	0.38–2.91
Hawke's Bay	37	37.94	1.44	0.69–3.00
MidCentral	44	34.89	1.32	0.67–2.61
Whanganui	12	30.76	1.17	0.33–4.19
Hutt Valley	26	27.52	1.04	0.44–2.50
Capital & Coast	62	26.86	1.02	0.57–1.81
Wairarapa	10	42.42	1.61	0.40–6.41
Nelson Marlborough	20	26.59	1.01	0.37–2.72
South Canterbury	<10	s	s	s
Canterbury	85	23.99	0.91	0.55–1.50
West Coast	<10	s	s	s
Southern DHB	47	19.89	0.75	0.39–1.46
New Zealand	821	26.34	1.00	

Numerator: National Cancer Registry, Denominator: Statistics NZ Estimated Resident Population. Cancer includes all malignant tumours (ICD-10-AM codes C00–C97) and other neoplasms of uncertain/unknown behaviour (D45–D47); ASR = Age standardised rate (standardised to 2013 NZ Census population)

Evidence for good practice

Possibilities for prevention

Unlike adult cancers, very few childhood cancers have known preventable causes.[6] One of the few preventable causes is exposure to ionising radiation in utero from diagnostic radiography.[6] Since this association was recognised in the 1950s, health services have been careful to minimise women's radiation exposure during pregnancy.[6]

While childhood cancers are largely not preventable, there are important preventive measures that children and young people can take to reduce their risks of getting cancer later in life. Getting sunburnt in childhood is associated with an increased risk of developing melanoma skin cancer in adulthood.[7] There has been little research on sun protection specifically for children but evidence from studies in adults suggests that the following measures are effective: avoiding sun exposure when sun is at its highest; avoiding the use of artificial UV sources such as sun beds; wearing sun-protective clothing, wide-brimmed hats and sunglasses; and liberal use of sunscreen products on all exposed skin.[8]

Infection with human papilloma virus is the main cause of cervical cancer and it is also a significant cause of vaginal, vulvar, penile, anal and oropharyngeal cancers.[9] Vaccination against HPV (for both girls and boys) offers protection against HPV-related cancers.[9] Vaccination against hepatitis B reduces the incidence of hepatocellular carcinoma (a type of liver cancer), which is associated with chronic hepatitis B infection.[10]

Smoking is a cause of many types of cancer in adults: lung, larynx, oral cavity, pharynx, oesophagus, pancreas, bladder, kidney, cervix, and stomach, and acute myeloid leukaemia.[11] Since most adult smokers started smoking as teenagers or young adults, discouraging young people from taking up smoking is a very effective way to prevent cancers caused by smoking.[12]

Evidence-based health care for children and young people with cancer

Treatment for child cancer is complex, involving multiple types of treatment (chemotherapy, radiotherapy, surgery) that are delivered over several years by multidisciplinary teams.[13] There is evidence that better treatment outcomes for child cancer are achieved by high (vs. low) volume hospitals, high (vs. low) volume providers, and specialised (vs. non-specialised) hospitals, although the differences are relatively small.[14] Suggested explanations for this include 'practice makes perfect' and 'selective referral' (hospitals or physicians with good reputations receive more referrals).[15] These factors have not been explored in relation to child cancer treatment specifically and it is likely that both play a part.[14]

New Zealand's small population makes it challenging to provide high quality child cancer services nationwide. New Zealand currently has two specialist child cancer services, located in Auckland and Christchurch. These have shared care arrangements with the other DHBs so that a specialist paediatric oncology service is responsible for advising on and coordinating the initial diagnostic work-up, the provision of intensive therapy, and overall management of a child's care but once the child is stabilised, some components of treatment can be provided closer to the child's home.[13]

In developed countries, including New Zealand, children with cancer are generally treated according to protocols developed through international collaborative research studies.[16] Since the early 2000s, the rate of decrease in child cancer mortality has slowed considerably and it is probable that optimisation of currently available anti-cancer treatments has been achieved.[16] New therapies are being developed which target the molecular biomarkers associated with particular sub-types of cancer[17] but so far targeted drugs have been of benefit to only a very small proportion of child cancer patients.[18] (Biomarkers are substances, mostly proteins, that are produced by cancer cells or by other body cells in response to cancer, and that can be detected and measured in tissues or body fluids.[19])

Treatment for child cancer has both short term and long term adverse effects. Commonly experienced short term effects include fatigue, lack of energy, pain, anaemia, infection, lack of appetite, hair loss, bruising, nausea, vomiting, diarrhoea, and mucositis (painful mouth ulcers due to inflammation and ulceration of the lining of the digestive tract).[20] In the long term child cancer survivors are at lifelong risk of developing a subsequent primary cancer and of having multiple physical and psychosocial health problems including cardiovascular disease, cardiomyopathy, pulmonary fibrosis, renal dysfunction, obesity, mental health problems, and endocrinopathies (e.g. premature gonadal failure, thyroid disease, and osteoporosis).[21] Most of the serious health problems do not become apparent until decades after cancer treatment has ended.[22] Lifelong follow-up of survivors is therefore regarded as best practise.[23,24]

These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of cancer are suggested for further reading.

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APPENDICES

APPENDIX 1. EVIDENCE FOR GOOD PRACTICE

For most indicators in this report there is a section devoted to evidence for good practice. These sections comprise evidence summaries, references and links. They aim to provide readers with a starting point from which to consider the most effective interventions that are available to address particular child and youth health issues. Included are New Zealand policy documents such as Ministry of Health Strategies and Toolkits, New Zealand and international guidelines, and evidence-based reviews that are relevant to the prevention and management of child and youth health issues. The approach taken in these sections is intended to assist health professionals use the principles of evidence-based medicine (EBM), that is, to solve problems by using the best available research evidence and combining this with clinical expertise and patient values.[1] Evidence-based reviews, the best known of which are those produced by the Cochrane Collaboration, collate all the available evidence (published and unpublished trials, observational studies etc.) relevant to a particular health intervention, evaluate it in a rigorous manner, and publish the resulting synthesis of the evidence in a format that allows readers to quickly evaluate the effectiveness of the intervention.

When preparing the Evidence for Good Practice section for each indicator, the authors searched a number of EBM journals and databases (e.g. the Cochrane Library) as well as Ovid MEDLINE and PubMed for systematic reviews of population level interventions in child and youth health (see Text Box below). They also conducted smart searches in Google Scholar for journal articles and Google for government documents.

Methodology used in preparing policy/evidence of good practice sections

New Zealand (health) policy documents

Each review section provides a list of Ministry of Health (or where appropriate, other Government Agency) policy documents and strategies relevant to the area. Using Google.com a smart search was conducted of Ministry of Health and other government departments.
Example smart searches used:
        ("fetal alcohol syndrome" OR "fetal alcohol spectrum disorder" OR FAS OR FASD) site:.health.govt.nz
        ("fetal alcohol syndrome" OR "fetal alcohol spectrum disorder" OR FAS OR FASD) site:.govt.nz

Evidence of good practice

The databases listed below were searched for reviews assessing the effectiveness of population level interventions to prevent and/or manage each of the issues included in this report. These databases were chosen because of the high calibre of the institutions maintaining them. The search strategy concentrated on publications that attempted to synthesise all of the available evidence, thereby providing the broadest possible coverage of the relevant literature. In general, only literature from the last three years was searched, although earlier publications were included if there was a lack of more recent information. Individual trials and protocols were not specifically sought but if there was no other relevant information available, an attempt was made to locate individual research reports or recommendations. It is hoped that that, although the lists of references provided are not completely comprehensive, they will nevertheless provide a useful starting point for DHBs wishing to explore strategies to address particular child and youth health issues.

Evidence-Based Medicine Reviews
This database allows seven EBM resources to be searched at once including The Database of Reviews of Effects (DARE), Health Technology Assessments (HTA) and the NHS Economic Evaluation Database (NHSEED) all produced by National Health Services' Centre for Reviews and Dissemination at the University of York, U.K., The Cochrane Database of Systematic Reviews, and the ACP Journal Club.

National Guideline Clearinghouse http://www.guideline.gov
This is a searchable database of evidence-based clinical practice guidelines maintained by the Agency for Healthcare Research and Quality in the United States.

Centre for Reviews and Dissemination (CRD) http://www.york.ac.uk/inst/crd
This is a department of the University of York and is part of the National Centre for Health Research (NCHR). While CRD produces the database of Review Effects (DARE), captured in the Evidence-Based Medicine Review Database, searching the CRD site identifies other reviews not captured by DARE. This database is available through most local library services. Due to cessation of funding, no new records have been added to the database since March 2015.

National Institute for Health and Clinical Excellence (NICE) http://www.nice.org.uk
This is an independent organisation based in the United Kingdom which provides national guidance on the promotion of good health and the prevention and treatment of ill health.

Guide to Community Preventive Services: Systematic Reviews and Evidence Based Recommendations
This guide was developed by the non-federal Task Force on Community Preventive Services whose members are appointed by the Director of the Centres for Disease Control and Prevention (CDC). The Community Guide summarises what is known about the effectiveness, economic efficiency, and feasibility of interventions to promote community health and prevent disease http://www.thecommunityguide.org/about.

In addition to these databases the websites of the World Health Organization, and government health departments in Australia, the UK, the US, and Canada, often yielded relevant guidance, as did the sites of international clinical collaborations such as the European Cystic Fibrosis Society and the International Society for Pediatric and Adolescent Diabetes.

References

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APPENDIX 2. STATISTICAL METHODS

Inferential statistics are used when a researcher wishes to use a sample to draw conclusions about a larger population as a whole; for example, weighing a class of 10-year-old boys, in order to estimate the average weight of all 10-year-old boys in New Zealand. The findings obtained from the sample provide an estimate for the population, but will always differ from it to some degree, simply due to chance. Similarly, samples are used when a researcher questions whether the risk of developing a particular condition is different between two groups, and the fit of the estimate obtained from the samples to the actual population needs to be carefully considered. An example of this would be a study examining whether lung cancer is more common in smokers or non-smokers: researchers using sample groups would have to consider the possibility that some of 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. These measures can assign a level of confidence to estimates and conclusions drawn from samples, allowing researchers to assess, for example, whether the average weight of boys in the sample reflects the true weight of all 10-year-old boys, or the rates of lung cancer in smokers are really different to those in non-smokers. Two of the most frequently used statistical significance tests are:

P **values:** The *p* value from a statistical test measures the probability of finding a difference at least as large as the one observed between groups, if there were no real differences between the groups studied. For example, 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 if the $p<0.05$; that is, when the probability of the observed differences occurring by chance is less than 5%.[1]

Confidence Intervals: When sampling from a population a confidence interval is a range of values that contains the measure of interest. While a confidence interval for the average height of 10-year-old boys could be 20cm to 200cm, for example, the smaller range of 130cm to 150cm is a more informative statistic. 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.[1] Where the observed counts are small and the denominator is large, then a Poisson distribution has been utilised for both rate and confidence interval calculations,[2] in particular for the congenital anomaly and cancer indicators.

Crude rates: Measures the number of people with the condition of interest in relation to the number of people in the population. It is calculated by dividing the number of people with the condition of interest in a specific time period by the total number of people in the population in the same time period.

Age-specific rates: Measures the occurrence of an event within a defined age group in relation to the number of people in that group. Age-specific rate is calculated by dividing the number of people with the condition of interest in a specific age group and time period by the total number of people in the population in the same age group and time period.

Age-standardised rates (**ASR**)**:** Age-standardised rates (ASR): A statistical technique that makes it possible to make meaningful comparisons between populations with different age structures. Age-standardisation adjusts rates for differences in the age distribution of the population over time or differences in age distribution between population groups. The direct standardisation method has been used to calculate ASR in this report.[3] The population used as the standard population was the New Zealand 2013 Census Estimated Resident population (**Table 57**), which is different from the standard population used by the Ministry of Health. The cancer indicator presents age-standardised rates, as indicated in information accompanying the relevant tables and graphs. The indicators in this report are mainly presented using crude (unadjusted) rates or age group (age-specific) rates, with the exception of cancer which uses age-standardised rates.

Table 57. Individuals aged 0–24 years by age group, and sex, New Zealand 2013 census data

Age group (year)	Male	Female	Total	NZ population weightings
New Zealand 2013 Census population				
0–4	160,160	151,780	311,940	0.343
5–9	153,580	146,490	300,070	0.330
10–14	152,180	144,580	296,760	0.327
Total 0–14	465,920	442,850	908,770	
15–19	160,160	152,330	312,490	0.498
20–24	159,190	155,730	314,920	0.502
Total 15–24	319,350	308,060	627,410	

Source: Statistics NZ Estimated Resident Population 2013 Census

Statistical significance testing in this report

When tests of statistical significance have been applied in a particular section, the statistical significance of the associations presented has been signalled in the text with the words significant, or not significant. 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.

Several data sources are used in this report. In general they belong to one of two groups: 1) population surveys or 2) routine administrative datasets. The relevant statistical testing for each of these data sources are as follows:

Population surveys: Some of the indicators reported on here are derived from data from national surveys 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 included in the text accompanying graphs and tables. In a small number of cases, information on statistical significance was not available, and any associations described do not imply statistical significance.

Numbers derived from routine administrative data: A large number of the indicators included in this report are based on data from New Zealand's administrative datasets, for example the National Mortality Collection, which capture information on all of the events occurring in a particular category.

Rate ratios derived from routine administrative data: To facilitate comparisons between different time periods, and for examining the data from New Zealand in a wider context, whenever measures of association (rate ratios) are presented in this report, 95% confidence intervals have been provided.[4]

References

1. Webb P, Pirozzo S. 2005. Essential epidemiology: An introduction for students and health professionals Cambridge: Cambridge University Press.
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APPENDIX 3. DATA SOURCES

This report contains information derived from several national administrative datasets and population surveys. These are described briefly below, and limitations to be aware of when interpreting results drawn from these sources are outlined.

National Mortality Collection

The National Mortality Collection is a dataset managed by the Ministry of Health that contains information on the underlying cause, or causes, of death along with basic demographic data for all deaths registered in New Zealand since 1988. Fetal and infant death data are a subset of the Mortality Collection, with cases in this subset having additional information on factors such as birth weight and gestational age.[1] 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 information from other sources such as Coronial Services, Police, NZ Transport Agency, the New Zealand Cancer Registry (NZCR), the Institute of Environmental Science and Research, and Water Safety NZ.[2]

National Minimum Dataset

The National Minimum Dataset (NMDS) is a national hospital discharge dataset and is maintained by the Ministry of Health. It is used for policy formation, performance monitoring, and research purposes, providing key information about the delivery of hospital inpatient and day patient health services both nationally and on a provider basis. It is also used for funding purposes.[3]

Information in the NMDS includes principal and additional diagnoses, procedures, external causes of injury, length of stay and sub-specialty codes; and demographic information such as age, ethnicity and usual area of residence. Data have been submitted by public hospitals electronically since the original NMDS was implemented in 1993, with additional data dating back to 1988 also included. The private hospital discharge information for publicly funded events has been collected since 1997. The current NMDS was introduced in 1999.[3]

Birth Registration Dataset

Since 1995 all New Zealand hospitals and delivering midwives have been required to notify the Department of Internal Affairs within five working days of the birth of a live or stillborn baby. This applies to stillborn babies born at or more than 20 weeks gestation, or weighing 400g or more: prior to 1995, only stillborn babies reaching more than 28 weeks of gestation required birth notification. Information on the hospital's notification form includes maternal age, ethnicity, multiple birth status, and the baby's sex, birth weight and gestational age. In addition, parents must jointly complete a birth registration form as soon as reasonable practicable after the birth, and within two years of delivery, which duplicates the above information with the exception of birth weight and gestational age. 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 details.[4]

National Cancer Register

The New Zealand Cancer Registry (NZCR) is national population-based cancer/tumour registry maintained by the Ministry of Health. The role of the NZCR is:

1. to provide information on the incidence of, and mortality from, cancer
2. to provide a basis for cancer survival studies and research programmes

The NZCR holds information on individuals diagnosed in New Zealand with a new malignant tumour (invasive and in-situ; excluding squamous and basal cell skin cancers), including pathological information. Information in the NZCR includes tumour information (such as morphology, site, diagnostic test, multiplicity), staging information, and demographic information such as age, ethnicity and usual area of residence. The quality of the data has improved since 1998. Data have been collated from pathology reports submitted electronically by laboratories, and from the NMDS, and national mortality collection.[5,6,7]

New Zealand Health Survey

The Ministry of Health's New Zealand Health Survey (NZHS) became an annual survey in 2011. The survey is conducted by interviewing a sample of adults and children's parents or caregivers in New Zealand, and utilises a core set of questions that cover a range of health-specific indicator areas, including health behaviours, conditions and use of health services.[8] **Table 58** presents the number of participants selected for each NZ Health Survey conducted and the corresponding coverage rate. The coverage rate is a measure related to survey response and is defined as the ratio of the sum of the selection weights for the survey respondents to the known external population size.[8]

The NZ Health Survey utilised adjusted rate ratios to account for the potential influence of other demographic factors when undertaking demographic comparisons. Gender comparisons are adjusted for age, ethnic comparisons are adjusted for age and gender, and deprivation comparisons are adjusted for age, sex and ethnicity.[8]

Table 58. Number of survey participants and coverage, New Zealand Health Survey

Survey year (1 July–30 June)	Adults (15 years and over)		Children (2–14 year olds)	
	n	Coverage (%)	*n*	Coverage (%)
New Zealand Health Survey				
2006/2007	12,488	59	4,921	67
2011/2012	12,370	54	4,478	68
2012/2013	13,009	59	4,485	69
2013/2014	13,309	54	4,699	63
2014/2015	13,497	59	4,754	69

Source: New Zealand Health Survey Methodology reports 2006/07–2014/15

Estimated prevalence

The NZ Health Survey presents the demographic factors for each surveyed condition using unadjusted and adjusted prevalence rates, and the total estimated prevalence as calculated by the Ministry of Health have been presented in this report. The Ministry of Health has weighted the survey data used for calculating prevalence to ensure that each group is represented appropriately, i.e. not under or over represented, in the survey estimates. This is achieved by a weight being devised for each respondent based on their chance of selection, such that a lower change of selection results in a higher weight and vice versa, or survey response rate (e.g. lower response rate means a higher weighting).[8]

The prevalence of a condition, or the proportion of the population with the condition e.g. diabetes, was estimated by calculating the sum of the weights for the survey respondents with the condition divided by the sum of the weights of all survey respondents. For example, the sum of the weights for survey respondents with diabetes is divided by sum of the weights for all survey respondents.[8]

Further information on the prevalence estimates, methodology and interpretation of the NZ Health Survey are available on the NZ Health Survey pages of Ministry of Health website http://www.health.govt.nz/nz-health-statistics/national-collections-and-surveys/surveys/current-recent-surveys/new-zealand-health-survey

Data limitations

There are limitations when using any of these datasets. The following are of particular relevance to this report.

Clinical coding accuracy and coding changes over time

The quality of data submitted to the administrative national datasets may vary. While the data for the National Mortality Collection and the Birth Registration Dataset are coded by single agencies, the clinical information held in the NMDS is entered by health providers before being collated by the Ministry of Health. In a 2001 review of the quality of coding in the data submitted to the NMDS, 2,708 events were audited over ten sites during a three-month period. Overall the audit found that 22% of events required a change in coding, although this included changes at a detailed level. Changes to the primary diagnosis involved 11% of events, to additional diagnoses 23%, and to procedure coding, 11%. There were 1,625 external causes of injury codes, of which 15% were re-coded differently.[9] 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, the average 16% error rate indicated by the 2001 review may be an overestimate as, in the majority of the analyses undertaken in this report, only the primary diagnosis is used to describe the reason for admission.

Changes in the coding systems used over time may result in irregularities in time series analyses.[2] New Zealand hospitals use the clinical coding classification developed by the World Health Organization and modified by the National Centre for Classification in Health, Australia. The current classification is called The International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (ICD-10-AM), the Australian Classification of Health Interventions (ACHI) and Australian Coding Standards (ACS). The introduction of ICD-10-AM represented the most significant change in classification in over 50 years, expanding the number of codes from ~5,000 to ~8,000, to provide for recently recognised conditions and allow greater specificity about common diseases.

From 1988 until 1999, clinical information in the NMDS was coded using versions of the ICD-9 classification system. From July 1999 onwards, the ICD-10-AM classification system has been used. Back and forward mapping between the two systems is possible using predefined algorithms,[9] and for most conditions there is a good correspondence between ICD-9 and ICD-10-AM codes. Care should still be taken when interpreting time series analyses which include data from both time periods as some conditions may not be directly comparable between the two coding systems.

Variation in reporting hospitalisations to the NMDS

Historically, there have been differences in the way New Zealand's 20 district health boards (DHBs) have reported their emergency department (ED) hospitalisations to the NMDS, which can affect the interpretation of hospitalisation data. Inconsistent recording of ED cases has resulted from differing definitions of the time spent in the ED, and at what point this time constitutes an admission. This is important in paediatrics where hospitalisations for acute onset infectious and respiratory diseases in young children especially are mainly of short duration. In addition, there are regional differences in treatment processes for paediatric emergency cases.

This report includes all ED day cases in its analyses of hospitalisations for medical conditions. This approach differs from that commonly used by the Ministry of Health when analysing NMDS hospital discharge data, which the Ministry of Health uses to minimise the impact of the inconsistent reporting of ED cases. Short stay ED events are often excluded from the Ministry's analyses to improve comparability between regions. However, as noted above, the treatment of children in acute cases differs from that of adults, and the inclusion of ED day cases is justified when considering hospitalisations for medical conditions, despite inconsistencies in the dataset. The Ministry of Health's practice of filtering out ED day cases for hospitalisations for injuries is followed in this report as it is considered that the processes for injury assessments are relatively consistent around the country.

Further information on the details of the inconsistencies can be seen in earlier reports by the NZCYES www.otago.ac.nz/ncyes

Changes in the way ethnicity information has been recorded over time

Due to inconsistencies in the way ethnicity information was recorded in the health sector, and in census data before 1996, all ethnic group specific analyses in this report are for the year 1996 onwards. See **Appendix 4** for a brief review of the changes in the recording of ethnicity information over the past 35 years in New Zealand.

References

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8.   Ministry of Health. 2015. Methodology report 2014/15: New Zealand Health Survey.
        http://www.health.govt.nz/publication/methodology-report-2014-15-new-zealand-health-survey
        accessed September, 2016
9.   New Zealand Health Information Service. 2002. Coder's Update. In 2001/2002 Ministry of Health
        Data Quality Audit Program 1-4.

APPENDIX 4. DEMOGRAPHIC FACTORS

Ethnicity data

Because of inconsistencies in the manner in which ethnicity information in New Zealand was collected prior to 1996, all ethnic group specific analyses presented in this report are for the 1996 year onwards, and reflect self-identified concepts of ethnicity. Details of the changes made in the census question on ethnicity, and why they were made, can be found on the Statistics New Zealand website www.stats.govt.nz.

Unless otherwise specified, prioritised ethnic group has been used to ensure that each health event is only counted once. 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 as the potential still remains for Māori and Pacific children and young people to be undercounted in our national data collections.

The authors of Hauora IV developed a set of adjusters which could be used to minimise the bias such undercounting introduced when calculating population rates and rate ratios. These, or similar, adjusters were not utilised in this report because previous research has shown that ethnicity misclassification can change over time and ethnic misclassification may vary significantly by district health board.[1,2] Adjusters developed using national level data (as in Hauora IV) may not be applicable to district health board level analyses, with separate adjusters needing to be developed for each.

In addition, the development of adjusters requires the linkage of the dataset under review with another dataset for which more reliable ethnicity information is available, and 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 data presented in this report may undercount Māori and Pacific children to a variable extent depending on the dataset used, and in the case of the hospital admission dataset for Māori, this undercount may be as high as 5–6%.

NZ Deprivation Index

The NZ index of deprivation (NZDep) was first created using information from the 1991 census, and has been updated following each census. It is a small area index of deprivation, and is used as a proxy for socioeconomic status. The main concept underpinning small area indices of deprivation is that the socioeconomic environment in which a person lives can confer risks or benefits which may be independent of their own social position within a community.[3] They are aggregate measures, providing information about the wider socioeconomic environment in which a person lives, rather than information about their individual socioeconomic status.

The latest index, NZDep2013, combines nine variables from the 2013 census to reflect eight dimensions of material and social deprivation (Table 59). Each variable represents a standardised proportion of people living in an area who lack a defined material or social resource. These are combined to give a score representing the average degree of deprivation experienced by people in that area. Individual area scores are 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.[4]

The advantage of the NZDep2013 is its ability to assign measures of socioeconomic status to the older population, the unemployed and to children, to whom 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.[3] Despite these limitations, the NZDep2013 has been shown to be predictive of mortality and morbidity from a number of diseases in New Zealand.

Table 59. Variables used in the NZDep2013

Dimension	Variable in order of decreasing weight in the index
Communication	People aged < 65 with no access to the Internet at home
Income	People aged 18–64 receiving a means tested benefit
Income	People living in equivalised* households with income below an income threshold
Employment	People aged 18–64 unemployed
Qualifications	People aged 18–64 without any qualifications
Owned home	People not living in own home
Support	People aged <65 living in a single parent family
Living space	People living in equivalised* households below a bedroom occupancy threshold
Transport	People with no access to a car

*The setting of the household equivalised income threshold was based on two principles: 1) the proportion of the population identified as being socioeconomically deprived by the threshold should be broadly consistent with the other variables in the index, and 2) the threshold should be broadly consistent with other measures of income poverty.[4]

References

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APPENDIX 5. CLINICAL CODES

The following are the codes associated with the conditions presented in this report.

	ICD-10-AM	ICD-9-CM
Cancer	C00–C96, D45–D47	140–208, 235–238
Cystic fibrosis	E84	277.0
Eczema or dermatitis	L20–L30, B00.0, B00.1, H01.1	690–698, 0540, 3733
Diabetes		
Type 1 diabetes	E10	250.x1, 250.x3
Type 2 diabetes	E11	250.x0, 250.x2
Diabetes - other	E09, E12, E13, E14	
Chronic lower respiratory diseases	**J40–J47**	**490–494, 496**
CLRD: Bronchitis	J40–J42	490–491
CLRD: Emphysema	J43	492
CLRD: Asthma (incl Status asthmaticus & Wheeze)	J45–J46, R06.2	493
CLRD: Bronchiectasis	J47	494
CLRD: Other	J40–J47	490–494, 496
Digestive		
Constipation	K59.0	564.0
Crohn disease	K50	555
Ulcerative colitis	K51	556
Mental and behavioural		
Autism or other pervasive developmental disorders	F84	299
Intellectual disability and developmental delay	F70–F79, R62	317–319, 7834
Musculoskeletal		
Juvenile arthritis	M08–M09	714.3
Juvenile osteochondrosis	M91–M92, M42.0, M93.2	732.0–732.1, 732.3–732.7
Scoliosis	M41	737.43
Nervous system		
Cerebral palsy	G80	343
Epilepsy or status epilepticus	G40–G41	345
Nutritional and eating disorders		
Eating disorders	F50	307.1, 307.5
Obesity	E66	278.0

Congenital anomalies

	ICD–10–AM	ICD–9–CM/A
All anomalies	Q codes, P350, P351, P371	740–759
Non-chromosomal anomalies (NCA)	Q0–Q8	740–757, 759
Nervous system anomalies	Q00–Q07	740–742
Neural tube defects	Q00, Q01, Q05	740, 741, 742.0
Anencephalus and similar	Q00	740
Encephalocele	Q01	742.0
Spina bifida	Q05	741
Hydrocephalus	Q03	742.3
Microcephaly	Q02	742.1
Arhinencephaly/holoprosencephaly	Q04.1, Q04.2	742.2
Eye anomalies	Q10–Q15	743
Anophthalmos/microphthalmos	Q11.0, Q11.1, Q11.2	743.0, 743.1
Congenital cataract & lens anomalies	Q12.0	743.3
Congenital glaucoma	Q15.0	743.20
Ear, face and neck anomalies	Q16, Q18, Q17.8	744.0, 744.21, 744.24, 744.29, 744.4–744.9
Anotia (Absence of external ear)	Q16.0	744.01

	ICD–10–AM	ICD–9–CM/A
Congenital Heart Defects (CHD)	Q20–Q26	745–746, 747.0–747.4
Severe CHD	Q20.0, Q20.3, Q20.4, Q21.2, Q21.3, Q22.0, Q22.4, Q22.5, Q22.6, Q23.0, Q23.4, Q25.1, Q26.2	745.00, 745.10, 745.2, 745.3, 745.6, 746.1, 746.2, 746.00, 746.3, 746.7, 747.1, 747.42
Common arterial truncus	Q20.0	745.00
Transposition of great vessels	Q20.3	745.10
Single ventricle	Q20.4	745.3
VSD	Q21.0	745.4
ASD	Q21.1	'745.5
AVSD	Q21.2	745.6
Tetralogy of Fallot	Q21.3	'745.2
Tricuspid atresia and stenosis	Q22.4	746.1
Ebstein's anomaly	Q225	746.2
Pulmonary valve stenosis	Q221	746.01
Pulmonary valve atresia	Q22.0	746.00
Aortic valve atresia/stenosis	Q23.0	746.3
Hypoplastic left heart	Q23.4	746.7
Hypoplastic right heart	Q22.6	no code
Coarctation of aorta	Q25.1	747.1
Total anomalous pulmonary venous return (TAPVR)	Q26.2	747.42
Term PDA	Q25.0	747.0
Respiratory anomalies	Q30–Q34	748
Choanal atresia	Q30.0	748.0
Cystic adenomatous malformation of lung	Q33.80	no code
Oro-facial clefts	Q35–Q37,	749.0–749.2
Cleft lip with or without cleft palate	Q36, Q37	749.1, 749.2
Cleft palate with cleft lip	Q37	749.2
Cleft lip only	Q36	749.1
Cleft palate	Q35	749.0
Digestive system	Q38–Q45, Q79.0 and not Q38.1, Q38.2	750–751, 756.6 and not 750.0, 750.15
Oesophageal atresia (OA) with or without tracheo-oesophageal fistula (TOF)	Q39.0, Q39.1	750.30, 750.31
Duodenal atresia or stenosis	Q41.0	751.10
Atresia or stenosis of other parts of small intestine	Q41.1–Q41.8	751.11–751.12
Ano-rectal atresia and stenosis	Q42.0–Q42.3	751.21–751.24
Hirschsprung's disease	Q43.1	751.30–751.33
Atresia of bile ducts	Q44.2	751.65
Annular pancreas	Q45.1	751.72
Diaphragmatic hernia	Q79.0	756.61
Abdominal wall defects	Q79.2, Q79.3, Q79.5	756.7 and not 756.72
Omphalocele / Gastroschisis	Q79.2, Q79.3	756.7 and not 756.72
Gastroschisis	Q79.3	
Omphalocele	Q79.2	
Urinary	Q60–Q64, Q79.4	753, 756.72, 752.62
Bilateral renal agenesis (including Potter syndrome)	Q60.1, Q60.6	753.00
Renal dysplasia	Q61.4	753.16
Congenital hydronephrosis	Q62.0	753.20
Bladder exstrophy and/or epispadias	Q64.0, Q64.1	753.5, 752.62
Posterior urethral valve and/or prune belly	Q64.20, Q79.4	753.60
Genital	Q50–Q52, Q54–Q56 and not Q53	752.0–752.4, 752.6, 752.7–752.9 and not 752.5
Female genital anomalies	Q50–Q52	752.0–752.4
Hypospadias	Q54	752.6
Other penial anomalies	Q55	no code

	ICD–10–AM	ICD–9–CM/A
Indeterminate sex	Q56	752.7
Limb anomalies	Q65–Q74	754.3–754.8, 755
Limb reduction	Q71–Q73	754.3–754.8, 755.6
Upper limb reduction	Q71	755.2
Lower limb reduction	Q72	755.3
Complete absence of a limb	Q71.0, Q72.0, Q73.0	755.20, 755.30, 755.40
Club foot	Q66.0	754.50
Hip dislocation and/or dysplasia	Q65.0–Q65.2, Q65.80, Q65.81	754.30, 754.31, 754.35
Polydactyly	Q69	755.0
Syndactyly	Q70	755.1
Other anomalies / syndromes		
Skeletal dysplasias	Q78.2–Q78.8, Q74.02, Q77, Q78.00, Q87.16	no code
Craniosynostosis	Q75.0	756.00
Congenital constriction bands/amniotic band	Q79.80	751.42, 762.80
Situs inversus	Q89.3	759.3
Conjoined twins	Q89.4	759.4
Congenital skin disorders	Q80–Q81, Q82 and not Q82.5, Q82.8	757.1 and not 757.32, 757.39
Teratogenic syndromes with malformations	Q86, P35.0, P35.1, P37.1	760.71, 771.0, 771.1, 771.21
Fetal alcohol syndrome	Q86.0,	760.71
Valproate syndrome	Q86.80	no code
Maternal infections resulting in malformations	P35.0, P35.1, P37.1	771.0, 771.1, 771.21
Genetic syndromes + microdeletions	Q44.71, Q61.90, Q74.84, Q75.1, Q75.4, Q75.81, Q87, Q93.6	759.8
Sequences	Q60.6, Q64.10, Q79.4, Q79.80, Q87.03, Q87.08, Q87.24, Q89.80	no code
Chromosomal defects	Q90–Q93, Q96–Q99, Q95.2–Q95.9	758.0–758.3, 758.5–758.9
Down syndrome	Q90	758.0
Patau syndrome (trisomy 13)	Q91.4–Q91.7	758.1
Edwards syndrome (trisomy 18)	Q91.0–Q91.3	758.2
Turner syndrome	Q96	758.6
Klinefelter syndrome	Q98.0–Q98.4	758.7