

Chapter 13

Chronic Disease: Asthma, Cancer, Diabetes, and Oral health

Asthma

Key points

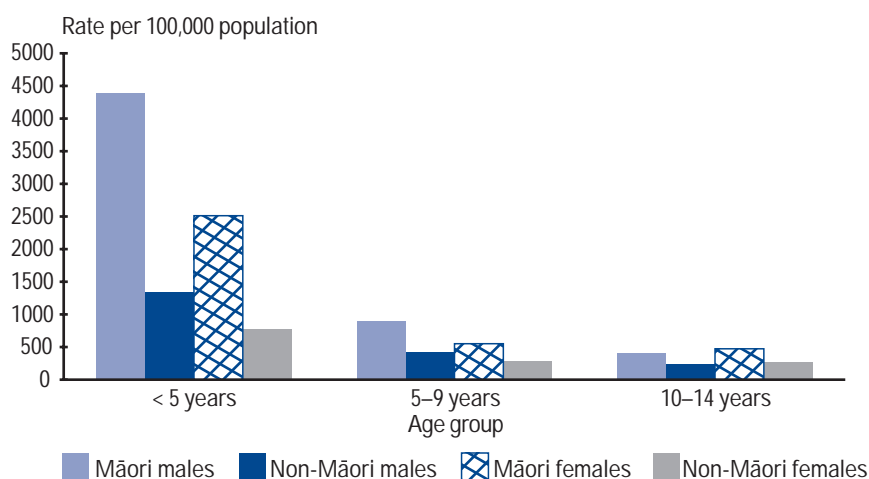
- In 1995, asthma accounted for 5869 hospitalisations among New Zealand 0–14-year-olds. Few New Zealand children now die from asthma. There were two deaths in 1994.
- An estimated 44 percent of New Zealand children experience asthma symptoms at some time before age 15. The prevalence of childhood asthma has increased over the last 20–30 years.
- There is a worldwide trend towards an increasing prevalence of asthma in childhood. The precise reason for this is unknown although multiple factors are thought to be contributing. Improved awareness of symptoms and diagnostic transfer do not fully account for the reported changes.
- Compared to other OECD countries, New Zealand and Australia appear to have high rates of childhood asthma.
- In New Zealand, ethnic differences in reported prevalence rates for childhood asthma are small and cannot account for the large disparity between the rates of hospitalisation for asthma evident for Māori and non-Māori. Between 1988 and 1995, Māori children were consistently hospitalised for asthma at more than twice the rate of non-Māori. Differential access to asthma care, including prophylactic medication, has been implicated in this disparity.
- The predisposition to asthma is genetically determined and is gender expressed, with males more likely to exhibit symptoms in childhood than females.
- Important allergens responsible for the development of asthma include house dust mites, the mould *Alternaria* and cats. These allergens are often more prevalent in sub-standard housing.
- Tobacco smoke is implicated as a factor contributing to the earlier onset and increased severity of childhood asthma.
- There is a critical period in early childhood when asthma may be induced in the genetically vulnerable child. Reduced exposure to allergens and other modifying factors such as maternal smoking, diet and infection early in life is thought to influence the development and severity of asthma.
- Adequate preventative medical care and asthma management by children and their families is important for effectively controlling asthma and preventing severe attacks.

Morbidity and prevalence

In 1995, there were 5869 hospitalisations of children due to asthma. Māori continue to be hospitalised at a higher rate than non-Māori, with the Māori rate being more than twice the non-Māori rate in 1995. Males aged 0–14 years are more likely to be hospitalised from asthma than are females. For the Māori population, the male rate was 69 percent higher than the female rate in 1995. For non-Māori, the male rate of hospitalisation from asthma exceeded the female rate by 53 percent (Ministry of Health 1997d).

Children aged 0–4 years accounted for 68 percent of all hospitalisations in the 0–14 year age group in 1995. Disparities between Māori and non-Māori and between males and females were most marked in this age group. Māori aged 0–4 years were three times as likely to be hospitalised for asthma as non-Māori in 1995 (Figure 13.1.1).

Figure 13.1.1: Hospitalisation due to asthma, 0–14-year-olds, 1995



Source of data:
Ministry of Health 1997d.

Over the last decade in New Zealand, a range of different measures of asthma prevalence and different tools for obtaining these measures has been used. This has resulted in wide variations in reported estimates of asthma prevalence. More recently, during the 1990s, a standard protocol for measuring asthma prevalence has been developed for the International Study of Asthma and Allergies in Childhood (ISAAC) (Asher et al 1995). The ISAAC protocol, which is now practised internationally, uses a video and a standard questionnaire to assess a range of respiratory symptoms such as ‘wheeze’ (Crane et al 1995).

Table 13.1.1 presents summary results from three recent New Zealand childhood asthma prevalence studies that have used the ISAAC protocol.

Table 13.1.1: Prevalence of asthma in New Zealand children, 1993–95

	<i>Ages 5–8 years</i>	<i>Ages 12–15 years</i>
Wheeze in last 12 months	26–28% ^{1,2,3}	22–28% ^{1,4}
Wheeze ever	44% ²	44% ^{2,4}
Asthma ever	24–28% ^{1,2}	18–30% ^{1,3}

1 *Moyes et al 1995*

2 *Wilkie et al 1995*

3 *Robson et al 1993*

4 *Pearce et al 1993*

Males aged 0–14 years are more likely than females to experience asthma symptoms. Consistently higher rates of asthma symptoms and diagnosed asthma have been reported for males (Mitchell 1983; Shaw et al 1994; Ministry of Health and Statistics New Zealand 1993; Horwood et al 1985). One study involving a birth cohort found that boys were twice as likely as girls to develop asthma by age six years (Horwood et al 1985).

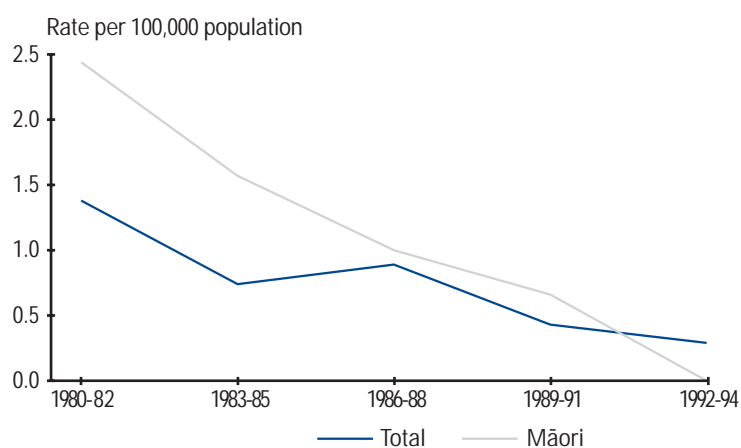
A number of New Zealand studies have reported on asthma prevalence by ethnicity. At most, there appear to be only minor differences in the prevalence of asthma between different ethnic groups in New Zealand. Any minor differences in the prevalence rates detected do not account for the high disparity between Māori and non-Māori rates of childhood hospitalisation for asthma. Nor does it appear that differences in socioeconomic status or passive smoking fully account for these differences. It is more likely that these differences are due to differential access and delivery of asthma care, including access to prophylactic medication (Robson et al 1993; Ministry of Health and Statistics New Zealand 1993; Shaw et al 1994; Pattermore et al 1989; Mitchell 1983).

Changes over time

There have been two epidemics of asthma deaths in New Zealand in the last 20 years, the first in the late 1970s and the second in the early 1980s. These epidemics have been attributed in part to the introduction and use of two specific asthma drugs (Beasley et al 1990; Pearce et al 1990; Pōmare et al 1992).

Few New Zealand children die each year due to asthma. In 1994, there were two asthma deaths recorded for children aged 0–14 years (Ministry of Health 1997b). The childhood asthma death rate has decreased since the early 1980s. Between 1980 and 1994 there was a five-fold decrease in the recorded death rate for 10–14-year-olds (Figure 13.1.2). A similar decline in the number of asthma deaths has occurred in both the total and Māori populations, as well as for both males and females (Ministry of Health 1997b).

Figure 13.1.2: Deaths due to asthma, 0–14-year-olds, 1980–94



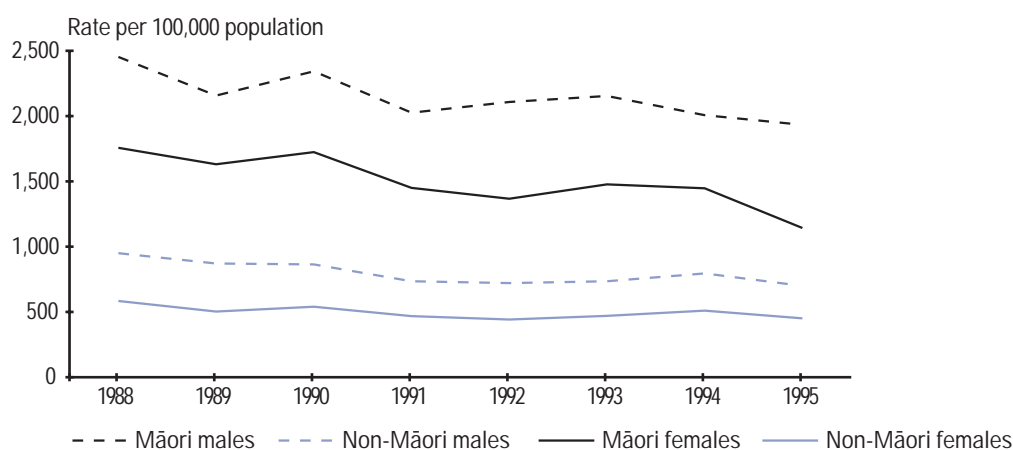
Source of data:
Ministry of Health 1997b.

Between 1988 and 1995, the hospitalisation rate for asthma decreased 25 percent for the 0–14 year age group. Māori hospitalisations for this age group also decreased in this period (by 27 percent), meaning that the disparity between the total and Māori asthma hospitalisation rates essentially remained unchanged (Figure 13.1.3).

The greatest rate decrease was recorded for Māori females aged 0–14 years. They had a 35 percent decrease in asthma hospitalisations between 1988 and 1995. This contributed to a decrease in the disparity between the Māori and non-Māori female rates. In 1988, Māori females were hospitalised at three times the rate of non-Māori females. By 1995 this disparity had decreased to 2.5 times. Over the same period the disparity between Māori and non-Māori males increased slightly (from 2.6 times in 1988 to 2.8 times in 1995).

Between 1988 and 1995, within the 0–14 year age group children aged 5–9 years experienced the largest drop in rates of hospitalisation for asthma, a 39 percent decrease (Ministry of Health 1997d).

Figure 13.1.3: Hospitalisation due to asthma, 0–14-year-olds, 1988–95



Source of data:
Ministry of Health 1997d.

The reported prevalence of childhood asthma is increasing worldwide. Surveys undertaken during the past 20 years using similar methods throughout have found a consistent increase in the prevalence of asthma (Newman-Taylor 1995; Mellis 1994).

A number of possible reasons have been postulated to account for this increase, although no single factor has yet been conclusively identified as the main contributor. Current reviews of available data indicate that this ubiquitous increase in asthma prevalence for children is likely to result from an interaction of multiple factors. Explanations proposed include an increase in allergen exposure (particularly to house-dust mites), higher rates of tobacco smoking in women of child-bearing age, and dietary changes (Newman-Taylor 1995).

While both physician and public awareness of the signs and symptoms of asthma have increased in the last 10–15 years, available data suggest that the increase in asthma prevalence in children cannot be fully accounted for by diagnostic transfer (Weiss et al 1993; Mellis 1994).

A number of studies have reported a significant increase in the prevalence of asthma symptoms in New Zealand children (Mitchell 1983; Mitchell and Asher 1994; Kljakovic 1991).

Mitchell and Asher (1994) reported a 33 percent increase in the prevalence of respiratory symptoms, with no significant increase in diagnosed asthma or severity indices. Mitchell (1983) found in an earlier study that the prevalence of asthma had doubled between 1969 and 1982. Kljakovic (1991) reported a 56 percent increase in the prevalence of 'wheeze' between 1971 and 1990.

International comparisons

During the asthma mortality epidemic of the 1980s, New Zealand children were dying from asthma at a higher rate than most other OECD countries. However, by the early 1990s, the New Zealand death rate from asthma compared favourably with the rest of the OECD (PHC 1994a).

Prevalence rates of asthma and respiratory symptoms appear to be higher for New Zealand and Australian children than for children in other OECD countries (Pearce et al 1993; Barry et al 1991; Burr et al 1994). A four-country comparative study of 12–15-year-olds using the ISAAC protocol found that Australian and New Zealand children reported higher rates of severe asthma than European children. However, rates of milder asthma symptoms did not vary significantly between countries (Pearce et al 1993). Two other studies found that Australian and New Zealand 12-year-olds had higher prevalence rates for a number of asthma symptoms than 12-year-olds from other OECD countries (Barry et al 1991; Burr et al 1994).

Risk and protective factors

The predisposition to asthma and atopy is genetically determined and is probably present in up to one-third of the population. The disease asthma is present once the pathological process responsible for altered airway responsiveness has developed. It is switched on by irritants, allergens or infectious agents. Studies have suggested that heritability could account for 60 or 70 percent of hay fever and asthma. This genetic susceptibility is also gender expressed, with males more likely than females to exhibit asthma symptoms early in childhood (Landau 1993).

Three important groups of allergens thought to be responsible for asthma in children include house-dust mites, the mould *Alternaria*, and cats (Woolcock et al 1995; Newman-Taylor 1995). Living in substandard housing often results in excess exposure to these indoor allergens (Malveaux and Fletcher-Vincent 1995).

The frequency of allergic disease is increased in the offspring of women who smoke. Babies born to women who smoke have diminished lung function compared to those born to non-smokers (Newman-Taylor 1995). Tobacco smoke has also been implicated in the earlier onset and increased severity of asthma (Weiss et al 1993; Landau 1993; Abramson 1995; Malveaux and Fletcher-Vincent 1995).

Viral infections are well recognised to be precipitating factors in asthma attacks, especially respiratory infections before the age of two (Weiss et al 1993, Landau 1993).

There is no substantive evidence that air pollution is an important factor inducing allergies and asthma (Newman-Taylor 1995; Weiss et al 1993).

The current belief is that there is a critical period in early infancy when asthma may be induced in the genetically vulnerable child. Reduced exposure to allergens, as well as other modifying factors such as maternal smoking, diet, and infection early in life, is thought to influence the development and severity of childhood asthma (Mellis 1994; Woolcock et al 1995; Newman-Taylor 1995; Landau 1993).

Other important contributing factors are thought to include the provision of appropriate preventative medical care, and children and their families possessing good asthma knowledge and management skills (Malveaux and Fletcher-Vincent 1995).

Cancer

Key points

- There were 126 newly diagnosed cancers registered in New Zealand among children aged 0–14 years in 1994. Childhood cancer deaths totalled 35 in 1994.
- Leukaemia and brain tumours are the most common childhood malignancies in New Zealand, accounting for about 30 percent and 20 percent of newly diagnosed cases respectively.
- Since the mid-1960s there has been a significant increase in the incidence of leukaemia among New Zealand children. This increase appears to be mostly confined to the 0–4 year age group. This is consistent with similar increases in the incidence of childhood leukaemia reported in other western populations.
- Survival rates for children with leukaemia have improved greatly in recent years. This has been attributed to improved treatment protocols.
- There has been a major increase in the incidence rate of brain cancer in New Zealand since the 1950s. In particular, a significant increase has been found in the 0–4 year age group. These findings are consistent with worldwide trends in the reported incidence of brain cancer. However, brain cancer rates have remained stable in New Zealand children in more recent years.

- Only about one quarter of all brain cancer patients survive five years.
- There are only three known causes of childhood cancer: ionising radiation, genetic factors (for specific types of cancer) and cancer chemotherapy drugs. Industrial and agricultural chemicals are being assessed as possible causes of childhood cancer, but it is too early to make definitive statements about whether they have a causal role.
- Some childhood exposures such as cigarette smoke, ultraviolet light, and viruses may contribute to cancers that develop many years after childhood.
- Children are thought to be more susceptible than adults to the carcinogenic effects of some types of exposures, such as radiation.

Mortality

All cancers

Cancer is the second most common cause of death (after injuries) for children aged 1–14 years (Ministry of Health 1997b). Thirty-five children under 15 years of age died from cancer in 1994 (an age-specific rate of 4.3 per 100,000). Males accounted for 18 of the 35 deaths and Māori deaths numbered seven (Table 13.2.1).

Table 13.2.1: Childhood cancers, incidence and mortality, by sex and ethnicity, 1994

	<i>Mortality (1994)</i>		<i>Incidence (1994)</i>	
	<i>No.</i>	<i>*Rate</i>	<i>No.</i>	<i>*Rate</i>
Total	35	4.3	127	15.5
Males	18	4.3	72	17.2
Females	17	4.3	55	13.8
Relative risk for males		1.0		1.2
Māori	7	6.5	19	17.7
Non-Māori	28	3.9	108	15.2
Relative risk for Māori		1.7		1.2

* Age-specific rate per 100,000 population.

Source of data:

Ministry of Health 1997b, 1998.

Leukaemia

Leukaemia (ICD-9 codes 204–208) accounted for 12 of the 35 childhood cancer deaths in 1994 (an age-specific rate of 1.5 per 100,000).

Brain cancer

Brain cancer (ICD-9 code 191) accounted for six of the 35 childhood cancer deaths in 1994 (an age-specific rate of 0.7 per 100,000). Only about a quarter of all brain cancer patients survive five years. Many of these patients remain impaired after aggressive surgical intervention or die later from a recurrence of the condition (Preston-Martin et al 1993).

Morbidity

Hospitalisations

In New Zealand in 1995, there were 1628 hospitalisation episodes involving children aged 0–14 years due to cancer (Ministry of Health 1997d). These cancer hospitalisations comprised 1 percent of all hospitalisations for this age group (excluding newborns).

Registrations

A national cancer register records newly diagnosed primary cancers in New Zealand. A statutory requirement to report cases to the register did not take effect until 1995. However, completeness of cancer registration was high even before then, at about 97 percent in the early 1990s (Dockerty et al 1997). Therefore, as in other countries in which cancer registries operate, cancer registration data are used to estimate cancer incidence in New Zealand. Registration data presented below exclude in situ cancers.

Childhood cancer registrations totalled 126 in 1994, an age-specific rate of 15 per 100,000. Males accounted for 71 (56 percent) of the 126 new cancers registered. Nineteen (15 percent) of the total registrations were for Māori children and 14 (11 percent) were Pacific children. Children aged 0–4 years accounted for 53 (42 percent) childhood cancers in 1994, compared with 38 (30 percent) aged 5–9 years and 35 (28 percent) aged 10–14 years.

There is a difference between Māori and non-Māori in the histological type of brain tumours diagnosed. Medulloblastoma is the most common paediatric brain-cancer among Māori. However, astrocytoma is the most common type among non-Māori children in New Zealand, as it is in most Western populations (Preston-Martin et al 1993).

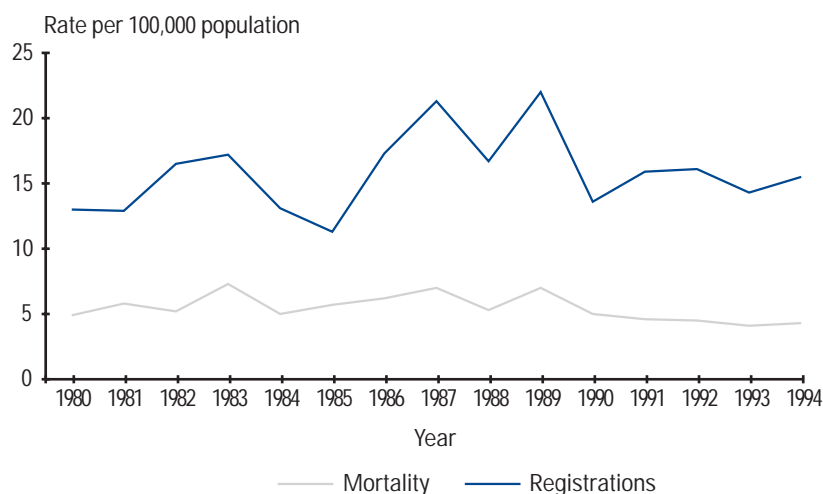
Changes over time

All cancers

Overall, childhood mortality rates for cancer decreased slightly in New Zealand between 1980 and 1994 (Figure 13.2.1). Between 1988 and 1995, an average annual increase in the rate of hospitalisations of 5 percent was recorded for children with cancer (Ministry of Health 1998). The proportion of hospitalisations that were daypatients increased across the period. However, the average inpatient length of stay decreased during this time.

Between 1980 and 1994, childhood cancer registration notes fluctuated from year to year, but there was no overall trend (Figure 13.2.1). The stable incidence yet lower mortality may indicate improvements in the treatment of childhood leukaemia over this period.

Figure 13.2.1: Childhood cancers, incidence and mortality, 1980–94



Source of data:
Ministry of Health 1997b, 1998.

Leukaemia

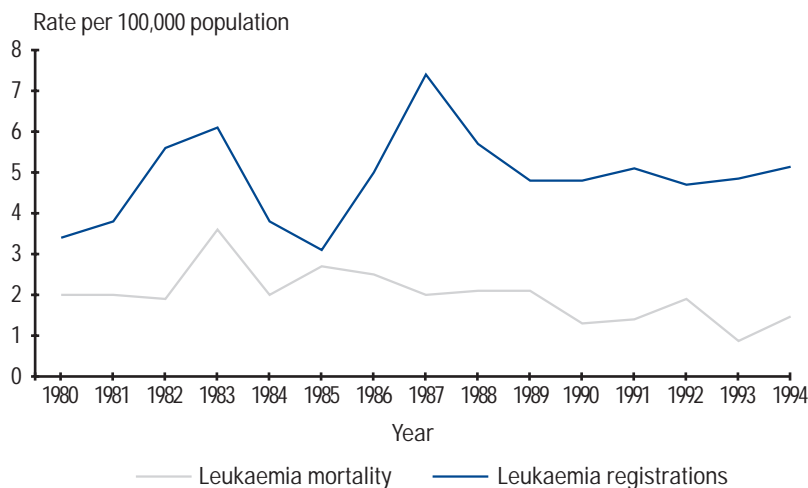
Mortality rates for childhood leukaemia remained relatively stable between 1980 and 1994 (Figure 13.2.2).

A recent study has analysed the time trends for childhood leukaemia using the New Zealand cancer registry data (Dockerty et al 1996). A highly significant increase was found in the incidence of leukaemia in children aged under five years between the mid-1960s and 1990. The increase was present in boys and girls and in the combined non-Māori population. For Māori, no overall statistically significant trend was detected. For the 5–9 and 10–14 year age groups, no statistically significant increases or decreases in the incidence of leukaemia were found.

The increase in incidence in the 0–4 year age group was attributed to an increase in acute lymphoblastic leukaemia (ALL). The incidence of acute non-lymphoblastic leukaemia (ANLL) decreased overall during the period being investigated. The risk of ALL was lower in the Māori than the non-Māori population. The risk of ANLL was higher among Māori. (Dockerty et al 1996). The increases in the incidence of ALL among children aged 0–4 or 1–4 years are consistent with findings reported in other countries (Dockerty et al 1996).

Survival rates for children diagnosed with leukaemia have improved greatly in recent years. One New Zealand study (Bailey and Lewis 1996) compared survival and disease-free survival of children with ALL over two periods, 1980–86 and 1986–92, when two different treatment protocols were used. Actuarial survival (life expectancy) increased from 53 percent to 93 percent and disease-free survival increased from 47 to 88 percent between the two periods. This significantly improved outcome was attributed to improved treatment.

Figure 13.2.2: Childhood leukaemia, incidence and mortality, 1980–94

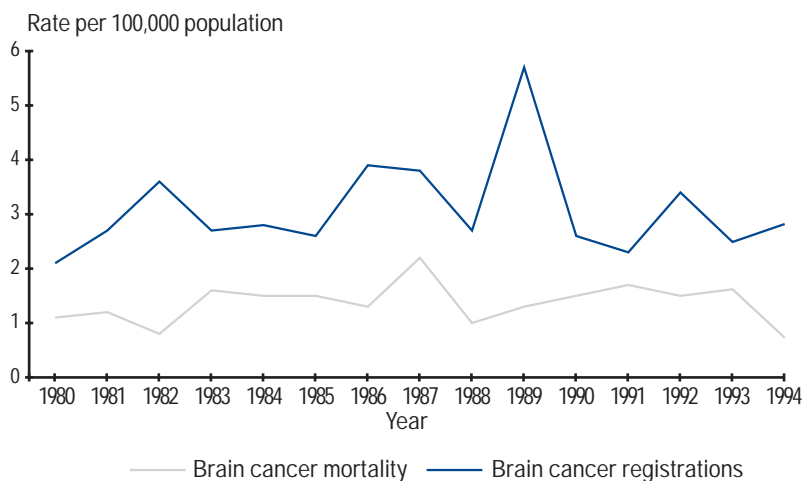


Source of data:
Ministry of Health 1997b, 1998.

Brain cancer

A worldwide increase in the incidence of brain cancer has been reported for recent decades. A New Zealand study analysed brain cancer registrations between 1948 and 1988 and found the age-standardised rate doubled over the 40 year time period. Statistically significant increases were found for several age groups, including the 0–4 year age group (Preston-Martin et al 1993). However, neither mortality rates nor registration rates of brain cancer in children changed significantly in more recent years (Figure 13.2.3).

Figure 13.2.3: Childhood brain cancer, incidence and mortality, 1980–94



Source of data:
Ministry of Health 1997b, 1998.

Risk factors

Environmental carcinogens

There are only three known causes of childhood cancer; ionising radiation, genetic factors (for specific types of cancer) and cancer chemotherapy drugs. Industrial and agricultural chemicals are being assessed as possible causes of childhood cancer, but it is too early to make definitive statements about whether they have a causal role.

Some childhood exposures, such as; cigarette smoke, ultraviolet light, and viruses, may contribute to the development of cancers that arise many years after childhood.

For some exposures, such as radiation and pesticides, there is limited evidence to suggest that children are more susceptible to the carcinogenic effects than adults who have been similarly exposed. There are also suggestions of possible interactions between environmental carcinogens and genetic susceptibility (Zahm and Devesa 1995).

The most well-established cause of childhood cancer is radiation, especially high dose radiation exposure such as that experienced by atomic bomb survivors and children receiving radiation therapy for cancer. The effects of lower dose radiation exposure are less well understood (Zahm and Devesa 1995).

The possibility that neonatal vitamin K prophylaxis increases the risk of leukaemia has not been excluded, but at most the risk is small (Von Kries 1996).

Sociodemographic factors

Several studies, but not all, have reported a higher risk of childhood leukaemia for first-born children. With a reduction in family sizes, the proportion of first-born children in the population will have increased (Dockerty et al 1996).

There is some evidence that children from the higher socioeconomic groups have an increased risk of childhood cancer, but findings are inconsistent (Dockerty et al 1996).

Diabetes

Key points

- Insulin-dependent diabetes mellitus (IDDM) is an important chronic childhood condition that is associated with major risks to health status in adulthood. It accounts for around 300 hospitalisations a year in the 0–14 year age group.
- The work on the epidemiology of IDDM in New Zealand is consistent with international work showing that a complex mixture of genetic and probable environmental factors (such as viral agents) are involved in IDDM causation.
- There is evidence that the incidence of IDDM in the 0–14 year age group is increasing in New Zealand.

Introduction

Insulin-dependent diabetes mellitus (IDDM) is a disorder in which there is a deficiency of the hormone insulin. This is due to autoimmune destruction of insulin-producing cells in the pancreas. IDDM is also known as type I diabetes or juvenile-onset diabetes. Without treatment, individuals with IDDM develop severe metabolic disturbances which can lead to death.

This section focuses entirely on IDDM, although a small proportion of diabetes in the 0–14 year age group could be classified as ‘maturity-onset diabetes of youth’ rather than IDDM.

Mortality

While diabetic ketoacidosis can result in death, diabetes causes fewer than one death per year in the 0–14 year age group in New Zealand (Ministry of Health 1997c). There were no deaths reported for 1994, the most recent year for which data were available at the time this document was written. However, diabetes is an important cause of premature death among adults and was the primary cause of death in an average of over 400 individuals per year in the period 1992–94 (Ministry of Health 1997c).

Morbidity

There is a significant burden associated with IDDM in terms of individuals requiring a daily injection of insulin, the need for them to closely monitor their metabolic control and the requirement for constant attention to dietary intake.

Incidence

Various estimates for the annual incidence of IDDM in children and adolescents are shown in Table 13.3.1.

Hospitalisations represent both new (incident) cases and readmissions for those with established IDDM. Between 1991 and 1995 there was an average of 296 hospitalisations per year for diabetes in the 0–14 year age group (an average annual rate of 36.8 hospitalisations per 100,000 population).

Table 13.3.1: Studies on the incidence of diabetes in New Zealand

<i>Population</i>	<i>Annual incidence per 100,000</i>	<i>Study</i>
0–15-year-olds, all NZ (1968–1972)	8.9	Crossley and Upsdell 1980
0–15-year-olds, Auckland (1977–84)	9.3	Elliott and Pilcher 1985
0–15-year-olds, Canterbury (1982–85)	10.2	Rewers et al 1988
0–14-year-olds, Canterbury (1982–92)	14.7	Brown 1993
0–19-year-olds, Canterbury (1990–92)	19.5	Forbes et al 1993

Hospitalisation rates are one indicator of the more severe end of the morbidity spectrum associated with diabetes. Hospitalisation data for Canterbury in 1983 showed that 15 percent of people with IDDM were hospitalised during the year (although 42 percent of these admissions were not recorded as being associated with diabetes) (Brown et al 1985).

Hospitalisations for ketoacidosis may also indicate how well IDDM in childhood is being managed. However, the recent data for New Zealand 0–14-year-olds indicate no significant trends. For the period 1988–90, the annual rate of hospitalisation for diabetic ketoacidosis was 9.8 per 100,000 children aged 0–14 years, compared to 10.4 per 100,000 children in 1991–93 and 9.4 per 100,000 children in 1994–95.

Prevalence

An estimate of the prevalence of IDDM in Canterbury in 1986 was 1.1 per 1000 population aged 0–19 years (Mason et al 1987). A more recent estimate for Auckland was 1.5 cases per 1000 children aged 0–14 years (Vogel et al 1996). This latter figure made diabetes the seventh most prevalent chronic childhood condition after asthma, mental retardation, autism, congenital heart disease, cerebral palsy, and cleft lip.

The prevalence of serious complications among young people with IDDM in New Zealand has not been described. Nevertheless, diabetes in adulthood is associated with complications from cardiovascular disease (including cerebrovascular disease), eye disease (retinopathy, glaucoma, cataracts, blindness), kidney disease, neuropathy, and foot and lower limb problems (Ministry of Health 1997a; Simmons 1996b).

Gender

For the period 1991–95, the average annual diabetes hospitalisation rate for females was higher than males (40.2 versus 33.4 per 100,000 population respectively) (Ministry of Health 1997d). No significant gender differences in IDDM incidence have been found in other New Zealand studies (Crossley and Upsdell 1980; Mason et al 1987).

Ethnicity

Lower incidence rates of IDDM for Māori and Pacific children are apparent in data for Auckland (Elliott and Pilcher 1985). In addition, hospitalisation rates are lower for Māori than non-Māori (21.6 versus 39.0 per 100,000 population respectively). Pacific peoples appear to have a generally low rate of IDDM in comparison to other ethnic groups (Unger and Foster 1992).

Age

Data from a Canterbury study (Scott and Brown 1991) show an increase in the annual incidence of IDDM with age, as does the national hospitalisation data (Table 13.3.2). The prevalence of IDDM more than doubles between age 0–9 years and 10–19 years.

Table 13.3.2: Incidence and hospitalisation rates for diabetes by age group (per 100,000 population)

Study details	Age (years)		
	0–4	5–9	10–14
Incidence rate – Canterbury (Scott et al 1992)	8.2	8.9	19.8
Annual hospitalisation rate – all New Zealand (primary diagnosis for 1991–95) (Ministry of Health 1998)	18.6	28.5	65.8

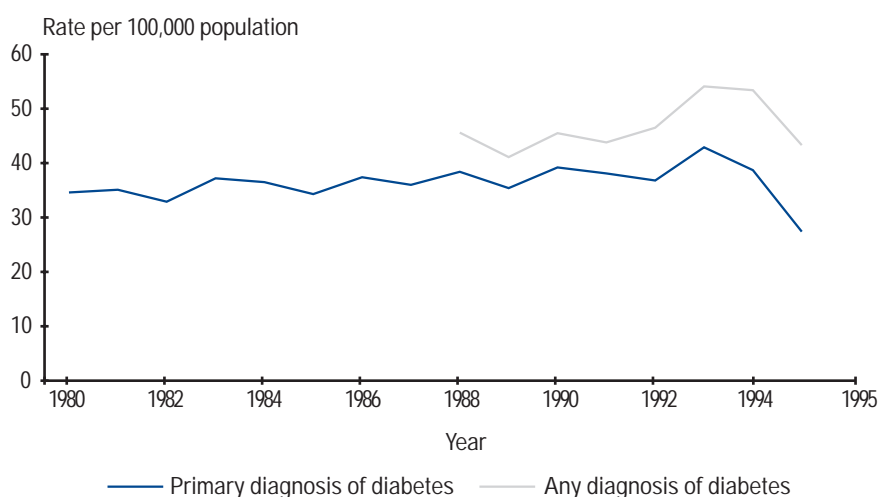
Changes over time

Data from Canterbury indicate no change in the incidence of IDDM among 0–19-year-olds over time (Scott et al 1992). However, data for Europeans in Auckland from 1977 to 1986 are suggestive of a 10 percent increase (but not in Māori and Pacific peoples) (DERI 1990). The national hospitalisation data shown in Figure 13.3.1 show no overall trend.

Hospitalisation rates are also influenced by changes in coding practices, especially where data are based on diabetes as the primary diagnosis alone (Simmons 1996a). The limitations of hospitalisation data for reflecting the true incidence of diabetes have been described before (Simmons 1996a).

The national trend data disguise the fact that within regions there are marked year-to-year fluctuations in risk and even the suggestion of ‘epidemics’ of IDDM in children (Brown 1993). A significant seasonal association to the onset of IDDM has also been found in boys, with incidence rates being significantly higher in winter than in summer (Scott et al 1992). Seasonal variation in the incidence of IDDM has been observed worldwide (reviewed by Dorman et al 1995).

Figure 13.3.1: Diabetes (ICD-9 code 250) hospitalisation, 0–14-year-olds, 1980–95



Source of data:
Ministry of Health 1997d.

Note:
Communication with New Zealand Health Information Service suggests that changes in the coding of diabetes occurred in 1995, with a shift from the use of diabetes in the primary diagnostic categories to its use as a secondary or tertiary diagnosis. Further work to clarify this issue is required.

International comparisons

Compared to the incidence in other countries, New Zealand is 'mid-ranked' with rates similar to those in Australia, the United Kingdom and North America, but lower than in Scandinavian countries (Karvonen et al 1993).

There is some evidence that IDDM is increasing globally (LaPorte et al 1995).

Risk factors

IDDM is the outcome of a complex mixture of genetic and environmental factors. However, worldwide at least 60 percent of childhood diabetes, and perhaps as much as 95 percent, is considered to be environmentally triggered and therefore potentially preventable (DERI 1987). The following discussion on risk factors briefly considers the major issues.

Genetic factors

Genetic factors are clearly important in the causation of IDDM (Dorman et al 1995). Around 20 percent of individuals with IDDM have a family history of IDDM. A genetic susceptibility for IDDM has been identified in children and adolescents in the Canterbury region of the South Island (Brown 1993; Forbes et al 1993).

Nutrition

A number of nutritional practices have been associated with an increased risk of IDDM. These include the consumption of smoked/cured mutton (Helgason and Jonasson 1981), high dietary nitrosamines (Dahlquist et al 1990) and high nitrate in water supplies (Kostraba et al 1992). There have also been numerous studies examining the early consumption of cow's milk and/or the absence of breastfeeding as possible risk factors for IDDM. Overall, it would seem that further studies are required to clarify the issue of cow's milk and other possible dietary factors.

Viruses

The global epidemiology of IDDM (WHO DIAMOND Project on Epidemics 1992) and occurrences of spatial clustering of IDDM (for example, Bodington et al 1995) are suggestive of a role for viral agents. Coxsackie viruses and cytomegalovirus infection have been implicated, but the evidence is still not conclusive and there may be complex host-environment interactions (Dorman et al 1995). Congenital rubella infection appears to be associated with the subsequent development of IDDM (for example, Forrest et al 1971). While the evidence for a role for rubella is quite good, it is equivocal for mumps (Dorman et al 1995).

New Zealand data supporting the importance of environmental causes for IDDM mainly derive from the epidemic nature of IDDM described in the South Island Canterbury region (Brown 1993) and the data suggestive of winter peaks (at least among boys (Scott et al 1992)).

Other risk factors

Older maternal age may be a risk factor for IDDM in the children of such women. Young maternal age (under age 25 years) may be a risk factor for children in the case of mothers with IDDM.

Other possible risk factors for IDDM that have limited relevance to the 0–14 year age group include: the occurrence of pregnancy and various other stressful life events. New Zealand research has indicated that women aged 15–19 years who become pregnant have more than a five-fold increased risk of developing IDDM compared to other women (Crossley and Upsdell 1980).

The evidence for the role of socioeconomic factors in IDDM is contradictory (Dorman et al 1995).

Oral health

Key points

- Major improvements in oral health of New Zealand children occurred in the 1970s and 1980s. Progress slowed in the 1990s and has worsened since 1994.
- In 1996, five-year-old children had an average of 1.7 missing or filled primary teeth (mft), and 55 percent had no dental caries. Form Two children had an average of 1.5 missing or filled permanent teeth (MFT), and 45 percent were caries-free.
- Form Two Māori children have on average 60 percent more MFT than non-Māori Form Two children.
- Children in socioeconomically disadvantaged families have more caries than others.
- Two important sources of fluoride to inhibit dental caries are fluoridated water and fluoride toothpaste. An estimated 47 percent of New Zealanders receive fluoridated water through reticulated supplies. Eighty-six percent of toothpaste sold in New Zealand contains fluoride.
- Children whose home water supply is fluoridated have 31 percent fewer mft at age five years and 26 percent fewer MFT at Form Two than those without access to fluoridated water.
- New Zealand children have better dental health than children in most other OECD countries.

Morbidity

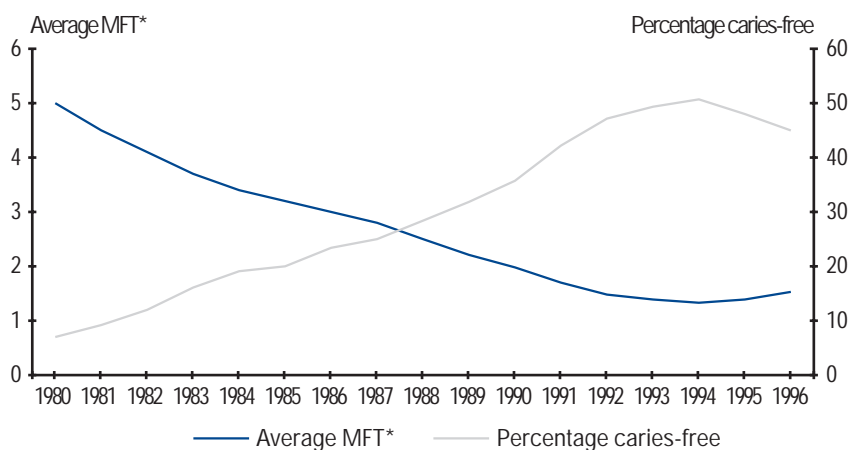
Dental caries (tooth decay) is a chronic, progressive, and largely irreversible disease. At a microscopic level, very early decay of tooth surfaces can be stopped and reversed. However, once cavitation has occurred, treatment and restorative dental procedures are required. Good dental care is essential for health, self-esteem and pain-free living. Poor oral health can lead to problems in eating and talking. Serious and persistent dental problems can disrupt school, work and social life. Untreated caries can result in the need for admission to hospital for acute medical complications.

Information on dental health of children at age five years and in Form Two (approximate 12 years of age) is collected annually by Crown Health Enterprise school dental services. In 1996 (the most recent year for which data are available), the average number of missing or filled primary teeth (mft) in five-year-old children was 1.7 per child. Fifty-five percent of five-year-olds were caries-free. In Form Two children, the average number of missing or filled permanent teeth (MFT) was 1.5 per child, and 45 percent were caries-free.

Changes over time

The dental health of children in New Zealand has been improving steadily for many years. Form Two MFT scores declined at approximately 8 percent per year from 1980 until the early 1990s, then rose slightly (Figure 13.4.1). The same trend is mirrored in the proportion of Form Two children with no caries. This proportion rose steadily from 1980 until the early 1990s, then declined after 1994.

Figure 13.4.1: Dental health of Form Two children, 1980–96



Source of data:
Ministry of Health 1997c.

Note:
* Missing or filled permanent teeth.

Possible explanations for the recent increase in caries in Form Two children include:

- greater use of radiography, which has enhanced sensitivity for detecting caries, by school dental services (Ministry of Health 1997c)
- increasing socioeconomic marginalisation of substantial sections of New Zealand society resulting in higher levels of disease (Thomson 1997).

International comparisons

As in New Zealand, child oral health has improved in most developed countries over the last quarter century (Newbrun 1992; Bjarnason et al 1993; Li et al 1993; Downer 1995; Nadavosky and Sheiham 1995). Improvements in the prevalence of dental caries in 12-year-old children has been greater in New Zealand than in most OECD countries. In the early 1970s, New Zealand ranked only 10th best out of 11 OECD countries for which data were available. By the early 1990s, New Zealand ranked fourth best out of 26 OECD countries that provided data (OECD 1997).

Risk and protective factors

Water fluoridation

In areas supplied with reticulated drinking water, water fluoridation is the most effective and efficient means of reducing dental caries (WHO 1994). A review of research in New Zealand as well as the United States, Australia, Britain, Canada, and Ireland concluded that water fluoridation:

- reduces dental caries by 30–60 percent in the primary teeth of children
- reduces caries by 20–40 percent in the mixed dentition in ages 8–12 years
- reduces caries by 15–35 percent in adolescents aged 14–17 years (Newbrun 1989).

Caries prevalence increases within a few years in communities where water fluoridation is discontinued (Stephen et al 1987; Attwood and Blinkhorn 1991; Ripa 1993).

In New Zealand, Form Two children who lived in areas supplied with fluoridated water had 26 percent fewer MFT than those in areas without fluoridated water, in 1996. At age five years, the difference was 31 percent. This is an underestimate of the protective effect of fluoride since children whose home water supply is not fluoridated have normally gained at least some degree of protection against dental caries by exposure to fluoride via:

- fluoridated water away from home (for example, at school)
- fluoridated water supplied to a previous home residence
- fluoride toothpaste
- fluoride tablets
- topical fluoride treatment by dentists and dental therapists
- dietary sources of fluoride.

Exposure to fluoride via drinking water has an advantage over other sources in that high fluoride exposure is less likely. High exposure to fluoride can cause dental fluorosis (mottling of tooth enamel).

Water fluoridation is of greatest benefit to those with poorer dental health, including Māori and lower socioeconomic groups. Thus, water fluoridation contributes to equity of health outcomes (PHC 1994b). The estimated effectiveness of water fluoridation in preventing decayed, missing or filled primary teeth (dmft) in five-year-olds in New Zealand is shown in Table 13.4.1 (Treasure and Dever 1992).

Table 13.4.1: Protective effect of water fluoridation against dental caries in 5-year-old New Zealand children, by socioeconomic group

<i>Socioeconomic group*</i>	<i>dmft** prevented per child</i>
Groups 1 and 2 (least socioeconomic disadvantage)	0.2
Groups 3 and 4	2.5
Groups 5 and 6 (greatest socioeconomic disadvantage)	3.5

* Determined by parental socioeconomic status (Elley and Irving 1985).

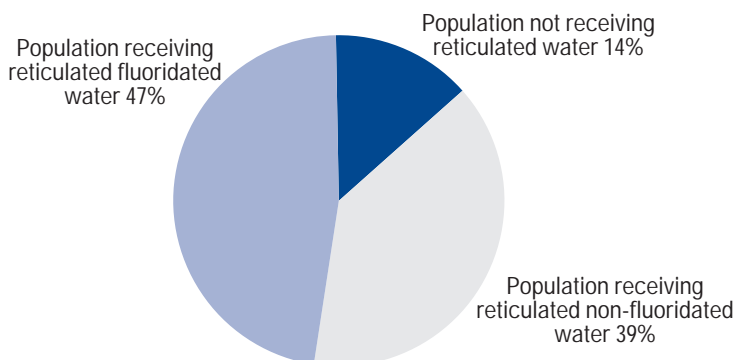
** Decayed, missing or filled primary teeth.

Source of data:

Treasure and Dever 1992.

Nearly half of all New Zealanders were supplied with reticulated fluoridated water in 1996 (Ministry of Health 1997c, Figure 13.4.2).

Figure 13.4.2: Population receiving fluoridated and non-fluoridated reticulated water, and population not receiving reticulated water, 1996



Source of data:

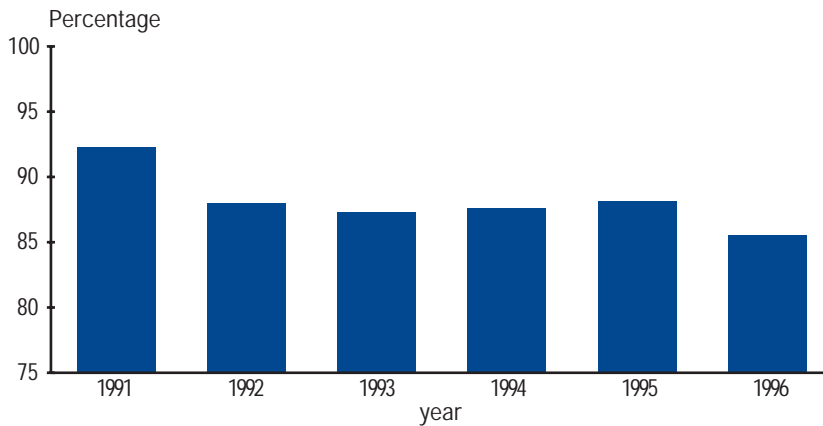
Ministry of Health 1997c.

Fluoride toothpaste

Regular use of fluoride toothpaste is an effective method of reducing dental caries. For people whose drinking water is fluoridated, regular use of fluoride toothpaste provides an additional degree of protection against tooth decay. In non-fluoridated areas, it is the prime method of preventing decay (Murray et al 1991).

The percentage of toothpaste sold that contains fluoride decreased from 92 percent in 1991 to 86 percent in 1996 (Figure 13.4.3).

Figure 13.4.3: Percentage (by weight) of fluoride toothpaste out of all toothpaste sold in New Zealand, 1991–96



Source of data:
Ministry of Health, 1997c.

Other factors

Apart from water fluoridation and fluoride toothpaste, other factors known to be associated with higher prevalence of dental caries include Māori ethnicity, low socioeconomic status (NHMRC 1991; Treasure and Dever 1992), and frequent or high dietary intake of simple sugars (Glinsmann et al 1986).

Separate MFT data for Māori children have been reported for some New Zealand regions (Ministry of Health 1997c). These indicate that Form Two Māori children had, on average, 60 percent more MFT than non-Māori in 1995. A study in the Manawatu-Wanganui Area Health Board showed that at age five years Māori children were one-third as likely to be caries-free as non-Māori, and were three times more likely to have high (five or more MFT) caries experience than non-Māori children (Thomson 1993). Māori children also had higher caries prevalence at Form Two (approximately age 12 years). Prevalence of caries in Pacific children lay between those for Māori and non-Māori children.

Dental caries is a disease with a strong socioeconomic gradient, with those most disadvantaged having greater disease experience. Such a gradient has been found in five out of six Australian studies since 1980 (NHMRC 1991), and also in New Zealand (Treasure and Dever 1992).

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