Part IV

Scope for Health Gain: The Potential for Health Improvement

The first three parts of this report have described the historical trends and the current status of population health in New Zealand, using a variety of indicators. First, the health of New Zealanders was analysed in terms of their experience of mortality – the quantity of life dimension (Part I). The indicators used included all-cause and cause-specific mortality rates and probabilities of survival, life expectancy at different ages, and years of life lost as a result of presenescent and premature death.

Then, the quality of life dimension was analysed, in terms of subjective health, disability, and morbidity (Part II). The indicators used included the global self rated health item, the SF-36 profile and physical and mental health component summary scores, prevalence rates of disability by type and severity, and the incidence and prevalence of specific diseases and injuries.

In Part III, integrated indexes of health that combine both quantity and quality of life dimensions into single measures of health were constructed. The first of these involved generalising life expectancy to *health* expectancy. The specific health expectancy indicator analysed was independent life expectancy, derived by integrating mortality with (dependent) disability rates. The second indicator was a health gap measure, the DALY, derived by generalising years of life lost to years of *healthy* life lost.

Part IV examines the scope that still remains to improve on current levels of health, as measured using these indicators. This analysis is intended to provide a context or framework within which detailed analyses of the costs, benefits and risks of specific interventions can be undertaken, and decisions made about targeting resources to specific population groups.

Three different yet complementary approaches are used to do this. The first approach is *benchmarking*: comparing current levels of health with those already achieved by some reference group. Information already presented in earlier chapters on intra- and (especially) international comparisons are summarised and extended. International comparisons are subject to bias from non-standard definitions and varying data collection methods, and for this reason have been restricted to mortality-based outcomes only. Such data quality problems do not apply to intra-national comparisons, where examination of socioeconomic and ethnic gradients in health are particularly informative.

Benchmarking approaches necessarily underestimate the scope for health gain, since they assume that the reference group is itself incapable of further progress – that is, they lack an absolute standard of comparison and are subject to the 'moving goalposts' problem. Benchmarking may be more useful at the intra-national level, where subgroup comparisons serve as an equity tool rather than as a health gain measure, identifying priority populations for targeting.

The second approach used is based on categorical attribution of causes. Building on previous work by Charlton and others, all ICD codes have been categorised as 'avoidable' or 'unavoidable', based on their responsiveness to currently available health sector interventions. These codes are further subcategorised based on the level of intervention involved, so allowing the potential contribution of different health sector strategies to population health gain to be quantified. As is conventional in this field, an arbitrary upper age limit is imposed (75 years here), beyond which health outcomes are not considered avoidable. This simply reflects the difficulty of assessing avoidability in old age, and inevitably leads to underestimation of the true scope for health gain.

The third approach taken moves beyond direct causes of health outcomes – the specific diseases, injuries or other morbid processes involved – to more remote causes: *risk factors* or determinants of health. By understanding the hazard functions linking causes to outcomes, and the exposure of the population to these causes, the impact of reduction in these exposures can be modelled by counterfactual analysis (that is, by estimating population attributable risks (PARs)). At present, data limitations restrict this approach to simple univariate analyses of behavioural and physiological risk factors. However, it should be emphasised that the social context constrains lifestyle choices and shapes physiological responses.

For this report, the most recent estimates of the prevalence of major lifestyle and physiological risk factors have been extracted from the 1996–97 health and nutrition surveys (and other sources when necessary). These are combined with the best estimates of the relative risks (hazard ratios) of these exposures for different health outcomes (mainly mortality) available from local and relevant overseas studies, to produce estimates of current PARs for New Zealand. These univariate PARs over-estimate the health gain that would be achieved were exposure to each risk factor to be reduced, because they do not allow for interaction or clustering among risk factors. Nevertheless, even this simplified and decontextualised analysis provides an estimate of the remaining scope for health gain through modification of risk exposures.

Chapter 9: Benchmarking health

Introduction

This chapter locates the health status of New Zealanders in an international context by comparing selected health indicators for New Zealand with those of other OECD nations. This analysis is intended to estimate the scope for improving health outcomes, assuming that New Zealand has the potential to match the best health outcomes found currently in other economically advanced countries.

Eleven mortality based health outcomes are compared. They include life expectancy at birth and at age 65, all-cause mortality rates, perinatal and infant mortality rates, mortality rates for selected conditions (such as ischaemic heart disease) and measures of potential years of life lost (YLL). Mortality based measures have been used, as these data are compiled in a standardised way across most of the OECD countries. This is in contrast to morbidity, disability and quality of life outcomes, data for which tend to be collected, classified or reported in different ways in different countries, thus making valid inter country comparisons problematic.*

Data sources and methods

The international health statistics that form the basis of this chapter have been extracted from data collections compiled by the OECD and WHO. In some cases, relevant data have been extracted directly from these collections; in others, data have been obtained from recently published reports that draw upon and extend these collections (de Looper and Bhatia 1998; Anderson and Poullier 1999). For most indicators the most recent comparative data available are for 1994–96 (OECD 1999).

Recent data for New Zealand, including gender and ethnic subgroups, are compared with the latest available data from 22 other economically advanced countries (for the three year period centred around the year to which the New Zealand data refers). Where reliable data exist, trends over time are considered. The main comparison countries used for trend analysis are Australia, Canada, Britain and the United States – all predominantly English speaking countries with living standards and political, economic and social systems similar to those of New Zealand. These international comparisons are the benchmarks used to assess the scope for health gain in this country.

It should be noted that population health outcomes are highly sensitive to the level of economic development of a country, but this relationship attenuates rapidly as the level of economic development increases (World Bank 1993). Thus comparison of population health status in New Zealand with that in non-industrialised countries would be of little interest. However, when comparing New Zealand's health outcomes with those of other industrialised countries, differences cannot simply be attributed to differences in per capita gross domestic product (Wilkinson 1996).

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* One non-mortality based outcome (self rated health status) is compared for selected OECD countries in Chapter 4. These data are not repeated here.

Life expectancy

In 1995–97 New Zealand males and females had a life expectancy at birth of 74.3 and 79.6 years respectively. This positioned New Zealand in the middle of the range of male life expectancies and in the lower range of female life expectancies in the OECD (Figure 133a). When life expectancy at age 65 is examined, New Zealand ranks close to the median of the OECD countries compared, for both genders (Figure 133b).

Figure 133a: Life expectancy at birth, New Zealand and selected OECD countries, 1995–97

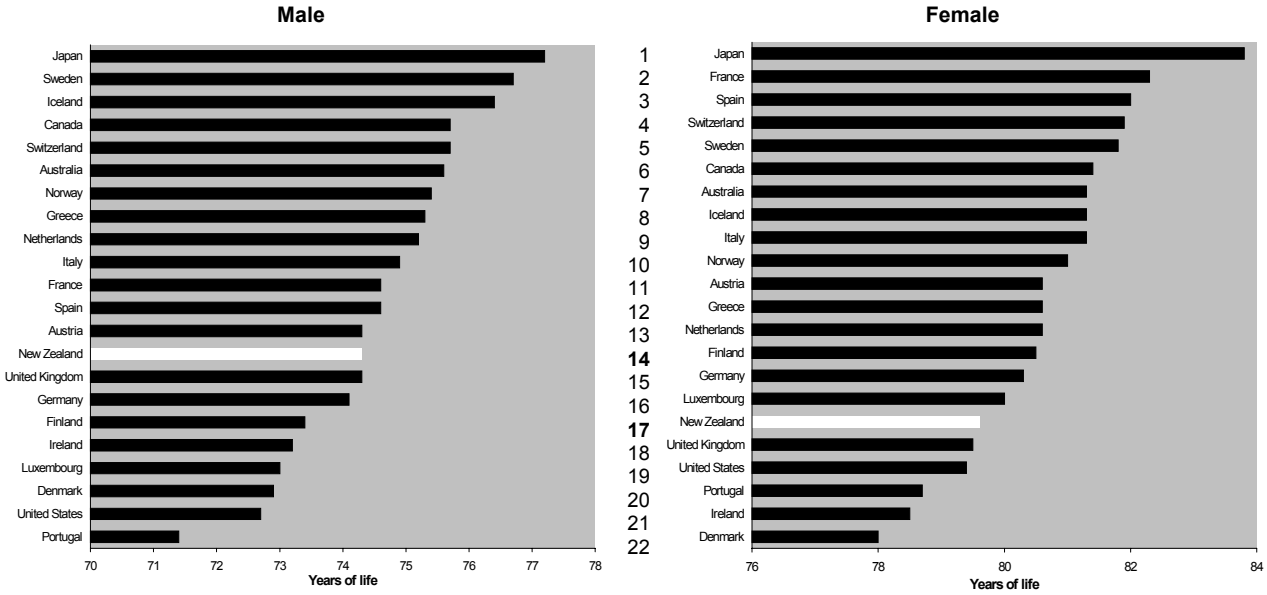
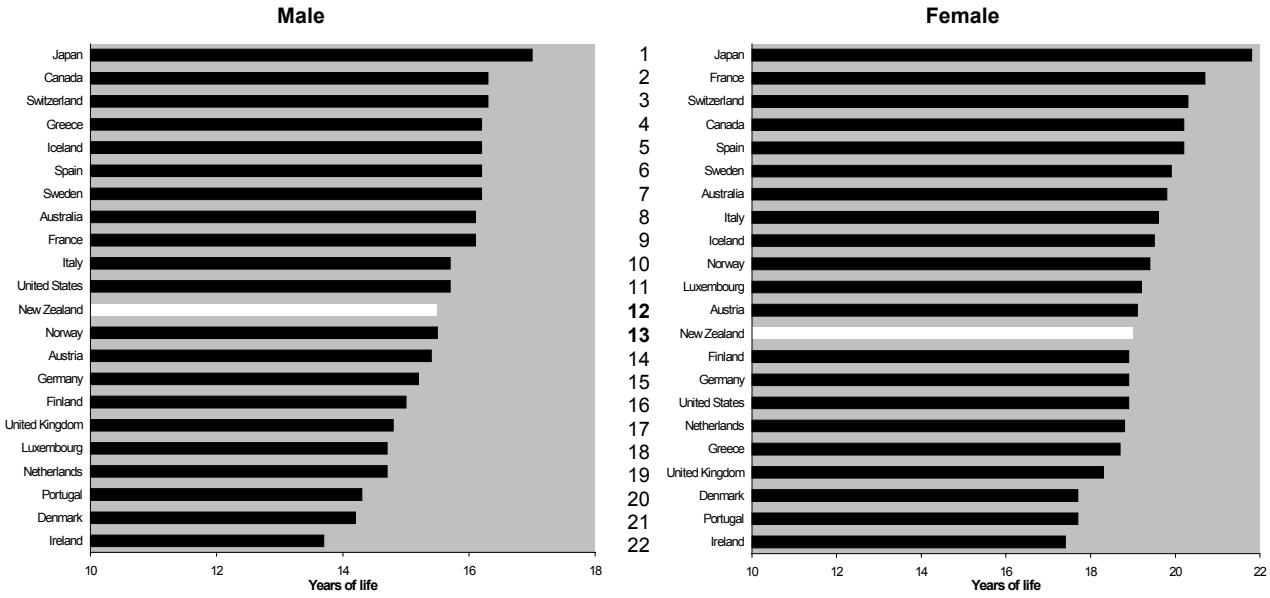


Figure 133b: Life expectancy at age 65, New Zealand and selected OECD countries, 1995–97



Source of base data: OECD
Note: scale varies between charts; broken axes.

The country recording the highest male and female life expectancies at birth and at age 65 in recent years is Japan. In 1996 Japanese males at birth could expect to live on average nearly three years longer than New Zealand males, and Japanese females had a life expectancy almost four years longer than New Zealand females.

In New Zealand, as in all OECD countries, males have a lower life expectancy than females. Males born in 1995–97 are likely to die 5.3 years earlier than females born in the same period. This gender gap in life expectancy is not as wide as in many other countries – for example, Finland and France, where men die between 7.1 and 7.7 years earlier than women. But neither is the gap as small as that found in Iceland or Sweden, where men die on average only about 5.0 years earlier than women.

At the intra national level, Māori have considerably lower life expectancies than the rest of the New Zealand population. In 1995–97 life expectancy for Māori males was about eight years lower than for non-Māori males (67.2 years versus 75.3 years), and Māori females had a life expectancy nine years lower than non-Māori females (71.6 years versus 80.6 years). The indigenous peoples of Australia also have life expectancies markedly lower than those of other Australians. In the period from 1991 to 1996, Indigenous Australian life expectancy at birth for males was estimated to be 57 years and for females 62 years (AIHW 1998), 18 to 19 years lower than the rest of Australia's population. In the United States, non-Hispanic whites have higher life expectancies at birth than non-Hispanic African Americans. In 1996 the life expectancy for white males and females was 73.9 and 79.7 years respectively, compared with 66.1 and 74.2 years for African American males and females, a gap of over seven years between white and black males and a gap of over five years between white and black females (National Center for Health Statistics 1998). This gap in life expectancy at birth is smaller than the gap between Māori and non-Māori for both genders.

Trends over time

As in other OECD countries, life expectancies have improved significantly for New Zealanders in recent decades. Compared with males and females born in 1950, males born in the mid 1990s enjoy about an extra seven years and females an extra eight years of life expectancy. These gains have been achieved through reductions in mortality rates at most ages, including substantial falls in mortality in the 65 and older age group over the past two decades.

In the last 20 years the life expectancies of New Zealand males and females have not improved as rapidly as in countries such as Australia, Canada, Britain and the United States, however (Figures 134 and 135). In Australia, the country considered to be most similar to New Zealand, male and female life expectancies at birth are now respectively 1.3 and 1.7 years higher than the New Zealand levels. This is in sharp contrast to the 1960s and early 1970s, when Australian male and female life expectancies were lower than those in New Zealand.

Figure 134a: Life expectancy at birth in New Zealand and similar countries, 1960–97

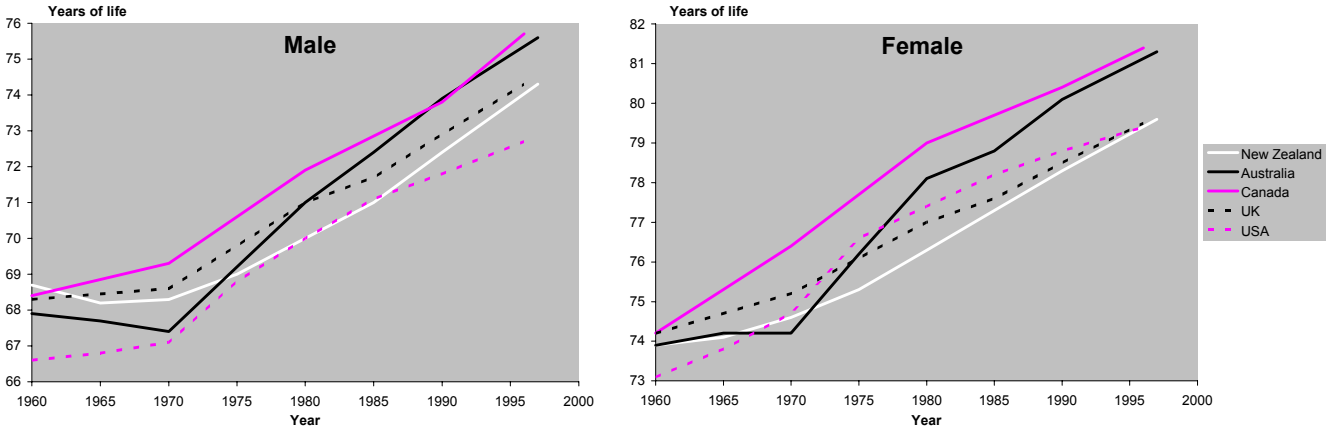
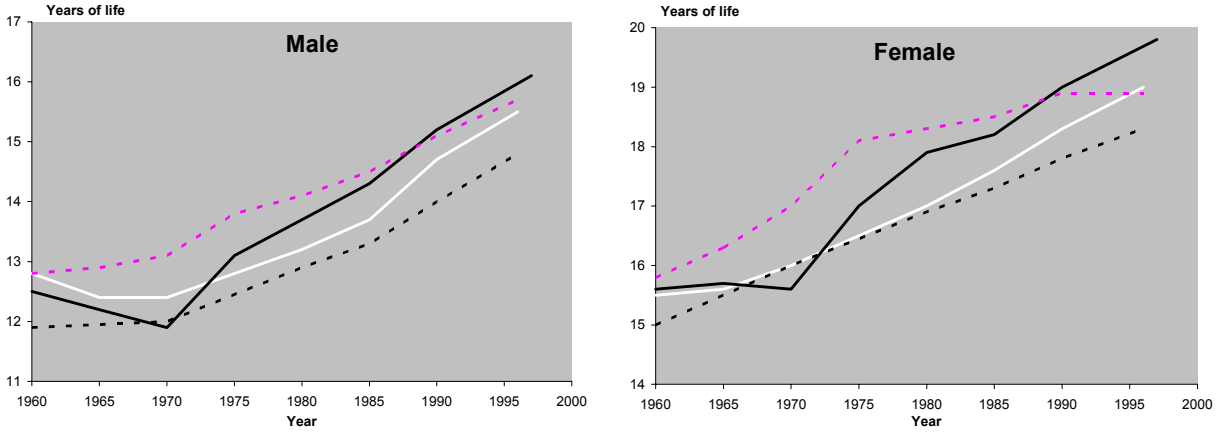


Figure 134b: Life expectancy at age 65 in New Zealand and similar countries, 1960–97



Source of base data: OECD
Note: scale varies between charts; broken axes.

Figure 135a: Change in life expectancy at birth in New Zealand and similar countries, 1960–97

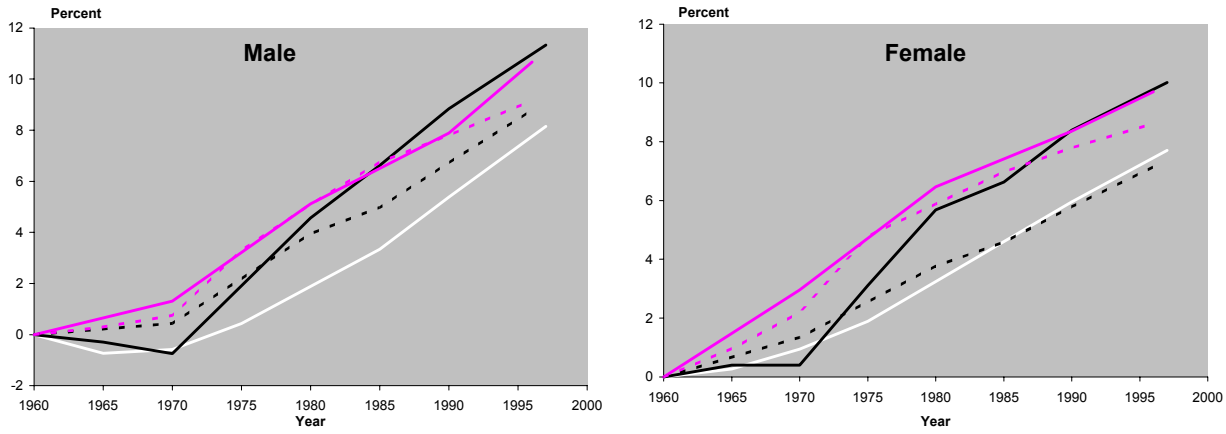
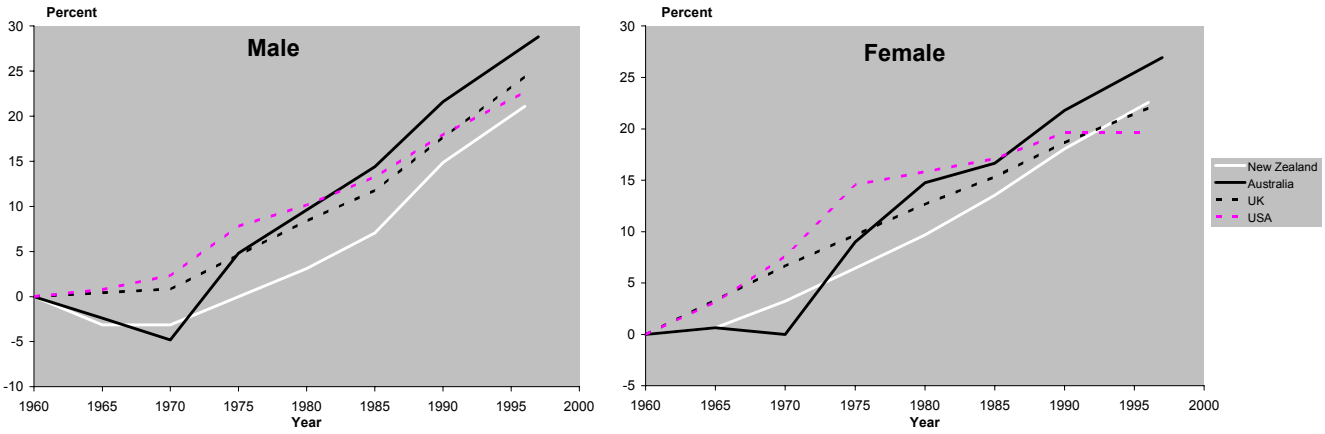


Figure 135b: Change in life expectancy at age 65 in New Zealand and similar countries, 1960–97



Source of base data: OECD
Note: scale varies between charts, broken axes.

Probabilities of survival

Another indicator that can be obtained from a life table is the probability of dying within any age interval. The probability of dying between ages 15 and 59 (the probability of not reaching one's 60th birthday, having reached one's 15th birthday) for 19 economically advanced countries in 1998 has been estimated by WHO (WHO 1999) (Table 77).

Table 77: Probability of dying between ages 15 and 59, by gender, selected OECD countries, 1998

Country	Male (%)	Female (%)	All (%)
Japan	9.9	5.0	7.5
Greece	11.0	4.9	8.0
Sweden	9.7	6.3	8.0
Italy	10.8	5.4	8.1
Australia	10.4	5.9	8.1
Israel	10.2	6.1	8.2
Norway	10.7	5.9	8.3
Netherlands	10.2	6.5	8.4
Canada	10.8	6.1	8.5
Switzerland	11.4	6.0	8.7
United Kingdom	11.0	6.9	9.0
Ireland	11.4	6.6	9.0
Spain	12.9	5.4	9.2
Singapore	11.8	7.8	9.8
Germany	13.2	6.6	9.9
New Zealand	**12.5**	**7.9**	**10.2**
France	14.5	6.3	10.4
United States	15.4	7.9	11.7
Denmark	14.1	9.6	11.9

Source of base data: WHO

Scope for health gain

Countries such as Japan demonstrate how high it is possible for life expectancy at birth to reach. In 1995–97 Japanese life expectancy at birth was three years higher than non-Māori and 11 years higher than Māori for males, and four and 13 years higher respectively for females. It is of interest that the analysis of life expectancy by socioeconomic status (NZDep96 decile) reported in Chapter 2 shows that European/Other New Zealanders living in the least deprived areas have life expectancies at birth very close to those currently achieved by the Japanese population as a whole.

Mortality rates

Age standardised mortality rates provide another way of summarising the age specific mortality rates experienced by a population in a given year. Unlike life table methods, however, age standardisation requires the arbitrary selection of a standard or reference population, and some distortion of the data is inevitable as a result. The values of the rates calculated, and even the precise ranking of countries, will vary depending on which population is selected as the standard. However, disaggregating mortality rates by cause is straightforward, which is an advantage over life table methods. The data shown here have been standardised to the OECD reference population, which is the total OECD population in 1980.*

In 1994, the latest year for which age standardised mortality data are available for the 22 comparison countries shown in Figure 136, New Zealand's female all-cause mortality rate of 572 per 100,000 ranked 17th, just above the United States and Britain. Japan's female all-cause mortality rate was 400 per 100,000, the best in the group and 30 percent lower than New Zealand's rate.

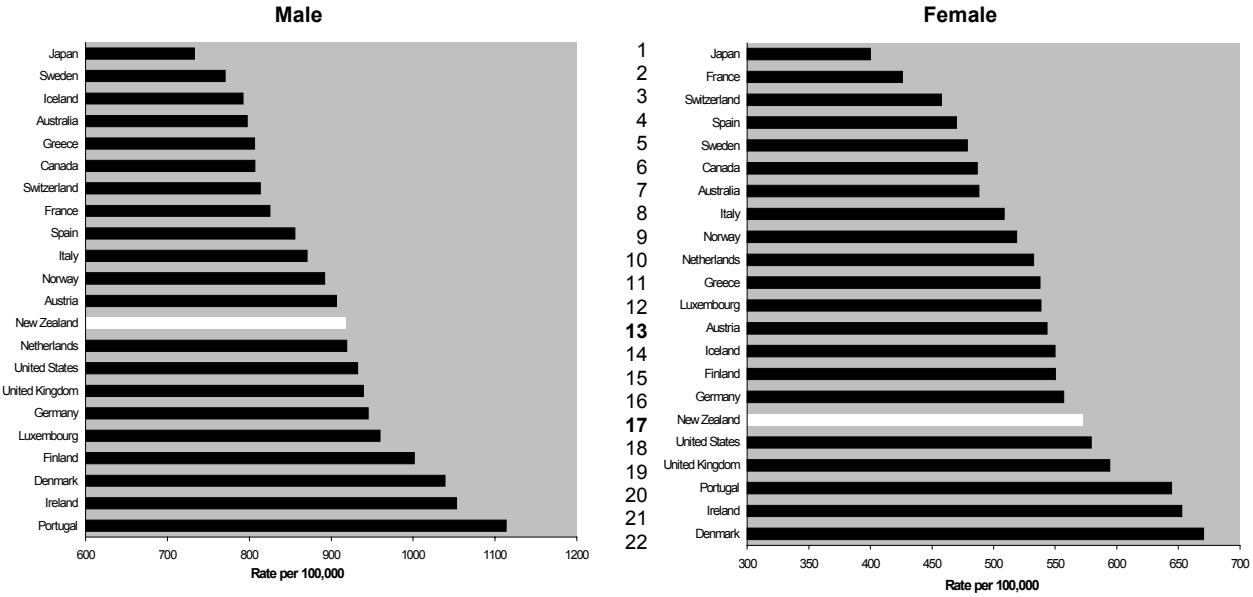
In the same year, New Zealand had an all-cause mortality rate for males of 918 per 100,000, placing it 13th, again above the United States and Britain. Japan had the lowest male all-cause mortality rate (733 deaths per 100,000 males), 20 percent lower than the New Zealand rate.

Of the countries in the group most similar to New Zealand, Canada and Australia had lower all-cause mortality rates, with Australia's rate being 13–15 percent lower than New Zealand's (for males and females respectively). New Zealand, Britain, the United States, Canada and Australia all had rates of male all-cause mortality that were 58 to 66 percent higher than the corresponding female rates. Some of the widest gender disparities in all-cause mortality in recent years have been recorded in Japan and France, where, by international standards, females have especially low all-cause mortality rates.

In comparison, in 1996 Māori had an age standardised all-cause mortality rate 1.9 times that of European/Other New Zealanders for males (1047 per 100,000 and 564 per 100,000 respectively, standardised to Segi's world population) and 2.1 times for females (754 per 100,000 and 361 per 100,000 respectively). In the same year, Pacific males and females had corresponding rate ratios of 1.6 for both genders (standardised rates of 903 per 100,000 and 573 per 100,000 respectively).

* The OECD database does not include the age specific data necessary to re-standardise against other reference populations; note that these rates are very different numerically from other rates presented in this report, which have been age standardised to Segi's world population.

Figure 136: All-cause mortality, New Zealand and selected OECD countries, 1994



Source of base data: OECD
Note: rate is age standardised to the OECD reference population; scale varies between charts; broken axes.

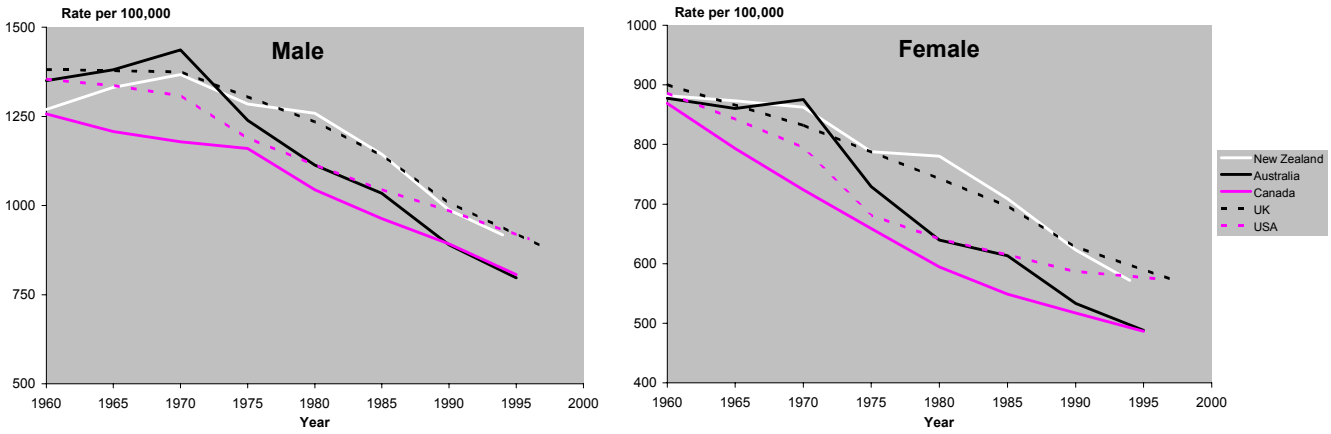
In Australia, Aboriginal and Torres Strait Island people have all-cause mortality rates more than three times as high as the rest of the population. Mortality rates for these two ethnic groups have not declined significantly in recent years, unlike those for the non-Aboriginal population (AIHW 1995). In 1993, for Aboriginal people, the male age standardised mortality rate was 1480 per 100,000 and the female rate was 1180 per 100,000 (standardised to the 1991 Australian population). These rates are very likely to be underestimates, as Aboriginal people are not always identified as such on death certificates, especially in the states of New South Wales and Victoria.

In the United States in 1994–96, African Americans had an age standardised (to the 1940 USA population) mortality rate of 759 per 100,000, more than 1.5 times higher than the rate for white people (National Center for Health Statistics 1998). In all these countries, indigenous peoples and ethnic minorities have much higher all-cause mortality rates than the majority European/ Other population.

Trends over time

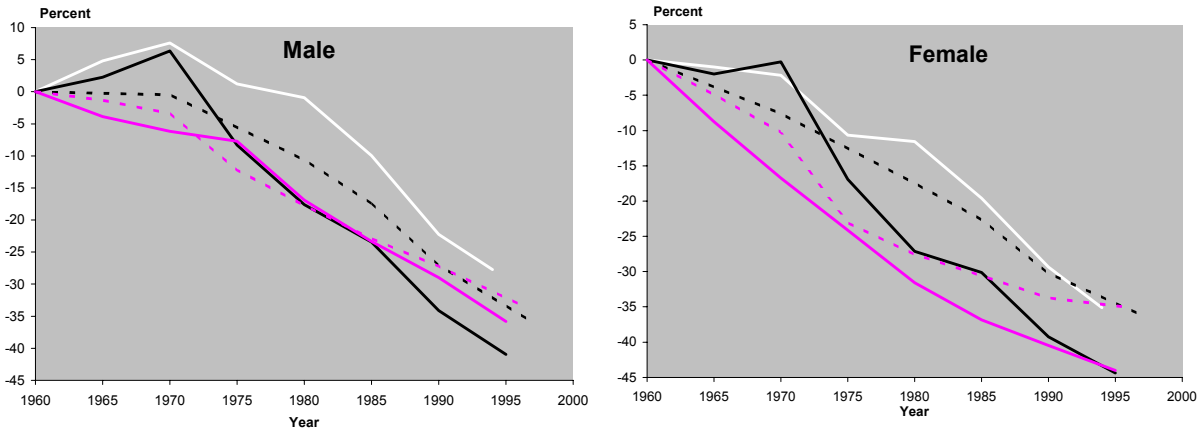
From 1960 to 1994, male all-cause mortality rates generally declined in all OECD countries, including New Zealand, although in New Zealand and Australia the rate initially increased throughout the 1960s. New Zealand's overall 28 percent reduction in male all-cause mortality was not as large as Canada's or Australia's, where male mortality declined by 41 percent and 36 percent respectively over this period (Figures 137a and 137b). New Zealand's female all-cause mortality rates also declined from 1960 to 1994, falling by 35 percent. This was a greater rate of improvement than the male trend, but was still modest compared to the 43–44 percent reduction in all-cause mortality experienced by Canadian and Australian females.

Figure 137a: All-cause mortality in New Zealand and similar countries, 1960–96



Note: rate is age standardised to the OECD reference population; scale varies between charts; broken axes; data shown are the most recent published for each country (OECD 1999).

Figure 137b: Change in all-cause mortality rates in New Zealand and similar countries, 1960–96



Source of base data: OECD
Note: scale varies between charts; broken axes; data shown are the most recent published for each country (OECD 1999).

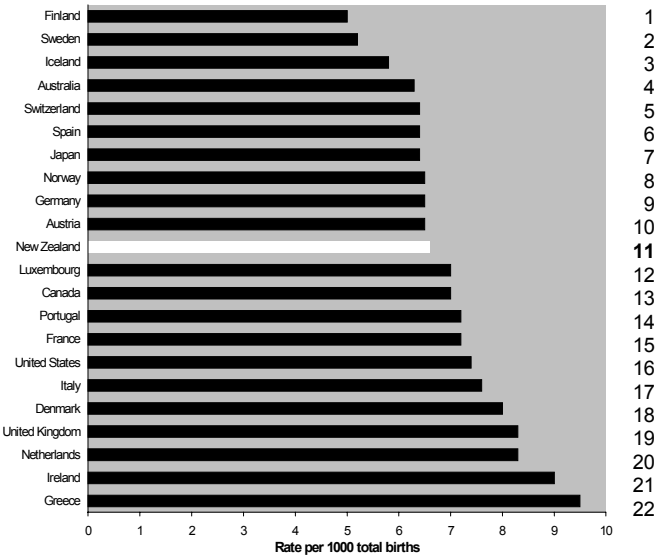
Scope for health gain

Japanese males and females have some of the lowest recorded all-cause mortality rates in the world. If in future the New Zealand population could attain mortality rates similar to those recorded in recent years in Japan, New Zealand's male all-cause mortality rate would fall by 20 percent and its female all-cause mortality rate by 30 percent. New Zealand European/Others would experience a 9 percent mortality reduction for males and a 22 percent reduction for females. Similarly, if the mortality rates for Pacific people matched those of the Japanese population, Pacific males and females would experience 43 percent and 51 percent lower mortality respectively. If Māori mortality rates matched those of the Japanese population, Māori males and females would experience 51 percent and 63 percent reductions in mortality respectively.

Perinatal mortality

The perinatal period begins at 28 weeks' gestation and ends seven days after birth. In 1996, the latest year for which comparable international statistics are available, New Zealand had a rate of 6.6 perinatal deaths per 1000 total births (Figure 138). The average for the 22 OECD countries in 1996 was 7.0 per 1000, with the highest perinatal mortality rates (9 or more per 1000) recorded in Greece and Ireland.

Figure 138: Perinatal mortality in New Zealand and selected other OECD countries, 1996



Source of base data: OECD

A comparison of ethnic specific perinatal mortality rates in New Zealand shows little difference between ethnic groups (NZHIS 1999a).

In 1996 reporting of perinatal mortality in New Zealand was extended to include deaths in the 20th to the 27th week of gestation. Using this extended definition, the perinatal mortality rate in New Zealand in 1996 was 10.2 per 1000 total births (NZHIS 1999a). However, for international comparisons the older definition must continue to be used.

Trends over time

Since 1960, when the perinatal mortality rate was 27.0 deaths per 1000, there has been a reduction of nearly 80 percent in the New Zealand rate (Figures 139a and 139b). Similar levels of improvement occurred in Australia, Canada, Britain and the USA, although in the mid 1990s perinatal mortality rates in the three latter countries were not quite as low as those in Australia and New Zealand.

Figure 139a: Trends in perinatal mortality in New Zealand and similar countries, 1960–96

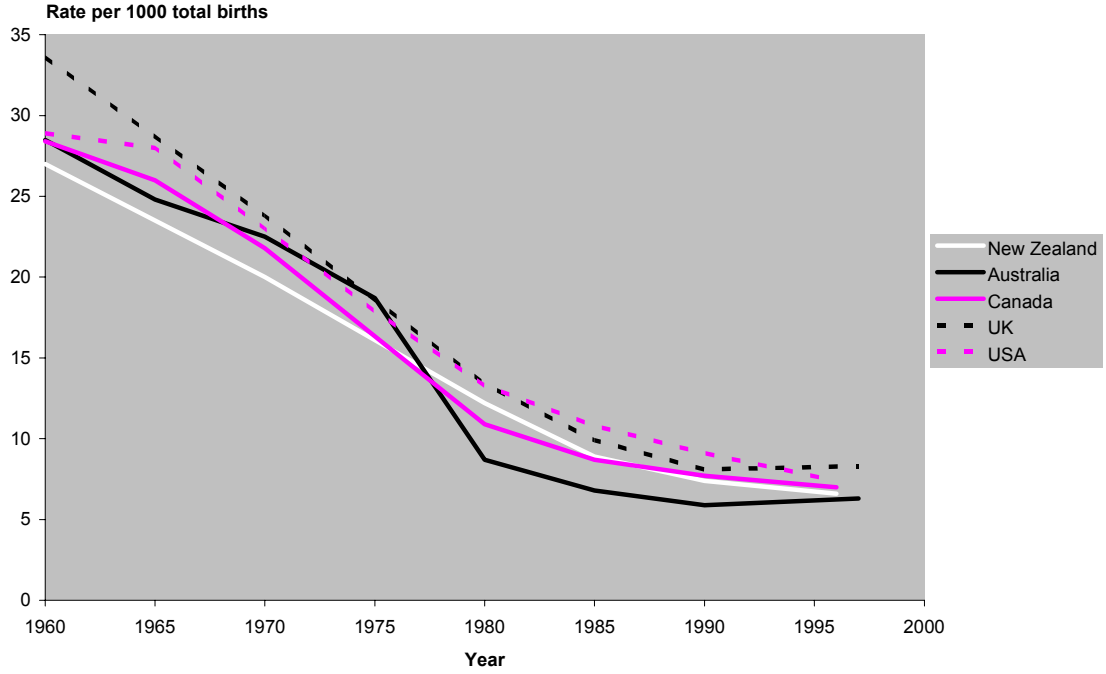
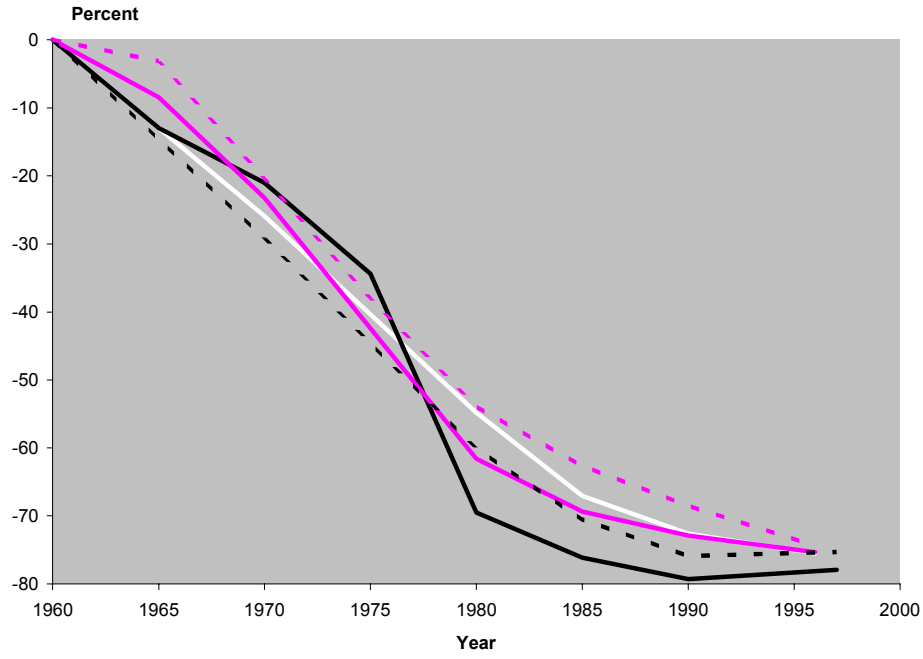


Figure 139b: Change in perinatal mortality rates in New Zealand and similar countries, 1960–96



Source of base data: OECD
Note: data shown are the most recent published for each country (OECD 1999).

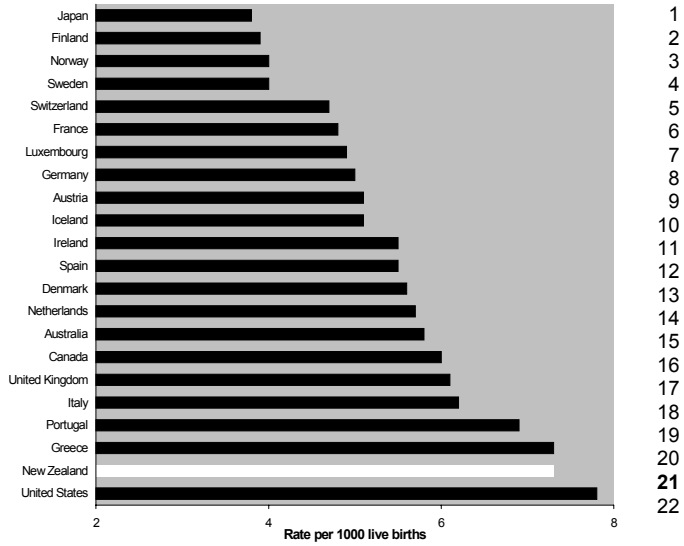
Scope for health gain

Australia's lowest recorded perinatal mortality rate in the early 1990s was 5.4 deaths per 1000. To achieve this level, New Zealand's perinatal mortality rate would need to improve by approximately 18 percent.

Infant mortality

Infant mortality has long been used as a yardstick for comparing the health status of children across different countries. In 1996, the latest year for which infant mortality data are available for the OECD countries, New Zealand's infant mortality rate was 7.3 deaths per 1000 live births (although the rate has since improved to 6.7 per 1000 in 1997 and 5.7 per 1000 in 1998 (provisional data for 1997 and 1998)). In 1996 New Zealand ranked 21st out of 22 OECD countries, above only the United States (Figure 140). The average infant mortality rate for the 22 countries was 5.4 deaths per 1000 live births, 24 percent lower than New Zealand's rate. Japan and Finland had the lowest infant mortality rates in the group, at under 4.0 deaths per 1000 live births.

Figure 140: Infant mortality in New Zealand and selected other OECD countries, genders pooled, 1996



Source of base data: OECD
Note: broken axis.

In 1996 Māori infants were more than twice as likely to die as European/Other infants (11.6 deaths per 1000 compared with 5.3 deaths per 1000). The infant mortality rate for the Pacific population varies from year to year since it is based on very small numbers; in 1996 it was 7.3 per 1000, about one third higher than the European/Other rate (NZHIS 1999a). Provisional rates for 1997 are 10.7 per 1000 and 8.8 per 1000 for Māori and Pacific infants respectively. The corresponding rates for 1998 are 9.5 per 1000 and 7.4 per 1000, indicating a significant decline at least for Māori infant mortality.

Trends over time

New Zealand's infant mortality rate has steadily improved over the last 40 years, down from the rate of nearly 23 per 1000 live births recorded in the early 1960s (Figure 141a). However, especially since the mid 1970s, the rate of improvement has not kept pace with that of many other countries and has slowed further in the 1990s at least until 1996 (Figure 141b). In 1996 Australia and Canada had infant mortality rates of 5.8 and 6.0 deaths per 1000 live births respectively, rates 15–18 percent lower than New Zealand's. Since 1996, however, New Zealand's infant mortality rate has again begun to fall sharply, improving to 6.7 per 1000 live births in 1997 and 5.7 per

1000 in 1998 (provisional data). A major contributor to this recent trend has been a sharp reduction in Māori SIDS mortality.

Figure 141a: Trends in infant mortality in New Zealand and similar countries, 1960–98

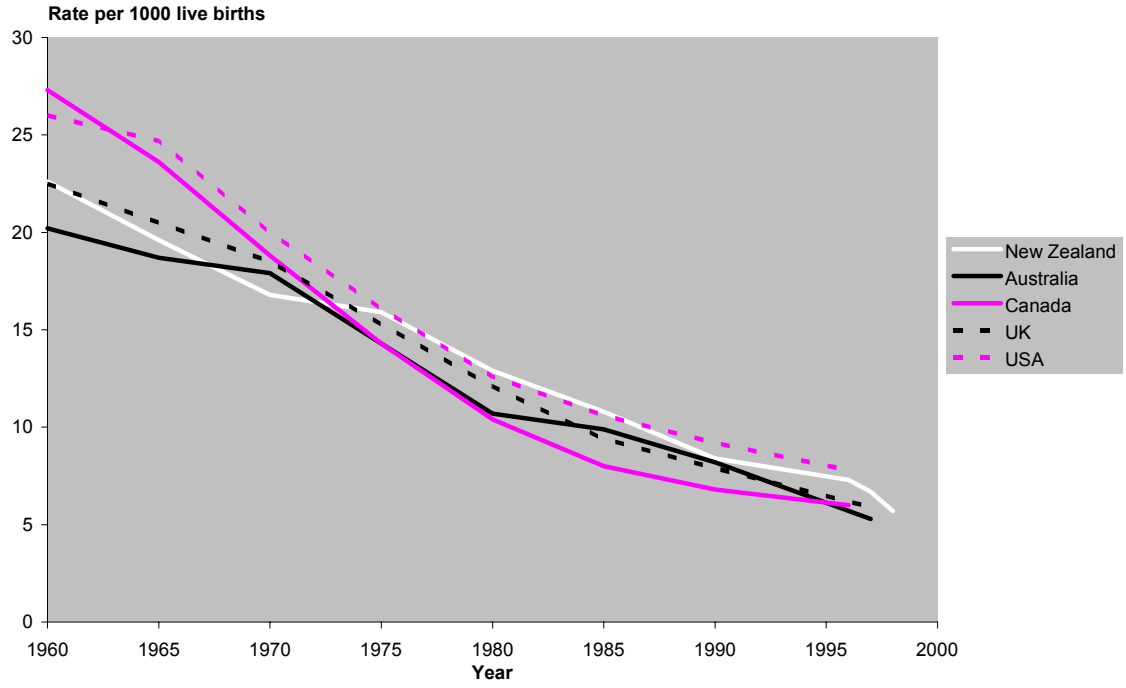
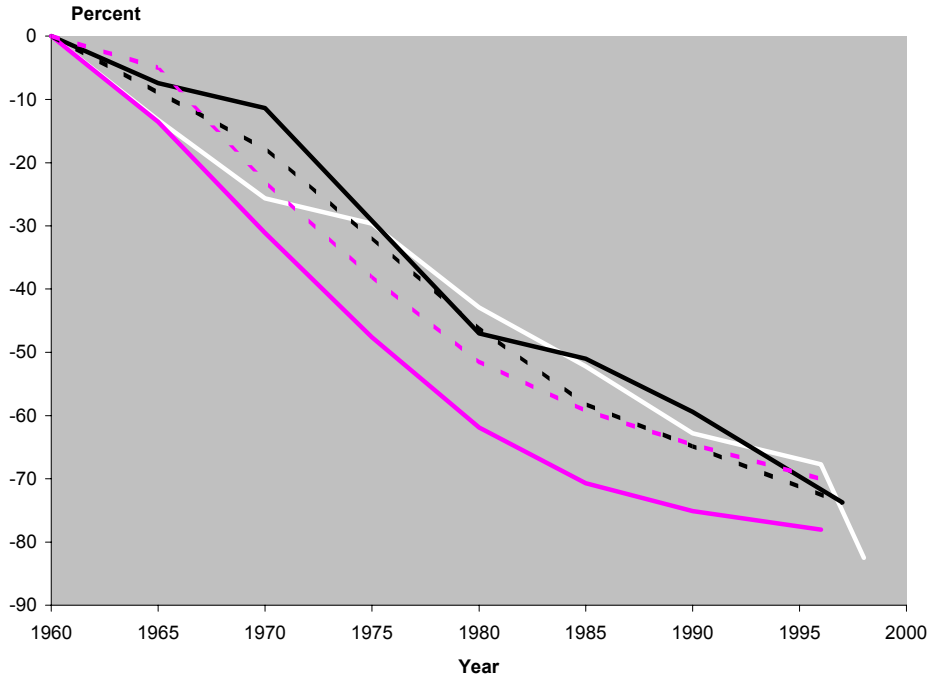


Figure 141b: Change in infant mortality rates in New Zealand and similar countries, 1960–98



Source of base data: OECD and NZHIS (New Zealand 1997 and 1998 data are provisional)

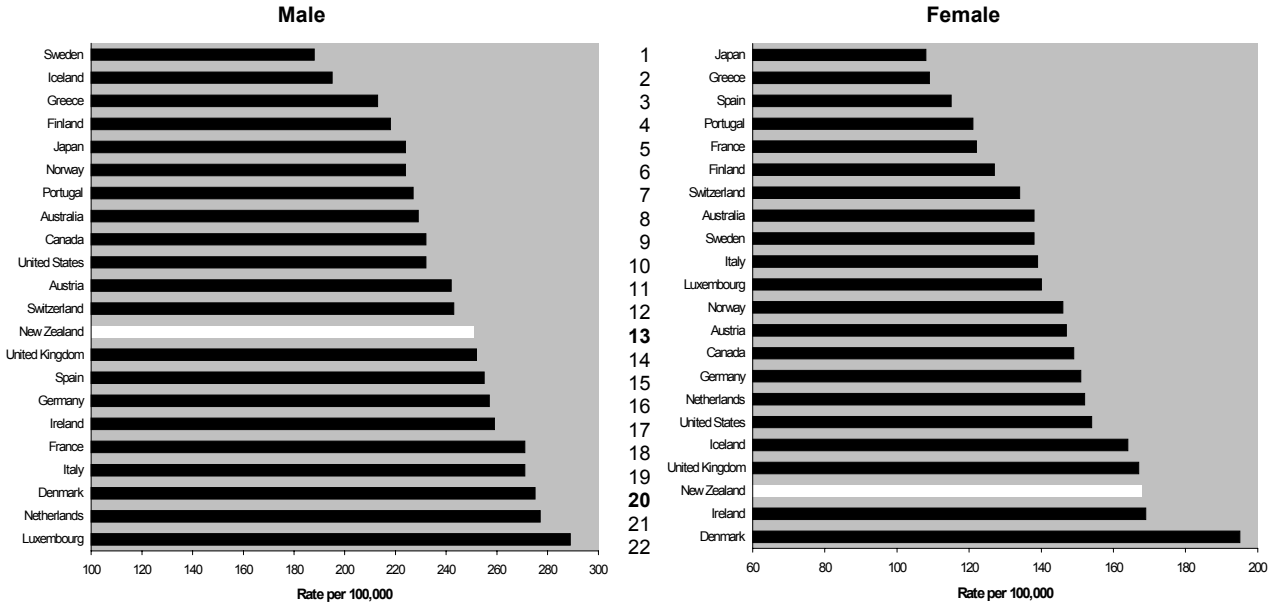
Scope for health gain

New Zealand's relatively high infant mortality rate is the result of high mortality in the post neonatal period (the period beginning 28 days after birth and ending at 12 months of age). Sudden Infant Death Syndrome (SIDS) is the leading cause of death in this period, with Māori infants making up two thirds of all infants who died from SIDS in 1996 (NZHIS 1999a). At least half of SIDS mortality may be attributable to parental smoking (Mitchell et al 1997). If New Zealand's 1996 infant mortality rate had been the same as Japan's (3.8 per 1000 live births), the number of infant deaths in that year would have nearly halved, saving approximately 200 infant lives. By OECD standards, New Zealand's Māori infant mortality rate is very high – over three times that of Japan, the country with the lowest infant mortality rate in 1996. The recent improvement in New Zealand's infant mortality rate since 1996 is partly the result of a significant reduction in SIDS among Māori infants.

Cancer

In 1993–95 New Zealand's male cancer mortality rate was near the median when compared with that of 21 other OECD countries (Figure 142). New Zealand's female rate was 20th, just below the United States and Britain and just above Ireland.

Figure 142: Cancer mortality in New Zealand compared with selected OECD countries, 1994



Source of base data: OECD
Note: rate is age standardised to the OECD reference population; scale varies between charts; broken axes.

The lowest cancer mortality rate for males was recorded in Sweden, 25 percent lower than New Zealand's. The lowest cancer mortality rate for females was recorded in Japan, 33 percent lower than the New Zealand rate. In 1993–95, New Zealand male and female cancer mortality rates were very similar to British rates. However, New Zealand cancer mortality rates were somewhat higher than those of Australia, Canada and (to a lesser extent) the United States, three countries that New Zealand might reasonably expect to match in terms of achieving reductions in cancer related mortality.

Comparing male mortality rates in these same five countries for different types of cancers shows that New Zealand males, like Australian males, had a relatively low rate of lung cancer mortality

in 1994 (de Looper and Bhatia 1998). However, New Zealand's male mortality rates for skin (including melanoma), prostate and colorectal cancer were higher than Canadian, British and American rates. New Zealand females had a lower lung cancer mortality rate than their counterparts in Canada, the United States and Britain, although not as low as Australian women. However, New Zealand females had the highest colorectal cancer and skin cancer mortality rates in the five countries, and the second highest breast cancer mortality rate.

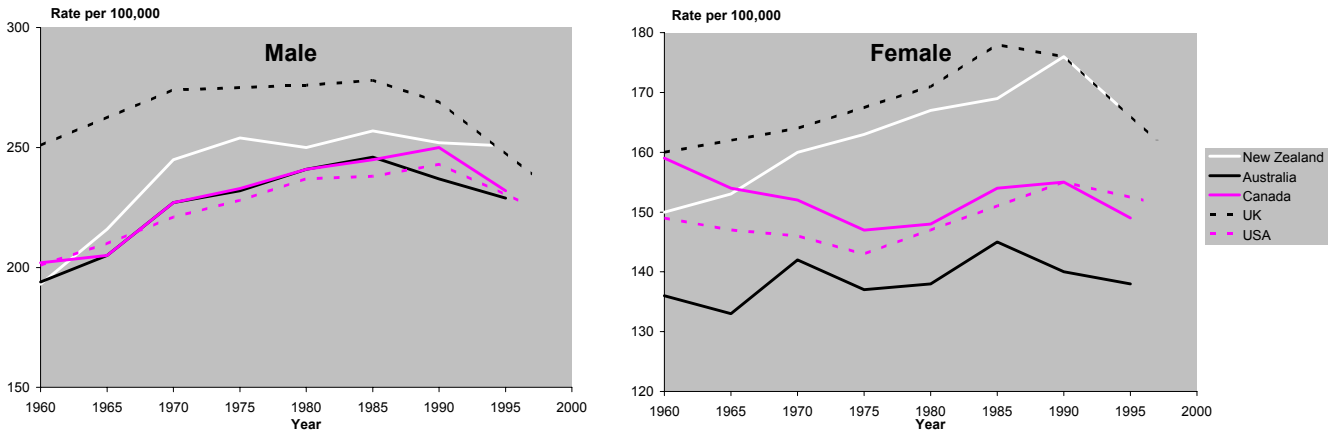
Examining cancer mortality across New Zealand's main ethnic groups, Māori women have a higher mortality rate from breast cancer than non-Māori women (32 deaths per 100,000 compared with 25 deaths per 100,000 in 1996, age standardised to Segi's world population). Māori men and women also have higher lung cancer mortality rates than European/Other and Pacific males and females. For some other types of cancers, non-Māori people have a higher rate of death than Māori. Melanoma incidence and mortality are dominated by European/Other people, and prostate cancer mortality is higher in non-Māori males than Māori males, especially in the 75 and over age groups. Colorectal cancer mortality is also higher in the non-Māori population.

Trends over time

Across the OECD countries, death rates for cancer have generally increased among males in the 35 years since 1960 (Figure 143a). Reflecting this trend, New Zealand's male cancer mortality rate increased by 30 percent (after adjusting for age), although most of this increase occurred in the first half of the period (Figure 143b). On the other hand, female cancer mortality rates have generally declined in the selected OECD countries. New Zealand is one of a minority of countries in this group to have recorded an increase in female cancer mortality during this period (a 12 percent increase); Britain also recorded a similar percentage increase in female cancer mortality at least until the early 1990s, since when the rate declined again, while the Australian and American rates remained essentially stable and Canada's showed a decrease.

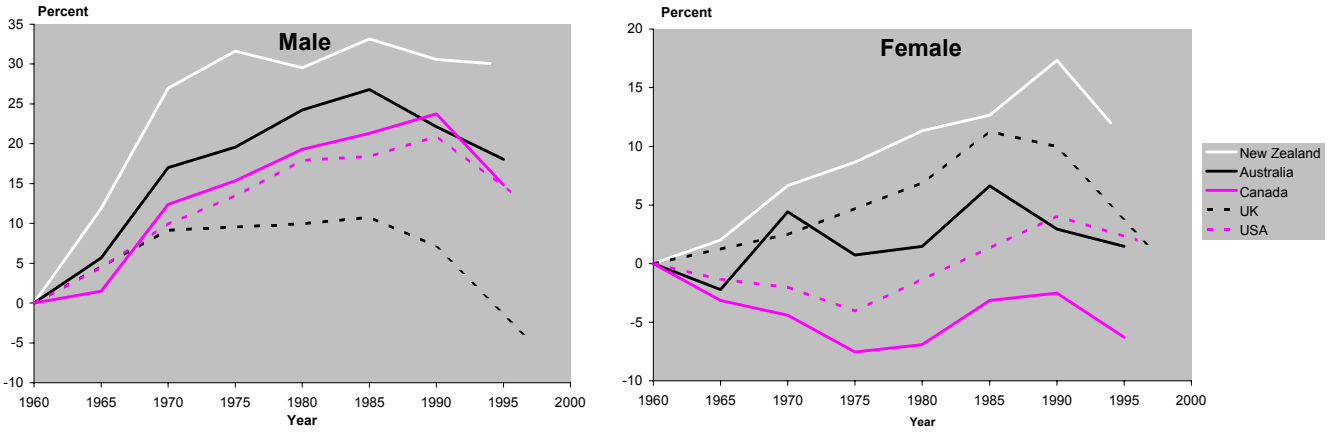
To some extent, these gender specific patterns represent the movement through the population of birth cohorts with different exposures to tobacco. However, smoking patterns are not the full explanation and other exposures (such as unhealthy diet and physical inactivity) also contribute to the complex interplay of age, period and cohort effects within each gender in each country. Changes in the effectiveness of and access to cancer treatment also influence cancer mortality – as opposed to incidence – rates.

Figure 143a: Trends in cancer mortality in New Zealand and similar countries, 1960–96



Note: rate is age standardised to OECD reference population; scale varies between charts; broken axes; data shown are the most recent published for each country (OECD 1999).

Figure 143b: Change in cancer mortality rates in New Zealand and similar countries, 1960–96



Source of base data: OECD
Note: scale varies between charts; data shown are the most recent published for each country (OECD 1999).

Scope for health gain

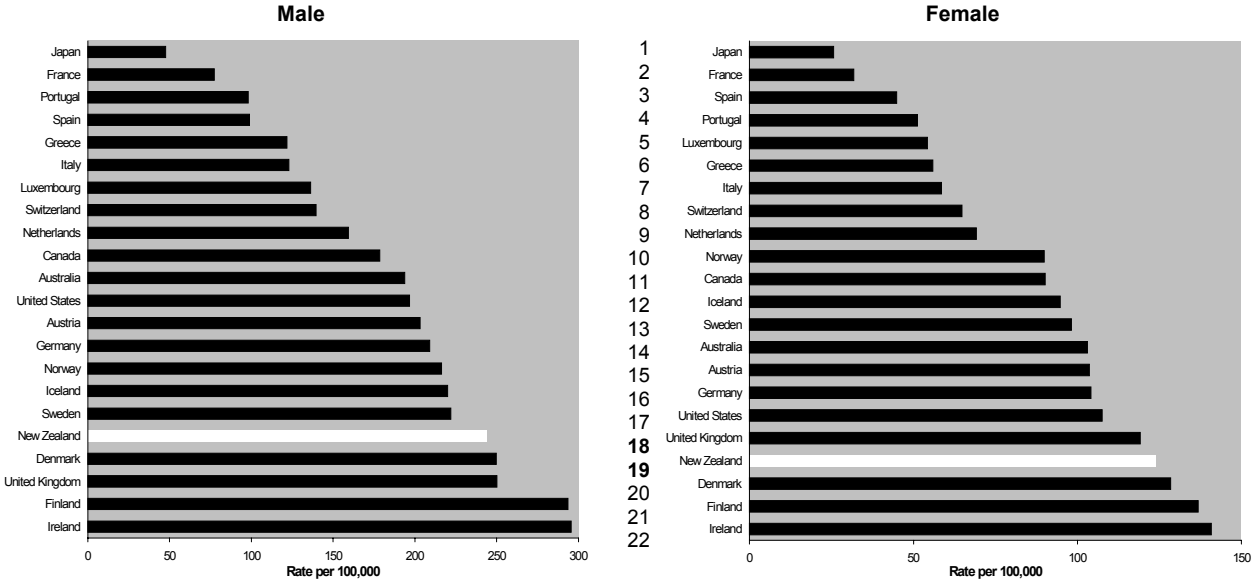
Comparisons of international cancer mortality rates indicate considerable scope for further health gain in New Zealand. Rates of cancer death among women appear to be particularly high compared with other similar countries, including Australia. Some of the greatest reductions in the female cancer burden are likely to be gained through lowering mortality from breast cancer and colorectal cancer, and blunting any further increase in lung and other smoking associated cancers. There is also considerable scope to reduce cancer mortality rates in New Zealand males, especially by reducing the comparatively high incidence of lung cancer in Māori and Pacific males and the comparatively high rates of colorectal cancer and skin cancer mortality among European/Other males. Most of the major cancers are amenable to one or more primary prevention strategies, such as prevention or cessation of smoking, limiting sun (ultraviolet light) exposure, improving diet (especially fruit and vegetable intake) and increasing physical activity (US DHHS 1996). Secondary prevention (screening mammography) is able to reduce mortality from breast cancer by up to one third among women over 50 years of age (Kerlikowske et al 1995).

Ischaemic heart disease

In 1993–95, the latest period for which comparable international IHD mortality data are available, New Zealand's male and female IHD mortality rates ranked 18th and 19th respectively out of 22 OECD countries (Figure 144), with age standardised rates very similar to those experienced in Britain.

Over this period some of the lowest male and female IHD mortality rates were recorded in Japan, France, Portugal and Spain (rates of less than 100 deaths per 100,000 males and 50 deaths per 100,000 females, age standardised to the OECD reference population). However, there is evidence of coding variations in the recording of cardiovascular deaths, and the true rates of IHD mortality in these countries may be as much as twice the recorded rate (Murray 1997).

Figure 144: Ischaemic heart disease mortality in New Zealand and selected OECD countries, 1994



Source of base data: OECD
Note: rate is age standardised to the OECD reference population; scale varies between charts.

Male IHD mortality rates were between 1.8 and 2.6 times higher than female IHD mortality rates across the 22 comparison countries, including those countries with very low male IHD rates. In keeping with this trend, New Zealand's male IHD mortality rate was exactly twice as high as its female rate. When compared with Australia, Canada, Britain and the United States (countries with similar cardiovascular disease coding systems to New Zealand), in 1994 New Zealand had the second highest male and the highest female IHD mortality rates, with only Britain having slightly higher rates for males.*

Māori IHD mortality rates are substantially higher than those of the rest of the New Zealand population. In 1996 the Māori male IHD mortality rate was over 65 percent higher than the rate for the total male population (257 per 100,000 compared with 150 per 100,000 when standardised to Segi's world population), and the rate for Māori females was more than double that for the total female population (150 per 100,000 compared with 69 per 100,000) (Ministry of Health 1998b).

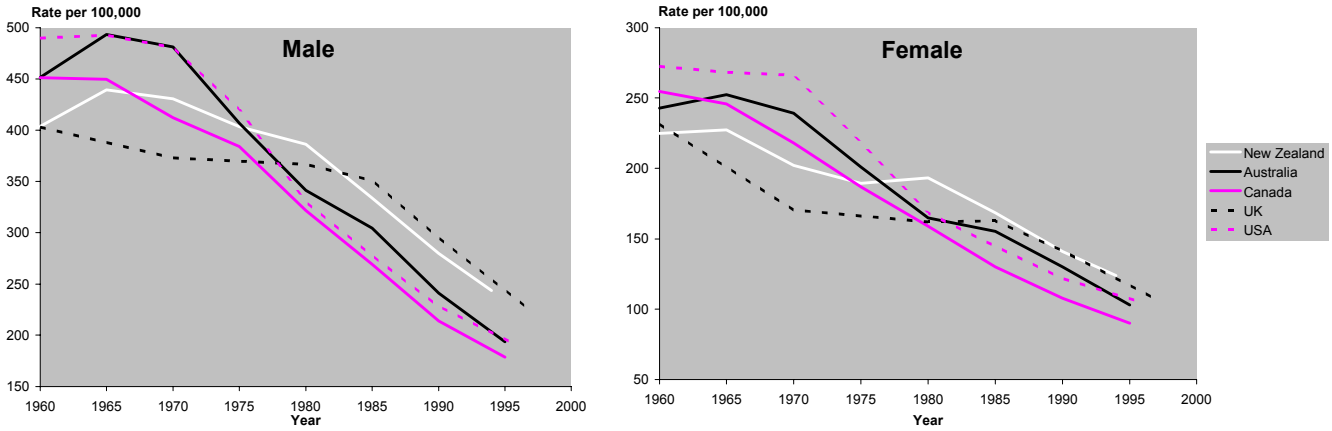
Trends over time

After peaking in the 1970s, New Zealand's IHD mortality rates have declined steadily (Figure 145a). A similar trend occurred in Britain, Australia, Canada and the United States, with the three latter countries all experiencing greater reductions in male IHD mortality than New Zealand over the 1980s and early 1990s (Figure 145b).

* A WHO study has monitored trends in cardiovascular disease in selected regions of the world. Results indicate that in 1985–87, Auckland men and women aged 35–64 years had IHD event rates (age standardised to Segi's world population) in the top 50 percent of the 20 sites selected for comparison. Auckland men had an IHD event rate of 466 per 100,000 population, and women had a rate of 128 per 100,000, or about a quarter of the male rate. These rates compared with a low for men of 187 IHD events per 100,000 and a low for women of 30 IHD events per 100,000 in the Catalonia region of Spain (de Looper and Bhatia 1998).

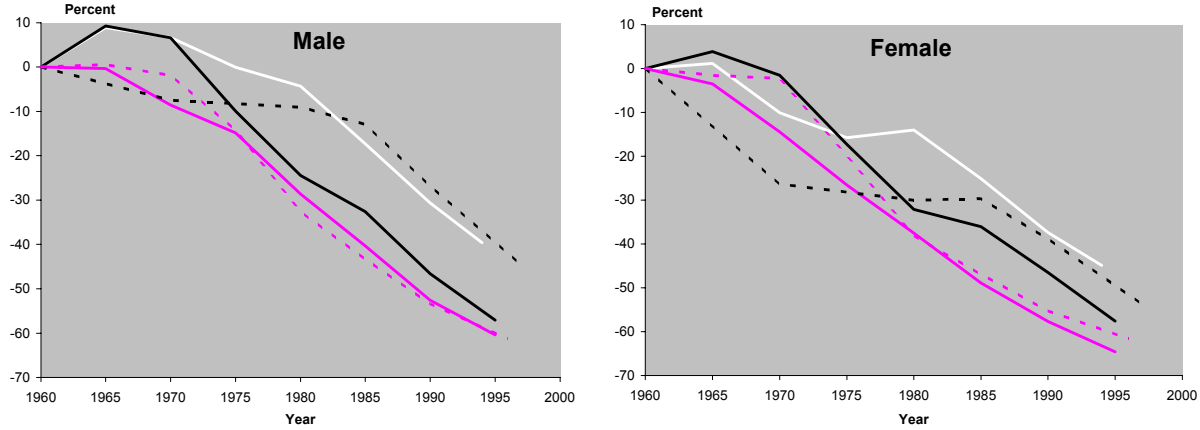
New Zealand's IHD mortality rates began to decline later than those of comparable countries, except Britain, but since about 1980 have declined at about the same rate as the comparison countries.

Figure 145a: Trends in ischaemic heart disease mortality in New Zealand and similar countries, 1960–96



Note: rate is age standardised to the OECD reference population; scale varies between charts; broken axes; data shown are the most recent published for each country (OECD 1999).

Figure 145b: Change in ischaemic heart disease mortality rates in New Zealand and similar countries, 1960–96



Source of base data: OECD
Note: data shown are the most recent published for each country (OECD 1999).

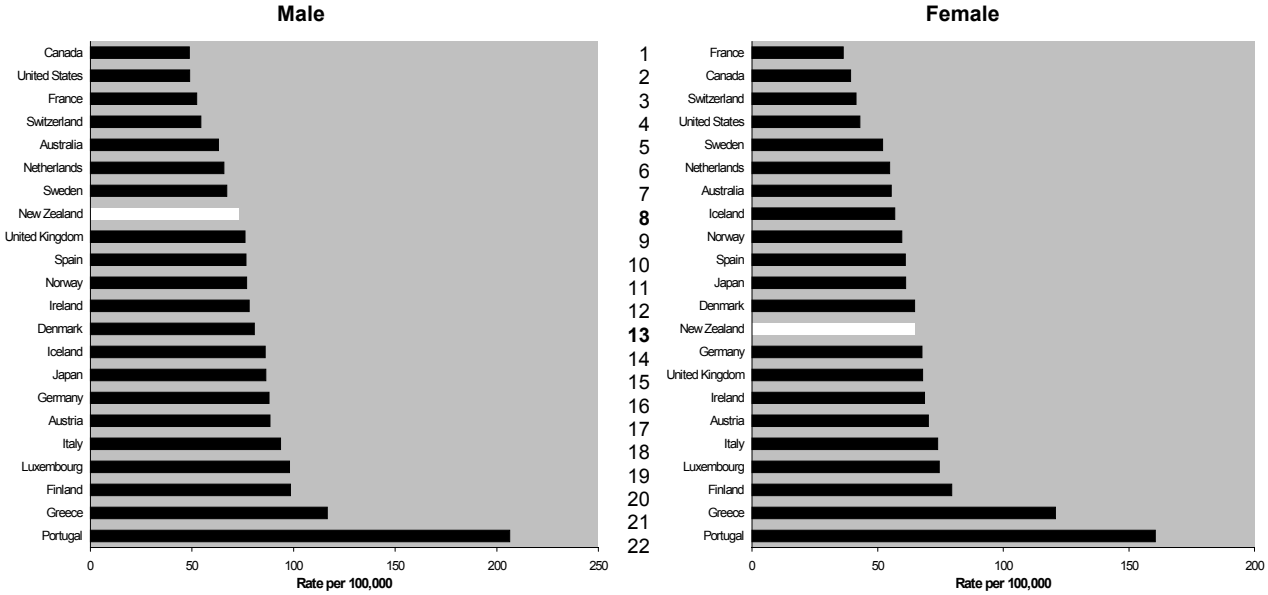
Scope for health gain

If New Zealand's IHD mortality rates could be reduced to twice the rates recorded for Japanese males and females in 1994, males in this country would experience a further 61 percent reduction and females a further 59 percent reduction in IHD mortality. If IHD mortality rates in New Zealand's Māori population could be reduced to similar levels, Māori would experience a further 80 percent reduction in IHD mortality.

Stroke

New Zealand's male age standardised stroke mortality rate of 73 per 100,000 in 1994 (standardised to the OECD reference population) placed it in the top half (eighth) of the 22 comparison countries shown in Figure 146, just above Britain. By comparison, New Zealand's female stroke mortality rate of 65 per 100,000 was ranked 13th (but still better than Britain).

Figure 146: Stroke mortality in New Zealand and selected OECD countries, 1994



Source of base data: OECD
Note: rate is age standardised to the OECD reference population; scale varies between charts.

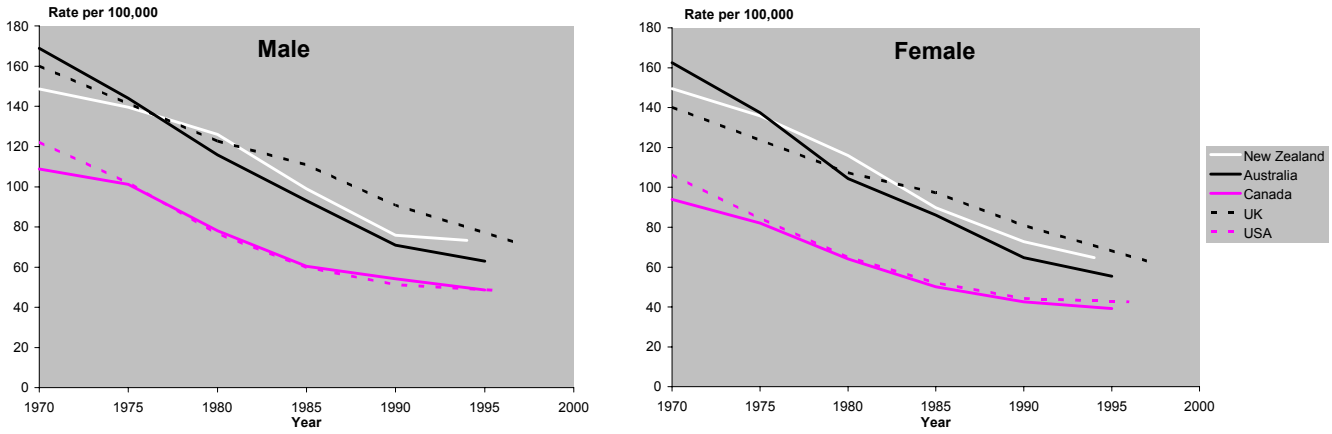
In the mid 1990s, Canada, the United States, France and Switzerland all had particularly low male and female stroke mortality rates. In these countries, age standardised stroke mortality rates were less than 55 per 100,000 for both genders. In the 22 comparison countries, stroke mortality rates were generally 20 percent higher for males than females. The average male stroke mortality rate was 83 deaths per 100,000, nearly 16 per 100,000 higher than the female average of 67 per 100,000. The gender gap was similar in New Zealand.

In New Zealand, stroke incidence and case fatality rates are higher in Māori and Pacific people than in the rest of the population. In 1996 Māori males and females aged 55 years and over had stroke mortality rates approximately 30 and 66 percent higher than the corresponding rates for European/Other males and females respectively.

Trends over time

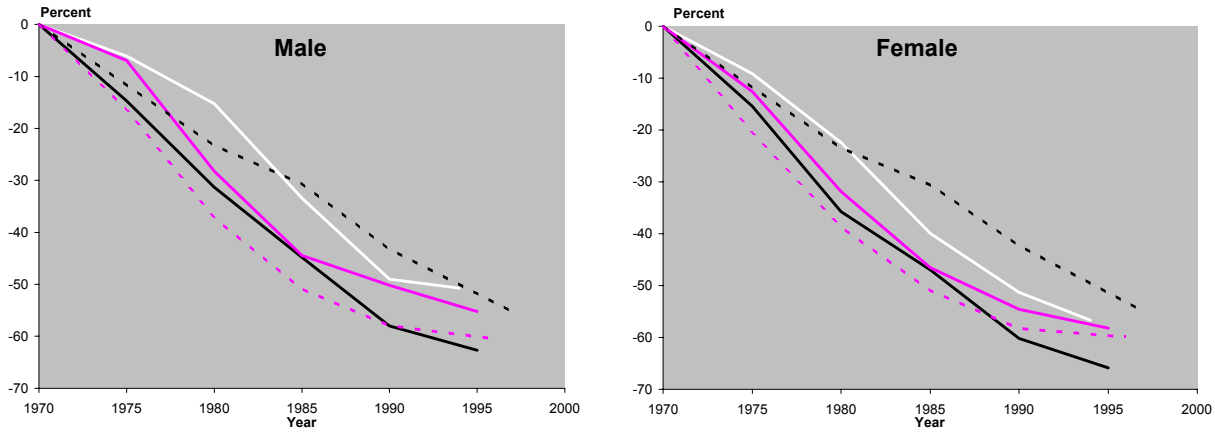
In most OECD countries, including the United States, Canada and Britain, stroke mortality rates have declined steadily for both males and females over the last 50 years. However, in New Zealand and Australia there has been a different pattern, with male and female stroke mortality rates increasing over the two decades from the early 1950s, after which they declined. Since 1970, the rates of decline in stroke mortality have been similar in all of the comparison countries (Figures 147a and 147b).

Figure 147a: Trends in stroke mortality in New Zealand and similar countries, 1970–96



Note: rate is age standardised to the OECD 1980 population; data shown are the most recent published for each country (OECD 1999).

Figure 147b: Change in stroke mortality rates in New Zealand and similar countries, 1970–96



Source of base data: OECD
Note: data shown are the most recent published for each country (OECD 1999).

New Zealand's recent reductions in stroke mortality are likely to be the result of a combination of factors, including improved medical care and the effects of primary prevention strategies (Ministry of Health 1998b). Data from regional studies in Auckland indicate increasing survival rates for stroke over the 10 years to 1991–92, although the actual incidence of stroke may not have declined significantly (Bonita et al 1993).

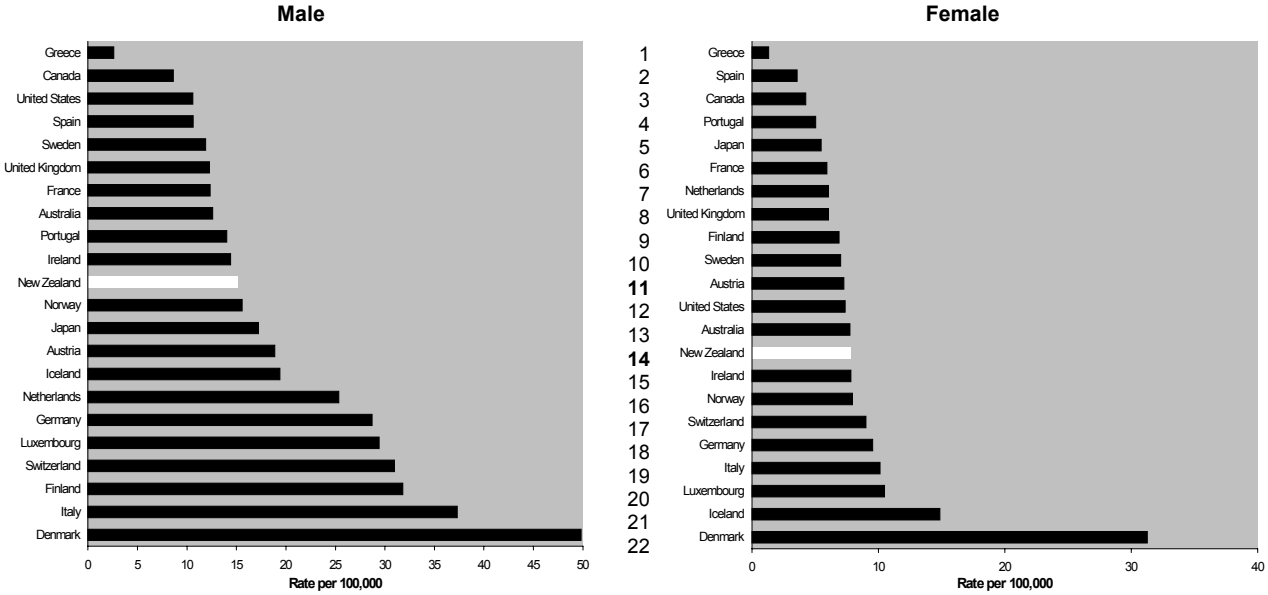
Scope for health gain

Assessed in an international context, there is still potential for further reduction in stroke mortality in New Zealand, particularly for females. If, in future, New Zealand succeeded in matching the lowest rates of male and female stroke mortality currently recorded in the comparison countries, a 34 percent reduction in male stroke deaths and a 44 percent reduction in female stroke deaths would be achieved.

Chronic respiratory disease

This cause category refers mainly to CORD (chronic bronchitis and emphysema) and as such reflects the smoking experience of older cohorts in the different countries. In 1994 New Zealand males and females had age standardised (OECD reference population) chronic respiratory disease mortality rates of 15 and 8 per 100,000 respectively (Figure 148). This ranked New Zealand near the middle of the 22 comparison countries (11th for males and 14th for females), but below Australia, Canada, Britain and the United States for both genders.

Figure 148: Chronic respiratory disease mortality in New Zealand and selected OECD countries, 1994



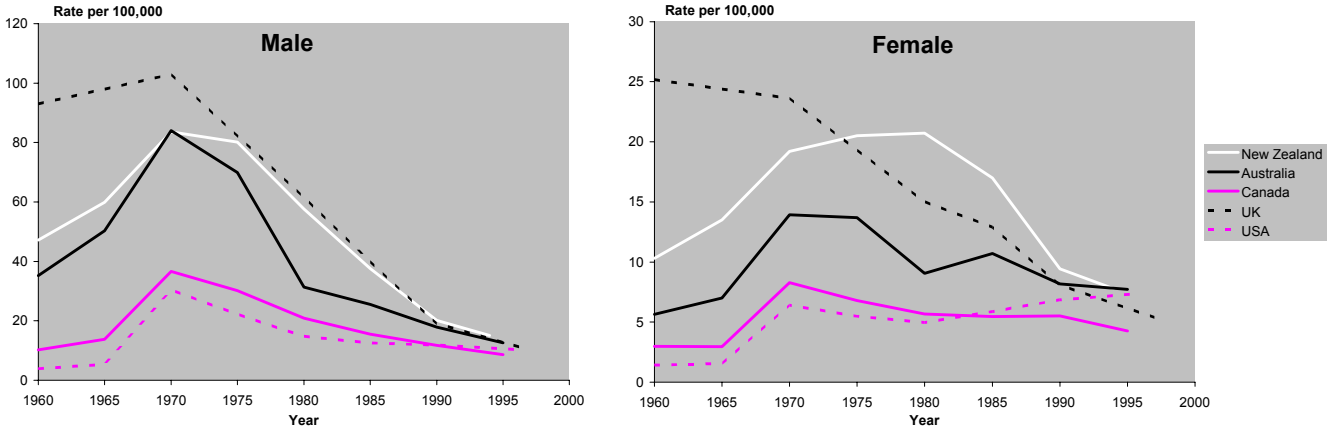
Source of base data: OECD
Note: rate is age standardised to the OECD reference population; scale varies between charts.

In the comparison countries, male rates of respiratory disease mortality were generally twice as high as female rates. This probably partly reflects differential exposure to tobacco.

Trends over time

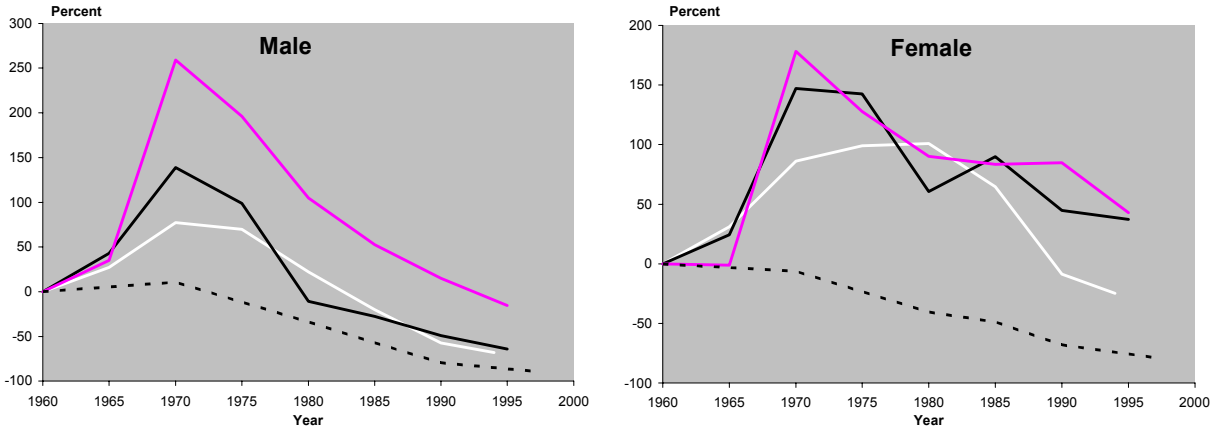
During the 1950s and 1960s New Zealand male and female respiratory disease mortality rates increased, then stabilised in the early 1970s, after which they declined (later for females). Similar broad trends occurred for males in Britain, the United States, Canada and Australia and for females in Britain (Figures 149a and 149b).

Figure 149a: Trends in respiratory disease mortality in New Zealand and similar countries, 1960–96



Note: rate is age standardised to the OECD reference population; scale varies between charts; data shown are the most recent published for each country (OECD 1999).

Figure 149b: Change in respiratory disease mortality rates in New Zealand and similar countries, 1960–96



Source of base data: OECD
Note: scale varies between charts; data shown are the most recent published for each country (OECD 1999).

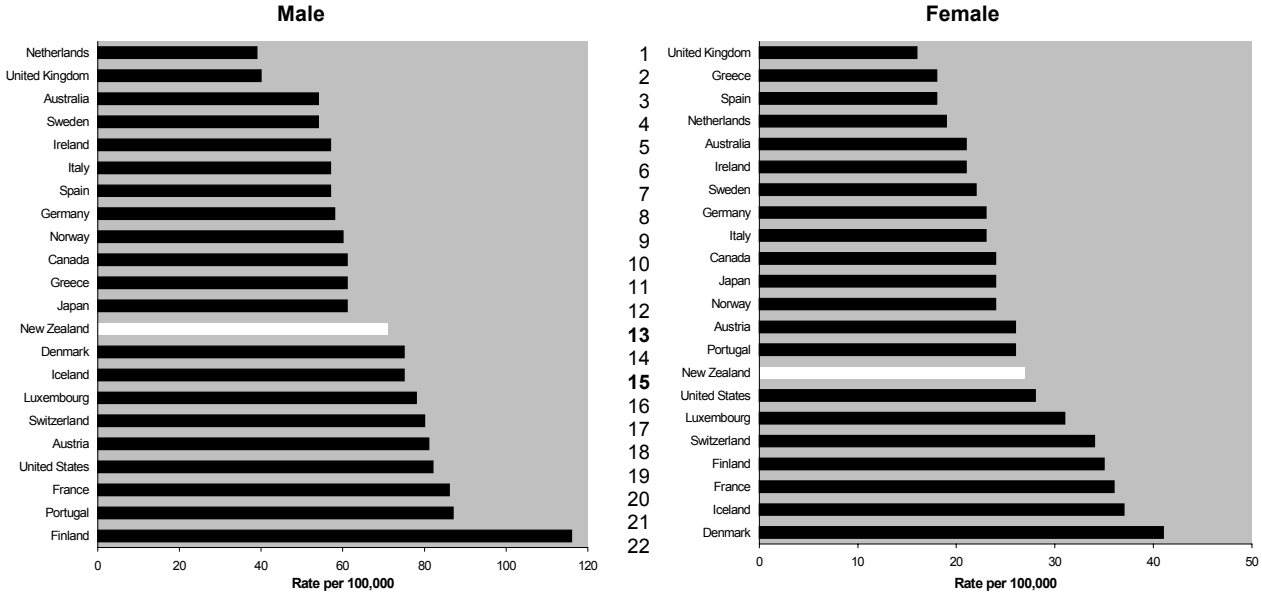
Scope for health gain

These international comparisons suggest that New Zealand has the potential to further reduce rates of respiratory disease mortality. If New Zealanders could match the CORD mortality rates of Canada, mortality from this cause would decrease by approximately 50 percent in both genders.

Injury

In 1994 New Zealand's injury* mortality rates for males (71 per 100,000 age standardised to the OECD reference population) and females (27 per 100,000) ranked 13th and 15th respectively out of the 22 comparison countries (Figure 150). In the same period, some of the lowest injury mortality rates were recorded in the Netherlands and Britain, both of which had rates of less than 40 per 100,000 for males and less than 20 per 100,000 for females.

Figure 150: Injury mortality in New Zealand and selected OECD countries, 1994



Source of base data: OECD
Note: rate is age standardised to the OECD reference population; scale varies between charts.

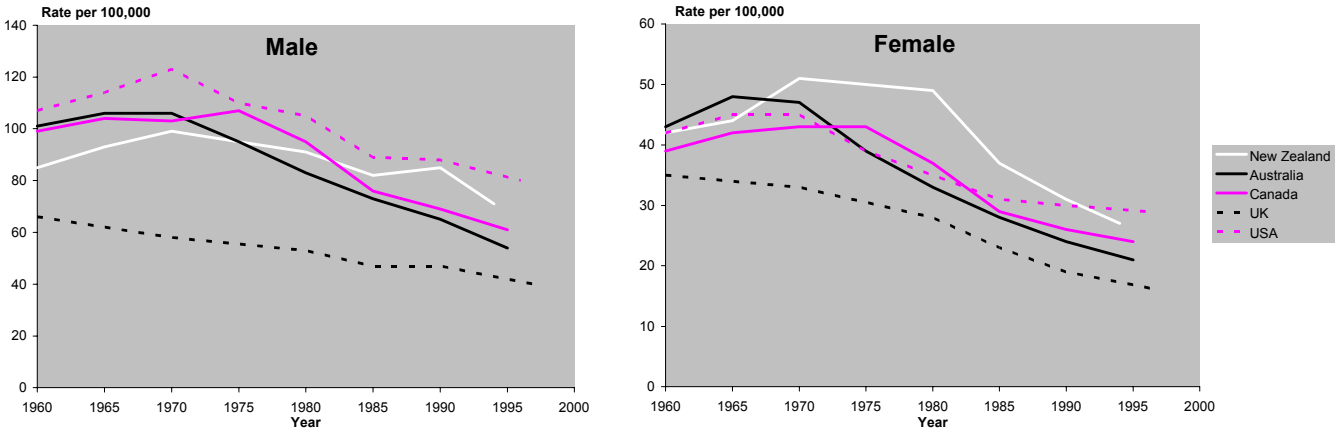
Across the 22 countries, male injury mortality rates were generally about 2.5 times higher than female injury mortality rates. The male–female gap in New Zealand injury mortality rates was only slightly larger than the average for all 22 countries. The Māori population has a high injury death rate by world standards. In 1996 injury mortality rates among Māori males and females were 70 percent higher than European/Other rates. By contrast, injury mortality rates among Pacific people in New Zealand were only slightly higher than the rates for European/Others.

Trends over time

After peaking in the 1970s, New Zealand's male and female injury mortality rates have generally declined, although not as rapidly as those of Australia and Canada (Figures 151a and 151b). These countries had injury mortality rates similar to or higher than New Zealand's in the 1970s, but significantly lower rates for both males and females by the mid 1990s. Of the five countries shown in Figure 151b, Britain has consistently recorded the lowest male and female injury mortality rates since 1960, while the United States has consistently recorded the highest injury mortality rates for males. In recent decades New Zealand and the United States have had the two highest female injury mortality rates in the group.

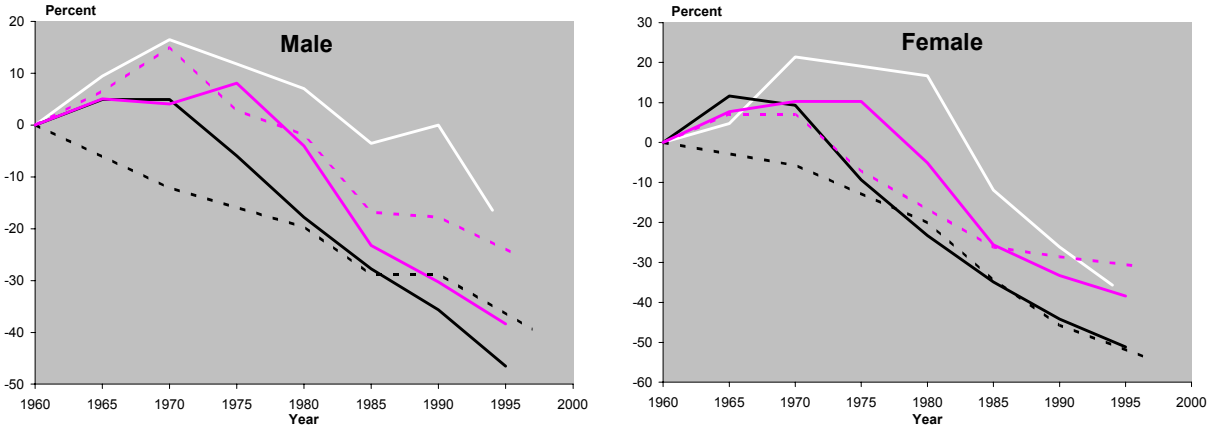
* includes unintentional injury, intentional injury and adverse effects of health care

Figure 151a: Trends in injury mortality in New Zealand and similar countries, 1960–96



Note: rate is age standardised to the OECD reference population; scale varies between charts; data shown are the most recent published for each country (OECD 1999).

Figure 151b: Change in injury mortality rates in New Zealand and similar countries, 1960–96



Source of base data: OECD
Note: scale varies between charts; data shown are the most recent published for each country (OECD 1999).

Scope for health gain

By world standards, considerable scope still exists for New Zealand to reduce its injury mortality rates. If this country could attain the same injury mortality rates as Britain did in 1994, this would result in a near halving of the male injury mortality rate and a 40 percent reduction in the female injury mortality rate. If Britain's recent injury mortality rates were matched in the Māori population, male and female deaths from injury would be reduced by more than 60 percent.

Progress in realising this potential for health gain will require further reductions in fatal road traffic injuries (the major risk factors for which are speed and alcohol), suicide, and falls in older people. Major efforts are already in place to reduce the road toll (LTSA 1996), a national strategy to reduce youth suicide has been implemented recently (Ministry of Youth Affairs et al 1998), and recommendations for reducing falls in older people have been prepared by the National Health Committee (Norton 1997).

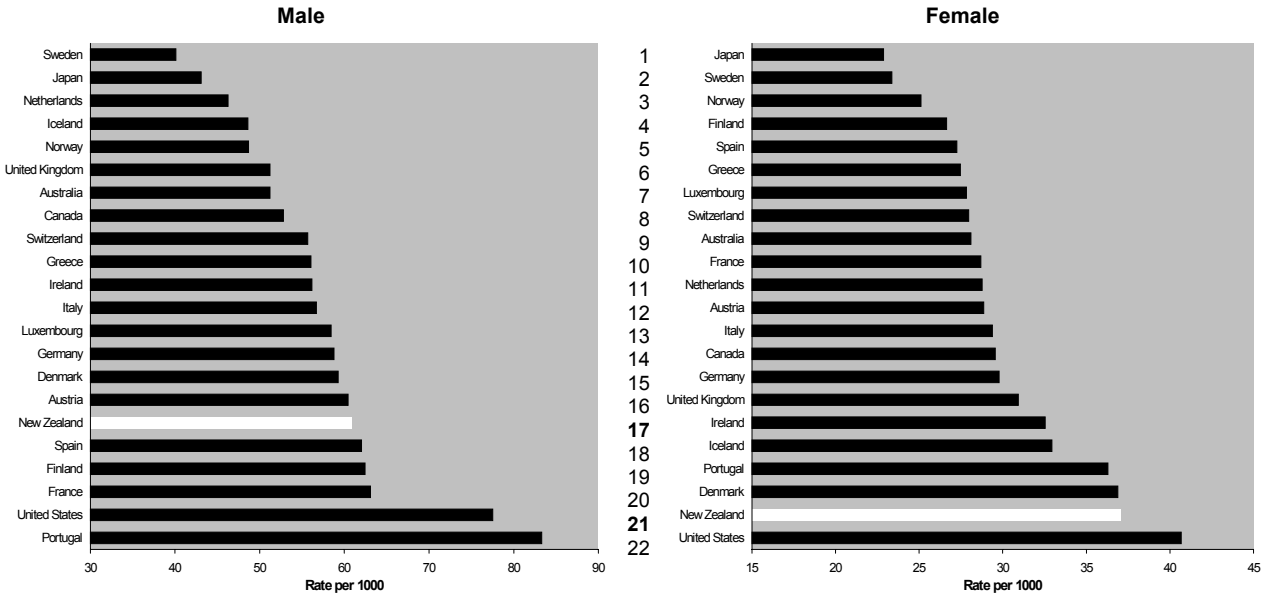
Potential years of life lost

Potential years of life lost (YLL) is a measure of *premature* mortality – that is, the social burden of fatal diseases and injuries. It is calculated here by selecting a particular age (70 years) as the arbitrary upper age limit beyond which death will not be considered premature. YLL measures differ from measures of mortality that are based solely on age specific mortality rates in that they weight deaths according to the age at death. As with age specific death rates, YLL rates still need to be age standardised if fair comparisons are to be made across populations with different age structures.

In Chapter 3 two measures of years of life lost are applied, one using age 65 as the upper age limit ('presenescent mortality') and the other calculating the gap between the actual age at death and the life expectancy remaining at this age ('premature mortality'). These indicators are also discounted at a rate of 3 percent per year. The present chapter uses an arbitrary upper age limit of 70 years and no discounting, as the OECD database does not permit calculation of other YLL indicators.

In 1996, the latest year for which comprehensive international YLL data for all-cause mortality are available, New Zealand males had an age standardised (OECD reference population) YLL_{70} rate of 61 years of life lost per 1000 population. The corresponding rate for females was 37 years of life lost per 1000. Compared with other OECD countries, New Zealand's YLL_{70} rates were relatively high, with the male rate ranked 17th out of 22 countries and the female rate 21st (Figure 152). This indicates that in recent years New Zealanders have experienced relatively high death rates at ages less than 70 years.

Figure 152: Potential years of life lost before age 70 from all causes in New Zealand and selected OECD countries, 1996



Source of base data: OECD
Note: rates are age standardised to the OECD reference population; scale varies between charts; broken axes.

In 1996 New Zealand's all-cause YLL_{70} rate for females was just under two thirds the male rate – a gap of 24 years of life lost per 1000. This gap was slightly smaller than the average for all 23 OECD countries. Iceland, Sweden, the Netherlands and Japan were among the countries with smaller gender gaps of between 15 and 18 years of life lost per 1000.

Although the OECD database includes a time series of YLL_{70} data, the earlier data are not considered to be robust because of variations between countries in the method used to estimate and standardise this indicator. For this reason, YLL estimates for earlier years are not reported here. Cause specific YLL rates are not reported here for the same reason.

Applying the YLL_{65} measure rather than the YLL_{70} measure, in 1996 Māori lost more than twice as many potential years of life per capita than the European/Other population. By this same measure, Pacific people lost potential years of life at more than 1.5 times the rate of European/ Other New Zealanders.

Scope for health gain

Data from other countries indicate that age standardised (OECD reference population) YLL_{70} rates for males of 40–45 per 1000 and rates for females of 25–30 per 1000 are achievable. If New Zealand could match these rates in the years ahead, it would amount to a reduction in years of life lost by both males and females aged 0–69 years of about one third. New Zealand's Māori and Pacific people experience a greater loss of potential years of life than do European/Others. As Chapter 3 indicates, if all subgroups in the New Zealand population had YLL rates similar to those of European/Other females, the total life years lost annually by the New Zealand population would again be reduced by almost one third.

Summary and conclusions

Since 1950 New Zealanders, like people in virtually all other OECD countries, have experienced substantial improvements in life expectancy and marked reductions in rates of all-cause mortality and potential years of life lost. Associated with this have been significant overall reductions in mortality related to perinatal and infant conditions, stroke, IHD and unintentional injury.

Although the scale of these improvements should not be underestimated, since the mid 1970s many of New Zealand's key health indicators have not improved as rapidly as those of other similar countries, such as Australia and Canada. As a consequence, during the last 20 years, for virtually all of the health indicators considered in this chapter, New Zealand's relative position has declined. By the mid 1990s, for many key mortality based measures – such as life expectancy at birth, infant mortality, female cancer mortality, male and female IHD mortality, female injury mortality, and male and female rates of potential years of life lost from all causes – New Zealand ranked below the median of the countries considered here.

When the health of New Zealand's ethnic groups is assessed in an international context, it is clear that the Māori population, despite marked improvements in life expectancy since 1950, still experiences survival chances significantly below those enjoyed by most people in the OECD. Relatively low levels of health are also found among indigenous peoples and ethnic minorities in other OECD countries. For example, the ethnic gaps in New Zealand are smaller than the Indigenous–non-Indigenous gap in Australia, but larger than the black-white differential in the United States, at least with respect to life expectancy.

The precise ranking of New Zealand relative to other countries depends on the indicator selected and the method chosen for its calculation (for example, the reference population selected for age standardisation): comparison of age standardised mortality rates and of YLL rates is liable to distortion by the reference or standard population selected. The fairest comparison is probably life expectancy, since life table methods adjust for differences in age distribution between groups without imposing any arbitrary standard. New Zealand fairs best (relatively) on this indicator, with males ranking 13th and females 17th out of 22 selected comparison countries for life expectancy at birth in 1995–97.

Whatever New Zealand's precise mortality based ranking may be – and this is subject to data quality issues and random variations from year to year – a more relevant question is whether the rankings for non-fatal health outcomes would show a similar pattern to the mortality-based rankings. Unfortunately, cross country comparisons of self reported (subjective) health status, disability and morbidity (such as hospitalisation rates) are either insufficiently standardised to be reliable or subject to cultural variation (for example, in willingness to acknowledge disability or in thresholds for admission to hospital). For the same reasons, international comparisons of integrated health measures (health expectancies or health gap measures) are also problematic at present. Nevertheless, limited data on levels of disability described in Chapter 5 of this report suggest that the country rankings are likely to be similar to those based on mortality measures. Certainly, there is no reason to believe that New Zealand is doing less well with respect to mortality based indicators but better with respect to non-fatal health outcomes.

In summary, the comparative data presented here are indicative of significant scope for improvement in health outcomes in New Zealand, most probably including both fatal and non-fatal outcomes. This applies to the population as a whole, but more especially to the Māori and Pacific ethnic groups. And although New Zealand males (like males in other OECD countries) have lower levels of health than females on many indicators, the international context reveals that New Zealand females are further behind than their male counterparts.

Although cross country and intra-national comparisons provide useful indicators of the potential for improvement, benchmarking has limitations as an indicator of scope for health gain. Firstly, comparisons with the 'best in class' for different indicators fail to acknowledge that doing well on one indicator may involve trade-offs with respect to other indicators. To aim simultaneously for Japanese male life expectancy, French female life expectancy, Australian perinatal mortality, Finnish infant mortality, Greek cancer mortality and British injury mortality is unrealistic because it makes no allowance for substitute mortality (if the mortality rate from one cause is reduced, mortality rates from other causes must increase, albeit at later ages – we all must die some time). Secondly, what works in one society may fail in another, with a different social structure and cultural milieu: Japan's significant achievements in extending life may hold few lessons for New Zealand.

On the other hand, benchmarking may underestimate the scope for progress, since it assumes that the reference country (the 'best in class') is itself incapable of further improvement, which is clearly false. For instance, Japanese life expectancy continues to improve, so far with no sign of slowing down. In fact, benchmarking may be better considered an equity tool rather than an indicator of scope for health gain for the population as a whole. On this basis, the inequalities in health outcomes described throughout this report – across age groups, genders, ethnic groups and, perhaps most importantly, socioeconomic groups – may be more relevant for New Zealand health and social policy than international benchmarking.

Chapter 10: Avoidable mortality and morbidity

Introduction

Chapter 9 analysed the scope for health gain by comparing health outcomes between population groups. This chapter assesses the scope for health gain by analysing the causal structure of health outcomes at the level of diseases and injuries – the proximal causes of these outcomes.

This analysis depends on two principles: firstly, the accurate assignment of cause of death or hospitalisation (or some other morbidity measure); and, secondly, categorical attribution of each cause as 'avoidable' or 'unavoidable'. Categorical attribution of diseases and injuries was first proposed by Ruttstein et al (1976). The first widely accepted list of causes of avoidable mortality was assembled by Charlton et al (1983), although this list was restricted to conditions amenable to medical treatment only and was intended to serve as a health care system performance indicator (Holland et al 1994). Variations of Charlton's list have been used in previous studies of avoidable mortality in New Zealand (Marshall and Keating 1989; Salmond and Malcolm 1993; Jackson et al 1998). The approach has also been extended from mortality to hospitalisation (Weissman et al 1992; Billings et al 1996).

For this analysis, in order to update and extend Charlton's original list, an extensive reassessment of the categorical attribution of ICD 9 codes was undertaken by reviewing published updates (for example, Manitoba Department of Health 1992; Tengs et al 1995) and key references for each condition. The extended list covers causes amenable to population-based and individual-based preventive interventions as well as those amenable to medical or surgical treatment, and it reflects recent developments in health promotion and disease prevention practice as well as advances in health care technology. As such, the approach adopted here is broader than that originally conceptualised by Charlton and is intended to serve as a measure of the scope for health gain – not as a performance indicator for the health care sector.* The avoidable causes have been further categorised according to the level of intervention involved, building on earlier work by Albert (1995) and Simonato et al (1998). The subcategories differ for avoidable mortality and avoidable hospitalisation.

Avoidable mortality

A potentially avoidable death is one that, theoretically, could have been avoided given current understanding of causation and currently available disease prevention and health care technologies. Three subcategories of avoidable causes of death are distinguished for this analysis:

* The list of avoidable conditions used in this report, together with subcategories and key references, is included in Appendix 3.

- *Primary avoidable mortality* (PAM) – conditions that are preventable, whether through individual behaviour change (lifestyle modification) or population level intervention (healthy public policy). The condition is prevented before it develops by addressing its risk or protective factors: 'primary prevention'.
- *Secondary avoidable mortality* (SAM) – conditions that respond to early detection and intervention, typically in a primary health care setting. As well as clinical preventive services such as cancer screening, it includes chronic disease management intended to delay the progression of diseases such as diabetes or the recurrence of events such as heart attacks or strokes (for example, through the monitoring and management of high blood pressure). This approach constitutes 'secondary prevention'.
- *Tertiary avoidable mortality* (TAM) – those conditions whose case fatality rate can be significantly reduced by existing medical or surgical treatments (typically, but not necessarily, in a hospital setting), even when the disease process is fully developed. This constitutes 'tertiary prevention'.

Inevitably there is overlap among the three subcategories, and judgement had to be applied in some cases to partition conditions across the three groups. For example, data from the ARCOS study (Beaglehole et al 1997) was used to attribute 50 percent of ischaemic heart disease mortality to primary avoidable mortality, 25 percent to secondary avoidable mortality, and 25 percent to tertiary avoidable mortality. By contrast, partitioning was not used in the initial categorisation of conditions into 'avoidable' or 'unavoidable'. Thus avoidable mortality should be interpreted as mortality that is *potentially* or theoretically avoidable, rather than as mortality that could realistically be prevented in all cases, at least in the short to medium term. For example, road traffic injuries are considered avoidable, although the total prevention of such deaths may well involve a greater trade off or sacrifice of mobility for health than society is willing to make.

It should be emphasised once again that the concept of 'avoidability' applied in this report is wider than that originally developed by Ruttstein and Charlton (Ruttstein et al 1976; Charlton et al 1983), which was restricted to conditions amenable to treatment rather than to prevention. Ruttstein proposed avoidable mortality as a subset of premature mortality (since, by definition, only deaths occurring prematurely are potentially avoidable) and employed an arbitrary upper age limit of 65 years. For this analysis, the age limit has been extended to 75 years (roughly, the current New Zealand life expectancy at birth), so allowing a higher proportion of deaths (46 percent in 1996) to be categorisable. The justification for doing so is that cause of death coding is considered to be reliable up to age 75; beyond this age, co-morbidity makes disentangling avoidable from unavoidable deaths problematic. Thus the concept of 'avoidability' applied here is wider than the original conception, both in the range of conditions considered potentially avoidable and in the (arbitrary) upper age limit applied to the category.

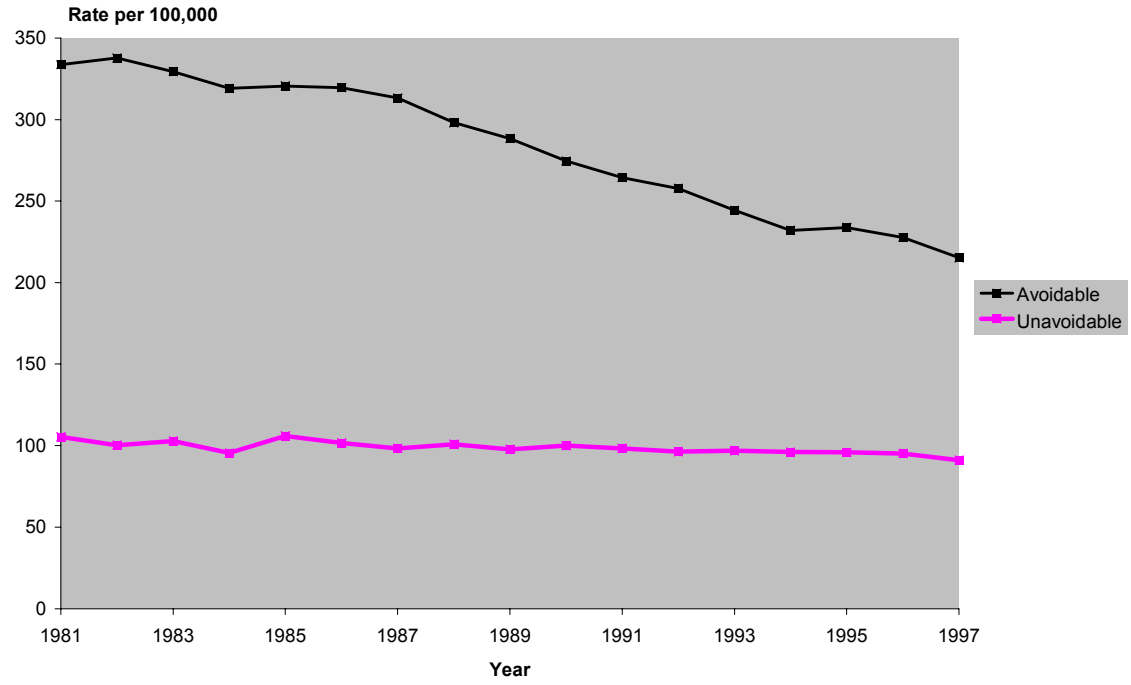
Data sources and methods

The analysis is based on mortality data for 1981–97 (the 1997 data are provisional) supplied by the New Zealand Health Information Service (NZHIS). ICD coding has been consistent over this period (except for minor changes in 1988 and 1995). Age standardisation has been carried out using the direct method, with Segi's world population as the reference. Because ethnicity coding changed significantly in 1995, an ethnic specific time series cannot be calculated. It should be noted that this analysis uses a wider age range and a more extensive set of conditions (especially injuries) categorised as 'avoidable' than previous New Zealand studies, and so cannot be directly compared with those studies.

Avoidable mortality in the whole population

From 1981 to 1997 all-cause mortality in the 0–74 age group fell by 31 percent (Figure 153). This was made up of a 9 percent fall in unavoidable mortality and a 38 percent fall in avoidable mortality. The reduction in unavoidable mortality provides a baseline against which to interpret the reduction in avoidable mortality. The 'excess' (greater than expected) reduction in avoidable mortality – a fall of 29 percent over the period, or over 1.5 percent per year on average – represents the 'added value' of the health system (including health promotion and disease prevention as well as treatment services). In absolute terms this represents a gain of approximately 300 deaths avoided per year, or almost 5000 fewer deaths in 1997 than expected based on 1981 mortality (adjusting for the underlying trend in unavoidable mortality).

Figure 153: Avoidable and unavoidable mortality, ages 0–74 years, 1981–97



Source of base data: NZHIS
Note: rate is age standardised to Segi's world population.

Although the historical trend provides a measure of health system performance, the current level of avoidable mortality indicates the scope that remains for further health gain through health promotion, disease prevention and treatment. In 1996 or 1997, approximately 9000 potentially avoidable deaths occurred per year – about 70 percent of all deaths occurring in people under 75 years of age (Table 78). The majority of these potentially avoidable deaths are preventable (PAM), with early intervention (SAM) and medical treatment (TAM) making smaller contributions. The precise contributions of the three levels of intervention vary with age, gender, ethnicity and socioeconomic status.

Table 78: Avoidable deaths, ages 0–74, average of 1996 and 1997

	Number of deaths	Age standardised rate per 100,000	Percentage of total deaths in 0–74 age group	Percentage of avoidable deaths
Avoidable mortality	9025	223	70	100
PAM	4741	116	37	53
SAM	2302	56	18	26
TAM	1982	50	15	22
Unavoidable mortality	3861	94	30	
Total mortality	12,886	316	100	

Source of base data: NZHIS (1997 data are provisional)
Note: number and rate of deaths is the average for 1996 and 1997; PAM = primary avoidable mortality; SAM = secondary avoidable mortality; TAM = tertiary avoidable mortality.

Variations by age

In each age group under 75 years, between 65 and 81 percent of all deaths are avoidable (Table 79). In absolute terms, avoidable mortality is therefore predominantly a feature of middle and (early) old age, with almost 80 percent of avoidable deaths occurring in people aged 45 years and over (1996–97 average). Age specific rates of avoidable mortality are low until middle age (then rise exponentially), with the exception of infancy. However, the high infant rate (and proportion) may be partly an artefact: routine datasets do not hold all the variables needed to make firm categorical attributions of perinatal and infant deaths (Langhoff Ross et al 1996; Petrini et al 1998); as a result avoidable infant mortality may have been overestimated.

In all age groups, preventable conditions (PAM) dominate the pattern of avoidable mortality. Indeed, there is surprisingly little variation in the relative shares of the three subcategories across the different stages of the life cycle, apart from a slight rise in the proportion of TAM in the younger age groups.

Table 79: Avoidable mortality, by age, 1996–97

	Number of deaths (and rate)						
	Age: < 1	1–14	15–24	25–44	45–64	65–74	Total
Avoidable mortality	325 (581)	139 (18)	393 (73)	926 (84)	3043 (422)	4200 (1703)	9025
PAM	168 (300)	68 (9)	223 (42)	473 (43)	1564 (217)	2247 (911)	4741
SAM	53 (95)	18 (2)	63 (12)	228 (21)	814 (113)	1125 (456)	2302
TAM	104 (186)	53 (7)	107 (20)	224 (20)	665 (92)	829 (336)	1982
Unavoidable mortality	78 (139)	77 (10)	114 (21)	422 (38)	1334 (185)	1838 (745)	3861
Total mortality	403 (721)	216 (28)	506 (95)	1347 (122)	4377 (606)	6038 (2448)	12,886
AM %	81	65	78	69	70	70	70

Source of base data: NZHIS (1997 data are provisional)
Note: number and rate of deaths is the average for 1996 and 1997 (age specific rate per 100,000 in brackets).

Among infants, the standout conditions are SIDS and low birthweight (Table 80), with maternal smoking being the common preventable exposure. Birth defects are sensitive to maternal folate and vitamin consumption, and birth trauma and asphyxia responds to effective obstetric care.

Among children and youth, injuries dominate the avoidable mortality picture. Averaging over 1996 and 1997, road traffic injuries accounted for 20 percent of all deaths (and 32 percent of avoidable deaths) in the under 15 age group. The corresponding proportions for youth (15–24) were 33 percent and 43 percent. Suicide accounted for a further 29 percent of all deaths (37 percent of avoidable deaths) among young people. Were it not for these two causes, only 20 percent of (remaining) deaths in this age group would have been considered avoidable. Injury (especially suicide and road traffic injury) remains the leading contributor to avoidable mortality among young adults. Ischaemic heart disease (IHD) emerges for the first time, becoming the leading cause in middle and old age. The common cancers (breast, colorectal and lung) and (smoking related) chronic obstructive respiratory disease (CORD) occupy the remaining rankings in the adult age groups.

Table 80: Major causes of avoidable mortality, by age, 1996–97

Age (years)	Condition	Deaths	Percentage
< 1	SIDS	89	22
	Low birthweight	67	17
	Congenital anomalies	43	11
	Birth trauma and asphyxia	35	9
1–14	Road traffic injury	44	20
	Leukaemia	16	7
	Congenital anomalies	12	5
	Fire	11	5
15–24	Road traffic injury	169	33
	Suicide	147	29
	Drowning	16	3
25–44	Suicide	242	18
	Road traffic injury	171	13
	IHD	108	8
	Breast cancer	63	5
45–64	IHD	947	22
	Lung cancer	383	9
	Colorectal cancer	296	7
	Breast cancer	253	6
65–74	Ischaemic heart disease	1513	25
	Lung cancer	545	9
	CORD	459	8
	Colorectal cancer	351	6

Source of base data: NZHIS
Notes: deaths are per year averaged over two years 1996–97; percentage is of all deaths (including unavoidable deaths) in that age group.

Although medical treatment could achieve considerable health gains, primary preventive strategies (such as reducing smoking and improving diet and physical activity) and secondary preventive services (such as management of high blood pressure, diabetes and cancer screening) appear to hold the key to substantive reductions in these causes of death (Table 81).

Table 81: Major causes of avoidable mortality, by age and intervention category, 1996–97

Age	PAM		SAM		TAM	
(years)	Condition	Deaths	Condition	Deaths	Condition	Deaths
< 1	SIDS	89	Birth trauma	14	Congenital anomalies	30
	Low birth weight	34	Congenital anomalies	9	Low birth weight	27
1–14	Road traffic injury	26	Epilepsy	5	Road traffic injury	17
	Fire	11	Other infections	3	Leukaemia	14
15–24	Road traffic injury	101	Suicide	44	Road traffic injury	67
	Suicide	88	Epilepsy	5	Suicide	15
25–44	Suicide	145	Suicide	73	Road traffic injury	68
	Road traffic injury	102	Epilepsy	27	Breast cancer	31
45–64	IHD	474	IHD	253	IHD	237
	Lung cancer	364	Colorectal cancer	148	Breast cancer	127
65–74	IHD	757	IHD	378	IHD	378
	Lung cancer	518	Colorectal cancer	175	Breast cancer	69

Source of base data: NZHIS (1997 data are provisional)
Notes: deaths are per year averaged over two years ie, 1996–97.

Since 1981 avoidable mortality rates have fallen in every age group (Table 82). The largest percentage reductions have occurred at both extremes of the 0–74 age range. The lowest reduction has been in the 15–44 age group; indeed, unavoidable causes have declined faster than avoidable causes in this age group. Given the dominance of injury among avoidable causes in those aged 15–44, this pattern reflects the net effect of moderate improvements in road safety offset by worsening suicide rates over this period.

Table 82: Change in mortality rates, by age and intervention category, 1981–97

	Percentage fall in rates					
	Age: <1	1–14	15–24	25–44	45–64	65–74
PAM	60	39	6	5	44	36
SAM	63	44	60	39	45	41
TAM	44	35	5	28	46	34
Total avoidable mortality	56	39	11	11	44	36
Unavoidable mortality	18	27	28	18	12	1
Total mortality	51	35	15	13	37	28

Source of base data: NZHIS (1997 data are provisional)

Variations by gender

Males have a higher rate of avoidable mortality than females: age standardised rates of 270 per 100,000 and 175 per 100,000 in 1996–97 respectively.* This represents a 54 percent male excess, compared to a 40 percent male disadvantage in unavoidable mortality. The gender difference in avoidable mortality is largely attributable to a higher rate of preventable deaths (PAM) (Table 83).

* age standardised to Segi's world population

Table 83: Avoidable mortality, ages 0–74, by gender, 1996–97

	Number of deaths per year			Age standardised rate		Ratio
	Male	Female	Total	Male	Female	M:F
PAM	3009	1642	4741	150	83	1.8
SAM	1330	972	2302	63	48	1.3
TAM	1140	842	1982	57	44	1.3
Total avoidable mortality	5569	3456	9025	270	175	1.5
Unavoidable mortality	2298	1563	3861	109	78	1.4
Total mortality	7867	5019	12,886	379	253	1.5
Avoidable mortality as % all deaths	71	69	70			

Source of base data: NZHIS (1997 data are provisional)
Notes: age standardised rate is per 100,000 population per year (standardised to Segi's world population).

Table 84 lists the most common causes of avoidable death in the 0–74 age group for each gender. The higher male avoidable mortality rate partly reflects the differential magnitude of the IHD epidemic in the two genders: if males and females had experienced the same IHD mortality rates in 1996 or 1997, approximately 1200 fewer males (aged less than 75 years) would have died each year. Another contributor to the gender inequality in avoidable mortality is injury, males having much higher death rates than females for both road traffic injuries and suicide.

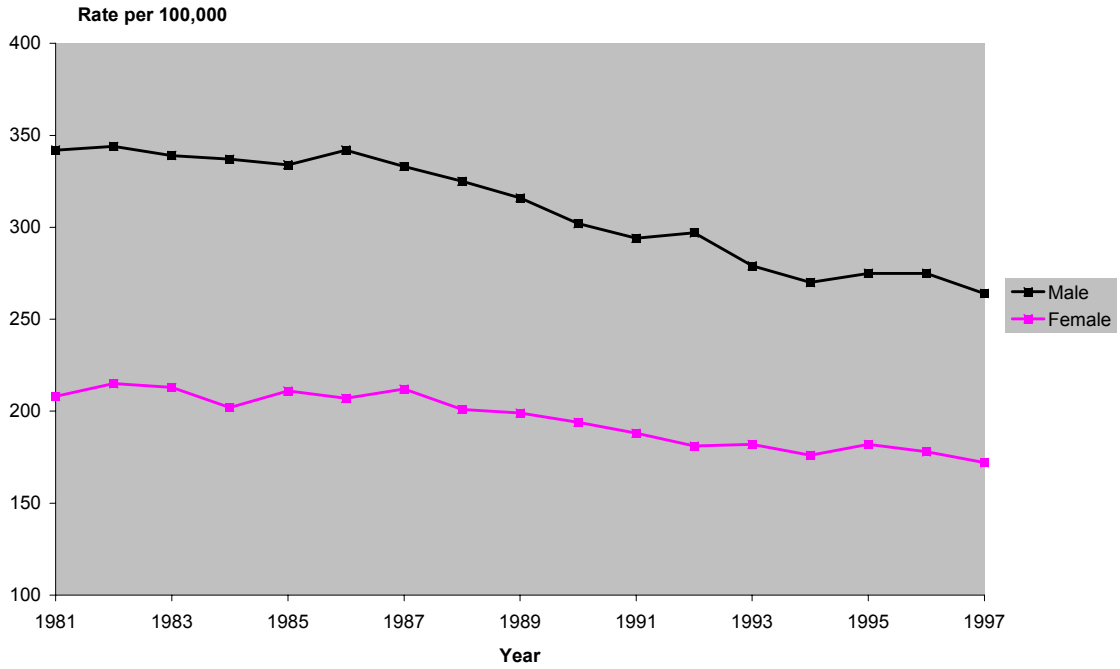
Table 84: Leading causes of avoidable mortality, ages 0–74, by gender, 1996–97

	Condition	Deaths per year	Percentage all 0–74 deaths	Male 'excess' deaths
Male	IHD	1877	24	1160
	Lung cancer	597	8	210
	Suicide	420	5	290
	Colorectal cancer	387	5	90
	Road traffic injury	360	5	210
Female	IHD	693	14	
	Breast cancer	453	9	
	Lung cancer	358	7	
	Colorectal cancer	284	6	
	CORD	255	5	

Source of base data: NZHIS (1997 data are provisional)
Notes: percentage is of all 0–74 deaths (including unavoidable deaths) by gender.

However, analysis of the trends in avoidable mortality since 1981 shows that there has been a faster rate of improvement for males than females, so narrowing the gap significantly (Figure 154). These trend differences may reflect the differential timing of the tobacco epidemic in the two genders, with smoking-related mortality falling in males (from a high base) but increasing in females (from a lower base) over the period concerned.

Figure 154: Avoidable mortality, by gender, 1981–97



Source of base data: NZHIS (1997 data are provisional)
Note: rate is age standardised to Segi's world population; broken axis.

Variations by ethnicity

Māori and Pacific people aged 0–74 years have much higher rates of avoidable deaths than European/Others (Table 85): in 1996–97 the Māori avoidable death rate was 2.5 times and the Pacific rate 1.9 times that of European/Other New Zealanders. Had these rates been the same, Māori would have experienced 970 fewer deaths and Pacific people 210 fewer deaths each year than actually occurred; this represents 45 percent and 35 percent of all Māori and Pacific deaths in the 0–74 age group respectively.

Since the majority of avoidable deaths in all ethnic groups fall into the PAM subcategory, the scope for health gain – and equity – is clearly greatest through primary prevention strategies. Yet the largest *relative* difference is in SAM, where the rates are approximately 2.5 times greater for both Māori and Pacific people. This subcategory includes conditions such as diabetes, high blood pressure, rheumatic heart disease and screenable cancers, all of which are amenable to early intervention and ongoing management in the primary or integrated care setting. At present, health care providers do not appear to be fully meeting the needs of Māori and Pacific people in this respect, although sufficient time has not elapsed for recent innovations in the funding and delivery of primary and integrated care to take effect (Malcolm 1999).

Table 85: Avoidable mortality, ages 0–74, by ethnicity, 1996–97

	Number			Rate			Ratio		Excess	
	Māori	Pacific	Eur	Māori	Pacific	Eur	Māori	Pacific	Māori	Pacific
PAM	855	202	3685	254	179	100	2.5	1.8	520	90
SAM	400	121	1781	125	110	48	2.6	2.3	250	70
TAM	346	102	1534	98	85	44	2.2	1.9	190	50
Total avoidable mortality	1601	424	7000	477	374	192	2.5	1.9	970	210
Unavoidable mortality	543	168	3150	163	148	85	1.9	1.7	260	70
Total all mortality	2144	592	10,150	640	523	278	2.3	1.9	1240	280
Avoidable mortality as % all mortality	75	72	69							

Source of base data: NZHIS (1997 data are provisional)
Notes: number = number of deaths, averaged over 1996 and 1997; rate = age standardised rate per 100,000; ratio = ratio of rate to Eur (= European and other ethnic groups); excess = number of excess deaths in group compared to European/Other group.

The major contributor to avoidable mortality in all ethnic groups is IHD (Table 86). Higher smoking rates, poorer diet and lower levels of physical activity all contribute to this ethnic differential. Higher Māori smoking rates also contribute to other causes of avoidable mortality, including lung cancer and CORD. Diabetes, itself related to diet and physical activity levels, also makes a major contribution to the gap in avoidable mortality (for both Māori and Pacific people), both as a direct cause of death and indirectly as a risk factor for IHD and stroke. The differential impact of diabetes on mortality mainly reflects higher incidence. Another factor is less access to high quality primary health care, a situation that leads to more rapid progression to complications and higher case fatality (Simmons 1996a). Higher rates of fatal road traffic injuries (70 excess deaths per year in the 0–74 age group), SIDS (50 excess deaths) and rheumatic heart disease (30 excess deaths) also contribute to the high Māori burden of avoidable mortality.

Table 86: Major causes of avoidable mortality, ages 0–74, by ethnicity, 1996–97

	Top four conditions, by number of deaths 1996–97	Deaths per year	Percentage of deaths	Excess deaths
Māori	IHD	379	18	230
	Lung cancer	179	8	130
	Road traffic injury	131	6	70
	Diabetes	120	6	110
Pacific	IHD	102	17	50
	Diabetes	32	5	30
	Lung cancer	30	5	10
	Stroke	26	4	20
European/Other	IHD	1969	20	
	Lung cancer	717	7	
	Colorectal cancer	601	6	
	CORD	496	5	

Source of base data: NZHIS (1997 data are provisional)
Notes: percentage is of all 0–74 deaths (including unavoidable deaths) by ethnic group; 'excess deaths' are in comparison to European/Other.

Variations by socioeconomic status

For this analysis, the NZDep96 index was used to stratify all avoidable and unavoidable deaths occurring in 1996 and 1997 (provisional). The NZDep96 scores were then grouped into deciles. To increase statistical stability and ease data presentation, for some analyses the deciles have been collapsed asymmetrically (reflecting the curvilinear relationship between deprivation and mortality) to create five deprivation strata: deciles 1–4 (least deprived group), deciles 5–6, deciles 7–8, decile 9, and decile 10 (most deprived group).

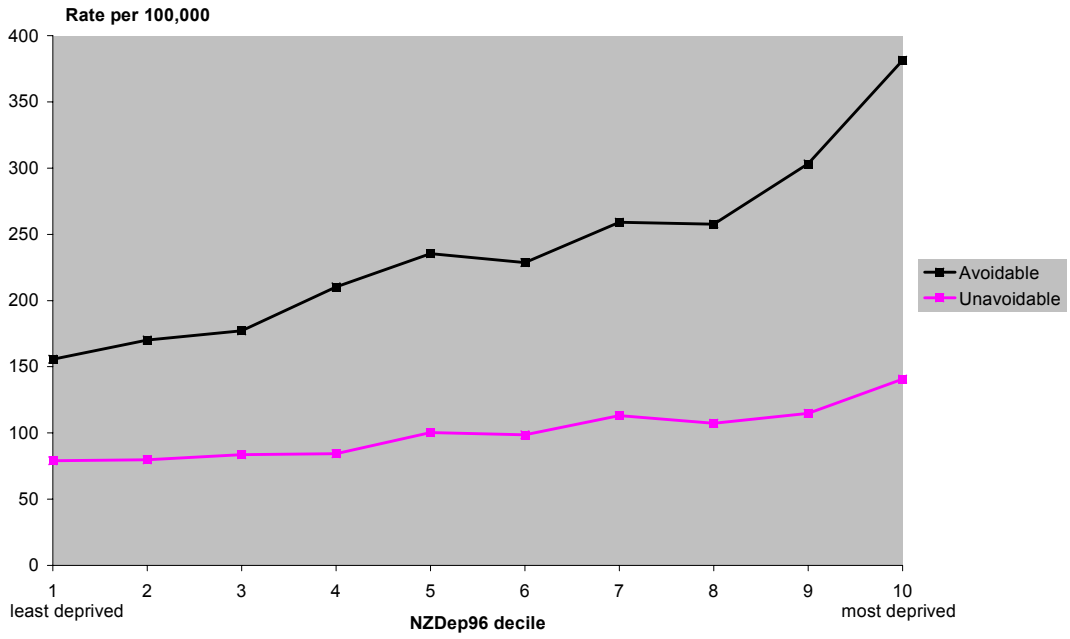
Table 87 and Figure 155 illustrate the strong relationship between deprivation and both avoidable and unavoidable mortality.

Table 87: Avoidable mortality, ages 0–74, by NZDep96 decile, 1996–97

	Number of deaths per year, by NZDep96 decile					Rate per 100,000, by NZDep96 decile					Ratio
	1–4	5–6	7–8	9	10	1–4	5–6	7–8	9	10	
PAM	1403	925	1080	597	690	92	121	136	165	202	2.2
SAM	697	457	526	267	337	45	58	66	73	98	2.2
TAM	622	388	436	234	277	43	53	57	66	81	1.9
Total avoidable mortality	2722	1770	2042	1098	1304	180	232	259	303	382	2.1
Unavoidable mortality	1269	770	876	419	485	82	99	110	115	141	1.7
Total mortality	3991	2539	2918	1517	1789	261	332	369	418	522	2.0
AM as % of all deaths	68	70	70	72	73						

Source of base data: NZHIS (1997 data are provisional)
Notes: ratio is decile 10 to decile 1–4; rate is age standardised to Segi's world population.

Figure 155: Avoidable and unavoidable mortality, by NZDep96 decile, ages 0–74, 1996–97



Source of base data: NZHIS (1997 data are provisional)
Note: rate is age standardised to Segi's world population.

One measure of health system performance (including health promotion and disease prevention as well as treatment and rehabilitation services) is the extent to which the system is able to mitigate the impact of social inequality on the health of the population. This is reflected in the relative steepness of the socioeconomic mortality gradients.

In New Zealand in 1996–97, the slope of the socioeconomic gradient in mortality for avoidable causes was steeper than that for unavoidable causes. Among avoidable causes, TAM had the shallowest gradient, suggesting that treatment services are being accessed more equitably than preventive services. The gradient was slightly steeper for SAM and PAM: people living in the most deprived areas had age standardised SAM and PAM rates more than double those of people living in the least deprived areas. This suggests that the health sector could do more to meet the preventive and chronic disease management needs of low income and less educated people (especially in primary health care settings). Much the same applies to PAM: affluent people are better able to respond to health education messages and have the necessary resources to make and sustain lifestyle changes. Smoking, a high fat low fruit and vegetable diet, physical inactivity, obesity, unrecognised or poorly controlled high blood pressure and type 2 diabetes are now becoming increasingly concentrated into poorer neighbourhoods (Ministry of Health 1999d; 1999a).

A complex interaction exists between ethnicity and deprivation in New Zealand: Māori and Pacific people are heavily concentrated into the more deprived NZDep96 strata (see Figure 25). This analysis does not attempt to disentangle ethnic or cultural from socioeconomic or structural effects. The analysis of life expectancy presented in Chapter 2, as well as earlier work (Pearce et al 1993), suggests that both sets of factors contribute independently and jointly to mortality differentials.

The impact of socioeconomic inequality on mortality is summarised in Table 88. The size of this impact is noteworthy: if mortality in all socioeconomic groups equalled that of the least deprived neighbourhoods (NZDep96 deciles 1–4), approximately 2850 fewer deaths would have occurred in New Zealand in 1996 or 1997 in the 0–74 age group, including 2160 fewer 'avoidable' deaths. This represents 18 percent of unavoidable and 24 percent of avoidable deaths.

Table 88: Excess deaths from avoidable causes, by NZDep96 decile, 1996–97

	Excess deaths, by NZDep96 decile					Percentage
	5–6	7–8	9	10	Total	of deaths
PAM	220	350	260	380	1220	26
SAM	110	170	100	180	560	25
TAM	70	110	80	130	390	20
Total avoidable deaths	400	620	450	690	2160	24
Unavoidable deaths	140	230	120	200	690	18
Total all deaths	540	850	570	890	2850	22

Source of base data: NZHIS (1997 data are provisional)
Notes: excess deaths are relative to least deprived group (deciles 1–4); percentage is by row.

With few exceptions (certain cancers such as melanoma, and birth defects) all the conditions included in the 'avoidable mortality' category show a socioeconomic gradient in cause specific mortality. About half the excess avoidable deaths among people living in deprived areas can be attributed to IHD, stroke, diabetes, lung cancer and CORD. These conditions are common and have steep socioeconomic gradients, with at least a two- to threefold difference in age standardised mortality rates between extreme groups (see Box 26). Table 89 summarises the

10 major contributors to the excess avoidable mortality of deciles 5–10 relative to deciles 1–4. Smoking contributes to each of the top three causes, and to several others as well. Diabetes is also a major risk factor, contributing to IHD and stroke deaths as well as directly to excess avoidable mortality.

Table 89: Major causes of excess mortality, by NZDep96 decile, ages 0–74, 1996–97

	Number of excess deaths per year, by NZDep96 decile					Percentage of all deaths from cause
	5–6	7–8	9	10	Total	
IHD	100	180	110	180	570	22
Lung cancer	60	70	70	80	280	30
CORD	50	70	50	50	220	36
Diabetes	20	40	20	70	150	48
Stroke	20	40	30	40	120	26
Colorectal cancer	40	40	10	10	90	14
Road traffic injury	20	20	30	30	90	19
Suicide	10	30	10	20	70	13
SIDS	10	20	10	20	60	67
Respiratory infections	5	20	10	20	50	41
Top 10 contributors	330	520	350	520	1720	

Source of base data: NZHIS (1997 data are provisional)
Notes: percentage is of all deaths from the cause; totals may not sum due to rounding; excess deaths are relative to least deprived group (NZDep decile 1–4).

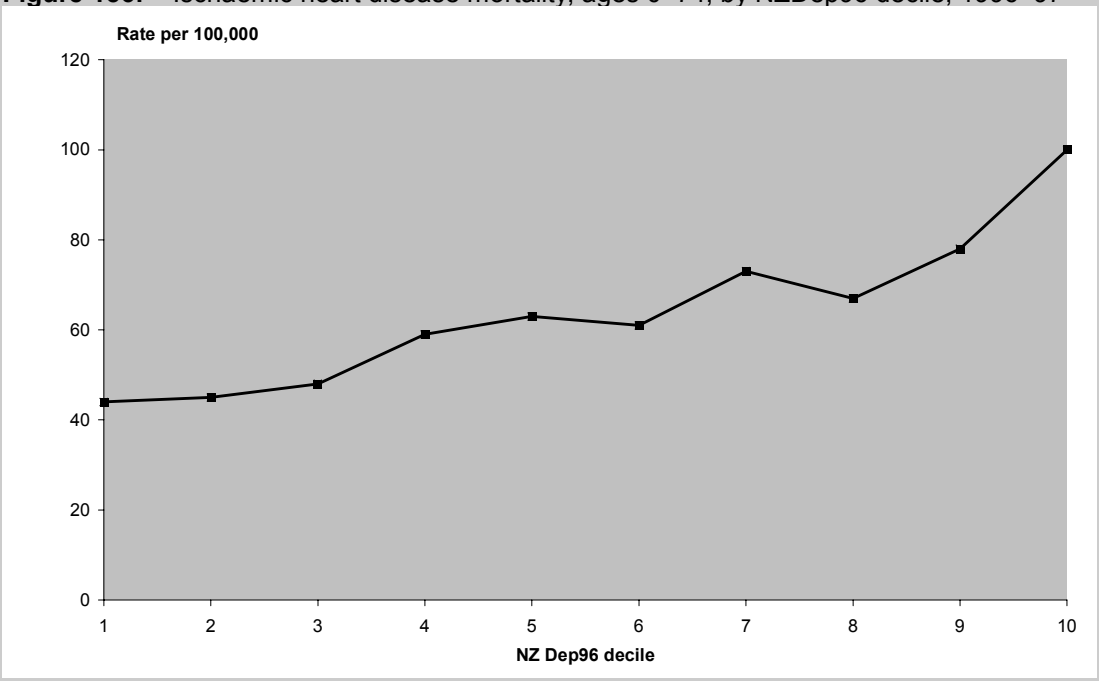
Unintentional injury also shows a steep socioeconomic gradient in avoidable mortality, with road traffic injuries being ranked within the top 10 causes. Factors involved include the quality of the fleet (new vehicles are safer), use of motorbikes (which are cheaper than cars), and behavioural factors (average speeds, drink drive rates). For children, differential exposures include traffic calming, traffic density, safe play areas, fencing, car seat, seat belt and cycle helmet use, and parental supervision.

Another cause worthy of note is SIDS: no fewer that two thirds of SIDS deaths would have been avoided if the risk in all areas were the same as that in the least deprived areas, reflecting a six fold variation in SIDS rates across NZDep96 strata. Up to half of SIDS may currently be attributed to maternal smoking behaviour (Mitchell et al 1997), which would explain part but not all of this gradient.

Box 26: Socioeconomic gradients in avoidable IHD mortality

IHD is by far the largest single contributor to the socioeconomic gradient in avoidable mortality. In 1996–97 it caused an average of 2541 deaths per year in the 0–74 age group – approximately 20 percent of total mortality in this age range. IHD mortality shows a statistically significant gradient from an age and gender standardised rate of 49 per 100,000 in the least deprived decile to 100 per 100,000 in the most deprived – a rate ratio between extreme deciles of almost exactly two (Figure 156).

Figure 156: Ischaemic heart disease mortality, ages 0–74, by NZDep96 decile, 1996–97



Source of base data: NZHIS (1997 data are provisional)
Note: rate is age standardised to Segi's world population; decile 1 = least deprived, decile 10 = most deprived.

Approximately 570 deaths occurring in 1996 or 1997 might have been postponed had all areas experienced the IHD mortality of the least deprived stratum. This represents 22 percent of all IHD mortality in the 0–74 age group.

This scope for health gain remains despite dramatically falling IHD mortality over the past two or three decades in New Zealand, suggesting that the trend may have been largely restricted to people living in more advantaged circumstances (Kawachi et al 1991). Evidence in support of this comes from surveys indicating declining rates of smoking and lower mean blood pressure among higher income or more educated adults, but stable or even rising levels of these risk factors among more deprived people (Ministry of Health 1999d). Similarly, the prevalence of obesity has risen dramatically over the past one or two decades, but probably less so among the more advantaged (Ministry of Health 1999a).

The analysis presented here supports the view that lifestyle changes are more easily made by better educated and more affluent people, who have the necessary resources to make and sustain such changes. By contrast, less advantaged New Zealanders may require additional support, often at a policy level, to gain similar benefit.

Avoidable morbidity

Introduction

The concept of avoidability can be extended from fatal to non-fatal outcomes. Limitations in the measurement of the latter, however, necessitate the use of hospitalisation as a proxy for disease (or injury) severity: a potentially avoidable hospitalisation therefore signals the occurrence of a severe illness or injury that, theoretically, could have been avoided.

Potentially avoidable hospitalisations fall into two subcategories:

- *preventable hospitalisations* (PH) – hospitalisations resulting from diseases preventable through population-based health promotion strategies (eg, tobacco excise tax, smokefree laws)
- *ambulatory sensitive hospitalisations* (ASH) – hospitalisations resulting from diseases sensitive to prophylactic or therapeutic interventions deliverable in a primary health care setting (such as vaccine preventable diseases, early recognition and excision of melanoma, effective glycaemic control in people with diabetes).

As with avoidable mortality, all causes of hospitalisation can be categorically attributed as (potentially) avoidable* or unavoidable, and the former further subdivided into the subcategories of 'preventable' and 'ambulatory sensitive'. For some causes, there is extensive overlap between the two subcategories, and judgement had to be applied to partition cause specific hospitalisations between them. The majority of categories attributed to preventable hospitalisation are those identified as causes of primary avoidable mortality; others are derived from the literature review. The ambulatory sensitive codes are largely derived from lists prepared by earlier workers (Weissman et al 1992; Begley et al 1995; Billings et al 1996; Jackson et al 1998), and were extended where necessary to reflect recent developments in health care technology and New Zealand practice patterns. A list of the conditions included and the key references used is appended (Appendix 3).

In the analysis that follows, injury admissions have been separated from other cause groups of preventable hospitalisations to reflect the different epidemiology of injury versus (preventable) disease: injuries have different risk and protective factors and respond to different prevention strategies. The analysis therefore presents avoidable hospitalisations in three subcategories:

- preventable hospitalisations (excluding injuries) (PH)
- ambulatory sensitive hospitalisations (ASH)
- hospitalisations avoidable through injury prevention (IP).

Ambulatory sensitive hospitalisations are sometimes monitored as a performance indicator for primary health care. In this report, however, this measure is used purely as an indicator of the scope for health gain – the potential to reduce the incidence of severe disease in the population. As with avoidable mortality, the analysis is restricted to the population aged less than 75 years.

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* A potential cause of confusion is the categorisation of admissions as 'discretionary' or 'non-discretionary'. These terms are not synonymous with the concept of avoidability. For example, an admission for appendicitis is non-discretionary and unavoidable, but an admission for ruptured appendix is non-discretionary yet avoidable.

Method and data sources

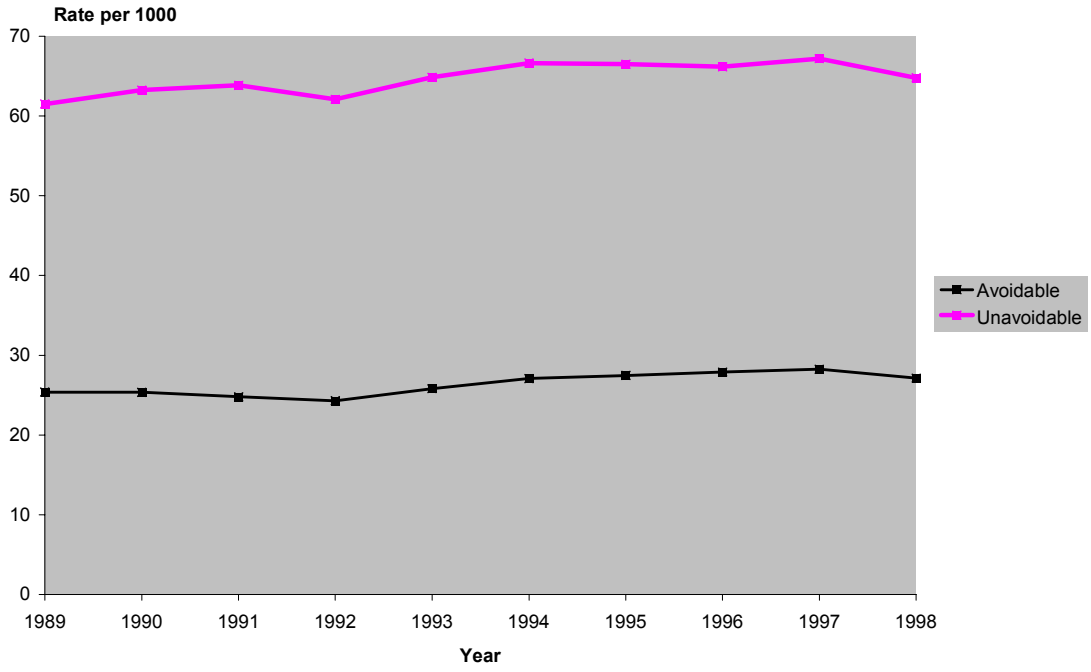
Discharges of persons aged 0–74 years from public hospitals for 1989–98 were analysed (1998 data are provisional). Detailed analysis of current levels uses the average of the two year period 1997–98. The discharge dataset provided by NZHIS was cleaned using a data filtering process developed by the Ministry of Health (1998c). Day cases, small rural hospitals, private hospitals, maternity and neonatal services, mental health services, and disability support services are excluded.

Avoidable hospitalisations in the whole population

In 1997–98 there were an annual average of 329 659 inpatient discharges from public hospitals (excluding the hospitals and services listed above) among persons aged 0–74 years. Of these, 97,390 or 30 percent are considered potentially 'avoidable'. Preventable hospitalisations accounted for 15,932 discharges (4.8 percent of total discharges within the age range), ambulatory sensitive hospitalisations for 63,721 (19.3 percent), and injuries for 17,736 (5.4 percent).

Since 1989, the age standardised rate of hospitalisation (0–74 age group) has *increased*. Both unavoidable and avoidable hospitalisations have shown a similar rising trend (Figure 157).

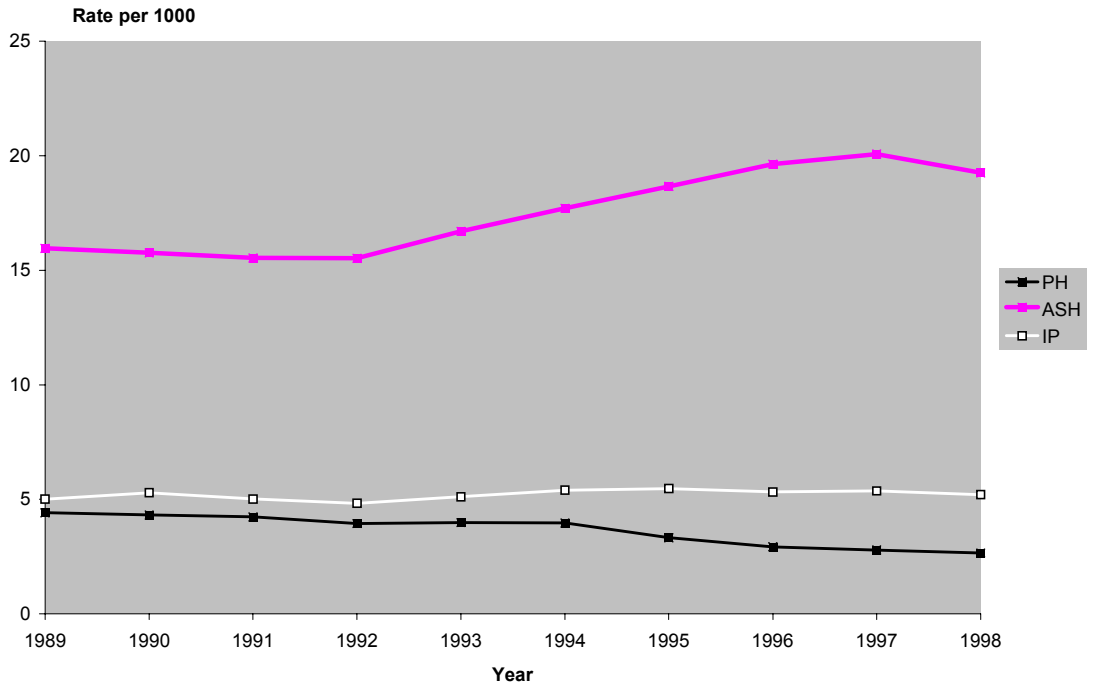
Figure 157: Avoidable and unavoidable hospitalisations, ages 0–74, 1989–98



Source of base data: NZHIS (1998 data are provisional)
Note: rate is age standardised to Segi's world population.

However, when avoidable hospitalisations are disaggregated into the three subcategories (Figure 158), it can be seen that the PH rate has in fact declined over the decade. By contrast, the ASH rate has risen dramatically. IP hospitalisations have also increased, but much less steeply.

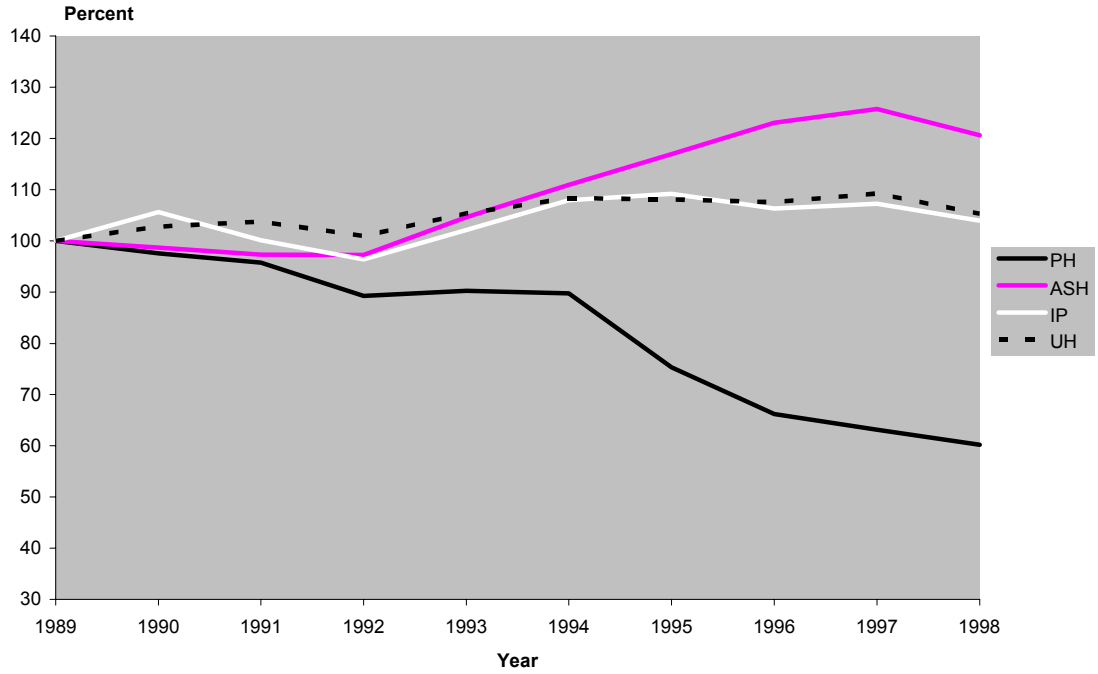
Figure 158: Avoidable hospitalisations, by subcategory, ages 0–74, 1989–98



Source of base data: NZHIS (1998 data are provisional)
Note: rate is age standardised to Segi's world population; PH = preventable hospitalisation; ASH = ambulatory sensitive hospitalisation; IP = hospitalisation avoidable by injury prevention.

These trends can be seen more clearly in Figure 159, which indexes rates to a base of 100 in 1989. The figure also shows the relative trend in unavoidable hospitalisations for comparison.

Figure 159: Relative trends in categories of hospitalisations, ages 0–74, 1989–98



Source of base data: NZHIS (1998 data are provisional)
Note: scaled to 1989 = 100; UH = unavoidable hospitalisations; broken axis.

The 40 percent fall in preventable hospitalisations (PH) over the decade is a measure of the success of health promotion in reducing disease incidence. However, this success has been more than offset by the 25 percent increase in ambulatory sensitive hospitalisations (ASH) over the same period. The 6 percent rise in injury hospitalisations exactly mirrors the trend in the 'baseline' of unavoidable hospitalisations, and contributes little to the overall increase in the avoidable hospitalisation rate.

Almost all of the rise in avoidable hospitalisations – and over half of the rise in total hospitalisations (all categories combined) – can therefore be attributed to the trend in ASH, especially since 1993. A proportion of this apparent increase may be an artefact of improvements in data management and coding systems in hospitals, including the introduction of the Australian coding standards in 1995 and the introduction of case mix funding, which may have shifted some cases from the PH to the ASH subcategories. This could partly explain the sharp decline in PH at about the same time as the sharp rise in ASH.

Changes in the funding and delivery of health care are likely to have also contributed to the rising trend in ASH, including altered incentives for GPs and hospitals to refer or admit patients respectively arising from the health reforms of 1992–93, higher perceived risk of litigation if hospitalisation is deferred, and possibly increased barriers (geographic, cultural or socioeconomic) to access to primary health care experienced by some sections of the population (Ministry of Health 1998c).

The rising trend in ASH also reflects, to some (unquantified) extent, real increases in the prevalence of some chronic conditions. In a few cases this may reflect increasing incidence (for example, asthma, diabetes), but in most it reflects reductions in both cause specific and/or other cause mortality: people are living longer with the disease and so are admitted to hospital more often, or are surviving long enough to develop the disease in the first place.

Whatever the contributions of coding changes, changing patterns of health care funding and delivery, and real changes in chronic disease epidemiology, the result of this trend has been that, by 1997 or 1998, one in three hospitalisations of people aged 0–74 years were theoretically avoidable, two thirds of these through more effective primary health care. Although in practice all of these potentially avoidable hospitalisations could not realistically have been avoided, these estimates nevertheless reveal considerable scope for further reduction in the incidence of serious disease and injury. However, these whole of population estimates disguise significant variation among subgroups of the population.

Variations by age

Unlike avoidable mortality, avoidable hospitalisations are more evenly distributed across the 0–74 age range (Tables 90 and 91). The rate of AH is highest in infancy, with just over 1 percent of all infants being admitted in 1997 or 1998 for an avoidable cause, most commonly an infectious disease (gastroenteritis or respiratory tract infection). However, avoidable causes account for less than 20 percent of total hospitalisations in infancy. For children aged 1–14 years, the rates of avoidable and unavoidable hospitalisations are much lower, yet avoidable causes account for almost half (45 percent in 1997 or 1998) of the total – the highest proportion in any age group. The single leading cause of AH in childhood is asthma, which accounted for 18 percent of AH in this age group in 1997–98.

Avoidable causes account for 25 percent of all hospitalisations of youth (15–24 years). Injury admissions peak in this age group, with road traffic injuries and sports injuries heading the list. Road traffic injuries remain the leading cause of avoidable hospitalisation in young adults (25–44

years). Cellulitis (a type of skin infection, often complicating minor lacerations or abrasions) is a surprisingly common cause of AH for people aged 15–44. Pelvic inflammatory disease and ectopic pregnancy – both often complications of sexually transmitted infections, especially chlamydia – are common avoidable reasons for admission among females aged 25–44.

From middle age the AH rate begins to rise with the emergence of chronic diseases, reflecting cumulative exposure to smoking, poor diet and physical inactivity over many decades. This pattern is further accentuated in old age (65–74 years). From age 45 onwards, one in three hospitalisations is potentially avoidable, mostly through effective chronic disease and risk factor management in the primary care setting. It is worth noting that both the sharp upward trend in ASH from 1993 onwards and the sharp downward trend in PH from 1994 onwards are largely restricted to the 45 and over age groups.

Table 90: Avoidable hospitalisations, by age, 1997–98

	Discharges per year (number)					
	Age: < 1	**1–14**	**15–24**	**25–44**	**45–64**	**65–74**
PH	423	660	386	1745	6387	6331
ASH	5213	14,414	5346	10,932	15,312	12,505
IP	80	4970	4467	5559	1995	666
AH (total)	5716	20,044	10,198	18,236	23,695	19,503
UH	24,431	24,127	29,925	77,904	44,559	31,324
Total	30,147	44,171	40,123	96,139	68,253	50,827
AH as % total	19	45	25	19	35	38
	Discharges per year (age specific rate)					
	Age: < 1	**1–14**	**15–24**	**25–44**	**45–64**	**65–74**
PH	8	1	1	2	9	26
ASH	93	19	10	10	21	51
IP	1	6	8	5	3	3
AH (total)	102	26	19	16	33	79
UH	437	31	56	70	62	127
Total	539	57	75	87	95	206

Source of base data: NZHIS (1998 data are provisional)
Notes: rate is per 1000 population, UH = unavoidable hospitalisations.

Table 91: Major causes of avoidable hospitalisations, by age, 1997–98

Age (years)	Condition	Number of discharges per year	Percentage
< 1	Gastroenteritis	1579	5
	Respiratory infection	1441	5
	Failure to thrive	477	2
	Kidney/urinary infections	394	1
1–14	Asthma	3627	8
	Respiratory infection	3263	7
	Gastroenteritis	1987	4
	Road traffic injury	1871	4
15–24	Road traffic injury	2157	5
	Sport injury	1126	3
	Cellulitis	998	2
	Asthma	888	2

Table 91 continued

Age (years)	Condition	Number of discharges per year	Percentage
25–44	Road traffic injury	2501	3
	Cellulitis	1929	2
	Sexually transmissible infections	1394	1
	Self harm	1332	1
45–64	Angina	4418	6
	Myocardial infarction	2683	4
	Respiratory infection	1497	2
	CORD	1424	2
65–74	Angina	3984	8
	Myocardial infarction	2279	4
	CORD	2200	4
	Congestive heart failure	1528	3

Source of base data: NZHIS (1998 data are provisional)
Notes: percentage is of total hospitalisations for each age group; IHD has been subdivided into myocardial infarction, angina and heart failure, to distinguish first episodes of IHD (which are preventable) from subsequent management of chronic IHD (which is ambulatory sensitive); sexually transmissible infections include complications (pelvic inflammatory disease, ectopic pregnancy).

Variations by gender

Among people aged 0–74 in 1997 or 1998, males had a rate of AH that was 18 percent higher than that of females (Table 92).

Table 92: Avoidable hospitalisations, by gender, ages 0–74, 1997–98

	Number of discharges per year			Rate		Ratio	Male
	Male	Female	Total	Male	Female	M:F	excess (deficit)
PH	9416	6516	15,932	4.6	3.4	1.4	2610
ASH	32,272	31,449	63,721	19.2	18.4	1.0	1430
IP	10,936	6799	17,735	6.6	4.1	1.6	4190
AH (total)	52,624	44,764	97,388	30.5	25.8	1.2	8080
UH	93,045	139,220	232,265	55.5	78.4	0.7	(38,470)
Total	145,669	183,984	329,653	86.0	104.2	0.8	(30,940)
AH as % total	36	24	30				

Source of base data: NZHIS (1998 data are provisional)
Note: age standardised rates per 1000 population are standardised to Segi's world population.

The leading causes of AH for each gender are shown in Table 93. Respiratory infections (mainly pneumonia and influenza) are the leading overall cause over the 0–74 age range for both males and females. Road traffic injury ranks second for males and fourth for females: had males experienced the same rate of road traffic injury hospitalisation as females in 1997, 2400 fewer males would have been hospitalised. Angina is ranked high among males, reflecting their higher prevalence of IHD. Urinary tract infections are highly ranked for females. Although cellulitis ranks highly for both, the male rate is 50 percent higher than the female rate, corresponding to an excess of approximately 1300 hospitalisations per year.

Table 93: Major causes of avoidable hospitalisation, by gender, ages 0–74, 1997–98

		Number of discharges per year	Rate	Male excess (deficit)
Male	Respiratory infections	4953	6.5	890
	Road traffic injury	5154	6.1	2380
	Angina	5675	5.4	2320
	Asthma	3455	4.8	(150)
	Cellulitis	3925	4.6	1270
	Sports injury	2754	3.3	1980
Female	Respiratory infections	4219	5.4	
	Asthma	4139	5.1	
	Gastroenteritis	3137	4.1	
	Road traffic injury	2762	3.3	
	Angina	3259	3.2	
	Urinary tract infection	2287	2.8	

Source of base data: NZHIS (1998 data are provisional)
Note: rate is age standardised (to Segi's world population) per 1000 population.

Variations by ethnicity

Māori and Pacific people experienced age standardised rates of AH 60 percent and 70 percent higher than European/Other New Zealanders in 1997–98 (Table 94). Had the ethnic specific rates been equal, 6800 fewer Māori and 2800 fewer Pacific people aged 0–74 years would have been hospitalised. The scope for equity gain is greatest for ASH, because this subcategory has both the highest rate ratio (approximately 2.0) and accounts for the largest proportion of total AH in all three ethnic groups.

Table 94: Avoidable hospitalisations by ethnicity, ages 0–74, 1997–98

	Number of discharges per year			Rate		
	Māori	**Pacific**	**European/Other**	**Māori**	**Pacific**	**European/Other**
PH	1849	633	13,007	5.4	5.6	3.7
ASH	12,872	5,040	44,333	28.5	33.7	16.1
IP	3304	878	12,493	6.0	4.8	5.2
AH (total)	18,025	6551	70,208	40.0	44.1	25.0
UH	36,854	15,472	173,716	75.8	92.0	61.6
Total	54,878	22,023	243,924	115.7	136.0	86.6
AH as % total	33	30	29	35	32	29
	Ratio of rates			**Number of excess discharges**		
PH	1.5	1.5		580	210	
ASH	1.8	2.1		5590	2630	
IP	1.2	0.9		550	(50)	
AH (total)	1.6	1.8		6760	2840	
UH	1.2	1.5		6880	5100	
Total	1.3	1.6		13,830	8010	

Source of base data: NZHIS (1998 data are provisional).
Notes: ratio of rates is to European/Other ethnic group; rates are per 1000, age standardised to Segi's world population; UH = unavoidable hospitalisations.

The leading causes of avoidable hospitalisations (across the whole 0–74 age range) for each ethnic group are summarised in Table 95. For both Māori and Pacific people, three of the top five causes are infectious diseases – respiratory infections, gastroenteritis and cellulitis – reflecting both social conditions and primary health care access (for example, immunisation, antibiotics, oral rehydration, skin care). Māori and Pacific people also have high rates of hospitalisation for asthma, diabetes and other chronic diseases. These causes again reflect socioeconomic status, but are also responsive to primary health care (effective chronic disease and risk factor management), a situation similar to that already described for SAM.

Table 95: Top five causes of avoidable hospitalisation, by ethnicity, ages 0–74, 1997–98

		Number of discharges per year	Percentage	Excess (number)
Māori	Respiratory infection	2357	4	1240
	Asthma	2107	4	1030
	Cellulitis	1531	3	790
	Road traffic injury	1456	3	230
	Gastroenteritis	938	2	(250)
Pacific	Respiratory infection	1127	5	760
	Cellulitis	879	4	640
	Asthma	633	3	300
	Gastroenteritis	351	2	(20)
	Road traffic injury	314	1	(100)
European/ Other	Angina	7770	3	
	Road traffic injury	5611	2	
	Respiratory infection	5296	2	
	Myocardial infarction	4558	2	
	Asthma	4525	2	

Source of base data: NZHIS (1998 data are provisional)
Notes: percentage is of total hospitalisations for each ethnic group; IHD is divided into subcategories (angina, myocardial infarction, congestive heart failure).

Variations by socioeconomic status

As for mortality, the NZDep96 index was used to assign a deprivation score (aggregated into deciles 1–4, 5–6, 7–8, 9 and 10) to each discharge based on the domicile of residence. People living in the most deprived areas (decile 10) had twice the probability of being hospitalised for an avoidable cause as people living in the least deprived areas (deciles 1–4) (Table 96 and Figures 160a and 160b). Had all New Zealanders enjoyed the AH rates of deciles 1–4, 28 percent fewer avoidable hospitalisations would have occurred in 1997 or 1998 – an annual 'saving' of approximately 26,000 hospital admissions. This is equivalent to the annual inpatient throughput of a middle sized hospital such as Whangarei or Palmerston North.

Table 96: Avoidable hospitalisations, by NZDep96 decile, ages 0–74, 1997–98

	Number of discharges per year, by NZDep96 decile					Rate per 1000					Rate ratio
	1–4	5–6	7–8	9	10	1–4	5–6	7–8	9	10	
PH	4713	3234	3628	2056	2097	6.3	8.6	9.7	11.2	12.4	1.9
ASH	17,248	12,633	14,015	8453	10,675	27.8	39.9	44.3	50.8	63.4	2.3
IP	5291	3639	3848	2309	2447	8.9	11.9	12.5	13.6	14.2	1.6
AH (total)	27,252	19,506	21,491	12,819	15,219	43	60	66	76	90	2.1
UH	70,123	46,996	50,427	28,898	33,106	110	144	154	168	194	1.8
Total	97,375	66,501	71,917	41,717	48,324	153	205	221	243	284	1.9
AH as % total	28	29	30	31	31	28	29	30	31	32	

Source of base data: NZHIS (1998 data are provisional)
Notes: NZDep96 deciles are grouped into five strata: deciles 1–4 is the least and decile 10 the most deprived; rate ratio is decile 10 to deciles 1–4.

Figure 160a: Avoidable and unavoidable hospitalisations, by NZDep96 decile, ages 0–74, 1997–98

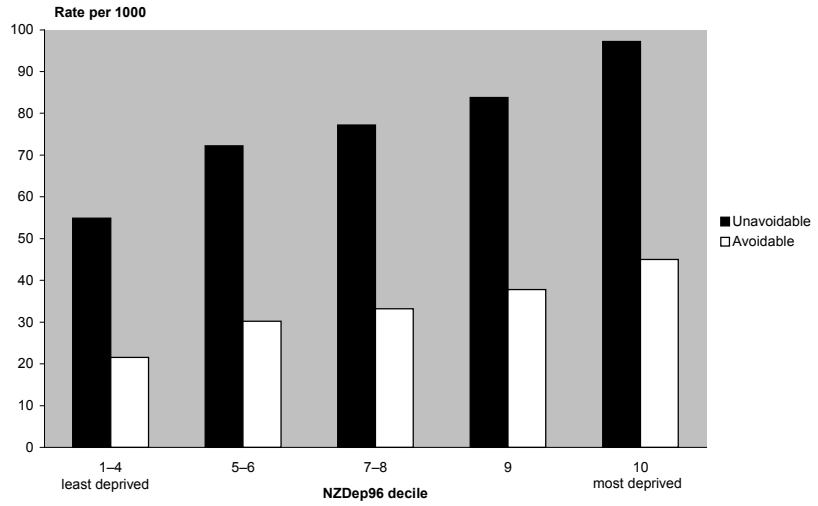
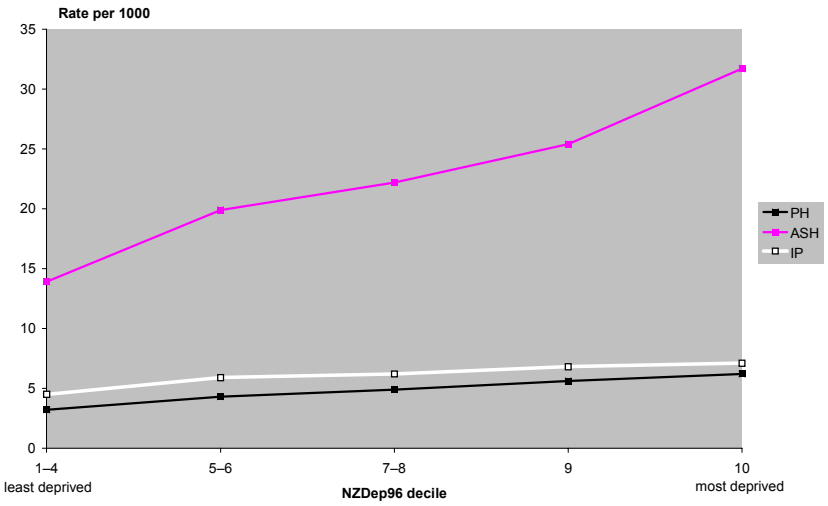


Figure 160b: Subcategories of avoidable hospitalisations, by NZDep96 decile, ages 0–74, 1997–98



Source of base data: NZHIS (1998 data are provisional)
Note: rate is age standardised to Segi's world population.

Higher rates of both avoidable and unavoidable hospitalisation are seen for people living in deprived areas. However, the excess varies depending on subcategory (Tables 96 and 97). For unavoidable hospitalisations, the ratio of age standardised rates between people in NZDep96 decile 10 and those in deciles 1–4 was 1.8 in 1997–98. For both injury and other preventable hospitalisations, the corresponding ratio was close to or less than 1.8, suggesting some success in ameliorating the impact of social inequality with respect to these causes. However, for ASH the corresponding ratio was 2.3 – significantly higher than the 1.8 'baseline'. This suggests that people from deprived areas may be benefiting less from (effective) primary health care than their more advantaged counterparts (especially with regard to preventive services, including chronic disease management).

Table 97: Excess discharges, by NZDep96, ages 0–74, 1997–98

	Excess discharges by NZDep96 decile, compared with deciles 1–4					Percentage of discharges
	5–6	7–8	9	10	Total	
PH	840	1250	890	1020	4000	25
ASH	3810	5200	3820	5990	18,820	30
IP	920	1090	790	910	3720	21
AH (total)	5580	7530	5510	7930	26,550	28
UH	11,270	14,590	9980	14,410	50,260	22
Total	16,860	22,140	15,490	22,350	76,840	24

Source of base data: NZHIS (1998 data are provisional)
Note: percentage is of total hospitalisations for each deprivation stratum.

The major causes contributing to the socioeconomic gradient in AH are listed in Table 98. Respiratory infections were the single largest contributor to the gradient in 1997–98. However, if all forms of IHD were combined into a single category, this would be the leading cause. (These conditions have been separated to distinguish first episodes of IHD, which are preventable, from subsequent management of chronic IHD, which is ambulatory sensitive.) Other respiratory disorders also feature prominently, with asthma in second place and CORD ranked fifth. Diabetes would rank even more highly if its macrovascular complications were included within this category. Although injury is also a significant contributor to the socioeconomic gradient in AH, the only injury type to appear in the top 10 causes is road traffic injury, accounting for 1580 excess hospitalisations (20 percent of total hospitalisations for this cause).

Table 98: Major causes of excess hospitalisation, by NZDep96 decile, ages 0–74, 1997–98

	Excess discharges per year by NZDep96 decile, compared with deciles 1–4					Percentage of total for cause
	5–6	7–8	9	10	Total	
Respiratory infection	520	750	580	1200	3050	34
Asthma	490	690	530	750	2470	33
Cellulitis	410	550	430	880	2280	35
Angina	660	740	420	410	2240	25
CORD	300	440	380	500	1620	41
Road traffic injury	410	430	320	410	1580	20
Congestive heart failure	190	270	240	440	1140	41
Epilepsy	230	330	210	310	1080	30
Myocardial infarction	240	340	190	210	980	18
Diabetes	140	260	190	340	920	41
Top 10 causes	3600	4800	3500	5450	17,350	18

Source of base data: NZHIS (1998 data are provisional)
Note: percentage is proportion of total hospitalisations for the specific cause in 1997–98 (averaged).

Summary and conclusions

Categorical attribution of diseases and injuries (coded as causes of death or hospitalisation) as 'avoidable' or 'unavoidable' provides one way to identify the scope for health gain. This categorisation is not meant to imply that every death or hospitalisation classed as 'avoidable' could in fact have been avoided – merely that the potential to do so exists. For this analysis, the categorisation of causes has been taken one step further, subdividing both avoidable mortality and avoidable hospitalisations into three subcategories, representing different levels of intervention within the health sector.

An upper age limit of 75 years has been used in this analysis. This does not mean that some deaths or hospitalisations involving people older than 75 years could not have been avoided; only that disentangling avoidable from unavoidable causes becomes problematic as the prevalence of co-morbidity increases. Indeed, it is conventional in this field to use a cut-off of 65 years; however, a higher upper age limit better reflects recent advances in coding and provides an analysis of greater policy relevance.

Mortality

Avoidable mortality in the 0–74 age range declined by 38 percent from 1981 to 1997, compared with a decline of only 9 percent in unavoidable mortality. This difference in trend (amounting to approximately 5000 fewer deaths in 1997 than expected, based on 1981 mortality rates) is a measure of the success of the health system (including population-based interventions) in reducing fatal outcomes. Even so, in 1996–97 almost 70 percent of deaths in the 0–74 age range (approximately 9000 deaths per year) were still considered to be potentially avoidable.

Although the proportion of deaths categorised as avoidable does not vary much across age groups, the exponential rise in mortality with age means that almost 80 percent of all avoidable deaths occur in the 45–74 age group. These deaths are dominated by the emergence of chronic diseases such as IHD, diabetes and smoking related cancers. In younger age groups, injury (including suicide) dominates avoidable mortality. Not surprisingly, these age groups have experienced less improvement in avoidable mortality rates over the past one to two decades than have the older age groups.

Males experience a greater burden of avoidable mortality than females – a relative excess of 54 percent (corresponding to approximately 2000 excess avoidable deaths) in 1996–97. The gender difference is largely attributable to diseases and injuries amenable to primary prevention, with the largest single contribution coming from IHD. The downward trend in avoidable mortality since 1981 has been steeper for males than females, narrowing the gender differential.

The ethnic gap in avoidable mortality remains wide: rates for Māori and Pacific people were 2–2.5 times higher than European/Other rates in 1996–97. In absolute terms, the greatest scope for narrowing the ethnic gap is in primary prevention – reducing disparities in socioeconomic status and in lifestyle (smoking, diet, physical activity).

Similar gradients are seen with socioeconomic status, using the NZDep96 index of deprivation. Eliminating the socioeconomic gradient in AM would postpone over 2000 deaths per year. The gradient is steepest for SAM and flattest for TAM, mirroring the ethnic differences. People with lower socioeconomic status, and Māori and Pacific people, appear less able to access preventive/ primary health care services. Health education messages may be less relevant or culturally appropriate for these groups; alternatively, the resources needed to respond to these messages may be less readily available.

Morbidity

Unlike mortality, over the past decade the age standardised rate of hospitalisation has steadily increased, and this rising trend applies to both avoidable and unavoidable causes. But disaggregating avoidable hospitalisations into subcategories reveals a more complex pattern: injury hospitalisation rates have increased in line with unavoidable hospitalisations, preventable hospitalisation rates have declined by 40 percent, and ambulatory sensitive hospitalisations (the largest subgroup of AH) have increased by 25 percent (most of this increase involving the 45–74 age group and occurring since 1993).

The increase in ASH has multiple causes, including changes in incentive structures and practice patterns emanating from the health reforms of 1992–93, improvements in hospital technology, increases in the incidence and/or prevalence of some chronic diseases, and artefact arising from coding changes.

Whatever the contributions of each of these causes, the trends described in this chapter indicate that significant scope exists to reduce hospitalisation rates, mainly through (integrated) primary care. In 1996–97, almost 100,000 potentially avoidable hospitalisations occurred – about 30 percent of total inpatient admissions in the 0–74 age range (excluding psychiatric and maternity services, small rural hospitals and private hospitals). Almost two thirds of these were judged to be ambulatory sensitive.

These avoidable hospitalisations, unlike avoidable deaths, were relatively evenly spread throughout the age range. The proportion of hospitalisations characterised as avoidable varied from 20 percent among infants to 45 percent among older children. Infections dominated this category among infants, asthma ranked first among older children, and injury dominated among the 15–44 age range (mainly road traffic and sports injuries). From middle age, chronic diseases emerge as cumulative exposure to tobacco, poor diet and physical inactivity – often over decades – begins to take its toll. From 45–74 years at least one in three hospitalisations could be avoided through a combination of health promotion and clinical preventive services, including effective management of chronic diseases and their risk factors.

The ethnic and socioeconomic inequalities seen in avoidable mortality are also present in avoidable hospitalisation. Māori and Pacific people have age standardised AH rates approximately 60 percent higher than those of European/Other New Zealanders, corresponding to 6600 and 2800 excess hospitalisations among Māori and Pacific people respectively in 1997. The best opportunity for narrowing the ethnic gap is in the ASH subcategory, which has the highest ethnic excess (almost twofold) and accounts for the largest proportion of avoidable hospitalisations. This means improving the access of Māori and Pacific people to culturally appropriate and effective primary health care.

As with mortality, both avoidable and unavoidable hospitalisation rates are higher for people living in more deprived areas. The slope of the gradient is shallower for injury and other preventable hospitalisations than it is for unavoidable hospitalisations, but significantly steeper for ASH. As with SAM, this suggests that the health system, and in particular the primary care sector, could do more to mitigate the impact of social inequality on health outcomes.

This analysis of avoidable mortality and morbidity in New Zealand in the mid to late 1990s has revealed significant scope for the health sector to contribute to population health gain and, in particular, to improvement in equity of outcomes across ethnic and socioeconomic groups.

Chapter 11: Risks to health

Introduction

This chapter shifts the focus from 'avoidable' mortality and morbidity – health outcomes amenable to health sector intervention – to health risks. It aims to identify modifiable risk factors and the scope for health gain possible from further reductions in exposure of the population (or subgroups) to these risks, whether through health sector or intersectoral intervention.

Risks to health arising from exposure to biological (such as obesity) or behavioural (such as smoking) factors are quantified by counterfactual analysis using the epidemiological measure 'population attributable risk' (PAR). This concept, and its derivative measures – the attributable fraction (AF) and impact fraction (IF) – are explained in Box 27.

This is not the first use of attributable risk in New Zealand. Of particular relevance are two previous studies: the PARs for alcohol consumption were calculated by Scragg (1995), and those for a range of modifiable risk factors in relation to a number of chronic diseases by Galgali et al (1998). This report builds on these earlier studies and draws on more recent prevalence data from three sources: the 1996 Census of Population and Dwellings, the 1996–97 New Zealand Health Survey (Ministry of Health 1999d) and the 1997 National Nutrition Survey (Ministry of Health 1999a). In addition, the substantial Australian work of English et al (1995) provided a sound basis for developing estimates of attributable fractions for a large number of conditions associated with tobacco smoking and alcohol consumption.

Although attributable risk is decontextualised for this analysis, in reality risks are embedded within a social context. Indeed, policies aimed at modifying lifestyles and changing habitual behaviours could actually worsen health inequalities unless they are designed to be sensitive to different sociocultural contexts and to address the underlying social inequalities themselves.

Method and data sources

Risk factor prevalence

Three criteria were used to select risk factors for inclusion in this study:

- The risk factor was known on the basis of solid evidence to be causally related to at least one major category of diseases or injuries.
- Relative risk estimates were available from high quality, recent epidemiological studies, either carried out in New Zealand or in similar populations elsewhere.
- High quality, nationally representative estimates of prevalence of the risk factor in the New Zealand population (and its major demographic subgroups) were available from recent (1996 or later) surveys.

Based on these criteria, eight risk factors for a range of chronic diseases were selected (other than alcohol consumption, no injury risk factors met the selection criteria). The risk factors included in the study are:

- behavioural risk factors: smoking, alcohol consumption, inadequate fruit and vegetable consumption, and physical inactivity
- biological risk factors: diabetes (type 2), obesity, high blood pressure, and high blood cholesterol.

Fruit and vegetable consumption was the only dietary risk factor that could be included. However, the effect of diet is captured indirectly through several of the other risk factors (such as obesity for energy balance, cholesterol for fat intake and blood pressure for salt intake).

For each risk factor, the most recent survey was analysed. Where more than one data source was available, the most statistically robust was selected. Data smoothing techniques (for example, fitting of polynomial curves by least squares) were used for some age, gender and ethnic cells, where survey numbers were too small to provide robust estimates directly. The main surveys used were the 1996–97 New Zealand Health Survey and the 1997 National Nutrition Survey. Smoking data were obtained from the 1996 Census.

Relative risk estimates

An extensive review of New Zealand and international literature was carried out. Relative risk estimates were selected from published meta analyses where possible or, alternatively, from major cohort studies. To the extent possible, the relative risk estimates selected had been adjusted (de-confounded) for other risk factors, in order to represent the independent effect of the risk factor concerned in the absence of other risk factors. The relative risk estimates used are therefore conservative, leading to under-estimation of the corresponding attributable fractions.

Although some studies provided gender and age specific relative risks, the desired level of data differentiation was not always available; in these cases, modelling techniques were used to further differentiate the relative risk estimates by gender and age group. In the absence of data to the contrary, it had to be assumed that relative risks for both Māori and non-Māori ethnic groups were similar. Differences in attributable fractions between these ethnic groups are thus driven by differences in exposure to risk factors (prevalence) rather than by differences in biological responses to such exposures.

Attributable fractions

For estimation of attributable fractions (AFs), both the prevalence and the relative risk data must relate to similar risk categories. For this analysis, most risks were simply dichotomised. In reality, risks do not display clear thresholds but tend to be continuous. Recognising only two or three risk categories therefore loses some information but makes it easier to align categories used in prevalence and relative risk studies. The risk thresholds selected were those identified in current New Zealand guidelines, typically based on evidence showing a sharp increase in disease occurrence at levels of exposure above the threshold. For alcohol, three levels of exposure (and relative risks) were selected to accommodate the different levels of risk and protective effects; smoking included both current and past (ex) smoking exposure. The method of calculating attributable fractions is described in Box 27.

Deaths and years of life lost (YLL) attributable to each risk factor were estimated by applying the calculated attributable fractions to the 1996 mortality data. However, the number of deaths calculated by this method does not accurately represent the number actually caused by the risk factor in 1996; this is because *current* chronic disease mortality reflects exposure to risk factors some years in the *past* (when the risk factor prevalences may have differed from their current levels). Rather, the estimates indicate the number of deaths that could be prevented in the *future* were exposure to the risk factor to be eliminated, assuming no change in population size, age structure or ethnic composition.

The univariate analysis applied here assumes that risk factors are independently distributed and do not interact with each other. In reality, people are exposed to several risk factors simultaneously, and these may interact in complex ways; for example, the Auckland Heart and Health Study found that 29 percent of males and 33 percent of females had more than one cardiovascular risk factor (Bullen et al 1998). More sophisticated multivariate models are necessary to fully account for joint exposures to multiple risk factors. It should also be noted that the focus in this report is on mortality (data are unavailable for non-fatal outcomes in many cases). It cannot be assumed that the relative risk for disease incidence (or consequential disability) is the same as that for mortality.

Finally, in some cases exposure is protective (for example, alcohol and ischaemic heart disease (IHD), or smoking and Parkinson's disease). In these cases the PAR is negative and the attributable fraction can be used to calculate the deaths that would be avoided in the future if current exposure to that protective factor continued unchanged.

As well as simple counts (numbers of deaths), the results are analysed in terms of years of life lost (YLL, using the remaining life expectancy method). It is recognised that there is debate concerning discounting, and this chapter provides both undiscounted years of life lost (YLL) and discounted years of life lost (YLL_d, discounted at 3 percent per year). The attributable fractions are also applied to the disability adjusted life years (DALYs) calculated in Chapter 8, although in many cases relative risk estimates for incidence were unavailable, making estimation of attributable fractions for the YLD component of the DALY problematic (these latter results are reported in Chapter 8 and are not repeated here).

Potential future impact of risk reduction

The attributable fraction indicates the potential scope for health gain from eliminating the risk factor, but more realistic scenarios involving modest reductions in risk factor exposure are also worth exploring.

Using 1996 as the base year (the latest year for which data are available), impact fractions were estimated for the year 2006. A single scenario is reported here: a 10 percent reduction from current prevalence for most risk factors (for example, a risk factor with a 50 percent prevalence in 1996 is assumed to have a 45 percent prevalence in 2006). This scenario projects typical historical trends in risk factor prevalences over the past one or two decades, and for most risk factors is thought to represent an achievable goal. The reduction in exposure is assumed to occur uniformly across all age, gender and ethnic groups. For alcohol, exposure reduction was applied only to 'hazardous to harmful' levels of consumption, with a consequent increase in 'low to moderate' consumption levels; no change was modelled in the prevalence of abstainers. For tobacco, the modelled reduction in current smoking prevalence is assumed to result in a concomitant increase in ex smoking prevalence.

For two risk factors – diabetes and obesity – the historical trend is different: these risk factors have been steadily *increasing* in prevalence over the past several decades. So, for diabetes the scenario reported is one in which prevalence is held to current (1996–97) levels – that is, further increases in prevalence are avoided (through appropriate interventions). Therefore, the impact fractions for diabetes are calculated using the difference between projected prevalence levels for 2006 and the 1996–97 prevalence levels. The 2006 prevalence of type 2 diabetes was predicted using a multi state life table model (Tobias and Roberts 1999). For obesity, the prevalence in 2006 was obtained by simple linear projection of the historical trend in obesity prevalence over the period 1989–97. A 10 percent reduction from this predicted 2006 prevalence was then modelled.

The impact fractions were applied to the projected 2006 population (Statistics New Zealand medium projection). This provides an estimate of the deaths and YLL that could be avoided in 2006, given the scenario outlined above for each risk factor, and taking into account expected demographic trends (trends in all-cause mortality, population growth, population ageing, and changes in the ethnic composition of the population).

Box 27: Population attributable risks (PARs)

The usual measure of effect in epidemiology is the relative risk (risk or rate ratio):

$$RR = \frac{I_e}{I_u}$$

where I_e is the incidence of the disease of interest in the people exposed to the risk factor, I_u is the incidence in those not exposed (that is, the baseline risk or rate), and RR is the relative risk.

Yet relative measures are inadequate for policy makers. A high relative risk may be associated with a low baseline risk, and so the risk among the exposed may still be low in absolute terms. Also, the proportion of the total population exposed may be low, making the impact of exposure on the whole population small despite a high relative risk. Therefore absolute measures are needed, and indicators that take account not only of the strength of the association between cause and effect (the RR) but also the extent of exposure of the population to the risk (the prevalence).

Population attributable risk (PAR) is the absolute risk experienced by the population, derived by determining the difference in risk (or rate of disease) in the whole population compared with those in the population not exposed to an identified risk factor:

$$PAR = I_t - I_u$$

where I_t is the incidence in the total population, including both exposed and unexposed subgroups. But I_t is simply the sum of the incidence in each of these two population subgroups (exposed and unexposed), weighted by the relative size of each in the population (the prevalence of the risk exposure):

$$I_t = pI_e + (1-p)I_u$$

where p is the proportion of the population exposed to the risk factor (the prevalence).

Combining the above provides an expression for PAR in terms of the prevalence of the risk factor and its relative risk for the outcome of interest:

$$PAR = p(RR-1)I_u$$

Relating this to the incidence of the outcome in the total population gives the attributable fraction:

$$AF = \frac{p(RR-1)I_u}{I_t}$$

or

$$AF = \frac{p(RR-1)}{p(RR-1)+1}$$

which can be extended to cover multiple exposure levels of the risk factor.

The attributable fraction (AF) is conventionally interpreted as the proportion of current disease (or mortality) attributable to the risk factor concerned. But this is not strictly correct: it would only be the case if the prevalence used to calculate the attributable fraction reflected the prevalence of the risk factor at an appropriate period in the past (for chronic diseases, many years may elapse between onset of exposure and disease). However, another interpretation is possible if current prevalence is used: the attributable fraction indicates the proportion of current disease or mortality that would be prevented in the *future* if exposure to the risk factor were eliminated. This second interpretation* is more useful for policy and is the interpretation favoured for this report.

At the same time, total elimination of a risk factor is unrealistic, at least in the short to medium term. Instead, the concept can readily be extended to estimate the health gain that would result from a partial reduction in risk factor prevalence. The reduction in prevalence anticipated can be estimated through historical trend projections or it can be predicted by modelling. This is known as the 'impact fraction' (IF), calculated by:

$$IF = \frac{\Sigma(p1-p2)RR}{\Sigma p1 RR}$$

where p1 is the current (observed) prevalence and p2 is the predicted prevalence of the risk factor at some future time. This concept has been utilised in the last section of this chapter, in which a future scenario is developed.

* referred to by Murray (1999) as the 'avoidable' fraction. This terminology is not used here, to prevent confusion with the concept of 'avoidability' applied in Chapter 10.

Risk factor prevalences

Smoking

Tobacco smoking is a well recognised risk factor for many cancers and respiratory and cardiovascular diseases. In addition, exposure to environmental tobacco smoke (particularly maternal smoking) has been identified as a major risk factor for SIDS (Mitchell et al 1997) and other respiratory problems in children. Internationally, smoking has been identified as the major cause of preventable death in OECD countries (World Health Organization 1997).

Prevalence

There were three potential sources of recent data on smoking prevalence in the New Zealand population:

- The 1996–97 New Zealand Health Survey (Ministry of Health 1999d) involved face to face interviews with 7862 people aged 15 years or over, with an adult response rate of 73.8 percent to participation in the survey and a non-response rate from survey participants of 0.2 percent for the smoking questions. Of participants, 24.9 percent (95 percent CI: 23.5–26.3) were current smokers.
- Data from AC Nielsen (NZ) Ltd involved quarterly face to face interviews with approximately 2500 people aged 18 years and over. This information has been collected regularly for some time, and is used as the basis for monitoring the trends in smoking in *Progress on Health Outcome Targets* (Ministry of Health 1998b). The 1997 prevalence was identified to be approximately 26 percent.
- The 1996 Census of Population and Dwellings provides a self reported census of the entire population, with a non-response rate of 8 percent to the questions on smoking. The 1996 Census identified 23.7 percent of the population overall as current smokers, which lies within the 95 percent confidence interval of the 1996–97 health survey results.

The 1996 Census smoking data for people usually resident in New Zealand were chosen primarily because the large size of the survey enabled the calculation of prevalence by age, gender and ethnic subgroups (Table 99). However, it is recognised that self reported surveys tend to underestimate smoking prevalence (Tappin et al 1997). Using census data, therefore, is likely to provide a conservative estimate of true smoking prevalence and result in conservative attributable fraction estimates.

Population smoking prevalence is not appropriate for calculating attributable fractions for deaths from SIDS, stillbirths, low birth weight, intrauterine growth retardation and premature rupture of membranes – the prevalence of maternal smoking during pregnancy is needed. Prevalence of exposure to maternal smoking during pregnancy is derived from data on 1995–96 Plunket visits at three months, which identify a prevalence of 49 percent for Māori , 18 percent for non-Māori, and 26 percent for all infants (Ministry of Health 1998b). This compares with two other studies: a Canterbury study of serum cotinine, from antenatal blood samples taken in 1993–94, which found that 26.8 percent of pregnant women smoked in their third trimester (Tappin et al 1997); and the 1996 Census, in which 28.6 percent of all women aged 15–39 years reported being current smokers. Although the representativeness of the Plunket survey is not ideal, the estimated prevalence from this survey was used.

For fire injuries, anonymous data were obtained from the New Zealand Fire Service on deaths caused by fires, and compared with 1996 mortality data recorded by the New Zealand Health Information Service (NZHIS). The proportions of fire deaths likely to be related to smoking (including fires started by matches and cigarette lighters) were identified as 14 percent for males and 15 percent for females. These proportions equal the attributable fractions for fire injuries, as they are direct estimates of the proportion of fire deaths attributable to smoking.

At risk categories

Two risk categories – current and ex smoker – were defined for all outcomes, except Parkinson's disease. For the latter, the protective effect of smoking appears to dissipate so slowly that the prevalence of 'ever smoking' is considered a more appropriate risk category.

Table 99: Prevalence of smoking, 1996

Age (years)	Prevalence of current smoking (%)					Prevalence of ever smoking (%)					Unknown smoking status (%)
	Māori	Pacific	Asian	European /Other	Total	Māori	Pacific	Asian	European /Other	Total	
Female											
15–19	41	17	4	18	22	49	22	5	24	29	8
20–24	54	32	7	28	33	67	40	10	40	45	8
25–29	55	32	7	27	32	72	41	10	45	49	7
30–34	54	28	5	23	28	73	39	8	44	48	7
35–39	53	30	5	22	27	74	39	9	45	49	7
40–44	49	25	4	20	24	70	34	7	42	45	7
45–49	46	22	4	20	22	68	30	7	43	45	7
50–54	43	20	5	19	22	65	28	9	44	45	7
55–59	36	15	4	16	18	59	23	9	39	40	7
60–64	29	13	6	14	15	55	22	10	39	39	8
65–69	24	14	5	12	13	49	25	9	38	38	8
70–74	19	10	6	10	10	44	22	10	36	36	9
75–79	13	10	3	8	8	38	22	11	30	31	9
80 84	10	8	6	6	6	34	21	16	25	25	10
85+	7	2	5	3	3	26	11	16	18	18	12
Female: total 15+	47	25	5	19	23	65	33	8	39	42	8
Male											
15–19	31	19	9	17	20	35	22	12	22	25	9
20–24	44	35	20	28	31	52	41	25	37	40	9
25–29	46	40	23	29	33	59	50	29	42	46	9
30–34	47	39	22	27	30	63	49	32	44	47	8
35–39	45	40	24	26	29	67	52	36	48	50	8
40–44	43	39	22	24	26	67	52	36	50	52	7
45–49	40	39	20	23	25	66	52	36	53	54	7
50–54	37	37	18	23	24	67	52	35	57	58	7
55–59	33	36	17	20	22	64	51	35	58	58	7
60–64	28	31	15	18	19	60	48	35	61	61	8
65–69	23	28	16	15	16	59	48	40	65	64	8
70–74	21	22	16	12	13	61	45	35	67	67	8
75–79	15	24	12	9	10	55	51	42	64	63	8
80–84	14	18	19	8	8	51	39	42	59	59	10
85+	12	16	18	6	6	49	52	43	54	54	11
Male: total 15+	40	35	19	22	25	57	45	29	49	50	8

Source of base data: 1996 Census (SNZ)
Notes: 'Asian' ethnic group is included to illustrate the low rate of smoking prevalence in Asian women, but due to other data limitations no attributable fraction calculations have been undertaken separately for Asian people in New Zealand; 'total' includes all ethnicities including those where ethnicity is unknown; 'European/Other' category includes New Zealand Europeans, Asians and others but excludes those whose ethnicity is unknown.

Alcohol consumption

Alcohol is an important cause of morbidity and mortality. Cirrhosis and other alcoholic liver disease, pancreatitis, endocrine disorders, cardiomyopathy, gastritis, high blood pressure, haemorrhagic stroke, some cancers, intentional and unintentional injuries (such as road traffic crashes), unsafe sexual practices, domestic violence, criminal offending, and mental disorders (such as alcohol dependence and abuse, amnesia, psychosis and dementia) are all recognised adverse effects of alcohol consumption (Ministry of Health 1998b). Alcohol use in pregnancy can result in birth defects (foetal alcohol syndrome).

However, different levels of consumption of alcohol are associated with different levels of health hazard, and alcohol can also be protective (for example, against IHD and other diseases with similar pathophysiology, in particular ischaemic stroke). In order to accommodate these differences, alcohol prevalence requires subdivision into three consumption levels associated with differing risks: 'zero to minimal' consumption of alcohol (regarded as 'abstinence' for the purposes of this report); 'low to moderate'; and (potentially) 'hazardous to harmful' consumption. The attributable fractions are calculated using both low to moderate as well as hazardous to harmful levels of exposure, as appropriate for the outcome concerned.

Prevalence

The 1996–97 New Zealand Health Survey was used as the basis for estimating prevalence of alcohol consumption (Ministry of Health 1999d). This survey used the AUDIT (Alcohol Use Disorders Identification Test) questionnaire (Barbor 1989) to examine alcohol use. The AUDIT is a 10 item questionnaire covering alcohol consumption, alcohol related problems and drinking behaviour. Each question is scored from 0 to 4 and the questionnaire has a maximum possible score of 40; the higher the AUDIT score, the more problematic the pattern of alcohol consumption is considered to be. Individuals with an AUDIT score of 8 or above are likely to suffer physical, mental and/or social effects as a consequence of their alcohol consumption. It should be noted, however, that the AUDIT questionnaire has not been used extensively to monitor the use of alcohol in general populations internationally; in addition, the 1996–97 New Zealand Health Survey was the first time that AUDIT had been used in a national survey in New Zealand.

One of the methodological issues involved in calculating attributable fractions is that the definition of the risk categories for the estimation of relative risks should correspond as closely as possible with the exposure categories used to measure risk factor prevalence. As studies measuring relative risks for alcohol usually define risk categories in terms of quantities of alcohol consumed (grams of alcohol or number of standard drinks) and/or frequency of consumption, the decision to utilise prevalence categories based on AUDIT scores requires that equivalence be demonstrated between these ways of categorising risk.

First, AUDIT has been designed to identify the effects of alcohol on the individual, taking account of both the quantity consumed and the pattern of drinking, and should therefore provide a more accurate descriptor of potentially hazardous drinking behaviour. 'Hazardous drinking' is defined as an established pattern of drinking that carries with it a high risk of future damage to physical or mental health (Saunders et al 1993). Second, the 1996–97 survey included questions related to the frequency of drinking; a significant correlation ($p < 0.05$) was found between an AUDIT score of 8 or more and individuals who drank over six drinks on one occasion at least weekly, as well as individuals who consumed five or more drinks in a single day when drinking (Table 100). Third, data were obtained from the 1995 National Survey on Drinking in New Zealand (S Casswell, personal communication 1999), and grouped according to the categories of

English et al (1995) for comparison. These data were derived from a survey of over 4000 people aged 14–65 years. Although significant differences were found between these two surveys for some age gender cells, overall patterns were similar.*

These comparisons support the use of an AUDIT score of 8 or more as an indicator of potentially harmful drinking and, further, one that offers advantages over risk categorisations based solely on quantity of alcohol consumed.

Table 100: Comparison between AUDIT and other alcohol consumption measures

	Age (years)	AUDIT > 8 (%)	Five or more drinks per day when drinking (%)	Six or more drinks on one occasion at least weekly (%)
Female	15–24	25.80	40.40	15.20
	25–44	8.90	14.20	3.60
	45–65	3.50	4.10	3.10
	65+	0.60	1.80	0.20
Male	15–24	40.70	56.40	33.40
	25–44	27.40	29.70	19.30
	45–65	20.50	20.50	21.80
	65+	8.80	7.90	8.10

Source of base data: NZHS 1996–97

Table 101 identifies the prevalence of AUDIT scores by age group and gender, as used for the calculation of attributable fractions for alcohol. Data limitations prevented further breakdown by ethnicity.

Table 101: Prevalence of alcohol consumption, 1996–97

Age (years)	Female prevalence (%)			Male prevalence (%)		
	Abstainers	AUDIT score under 8	AUDIT score 8 or more	Abstainers	AUDIT score under 8	AUDIT score 8 or more
15–19	61	18	22	55	15	30
20–24	34	42	24	28	27	45
25–29	49	40	11	29	37	34
30–34	49	43	8	26	51	23
35–39	47	47	6	27	50	23
40–44	47	48	5	29	50	21
45–49	47	50	4	26	54	20
50–54	47	49	5	32	46	22
55–59	45	54	1	28	49	23
60–64	55	44	2	32	56	12
65–69	53	47	0	32	54	14
70– 74	64	35	1	32	61	7
75–79	65	35	0	42	50	8
80–84	76	24	0	50	50	0
85+	80	20	0	75	22	3
Total 15+	50	42	8	32	44	24

Source of base data: NZHS 1996–97
Note: 'abstainers' includes people with minimal alcohol intake (based on alcohol consumption in the category of 'monthly or less').

* Since this analysis was done, the results of the 1998 National Drug Survey have become available. This will allow further comparison of the different methods for quantifying drinking patterns.

For deaths related to road traffic injuries, national statistics were used to establish that 28 percent of road traffic deaths were alcohol-related in 1996 (LTSA 1998). This proportion is a direct estimate of the attributable fraction for road traffic deaths. Similarly, the attributable fraction for drowning was estimated directly, using data supplied by Water Safety New Zealand.

Physical inactivity

Regular moderate physical activity is recognised as being beneficial for health (US Department of Health and Human Services 1996; National Health Committee 1998a). Conversely, a lack of physical activity is a risk factor for heart disease, stroke, hypertension, type 2 diabetes, colon cancer and premature death. Currently, it is recommended that adults participate in at least 2.5 hours of leisure time physical activity per week of an intensity equivalent to brisk walking (National Health Committee 1998a). This was the threshold chosen for determining the prevalence of physical inactivity for PAR estimation.

Prevalence

The most recent dataset available to enable prevalence of inactivity to be estimated by age, gender and ethnicity was the 1996–97 New Zealand Health Survey (Figure 161 and Table 102). However, the numbers of older Māori and Pacific people in the survey were relatively small, and so these data should be treated with caution. The only other similar survey which is suitable for comparison is the Sport and Physical Activity Survey undertaken by the Hillary Commission (1997). The health survey identified fewer people as meeting the recommended level of physical activity across all age groups (18 years and over), with a difference of 5–7 percent for each age group, except for those aged 35–49 years, where the difference was approximately 9 percent. Health survey data therefore give higher attributable fraction estimates than those calculated using data from the Hillary Commission survey.

Figure 161: Prevalence of physical inactivity, 1996–97

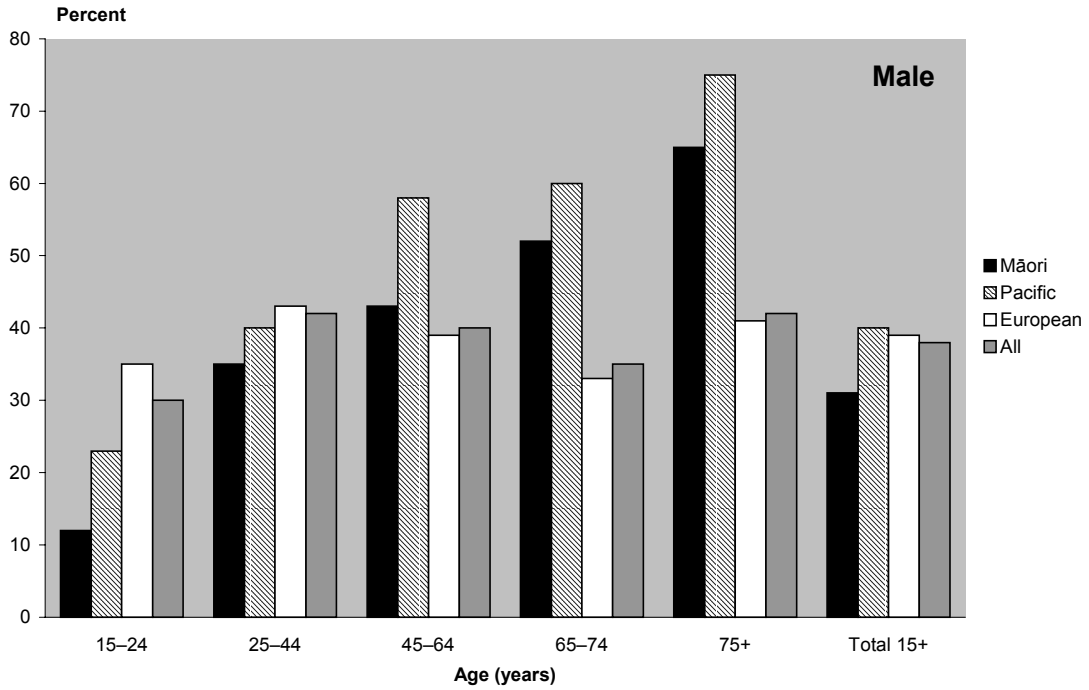
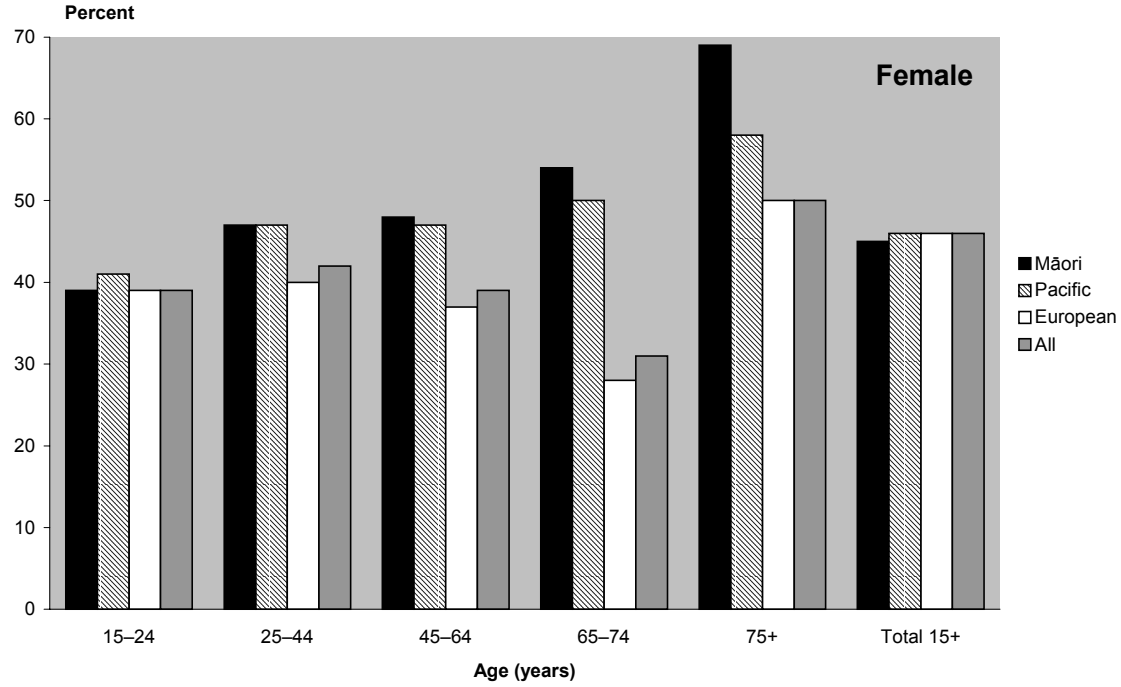


Figure 161 continued



Source of base data: NZHS 1996–97
Note: scale varies between charts.

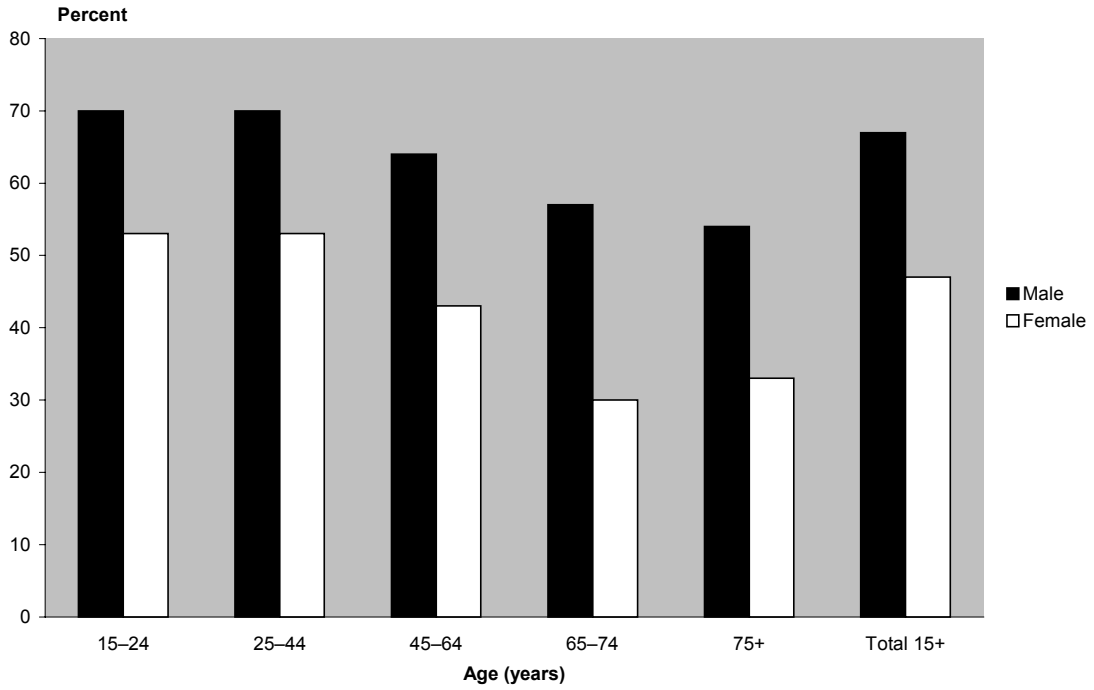
Inadequate fruit and vegetable consumption

There is increasing epidemiological evidence that fresh fruit and vegetable consumption offers protection against cancer at many sites, especially the colon and lung (Block et al 1992), and diets high in fruit and vegetables are also protective against IHD (Miller et al 1997). Currently, New Zealand adults are recommended to eat five or more servings of fresh fruit and vegetables each day (Nutrition Taskforce 1991).

Prevalence

The prevalence of those consuming less than five servings of fruit and vegetables per day was derived from 1997 National Nutrition Survey data, using an algorithm to combine different levels of fruit consumption with different levels of vegetable consumption. This algorithm provides a conservative estimate of total consumption. Due to data limitations, no ethnic breakdown was undertaken. The prevalence estimates are summarised in Figure 162.

Figure 162: Prevalence of consuming less than five servings of fruit and vegetables per day, 1996–97



Source of base data: NNS 1996–97

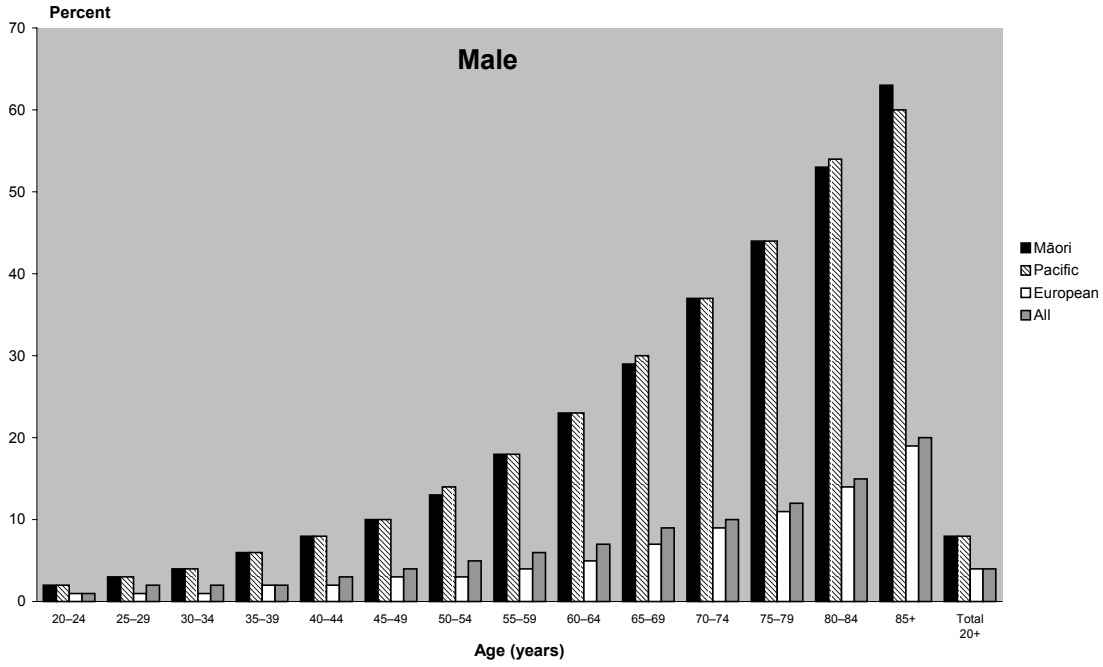
Diabetes (type 2)

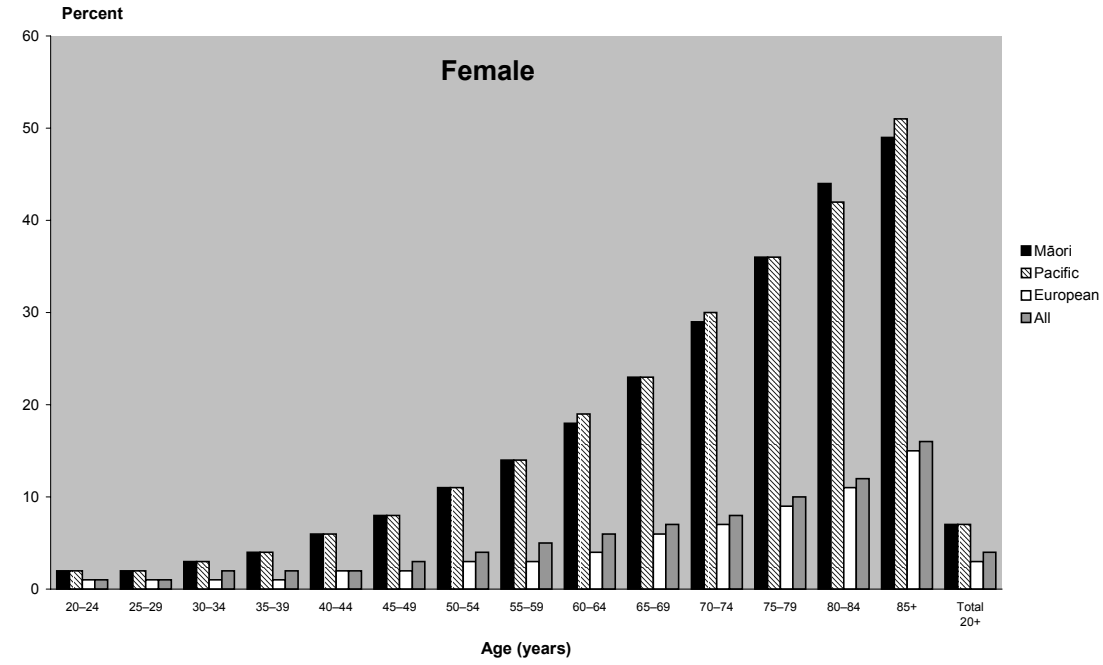
Type 2 diabetes (formerly called non-insulin dependent diabetes and typically having onset in adulthood) is recognised as a significant public health problem. Its health consequences can include retinopathy (which can progress to blindness), kidney disease, peripheral and autonomic neuropathy, IHD, hypertension, stroke and peripheral vascular disease. Diabetic nephropathy is now the leading cause of end stage renal failure in New Zealand (Simmons 1996a). Pre menopausal women with diabetes lose the protection normally enjoyed by younger women against atherosclerosis (Larosa 1997). Gestational diabetes is also associated with adverse reproductive outcomes.

Although often present with other risk factors, such as obesity and lack of physical activity, diabetes has been found to be an independent risk factor for cardiovascular disease (Webster 1997). As a result, diabetes has been identified in this report both as a risk factor for disease, and as a disease outcome of other risk factors.

The prevalence of type 2 diabetes has been estimated from data collected in the 1996–97 New Zealand Health Survey, using a mathematical model (Tobias and Roberts 1999). As this provides estimates only for the prevalence of symptomatic (clinical) diabetes, attributable fractions based on these estimates will be conservative.

Figure 163: Prevalence of type 2 diabetes, 1996 (modelled)





Source of base data: Tobias and Roberts 1999
Note: scale varies between charts.

Obesity

Obesity is associated with increased all-cause, cardiovascular, stroke and type 2 diabetes mortality. Relationships have also been identified between increasing body mass and increased blood pressure, cholesterol levels, gallstones, obstructive sleep apnoea, some cancers, osteoarthritis, and some female reproductive disorders (Bray 1996).

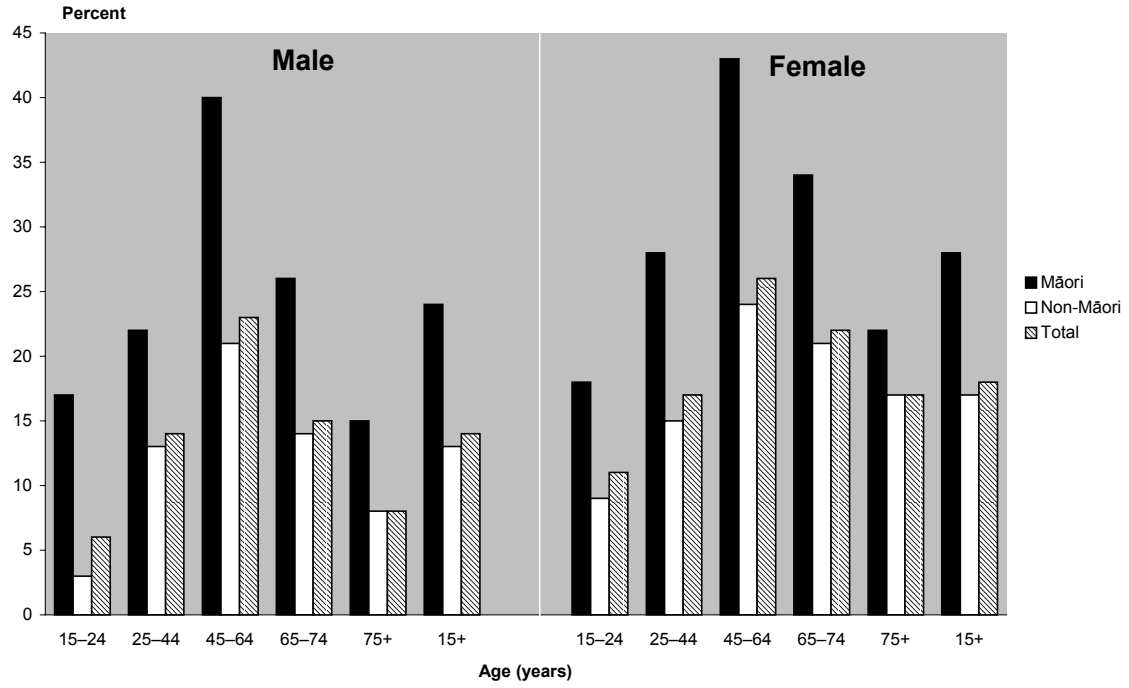
A commonly used obesity measure is the body mass index (BMI), calculated by weight (in kilograms) divided by height (in meters) squared. However, there are a number of different ways of defining and measuring obesity and these are not without debate. Rimm et al (1995), for example, has identified that BMI has a weaker association with IHD than the waist to hip ratio or waist circumference. BMI was used in this study because of the availability of matching data. Using BMI as the measure of obesity, Auckland data suggest that from 1982 to 1993 an additional 3 percent of women and 6 percent of men became obese (Simmons 1996a). Comparison of the 1989 Life in New Zealand Survey (Hillary Commission 1990) and the 1997 National Nutrition Survey suggests a similar rate of increase over the 1990s: from 11 percent to 17 percent for the adult population as a whole (Ministry of Health 1999a).

For this study, obesity was defined as a BMI greater than or equal to 30 for European/Others; for Māori and Pacific people 32 was adopted as the obesity threshold to reflect ethnic variations in body composition (Swinburn 1997, Swinburn 1996).

Prevalence

Data were extracted from the 1997 National Nutrition Survey where height and weight were measured on three occasions, and mean results were used to calculate BMI. Due to data limitations, only prevalences for Māori were provided separately; however, Pacific people were included in the non-Māori category (with BMI ≥ 32; for all others BMI ≥ 30). Some modelling of the prevalence in older Māori men was required; these prevalences were modelled to reflect the overall patterns found within the national and Auckland regional surveys (Bonita et al 1998) for older age groups.

Figure 164: Prevalence of obesity, 1996–97



Source of base data: NNS 1997 and Bonita et al 1998
Note: BMI ≥ 32 for Māori and for Pacific people (within non-Māori category); all other ethnicities BMI ≥ 30.

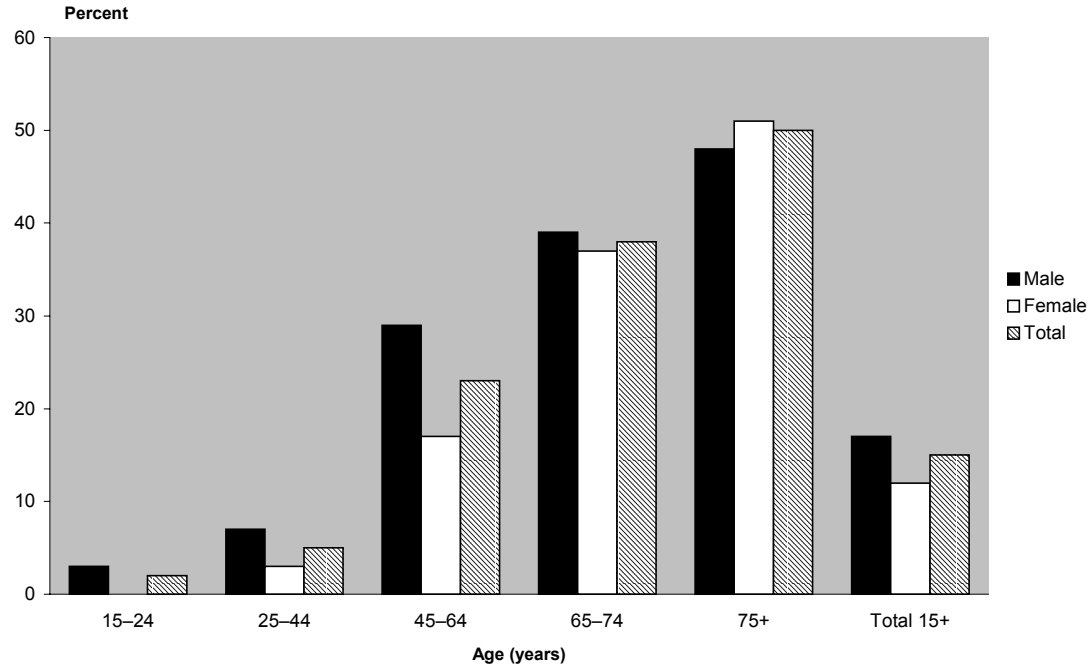
High blood pressure

High blood pressure (hypertension) is an important risk factor for heart disease and stroke. Hypertension is associated with obesity, physical inactivity, high alcohol and salt intake, and low potassium intake (Ministry of Health 1998b). The prevalence of hypertension in New Zealand appears to have declined since the early 1980s, possibly due to increased physical exercise, or from a reduction of alcohol or salt intake (Trye et al 1996), as well as greater recognition and pharmacologic treatment of the risk factor in primary health care.

Prevalence

For this study, hypertension was defined as systolic pressure 160 mm Hg or higher and/or diastolic pressure of 95 mm Hg or higher. People on anti hypertensive medication with measured pressures below these levels were not considered hypertensive. It should be noted that this underestimates the true attributable fraction because people with hypertension, even following treatment in which the blood pressure is successfully lowered, remain at a (slightly) increased risk compared with normotensive people (Barendregt et al 1998). Data from the 1997 National Nutrition Survey were used, but due to data limitations no ethnic breakdown was undertaken (Figure 165).

Figure 165: Prevalence of measured hypertension, 1996–97



Source: NNS 1997
Note: hypertension is defined as systolic blood pressure ≥ 160 mm Hg and/or diastolic blood pressure ≥ 95 mm Hg.

High blood cholesterol

There is an extensive body of scientific literature on blood lipids and risk of IHD. The underlying cause of abnormal blood lipids (other than genetic factors) is a diet high in animal fats (Bonita et al 1998). Although the attributable fractions for a number of different lipid components could be considered, total blood cholesterol was chosen because it has been identified in a number of studies as being predictive of IHD events occurring 10 or more years

after initial measurement (Wannamethee et al 1999). Although mean cholesterol levels do increase with age, a cut off point of 6.5 mmol/L or greater for all ages was used to define high blood cholesterol (hypercholesterolaemia).

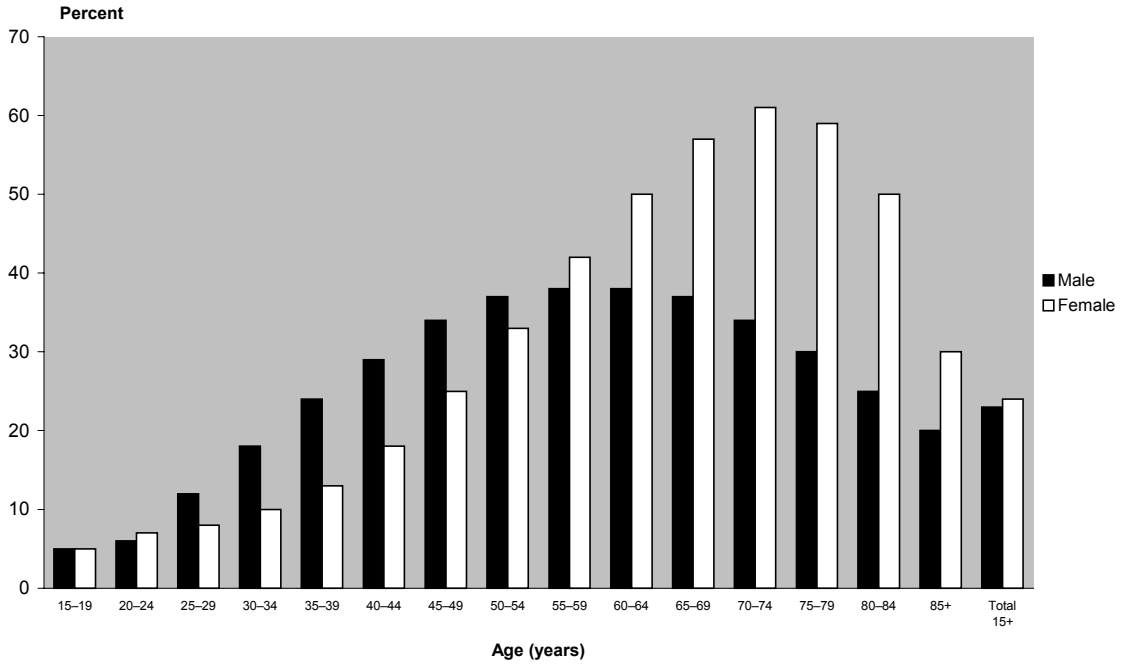
Prevalence

The most recent national survey of blood cholesterol levels in New Zealand was undertaken as part of the 1997 National Nutrition Survey.

For most age–gender cells, the mean total cholesterol levels were lower than those found in earlier New Zealand studies. For example, comparison with the Life in New Zealand survey (Hillary Commission 1990) suggests an overall decline in the prevalence of hypercholesterolaemia of about 25 percent (from approximately 30 percent to 23 percent) over the decade.

Scragg et al (1993) also identified that Māori and Pacific people both had a slightly lower prevalence of total blood cholesterol ≥ 6.5 compared with European/Others (male: Māori 39.3 percent, Pacific people 34.4 percent, European/Other 40.1 percent; female: 30.9 percent, 32.9 percent and 35.9 percent respectively). However, due to data limitations, it was not possible to further subdivide the 1997 National Nutrition Survey data, and so no ethnic specific prevalences were calculated. Even using only age gender subdivision, some smoothing of the prevalence dataset was still necessary (Figure 166).

Figure 166: Prevalence of total blood cholesterol ≥ 6.5 mmol/L, 1996–97



Source of base data: NNS 1997

Relative risk estimates

Smoking

The substantial work of English et al (1995) was used as the basis of the relative risk estimates for most of the outcomes causally related to smoking. The individual causes of death included those judged by these authors to have at least limited evidence of being caused by smoking, and for which there were deaths in 1996 in New Zealand, as listed in Table 102. For IHD, the modelled age specific relative risk estimates of Barendregt and Bonneux (1998) were utilised (Table 104). All-cause mortality was summed from the results of calculations for the causes of death listed in Table 102; this provides a conservative estimate of smoking related mortality, as potentially there are many other diseases to which smoking contributes yet the epidemiological evidence remains inadequate. In addition, the effects of passive smoking have not been included (except for SIDS).

Table 102: Relative risks for current smoking, ages 15 and over

Cause of death	Relative risk of current smokers	Relative risk of ex-smokers
Cancers		
Oropharyngeal	4.55	1.76
Oesophageal	4.01	1.79
Stomach	1.41	1.11
Anal	3.18	1.83
Pancreatic	1.86	1.15
Larygneal	7.48	2.86
Lung	f 11.4; m 13	f 5.07; m 6.75
Endometrial	0.53	0.91
Cervical	1.75	1.31
Vulvar	3.42	1.37
Penile	1.80	1.60
Bladder	2.72	1.66
Renal parenchymal	1.64	1.61
Renal pelvic	3.96	1.95
Cardiovascular diseases		
Atherosclerosis	2.54	1.82
Cardiac dysrhthmias	as for IHD	as for IHD
Heart failure	two thirds of IHD	two thirds of IHD
IHD	see Table 104	< 65y 1.45; 65y+ 1.12
Pulmonary circulatory disease	9.8	6.70
Stroke	< 65y 3.12; 65y+ 1.65	< 65y 1.30; 65y+ 1.15
Others		
CORD	9.80	6.70
Pneumonia	1.47	1.29
Parkinson's disease	0.57	0.57
Peptic ulceration	2.07	2.24
Crohn's disease	m 1.92; f 3.27	m 1.92; f 1.60
Ulcerative colitis	0.63	1.71
SIDS	2.76	
Stillbirth	2.76	
Low birthweight, IUGR, prematurity	2	
Fire injuries (attributable fraction)	m 0.14; f 0.15	

Source of base data: English et al 1995

Alcohol consumption

As with smoking, the work of English et al (1995) was used as the basis of the relative risk estimates for most of the outcomes causally related to alcohol (Table 103). However, for IHD the focus of these authors was on the impact of hazardous/harmful alcohol consumption; the protective effect of alcohol consumption on IHD (compared with abstainers) was neglected. Evidence of an independent protective effect of alcohol against IHD and some other diseases, particularly at low to moderate levels of alcohol consumption, is supported by more recent reviews, including Doll (1998) and Thun et al (1997), and these studies have been used for this report.

Table 103a: Relative risks for alcohol consumption

Cause of death	Low/moderate consumption	Hazardous/harmful consumption	
	Male and female	Female	Male
Oropharnygeal cancer	1.5	3.0	4.1
Oesophageal cancer	1.8	3.0	3.6
Liver cancer	1.5	3.2	3.4
Laryngeal cancer	1.8	4.2	4.5
Female breast cancer	1.1	1.4	na
Hypertension	f = 0.9; m = 1.0	1.4	1.8
IHD	0.8	0.9	0.9
Supraventricular tachycardias	1.5	2.2	2.2
Stroke	0.6	2.9	1.5
Unspecified liver cirrhosis	1.3	9.5	9.5
Cholelithiasis	0.8	0.6	0.6
Suicide	1.4	2.4	2.4

Table 103b: Directly calculated attributable fractions for alcohol consumption

Cause of death	Attributable fraction (male and female)
Alcohol dependence syndrome	1
Alcohol abuse	1
Alcoholic liver cirrhosis	1
Acute pancreatitis	0.24
Chronic pancreatitis	0.84
Road traffic injury	0.28*
Fall injuries	0.34
Fire injuries	0.23
Drowning	0.34**
Occupational and machine injuries	0.07
Assault	0.47

Principal source of base data: English et al 1995
* data from LTSA (1998)
** data from Water Safety New Zealand

Other risk factors

A number of challenges were encountered in identifying and selecting suitable relative risk estimates for many of the other risk factors – principally, definitions of exposure categories that did not correspond with those used to measure risk factor prevalences in the surveys. For example, obesity relative risks need to be based on the BMI > 30 threshold to be compared with the 20–25 baseline category. A BMI of 25–30 is in an intermediate risk category, and there is some evidence that a BMI of less than 20 may increase risk for some causes of mortality; for dichotomisation of risk, these categories had to be combined. Often studies provided comparisons between the lowest and highest quintiles or quartiles only, which could not be readily translated into the above BMI categories.

For fruit and vegetable consumption, the recommended consumption is five or more servings, and the ideal relative risks are based on comparisons above and below this level. However, most studies compared highest and lowest quintiles or quartiles; relative risks therefore had to be re-estimated to encompass all levels of consumption and not just the two extremes.

The tables showing the attributable fractions also identify the relative risks used in this study. A list of references that the authors found useful in developing these relative risk estimates is available from the Ministry of Health.

Attributable fractions

Smoking

Because it has both high relative risks and prevalences, smoking has the highest attributable fractions for many outcomes and population subgroups. Tables 110 to 113 detail the relative risks and attributable fractions of IHD, stroke, lung cancer and CORD for smoking.

The very high attributable fractions at younger ages for females reflect the relative risks and prevalence of smoking at these ages, particularly for Māori women.

It should be noted that in these and other tables of attributable fractions, estimates are not shown for young adult ages if the outcome concerned has negligible incidence at these ages.

Table 104a: Relative risks and attributable fractions for smoking and IHD, 1996

Age (years)	RR female ex-smoker	RR female current smoker	Attributable fractions: female				RR male ex-smoker	RR male current smoker	Attributable fractions: male			
			Māori (%)	Pacific (%)	European/ Other (%)	Total female (%)			Māori (%)	Pacific (%)	European/ Other (%)	Total male (%)
40–44	1.5	7.7	77	63	59	63	1.5	3.7	56	53	43	46
45–49	1.5	5.4	68	50	50	52	1.5	3.1	49	47	38	39
50–54	1.5	4.2	60	41	42	45	1.5	2.7	43	41	34	36
55–59	1.5	3.4	49	28	33	35	1.5	2.4	37	36	31	32
60–64	1.5	2.8	39	22	27	28	1.5	2.2	32	31	29	29
65–69	1.1	2.6	29	19	18	19	1.1	2.0	22	24	18	18
70–74	1.1	2.3	22	13	14	14	1.1	1.9	19	19	15	15
75–79	1.1	2.2	16	12	11	11	1.1	1.8	15	18	12	13
80–84	1.1	2.1	12	9	8	8	1.1	1.7	13	14	11	11
85+	1.1	2.0	8	3	5	5	1.1	1.7	11	13	9	9

Table 104b: Relative risks and attributable fractions for smoking and stroke, 1996

Age (years)	RR female ex-smoker	RR female current smoker	Attributable fractions: female				RR male ex-smoker	RR male current smoker	Attributable fractions: male			
			Māori (%)	Pacific (%)	European/ Other (%)	Total female (%)			Māori (%)	Pacific (%)	European/ Other (%)	Total male
40–44	1.3	3.1	53	36	33	36	1.3	3.1	49	47	37	39
45–49	1.3	3.1	51	33	33	35	1.3	3.1	48	47	36	38
50–54	1.3	3.1	50	31	33	35	1.3	3.1	47	46	37	38
55–59	1.3	3.1	45	26	29	31	1.3	3.1	44	45	35	36
60–64	1.3	3.1	41	23	27	28	1.3	3.1	41	41	34	35
65–69	1.2	1.7	16	10	11	11	1.2	1.7	17	18	15	15
70–74	1.2	1.7	14	8	9	9	1.2	1.7	16	15	14	14
75–79	1.2	1.7	11	8	8	8	1.2	1.7	14	16	12	12
80–84	1.2	1.7	9	7	6	6	1.2	1.7	13	13	11	11
85+	1.2	1.7	7	3	4	4	1.2	1.7	12	14	10	10

Table 104c: Relative risks and attributable fractions for smoking and lung cancer, 1996

Age (years)	RR female ex-smoker	RR female current smoker	Attributable fractions: female				RR male ex-smoker	RR male current smoker	Attributable fraction: male			
			Māori (%)	Pacific (%)	European/ Other (%)	Total female (%)			Māori (%)	Pacific (%)	European/ Other (%)	Total male (%)
40 44	5.1	11.4	86	75	75	77	6.8	13.0	87	85	81	82
45–49	5.1	11.4	85	72	75	76	6.8	13.0	86	85	82	82
50–54	5.1	11.4	84	71	75	76	6.8	13.0	86	84	82	83
55–59	5.1	11.4	82	66	72	74	6.8	13.0	85	84	82	82
60–64	5.1	11.4	80	63	71	72	6.8	13.0	84	82	82	82
65–69	5.1	11.4	78	66	70	70	6.8	13.0	83	82	82	82
70–74	5.1	11.4	75	60	67	68	6.8	13.0	83	80	82	82
75–79	5.1	11.4	70	60	63	63	6.8	13.0	81	81	81	81
80–84	5.1	11.4	67	58	58	58	6.8	13.0	79	77	80	79
85+	5.1	11.4	60	36	48	48	6.8	13.0	78	80	78	78

Table 104d: Relative risks and attributable fractions for smoking and CORD, 1996

Age (years)	RR female ex-smoker	RR female current smoker	Attributable fractions: female				RR male ex-smoker	RR male current smoker	Attributable fractions: male			
			Māori (%)	Pacific (%)	European/ Other (%)	Total female (%)			Māori (%)	Pacific (%)	European/ Other (%)	Total male (%)
40–44	6.7	9.8	85	73	75	77	6.7	9.8	84	81	78	79
45–49	6.7	9.8	84	70	75	76	6.7	9.8	83	81	79	79
50–54	6.7	9.8	84	69	76	76	6.7	9.8	83	80	80	80
55–59	6.7	9.8	82	64	73	74	6.7	9.8	82	80	80	80
60–64	6.7	9.8	80	62	72	73	6.7	9.8	81	79	80	80
65–69	6.7	9.8	78	65	72	72	6.7	9.8	80	78	81	81
70–74	6.7	9.8	76	61	70	70	6.7	9.8	81	77	81	81
75–79	6.7	9.8	72	61	66	66	6.7	9.8	78	78	80	80
80–84	6.7	9.8	69	59	62	62	6.7	9.8	77	73	78	78
85+	6.7	9.8	63	41	53	53	6.7	9.8	76	78	77	77

Principal source of base data: English et al 1995 and 1996 Census

Table 105a summarises the overall impact of smoking on mortality: each year, over 1500 female deaths (11 percent of deaths in females aged 15 years or older) and over 2700 male deaths (19 percent) are attributable to smoking (based on 1996 mortality and prevalence rates). Annually, this amounts to over 34,900 discounted years of life lost among males and 18,600 discounted years among females (Table 105b). For IHD and stroke, the proportions of attributable deaths for Māori males are approximately twice the proportion for European/Others; for females, a three- to four-fold difference exists. These estimates are underestimates of the real impact of smoking, as the calculations do not include the impacts of passive smoking, apart from SIDS (Table 105c).

Table 105a: Deaths caused by smoking, 1996

	Attributable deaths							
	Māori		Pacific		European/Other		Total	
	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Male								
IHD	98	31	29	34	545	16	672	18
Stroke	16	29	4	18	136	14	156	15
Lung cancer	98	84	21	81	621	81	740	82
CORD	51	80	11	79	672	79	734	79
Other causes	31	13	13	20	392	23	476	24
All causes	294	22	78	19	2366	19	2778	19
Female								
IHD	62	31	11	23	250	9	323	11
Stroke	16	26	3	10	110	7	129	8
Lung cancer	68	79	5	71	270	66	343	68
CORD	38	78	3	60	385	64	426	65
Other causes	35	16	5	10	230	12	303	14
All causes	219	21	27	8	1245	10	1524	11

Table 105b: YLL attributable to smoking, 1996

	Māori		Pacific		European/Other		Total	
	YLL_d	YLL	YLL_d	YLL	YLL_d	YLL	YLL_d	YLL
Male								
IHD	1779	2718	540	847	7261	10,060	9580	13,625
Stroke	276	412	63	89	1490	2006	1829	2506
Lung cancer	1507	2112	346	509	7684	10,180	9537	12,801
CORD	701	993	125	180	6172	7617	6999	8790
Other causes	547	795	303	672	4894	7391	6995	12239
All causes	4810	7029	1378	2296	27,501	37,254	34,939	49,960
Female								
IHD	1032	1529	195	306	2599	3372	3826	5208
Stroke	280	420	53	79	1177	1625	1510	2124
Lung cancer	1087	1559	78	107	3441	4622	4606	6289
CORD	504	683	45	62	3723	4623	4272	5368
Other causes	608	952	147	370	2649	4233	4441	8359
All causes	3511	5144	518	923	13,589	18,476	18,655	27,347

Table 105c: SIDS deaths attributable to smoking, 1996

Group	Relative risk	Prevalence of maternal smoking (%)	Attributable fraction (%)	Total 1996 deaths	Deaths attributable to smoking	YLL_d
Female						
Māori	2.76	49	46	31	14	427
Pacific	2.76	18	24	3	1	31
Other	2.76	18	24	11	3	92
Total				45	18	550
Male						
Māori	2.76	49	46	42	19	580
Pacific	2.76	18	24	8	2	61
Other	2.76	18	24	14	3	92
Total				64	24	733

Principal sources of base data: English et al 1995 and 1996 Census

Alcohol consumption

There is now good epidemiological evidence that alcohol exerts a protective effect against mortality from IHD (Doll 1998; Thun et al 1997) (illustrated by the negative attributable fractions in Table 106a), and at low to moderate levels of consumption there appears to be some protection against ischaemic stroke* as well (Table 106b). Thus, the results for alcohol are different from other risk factors, in that the harmful effects of alcohol are at least partially offset by its protective effects against IHD and stroke (and a number of other, less important conditions); these are separately detailed in Table 106c.

Table 106a: Relative risks and attributable fractions for alcohol and IHD, 1996

Age (years)	RR female low/moderate	RR female hazardous/harmful	AF female (%)	RR male low/moderate	RR male hazardous/harmful	AF male (%)
40–44	0.82	0.85	–10	0.82	0.87	–13
45–49	0.82	0.85	–10	0.82	0.87	–14
50–54	0.82	0.85	–10	0.82	0.87	–13
55–59	0.82	0.85	–11	0.82	0.87	–13
60–64	0.82	0.85	–9	0.82	0.87	–13
65–69	0.82	0.85	–9	0.82	0.87	–13
70–74	0.82	0.85	–7	0.82	0.87	–14
75–79	0.82	0.85	–7	0.82	0.87	–11
80–84	0.82	0.85	–4	0.82	0.87	–10
85+	0.82	0.85	–4	0.82	0.87	–5

Principal sources of base data: English et al 1995, Thun et al 1997, NZHS 1996–97.
Note: RR < 1 indicates a protective effect; the corresponding attributable fraction is therefore negative.

* While alcohol may act as a protective factor against ischaemic stroke, it is a risk factor for haemorrhagic stroke through its effect on blood pressure. The PAR estimated here is the best current estimate of alcohol's net effect on stroke overall.

Table 106b: Relative risks and attributable fractions for alcohol and stroke, 1996

Age (years)	RR female low/moderate	RR female hazardous/harmful	AF female (%)	RR male low/moderate	RR male hazardous/harmful	AF male (%)
40–44	0.58	2.87	–13	0.60	1.47	–11
45–49	0.58	2.87	–17	0.60	1.47	–14
50–54	0.58	2.87	–13	0.60	1.47	–9
55–59	0.58	2.87	–26	0.60	1.47	–10
60–64	0.58	2.87	–18	0.60	1.47	–21
65–69	0.58	2.87	–23	0.60	1.47	–18
70–74	0.58	2.87	–14	0.60	1.47	–27
75–79	0.58	2.87	–16	0.60	1.47	–19
80–84	0.58	2.87	–11	0.60	1.47	–25
85+	0.58	2.87	–9	0.60	1.47	–8

Principal sources of base data: English et al 1995, Thun et al 1997, NZHS 1996–97
Note: RR < 1 indicates a protective effect, resulting in a negative attributable fraction.

Table 106c: Deaths caused and prevented by alcohol, 1996

Age (years)	Deaths caused	Deaths prevented	Net attributable deaths		Net attributable YLL_d	Net attributable YLL
			Number	Percentage		
Female						
15–19	20	0	20	23	575	1320
20–24	13	0	13	19	364	794
25–29	8	0	8	10	217	450
30–34	11	0	11	10	288	564
35–39	11	0	11	9	276	510
40–44	8	–2	6	4	143	250
45–49	16	–6	10	3	223	368
50–54	13	–5	8	2	165	257
55–59	15	–13	2	0	38	55
60–64	9	–16	–7	–1	–117	–162
65–69	16	–31	–15	–1	–215	–281
70–74	22	–43	–21	–1	–248	–307
75–79	15	–73	–58	–3	–539	–632
80–84	22	–70	–48	–2	–334	–374
85+	41	–105	–64	–1	–241	–256
Total 15+	240	–364	–124	–1	593	2557
Male						
15–19	39	0	39	23	1121	2574
20–24	55	0	55	28	1540	3361
25–29	48	0	48	24	1304	2698
30–34	41	–2	39	19	1021	2001
35–39	29	–5	24	11	601	1114
40–44	18	–7	11	5	261	458
45–49	27	–17	10	3	223	368
50–54	30	–21	9	2	185	289
55–59	40	–34	6	1	113	166
60–64	39	–52	–13	–1	–217	–300
65–69	45	–81	–36	–2	–515	–673
70–74	48	–121	–73	–4	–863	–1066
75–79	29	–108	–79	–4	–734	–861
80–84	24	–118	–94	–4	–654	–733
85+	41	–46	–5	0	–19	–20
Total 15+	553	–612	–59	0	3367	9373

Principal sources of base data: English et al 1995, Thun et al 1997, NZHS 1996–97, NZHIS
Note: negative numbers indicate deaths prevented. Percentages are of deaths from all causes in age group.

For both males and females there is a net saving in number of lives of 124 for females and 59 for males at 1996 levels of alcohol consumption (Table 106c). Importantly, however, there are still net years of life lost associated with current patterns of alcohol consumption, even when discounted at 3 percent per year – 593 for females and a more substantial 3367 for males in 1996 (or 2557 and 9373 net years of life lost respectively if undiscounted).* This is because a high proportion of the deaths related to alcohol occur at younger ages, principally injuries (particularly road traffic injuries, but also including suicides, falls, drownings, assaults and fire injuries), whereas the protective effect of alcohol on (mainly) IHD and stroke occurs later in life. This differential impact is illustrated in Figure 167.

Figure 167a: Deaths caused and prevented by alcohol consumption, 1996

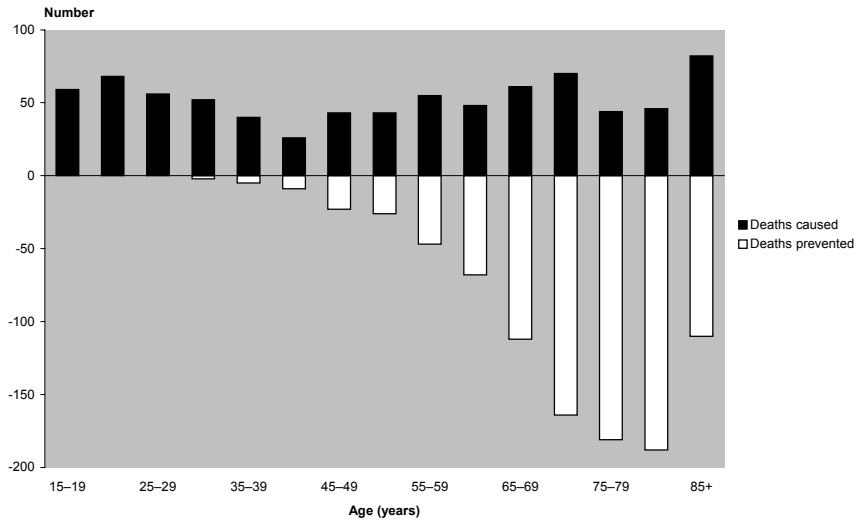
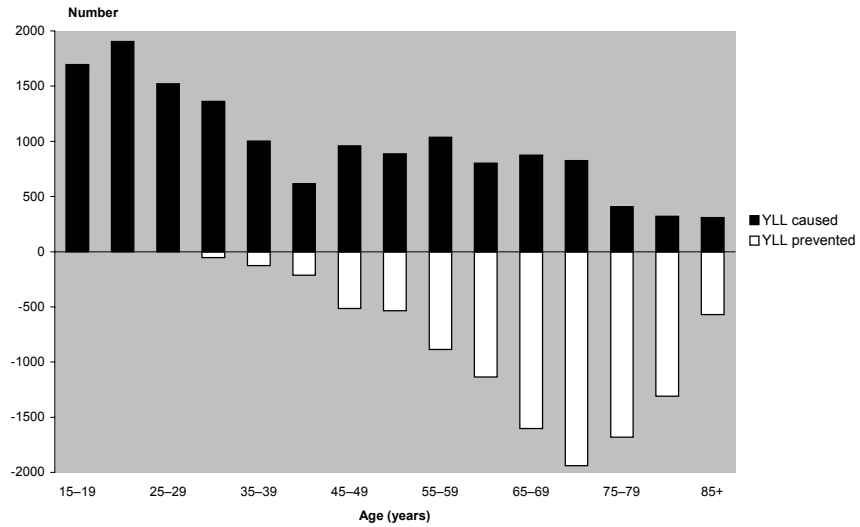


Figure 167b: YLL caused and prevented by alcohol consumption, 1996



Principal sources of base data: English et al 1995, Thun et al 1997, NZHS 1996–97, NZHIS
Note: YLL discounted at 3 percent per year; YLL calculated by the remaining life expectancy method with West level 26 as the standard.

* The precise number estimated will depend on the method used to calculate YLL, as well as the methods and data sources used to estimate PARs for alcohol (ie, to obtain estimates for both relative risks and prevalences of alcohol consumption patterns).

Inadequate fruit and vegetable consumption

Estimated attributable fractions related to low fruit and vegetable consumption (less than five servings per day) are detailed in Table 107. The combination of higher relative risks at younger ages and the greater prevalence of those with inadequate fruit and vegetable consumption result in the highest attributable fractions occurring in younger age groups. Although some deaths relate to IHD and stroke (Table 108), most are attributable to cancer among both males and females. Indeed, almost 9 percent of all cancer mortality may be attributable to this cause.* In total, it is estimated that each year almost twice as many deaths (570) among males are associated with an inadequate consumption of fruit and vegetables as among females (290).

Table 107: Relative risks and attributable fractions for inadequate fruit and vegetable consumption, 1996

Age (years)	All cancer mortality			IHD mortality		
	Relative risk	Attributable fraction (%)		Relative risk	Attributable fraction (%)	
		Female	Male		Female	Male
25–44	1.40	18	22	1.18	9	11
45–64	1.30	11	16	1.18	7	10
65–74	1.20	6	10	1.10	3	5
75+	1.10	3	5	1.00	0	0

Principal sources of base data: NNS 1997 (prevalence); RR estimates derived from multiple sources

Table 108: Deaths and YLL caused by inadequate fruit and vegetable consumption, 1996

	Attributable deaths		Attributable YLL_d	Attributable YLL
	Number	Percentage		
Male (15+)				
All cancers	403	10	6300	9241
IHD	147	4	2536	3725
Stroke	24	2	374	526
All causes	574	4	9210	13,492
Female (15+)				
All cancers	250	7	4204	6424
IHD	31	1	518	745
Stroke	12	1	200	292
All causes	293	2	4922	7461

Principal sources of base data: NNS 1997 (prevalence); NZHIS (mortality); RR estimates derived from multiple sources

* Note however that the evidence base for a causal role for fruit and vegetable consumption in protection against cancers or IHD is less well established than that for many of the other risk factors considered here.

Physical inactivity

The majority of deaths attributable to physical inactivity are related to cardiovascular diseases, with over 740 deaths among males and over 640 deaths among females from IHD and stroke attributable to this cause in 1996 (Table 110a). Significant losses were also associated with cancer and diabetes. For all causes, over 2100 people are estimated to have died prematurely because of physical inactivity in 1996 or 1997, amounting to over 29,000 discounted years of life lost per year (Table 110b). Over 50 percent of YLL occur in middle age (45–64 years), although the impact is evident even in the 25–44 age group.

Table 109a: Relative risks and attributable fractions for physical inactivity and IHD mortality, 1996

Age (years)	RR female	Attributable fractions for IHD: female (%)				RR male	Attributable fractions for IHD: male (%)			
		Māori	Pacific	European /Other	Total female		Māori	Pacific	European/ Other	Total male
45–64	1.8	28	27	23	24	1.8	26	32	24	24
65–74	1.4	18	17	10	11	1.8	27	30	19	19
75+	1.3	17	15	13	13	1.3	16	18	11	11

Table 109b: Relative risks and attributable fractions for physical inactivity and diabetes mortality, 1996

Age (years)	RR female	Attributable fractions for diabetes: female (%)				RR male	Attributable fractions for diabetes: male (%)			
		Māori	Pacific	European /Other	Total female		Māori	Pacific	European/ Other	Total male
25–44	1.4	16	16	14	14	1.4	12	14	15	14
45–64	1.4	16	16	13	13	1.4	15	19	14	14
65–74	1.3	14	13	8	8	1.3	14	15	9	9
75+	1.2	12	10	9	9	1.2	12	13	8	8

Note: RRs are conservative estimates of the effect of physical inactivity on diabetes mortality independent of other risk factors, including obesity.

Table 109c: Relative risks and attributable fractions for physical inactivity and all-cause mortality, 1996

Age (years)	RR female	Attributable fractions: all-cause mortality: female (%)				RR male	Attributable fractions: all-cause mortality: male (%)			
		Māori	Pacific	European /Other	Total female		Māori	Pacific	European/ Other	Total male
25–44	1.4	16	16	14	14	1.4	12	14	15	14
45–64	1.3	12	12	10	10	1.3	11	15	11	11
65–74	1.2	10	9	5	6	1.2	9	11	6	6
75+	1.2	9	8	7	7	1.2	9	10	6	6

Principal sources of base data: US DHHS 1996, NZHS 1996–97

Table 110a: Deaths caused by physical inactivity, 1996

	Māori Attributable deaths		Pacific Attributable deaths		European/Other Attributable deaths		Total Attributable deaths	
	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Male (15+)								
All cancers	14	4	4	4	131	4	149	4
Diabetes	27	14	3	14	32	10	62	12
IHD	77	24	25	29	526	16	628	17
Stroke	8	15	4	18	105	11	117	11
All causes	139	10	48	13	910	7	1097	8
Female (15+)								
All cancers	16	5	4	5	125	4	145	4
Diabetes	33	15	2	10	42	10	77	11
IHD	42	21	10	21	349	13	401	14
Stroke	15	24	7	23	218	14	240	15
All causes	122	12	33	11	891	7	1046	8

Table 110b: YLL attributable to physical inactivity, 1996

Outcome	Māori		Pacific		European/Other		Total	
	YLL_d	YLL	YLL_d	YLL	YLL_d	YLL	YLL_d	YLL
Male (15+)								
All cancers	222	323	60	84	1668	2290	1951	2697
Diabetes	468	704	47	68	469	669	984	1441
IHD	1299	1934	423	626	6866	9331	8588	11,890
Stroke	114	160	53	71	1080	1377	1247	1608
All causes	2519	4040	865	1381	12,897	19,307	16,280	24,728
Female (15+)								
All cancers	262	394	77	123	1525	2079	1865	2596
Diabetes	531	773	32	46	496	663	1059	1482
IHD	635	909	153	225	3323	4094	4110	5228
Stroke	226	319	93	125	2196	2819	2515	3263
All causes	2105	3333	579	933	10,173	14,058	12,857	18,324

Principal sources of base data: US DHHS 1996, NZHS 1996–97, NZHIS
Note: 'all-cause' includes minor causes not shown separately.

Diabetes (type 2)

Diabetes is associated with significant risks for a number of causes of death. Tables 111a and 111b detail the relative risks and attributable fractions for all-cause mortality and IHD related to type 2 diabetes. Ethnic differences are driven by prevalence and, proportionately, three to four times as many deaths among Māori and Pacific people are attributable to diabetes compared with those among European/Others.

Over 600 male deaths and almost 780 female deaths are attributable to type 2 diabetes (respectively, 4 percent and 6 percent of all deaths in people aged 20 and older), based on 1996 prevalence and mortality rates.

Table 111a: Relative risks and attributable fractions for type 2 diabetes and all-cause mortality, 1996

Age (years)	RR female	Attributable fractions for all-cause mortality: female (%)				RR male	Attributable fractions for all-cause mortality: male (%)			
		Māori	Pacific	European /Other	Total female		Māori	Pacific	European/ Other	Total male
30–34	3.0	6	6	2	3	4.7	13	14	5	7
35–39	3.0	8	8	3	4	4.7	17	17	6	8
40–44	3.0	11	11	3	5	4.7	22	22	7	10
45–49	3.4	16	16	5	7	2.3	12	12	4	5
50–54	3.4	21	21	6	8	2.3	15	16	4	6
55–59	2.3	16	16	4	6	2.1	17	17	5	6
60–64	2.3	19	20	5	7	2.1	20	20	6	8
65–69	2.3	22	22	7	8	1.5	13	13	3	4
70–74	2.3	27	28	8	9	1.5	16	16	4	5
75–79	1.3	10	10	3	3	1.1	5	5	1	2
80 84	1.3	12	12	3	4	1.1	6	7	2	2
85+	1.3	14	14	3	5	1.1	8	7	2	3

Table 111b: Relative risks and attributable fractions for type 2 diabetes and IHD, 1996

Age (years)	RR female	Attributable fractions for IHD: female (%)				RR male	Attributable fractions for IHD: male (%)			
		Māori	Pacific	European /Other	Total female		Māori	Pacific	European/ Other	Total male
30–34	12.0	26	27	10	15	3.9	11	11	4	5
35–39	11.0	31	31	12	16	3.6	13	13	4	6
40–44	10.0	35	35	13	18	3.3	15	15	5	6
45–49	8.2	37	37	14	18	3.0	17	17	5	7
50–54	6.7	38	38	13	17	2.7	19	19	6	7
55–59	5.4	38	38	13	17	2.4	20	20	6	8
60–64	4.3	37	38	13	16	2.2	22	22	6	8
65–69	3.5	36	36	12	15	2.0	23	23	7	8
70–74	2.9	36	36	12	13	1.8	23	23	7	8
75–79	2.6	36	36	12	13	1.6	22	22	7	7
80 84	2.5	39	38	14	15	1.5	21	21	6	7
85+	2.3	39	40	16	17	1.4	19	18	6	7

Principal sources of base data: RR compiled from multiple sources; prevalence modelled from NZHS 1996–97

Table 112a: Deaths caused by type 2 diabetes, 1996

Outcome	Māori Attributable deaths		Pacific Attributable deaths		European/Other Attributable deaths		Total Attributable deaths	
	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Male (20+)								
IHD	63	20	18	21	214	6	295	8
Stroke	8	15	2	9	40	4	50	5
Other causes	110	8	31	2	127	9	268	3
All causes	181	14	51	14	381	3	613	4
Female (20+)								
IHD	73	37	18	38	377	14	468	16
Stroke	11	18	6	19	59	4	76	5
Other causes	89	7	21	1	125	5	235	3
All causes	173	17	45	16	561	5	779	6

Table 112b: YLL attributable to type 2 diabetes, 1996

Outcome	Māori		Pacific		European/Other		Total	
	YLL_d	YLL	YLL_d	YLL	YLL_d	YLL	YLL_d	YLL
Male (20+)								
IHD	977	1396	296	431	2301	2982	3574	4809
Stroke	115	158	26	33	369	458	511	649
Other causes	2142	3511	585	960	2422	3864	5149	8336
All causes	3234	5065	908	1425	5092	7304	9234	13,793
Female (20+)								
IHD	960	1307	214	290	2652	3148	3826	4744
Stroke	155	217	73	94	491	605	720	917
Other causes	1483	2202	364	5556	2151	3099	3999	5857
All causes	2599	3726	651	940	5294	6852	8544	11,518

Principal sources of base data: NZHS 1996–97, NZHIS, RR estimates compiled from multiple sources
Notes: all-cause percentages calculated only in relation to mortality for people 20 years and over.

Obesity

In 1996 obesity was estimated to account for over 15,000 discounted years of life lost (Table 114b), from approximately 1070 deaths (Table 114a). Obesity is of greatest significance in relation to diabetes: at least one third of all diabetes related deaths are attributable to obesity, although this proportion is much higher for young Māori (Table 113b). Other major causes of death associated with obesity are IHD (Table 113a) and stroke. For all-cause mortality, approximately 11 percent of all Māori deaths in the 45–64 age group are attributable to obesity compared with 6–7 percent for non-Māori (Table 113c).

Table 113a: Relative risks and attributable fractions for obesity and IHD mortality, 1996

Age (years)	RR female	AF for IHD: female (%)			RR male	AF for IHD: male (%)		
		Māori	Non-Māori	Total		Māori	Non-Māori	Total
45–64	1.7	23	14	15	1.7	22	13	14
65–74	1.5	15	10	10	1.5	12	7	7
75+	1.3	6	5	5	1.3	4	2	2

Table 113b: Relative risks and attributable fractions for obesity and diabetes mortality, 1996

Age (years)	RR female	AF for diabetes: female (%)			RR male	AF for diabetes: male (%)		
		Māori	Non-Māori	Total		Māori	Non-Māori	Total
25–44	7.0	63	48	51	4.5	44	31	33
45–64	5.0	63	49	51	3.8	52	36	38
65–74	3.5	46	34	35	3.0	34	22	23
75+	2.0	18	14	14	2.0	13	7	7

Note: relative risks are conservative estimates of the effect of obesity on diabetes mortality independent of other risk factors including physical inactivity.

Table 113c: Relative risks and attributable fractions for obesity and all-cause mortality, 1996

Age (years)	RR female	AF for all-cause mortality: female (%)			RR male	AF for all-cause mortality: male (%)		
		Māori	Non-Māori	Total		Māori	Non-Māori	Total
25–44	1.4	10	6	6	1.4	8	5	5
45–64	1.3	11	7	7	1.3	11	6	6
65–74	1.3	8	5	5	1.3	6	3	4
75+	1.2	4	3	3	1.2	3	1	2

Principal sources of base data: NNS 1997 (prevalence); RR estimates derived from multiple sources

Table 114a: Deaths caused by obesity, 1996

	Māori Attributable deaths		Non-Māori Attributable deaths		Total Attributable deaths	
	Number	Percentage	Number	Percentage	Number	Percentage
Male						
Diabetes	82	42	72	21	154	29
IHD	50	16	191	6	241	6
Stroke	5	9	29	3	34	3
All causes	105	8	375	3	480	3
Female						
Diabetes	112	50	120	26	232	34
IHD	30	15	165	6	195	7
Stroke	6	10	63	4	69	4
All causes	90	9	503	4	593	4

Table 114b: YLL caused by obesity, 1996

Outcome	Māori		Non-Māori		Total	
	YLL_d	YL	YLL_d	YLL	YLL_d	YLL
Male						
Diabetes	1501	2302	1161	1690	2663	3992
IHD	922	1404	2860	4043	3782	5447
Stroke	91	134	401	567	492	700
All causes	2037	3336	5649	8267	7687	11,603
Female						
Diabetes	1947	2916	1659	2303	3606	5219
IHD	478	687	1758	2237	2236	2924
Stroke	99	143	688	904	787	1047
All causes	1598	2491	6021	8225	7619	10,716

Principal source of base data: NNS 1997, NZHS 1996–97, multiple sources (for relative risk estimate)
Notes: 'all causes' includes minor causes not separately listed; 'diabetes' includes deaths coded to other causes.

It should be noted that this model may underestimate the impact of body weight on health for three reasons: people who are overweight (BMI 25–29.9) but not obese also have (slightly) elevated risks of adverse health outcomes but these are not captured by the model; central adiposity (as measured by waist circumference) is an independent risk factor within each BMI category but is not included in the model; and the model uses relative risk estimates at least partially adjusted for physical inactivity, high blood pressure and high blood cholesterol (to capture the independent effect of obesity) even though these variables may be on the causal pathway from obesity to outcome.

High blood pressure

Based on 1996 mortality, high blood pressure* is estimated to account for approximately 9 percent of all deaths in adults (15 years and older) – over 2400 deaths or 26,500 YLL_d – principally through IHD, stroke and hypertensive heart disease (Tables 115 and 116).

Table 115: Relative risks and attributable fractions for hypertension, 1996

Age (years)	IHD mortality				Stroke mortality			
	Relative risk		Attributable fraction (%)		Relative risk		Attributable fraction (%)	
	Female	Male	Female	Male	Female	Male	Female	Male
25–44	8.5	4.4	20	20	3.1	2.9	6	12
45–64	3.5	2.5	30	29	2.7	2.6	22	31
65–74	2.1	1.6	29	18	2.1	1.9	30	27
75+	1.4	1.5	19	19	1.7	1.9	26	31

Principal source of base data: prevalence – NNS 1997; RR – compiled from multiple sources
Note: hypertension: systolic ≥ 160 mm Hg and/or diastolic ≥ 95 mm Hg.

* Since blood pressure appears to be continuously related to IHD or stroke risk, considering only those with blood pressure above a threshold to be at risk will underestimate the total impact, but may better reflect the *modifiable* impact of this risk factor.

Table 116: Deaths and YLL caused by hypertension, 1996

Outcome	Attributable deaths		Attributable YLL_d	Attributable YLL
	Number	%		
Male (15+)				
IHD	782	21	10,051	13,697
Stroke	311	30	3192	4029
Hypertensive heart disease	122	100	1515	2091
All causes	1215	9	14,758	19,817
Female (15+)				
IHD	617	21	6339	7952
Stroke	413	26	3799	4602
Hypertensive heart disease	167	100	1605	1992
All causes	1197	9	11,743	14,546

Principal sources of base data: NNS 1997, NZHIS, multiple sources (relative risk estimates).
Notes: all cause is the sum of the listed specific causes.

High blood lipids (total cholesterol)

Based on 1996 prevalence and mortality levels, over 800 male deaths and 700 female deaths are attributable each year to hypercholesterolaemia (total blood cholesterol ≥ 6.5 mmol/L), amounting to 22 percent and 25 percent of all male and female IHD deaths respectively (Table 118). Because of the high relative risks and the increased prevalence at younger ages, the attributable fractions are significant even for younger ages (Table 117). Over 18,000 YLLd are attributable to hypercholesterolaemia. There is insufficient evidence to attribute any non-IHD deaths to hypercholesterolaemia.

The attributable fractions presented here may underestimate the impact of blood lipid levels on health for two reasons: the risk of IHD appears to be continuously related to blood cholesterol level, so considering only those above an arbitrary threshold level to be at risk will underestimate the population impact; and other lipid fractions – in particular low HDL cholesterol – have not been included even though they represent independent risk factors for IHD and possibly other diseases.

Table 117: Relative risks and attributable fractions for high blood cholesterol and IHD mortality, 1996

Age (years)	Relative risk		Attributable fraction (%)	
	Female	Male	Female	Male
35–39	4.0	4.0	28	42
40–44	3.5	3.5	31	42
45–49	3.0	3.0	32	40
50–54	2.6	2.6	34	37
55–59	2.3	2.3	36	34
60–64	2.1	2.1	36	30
65–69	2.0	2.0	37	27
70–74	1.9	1.9	35	23
75–79	1.8	1.8	32	19
80–84	1.7	1.7	26	15
85+	1.6	1.6	16	11

Principal source of base data: prevalence – NNS 1997; relative risk – compiled from multiple sources
Notes: high blood cholesterol defined as ≥ 6.5 mmol/L.

Table 118: Deaths and YLL caused by high blood cholesterol, 1996

	Attributable deaths		Attributable YLL	
	Number	Percentage	Discounted	Undiscounted
Male (15+)				
IHD	839	22	11,294	15,665
Female (15+)				
IHD	736	25	6860	8596

Principal sources of base data NNS 1997, NZHIS, multiple sources (relative risk)
Notes: percentage is of total IHD deaths, by gender in the adult population.

Potential future impact of risk reduction

All of the above results are based on 1996 prevalence and mortality levels, and the attributable deaths and YLL relate to the complete elimination of the risk factor. In reality, however, changes in prevalence occur over long periods of time, and both risk factor prevalences and their impacts on mortality influence – and are influenced by – underlying changes in the demography of the population: its size, age structure and ethnic composition in particular.

A scenario was developed for one decade from the current data point (1996–2006) in which a 10 percent reduction in prevalence was assumed for current smoking (with a concomitant increase in ex smoking prevalence), physical inactivity, hypercholesterolaemia, inadequate fruit and vegetable consumption, hypertension, and harmful/hazardous alcohol consumption (AUDIT 8 or more, with a concomitant increase in the prevalence of AUDIT less than 8, but assuming no change in 'abstinence' levels of consumption).

For diabetes and obesity, an upward trend is expected in future, and therefore it was unrealistic to assume a reduction from 1996 prevalence. Instead, the type 2 diabetes related health gain was modelled on the basis of holding the diabetes prevalence at the 1996 level. The health gain in 2006 would then be the difference between current (1996) diabetes related mortality and that projected for 2006 (as modelled by Tobias and Roberts 1999). For obesity, the 2006 prevalence was estimated by projecting a linear trend, based on the increase between the 1989 Life in New Zealand survey (Hillary Commission 1990) and the 1997 National Nutrition Survey (resulting in a 50 percent increase on 1996–97 prevalence for 2006). It was then assumed that additional intervention could realistically slow this rate of increase so that the prevalence reached in 2006 would be 10 percent less than that projected.

Table 119 summarises the results of this scenario: reducing physical inactivity and hypertension are identified as producing the greatest annual gains in terms of deaths or YLL prevented for both males and females.

Table 119: Future impact on mortality and YLL, 2006, from one risk factor reduction scenario

Risk factor	Male			Female			Total		
	Deaths prevented: number (%)	YLL_d saved	YLL saved	Deaths prevented: number (%)	YLL_d saved	YLL saved	Deaths prevented: number (%)	YLL_d saved	YLL saved
Behavioural									
Smoking	38 (1)	542	866	29 (2)	360	588	67 (1)	902	1454
Alcohol consumption	36 (5)	604	1082	9 (3)	108	194	45 (5)	712	1276
Inadequate fruit and vegetable	62 (7)	984	1423	34 (9)	558	827	96 (9)	1542	2250
Physical inactivity	134 (9)	1887	2791	128 (9)	1547	2186	262 (9)	3434	4977
Biological									
Diabetes (type 2)	20 (2)	239	318	32 (3)	341	442	52 (3)	580	760
Obesity	86 (9)	1340	1999	104 (9)	1310	1828	190 (9)	2650	3827
High blood pressure	129 (8)	1528	2038	127 (8)	1206	1480	256 (8)	2739	3518
Total cholesterol	83 (8)	1047	1428	69 (7)	593	736	152 (8)	1640	2164

Note: percentage is of all deaths attributable to the risk factor in 1996.

In the scenario modelled, smoking attributable mortality does not fall as rapidly as might be expected, especially when compared with the reduction in mortality expected from a complete elimination of smoking (see Tables 105a and 105b). This apparent discrepancy is due to the residual elevated risks of ex smokers. Over time, however, risks of ex smokers return to baseline – for example, after at least three years of non-smoking, the higher risk of IHD for ex smokers declines to risk levels equivalent to never smokers (Wilson 1994), although some risks take much longer than this to return to baseline. Over a longer timeframe, therefore, it is likely that a 10 percent reduction in smoking prevalence would result in a much greater reduction in the total attributable deaths than that identified in Table 119 – possibly approaching several hundred deaths per year. Further, impacts of passive smoking have not been included in these calculations, making this estimate a conservative one. On this basis, smoking remains the most significant risk factor of those examined in this study.

For alcohol, no change is modelled in the prevalence of 'abstainers'. If, however, efforts to reduce the level of hazardous/harmful alcohol consumption also result in an increase in 'abstainers', the deaths prevented by the reduction in more harmful levels of consumption will be offset by a reduction in the number of IHD deaths prevented. But it is also important to note that the impacts of alcohol consumption are significantly different at different ages, with the major adverse impact occurring at younger ages, particularly from injury (including suicide); at older ages, deaths are both caused and prevented at all levels of consumption. The scenario modelled here demonstrates that the effects of alcohol on population health status are complex.

It should be borne in mind that only a single scenario is presented here. If, over the next decade, risk factor prevalences could be reduced even further than suggested here, then the health gains could be significantly greater than those identified.

On the other hand, for most risk factors, the scenario involves a reduction in 1996 prevalence levels for each age, gender and ethnic group, and the estimated health gains will be different for population subgroups where this does not occur. One major area of concern is the increasing prevalence of smoking in young women: if this trend is not reversed there will be significant adverse impacts on female mortality over the next several decades. For smoking, there is also a significant impact on the health of many children and non-smoking adults from passive smoking (Bonita et al 1999), the impacts of which are not fully captured in this model. Similarly, the

increasing prevalence of obesity is of concern, particularly because of the already very high prevalence of type 2 diabetes in Māori and Pacific people and the very high mortality risk that diabetes confers.

Summary and conclusions

Population attributable risk (PAR) is a fundamental epidemiological measure of considerable relevance as a policy analysis tool. This report has made use of recent national risk factor prevalence surveys and an exhaustive literature search for appropriate relative risk estimates, in order to generate robust, highly differentiated PARs for New Zealand in the mid 1990s, with regard to eight major risk factors.

Even so, the prevalence and relative risk estimates are drawn from different data sources, and relate to different populations and different periods. Hence the absolute values calculated should be treated with caution. The method is more useful in quantifying the *relative* impacts of the different risk factors analysed. It also provides a basis for modelling different future scenarios, one of which has been presented here. Such modelling enables observed changes in outcomes to be compared with those predicted based on achievable reductions in risk exposures.

The most interesting results are perhaps those obtained by applying the 1996 attributable fractions to the 1996 mortality data, although caution in interpreting these results as indicative of the current burden caused by each risk factor is necessary. These results are summarised in Table 120.

Table 120: Summary of results: mortality attributable to eight risk factors, New Zealand 1996

Risk factor	Population group (15 years and over)	Maximum impact on deaths (per year)	Maximum impact on years of life lost (per year, discounted)
Smoking	Male	2778 caused	34,939
	Female	1524 caused	18,655
	Total	4302 caused (15.2%)	53,594 (16.4%)
Alcohol consumption	Male	553 caused 612 prevented 59 prevented (net)	3367 (net) 593 (net) 3960 (net) (1.2%)
	Female	240 caused 364 prevented 124 prevented (net)	
	Total	793 caused 976 prevented 183 prevented (net) (–0.6%)	
Inadequate fruit and vegetable	Male	574 caused	9210
	Female	293 caused	4922
	Total	867 caused (3.1%)	14,132 (3.8%)
Physical inactivity	Male	1097 caused	16,280
	Female	1046 caused	12,857
	Total	2143 caused (7.6%)	29,137 (8.9%)
Diabetes (type 2)	Male	613 caused	9234
	Female	779 caused	8544
	Total	1392 caused (4.9%)	17,778 (5.4%)
Obesity	Male	480 caused	7687
	Female	593 caused	7619
	Total	1073 caused (3.8%)	15,306 (4.7%)

Table 120 continued

Risk factor	Population group (15 years and over)	Maximum impact on deaths (per year)	Maximum impact on years of life lost (per year, discounted)
High blood pressure	Male	1215 caused	14,758
	Female	1197 caused	11,743
	Total	2412 caused (8.7%)	26,501 (8.1%)
Total cholesterol	Male	839 caused	11,294
	Female	736 caused	6860
	Total	1575 caused (5.6%)	18,154 (5.6%)

Principal sources of data: NZHIS, census 1996, NZHS 1996–97, NNS 1997, English et al 1995
Note: percentage is of total deaths (28,377 in 1996); years of life lost are YLL_e discounted at 3 percent per year.

Smoking is by far the leading risk factor of the eight considered, with a total of approximately 4300 deaths attributable in 1996 (15 percent of all deaths). Physical inactivity ranks third (after high blood pressure) in terms of deaths but second in terms of years of life lost; both these causes were associated with approximately 2100 to 2400 deaths in 1996. The total elimination of high blood cholesterol and diabetes would each prevent approximately 1400 to 1600 deaths per year. If the prevalence of obesity could be reduced form current levels to zero, at least 1000 deaths would be prevented per year. Finally, if all New Zealanders consumed at least five servings of fresh fruit and vegetables per day, mortality would be reduced by over 800 deaths per year.

These results indicate significant scope for health gain through further risk reduction. This analysis also helps to place less well known risk factors in perspective, alongside better known ones such as smoking. However, the PAR analysis is only one dimension of the policy debate: information on the cost effectiveness and acceptability of available interventions capable of modifying risk exposure is also necessary.

Based on the impact assessment reported here, and the accumulating evidence on the cost effectiveness of interventions, it seems reasonable to conclude that the physical inactivity – obesity – diabetes nexus is emerging as worthy of the same level of societal concern as that already afforded to smoking and alcohol (see for example, US Department of Health and Human Services 1996, National Health Committee 1998a). This analysis also lends support to the notion that improved recognition and management of high blood pressure, high blood cholesterol and diabetes in primary health care settings represent a major potential contribution to population health. Finally, increasing the consumption of fruit and vegetables would appear to be an achievable target (Ministry of Health 1998b), one which may have a surprisingly large impact on population health, preventing over 800 deaths each year (approximately 8–9 percent of all cancer deaths and a small proportion of IHD deaths).

One approach to summarising risk factor impact information, which has been found useful by policymakers and analysts in the United States (McGinnis and Foege 1993), is the calculation of 'actual causes of death', and this analysis has been applied to the New Zealand 1996 mortality data (Box 28).

Box 28: 'Actual' causes of death in 1996

Cause of death analysis is usually carried out at the level of the diseases and injuries that are the direct (underlying) causes of death. Alternatively, deaths can be attributed to risk factors rather than diseases, by categorical attribution (CA) where possible and counterfactual analysis (PAR) where necessary.

In order to add estimates of attributable fractions of all-cause mortality for different risk factors, the assumption is made that the risk factors are independent. The probability of dying from other causes is then the product of the probabilities of *not* dying from each specified cause:

$$AF_0 = (1 - AF_1)\,(1 - AF_2)\,(1 - AF_3)\,...\,(1 - AF_n)$$

Because of this adjustment, the number of deaths attributed to each risk factor in Table 121 is lower than that estimated for each risk factor separately in Table 120 (for example, smoking is estimated to account for 3500 rather than 4300 deaths, because of competition between causes for the same pool of survivors).

The results of the PAR analysis reported here have been combined with causes of death assigned by categorical attribution to derive the results shown in Table 121 (rounded to the nearest hundred to reflect uncertainty in the estimates).

Table 121: Actual causes of death, New Zealand, 1996

Cause	Number of deaths (%)	Method used
Smoking	3500 (13%)	PAR (some CA)
Alcohol*	600 (2%)	PAR (some CA)
Lack of fruit and vegetables	700 (3%)	PAR
Physical inactivity	1700 (6%)	PAR
Diabetes	1100 (4%)	PAR
Obesity	900 (3%)	PAR
High blood pressure	1900 (7%)	PAR (some CA)
High blood cholesterol	1300 (5%)	PAR
Road traffic crashes	500 (2%)	CA
Other injury hazards** (eg, fire, firearms, machinery, poisons)	600 (2%)	CA
Violence (suicide, assault)	600 (2%)	CA
Microbes	1800 (6%)	CA (see Christie and Tobias 1998)
Radiation (ultraviolet)	200 (1%)	CA (skin cancer, melanoma)
Pollution/toxics	no data	Included in 'Other or unknown' below
Sexual behaviour	100 (< 0.1%)	CA (HIV/STDs, cervix cancer, maternal mortality, ectopic pregnancy)
Illicit drug use	100 (< 0.1%)	CA (heroin abuse, methadone abuse)
Other or unknown cause	12,400 (44%)	
All causes	28,000 (100%)	

Source of base data: NZHIS and multiple other sources
* deaths caused only – not net of deaths prevented
** unintentional injury hazards only

The eight risk factors considered in this report collectively account for approximately 12,000 of the 28,000 deaths registered in 1996 (about 40 percent). However, the model used does not adjust for clustering of risks, so biasing the estimate upwards. On the other hand, conservative estimates of relative risk and discretisation of risk categories bias the estimate downwards.

Data limitations do not allow 'diet' to be included in the table as an 'actual' cause of death. Yet if the contribution of diet to obesity, diabetes, blood lipid profile and high blood pressure could be estimated, and added to the estimate for cancer and other deaths attributable to inadequate intake of fruit and vegetables, diet may in fact rival smoking in terms of its impact on mortality.

This analysis provides a different perspective on priorities than that provided by analysis of cause of death data at disease level. It would, of course, be even more useful if the impact of these risk factors on morbidity (quality of life) outcomes could be estimated, in addition to their impact on fatal outcomes. Finally, the approach should be extended to include such 'risk factors' as socioeconomic status, unemployment, stress, access to primary health care – and indeed the full range of social, cultural and economic determinants of health, acting over the life course and at the group as well as the individual level. However, the necessary data and models to do so are still being developed.

PAR methodology may be even more useful as a policy analysis tool when applied to monitoring *trends* in risk exposures over time. This approach can help to evaluate national (or subgroup) progress when different risk factors are trending in opposite directions. For example, in New Zealand in the 1990s, obesity and diabetes increased in prevalence while high cholesterol and high blood pressure decreased and smoking essentially remained stable (decreasing in some age, gender and ethnic subgroups but increasing in others).

Although simple univariate PAR methods can illustrate the scope available for health gain through risk reduction, and even identify the relative impact of each risk factor, the method has major limitations. It requires the assumption of independence of risks, which is clearly violated in reality. It is essentially static rather than dynamic in a temporal sense. The assessment of exposure is generally highly simplified, with risk often being simply dichotomised, as done here. Although robust prevalence data were available, New Zealand specific relative risk estimates were largely unavailable. Thus the relative risk data come from different populations and often refer to different time periods. Finally, the only outcomes for which robust relative risk estimates could be obtained were mortality outcomes.

More sophisticated dynamic modelling methods are required to examine the impacts of multiple risk factors acting jointly on multiple fatal and non-fatal outcomes, within a realistic sociodemographic context (which is itself influenced by these impacts). Such methods are not yet available. Even if they were, better data would still be needed to input into the model. In particular, up to date prevalence data are required – especially for older people, who experience the highest rates of the outcomes of interest (such as premature death or disability), and for older Māori and Pacific people in particular. Also, New Zealand specific relative risks would significantly improve the validity of the estimates; large scale cohort studies are becoming less expensive as record linkage methods improve, and could usefully complement cross sectional prevalence surveys. Such cohort studies may also enable data on non-fatal as well as fatal outcomes to be collected.

Perhaps more importantly, the risk factors monitored and analysed need to be extended from the proximal biological and behavioural variables considered in this report to the more distal social, economic and cultural determinants of health – including group level as well as individual level causes. Until these analyses can be done, however, the results reported here may provide a valuable insight into the scope remaining for health gain through risk reduction.

Part IV summary and conclusions

Intra-national comparisons of health outcomes reveal significant scope for improving equity and, in so doing, achieving improvements in levels of health for the New Zealand population as a whole. Particularly noteworthy are the ethnic gaps and socioeconomic gradients (and their interactions) in both fatal and non-fatal health outcomes. Māori life expectancy at birth is nine years lower than non-Māori for females, and eight years lower for males. After adjusting the groups for age, Māori are about one third more likely to be dependently disabled than non-Māori. The gap in health expectancy (as illustrated by independent life expectancy, or ILE) is thus wider than that in life expectancy: approximately 10 years for females and 8.5 years for males at birth. For most major diseases and injuries – including IHD, stroke, lung cancer, diabetes and road traffic injuries – Māori incidence, prevalence and mortality rates are 50 to 100 percent higher than non-Māori rates once adjusted for age. Turning to socioeconomic inequalities, the gap in life expectancy at birth between the most and least deprived NZDep96 deciles is about nine years for males and almost seven years for females. A similar socioeconomic gradient is seen with respect to disability.

Yet such within-population comparisons do not locate the New Zealand population as a whole in a global context. Unfortunately, international comparisons have had to be restricted to mortality outcomes because the measurement of disability or morbidity outcomes generally is poorly standardised. Within this limited frame of comparison, New Zealand appears below average on most indicators in comparison with other OECD countries. Although exact rankings vary, depending on the indicator and also from year to year, New Zealand males fare somewhat better in the rankings than their female counterparts. For example, male life expectancy at birth in New Zealand is about one third of a year below the OECD average and a little over one year below that of Australian males. But New Zealand females are placed lower on the OECD rankings for this indicator, and in 1996 had a life expectancy at birth approximately one year below the OECD average and over 1.5 years below that of Australian females. The relatively narrow gender gap in New Zealand (with respect to mortality) thus reflects poor female rather than good male performance in terms of longevity.

A similar story is revealed if other mortality indicators are examined: YLL or mortality rates, both age specific (for example, infant mortality rates) and age standardised, both all-cause rates and rates for most major causes (for example, chronic diseases and injuries). The historical trend over the past quarter century does show that New Zealand has achieved significant improvement (for most age, gender and ethnic subgroups) for all indicators over this period. However, many other OECD countries have improved even more rapidly, with the result that New Zealand has fallen from near the top in the early 1970s to below the middle of the countries examined in the mid 1990s. Countries such as Australia, which had life expectancies below those of New Zealand in the early 1970s, have now overtaken us. International benchmarking thus indicates that New Zealand has scope to improve overall, but the intra national inequalities suggest that such improvement will only result from greater equity of health outcomes within New Zealand.

One way to estimate this potential for improvement in equity and in whole-of-population outcomes is to categorise diseases and injuries as ‘avoidable’ and ‘unavoidable’ – the concept of avoidability being understood in terms of responsiveness to health sectoral intervention (whether through health promotion, disease prevention, or treatment). With increasing age, avoidability becomes difficult to assign, so an arbitrary age cut off is applied in this approach, usually 65 years. However, the analysis presented here extends the upper age limit to 75, and also extends the range of conditions considered avoidable to reflect recent developments in health promotion and health care technology.

Within the 0–74 age range, it is estimated that 70 percent of all deaths could be categorised as potentially avoidable (approximately 9000 deaths in 1996–97). About half of these deaths are responsive to primary prevention strategies (health promotion and disease prevention) and thus are described as 'primary avoidable mortality', or PAM; approximately one quarter are sensitive to early intervention (for example, screening, effective chronic disease management delaying disease progression), typically in the primary health care setting ('secondary preventable mortality' or SAM); and the remaining quarter (approximately) could be avoided by effective medical and surgical treatment of established disease ('tertiary avoidable mortality', or TAM).

Although primary prevention would have the biggest impact on avoidable mortality, the highest relative inequalities are found in secondary avoidable mortality rates. Improving access to effective primary health care services for ethnic and socioeconomic subgroups with poorer health status is therefore of particular importance.

Analysis of avoidable hospitalisations tells a similar story. In 1997–98, almost one third of total hospitalisations in the 0–74 age group (approximately 100,000 per year, excluding maternity, mental and disability support services) were assessed as having been potentially avoidable, about one third of these through population-based (health promotion) interventions ('preventable hospitalisations' or PH) and two thirds through more effective primary health care ('ambulatory sensitive hospitalisations', or ASH). Māori and Pacific people have rates of avoidable hospitalisations approximately 60 percent higher than European/Others: taking differences in ethnic group age structures into account, this corresponds to 6600 and 2800 excess hospitalisations in 1997–98 respectively.

People aged 0–74 living in the most deprived areas of New Zealand had twice the risk of being admitted to a hospital for an avoidable cause than their more advantaged counterparts in 1997–98. Had all New Zealanders experienced the avoidable hospitalisation rates of the least deprived subgroup, some 26,000 fewer avoidable hospitalisations would have occurred among those aged 0–74 years.

The best opportunity for reducing both the ethnic and the socioeconomic gaps (which, of course, interact) in avoidable hospitalisations can be found in the subcategory of ASH. ASH has both the highest rate differentials and accounts for the largest share of avoidable hospitalisations. This mirrors the findings in regard to SAM. Indeed, the high ethnic and socioeconomic inequalities in both ASH and SAM rates indicate only limited success of the health sector (and especially of primary care) in mitigating the impact of socioeconomic and ethnic inequalities on health outcomes. At the same time, these findings also indicate the considerable scope for gains in both fatal and non-fatal health outcomes potentially available through improvements in primary health care services. A number of innovative developments in primary and integrated care are already in progress, but have not yet had sufficient time or become sufficiently diffused nationwide to influence outcomes at the population level. Both the Ministry of Health and the National Health Committee are currently consulting widely on strategies to improve primary health care.

In the final analysis, however, the scope for health gain through the agency of health sector organisations is limited, even if the sector is broadly defined (as here) to include a range of health promotion, health protection and disease prevention services in addition to treatment and rehabilitation services. Individuals, families, communities and all sectors of government at both local and central levels can act to reduce exposure to known chronic disease and injury risk factors. The analysis of 'population attributable risks' (PARs) presented here – albeit univariate, decontextualised and restricted to the major chronic disease risk factors – reveals that major gains are still possible through lifestyle modification.

Smoking rates appear to have stabilised at around 24 percent of the population overall, but with particularly worrying trends among youth. Total elimination of smoking would prevent approximately 4300 deaths each year. More realistically, a 10 percent reduction in smoking prevalence (from 24 percent to 21.5 percent approximately) over the next decade, assuming no change in smoking intensity, would reduce mortality by approximately 70 deaths per year in the short run, and much more in the long run once the excess risk of ex-smokers has had time to dissipate.

Alcohol consumption has both positive and negative effects on health. Overall, the negative consequences outweigh the positive, at least in terms of years of life lost. In 1996–97 a net loss of approximately 4000 years of life* is estimated. If the prevalence of hazardous drinkers declined by 10 percent from this level (without any change in the proportion of abstainers) by 2006, the net loss of life years would be decreased by approximately 700 each year.

The impact of diet is difficult to estimate from available data. Energy intake is partly captured in obesity rates, fat intake in blood cholesterol, and salt intake in the prevalence of high blood pressure. The only dietary variable measured directly was fruit and vegetable consumption. If everyone ate five or more helpings per day, mortality would reduce by over 800 deaths per year. More realistically, if the proportion of the population consuming at this level increased by 10 percent from the 1996–97 rate, then by 2006 about 90 fewer deaths would occur each year.

Improvements in physical activity levels would have direct benefits as well as acting indirectly through obesity and diabetes. Although New Zealand already has relatively high rates of participation in leisure time physical activity, at least one third of adults are currently insufficiently active. If all adults enjoyed at least the recommended minimum level of 2.5 hours per week of moderate intensity physical activity, approximately 2200 fewer deaths would occur per year. More realistically, if physical activity levels could be increased by 10 percent by 2006, mortality would decline by about 260 deaths per year.

Obesity rates have been increasing over the past decade (if not longer), as has the prevalence of type 2 diabetes. At present, obesity is conservatively estimated to be associated with at least 1000 deaths per year. If the rate of increase in the prevalence of obesity could be reduced by 10 percent by 2006, it is estimated that about 190 deaths per year would be prevented.

For type 2 diabetes – for which physical activity and obesity are major modifiable risk factors – stabilising prevalence at current levels would prevent approximately 50 deaths per year. At present, diabetes is estimated to be directly associated with approximately 1200–1400 deaths per year (over twice the number actually coded to diabetes on death records).

High blood pressure and high blood cholesterol are major cardiovascular risk factors, with very high prevalences in the older age groups. In both cases, there is some evidence that prevalence may be falling slowly as more older people are screened and appropriately managed, and as food choices and cooking practices are becoming healthier. Nevertheless, at current prevalence levels, total elimination of these risk factors would reduce mortality by about 2400 and 1600 deaths per year respectively. The more realistic scenario of a further 10 percent reduction in prevalence by 2006 would be associated with reductions in mortality of approximately 260 and 150 deaths per year respectively.

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* measured by the remaining life expectancy method using West level 26 model life table as the standard, and discounted at 3 percent per year

Such reductions in risk exposures could therefore make major contributions to population health gain. Nevertheless, it should not be forgotten that these behavioural (lifestyle) and physiological risk factors operate at the level of the individual and are based on studies that have examined inter individual differences in health outcomes. Thus they tell us who will develop the outcome, but may not identify the major causes of differences in the rates of these outcomes between social groups. That is, at the group level, other factors – the social, cultural and economic determinants of health – may be more relevant as the focus of policy intervention. Such determinants may operate by shaping social norms of behaviour or constraining lifestyle choices, or may act independently of the behavioural and biological risk factors that are so important at the individual level. Future editions of this report may be able to include analyses of these major group level variables alongside the individual level lifestyle (behavioural) and physiological risk factors.