

# **Nutrition and the Burden of Disease**

New Zealand 1997–2011

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## Foreword

Good nutrition is essential for good health. However, the full impact of non-optimal nutrition on New Zealanders' health has not been measured previously. Such an assessment is the main purpose of this joint study between the Ministry of Health and the University of Auckland.

The report assesses how many premature deaths and years of life lost are attributable to nutrition-related factors in this country. Overall, this report estimates that nutrition plays a role in about 11,000 deaths a year in New Zealand (two in every five deaths), of which approximately 8000–9000 reflect diet and 2000–3000 reflect physical inactivity. With over 4500 premature deaths in 1997 attributed to high cholesterol, the report highlights the health impact of prolonged high intake of saturated fat. This report also finds substantial burdens from obesity and overweight (due in part to decreasing levels of physical activity), high blood pressure and lack of fruit and vegetable intake.

While the burden from these nutrition-related risk factors is large, the potential benefits from modest improvements in our diet are also considerable. Perhaps not widely appreciated is just how quickly such benefits might accrue – even modest improvements in diet could prevent hundreds of deaths annually within just a few years.

The estimates of attributable and avoidable burden reported here support the decision to include improving nutrition and reducing obesity as priority objectives within the New Zealand Health Strategy. The results will contribute to developing *Healthy Eating – Healthy Action* implementation plans to achieve these objectives. This report also provides valuable baseline estimates for the burden of disease against which progress towards the Ministry of Health's societal and system outcomes of better health, reduced inequalities, and equity and access can be measured.

The findings of this report should also influence policy in many other sectors, from agriculture to urban planning, given the many different agencies that contribute to healthy nutrition and physical activity levels in New Zealand.

We invite readers to comment on the content, relevance and direction of this report and how its findings might be translated into policy and improved health for New Zealanders. Please direct any comments to Public Health Intelligence, Ministry of Health, PO Box 5013, Wellington.



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## Disclaimer

The opinions expressed are those of the authors and do not necessarily reflect the views of the Ministry of Health. The Ministry of Health accepts no liability for decisions or actions based on the contents of this report.

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# Executive Summary

Nutrition is a key determinant of health. However, while it is established that nutrition-related risk factors cause heart disease, diabetes, stroke, cancer and other major health problems, their full impact on the health of New Zealanders has never been accurately quantified. The objective of this study was therefore to estimate, for selected nutrition-related risk factors, the attributable burden of disease in 1997 as well as the burden of disease that could be avoided in 2011 if exposure to these risk factors were reduced through policy interventions.

To estimate the attributable and avoidable burdens, the study uses comparative risk assessment methodology developed by the World Health Organization and used in the *World Health Report 2002* (WHO 2002).

The risk factor–disease relationships studied were limited to those where good evidence for a causal association exists. In this report, the burden of disease is restricted to fatal outcomes only. Therefore, the measure of burden is mortality, expressed as deaths or years of life lost (YLL).

## Attributable burden

The attributable burden gauges the impact of historical levels of risk. It is defined as the proportion of current disease resulting from past risk factor exposure. Table 1 presents a summary of the attributable burden estimates for New Zealanders in 1997 for the selected risk factors.

**Table 1:** Attributable mortality for nutrition-related risk factors, New Zealand, 1997

Risk factor	Attributable mortality <sup>1,7</sup>					
	Ischaemic heart disease	Stroke <sup>2</sup>	Diabetes <sup>3</sup>	Cancer <sup>4</sup>	Total <sup>6</sup>	Percentage of all deaths
Total blood cholesterol	4,096 (3,919–4,246)	625 (286–870)	– –	– –	4,721 (4,205–5,116)	17 (15–19)
Systolic blood pressure	2,542 (2,033–2,898)	1,157 (810–1,407)	– –	– –	3,699 (2,843–4,305)	13 (10–16)
Body mass index	1,561 (1,243–1,852)	367 (174–517)	1,231 (1,208–1,247)	268 (232–303)	3,154 <sup>5</sup> (2,591–3,645)	11 (9–13)
Vegetable and fruit intake	1,171 (703–1,915)	179 (65–276)	– –	209 (112–404)	1,559 (880–2,595)	6 (3–9)

1 Number of deaths (95 percent confidence interval).

2 Ischaemic stroke only for total blood cholesterol, vegetable and fruit intake, and body mass index.

3 Numbers are not adjusted for cardiovascular disease/diabetes overlap (see text for details).

4 Vegetable and fruit intake – oesophageal, stomach, colorectal and lung cancers; body mass index – post-menopausal breast and colorectal cancers.

5 Numbers adjusted for cardiovascular disease/diabetes overlap (see text for details).

6 Estimates do not add across risk factors since disease events can be caused by the joint or sequential actions of two or more risk factors.

7 Years of life lost are not included in the table as percentages of years of life lost are similar to those of death counts.

These nutrition-related risk factors accounted for a substantial proportion of the chronic disease burden in New Zealand in 1997; for example, 70 percent or more of stroke and heart disease mortality can be attributed to the combined effects of these risks. More than 80 percent of diabetes can be attributed to higher than optimal body mass index. Non-optimal body mass index and fruit and vegetable intake were also responsible for over 6 percent of deaths due to cancer.

Ranking these nutrition-related risk factors relative to other major risk factors as causes of death demonstrates that the former are among the leading contributors to the burden of premature mortality in New Zealand. Only tobacco consumption ranked higher, with approximately 5000 deaths in 1997.

Approximately 11,000 deaths (40 percent of all deaths or 37 percent of years of life lost) were estimated to be attributable to the joint effects of these nutrition-related risk factors. Of these deaths, approximately 8000–9000 reflect diet and 2000–3000 reflect physical inactivity. About 34 percent of years of life lost due to nutrition occurred among middle-aged adults (45–64 years). Years of life lost were equally distributed between males and females.

## Avoidable burden

Of sometimes more relevance to policy than attributable burden, but also more difficult to estimate accurately, is the question of how many deaths can be avoided in the future. Avoidable burden is defined as the quantum of disease that could be prevented if future risk factor exposure is reduced as specified in an intervention scenario. Intervention scenarios were selected to represent modest changes in risk exposures towards the distribution conferring minimum risk (the theoretical minimum distribution) that could realistically be achieved given existing and potential interventions. Table 2 summarises the intervention scenarios selected.

**Table 2:** Intervention scenarios

Risk factor	Theoretical minimum distribution (mean)	Shift towards the theoretical minimum distribution under intervention scenario (%)	Change in risk factor <sup>1</sup>
Total blood cholesterol	3.8 mmol/L	10	0.1 mmol/L decrease (0.2–0.4 mol/L decrease in older age groups)
Systolic blood pressure	115 mmHg	16	0.5 mmHg decrease (2–3 mmHg decrease in older age groups)
Body mass index <sup>2</sup>	21 kg/m <sup>2</sup>	19	1.0 kg/m <sup>2</sup> increase
Vegetables and fruit	600 g/day	44	40 g increase (all age groups)

1 These changes are over and above what would be expected with continuation of current trends.

2 Unlike other risk factors, the intervention scenario for BMI did not include an improvement in the current situation since most experts believe the obesity epidemic has yet to peak. Consequently, the intervention scenario for BMI involves a smaller increase in BMI than that projected if current trends continue.



Table 3 presents a summary of the avoidable burden estimates for New Zealand in 2011, based on the selected intervention scenarios.

**Table 3:** Avoidable mortality for nutrition-related risk factors, 2011

Risk factor	Risk factor levels						Avoidable mortality in 2011 under intervention scenario (death count)	
	Current level, 1997 (mean)		Projected level in 2011 based on current trends		Projected level in 2011 under intervention scenario			
	Male	Female	Male	Female	Male	Female	Male	Female
Total blood cholesterol (mmol/L)	5.7	5.7	5.6	5.6	5.5	5.5	179	121
Systolic blood pressure (mmHg)	123.3	120.1	122.4	119.6	121.7	119.2	146	136
Body mass index (kg/m <sup>2</sup> )	26.2	26.1	27.5	27.4	27.2	27.1	182 <sup>1</sup>	203 <sup>1</sup>
Vegetable and fruit intake (g/day)	420	404	460	450	500	490	200	134

1 Numbers adjusted for cardiovascular disease/diabetes overlap – see text for details.

Under the specified intervention scenarios, approximately 300 deaths would be avoided in 2011 from feasible changes in each risk factor distribution. Changes such as those illustrated in the intervention scenarios or greater have been observed in New Zealand or internationally for these risk factors; for example, reductions of 0.3 mmol/L or more in total blood cholesterol have been observed over a decade in the USA, the UK, Australia and New Zealand, and recent studies of ‘5 a day’ campaigns have demonstrated increases in vegetable and fruit intake similar to the intervention modelled here.

## Policy implications

The formulation of specific policy options to improve nutrition, encourage physical activity and maintain healthy body weight is beyond the scope of this report. However, a number of policy approaches to achieve the modelled intervention scenarios are outlined in the report. For example, if more New Zealanders followed the *Food and Nutrition Guidelines for Healthy Adults* (Ministry of Health 2002b), this could reduce blood pressure and blood cholesterol, increase vegetable and fruit consumption and limit expected increases in BMI to the extent required by the intervention scenarios modelled. More targeted strategies highlighted in this study include reducing salt and butter intake to reduce blood pressure and total cholesterol, respectively, as indicated in the intervention scenarios.

## Conclusion

The results reported here provide for the first time reliable estimates of the mortality burden of nutrition-related risk factors in New Zealand. We estimate that as many as 11,000 deaths in 1997 (40 percent of all deaths) may have been attributable to the joint effect of sub-optimal diet and physical activity levels. This includes over 85 percent of

ischaemic heart disease, 70 percent of stroke mortality, 80 percent of diabetes mortality and 6 percent of all cancer mortality.

Our results confirm that two well-established nutrition-related risk factors – cholesterol and blood pressure – are, along with tobacco smoking, the three major modifiable causes of premature death in New Zealand. The estimates of avoidable burden reported here also show that feasible changes in vegetable and fruit intake and in BMI could have a major impact on population health within a decade.

The clear implication is that a wide focus of policy attention, extending to vegetable and fruit intake and healthy weight maintenance, is needed. Multiple strategies, acting at multiple levels, will be required to achieve sustainable change. Such strategies will be developed as part of the Ministry of Health's *Healthy Eating – Healthy Action* initiative (Ministry of Health 2003), for which this report provides part of the evidence base.

# 1. Introduction

Whether because of affordability, availability, convenience, preference or other reasons, the foods we consume may not be consistent with the dietary pattern that is optimal for our health. We have known for a long time that nutrition-related risk and protective factors play important roles in ischaemic heart disease, stroke, diabetes and certain cancers. Yet the burden of sub-optimal nutrition on the health of the New Zealand population has never been previously quantified. The ‘comparative risk assessment’ methodology developed for the World Health Organization *World Health Report 2002* (WHO 2002) has provided an opportunity to do so.

## 1.1 Objective

The objective of this report is to estimate, for selected nutrition-related risk factors:

- attributable burden of disease in 1997
- avoidable burden of disease in 2011.

## 1.2 Motivation

This report is motivated by the *New Zealand Health Strategy*, which includes nutrition and obesity among its 13 priority objectives (Minister of Health 2000). Specifically, the report contributes to the evidence base of *Healthy Eating – Healthy Action* (Ministry of Health 2003), an action-planning tool developed by the Ministry of Health to implement the nutrition, physical activity and body weight-related objectives of the *New Zealand Health Strategy*.

This report employs comparative risk assessment (CRA) methodology developed by the WHO (Murray and Lopez 1999) and used in the *World Health Report 2002* (WHO 2002), with minor modifications. In the *World Health Report 2002* New Zealand is included in the Western Pacific ‘A’ region, along with Australia, Japan, Singapore and Brunei, and country-specific burdens are not calculated. This report provides country-specific estimates of the burden of disease due to nutrition-related risk factors for New Zealand.

## 1.3 Comparative risk assessment

CRA is a systematic approach to estimating the current burden of disease attributable to a range of biological, behavioural and environmental risk factors, as well as the future burden of disease that could be avoided if exposure to these risk factors were reduced.

Because a systematic approach is used, the attributable and avoidable burdens due to different risk factors are comparable. The methodology uses standardised methods at each of the following levels:

- obtaining the best estimates of risk factor distributions
- obtaining the best estimates for risk factor–disease relationships, including adjustment for confounding
- estimating parameter uncertainty.

The CRA approach differs from previous methodology in that it recognises that risk factor–disease relationships are continuous rather than categorical. Therefore, rather than categorising people into ‘exposed’ and ‘unexposed’ groups, the CRA methodology takes into account the entire population risk factor distribution.

## Selection of risk factors and diseases

The risk factors assessed in this report are:

- systolic blood pressure, as a marker for several nutritional factors, primarily salt intake
- total blood cholesterol, as a marker for saturated fat intake
- vegetable and fruit intake
- body mass index, as a marker of energy balance.

These risk factors were chosen because of their potential impact and modifiability, as well as the availability of complete data on their distributions and disease relationships.

The disease outcomes assessed are:

- ischaemic heart disease (IHD)
- stroke
- diabetes (type 2)
- cancers (selected types).

Selection of these diseases was based on the strength of the evidence for their causal relationship to the chosen risk factors, their impact on population health, and the availability of time series incidence and mortality data for them. Table 4 summarises the risk-factor–disease relationships explored in this report.

**Table 4:** Risk factor–disease combinations

Outcome	Risk factor			
	Systolic blood pressure	Total blood cholesterol	Vegetable and fruit	Body mass index
Ischaemic heart disease	✓	✓	✓	✓
Stroke	✓	✓ <sup>1</sup>	✓ <sup>1</sup>	✓ <sup>1</sup>
Diabetes				✓
Cancer			✓ <sup>2</sup>	✓ <sup>3</sup>

1 Ischaemic stroke only

2 Oesophageal, stomach, colorectal and lung cancers

3 Post-menopausal breast and colorectal cancers

Collectively, the four disease groups chosen account for over half the total mortality in New Zealand and a significant proportion of morbidity.

## **Measure of burden**

In this report the burden of disease is restricted to fatal outcomes only. The measure of burden is deaths and years of life lost (YLL).

Ideally, non-fatal outcomes would also be included, and would be summarised along with fatal outcomes using an integrated measure of population health such as the disability-adjusted life year (DALY). However, limitations in the epidemiological data available to estimate disease incidence (as opposed to mortality), and the lack of New Zealand-specific health state valuations, make calculation of non-fatal burdens and DALYs problematic. For this report, we prefer to rely on estimates of mortality alone, recognising that this provides an incomplete view of the burden of disease associated with nutrition-related risk factors.

## 2. Data and Methods

This chapter provides an outline of the generic CRA methodology and data sources used. Details specific to each selected risk factor (and disease) are provided in the relevant chapter.

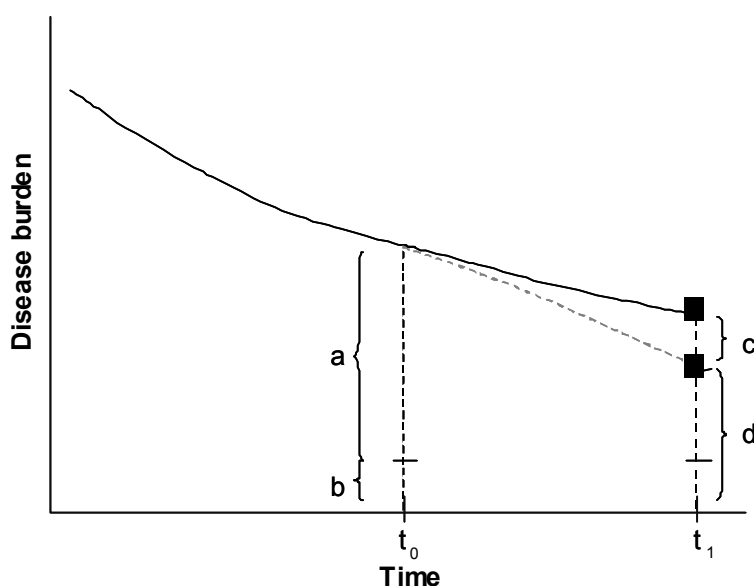
### 2.1 Definitions

*Attributable burden* is the difference between the burden currently observed and the burden that would have been observed under an alternative population distribution of exposure (Murray and Lopez 1999). The alternative (counterfactual) population distribution is that which confers the theoretical minimum population risk (not necessarily zero exposure).

*Avoidable burden* is the reduction in the future burden of disease that would occur if the current distribution of exposure to the risk factor were reduced to an alternative (counterfactual) distribution of exposure (Murray and Lopez 1999). In this report, the counterfactual distribution has been set to represent a specific intervention scenario.

The definition of attributable and avoidable burden may be illustrated using a simple diagram (Figure 1).

**Figure 1:** Attributable and avoidable burdens



a = disease at  $t_0$  attributable to prior exposure

b = disease at  $t_0$  not attributable to the risk factor (caused by other factors)

c = disease at  $t_1$  avoidable with a specified exposure reduction at  $t_0$  (ie, the avoidable burden)

d = disease at  $t_1$  after the specified reduction in risk exposure at  $t_0$

Attributable fraction at  $t_0$  due to prior exposure =  $a / a + b$

Avoidable fraction at  $t_1$  due to the specified reduction in exposure at  $t_0$  =  $c / c + d$

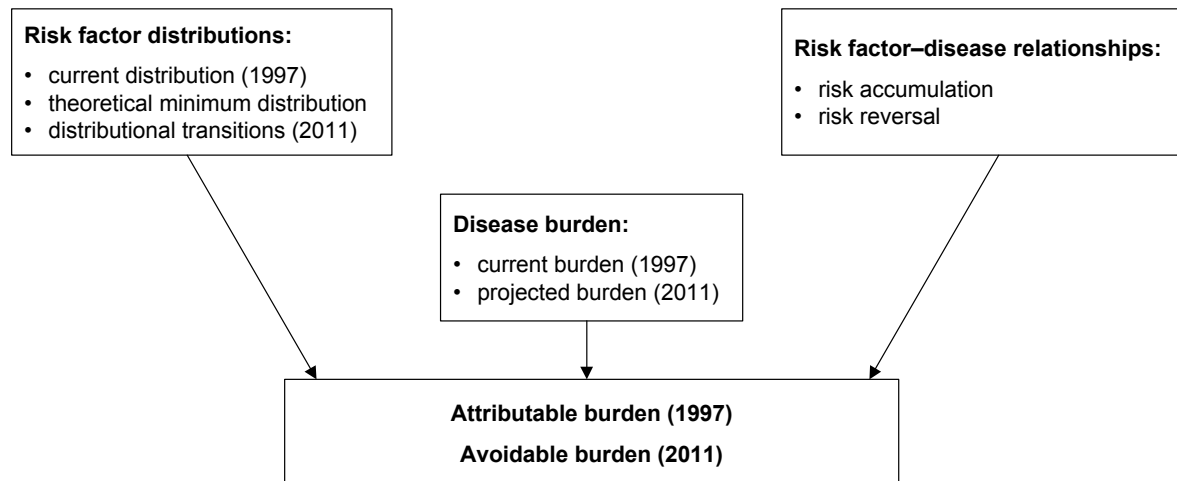
The solid line shows total disease burden based on a continuation of historical trends (ie, the 'business as usual' scenario). The dashed line shows total disease burden after specified reduction in exposure at  $t_0$  (ie, the 'intervention' scenario).

It is important to note that the avoidable burden is defined in a particular year ( $t_1$ ), not as cumulative burden avoided over a time interval (between  $t_0$  and  $t_1$ ).

Adapted from WHO 2002.

Both the attributable and avoidable burdens are calculated using potential impact fractions (PIFs). Potential impact fractions are a multi-level extension of attributable risk and take into account continuous risk factor–disease relationships. The estimation of potential impact fractions requires three main data inputs: information on risk factor distributions, risk factor–disease relationships, and disease burden. Figure 2 outlines the relationship between the three main data inputs.

**Figure 2:** Data inputs for comparative risk assessment



Adapted from WHO 2002.

## 2.2 Risk factor distributions

### Current distributions

Data on the population distribution of the selected risk factors, by 10-year age bands from age 25 years, gender and ethnicity (Māori/non-Māori), were extracted from the most recent available national or regional survey (see Appendix 1 for details of these surveys):

- 1997 National Nutrition Survey (Russell et al 1999) – systolic blood pressure, total blood cholesterol, vegetable and fruit intake, body mass index
- Auckland MONICA surveys (Trye et al 1996) – additional data for systolic blood pressure.

Polynomial models were used to smooth the raw survey data to reduce stochastic ‘noise’.

## Theoretical minimum distribution

To calculate the attributable fraction, the observed risk factor distribution must be compared to an alternative risk factor distribution conferring the lowest overall population risk of disease (ie, the theoretical minimum distribution) (Murray and Lopez 1999).

For some risk factors (eg, tobacco consumption) the theoretical minimum distribution is readily defined as zero exposure. However, this is not the case for the risk factors included in this report. Instead, for each risk factor, the theoretical minimum distributions were defined by expert working groups set up by WHO for the *World Health Report* based on one or more of the following.

- Meta-analyses of cohort studies (where possible), with the risk factor distribution associated with the lowest observed level of disease being selected as the theoretical minimum distribution, or by downward extrapolation of the (log linear) exposure–outcome curve to derive a plausible theoretical minimum distribution.
- Surveys of hunter-gatherer societies with little or no incidence of the disease of interest, with their observed risk factor distribution being taken as the theoretical minimum distribution.

In contrast to other risk factors, vegetable and fruit consumption is inversely associated with risk of disease. Therefore, for vegetables and fruit it was necessary to estimate the *maximum* consumption that would result in the theoretical minimum risk of disease.

## Distributional transitions

For a specific risk factor, distributional transitions are relative shifts away from the current distribution of the risk factor towards its theoretical minimum distribution (WHO Comparative Risk Assessment Working Group 2000). These distributional transitions are modelled to calculate the avoidable burden.

In this report, the avoidable burden is estimated from the difference between two distributional transitions: the ‘business as usual’ (BAU) scenario, representing a continuation in the historical trend for the risk factor; and the ‘intervention’ scenario, representing a change in trend reflecting implementation of new or intensified policies or programmes. For example, for systolic blood pressure in males, the avoidable burden was estimated by modelling an intervention shift equal to twice the anticipated BAU shift towards the theoretical minimum.

The distributional transitions for each risk factor were assumed to be the same in relative terms for all age and ethnic groups. However, the actual change in risk factor distributions varied by age, sex and ethnicity, depending on baseline risk factor levels.



## Business as usual scenarios

The business as usual (BAU) scenarios are intended to represent continuation over the next decade of historical trends in risk factor levels, taking age, period and cohort effects into account.

However, the time series available for the selected risk factors is short (typically data were available from the 1977 National Diet Survey, 1989 Life in New Zealand (LINZ) Survey and 1997 National Nutrition Survey (NNS) or just the two more recent surveys), so full age/period/cohort modelling could not be done. Instead, the distributional transitions under the BAU scenario were generally based on linear extrapolation over the surveys and analogy with the rates of change found over similar time periods in other economically developed societies.

## Intervention scenarios

For each risk factor, a distributional transition was modelled to represent the change in the risk factor distribution that could feasibly result, given:

- intensification or extension of coverage of existing policies
- implementation of novel interventions outlined in *Healthy Eating – Healthy Action* (Ministry of Health 2003).

The distributional transitions for the intervention scenarios were based on judgement, informed by:

- literature reviews on the effectiveness of existing interventions (in relation to intensity or coverage), as well as novel interventions not yet implemented in New Zealand
- examination of the risk factor distribution already achieved in other economically developed countries, together with current and projected rates of change in those countries.

The selected distributional transitions (intervention scenarios) are intended to be no more than illustrative of feasible changes in the population exposure distribution to each risk factor over the next decade and are conservative. Other scenarios could equally be run; indeed, a ‘sensitivity analysis’ of the burdens avoidable through intervention on risk factor exposures might be preferable to presenting the results of a single intervention scenario only. However, the approach adopted here is more relevant to policy formulation, provided that the calculated avoidable burdens are recognised as being illustrative only. In general the relationship between the distributional transition and the avoidable burden is approximately linear. So doubling the distributional transition will approximately double the avoidable burden.

## **2.3 Risk factor–disease relationships**

### **Risk accumulation**

Risk accumulation is the magnitude of, and lag in, the relationship between increasing (cumulative) exposure to a risk factor and resultant disease. Risk accumulation is expressed as the regression coefficient in the case of a continuous risk factor (the increase in incidence or mortality of the disease per unit increase in exposure, suitably lagged), or as the relative risk in the case of a categorical risk factor (the ratio of the disease incidence or mortality rate in the exposure category of interest to the rate in the lowest exposure or reference category).

Regression coefficients / relative risks may vary with gender and ethnicity, and generally will vary with age, often but not necessarily showing age attenuation. For this study age- and, in some cases, gender-specific estimates were used, but estimates were not differentiated by ethnicity. In many cases the assumption was also made that estimates were the same for non-fatal as for fatal outcomes (ie, risk coefficients or relative risks for incidence and mortality were the same). Where data were lacking for older age groups, a degree of age attenuation was modelled.

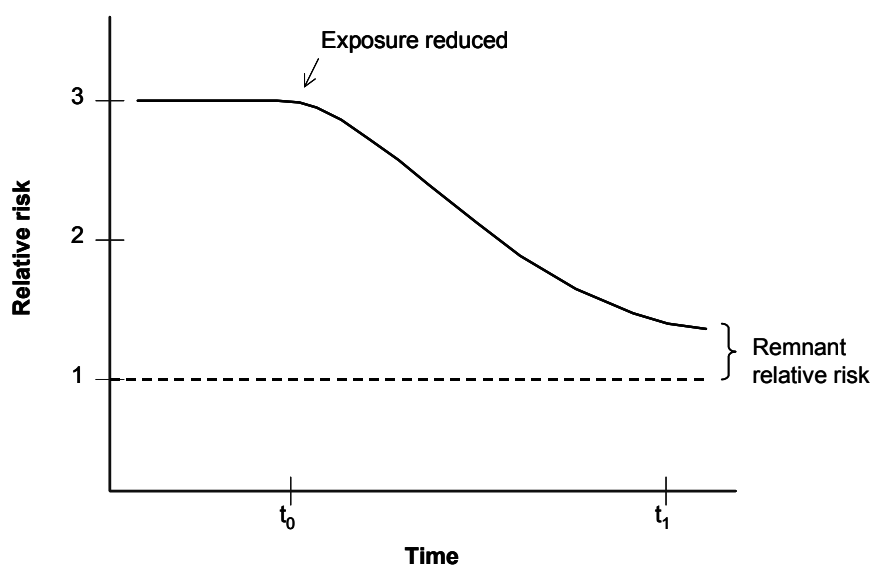
Risk coefficients and relative risks used in this report were estimated by the WHO expert working groups (WHO 2002) in three steps:

1. systematic literature reviews using strict inclusion and exclusion criteria to select the key studies (cohort studies where possible)
2. meta-analysis of the data from the cohort studies (such overviews provide more reliable estimates than any single study and are less subject to regression dilution bias)
3. standardised adjustment for confounding. Often adjustment had little impact on the estimate, but in some cases partial adjustment for covariates that are simultaneously independent risk factors and also mediating or pathway variables was required. Partial adjustment is necessary so that the full effect of the risk factor of interest is captured, yet is not overestimated.

### **Risk reversal**

Risk reversal is the magnitude and timing of the reduction in risk when the mean of the exposure distribution is reduced (Figure 3). This information is needed to calculate the avoidable burden that might result from the risk factor distributional transitions used in the BAU and intervention scenarios.

**Figure 3:** Illustration of risk reversal



Risk-reversal estimates used in this report were derived by the WHO expert working groups (WHO 2002) through the following:

- systematic literature reviews using strict inclusion and exclusion criteria to select the key clinical trials
- meta-analysis of the trial data
- adjustment for confounding, as done for risk accumulation.

Both risk accumulation and risk-reversal estimates had to be differentiated by 10-year age group and gender. Where estimates were available only for broader or different age groups from those used in this report, smoothing techniques were used to interpolate the values.

## 2.4 Disease burdens

### Current burden

The number of deaths by 10-year age group (from age 25), sex, and ethnicity (Māori/non-Māori) for each selected disease (ICD-9-CM code) for 1996–98 (to coincide with the risk factor data) were extracted from the New Zealand Health Information Service (NZHIS) mortality database. Average rates for 1997 were calculated using 1997 mid-year population estimates provided by Statistics New Zealand.

Simple extraction from the NZHIS mortality database could not be used for diabetes, since many deaths actually caused by diabetes are not coded to ICD 250 but rather to cardiovascular or renal codes, reflecting the immediate cause of death. Instead, a multi-state life table model (Ministry of Health 2002d) was used to estimate the number and distribution of deaths attributable to diabetes in 1997 irrespective of ICD code. The number of deaths attributable to diabetes using this latter method was approximately 120 percent greater than the number coded to ICD 250 overall.

## Projected burden

Generalised linear models (so-called age/period/cohort or APC models) were used to forecast mortality through to the year 2011 for each disease (Clayton and Schifflers 1987). The APC models were fitted to the mortality data for each disease (other than diabetes) from 1960 to 1999 by five-year age groups and five-year periods (quinary quinquennia) to give 10-year overlapping cohorts.

The goodness of fit of the models was tested using statistical tests and ex-post tests (empirical projections in which data from earlier periods only are used to fit the models and projections are obtained for the recent periods and compared with the observed data). In general, the models performed well for IHD and stroke, but tended to perform less well for the cancers.

For cancers, the APC models were supplemented with BAMP models (the Bayesian equivalent of the generalised linear models) and non-parametric generalised additive models (GAM). A model averaging process was then used to derive the ‘best’ projections from all models, subject to goodness of fit and empirical projection tests. Full details of the cancer forecasting methodology are provided in a separate publication (Ministry of Health 2002a).

APC models could not be used for diabetes because of the absence of a time series. Instead, a multi-state life table model developed by the Ministry of Health was used to forecast diabetes mortality in 2011 under a scenario of reduced case fatality (reflecting improvements in diabetes care) and increased incidence (reflecting continued growth of the obesity epidemic) (Ministry of Health 2002c).

The projections for stroke were based on APC modelling, but adjusted for undercount using a multi-state life table stroke model (Ministry of Health 2002d).

Because a robust time series for ethnic-specific mortality was absent (the result of changing concepts and classifications of ethnicity over the 30-year observation period), the models could not be made ethnic-specific; instead projections were made for the total population (by gender and five-year age group) only. The projections were then split into Māori and non-Māori estimates assuming that the ratio of ethnic-specific mortality rates in 1996–98 will be stable to the forecasting horizon.

Population projections, required to estimate mortality rates through to 2011, were obtained from Statistics New Zealand. Series 4 projections, assuming medium mortality and fertility and 5000 net migration gain per year from 2001, were used throughout.

## 2.5 Calculation of attributable and avoidable burdens

Attributable burdens were calculated by applying the relevant potential impact fraction (PIF) to the estimate of current burden. The PIFs were calculated from the estimates of the current risk factor distribution and risk accumulation (risk coefficients) using the formula (WHO 2002):

$$PIF = \frac{\int_{x=0}^m RR(x)P(x) - \int_{x=0}^m RR(x)P'(x)}{\int_{x=0}^m RR(x)P(x)}$$

where:

- $RR(x)$  = relative risk at exposure level  $x$
- $P(x)$  = population distribution of exposure
- $P'(x)$  = alternative or counterfactual distribution of exposure
- $m$  = maximum exposure level.

Further details of the methodology used to estimate attributable burden, with examples, can be found in Vander Hoorn et al (in press).

Avoidable burdens were calculated by extending the methodology of the PIF formula described above to include estimates of risk reversibility in the calculations.

Uncertainty was estimated by a simulation procedure incorporating sources of uncertainty relating to the risk factor distribution, the risk factor–disease relationship, and the projected total disease burden (WHO 2002). These input parameters were simultaneously varied within their respective distributions and the calculation of the PIF was reiterated for a total of 500 iterations. From this, 95 percent confidence intervals were derived. This method captures uncertainty in the parameters, but, ideally, data on exposure among those with disease should be used as well (such data were not available). Also, the method fails to adjust for competing risks, thereby overestimating the absolute attributable and avoidable burdens, although the degree of overestimation is probably slight.

## 2.6 Calculation of years of life lost

Mortality counts were translated into YLL counts using the ‘remaining life expectancy’ method, with Coale and Demeny model life table West level 26 as the standard for both genders (Murray and Lopez 1996). As recommended by WHO (Murray and Lopez 1996), YLL counts were discounted to the present using a 3 percent per annum discount rate.

Mortality and YLL rates were age standardised by the direct method for summarisation, using the WHO World population (WHO 2000a) as the standard population.

### **3. Current and Projected Disease Burdens**

This chapter summarises the mortality estimates (counts and years of life lost) for the selected diseases, both in 1997 and (projected) 2011. Full age-, sex- and ethnic-specific results are provided in Appendix 2.

These estimates of total burden constitute an essential data input necessary for the calculation of both attributable burdens in 1997 and avoidable burdens in 2011.

#### **3.1 Ischaemic heart disease**

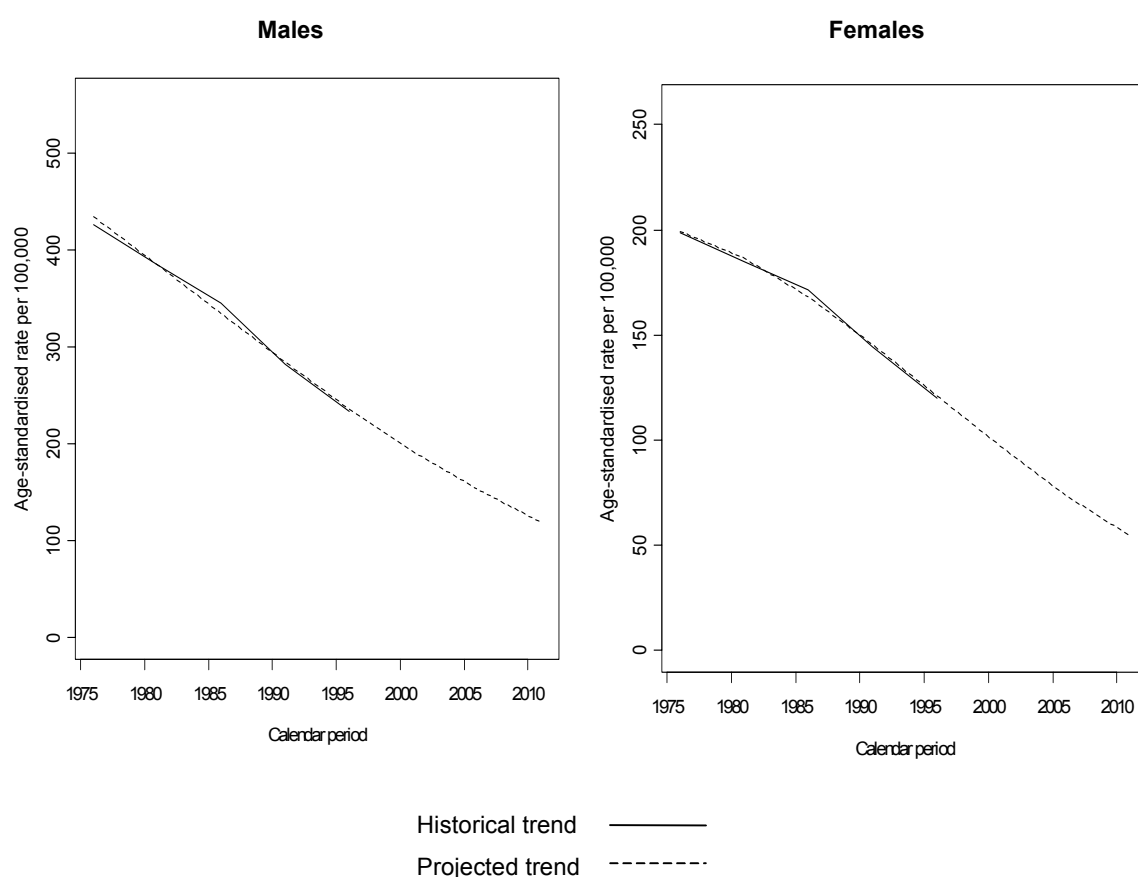
Total blood cholesterol (or the total cholesterol to HDL cholesterol ratio), blood pressure (systolic and diastolic), body fat mass (especially abdominal fat mass), and vegetable and fruit intake are all established risk factors for ischaemic heart disease (IHD), along with family history, tobacco consumption, physical activity and diabetes.

IHD remains by far the leading cause of death in New Zealand for both genders and all ethnic groups, accounting for almost one-quarter of all deaths in the late 1990s. Once adjusted for age, IHD mortality rates are higher in males than in females, and in Māori than in non-Māori.

IHD mortality rates have been declining steadily since the early 1970s, reflecting both changes in risk exposure (reduced incidence) and improvements in medical care (reduced in-hospital case fatality). The APC model projects a further rapid decline, assuming a continuation of these historical trends. However, this projection may be overly optimistic in view of the recent increases in obesity and (consequently) diabetes prevalence, which may not be fully captured in the period and cohort effects. The model is therefore conservative in that it may underestimate the avoidable burdens.

The age-standardised historical and projected IHD trends are illustrated in Figure 4. IHD mortality data for 1997 and 2011, by sex and ethnic group, are summarised in Tables 5 and 6.

**Figure 4:** Trends and projections of ischaemic heart disease mortality rates, by sex, 1976–2011



**Table 5:** Ischaemic heart disease mortality, by ethnic group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	3,303	30,081	278.6	2,652
	Māori	303	4,237	451.5	5,256
Females	Non-Māori	2,614	22,553	142.4	1,340
	Māori	180	2,315	257.1	2,951

\* Rate per 100,000, age-standardised to WHO World population

**Table 6:** Ischaemic heart disease mortality, by ethnic group and sex, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	2,424	20,885	147.3	1,338
	Māori	269	3,332	231.4	2,538
Females	Non-Māori	1,552	13,177	67.0	607
	Māori	149	1,670	115.1	1,228

\* Rate per 100,000, age-standardised to WHO World population

## 3.2 Stroke

Stroke includes ischaemic and haemorrhagic subtypes. Ischaemic stroke accounts for approximately 80 percent of all stroke incidence in New Zealand, but a lesser proportion of all stroke mortality. Risk factors for ischaemic stroke are similar to those for IHD, whereas risk of haemorrhagic stroke is dominated by blood pressure.

Due to limitations in the available data, only total stroke numbers were available for 1997 and (projected) 2011. With the exception of systolic blood pressure, risk coefficients were estimated for ischaemic stroke. Where this was the case, the burden of disease for ischaemic stroke was obtained by applying the ratio of ischaemic to haemorrhagic stroke in Australia and New Zealand to the total stroke numbers (Lawes et al, in press).

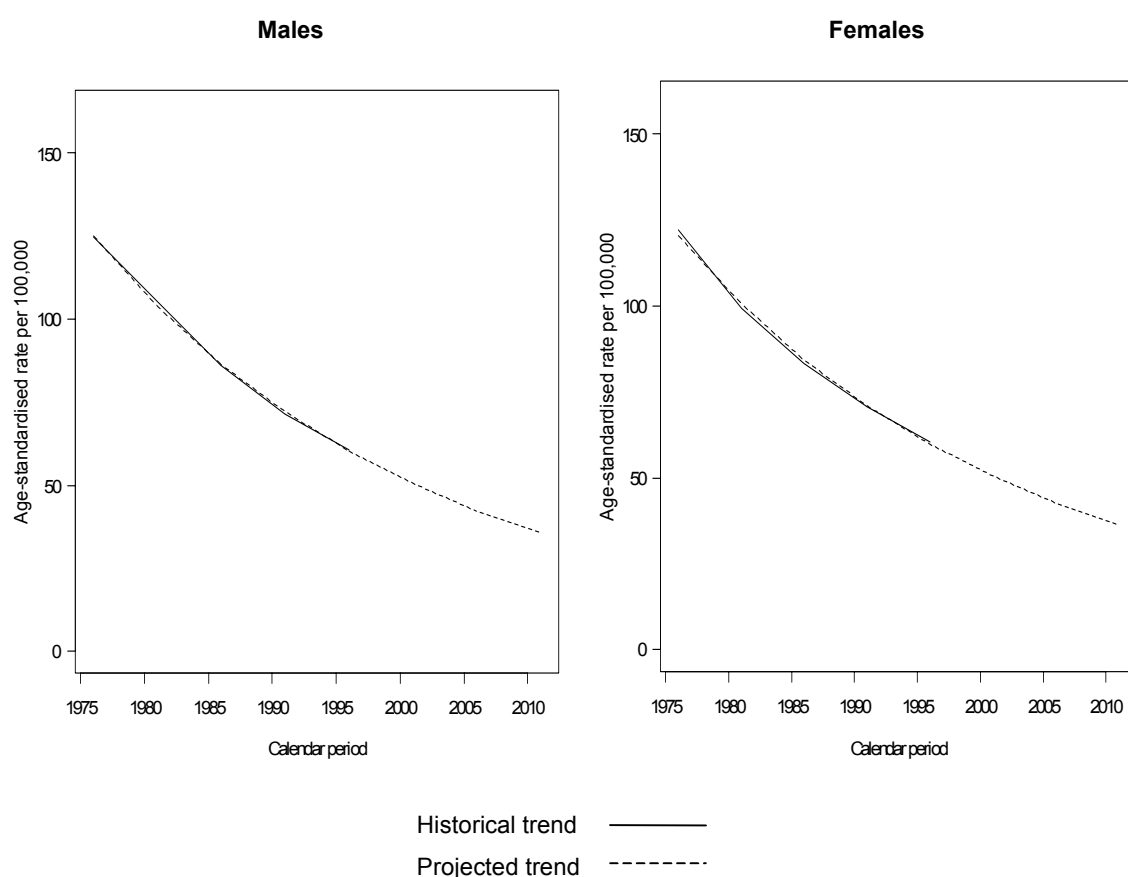
The clinical diagnosis of stroke is not always accurate. Accordingly, the Ministry of Health has built a multi-state life table model of stroke, based on data from a regional stroke register rather than routine hospital and mortality data. This model is considered to provide more accurate estimates of stroke mortality than routine death certificates (Ministry of Health 2002d). According to this model, stroke currently (1997) accounts for approximately 12 percent of total mortality (about half that of IHD). Almost 60 percent of deaths occur in females and 80 percent in older people (> 65 years). Māori rates exceed non-Māori rates (adjusted for age), although the ethnic differential is less marked than for IHD.

Stroke mortality has been declining in New Zealand for even longer than has IHD mortality. This trend may have occurred partly as a result of incidence reduction (mainly through blood pressure control), but more especially as a result of lower case fatality (reflecting reduced severity, slower progression and better treatment) (Bonita 1993). A continued decline in stroke mortality rates has been projected to 2011, albeit less rapidly than before.

The age-standardised historical and projected total stroke trends are illustrated in Figure 5. Total stroke data for 1997 and 2011, by sex and ethnic group, are summarised in Tables 7 and 8.



**Figure 5:** Trends in total stroke mortality rates in New Zealand, by sex, 1975–2011



**Table 7:** Stroke mortality, by ethnic group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	897	6,592	73.7	555
	Māori	44	501	82.9	727
Females	Non-Māori	1,425	11,693	74.3	652
	Māori	57	711	84.0	929

\* Rate per 100,000, age-standardised to WHO World population

**Table 8:** Stroke mortality, by ethnic group and sex, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	773	5,391	45.3	334
	Māori	52	517	50.8	442
Females	Non-Māori	1,120	8,950	46.1	389
	Māori	63	729	48.5	512

\* Rate per 100,000, age-standardised to WHO World population

### 3.4 Diabetes

Type 2 diabetes mellitus is a syndrome resulting from insulin resistance that leads, in genetically susceptible individuals, to a relative deficit in insulin secretion. By far the major cause of insulin resistance is increased body fat mass, especially in the visceral (abdominal) compartment. Physical inactivity, as well as contributing to increased body fat, is also an independent risk factor for insulin resistance.

At least 85 percent of diabetes in New Zealand is currently type 2 (Ministry of Health 1999b). Historically, type 2 diabetes was uncommon before middle age, but is now being diagnosed with increasing frequency in young adults and even adolescents, especially among Māori and Pacific ethnic groups (Moore and Lunt 2000).

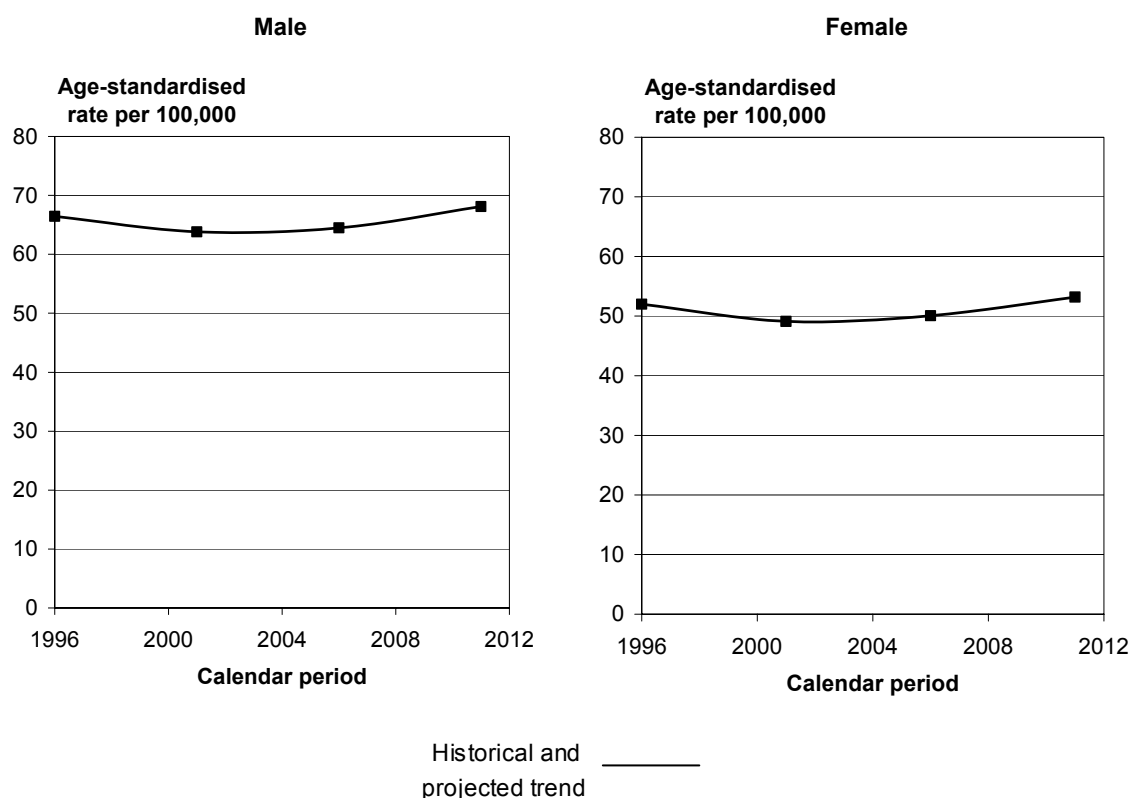
Mortality from diabetes will be seriously underestimated if only deaths coded to ICD 250 are included, since many deaths in diabetics are incorrectly coded to IHD or stroke without recognition of diabetes as the underlying cause of death. Accordingly, the Ministry of Health has built a multi-state life table model of diabetes (Ministry of Health 2002c), which provides a more accurate assessment of mortality due to diabetes.

According to this model, diagnosed type 2 diabetes accounted for over 5 percent of all deaths in 1996, of which only 42 percent had been coded to ICD 250. For Māori, the proportion of all deaths attributable to diabetes was almost 20 percent, while for Pacific people it was 16 percent. Pooling genders, Māori and Pacific people were approximately ten and seven times, respectively, more likely to die from diabetes than their European counterparts, after adjusting for age. Overall, slightly more than half of all diabetes-attributable deaths occurred in males and almost 60 percent in older people (> 65 years).

The model was used to project diabetes-attributable mortality to 2011 under varying scenarios. The 'most likely' future scenario projects an 80 percent increase in diabetes incidence rates and a 25 percent decrease in case fatality rates across all ages, genders and ethnic groups. Under this scenario, the mortality rate does not change appreciably, as the increase in diabetes incidence over the forecasting period is offset by an anticipated decline in case fatality. This anticipated decline in case-fatality is a reflection of both improved diabetes care and better cardiac and stroke care. However, the number of diabetes deaths increases about 40 percent, reflecting demographic trends.

The age-standardised historical and projected diabetes rates are illustrated in Figure 6. The rate remains almost constant over the projection period as decreasing case-fatality counterbalances increasing incidence. Diabetes mortality data for 1997 and 2011, by sex and ethnic group, are summarised in Tables 9 and 10.

**Figure 6:** Trends in diabetes mortality rates in New Zealand, by sex, 1996–2011



**Table 9:** Diabetes mortality, by ethnic group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	554	6,385	48.3	583
	Māori	238	3,501	331.3	4,324
Females	Non-Māori	428	5,243	31.4	422
	Māori	274	4,252	348.1	4,975

\* Rate per 100,000, age-standardised to WHO World population

**Table 10:** Diabetes mortality, by ethnic group and sex, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	721	8,106	46.4	551
	Māori	403	5,529	326.7	4,094
Females	Non-Māori	526	6,519	29.7	397
	Māori	454	6,736	316.1	4,433

\* Rate per 100,000, age-standardised to WHO World population

### 3.3 Cancer

Several of the selected risk factors are causally related to major cancers: BMI with post-menopausal breast and colorectal cancer, and vegetable and fruit intake with smoking-related cancers (especially cancer of the lung in smokers) and cancers of the gastro-intestinal tract (including oesophageal, stomach and colorectal cancers). The selection of cancers included in this study is based on those selected by the WHO expert working groups for the *World Health Report 2002* (Ezzati et al 2002).

Breast cancer is currently the leading cause of cancer death in females, accounting for almost 20 percent of all cancer deaths in this gender. Over 80 percent of breast cancer deaths are post-menopausal. Māori breast cancer rates are slightly higher than non-Māori rates, after adjusting for age. In this report we examine the relationship between BMI and post-menopausal breast cancer.

Colorectal cancer is the second leading cause of cancer deaths in both genders at present, accounting for approximately 15 percent of all cancer deaths in each. Mortality from colorectal cancer increases steeply with age, from about age 40 years. Colorectal cancer mortality rates are lower in Māori than in non-Māori (adjusted for age). This difference in mortality rates could be explained by undercounting of Māori cancer mortality (Ajwani et al 2003).

Lung cancer is the leading cause of cancer death among males (20 percent of all cancer deaths in 1997), and is already the third-ranked cause among females (accounting for over 14 percent of all female cancer deaths in 1997). Lung cancer mortality rates increase sharply with age, and are higher among Māori than non-Māori (adjusted for age). Cigarette smoking is causally associated with at least 80 percent of lung cancer incidence in New Zealand at present.

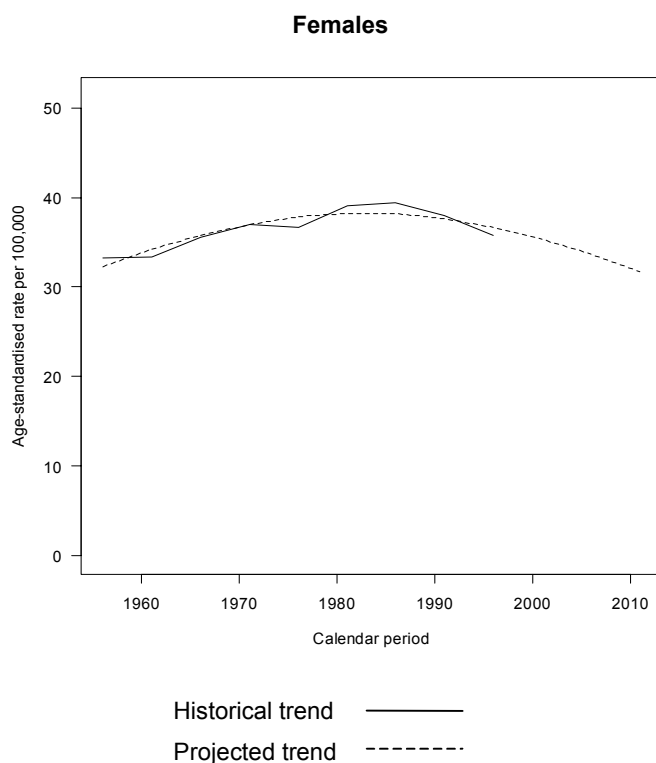
Vegetable and fruit intake confers a protective effect against lung cancer, as well as other less common tobacco-associated cancers. It also protects against colorectal cancer and other gastro-intestinal cancers, of which oesophageal (2 percent of total cancer mortality in 1997) and stomach (4 percent of total cancer mortality in 1997) cancers are sufficiently common to warrant inclusion in this report.

Estimates of current (1997) cancer mortality (from the New Zealand Cancer Registry) and projections to 2011 (based on averaging across APC, BAMP and GAM models as outlined in chapter 2) (Ministry of Health 2002a) are summarised in Tables 11 to 20. Figures 7 to 11 show historical and projected age-standardised cancer mortality rates. In brief, breast cancer mortality is projected to decline slowly, colorectal cancer to decline more sharply (both genders), lung cancer mortality to decline rapidly among males but increase among females, oesophageal cancer mortality to stabilise among males but decline slowly among females, and stomach cancer mortality to continue its historical downward trend among both genders, but less rapidly than in the past.

## Post-menopausal breast cancer

Figure 7 shows observed trends in the age-standardised breast cancer mortality rate in New Zealand women from 1960 to 1997, and projected to 2011.

**Figure 7:** Trends in breast cancer mortality rates in New Zealand, 1960–2011



The following tables show current (1997) and future (2011) levels of breast cancer mortality in post-menopausal women.

**Table 11:** Post-menopausal breast cancer mortality, by ethnic group, 1997

Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Non-Māori	487	6,023	93.6	1,300
Māori	35	523	116.9	1,648

\* Rate per 100,000, age-standardised to WHO World population

**Table 12:** Post-menopausal breast cancer mortality, by ethnic group, 2011

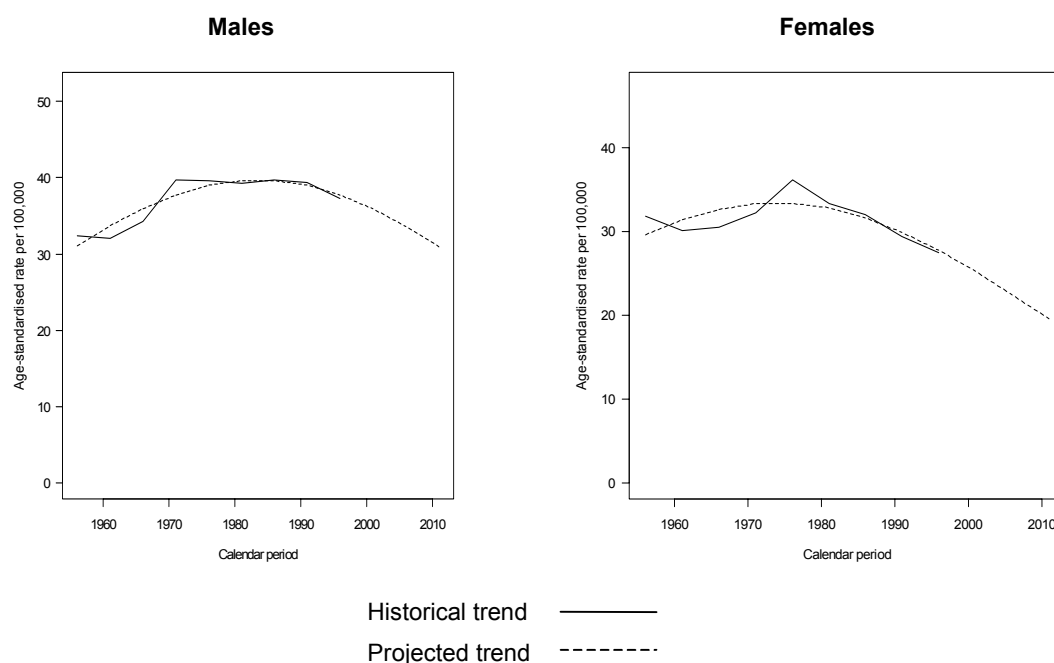
Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Non-Māori	586	7,338	81.2	1,105
Māori	60	881	101.4	1,400

\* Rate per 100,000, age-standardised to WHO World population

## Colorectal cancer

Figure 8 shows observed trends in the age-standardised colorectal cancer mortality rate in New Zealand from 1960 to 1997, and projected to 2011.

**Figure 8:** Trends in colorectal cancer mortality rates in New Zealand, by sex, 1960–2011



The following tables (Tables 13 and 14) show current (1997) and projected (2011) colorectal cancer mortality, by sex and ethnic group.

**Table 13:** Colorectal cancer mortality, by ethnic group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	553	5,952	47.7	535
	Māori	23	307	34.7	393
Females	Non-Māori	520	5,879	35.1	448
	Māori	18	246	22.5	309

\* Rate per 100,000, age-standardised to WHO World population

**Table 14:** Colorectal cancer mortality, by ethnic group and sex, 2011

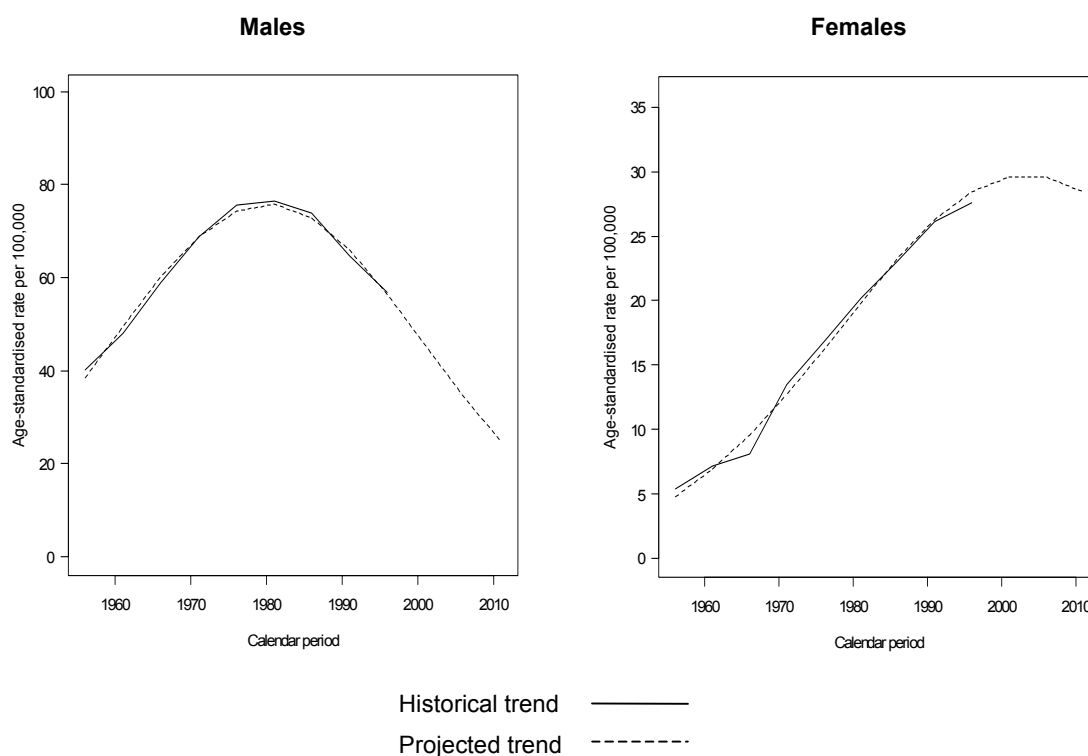
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	641	5,793	38.4	370
	Māori	31	364	27.3	269
Females	Non-Māori	518	5,373	25.0	286
	Māori	22	250	14.9	183

\* Rate per 100,000, age-standardised to WHO World population

## Lung cancer

Figure 9 shows observed trends in lung cancer mortality rates in New Zealand from 1960 to 1997, and projected lung cancer mortality rates to 2011.

**Figure 9:** Trends in lung cancer mortality rates in New Zealand, by sex, 1960–2011



The following tables (Tables 15 and 16) show both current (1997) and projected (2011) lung cancer mortality, by sex and ethnic group.

**Table 15:** Lung cancer mortality, by ethnic group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	767	7,745	65.4	686
	Māori	113	1,509	168.3	1,979
Females	Non-Māori	421	5,140	30.2	406
	Māori	98	1,486	123.4	1,691

\* Rate per 100,000, age-standardised to WHO World population

**Table 16:** Lung cancer mortality, by ethnic group, age group and sex, 2011

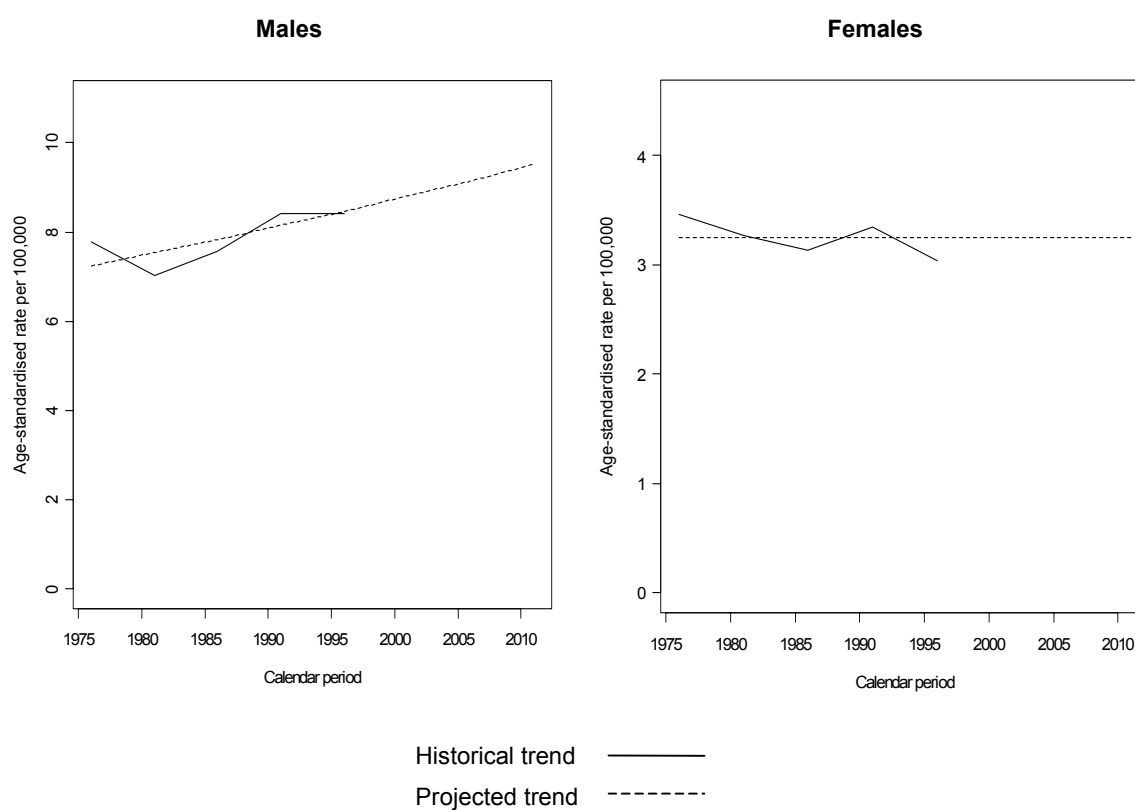
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	686	6,244	41.0	391
	Māori	121	1,464	99.9	1,083
Females	Non-Māori	654	7,720	34.3	444
	Māori	209	2,930	139.7	1,844

\* Rate per 100,000, age-standardised to WHO World population

## Oesophageal cancer

Figure 10 shows observed trends in the oesophageal cancer age-standardised mortality rate in New Zealand from 1975 to 1997, and projected to 2011.

**Figure 10:** Trends in oesophageal cancer mortality rates in New Zealand, by sex, 1975–2011





The following tables (Tables 17 and 18) show current (1997) and projected (2011) oesophageal cancer mortality by sex and ethnic group.

**Table 17:** Oesophageal cancer mortality, by ethnic group, age group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	116	1,234	10.0	111
	Māori	8	113	10.6	134
Females	Non-Māori	61	595	3.6	39
	Māori	4	53	5.2	68

\* Rate per 100,000, age-standardised to WHO World population

**Table 18:** Oesophageal cancer mortality, by ethnic group, age group and sex, 2011

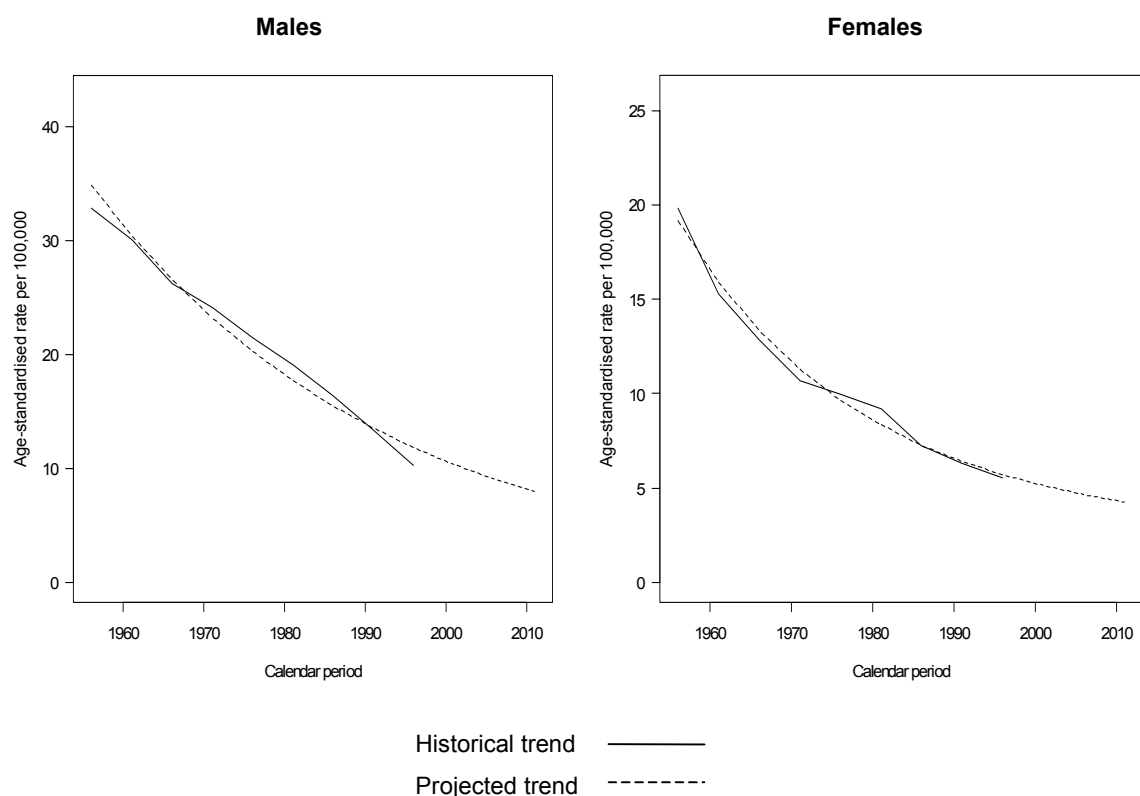
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	170	1,728	10.4	111
	Māori	14	200	10.9	138
Females	Non-Māori	74	744	3.4	37
	Māori	7	81	4.8	62

\* Rate per 100,000, age-standardised to WHO World population

## Stomach cancer

Figure 11 shows observed trends in the age-standardised stomach cancer mortality rate in New Zealand from 1960 to 1997, and projected to 2011.

**Figure 11:** Trends in stomach cancer mortality rates in New Zealand, by sex, 1960–2011



The following tables show both current (1997) and projected (2011) stomach cancer mortality, by sex and ethnic group.

**Table 19:** Stomach cancer mortality, by ethnic group and sex, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	144	1,419	12.2	125
	Māori	27	421	36.6	467
Females	Non-Māori	100	1,055	6.4	78
	Māori	17	245	23.0	285

\* Rate per 100,000, age-standardised to WHO World population

**Table 20:** Stomach cancer mortality, by ethnic group, age group and sex, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	129	1,309	7.9	84
	Māori	33	452	24.4	321
Females	Non-Māori	85	928	4.2	51
	Māori	22	294	15.2	192

\* Rate per 100,000, age-standardised to WHO World population

### 3.5 Summary

Collectively, the diseases selected for this report include over 50 percent of all deaths in 1997 (genders pooled), although a slightly smaller proportion of years of life lost. The proportion in 2011 is projected to be lower, but still very substantial.

By combining the above estimates of current (1997) and projected (2011) total burden for the selected diseases with the estimates of risk factor distributions and risk factor–disease relationships, attributable and avoidable fractions and burdens can be calculated. As uncertainty estimates were also derived for all the input data, uncertainty in the estimates of attributable and avoidable fractions and burdens can also be quantified.

## 4. Nutrition, Blood Pressure and the Burden of Disease

### 4.1 Introduction

High blood pressure (hypertension) is an important risk factor for cardiovascular disease, particularly stroke. Traditionally, hypertension has been defined as systolic blood pressure  $\geq 140$  mmHg, or diastolic blood pressure  $\geq 90$  mmHg, or treatment with anti-hypertensive medication. However, the association between blood pressure and cardiovascular disease is continuous with no lower threshold, and many cardiovascular events occur in individuals with blood pressure levels below the cut-off for hypertension (MacMahon et al 1990; Prospective Studies Collaboration 1995, 2002; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration 1999). Therefore, many individuals with blood pressure levels in the normal range (normotensive) are at increased risk of cardiovascular disease and would benefit from blood pressure lowering.

The focus of this chapter will be on systolic blood pressure (SBP) as the majority of analyses have demonstrated a stronger association between this variable and cardiovascular disease than for diastolic blood pressure. Other measures such as mean arterial pressure and pulse pressure have not been shown to be conclusively superior to SBP (Mitchell et al 1997; Sesso et al 2000; Franklin et al 2001; Miura et al 2001; Asia Pacific Cohort Studies Collaboration 2003a).

### 4.2 Determinants of blood pressure

Blood pressure is influenced by a combination of genetic and environmental factors. In Western societies, blood pressure increases steadily with age. However, the age-related increase in blood pressure is largely due to environmental factors, as blood pressure does not increase with age in economically undeveloped countries or hunter-gatherer societies (Law et al 1991c). As discussed in this chapter, modifiable determinants of blood pressure include diet, body weight or BMI, physical activity and alcohol intake. The focus of this chapter will be on dietary factors, particularly sodium intake.

#### Sodium

##### *Sodium and systolic blood pressure*

Dietary sodium (principally from salt) is an important determinant of blood pressure. A vast number of observational studies and randomised controlled feeding trials have investigated the relationship between sodium and blood pressure. The results of these studies have been somewhat variable, and as a result the association between salt and blood pressure remains controversial. However, much of this controversy arises from flaws in study design, analysis or interpretation. Many studies were underpowered and so were unable to detect small differences in blood pressure, or dismissed them as unimportant. Many studies failed to correct for regression dilution bias, a type of measurement error associated with single measures of exposure. Because salt intake and blood pressure are variable within individuals from day to day, failure to correct for

within-person variation reduces the apparent extent of between-person variation, and leads to underestimation of the strength of the association between salt and blood pressure. Many studies did not consider the impact of BMI, which is correlated with salt intake and independently associated with blood pressure. However, although BMI confounds the relationship between sodium intake and blood pressure and should be controlled for, it is measured with much more accuracy than salt intake and therefore controlling for BMI by conventional methods leads to over-adjustment. The true association between sodium intake and blood pressure is likely to be somewhere between the BMI-adjusted and BMI-unadjusted estimates.

Of studies that avoided the problems described, the reanalysis of the Intersalt study data is the most important (Elliott et al 1996). The Intersalt study was a large observational study involving 10,074 adults aged 20–59 years from 52 population samples in 32 different countries. After correction for regression dilution bias and adjustment for age, sex, urinary potassium and alcohol intake, summary estimates suggest that a 100 mmol/day lower sodium intake is associated with a 3.1/6.0 mmHg lower systolic blood pressure (SBP) (with/without adjustment for BMI).

These estimates agree closely with that obtained from a meta-analysis of observational studies in 24 communities (47,000 people), which found that a 100 mmol/day difference in sodium intake was associated with a 6 mmHg difference in SBP among populations with a mean age of 40 years (Law et al 1991a).

Feeding trials provide more direct evidence than observational studies of the effects of sodium on blood pressure. Law et al (1991b) analysed 78 sodium restriction trials and found that in the 45 trials of short duration (under four weeks), observed reductions in blood pressure were less than those predicted based on observational study data, suggesting longer time was needed to attain full benefit. In the 33 trials lasting five weeks or longer, observed reductions in blood pressure were similar to those predicted based on observational study data.

Several other meta-analyses of sodium restriction trials have been undertaken (Midgley et al 1996; Cutler et al 1997; Graudal et al 1998; Ebrahim and Davey Smith 1998; Alam and Johnson 1999; He and MacGregor 2002; Hooper et al 2002). All meta-analyses found that decreases in 24-hour sodium excretion were associated with decreases in SBP, although the magnitude of the decrease varied, depending on the duration of the trials, the level of sodium restriction, and baseline blood pressure levels.

The most important recent trial of sodium restriction was the follow-up Dietary Approaches to Stop Hypertension (DASH) trial (Sacks et al 2001). The DASH trial was a multi-centre randomised controlled trial comparing the effects of two dietary patterns and three levels of sodium intake on blood pressure in people with normal and high normal blood pressure (ie, above optimal level but below threshold for treatment). Participants (n = 412) were randomised to either the control diet (typical of what many Americans eat) or the DASH diet (rich in vegetables, fruit, low-fat dairy products, whole grains, poultry, fish and nuts, and low in red meat, sweets and sugar-containing beverages). Within the assigned diet, participants ate food with sodium levels that were high (typical of current consumption), intermediate (upper limit of current recommendations) and low (potentially optimal levels) for 30 consecutive days, in random order. Reducing sodium intake was

found to lower SBP, with the magnitude of blood pressure lowering depending on baseline blood pressure, baseline sodium intake and type of diet. Of most relevance for this analysis is the effect size for participants on the control diet with sodium intake reduced from high to intermediate level, as this most closely reflects changes feasible in the general population. For these participants (mean age 47 years), body weight remained constant while sodium intake decreased from 141 to 106 mmol/day. This 35 mmol/day decrease in sodium intake was associated with a fall in SBP of 2.1 mmHg, which is equivalent to a 6.0 mmHg decrease in SBP for a 100 mmol/L decrease in sodium intake.

Another important sodium restriction study was the Trials of Hypertension Prevention (1987–1990) study (Cook et al 1998). Participants (mean age 43 years) with high normal diastolic blood pressure were randomised to the sodium restriction (n = 327) or control group (n = 417) and followed for 18 months. After correction for regression dilution bias, the decrease in SBP for a 100 mmol decrease in sodium excretion was 3.5/4.4 mmHg (with/without adjustment for BMI).

Taken together, these data suggest that a decrease in dietary sodium of 100 mmol/day could lower SBP by up to 6 mmHg, depending on age and baseline blood pressure.

## **Sodium in the diet**

Sodium chloride (salt) is the principal source of sodium in the diet. Although sodium is an essential nutrient, intakes of sodium in developed countries greatly exceed those required to meet daily requirements (10–20 mmol/day). Sodium intake from food is difficult to measure given the amount of salt added to foods during cooking and at the table varies between individuals and is difficult to quantify. Sodium intake is more accurately measured by 24-hour urinary sodium excretion, with approximately 90 percent of ingested sodium being excreted in the urine. However, collection of 24-hour urine samples is generally not done as part of national nutrition surveys and therefore data from smaller surveys have been used to estimate sodium intake in New Zealand.

The largest surveys of urinary sodium excretion in New Zealand were conducted in Milton in 1975 and 1981. The 1975 survey, which involved 1209 subjects (95.7 percent European), found the mean 24-hour sodium excretion was 173 mmol for males and 140 mmol for females (Simpson et al 1978). The 1981 survey (1058 subjects) found similar results: 172 mmol for males and 134 mmol for females (Simpson et al 1982).

More recently, 24-hour urinary samples from 724 participants in studies conducted from 1993 to 1998 in Dunedin, Waikato and Taranaki were analysed for sodium. Mean 24-hour sodium excretion was 167 mmol in males and 122 mmol in females (Thomson and Colls 1998). The slightly lower sodium excretion found in this study compared to the Milton studies may be due to a real decrease in sodium intake over time, or differences in the study populations (Milton participants were predominately rural, whereas participants in the more recent studies were predominately blood donors and probably more health conscious than the general population).

As shown in Table 21, sodium intakes in New Zealand are similar to intakes in Australia and Britain. The higher sodium intakes in males than females is a common finding, as

sodium intake is highly correlated with energy intake, and males usually have higher energy requirements due to their larger body size.

**Table 21:** International comparisons of mean 24-hour urinary sodium excretion and salt intake

Country, year	24-hour sodium excretion (mmol)		Equivalent salt intake (g/day)	
	Males	Females	Males	Females
Milton, New Zealand, 1975	173	140	10.1	8.2
Milton, New Zealand, 1981	172	134	10.1	7.8
New Zealand, 1993-98	167	122	9.8	7.1
Hobart, Australia, 1989	160	124	9.4	7.3
Hobart, Australia, 1995	170	118	9.9	6.9
Sydney, Australia, 1992	164	133	9.6	7.8
Britain, 1986-87	173	132	10.1	7.7

Sources: Simpson et al 1978; Simpson et al 1982; Thomson and Colls 1998; Beard et al 1992; Beard et al 1997; Notowidjojo and Truswell 1993; Gregory et al 1990.

Taken together, these data suggest that daily sodium intake in New Zealand is approximately 150 mmol/L overall. A daily sodium (salt) intake of 150 mmol (9 g) is considerably higher than the recommended intake of 40–100 mmol (2.3–5.9 g) per day (Bullock 1990). Reducing sodium intake from 150 to 100 mmol/day could substantially lower mean SBP.

The majority of dietary sodium intake (at least 75 percent) is non-discretionary, and comes from salt added to food during its manufacturing and processing (James et al 1987; Mattes and Donnelly 1991). Discretionary use of salt during cooking or at the table accounts for about 15 percent of dietary sodium intake, while sodium naturally present in foods provides the remaining 10 percent (James et al 1987; Mattes and Donnelly 1991). This suggests that in the New Zealand diet, manufactured foods provide about 7 g/day of salt, and salt added during cooking or at table provides about 1.5 g/day.

DIAMOND (Dietary Modelling of Nutritional Data), a software program developed by Food Standards Australia New Zealand (FSANZ), was used to determine the principal sources of sodium in the New Zealand diet, excluding the use of ‘table’ salt. DIAMOND combines food consumption data with food chemical concentration data (including nutrients) to estimate dietary intake. DIAMOND uses food consumption data from the 1997 NNS and nutrient concentration data derived from the New Zealand Food Composition Database.

As shown in Table 22, bread is the leading source of sodium in the New Zealand diet, accounting for one-quarter of all dietary sodium. Other manufactured foods that are major sources of sodium in the New Zealand diet include processed meats (10.3 percent), sauces (6.7 percent), breakfast cereals (5.8 percent), cakes, muffins and biscuits (4.5 percent), bread-based dishes (4.3 percent), and butter and margarine (3.8 percent).

**Table 22:** Food groups contributing to sodium intake in New Zealand

Food group	Percentage of sodium intake	Notes
Breakfast cereals	5.8	Includes porridge
Grains and pasta	3.2	Rice, flour, pasta, noodles
Breads	25.7	
Bread-based dishes	4.3	Sandwiches, burgers, pizza, tortilla, etc
Cakes, muffins and biscuits	4.5	Includes crackers
Pies and pasties	3.1	Meat pies, pasties, sausage rolls, etc
Processed meats and sausages	10.3	Includes ham and bacon
Meat and poultry	5.0	Beef and veal, lamb & mutton, pork, poultry
Fish and seafood	3.2	
Eggs	0.9	
Cheese	2.8	
Butter and margarine	3.8	
Milk and dairy	4.3	Cream, ice cream, sour cream, yoghurt
Fats and oils	0.0	
Potatoes and kumara	6.7	Excludes potato crisps, includes chips
Vegetables	3.5	
Fruit, nuts and seeds	0.5	
Sauces	6.3	
Stocks and soups	1.7	
Snack foods	1.0	Includes potato crisps
Beverages	2.0	
Sugar/sweets and puddings	0.7	
Herbs and spices	0.6	

Source: 1997 NNS data modelled using DIAMOND

## Potassium

### *Potassium and systolic blood pressure*

In contrast to sodium, diets high in potassium help regulate blood pressure. The Intersalt study found a negative relationship between potassium intake and SBP in most of the 52 centres in the Intersalt study (Intersalt Co-operative Research Group 1988). Subsequent reanalysis of the data, which adjusted for potential confounders and regression dilution bias, showed that a 50 mmol/day higher urinary potassium excretion was associated with a 3.4/3.3 mmHg lower SBP (with/without adjustment for BMI) (Stamler 1997).

Randomised controlled feeding trials provide more direct evidence than observational studies of the effects of potassium on blood pressure. Whelton et al (1997) undertook a meta-analysis of 33 randomised controlled potassium supplementation trials (2609 participants) and found a significant 2.9 mmHg reduction in mean SBP for a 50 mmol/day increase in potassium. The small net changes in urinary sodium (-7 mmol/day) and body weight (-0.1 kg) may have contributed slightly to the decrease in SBP associated with potassium supplementation.



Taken together, data from observational studies and controlled feeding trials suggest an increase in dietary potassium of 30 mmol/day could lower SBP by up to 1.5 mmHg.

Given the opposing effects of sodium and potassium, it is the ratio of the two intakes that is in fact the main dietary determinant of blood pressure. Thus sodium restriction can partially offset the impact of low vegetable and fruit consumption (a major source of potassium), while increasing vegetable and fruit intake can partly compensate for a high salt diet. More importantly, simultaneously reducing salt and increasing vegetable and fruit intake will have a synergistic effect on blood pressure.

### ***Potassium in the diet***

Vegetables and fruit are a good source of potassium. Diets high in vegetables and fruit are associated with lower risk of cardiovascular disease, and much of this benefit is mediated via the beneficial effects of potassium on blood pressure (Law and Morris 1998). The original DASH trial, which assessed the effects of dietary patterns on blood pressure, shows that high intakes of vegetables and fruit lower blood pressure (Appel et al 1997). The eight-week DASH trial compared three diets: control diet (typical US diet); vegetable and fruit diet (enriched in vegetables and fruit but otherwise similar to the control diet); and the DASH diet (rich in vegetables, fruit, low-fat dairy products, whole grains, poultry, fish and nuts and low in red meat, sweets and sugar-containing beverages). Sodium intake and body weight were kept constant throughout this phase of the DASH trial. Compared to the control diet, the vegetable and fruit diet lowered SBP by 2.8 mmHg.

Increasing vegetable and fruit consumption has also been shown to lower blood pressure in the general population. In a recent randomised controlled dietary intervention trial among free-living adults in England, daily vegetable and fruit intake increased by 1.4 portions over the six-month intervention period, and this was associated with a 4.0 mmHg decrease in systolic blood pressure (John et al 2002).

In addition to vegetables and fruit, dietary sources of potassium include meat and dairy products. In 1997, daily intakes of potassium in New Zealand adults were 89 mmol (102 mmol in males and 77 mmol in females) (Russell et al 1999). Recommended intakes are 50–140 mmol/day for males aged 19–64 years and females aged 19–54 years, 40–130 mmol/day for males aged 65 years and over, and 40–100 mmol/day for females aged 55 years and over (Truswell 1990).

## **Alcohol**

### ***Alcohol and systolic blood pressure***

The Intersalt study found heavy use of alcohol was directly and independently related to SBP, both within and between populations (Intersalt Co-operative Research Group 1988). Subsequent reanalysis of the data, which adjusted for confounding variables and regression dilution bias, showed that high alcohol intake ( $\geq 300$  ml/week) was associated with a 3.5/3.2 mmHg higher SBP (with/without adjustment for BMI) (Stamler 1997). The Multiple Risk Factor Intervention Trial (MRFIT), a randomised primary prevention trial among 12,866 men aged 35–57 years, found alcohol intake was significantly and positively correlated with SBP, with a one energy percentage point change in alcohol intake associated with a 0.06 mmHg change in SBP (Stamler et al 1997). A New Zealand study found males with high alcohol intakes ( $\geq 300$  ml/week) had SBP measurements 9.8 mmHg higher than non-drinkers, after adjustment for age and BMI (Paulin et al 1985).

Randomised controlled alcohol restriction trials demonstrate that reducing alcohol intake lowers blood pressure. A meta-analysis of 15 randomised controlled trials (2234 participants) in which alcohol reduction was the only intervention found that among heavy drinkers (three to six drinks per day) an average reduction in alcohol intake of 67 percent (ie, to one to three drinks per day) reduced SBP by 3.31 mmHg (Xin et al 2001). The dose–response effect of alcohol reduction on blood pressure was consistent across subgroups, including those without hypertension.

These data suggest that reducing alcohol intake by one drink per day lowers SBP by 1.0 mmHg in high alcohol consumers (over three drinks per day).

### ***Alcohol in the diet***

In 1997 alcohol provided 4 percent of total energy, and principal sources of alcohol in the New Zealand diet were beer (53 percent) and wine (32 percent) (Russell et al 1999). A national survey of alcohol consumption in New Zealanders aged 14–65 years found that 85 percent of people surveyed were drinkers in 2000 (Habgood et al 2001). On average, males drank 20 drinks per week and females drank nine. Twenty-nine percent of the sample consumed in excess of 10 litres of absolute alcohol annually (equivalent to 1.8 drinks per day), and 14 percent consumed in excess of 20 litres of absolute alcohol annually (equivalent to 3.7 drinks per day). This suggests that approximately 17 percent of adults are heavy drinkers (over three drinks per day). Therefore, reducing alcohol consumption by one drink per day in high-alcohol consumers could lower SBP by 1.0 mmHg in high alcohol consumers, which translates into a 0.15 mmHg lower mean population SBP.

## **Body mass index**

### ***Body mass index and systolic blood pressure***

In observational studies, blood pressure has been consistently found to increase linearly with increasing BMI. The nationwide screening of one million US adults showed a much greater prevalence of hypertension in overweight people compared to normal-weight people (Stamler et al 1978). The Intersalt study found that BMI was significantly and positively correlated with SBP (Intersalt Co-operative Research Group 1988). Further analyses of the Intersalt study (Stamler 1997) showed that a three-unit-higher BMI was associated with a 2.2 mmHg higher SBP (after controlling for potential confounders and regression dilution bias), which is equivalent to a 0.7 mmHg higher SBP for a one-unit-higher BMI. Based on data from NHANES III (1988–94), mean SBP was about 9 mmHg higher for males and 11 mmHg higher for females in the highest BMI category ( $\geq 30$ ) compared with the lowest BMI category ( $< 25$ ) (Brown et al 2000).

Prospective studies confirm that increases in body weight or BMI are associated with an increase in SBP. A study of over 9000 adults aged 45–64 years in the United States showed that for every 1 kg increase in body weight per year, the mean annual increase in SBP was approximately 0.5 mmHg (Juhaeri et al 2002). In the Nurses' Health Study, BMI at age 18 years and midlife were positively associated with the incidence of hypertension, and weight gain was found to dramatically increase the risk of hypertension (Huang et al 1998).

Although weight loss trials have been shown to reduce blood pressure, few attempts have been made to quantify the effect of weight loss on blood pressure, probably because weight loss is generally associated with several dietary changes that could influence blood pressure. A 1988 meta-analysis of 12 studies, including five randomised controlled trials, found that a 1 kg decrease in body weight in obese hypertensive patients was associated with a 2.4 mmHg decrease in SBP (Staessen et al 1988), although the authors acknowledged that a decrease in salt intake may have contributed to blood pressure lowering.

A recent Cochrane systematic review updated the Staessen meta-analysis, focusing on randomised controlled trials (Mulrow et al 2001). The 18 identified trials included 2611 hypertensive participants with an average body weight of 84 kg. The data suggest that weight loss in the range of 4 to 8 percent of body weight produces an average reduction in SBP of 3.0 mmHg. The authors did not quantify the specific blood pressure decrease per kilogram of weight loss as the finding was not statistically significant; however, the findings suggest a 1 kg decrease in body weight is associated with a 0.5–1.0 mmHg decrease in SBP.

Taken together, these data suggest a 1 kg weight loss could lower SBP by at least 0.5 mmHg.

### ***Body mass index in New Zealand***

In 1997 mean body weight in adults was 74.5 kg and mean BMI was 26.1 kg/m<sup>2</sup> (Russell et al 1999), which represents a substantial increase since 1989, when mean body weight was 71.3 kg and mean BMI was 25.0 kg/m<sup>2</sup> (Russell et al 1999). The prevalence of obesity (BMI ≥ 30.0 for Europeans and Others and BMI ≥ 32.0 for Māori and Pacific peoples) increased from 11.1 to 17.0 percent between 1989 and 1997 (Russell et al 1999). The expected continued increase in BMI is likely to impact unfavourably on population blood pressure levels.

### **Physical activity**

#### ***Physical activity and systolic blood pressure***

Increasing physical activity levels has been shown to lower blood pressure independently of its effect on body weight. A recent meta-analysis of 54 randomised controlled trials (2419 participants with sedentary lifestyles), found that aerobic exercise was associated with a significant 3.8 mmHg reduction in SBP (Whelton et al 2002). The impact of an overall intervention-related weight loss of 0.42 kg was not statistically significant and was not considered biologically important. The reduction in blood pressure occurred in both overweight and normal-weight participants, and was slightly greater in hypertensive than normotensive participants. The SBP reduction related to aerobic exercise did not differ significantly according to exercise intensity, but was slightly greater in those who exercised for longer durations (2.8 mmHg for less than 120 minutes per week, 4.7 mmHg for 120–150 minutes per week, and 5.1 mmHg for more than 150 minutes/week). In a separate meta-analysis of 47 randomised controlled trials (2543 subjects), aerobic exercise was found to decrease systolic blood pressure by 6 mmHg in hypertensive and 2 mmHg in normotensive subjects (Kelley et al 2001a).

Walking and resistance exercise also appear to lower blood pressure. In a meta-analysis of 16 studies (650 subjects), walking was found to significantly decrease systolic blood pressure by 3 mmHg (approximately 2 percent) in adults (Kelley et al 2001b). A meta-analysis of 11 studies (320 subjects) indicates that resistance exercise lowers systolic blood pressure by approximately 3 mmHg (Kelley and Kelley 2000).

Taken together, these data suggest that increasing physical activity among inactive adults can lower SBP by 3 mmHg.

#### ***Physical activity in New Zealand***

In 1996/97 15 percent of adults were inactive and a further 24 percent were relatively inactive (less than 150 minutes per week activity) (Ministry of Health 1999a). Given that a portion of the population will be inactive due to disability or illness, reducing the proportion of the population who are inactive to 10 percent is a realistic goal. Therefore, reducing the proportion of adults who are inactive from 15 to 10 percent could lower SBP by 3 mmHg in inactive people, which translates into a 0.15 mmHg lower mean population SBP.

## Summary

Summary estimates of the change in mean population SBP expected following a given change in modifiable determinants of blood pressure are shown in Table 23.

**Table 23:** Decrease in mean population systolic blood pressure for a given change in key determinants

Determinant	Current level (mean)	Change in determinant	Decrease in mean systolic blood pressure
Sodium (salt)	150 mmol (9 g)	↓ 50 mmol (3 g)	2.50 mmHg
Potassium	89 mmol	↑ 30 mmol	1.50 mmHg
Weight (BMI)	74.5 kg (26.1 kg/m <sup>2</sup> )	↓ 1 kg (0.35 kg/m <sup>2</sup> )	0.50 mmHg
Alcohol	17% high alcohol*	↓ 1 drink/day	0.15 mmHg
Physical activity	15% sedentary	↓ sedentary by 5%	0.15 mmHg

\* More than three drinks per day.

## 4.3 Blood pressure distributions

### *Current distribution*

It was intended that data on the current blood pressure distribution (mean and standard deviation) in New Zealand would be obtained from the 1997 NNS. However, blood pressure measurements from this survey were considered ‘unreliable’ by investigators (N Wilson, personal communication, November 2001) due to difficulty experienced in calibrating the instrument used to measure blood pressure in the survey, an Omron 706c smart-inflate blood pressure monitor. This change in instrument may be the explanation for the unexplained increase in blood pressure between the 1989 Life in New Zealand (LINZ) survey (Mann et al 1991) and the 1997 NNS (Russell et al 1999), an increase that was contrary to trends in blood pressure in Auckland during the late 1980s and early 1990s (Trye et al 1996), as well as trends observed in many similar populations internationally (Evans et al 2001).

SBP measurements from the 1997 NNS were therefore adjusted downward, to bring them in line with data from three cross-sectional surveys undertaken in Auckland as part of the WHO MONICA project during the 1980s and early 1990s. The three surveys were as follows: the 1982 Auckland Risk Factor Study involving 1568 people, the 1986–88 Auckland Heart Study involving 888 people, and the 1993–94 Auckland Heart and Health Study involving 1350 people. Reported blood pressure trends were restricted to Europeans aged 35–64 years (Trye et al 1996).

The age-standardised SBP measurements from the 1982, 1986–88 and 1993–94 Auckland studies were 132.2, 129.5 and 126.3 mmHg for males; and 125.9, 126.7 and 121.7 mmHg for females. A linear regression model was used to project MONICA age-standardised SBP measurements for 1997 (124.8 and 121.3 mmHg for males and females, respectively). These values were divided by the 1997 NNS age-standardised SBP measurements for Europeans aged 35–64 years to give adjustment factors of 0.90 and 0.94 for males and females, respectively. The sex-specific adjustment factors were applied to all 1997 NNS age and ethnic groups to provide adjusted estimates of current SBP (Tables 24 and 25).

**Table 24:** Mean systolic blood pressure (mmHg), by ethnicity, sex and age group, 1997

	Sex	Age group (years)								
		15–24	25–34	35–44	45–54	55–64	65–74	75+	Total	Adjusted <sup>1</sup>
Non-Māori	Males	114	115	119	126	133	140	144	123	122
	Females	106	108	114	123	133	143	152	121	118
Māori	Males	114	117	124	132	139	146	145	122	126
	Females	104	108	119	132	148	150	146	116	122
Total	Males	114	116	120	127	134	140	144	123	123
	Females	106	108	114	123	134	144	152	120	118

<sup>1</sup> Age-standardised to WHO World population

**Table 25:** Standard deviation systolic blood pressure (mmHg), by ethnicity, sex and age group, 1997

	Sex	Age group (years)								
		15–24	25–34	35–44	45–54	55–64	65–74	75+	Total	Adjusted <sup>1</sup>
Non-Māori	Males	14	11	13	16	19	20	22	18	15
	Females	10	12	14	19	20	24	27	23	15
Māori	Males	10	12	15	18	21	30	22	17	16
	Females	10	14	18	27	22	15	21	23	17
Total	Males	13	11	14	16	19	20	22	18	15
	Females	10	12	15	20	20	24	27	23	16

<sup>1</sup> Age-standardised to WHO World population.

The estimated current age- and sex-specific SBP measurements were compared to data from the 1989 LINZ survey to ensure the decreases from 1989 and 1997 were plausible. The 1997 age-standardised SBP estimates (123 mmHg males, 118 mmHg females) were considerably lower than age-standardised SBP measurements in the 1989 LINZ survey (129 mmHg males, 124 mmHg females) (Mann et al 1991). The 1997 SBP estimates, particularly for males, are also lower than in many other countries (see next section), suggesting they may have been adjusted too far downward. However, it was decided to proceed with the 1997 estimates as the lower SBP measurements will provide more conservative results with respect to mortality attributable to high blood pressure.

## Trends and international comparisons

SBP has been declining in most developed countries for the last few decades. In New Zealand, national trends in SBP could not be determined due to the unreliability of the 1997 NNS data (discussed previously). However, the three cross-sectional Auckland surveys undertaken for the WHO MONICA project showed that age-standardised SBP measurements declined from 1982 to 1993–94 in New Zealand Europeans, from 126.3 to 123.2 mmHg in males and from 125.9 to 121.7 mmHg in females (Trye et al 1996).

In English adults aged 16 years and older, SBP declined from 139 to 136 mmHg in males and from 136 to 132 mmHg in females from 1993 to 2001 (Department of Health [London] 2003). In Scottish adults aged 16–64 years, there was no real change in SBP values from

1995 to 1998 (The Scottish Executive Department of Health 2000). In Scottish adults aged 16–74 years, SBP values in 1998 were 132 and 127 mmHg in males and females respectively (The Scottish Executive Department of Health 2000). Based on data from NHANES III (1988–94), mean SBP is 125 mmHg in US males and 120 mmHg in US females aged 20 years and older (Brown et al 2000).

Information on trends in mean SBP is not available from Australia, but the prevalence of hypertension (systolic  $\geq 140$  mmHg or diastolic  $\geq 90$  mmHg or taking anti-hypertensive medication) is declining. In Australian males aged 25–64 years, the prevalence of hypertension fell from 45 percent in 1980 to 22 percent in 1999–2000 (AIHW 2001). In Australian females aged 25–64 years, the prevalence of hypertension decreased from 29 percent in 1980 to 16 percent in 1995, but has not changed since then. In the United States, the prevalence of hypertension decreased between 1976–80 and 1988–94, from 45 to 26 percent in males and from 36 to 21 percent in females (National Center for Health Statistics 2002).

Table 26 shows current SBP levels and/or the prevalence of hypertension in New Zealand and other similar countries.

**Table 26:** International comparisons of mean systolic blood pressure

Country, year	Systolic blood pressure (mmHg)	
	Males	Females
New Zealand, 1997 <sup>2</sup>	123	121
England, 2001	136	132
Scotland, 1998	132	127
United States, 1988–94	125	120

Sources: Department of Health [London] 2003; The Scottish Executive Department of Health 2000; Brown et al 2000; National Center for Health Statistics 2002.

1 Systolic  $\geq 140$  mmHg or diastolic  $\geq 90$  mmHg or taking anti-hypertensive medication.

2 Estimate.

The decline in SBP in Auckland from 1982 to 1993–94 was not explained by changes in pharmaceutical treatment, but coincided with small increases in leisure time physical activity, and reductions in smoking, alcohol consumption and possibly the amount of salt added to manufactured foods (Trye et al 1996). A recent New Zealand study showed that in order to meet the National Heart Foundation *Pick the Tick* criteria, food manufacturers substantially reduced the sodium content of selected breads, breakfast cereals and margarines (Young and Swinburn 2002). However, food manufacturers stated that the sodium content of these foods was not otherwise being reduced over time.

In the United States, the food industry is reducing the sodium content of foods very gradually, by approximately 1 percent per year (Jacobson and Liebman 1996). However, mean daily sodium intakes from foods appear to have increased slightly between 1980–82 and 1990–92 in the United States (Engstrom et al 1997), perhaps due to a change in food consumption patterns. For example, foods eaten or prepared outside the home, which are usually higher in sodium than foods prepared at home, represent an increasing proportion of overall food intake. Analysis of a selection of common restaurant foods in the United States showed that the salt content of some single meals exceeded the recommendations for an entire day (Liebman and Jacobson 1997). The increased sodium intake as well as the increase in obesity may explain why the most recent data from the United States show that the prevalence of hypertension has increased in recent years (CDC 2002).

### **Theoretical minimum distribution**

The theoretical minimum distribution is the risk factor (ie, SBP) distribution that would yield the lowest population risk of adverse health outcomes. In developed countries, the relationship between SBP and cardiovascular disease is continuous, with no evidence of a lower threshold, although the relationship has seldom been demonstrated below an SBP measurement of 115 mmHg in Western populations (MacMahon et al 1990; Prospective Studies Collaboration 1995, 2002; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration 1999).

Populations with little or no cardiovascular disease provide an alternative source of data on the SBP level associated with the lowest risk of disease. The Intersalt study included several such populations: rural villagers in remote areas of Kenya, Yanamamo Indians of the Amazon rain forest, Xingu Indians of Brazil, and rural villagers in remote areas of Papua New Guinea (Carvalho et al 1989). Mean SBP in these populations ranged from 105–115 mmHg, and in contrast to Western populations there was virtually no increase in blood pressure with age.

Taken together, these data suggest that an SBP of 110–115 mmHg would be an appropriate theoretical minimum for all age groups. For this study, calculations of attributable and avoidable mortality will be based on a theoretical minimum SBP distribution of  $115 \pm 6$  mmHg for all age, sex and ethnic groups. This theoretical minimum is based on that chosen by the WHO blood pressure expert working group for the *World Health Report 2002* (Ezzati et al 2002; Lawes et al, in press).

### **Distributional transitions**

Two distributional transitions for SBP were estimated: a business as usual (BAU) scenario (historical trend) and an intervention scenario (deviation from the historical trend, reflecting policy change). Distributional transitions for SBP were based on trends in New Zealand, as indicated by the Auckland MONICA studies, as well as overseas trends in SBP and/or the prevalence of hypertension. Trends in key determinants of blood pressure were also taken into account when estimating the distributional transitions.



Distributional transitions are expressed as a percentage shift from the current distribution towards the theoretical minimum. For SBP, distributional transitions were assumed to be the same for all age, sex and ethnic groups. However, absolute changes in SBP vary by age, sex and ethnicity depending on baseline SBP levels.

The avoidable burden is the difference between the projected BAU scenario and the intervention scenario, with the shift between the two scenarios being maintained over time.

### ***Business as usual scenario***

Given that current evidence suggests SBP measurements will continue to decline (although more slowly than in the past), under the BAU scenario we estimated an overall 0.5 mmHg decrease in the mean population SBP by 2011. This 0.5 mmHg decrease is equivalent to an 8 percent shift in the current SBP distribution towards the theoretical minimum. Because SBP increases with age, an 8 percent distributional transition is equivalent to a 2–3 mmHg decrease in SBP in adults aged 65 years and over. Table 27 shows the projected SBP levels in 2011 by ethnicity, sex and age under the BAU scenario.

**Table 27:** Projected 2011 systolic blood pressure levels under the business as usual scenario

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Non-Māori	Males	115.0	118.7	125.1	131.6	138.0	141.7	122.4
	Females	108.6	114.1	122.4	131.6	140.8	149.0	120.5
Māori	Males	116.8	123.3	130.6	137.1	143.5	142.6	121.4
	Females	108.6	118.7	130.6	145.4	147.2	143.5	115.9
Total	Males	115.9	119.6	126.0	132.5	138.0	141.7	122.4
	Females	108.6	114.1	122.4	132.5	141.7	149.0	119.6

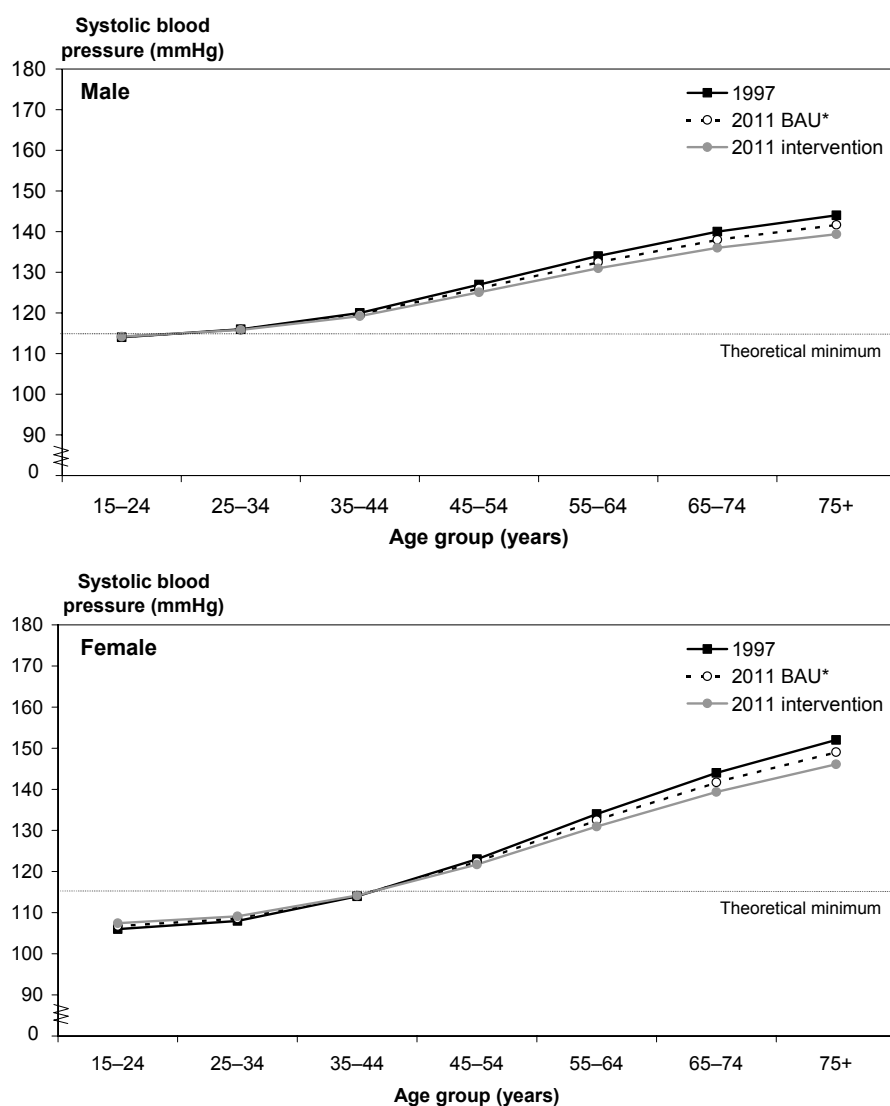
### ***Intervention scenario***

Under the more optimistic intervention scenario, we estimated an overall 1.0 mmHg decrease in the mean population SBP by 2011. This 1.0 mmHg decrease is roughly equivalent to a 16 percent shift in the current SBP distribution towards the theoretical minimum. Because SBP increases with age, a 16 percent distributional transition is equivalent to a 4–6 mmHg decrease in SBP in adults aged 65 years and over. In this older age group, the intervention scenario would therefore result in a 2–3 mmHg decrease over and above that estimated in the BAU scenario. Table 28 shows the projected SBP levels in 2011 by ethnicity, sex and age under the intervention scenario.

**Table 28:** Projected 2011 systolic blood pressure levels under the intervention scenario

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Non-Māori	Males	115.0	118.4	124.2	130.1	136.0	139.4	121.7
	Females	109.1	114.2	121.7	130.1	138.5	146.1	120.0
Māori	Males	116.7	122.6	129.3	135.2	141.0	140.2	120.9
	Females	109.1	118.4	129.3	142.7	144.4	141.0	115.8
Total	Males	115.8	119.2	125.1	131.0	136.0	139.4	121.7
	Females	109.1	114.2	121.7	131.0	139.4	146.1	119.2

Figure 12 shows current (1997) SBP levels, as well as projected 2011 SBP levels under the BAU and intervention scenarios (separately for males and females) in relation to the theoretical minimum.

**Figure 12:** Current (1997) and projected future (2011) systolic blood pressure levels

\* BAU = business as usual.

## 4.4 Risk factor–disease relationships

### Disease outcomes

The outcomes assessed were based on those selected for the *World Health Report 2002* (WHO 2002):

- ischaemic heart disease
- stroke.

### Risk accumulation

Risk accumulation refers to the nature and strength of the association between an exposure (ie, blood pressure) and a disease, and is expressed as the regression (risk) coefficient in the case of a continuous risk factor. In other words, the regression coefficient is the increase in incidence or mortality of the disease per unit increase in exposure.

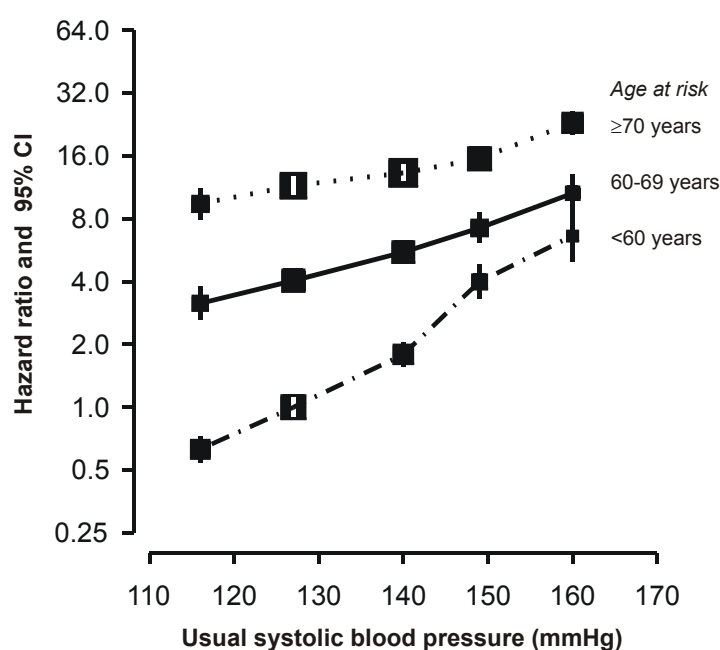
Several major overviews of observational studies investigating the relationship between SBP and cardiovascular disease (IHD and stroke) endpoints have been undertaken (MacMahon et al 1990; Prospective Studies Collaboration 1995, 2002; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration 2003b).

For this study, we used the Asia Pacific Cohort Studies Collaboration (APCSC) overview for risk accumulation estimates. The APCSC study combined data from 37 prospective cohorts from eight countries in the Asia–Pacific region, including four cohorts from Australia and one from New Zealand. A major advantage of the APCSC overview was that it included individual participant data and was therefore able to provide more reliable estimates of risk accumulation for different age groups and was better able to control for confounding. For blood pressure and cardiovascular disease, the APCSC overview included data on 425,251 participants (57 percent male) aged 20–107 years. During the follow-up period (mean seven years), there were 3560 IHD and 4355 stroke events (Asia Pacific Cohort Studies Collaboration 2003b).

### *Ischaemic heart disease*

Data from the APCSC overview show that the risk of IHD increases with increasing SBP, and that the association is roughly linear when plotted on a log scale (Figure 13). The association is continuous, with no evidence of a lower threshold below which lower levels of SBP are not associated with a decreased risk of IHD (down to 115 mmHg). There is no evidence of an upper threshold either (Asia Pacific Cohort Studies Collaboration 2003b).

**Figure 13:** Usual systolic blood pressure and risk of ischaemic heart disease, by age



Source: Asia Pacific Cohort Studies Collaboration 2003

As shown in Figure 13 and Table 29, the association between SBP and IHD varies with age: it is strongest in young adults and then attenuates with age. Since the APCSC data provide no evidence that the strength of the association between SBP and IHD varies by sex, it was not necessary to have different estimates for males and females. It was assumed that the relationship between SBP and IHD was the same for all ethnic groups. The APCSC data provided no evidence of a differential association for fatal and non-fatal IHD endpoints.

A 1 mmHg lower SBP was associated with a lower risk of IHD of 6.3 percent in younger adults, dropping to a 1.1 percent lower risk in adults aged 75 years and over.

**Table 29:** Risk coefficients for ischaemic heart disease for a 1 mmHg lower systolic blood pressure

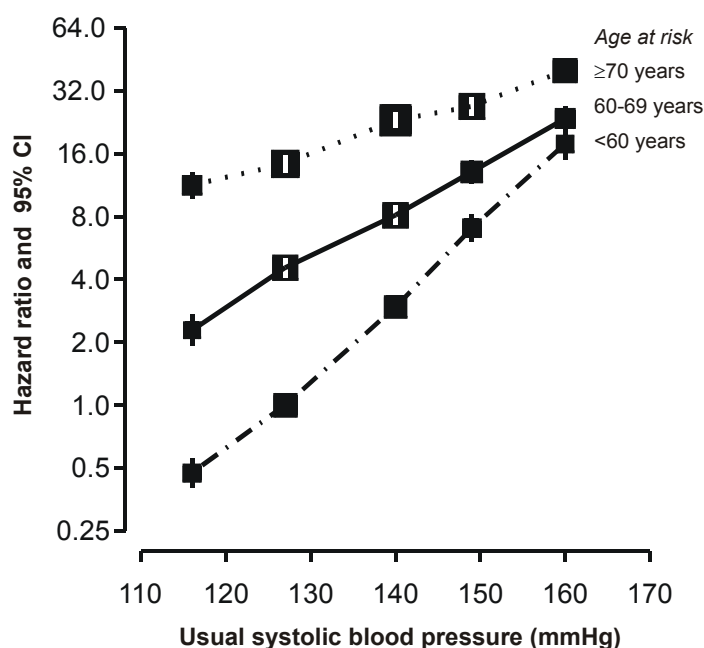
Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.937	6.3
35–44	0.937	6.3
45–54	0.950	5.0
55–64	0.961	3.9
65–74	0.975	2.5
75+	0.989	1.1

Source: Asia Pacific Cohort Studies Collaboration

## Stroke

Data from the APCSC overview show that the risk of stroke increases with increasing SBP, and that the association is roughly linear when plotted on a log scale (Figure 14). The association is continuous, with no evidence of a threshold below which lower levels of SBP are not associated with decreased risk of stroke (down to 115 mmHg). There is no evidence of an upper threshold either (Asia Pacific Cohort Studies Collaboration 2003b).

**Figure 14:** Usual systolic blood pressure and risk of stroke, by age



Source: Asia Pacific Cohort Studies Collaboration 2003

The proportions of stroke subtypes (haemorrhagic and ischaemic) differs between cohorts in Asia and Australia / New Zealand (ANZ). Therefore, although the total APCSC cohort has greater numbers and therefore better power, for this study risk estimates for total stroke are based on the ANZ data. As the risk estimates based on the ANZ data are lower than those based on the entire APCSC cohort, we may have underestimated that association between SBP and stroke.

As shown in Figure 14 and Table 30, the association between SBP and stroke varies with age: it is strongest in young adults and attenuates with age. Since the APCSC data provide no evidence that the strength of the association between SBP and IHD varies by sex, it was not necessary to have different estimates for males and females. It was assumed that the relationship between SBP and stroke was the same for all ethnic groups.

A 1 mmHg lower SBP was associated with a lower risk of stroke of 5.6 percent in younger adults, dropping to a 1.8 percent lower risk in adults aged 75 years and over.

**Table 30:** Risk coefficients for stroke for a 1 mmHg lower systolic blood pressure

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.944	5.6
35–44	0.944	5.6
45–54	0.957	4.3
55–64	0.966	3.4
65–74	0.976	2.4
75+	0.982	1.8

Source: Asia Pacific Cohort Studies Collaboration

## Risk reversal

Risk reversal refers to how quickly and how completely risk of cardiovascular disease is reversed following blood pressure lowering. Randomised controlled trials provide information on risk reversibility. A vast number of trials have studied the impact of blood pressure lowering on cardiovascular disease. However, individual studies usually lack sufficient power to reliably detect moderate changes in events, and results from overviews are therefore more reliable. It was assumed that risk reversibility was the same for both sexes and all ethnic groups.

### *Ischaemic heart disease*

Overviews of randomised controlled trials have all confirmed that a reduction in SBP reduces the risk of IHD (Collins et al 1990; MacMahon and Rodgers 1993a, 1993b; Psaty et al 1997; He and Whelton 1999; Blood Pressure Lowering Treatment Trialists' Collaboration 2000; Pahor et al 2000). Three of these studies suggest a 10 mmHg decrease in SBP is associated with a 15–20 percent reduction in IHD (Collins et al 1990; MacMahon and Rodgers 1993a; He and Whelton 1999), with one overview finding similar reductions for fatal and non-fatal IHD (He and Whelton 1999). The reduction in risk occurred after two to three years of blood pressure treatment (MacMahon and Rodgers 1993a; He and Whelton 1999), with approximately two-thirds (not all) of the risk of IHD reversed in this timeframe.

These estimates of risk reversibility are slightly less than the 20–25 percent lower risk of IHD associated with a 10 mmHg lower SBP found in cohort studies (MacMahon et al 1990; Prospective Studies Collaboration 2002; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration 2003b). Cohort studies showed a clear pattern of age attenuation, with the percentage reductions in IHD with SBP lowering being smaller in older age groups. There was no evidence of age attenuation in the trials, with relative risk estimates similar for those aged over 60 years and under 60 years (MacMahon and Rodgers 1993a, 1993b). However, this does not exclude the possibility of age attenuation, as the trials were often insufficiently powered to undertake subgroup analyses by multiple age groups.

In summary, our risk-reversal estimate is that SBP lowering is associated with complete reversal of the initial IHD risk after five years, whatever the initial level of risk may have been. This risk-reversal summary is based on that chosen by the WHO blood pressure expert working group for the *World Health Report 2002*, details of which will be published in a forthcoming technical report (Lawes et al, in press).

### **Stroke**

Overviews of randomised controlled trials have all confirmed that a reduction in SBP reduces the risk of stroke (Collins et al 1990; MacMahon and Rodgers 1993a, 1993b; Psaty et al 1997; He and Whelton 1999; Blood Pressure Lowering Treatment Trialists' Collaboration 2000; Pahor et al 2000). Major overviews of trials suggest a 10 mmHg decrease in SBP is associated with a 35–40 percent reduction in stroke (Collins et al 1990; MacMahon and Rodgers 1993a, 1993b). Subsequent overviews, which included additional trials, had similar results (Psaty et al 1997; He and Whelton 1999; Blood Pressure Lowering Treatment Trialists' Collaboration 2000), and suggest that potentially all of the excess risk of stroke due to higher than optimal blood pressure can be reversed. The timeframe for risk reversal was two to three years (MacMahon and Rodgers 1993a; He and Whelton 1999).

These estimates of risk reversibility are similar to the 30–40 percent lower risk of stroke associated with a 10 mmHg lower SBP found in cohort studies (MacMahon et al 1990; Prospective Studies Collaboration 1995, 2002; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration 2003b). Both trials and cohort studies suggest a similar reduction for fatal and non-fatal stroke.

As for IHD, our summary risk reversal estimate is that SBP lowering is associated with complete reversal of the initial stroke risk after five years. This risk-reversal estimate is based on that chosen by the WHO blood pressure expert working group for the *World Health Report 2002*, details of which will be published in a forthcoming technical report (Lawes et al, in press).

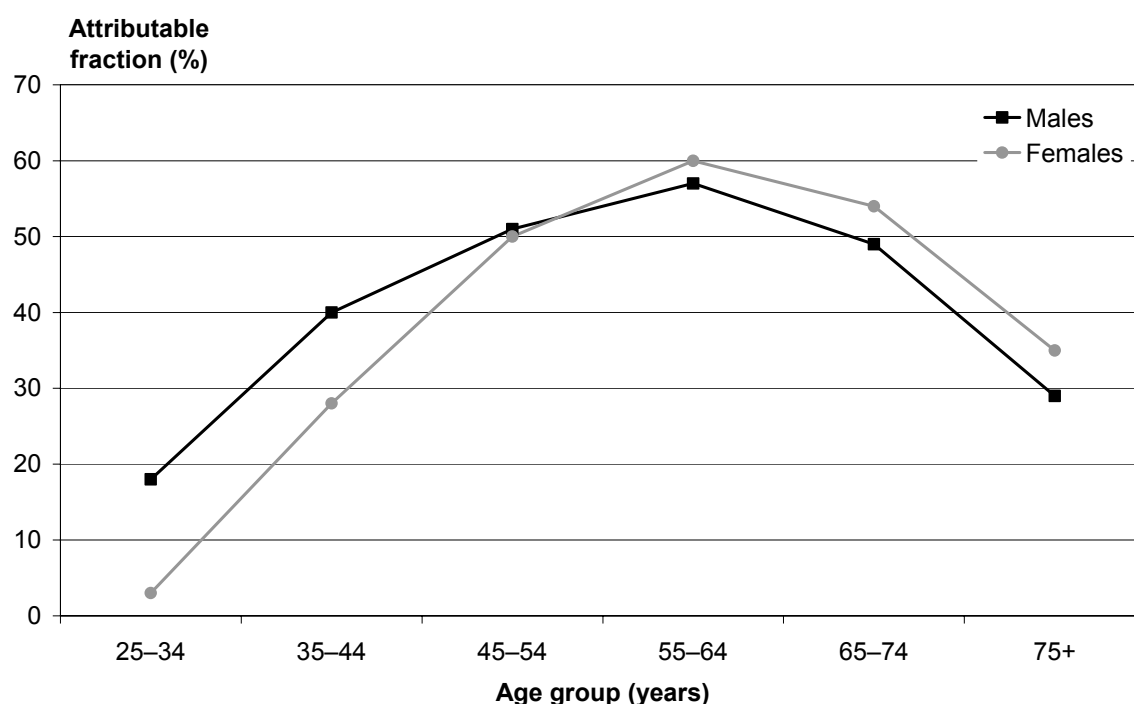
## **4.5 Attributable mortality**

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### **Ischaemic heart disease**

In the two oldest age groups, where the majority of IHD deaths occur, approximately 40 percent of IHD was attributable to 'high' SBP (Figure 15).

**Figure 15:** Attributable fractions (%) for systolic blood pressure and ischaemic heart disease, 1997



Overall, 2542 (95% CI 2033–2898) IHD deaths in 1997 (9 percent of all deaths) were attributable to ‘high’ SBP (Table 31). The majority (almost 80 percent) of IHD deaths attributable to ‘high’ SBP occurred in those aged 65 years and older. IHD mortality due to ‘high’ SBP contributed to 25,904 YLL in 1997.

The age-standardised IHD mortality rate attributable to ‘high’ SBP was twice as high in males as in females (Table 31). This difference primarily reflects the higher IHD mortality in males, as the attributable fractions were only slightly higher in males than in females. IHD mortality attributable to ‘high’ SBP in Māori was approximately twice as high as in non-Māori. This difference reflects a combination of higher IHD mortality and higher attributable fractions for Māori compared to non-Māori.

**Table 31:** Attributable mortality for systolic blood pressure and ischaemic heart disease, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	1,271	13,001	109.0	1,161
	Māori	166	2,467	225.1	2,934
	Total	1,437	15,468	117.6	1,303
Females	Non-Māori	1,010	9,109	57.3	567
	Māori	95	1,327	125.1	1,611
	Total	1,105	10,436	62.2	647

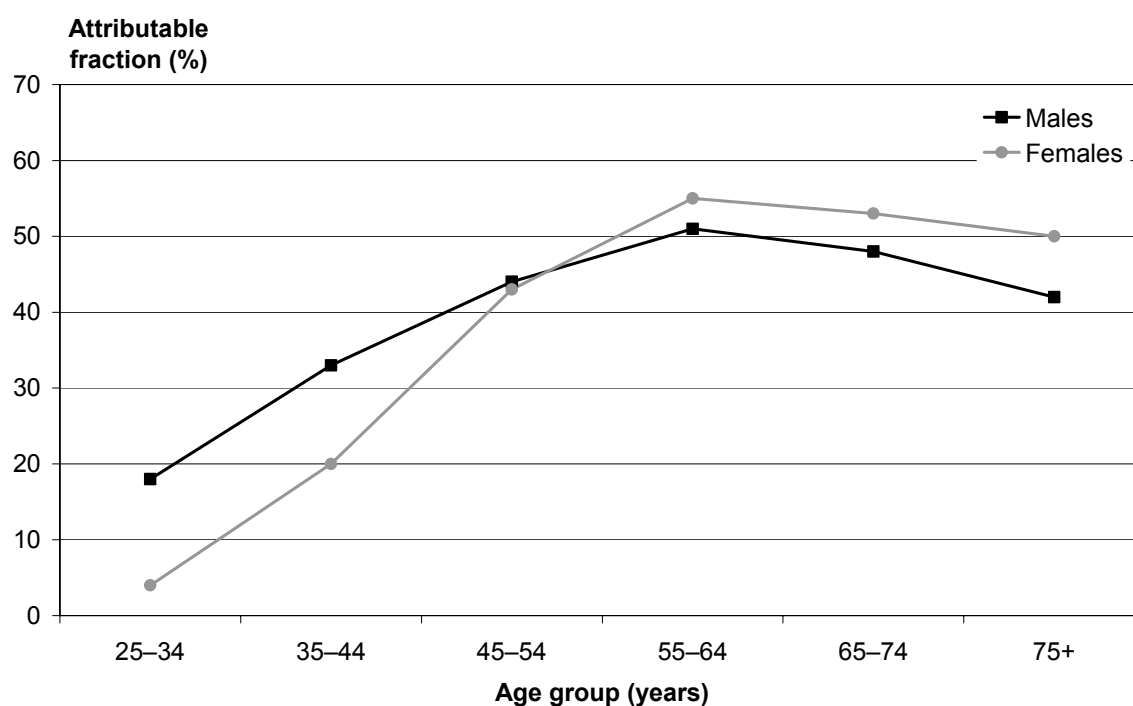
\* Rate per 100,000, age-standardised to WHO World population



## Stroke

In the two oldest age groups, where the majority of stroke deaths occur, approximately 45 percent of all strokes were attributable to 'high' SBP (Figure 16).

**Figure 16:** Attributable fractions (%) for systolic blood pressure and stroke, 1997



Overall, 1157 (95% CI 810–1407) stroke deaths in 1997 (4 percent of all deaths) were attributable to 'high' SBP (Table 32). The majority (almost 80 percent) of stroke deaths attributable to 'high' SBP occurred in those aged 75 years and older. A total of 9359 YLL were lost in 1997 from stroke due to 'high' SBP.

The age-standardised stroke mortality rate attributable to 'high' SBP was similar in males and females, and in Māori and non-Māori (Table 32).

**Table 32:** Attributable mortality for systolic blood pressure and stroke, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	390	2,902	32.1	246
	Māori	22	258	40.0	374
	Total	412	3,160	32.7	256
Females	Non-Māori	715	5,806	37.2	321
	Māori	30	393	43.5	505
	Total	745	6,199	38.2	340

\* Rate per 100,000, age-standardised to WHO World population

## Total attributable mortality

Overall, 'high' SBP contributed to a total of 3699 deaths (13 percent of all deaths) and 35,263 YLL in 1997 (Table 33).

**Table 33:** Attributable mortality for systolic blood pressure and all diseases, 1997

Sex	Ethnicity	Deaths		Years of life lost	
		Count	%*	Count	%*
Males	Non-Māori	1,661	13	15,903	12
	Māori	188	13	2725	11
	Total	1,849	13	18,628	12
Females	Non-Māori	1,725	14	14,915	14
	Māori	125	11	1720	9
	Total	1,850	14	16,635	12

\* % = percentage of all deaths and years of life lost, 1997.

## 4.6 Avoidable mortality

Estimates of IHD and stroke mortality in 2011 were forecast based on trends since 1976. As IHD and, to a lesser extent, stroke mortality declined steadily during this period for all birth cohorts, the forecasts indicate a continuation of this downward trend. However, it is possible that the obesity epidemic will slow this downward trend in years to come, meaning these forecasts may be somewhat optimistic. As a result, we may have underestimated avoidable mortality for 2011.

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### Ischaemic heart disease

With a decrease in SBP as outlined under the intervention scenario it is estimated that 3–11 percent of IHD deaths would be avoided each year from 2011. If SBP decreases to this extent, 179 IHD deaths and 1808 YLL could be avoided each year from 2011 (Table 34), over and above the mortality reduction expected under the BAU scenario.

**Table 34:** Avoidable mortality\* for systolic blood pressure and ischaemic heart disease, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	92	918
	Māori	17	225
	Total	109	1,142
Females	Non-Māori	60	545
	Māori	10	121
	Total	70	666

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario

## Stroke

With a decrease in SBP as outlined under the intervention scenario, 1–9 percent of stroke deaths would be avoided each year from 2011. The greatest proportion of deaths avoided will occur in the older age groups. If SBP decreases to this extent, 103 stroke deaths and 805 YLL could be avoided each year from 2011 (Table 35).

**Table 35:** Avoidable mortality\* for systolic blood pressure and stroke, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	34	240
	Māori	3	32
	Total	37	272
Females	Non-Māori	61	479
	Māori	5	54
	Total	66	533

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario

## Total avoidable mortality

If SBP levels were reduced as outlined under the intervention scenario, a total of 282 deaths and 2613 YLL could be avoided each year from 2011 (Table 36), over and above the mortality reduction expected under the BAU scenario.

**Table 36:** Avoidable mortality\* for systolic blood pressure and all diseases, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	126	1,158
	Māori	20	257
	Total	146	1,414
Females	Non-Māori	121	1,024
	Māori	15	175
	Total	136	1,199

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario

## 4.7 Discussion

We estimate that in 1997 ‘high’ SBP contributed to 3699 deaths (13 percent of all deaths) (Table 37). Although only a small incremental decrease in mean population SBP is proposed under the intervention scenario, this translates into significant decreases in both IHD and stroke mortality. If relevant policy initiatives were introduced now, such a change could prevent approximately 282 deaths each year from 2011 (Table 37). Some reduction in mortality would be seen even earlier, as the benefits of blood pressure lowering are seen within two to three years.

**Table 37:** Summary of attributable and avoidable mortality due to 'high' systolic blood pressure

	Attributable mortality (1997)		Avoidable mortality (2011)*	
	Deaths (count)	Years of life lost (count)	Deaths (count)	Years of life lost (count)
Ischaemic heart disease	2,542	25,904	179	1,808
Stroke	1,157	9,359	103	805
Total	3,699	35,263	282	2,613

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario

Although a single intervention scenario has been modelled here, the effect of alternative scenarios can be readily estimated. The relationship between the distributional transition and the avoidable burden is approximately linear. Therefore doubling the SBP shift (from 8 percent to 16 percent beyond the BAU scenario) would double the avoidable burden (from 280 to 560 deaths in 2011).

## Policy implications

The avoidable burden was calculated by shifting the current SBP distribution towards the theoretical minimum. This shift resulted in a 0.5 mmHg decrease in mean population SBP by 2011 over and above the BAU scenario. The shift in the SBP distribution was higher in the older age groups (2–3 mmHg reduction in SBP) due to higher blood pressure values in this group. Because the greatest number of events occur in the older age group, a reduction of 2 mmHg in older people is used to model possible policy approaches discussed in this section.

Two possible policy approaches for achieving the SBP reductions outlined under the intervention scenario have been modelled:

- a general shift toward the *Guidelines* diet (combined option)
- a specific effort to reduce use of salt as an additive in manufactured foods (salt option).

Of course these initiatives are by no means mutually exclusive. Other options for reducing blood pressure exist, but are not discussed here.

### Combined option

A range of dietary and lifestyle factors could reduce SBP. Promoting the *Food and Nutrition Guidelines for Healthy Adults* (Ministry of Health 2002b) is one way to achieve the blood pressure reduction outlined under the intervention scenario. The *Guidelines* recommend eating five or more servings of vegetables and fruit each day. They also recommend choosing foods low in salt and preparing foods with minimum added salt, drinking alcohol in moderation (if drinking), and maintaining a healthy body weight by regular physical activity and healthy eating.

If more New Zealanders followed the *Guidelines*, it is likely there would be a decrease in sodium (salt) intake, fewer high-alcohol consumers, an increase in potassium intake, little

or no further increase in the prevalence of overweight and obesity, and an increase in physical activity levels. All these changes would impact favourably on SBP.

For example, a reduction in sodium intake of 25 mmol/day (17 percent) could lower SBP by 1.25 mmHg. Increasing potassium intake by 10 mmol/day (11 percent) could lower SBP by a further 0.5 mmHg. Reducing alcohol consumption by one drink per day in those with high intakes (over three drinks per day) could reduce mean population SBP by 0.15 mmHg. Decreasing the proportion of the population who are sedentary from 15 to 10 percent could reduce mean population SBP by 0.15 mmHg. It is unlikely that the prevalence of overweight could be reduced in the short term; a more realistic aim would be to limit further increases in body weight.

Assuming these changes are additive, the overall impact would be a reduction in SBP similar to that outlined under the intervention scenario. Improved diagnosis and management of people with 'clinically' high blood pressure may also contribute to a change in the population blood pressure distribution.

### ***Salt option***

To achieve the blood pressure reduction outlined under the intervention scenario by reducing salt intake alone, sodium (salt) consumption would have to be decreased by 40 mmol/day (2.5 g/day). A reduction in salt intake from both manufactured foods and discretionary salt use needs to occur. Although a higher proportion of salt in the diet comes from manufactured foods, it is important to continue recommendations to reduce discretionary salt use as a New Zealand study found that sodium excretion was higher in adults that said they 'add salt regularly' compared with adults that said they 'never add salt' (Thomson and Colls 1998).

As current daily sodium (salt) intakes are about 150 mmol (9 g), a reduction as described above represents a 28 percent decrease in current intake, and would involve reducing the salt intake from manufactured foods from approximately 7 g/day to 5 g/day while simultaneously reducing discretionary salt use from 1.5 to 1.0 g/day.

This reduction in dietary salt intake could be achieved gradually by reducing the salt content of locally manufactured foods by about 4 percent per year over a period of 10 years (this allows for a lower rate of reduction in imported manufactured foods), and reducing discretionary salt use (by about one-third) at the same time.

It is unrealistic to expect salt not to be used as a food additive. In addition to its use as a flavouring agent, salt is added to foods for many technological purposes. For example, some salt is necessary in bread as it strengthens gluten by inhibiting the enzymes that catalyse the breakdown of proteins. If too little salt is added, the dough is tough and sticky. Salt is added to processed meats (eg, ham, bacon and salami) to inhibit microbial growth. However, salt is much cheaper than meat and it is also added to increase the moisture content, which increases the weight of the product.

Salt is currently added to many manufactured foods at levels far in excess of those required for quality (technology) and safety (microbial inhibition) reasons. The large variation in sodium levels within New Zealand breads (330–670 mg/100 g) and breakfast

cereals (2–1130 mg/100 g) (Athar et al 2001) suggests that sodium levels could be reduced substantially without affecting product stability and palatability. The typical sodium content of bread also varies considerably between countries, from about 200 mg/100 g in Asian countries to 500 mg/100 g in Western countries (Joossens et al 1994). Further evidence of the feasibility of reducing the salt content in manufactured foods comes from a recent New Zealand study. In one year, food manufacturers reduced the sodium content of four breakfast cereals by 61 percent, seven breads by 26 percent, and 12 margarines by 11 percent (Young and Swinburn 2002). The reduction in salt was achieved through formulation or reformulation in order to meet the National Heart Foundation *Pick the Tick* criteria, and food manufacturers stated that the sodium content of these foods was not otherwise being reduced.

A key argument against reducing the salt content of manufactured foods is a loss of palatability. However, an Auckland study found that participants could not tell the difference between reduced-salt bread compared to standard bread (Rodgers and Neal 1999). Furthermore, even if people notice a taste difference initially, the preferred level of salt can be lowered following a reduction in sodium intake (Bertino et al 1982). It is well known that people adapt to changes in the sodium content of the diet, and therefore reducing the salt content of manufactured foods gradually over 10 years is unlikely to be noticed by most people.

Another concern is that decreasing the sodium content of food will result in increased discretionary salt consumption. However, while there is evidence that discretionary salt use does increase slightly after the sodium content of foods is lowered, one study found that subjects compensated only partially (approximately 20 percent) for the reduction in salt intake (Beauchamp et al 1987). Simultaneous health promotion campaigns to discourage excessive discretionary salt consumption would also help to limit this potential loss of effectiveness.

Other objections to widespread salt restriction include possible deficiencies in intakes of other nutrients. A recent Finnish study shows that reducing daily salt intake by 5 g/day in free-living subjects with mildly elevated blood pressure had no adverse effects on nutrient intake (Korhonen et al 2000). In New Zealand, where iodine deficiency is a potential problem, a reduction in the use of iodised table salt could reduce iodine intake. Reducing the amount of salt added to manufactured foods is unlikely to have adverse effects on iodine status as almost all salt used in manufacturing is non-iodised. However, the effect of reducing salt intake on iodine status would need to be monitored and appropriate action taken if necessary.

It is likely that an important commercial concern of the food industry relates to the potential impact of salt restriction on soft drink consumption. It is known that there is a direct link between the salt content of manufactured foods and the consumption of soft drinks (MacGregor 1999). For this reason the salt, food and soft drink industries are very reluctant to reduce the salt content of processed foods. However, any ‘spin-off’ reduction in soft drink consumption would have added benefits in terms of healthy weight maintenance, especially among youth and young adults for whom soft drinks provide 5–9 percent of energy (Russell et al 1999).

Some countries have been successful in getting the food industry to lower the salt content of manufactured foods. One of the best-known examples is the North Karelia Project, a community-based cardiovascular disease prevention programme, which began in one province in eastern Finland in 1979 and was later expanded to the whole country (Narhinen and Cernerud 1995). One of the project initiatives was to reduce population salt intake, and the food industry was active in lowering the salt content of manufactured foods. The success of the initiative was considered to have resulted from co-operation between the government, food industry, medical and nutrition professions, and the media.

## **Limitations**

The need to recalibrate the 1997 NNS blood pressure data was an unexpected limitation of this study. The 1997 NNS SBP measurements were adjusted downward based on evidence from the Auckland MONICA studies that showed that blood pressure declined from 1982 to 1993/94. It was assumed that blood pressure measurements continued to decline through to 1997. However, although blood pressure measurements have been declining in most developed countries, at least up until the mid-1990s, there is some evidence that this declining trend has not continued. In the United States, the number of adults diagnosed with high blood pressure (self-reported) increased almost 10 percent between 1991 and 1999 (CDC 2002). The increase in the prevalence of hypertension occurred in almost all age, sex and ethnic groups, and is thought to be explained by the rapid increase in overweight and obesity. Improved detection and reporting of hypertension by health-care providers may also have contributed to the increase. Therefore, it is possible that we adjusted blood pressure measurements too far downward, which would result in underestimation of disease burden. A further limitation of using the Auckland MONICA studies to adjust our blood pressure measurements is that Auckland may not be representative of the general New Zealand population.

## **5. Nutrition, Total Blood Cholesterol and the Burden of Disease**

### **5.1 Introduction**

High blood cholesterol (hypercholesterolaemia) is an important risk factor for cardiovascular disease, particularly ischaemic heart disease (IHD). Traditionally, hypercholesterolaemia has been defined as total cholesterol  $> 6.5$  mmol/L. However, the association between blood cholesterol and cardiovascular disease is continuous and graded, from total blood cholesterol concentrations as low as 4.3 mmol/L (Stamler et al 1986). Therefore, many individuals with blood cholesterol concentrations below the 6.5 mmol/L threshold are at increased risk of cardiovascular disease and would benefit from cholesterol lowering.

### **5.2 Determinants of blood cholesterol**

Blood cholesterol concentrations are determined by a combination of genetic and environmental factors. Modifiable determinants of blood cholesterol include diet, body weight and physical activity levels. The focus of this chapter will be on dietary factors, particularly fat intake.

#### **Dietary fats**

Dietary fats are the most important environmental determinant of blood cholesterol concentrations. Triglycerides are the major component of dietary fats, and each triglyceride contains three (usually different) fatty acids attached to a glycerol backbone. Fatty acids are classified according to the number of double bonds on the hydrocarbon chain: saturated fatty acids (SFA) have no double bonds, monounsaturated fatty acids (MUFA) have one double bond, and polyunsaturated fatty acids (PUFA) have more than one double bond. Within each of these categories, individual fatty acids (characterised by length of the hydrocarbon chain and the position of any double bonds) have different effects on blood lipids and lipoproteins.

Dietary fats circulate in the blood bound to lipoproteins. Low-density lipoprotein (LDL) cholesterol is the predominant form of circulating cholesterol. High-density lipoprotein (HDL) cholesterol forms a much smaller portion of circulating cholesterol. These lipoprotein sub-fractions have different implications for cardiovascular disease: LDL cholesterol increases the risk of cardiovascular disease, whereas HDL cholesterol is protective. Although the total (or LDL cholesterol) to HDL cholesterol ratio is a more accurate predictor of cardiovascular risk, total blood cholesterol (also referred to in this report as blood cholesterol) was selected for this study because it is more suitable for time series analyses.



### ***Saturated fatty acids and blood cholesterol***

Observational studies show a strong positive correlation between SFA intake and total blood cholesterol. By comparing SFA intake and blood cholesterol concentrations in Britain and Japan, Law (2000a) found that a one percentage point decrease in energy intake from SFA will lower blood cholesterol by 0.05 mmol/L in young adults and 0.1 mmol/L in older adults. However, this may be an overestimate, as the potentially confounding effects of physical activity, BMI and other dietary factors were not taken into account.

Dietary intervention trials provide more direct evidence than observational studies of the effect of changes in dietary fats on total blood cholesterol. Dietary intervention trials have consistently shown that increasing or decreasing the contribution of SFA to dietary energy is followed by a rise or fall in blood cholesterol. Although the change in blood cholesterol for a given change in SFA intake varies between individuals and for particular saturated fatty acids, for groups it is predictable and quantifiable (Department of Health [London] 1994).

A recent meta-analysis of 395 dietary intervention studies conducted under controlled conditions that ensured compliance (ie, metabolic ward studies) found that isocaloric replacement of 1 percent of energy from SFA with carbohydrate resulted in total blood cholesterol falling by 0.052 mmol/L in healthy volunteers (Clarke et al 1997). Isocaloric replacement of 1 percent of energy from carbohydrate with PUFA was found to lower total cholesterol by 0.026 mmol/L, whereas replacement of carbohydrate with MUFA resulted in a small (0.005 mmol/L) increase in cholesterol (Clarke et al 1997). Therefore, isocaloric replacement of SFA with PUFA would decrease blood cholesterol by a total of 0.078 mmol/L, and replacement of SFA with MUFA would decrease cholesterol by 0.047 mmol/L.

Given that SFA intake varies little with age, but produces blood cholesterol concentrations that increase with age, absolute reductions in blood cholesterol for a given decrease in SFA intake may be greater in older adults (Law 2000a).

### ***Saturated fatty acid intake in New Zealand***

Foods of animal origin (meat and dairy products) are the major sources of SFA in the New Zealand diet. In 1997 SFA provided 15.1 percent of energy in males and 14.7 percent of energy in females (Skeaff et al 2001). Current levels of SFA intake are substantially lower than in 1977, when SFA provided approximately 20 percent of energy in males and females (Birkbeck 1979), and lower than in 1989, when saturated fat provided 16.4 percent of energy in males and 16.1 percent of energy in females (Skeaff et al 2001).

SFA intake in New Zealand is considerably higher than in Australia (12.7 percent in 1995) (McLennan and Podger 1998) and the United States (12.0 percent in 1988–91) (McDowell et al 1994), reflecting our higher consumption of animal products (FAO 1996). SFA intake is also higher than the year 2000 target set by the New Zealand Department of Health of 8–12 percent of energy from SFA (Department of Health 1991). Further decreases in SFA intake in New Zealand are clearly both feasible and desirable.

### ***Approaches for reducing saturated fatty acid intake***

Different dietary approaches can be used to reduce SFA intake. SFA can be replaced by carbohydrate or unsaturated fatty acids (MUFA or PUFA). As already shown, replacing 1 percent of energy from SFA with carbohydrate lowers total cholesterol by 0.052 mmol/L, whereas replacing SFA with PUFA or MUFA lowers total cholesterol by 0.078 mmol/L and 0.047 mmol/L, respectively (Clarke et al 1997).

Despite the blood cholesterol-lowering effects of PUFA compared to carbohydrate, it is generally recommended that intakes of PUFA do not exceed 10 percent of energy because PUFA are subject to peroxidation in the artery wall, and this has adverse consequences with respect to cardiovascular disease (Department of Health [London] 1994). Currently, PUFA provide 4.9 percent of energy, which represents a small decrease since 1989 when they provided just over 5 percent of energy (Skeaff et al 2001). Therefore, New Zealanders could replace up to 5 percent of energy from SFA with PUFA. Major sources of PUFA are cooking or salad oils and margarines.

Replacing SFA with carbohydrates rich in non-starch polysaccharide may have benefits over and above those attributed to a reduction in SFA alone. Foods rich in non-starch polysaccharide (eg, whole-grain breads and cereals, vegetables and fruit) have been shown to lower blood cholesterol (see dietary fibre, below). Some experts argue against replacing SFA with carbohydrates because high carbohydrate intakes have been shown to decrease HDL cholesterol and increase triglycerides (Katan et al 1997). However, in free-living populations, replacing SFA with a variety of carbohydrate-rich foods often results in a small decrease in energy intake and body weight, which impacts favourably on blood lipid concentrations (see overweight, below).

The decrease in total cholesterol that occurs when SFA are replaced with MUFA or carbohydrate is similar, although compared to carbohydrates MUFA have more favourable effects on HDL cholesterol. Recommended MUFA intakes are generally in the range of 10–20 percent of energy. Currently, MUFA provide 12 percent of energy, which is a small decrease since 1989 when they provided approximately 13 percent of energy (Skeaff et al 2001).

Although altering the type, but not the amount, of dietary fat (ie, replacing SFA with unsaturated fats, particularly PUFA) significantly decreases total blood cholesterol concentrations, this approach may not be ideal in the control of obesity as high fat intakes have been implicated in the development of obesity (Bray and Popkin 1998). Therefore in people who are above their ideal weight or already have high fat intakes (the majority of New Zealanders), where possible, foods high in SFA should be replaced with foods rich in non-starch polysaccharides (eg, replace some meat and dairy products with vegetables, fruit, and whole-grain breads and cereals). Foods high in SFA that cannot be totally replaced with carbohydrate-rich foods should be replaced with their unsaturated or low-fat equivalents (eg, replace butter and lard with low trans fatty acid margarine and unsaturated oils, replace full-fat milk and dairy products with reduced or low-fat alternatives). Replacing 1 percent of energy from SFA with a combination of carbohydrate and unsaturated fat would result in a decrease in total cholesterol of approximately 0.06 mmol/L.

## **Dietary cholesterol and blood cholesterol**

Dietary cholesterol is a less important determinant of total blood cholesterol than SFA. Based on the meta-analysis by Clarke et al (1997), the predicted decrease in total blood cholesterol for a 10 mg decrease in dietary cholesterol is 0.007 mmol/L. Thus a decrease in dietary cholesterol of 100 mg/day (about one-third of current intake) would have the same effect on blood cholesterol as replacing 1 percent of energy from SFA with PUFA.

### ***Dietary cholesterol intake in New Zealand***

Foods of animal origin (meat, eggs and dairy products) are the major sources of dietary cholesterol in the New Zealand diet. In 1997 mean daily intakes of dietary cholesterol were 381 mg in males and 261 mg in females (Russell et al 1999), which represents a large decrease since 1977, when daily intakes were approximately 560 mg in males and 360 mg in females (Birkbeck 1979), but a smaller decrease since 1989 when daily intakes were 396 mg in males and 263 mg in females (Skeaff et al 2001). Because sources of dietary SFA and cholesterol are similar, any decreases in dietary SFA are likely to result in a simultaneous decrease in dietary cholesterol.

## **Dietary fibre**

Particular types of dietary fibre have long been thought to decrease blood cholesterol. A recent meta-analysis of 67 randomised trials testing the effects of dietary soluble fibre at feasible intakes (2–10 g/day) on blood cholesterol found that soluble fibre was associated with a small but significant decrease in total cholesterol (0.045 mmol/L decrease per 1 g/day increase in soluble fibre) (Brown et al 1999). Although some of the change in blood cholesterol may be attributed to changes in body weight or dietary composition (eg, the substitution of fibre for SFA and cholesterol), dietary fibre is thought to have an independent effect on blood cholesterol. We estimate this effect to be a decrease of approximately 0.015 mmol/L in blood cholesterol per 1 g/day increase in soluble fibre intake.

## **Body weight**

Excess body fat (adiposity), as indicated by a high BMI or waist circumference, may be an important determinant of blood lipids. In 1997, increasing BMI in New Zealand males was associated with a marked increase in total blood cholesterol (Skeaff et al 2001). For females, the association between BMI and total blood cholesterol was less striking and no stronger at BMI  $\geq 30$  than at BMI 25–30. A similar relationship was found in the United States, where data from the third National Health and Nutrition Examination Survey (NHANES III, 1988–94) showed that blood cholesterol concentrations were consistently lower at BMI  $< 25$  for both males and females, but for females did not increase consistently with increasing BMI above 25 (Brown et al 2000). Possible reasons for the sex difference include a protective effect of oestrogen on blood cholesterol that is stronger than the variability in BMI in females, or the tendency for females to accumulate body fat on their lower body. Lower body fat is less strongly associated with blood lipid profiles than abdominal body fat, which is the predominant site of body fat accumulation in males.

Clinical trials involving weight loss have shown that reducing excess weight decreases total blood cholesterol. Although the decrease in total cholesterol is often small, it masks more important changes to cholesterol sub-fractions, such as an increase in HDL cholesterol. Quantifying the effects of weight loss on blood cholesterol can be difficult, given that weight loss is generally the result of multiple dietary changes. A meta-analysis of 70 studies investigating the effects of weight loss on total blood cholesterol demonstrated that for every kilogram decrease in body weight there was a 0.05 mmol/L decrease in total blood cholesterol (Dattilo and Kris-Etherton 1992). However, it is likely that weight loss diets were lower in total fat, SFA and cholesterol, so the decrease in total blood cholesterol due to weight loss alone is likely to be much smaller (we estimate 0.02 mmol/L).

Mean body weight, BMI and the prevalence of obesity increased substantially in New Zealand from 1989 to 1997 (Russell et al 1999). Body weight is likely to continue increasing over the next decade, which may impact unfavourably on declining trends in total blood cholesterol concentrations.

## Physical activity

A number of observational studies have found an inverse association between physical activity levels and blood cholesterol. A recent review of 51 clinical trials to determine the effect of moderate to vigorous intensity aerobic exercise on blood lipids found that the most common change was an increase in HDL cholesterol, with reduction in total cholesterol, LDL cholesterol and triglycerides being less common (Leon and Sanchez 2001).

## Summary

Summary estimates of the expected decrease in total blood cholesterol following a change in the key determinants of this risk factor are shown in Table 38. Since a reduction in SFA intake (ie, animal fat) is by far the most important determinant of total blood cholesterol, this dietary change forms the basis of scenarios to be discussed later in this chapter.

**Table 38:** Decrease in blood cholesterol for a change in key determinants

Determinant	Current level	Change in determinant	Decrease in TC
Saturated fatty acids	15% energy	↓ 1% energy (replace with carbohydrate) ↓ 1% energy (replace with MUFA <sup>1</sup> ) ↓ 1% energy (replace with PUFA <sup>2</sup> )	0.052 mmol/L 0.047 mmol/L 0.078 mmol/L
Dietary cholesterol	319 mg	↓ 10 mg	0.007 mmol/L
Soluble dietary fibre	10 g	↑ 1 g	0.015 mmol/L
Body weight	74.5 kg	↓ 1 kg	0.02 mmol/L

1 Monounsaturated fatty acids

2 Polyunsaturated fatty acids

## 5.3 Blood cholesterol distributions

### Current distribution

Data on current total blood cholesterol distributions (mean and standard deviation) were extracted from the 1997 NNS dataset. Total blood cholesterol measurements were made on a single blood sample using enzymatic methods. Methods complied with International Accreditation New Zealand approved quality control procedures. Current distributions of total blood cholesterol in New Zealand by age group, sex and ethnicity, are shown in Tables 39 and 40.

**Table 39:** Mean total blood cholesterol (mmol/L), by age group, sex and ethnicity, 1997

	Sex	Age group (years)								
		15–24	25–34	35–44	45–54	55–64	65–74	75+	Total	Adjusted <sup>1</sup>
Non-Māori	Males	4.7	5.3	5.9	6.2	6.3	6.1	5.8	5.7	5.6
	Females	4.9	5.2	5.6	6.0	6.5	6.8	6.8	5.8	5.7
Māori	Males	5.3	6.1	6.4	6.5	6.3	5.9	5.2	6.0	6.0
	Females	4.7	5.0	5.5	5.9	6.4	6.1	5.8	5.3	5.4
Total	Males	4.8	5.4	5.9	6.2	6.3	6.1	5.8	5.7	5.6
	Females	4.9	5.2	5.6	6.0	6.5	6.8	6.8	5.7	5.7

<sup>1</sup> Age-standardised to WHO World population

**Table 40:** Standard deviation for total blood cholesterol (mmol/L), by age group, sex and ethnicity, 1997

	Sex	Age group (years)								
		15–24	25–34	35–44	45–54	55–64	65–74	75+	Total	Adjusted <sup>1</sup>
Non-Māori	Males	1.0	1.0	1.1	1.2	0.9	1.0	1.1	1.2	1.0
	Females	1.0	1.0	1.1	1.1	1.2	1.2	1.3	1.3	1.1
Māori	Males	0.9	1.6	1.3	1.5	1.4	1.0	1.1	1.4	1.3
	Females	0.7	0.8	0.8	0.8	0.8	0.8	1.0	1.0	0.8
Total	Males	1.0	1.2	1.2	1.2	1.0	1.0	1.1	1.2	1.1
	Females	0.9	1.0	1.1	1.1	1.1	1.2	1.3	1.3	1.0

<sup>1</sup> Age-standardised to WHO World population.

### Trends and international comparisons

Total blood cholesterol concentrations have been declining in developed countries for the last two to three decades. In New Zealand, total blood cholesterol concentrations in adults aged 15 years and older decreased from 5.9 mmol/L in 1989 to 5.7 mmol/L in 1997 (Russell et al 1999). The decrease in total blood cholesterol was greater in females (6.0 to 5.7 mmol/L) than in males (5.8 to 5.7 mmol/L).

Although total blood cholesterol levels are declining in New Zealand, they are still considerably higher than the recommended range of 3.0 to 5.0 mmol/L (Dyslipidaemia Advisory Group 1996). They are also relatively high compared to other similar countries (Table 41).

**Table 41:** International comparisons of mean total blood cholesterol and prevalence of high cholesterol

Country, year	Total blood cholesterol (mmol/L)		High cholesterol* (%)	
	Males	Females	Males	Females
New Zealand, 1997	5.7	5.7	23.2	23.7
England, 1998	5.5	5.6	18.0	22.4
Scotland, 1995	5.6	5.6	22.0	21.0
US, 1988–94	5.3	5.3	18.8	20.5
Australia, 1999/2000	5.5	5.4	–	–

\*  $\geq 6.5$  mmol/L for all except the United States where  $\geq 6.2$  mmol/L

Sources: Russell et al 1999; Department of Health [London] 1999; The Scottish Office Department of Health 1997; National Center for Health Statistics 2002; AIHW 2001.

In Australia, total blood cholesterol concentrations in adults aged 25–64 years declined from 5.66 to 5.50 mmol/L in males and from 5.55 to 5.41 mmol/L in females between 1989 and 1999/2000 (AIHW 2001). In the United States, total blood cholesterol decreased from 5.5 to 5.3 mmol/L in males and from 5.6 to 5.3 mmol/L in females from 1976–80 to 1988–94 (National Center for Health Statistics 2002). From 1994 to 1998, total blood cholesterol concentrations in English adults aged 16 years and older declined from 5.8 to 5.5 mmol/L in males and from 6.0 to 5.6 mmol/L in females (Department of Health [London] 1999). In 1995, total blood cholesterol concentrations in Scotland were 5.6 mmol/L in both males and females aged 16–64 years (The Scottish Office Department of Health 1997).

The decline in total blood cholesterol observed in many developed countries is largely due to a decrease in total and saturated fat intake, and to a lesser extent a decrease in dietary cholesterol intake. In the United States, declines in total blood cholesterol concentrations between 1960 and 1990 parallel decreases in dietary total and saturated fat and dietary cholesterol (Ernst et al 1997).

Dietary changes that have contributed to the decrease in total and saturated fat intake include reduced consumption of meat, eggs and full-fat dairy products. Improved diagnosis and management of people with ‘clinically’ high blood cholesterol is also likely to have contributed to the decline in population blood cholesterol levels.

Based on our estimates, reported dietary changes between the 1989 and 1997 national nutrition surveys explain only 25 percent of the observed decline in total cholesterol in New Zealand over this period. However, total fat intake decreased by about three energy percentage points over the same period, suggesting that total fat intake may be important, in addition to the types of dietary fat. Inaccuracies in self-reported food intakes might also have led to underestimation of the impact of dietary change. Pharmacological treatment may have had a small impact, with a large increase in the use of statins over the period concerned (Metcalf and Moodie 2002).

## Theoretical minimum

The theoretical minimum is the risk factor (ie, total blood cholesterol) distribution that would yield the lowest population risk of adverse health outcomes. In developed countries, the relationship between total blood cholesterol and IHD is continuous, with no evidence of a lower threshold, although the relationship has seldom been demonstrated below a cholesterol level of 4.5–5.0 mmol/L in European and North American populations (Chen et al 1991). In contrast, in countries such as China, there is evidence that this relationship continues, without a threshold, to cholesterol concentrations less than 4.0 mmol/L (Chen et al 1991; Law and Wald 1994; Law et al 1994).

Populations with little or no cardiovascular disease provide an alternative source of data on the cholesterol concentration associated with the lowest risk of disease. In hunter-gatherer societies, mean cholesterol concentrations are as low as 3.0–3.5 mmol/L in males aged 45–64 years. In contrast, mean cholesterol concentrations in males aged 45–64 years in Western populations range from 5.5 to 7.0 mmol/L (Law and Wald 1994; Law 1999).

Taken together, these data suggest that a cholesterol concentration of 3.5–4.0 mmol/L would be an appropriate theoretical minimum for all age groups. For this study, calculations of attributable and avoidable mortality have been based on a theoretical minimum cholesterol concentration of  $3.8 \pm 0.5$  mmol/L for all age, sex and ethnic groups. This theoretical minimum is based on that chosen by the WHO cholesterol expert working group for the *World Health Report 2002* (Ezzati et al 200; Lawes et al, in press).

## Distributional transitions

Two distributional transitions for blood cholesterol were estimated: a business as usual (BAU) scenario (historical trend) and an intervention scenario (deviation from the historical trend reflecting policy change). Distributional transitions were based on trends in total blood cholesterol in New Zealand and other developed countries, as well as trends in key determinants of cholesterol.

Distributional transitions are expressed as a percentage shift from the current blood cholesterol distribution towards the theoretical minimum distribution. For blood cholesterol, distributional transitions were greater for females than males, but the same for all age and ethnic groups. However, absolute changes in blood cholesterol vary by age and ethnicity as well as by sex, due to differences in baseline cholesterol levels.

The avoidable burden is the difference between the projected BAU scenario and the intervention scenario, with the shift between the two scenarios being maintained over time.

### ***Business as usual scenario***

Given that current evidence suggests that total cholesterol concentrations will continue to decline, under the BAU scenario we estimated an overall decrease in mean population total blood cholesterol of 0.10 mmol/L in males and 0.15 mmol/L in females by 2011. These declines in the total blood cholesterol are equivalent to a shift in the current blood cholesterol distribution towards the theoretical minimum of 5.2 percent in males and 7.8 percent in females. Absolute changes in blood cholesterol vary by age, ranging from 0.05 to 0.13 mmol/L in males and 0.09 to 0.23 mmol/L in females. Table 42 shows projected total blood cholesterol levels in 2011 under the BAU scenario, by ethnicity, sex and age.

**Table 42:** Projected 2011 total blood cholesterol levels under the business as usual scenario

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Non-Māori	Males	5.2	5.8	6.1	6.2	6.0	5.7	5.6
	Females	5.1	5.5	5.8	6.3	6.6	6.6	5.6
Māori	Males	6.0	6.3	6.4	6.2	5.8	5.1	5.9
	Females	4.9	5.4	5.7	6.2	5.9	5.6	5.2
Total	Males	5.3	5.8	6.1	6.2	6.0	5.7	5.6
	Females	5.1	5.5	5.8	6.3	6.6	6.6	5.6

### ***Intervention scenario***

Under the more optimistic intervention scenario, we estimated a decrease in mean population total blood cholesterol of approximately 0.20 mmol/L in males and 0.25 mmol/L in females by 2011 (approximately 0.1 mmol/L over and above the BAU trend in males and females). These declines in the mean population total blood cholesterol are equivalent to shifts in the current blood cholesterol distribution towards the theoretical minimum of 9.1 percent in males and 11.7 percent in females. Absolute changes in blood cholesterol vary by age, ranging from 0.15 to 0.23 mmol/L in males and 0.16 to 0.35 mmol/L in females. Table 43 shows projected total blood cholesterol levels in 2011 under the intervention scenario, by ethnicity, sex and age.

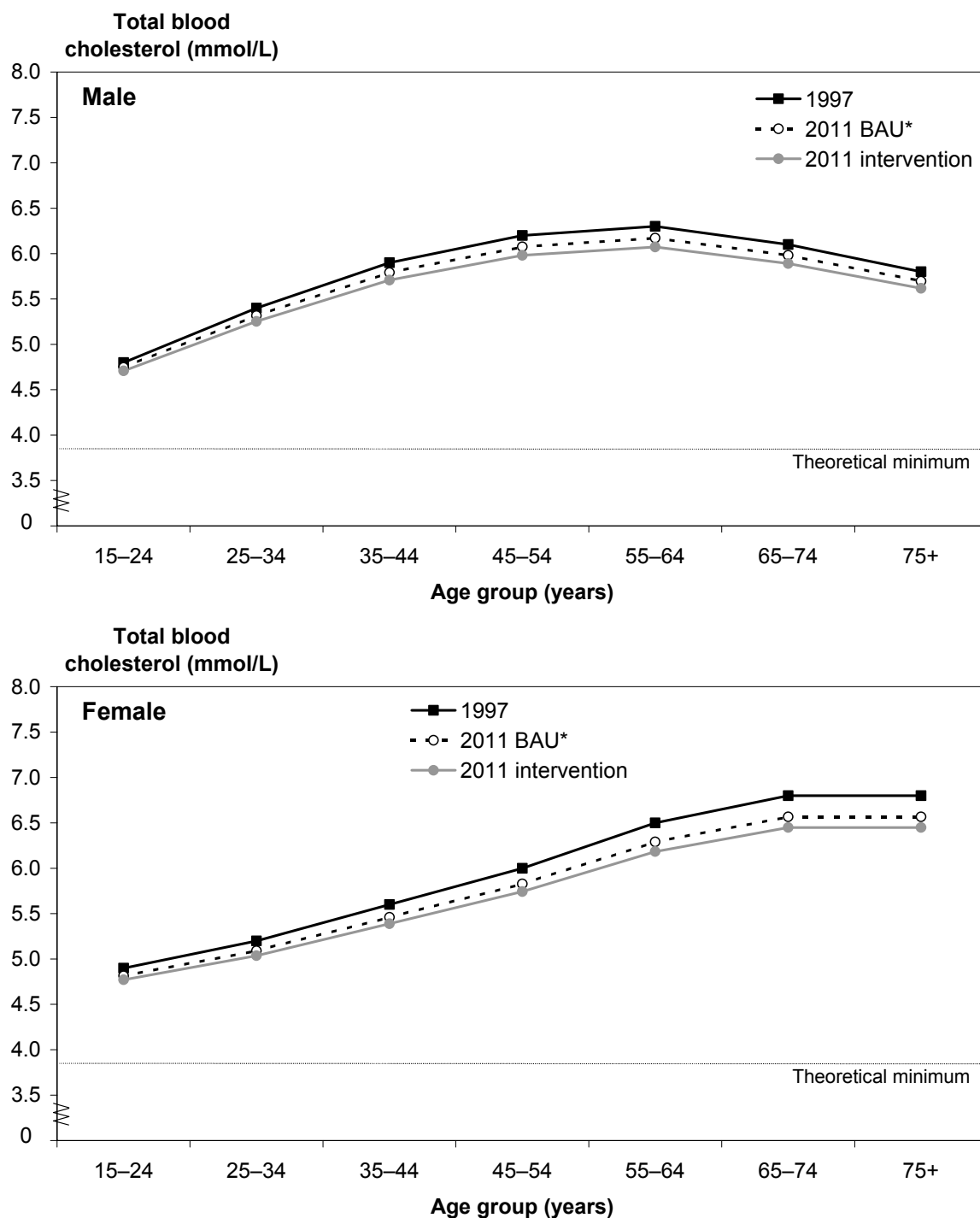
**Table 43:** Projected 2011 total blood cholesterol levels under the intervention scenario

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Non-Māori	Males	5.2	5.7	6.0	6.1	5.9	5.6	5.5
	Females	5.0	5.4	5.7	6.2	6.4	6.4	5.6
Māori	Males	5.9	6.2	6.3	6.1	5.7	5.1	5.8
	Females	4.9	5.3	5.7	6.1	5.8	5.6	5.1
Total	Males	5.3	5.7	6.0	6.1	5.9	5.6	5.5
	Females	5.0	5.4	5.7	6.2	6.4	6.4	5.5



Figure 17 shows current (1997) blood cholesterol levels, as well as projected 2011 blood cholesterol levels under the business as usual and intervention scenarios (separately for males and females) in relation to the theoretical minimum.

**Figure 17:** Current (1997) and projected future (2011) total blood cholesterol levels



\* BAU = business as usual

## 5.4 Risk factor–disease relationships

### Disease outcomes

The outcomes assessed were based on those selected for the *World Health Report 2002* (WHO 2002):

- ischaemic heart disease
- stroke.

### Risk accumulation

Risk accumulation refers to the nature and strength of the association between an exposure (ie, total blood cholesterol) and disease. Risk accumulation is expressed as the regression (risk) coefficient in the case of a continuous risk factor (ie, the increase in incidence or mortality of the disease per unit increase in exposure).

Four major overviews of observational studies investigating the relationship between cholesterol and cardiovascular disease (IHD and stroke) have been undertaken (Law et al 1994; Prospective Studies Collaboration 1995; Eastern Stroke and Coronary Heart disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration, in press).

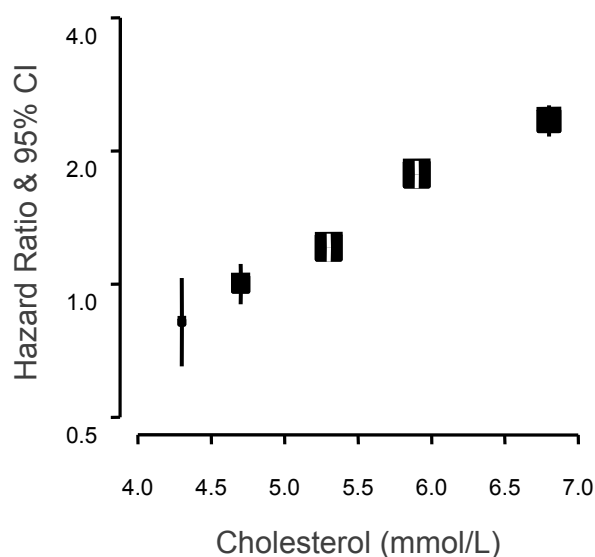
For cholesterol and IHD, we used the Law et al (1994) meta-analysis for risk accumulation estimates. This meta-analysis combined data on 494,804 participants (all male) aged 35–84 years from 10 cohorts. During the follow-up period (7–23 years), there were 18,811 IHD events.

For cholesterol and ischaemic stroke, we used the Asia Pacific Cohort Studies Collaboration (APCSC) overview for risk accumulation estimates. The APCSC study combined data from 29 prospective cohorts from eight countries in the Asia–Pacific region, including three cohorts from Australia and one from New Zealand. A major advantage of the APCSC overview was that it included individual participant data and was therefore able to provide more reliable estimates of risk accumulation for different age groups and was better able to control for confounding. The APCSC overview included data on 353,065 participants (57 percent male) aged 20–107 years. During the follow-up period (mean seven years), there were 2937 stroke events and 2838 IHD events.

### Ischaemic heart disease

Data from meta-analyses show that the risk of IHD increases with increasing blood cholesterol concentration, and that the association is roughly linear when plotted on a log scale (Law et al 1994, Asia Pacific Cohort Study Collaboration, in press). In almost all cohorts, the association was continuous, with no evidence of a lower threshold below which lower levels of cholesterol are not associated with a lower risk of IHD (down to almost 4.0 mmol/L) (Figure 18). There is no evidence of an upper threshold either.

**Figure 18:** Usual total blood cholesterol and ischaemic heart disease (ages pooled)



Source: Redrawn from published data in Asia Pacific Cohort Studies Collaboration, Cholesterol, coronary heart disease and stroke in the Asia Pacific region, in press.

As shown in Table 44, the association between blood cholesterol and IHD varies with age: it is strongest in young adults and then attenuates with age. Although none of the cohorts in the Law et al meta-analysis included females, the APCSC data provide no evidence that the strength of association between blood cholesterol and IHD varies by sex and therefore it was not considered necessary to have different risk estimates for males and females. It was assumed that the relationship between blood cholesterol and IHD was the same for all ethnic groups.

A 1 mmol/L lower total blood cholesterol was associated with a 73 percent lower risk of IHD in younger adults, dropping to a 30 percent lower risk in adults aged 75 years and over.

**Table 44:** Risk coefficients for ischaemic heart disease for a 1 mmol/L lower total blood cholesterol

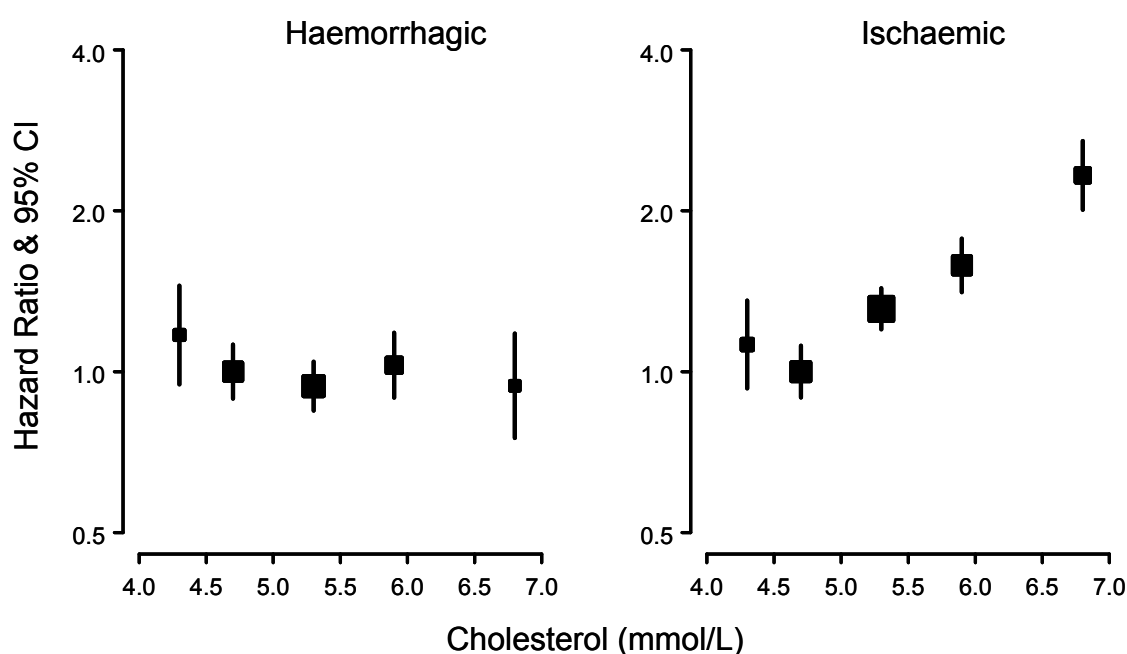
Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.27	73
35–44	0.27	73
45–54	0.48	52
55–64	0.56	44
65–74	0.67	33
75+	0.70	30

Source: WHO cholesterol expert working group.

### Ischaemic stroke

The relationship between blood cholesterol and stroke is more complex, largely due to a quantitatively different association between blood cholesterol and the two major types of stroke: haemorrhagic and ischaemic. While the APCSC data indicate there is a positive association between blood cholesterol and ischaemic stroke, there is a negative or null association between blood cholesterol and haemorrhagic stroke (Figure 19). Therefore, for this study the disease endpoint was restricted to ischaemic stroke mortality. Estimates of the proportion of stroke sub-types occurring in developed regions were used to estimate the proportion of total stroke deaths that were ischaemic stroke in the New Zealand population (Lawes et al, in press). Based on this data, ischaemic stroke accounts for approximately two-thirds of total stroke mortality overall, ranging from 43 percent in the youngest age group to 70 percent in the oldest age group.

**Figure 19:** Usual total blood cholesterol and risk of stroke (ages pooled)



Source: Redrawn from published data in Asia Pacific Cohort Studies Collaboration, Cholesterol, coronary heart disease and stroke in the Asia Pacific region, in press

As shown in Table 45, the association between blood cholesterol and ischaemic stroke varies with age: it is strongest in young adults and then attenuates with age. As the APCSC data provide no evidence that the strength of association between blood cholesterol and ischaemic stroke varies by sex, it is not necessary to have different risk estimates for males and females. It was assumed that the relationship between blood cholesterol and ischaemic stroke was the same for all ethnic groups.

A 1 mmol/L lower total blood cholesterol was associated with a 31 percent lower risk of ischaemic stroke in younger adults, dropping to a 16 percent lower risk in adults aged 75 years and over.

**Table 45:** Risk coefficients for ischaemic stroke for a 1 mmol/L lower total blood cholesterol

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.69	31
35–44	0.69	31
45–54	0.65	35
55–64	0.70	30
65–74	0.83	17
75+	0.84	16

Source: Asia Pacific Cohort Studies Collaboration

## Risk reversal

Risk reversal refers to how quickly and how completely risk of cardiovascular disease is reversed following cholesterol lowering. Randomised controlled trials provide information on risk reversal. A vast number of trials have studied the impact of cholesterol lowering on cardiovascular disease. However, individual studies usually lack sufficient power to reliably detect moderate changes in events. Results from overviews (meta-analyses) are therefore more reliable. It was assumed that risk reversal was the same for both sexes and all ethnic groups.

### *Ischaemic heart disease*

Overviews of cholesterol-lowering trials clearly demonstrate that a reduction in blood cholesterol reduces IHD mortality. A major overview of 28 trials (45,254 individuals, and 4421 IHD deaths) by Law et al (1994) demonstrated that a reduction in blood cholesterol was followed by a reduction in IHD risk of similar magnitude to that predicted based on data from observational studies (a 45 percent decrease in risk of IHD for a 1 mmol/L decrease in blood cholesterol), but only after five years. By that time, almost all the risk of IHD associated with high blood cholesterol had been reversed. The effects of cholesterol lowering on risk of IHD appear similar in males and females (Law et al 1994; LaRosa et al 1999; Byington et al 2001).

Several other overviews have also demonstrated that cholesterol lowering reduces the risk of IHD (Bucher et al 1998; LaRosa et al 1999; Ross et al 1999; Pignone et al 2000). However, unlike the meta-analysis by Law et al (1994), these overviews do not report IHD events according to duration of treatment, which is vital if the timing and extent of risk reversibility are to be assessed.

In summary, our risk-reversal estimate is that blood cholesterol lowering is associated with a complete (100 percent) reversal of IHD risk after five years, whatever the initial risk level. This risk-reversal summary is based on that chosen by the WHO cholesterol expert working group for the *World Health Report 2002*, details of which will be published in a forthcoming technical report (Lawes et al, in press).

### **Ischaemic stroke**

Overviews of early observational studies and trials did not find an association between blood cholesterol lowering and subsequent risk of stroke, possibly because they considered total stroke rather than assessing ischaemic and haemorrhagic stroke separately. Because early trials only achieved small reductions in blood cholesterol, and included few stroke events (which were mostly fatal), any effect of cholesterol lowering on the risk of stroke would have been difficult to detect (Crouse et al 1998; Law 1999).

The larger overviews of more recent trials have found that blood cholesterol lowering reduces the risk of stroke (Blauw et al 1997; Crouse et al 1997; Hebert et al 1997; Crouse et al 1998). The association between blood cholesterol and stroke appears to be stronger for non-fatal stroke (which comprises a higher proportion of ischaemic strokes) than fatal stroke (Blauw et al 1997; Ross et al 1999; Byington et al 2001). The effects of cholesterol lowering on risk of stroke appear similar in males and females (Law et al 1994; LaRosa et al 1999; Byington et al 2001).

Our summary risk-reversal estimate is that blood cholesterol lowering is associated with a complete (100 percent) reversal of the initial stroke risk after five years. This risk-reversal estimate is based on that chosen by the WHO cholesterol expert working group for the *World Health Report 2002*, details of which will be published in a forthcoming technical report (Lawes et al, in press). It was assumed that risk reversal is the same for both sexes and all ethnic groups.

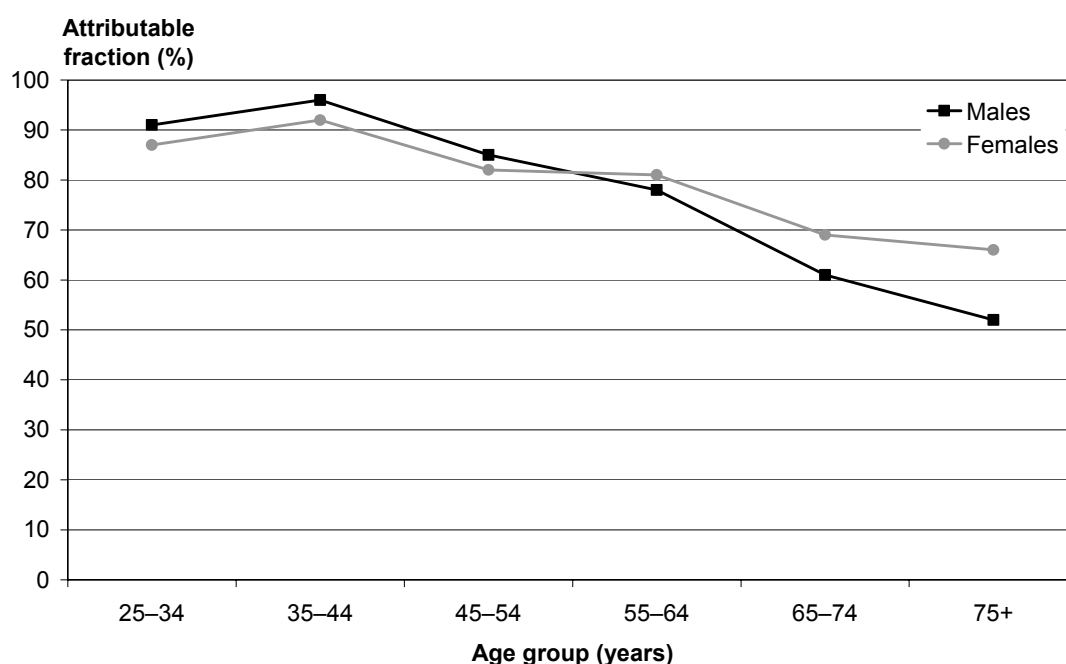
## **5.5 Attributable mortality**

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### **Ischaemic heart disease**

In the two oldest age groups, where the majority of IHD deaths occur, approximately 60 percent of IHD was attributable to 'high' blood cholesterol (Figure 20). The very high attributable fractions for blood cholesterol and IHD reflect the strong relationship between blood cholesterol and IHD, as well as the fact that almost the entire population has cholesterol concentrations exceeding the theoretical minimum (3.8 mmol/L).

**Figure 20:** Attributable fractions (%) for total blood cholesterol and ischaemic heart disease, by sex, 1997



Overall, 4096 (95% CI 3919–4246) IHD deaths in 1997 (15 percent of all deaths) were attributable to ‘high’ blood cholesterol (Table 46). The majority (almost 80 percent) of IHD deaths attributable to ‘high’ blood cholesterol occurred in those aged 65 years and older. IHD mortality due to ‘high’ blood cholesterol contributed to 40,511 YLL in 1997.

The age-standardised IHD mortality rate attributable to ‘high’ blood cholesterol was almost twice as high in males as in females (Table 46). This difference reflects the higher IHD mortality in males, as attributable fractions were similar in males and females. IHD mortality attributable to ‘high’ blood cholesterol was approximately 50 percent higher in Māori than in non-Māori. This difference primarily reflects higher IHD mortality in Māori compared to non-Māori, as attributable fractions were similar in both groups.

**Table 46:** Attributable mortality for total blood cholesterol and ischaemic heart disease, 1997

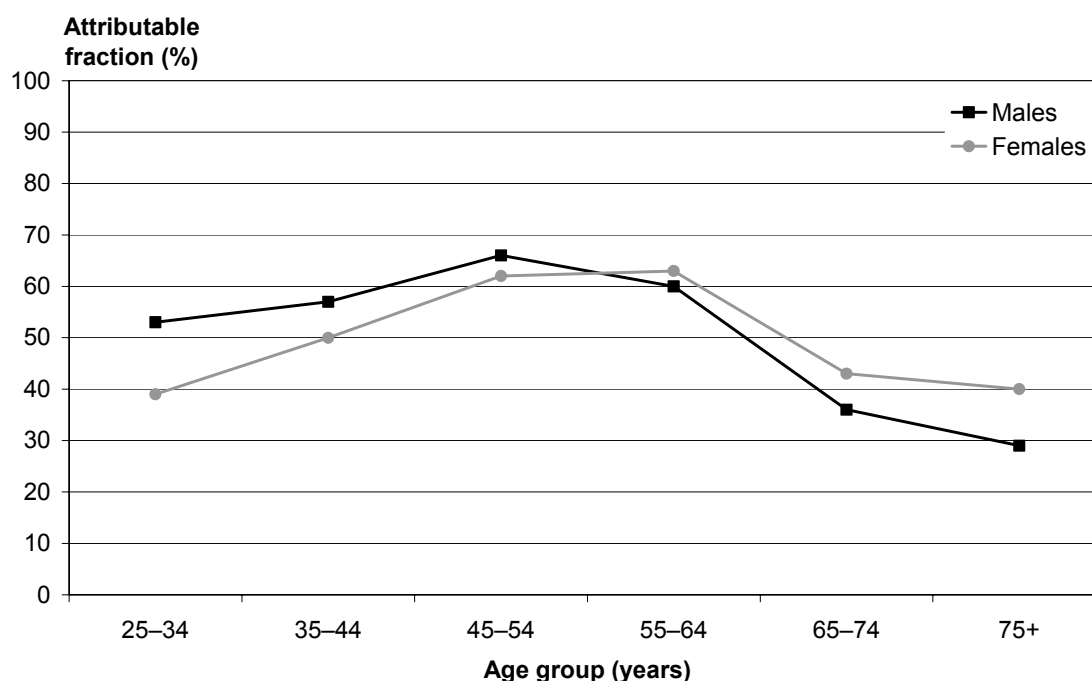
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	1,992	20,002	170.8	1,798
	Māori	214	3,312	282.7	3,779
	Total	2,206	23,314	180.5	1,970
Females	Non-Māori	1,774	15,593	98.1	948
	Māori	116	1,604	156.8	1,939
	Total	1,890	17,197	102.8	1,030

\* Rate per 100,000, age-standardised to WHO World population.

## Ischaemic stroke

In the two oldest age groups, where the majority of stroke deaths occur, 30–35 percent of all ischaemic strokes were attributable to ‘high’ blood cholesterol (Figure 21).

**Figure 21:** Attributable fractions (%) for total blood cholesterol and ischaemic stroke, by sex, 1997



Overall, 625 (95% CI 286–870) ischaemic stroke deaths in 1997 (2 percent of all deaths) were attributable to ‘high’ blood cholesterol (Table 47). The majority (almost 80 percent) of ischaemic stroke deaths attributable to ‘high’ blood cholesterol occurred in those aged 75 years and older. Ischaemic stroke mortality due to ‘high’ blood cholesterol contributed to 5171 YLL in 1997.

The age-standardised ischaemic stroke mortality rate attributable to ‘high’ blood cholesterol was slightly higher in females than in males, and similar in Māori and non-Māori (Table 47).

**Table 47:** Attributable mortality for blood cholesterol and ischaemic stroke, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	197	1,537	16.4	132
	Māori	10	131	16.7	174
	Total	207	1,668	16.6	137
Females	Non-Māori	404	3,318	21.2	187
	Māori	14	185	19.5	231
	Total	418	3,503	21.5	194

\* Rate per 100,000, age-standardised to WHO World population.



## Total attributable mortality

Overall, 'high' blood cholesterol contributed to a total of 4721 deaths (17 percent of all deaths) and 45,682 YLL in 1997 (Table 48).

**Table 48:** Attributable mortality for total blood cholesterol and all diseases, 1997

Sex	Ethnicity	Deaths		Years of life lost	
		Count	%*	Count	%*
Males	Non-Māori	2,189	17	21,539	16
	Māori	224	16	3,443	14
	Total	2,413	17	24,982	16
Females	Non-Māori	2,178	18	18,911	16
	Māori	130	11	1,789	9
	Total	2,308	17	20,700	15

\* % = percentage of all deaths and years of life lost, 1997.

## 5.6 Avoidable mortality

Estimates of IHD and stroke mortality in 2011 were forecast based on trends since 1976. As IHD and, to a lesser extent, stroke mortality declined steadily during this period for all birth cohorts, the forecasts indicate a continuation of this downward trend. However, it is possible that the obesity epidemic will slow this downward trend in years to come, meaning these forecasts may be somewhat optimistic. As a result, we may have underestimated avoidable mortality for 2011.

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### Ischaemic heart disease

With a decrease in total blood cholesterol as outlined under the intervention scenario (a reduction of 0.1 mmol/L above the BAU scenario), 5–10 percent of ischaemic heart disease deaths would be avoided. Approximately 6 percent of deaths would be avoided in the 75+ years age group. This shift in total blood cholesterol levels will mean that 261 IHD deaths and 2371 YLL could be avoided each year from 2011 (Table 49), as compared with the mortality expected under the BAU scenario.

**Table 49:** Avoidable mortality\* for total blood cholesterol and ischaemic heart disease, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	140	1,221
	Māori	23	297
	Total	163	1,518
Females	Non-Māori	89	743
	Māori	10	109
	Total	98	852

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

## Stroke

With a decrease in total blood cholesterol as outlined under the intervention scenario (a reduction of 0.1 mmol/L over and above the BAU scenario), approximately 2–6 percent of ischaemic stroke deaths would be avoided. This shift in total blood cholesterol levels would mean that 39 stroke deaths and 300 YLL could be avoided each year from 2011 (Table 50).

**Table 50:** Avoidable mortality\* for total blood cholesterol and stroke, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	15	100
	Māori	1	14
	Total	16	114
Females	Non-Māori	22	172
	Māori	1	14
	Total	23	186

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

## Total avoidable mortality

If total blood cholesterol levels were reduced as outlined under the intervention scenario, a total of 300 deaths and 2670 YLL could be avoided each year from 2011 (Table 51).

**Table 51:** Avoidable mortality\* for blood cholesterol and all diseases, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	155	1,321
	Māori	24	311
	Total	179	1,632
Females	Non-Māori	111	915
	Māori	11	123
	Total	121	1,038

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

## 5.7 Discussion

We estimate that in 1997 'high' blood cholesterol contributed to 4721 deaths (17 percent of all deaths) (Table 52). Although only a small incremental decrease in mean population blood cholesterol is proposed under the intervention scenario, this translates into significant decreases in both IHD and stroke mortality. If relevant policy initiatives were introduced now, such a change could prevent approximately 300 deaths each year from 2011. Some reduction in mortality would be seen even earlier as the benefits of cholesterol lowering are seen within two to three years.

**Table 52:** Summary of attributable and avoidable mortality due to 'high' blood cholesterol

	Attributable mortality (1997)		Avoidable mortality (2011)*	
	Deaths (count)	Years of life lost (count)	Deaths (count)	Years of life lost (count)
Ischaemic heart disease	4,096	40,511	261	2,371
Stroke	625	5,171	39	300
Total	4,721	45,682	300	2,670

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

Although a single intervention scenario has been modelled here, the effect of alternative scenarios can be readily estimated. The relationship between the distributional transition and the avoidable burden is approximately linear. Therefore, doubling the total blood cholesterol shift (from 4 percent to 8 percent beyond the BAU scenario) would double the avoidable burden (from 300 to 600 deaths in 2011).

## Policy implications

The avoidable burden was calculated by shifting the current cholesterol distribution towards the theoretical minimum. This shift resulted in a 0.1 mmol/L decrease in mean population total blood cholesterol by 2011 over and above the BAU scenario. The shift in the total blood cholesterol distribution was higher in the older age groups (up to 0.4 mmol/L reduction) due to higher blood cholesterol values in this group.

Two possible policy approaches for achieving the blood cholesterol reductions outlined under the intervention scenario have been modelled:

- a general shift toward the *Guidelines* diet (combined option)
- a specific effort to reduce butter consumption (butter option).

Of course these initiatives are by no means mutually exclusive. Other options for reducing blood cholesterol also exist, but are not discussed here.

### **Combined option**

A range of dietary and lifestyle factors could reduce blood cholesterol. Promoting the *Food and Nutrition Guidelines for Healthy Adults* (Ministry of Health 2002b) is one way to achieve the total cholesterol reduction outlined under the intervention scenario (a further 0.1 mmol/L reduction in mean population blood cholesterol over and above the BAU scenario). The *Guidelines* recommend eating foods low in fat, eating plenty of vegetables and fruit, as well as breads and cereals (preferably wholemeal and whole-grain), and maintaining a healthy body weight. The recommendation to eat foods low in fat would involve preparing foods with minimal added fat (eg, trimming visible fat off meat, choosing low-fat cooking methods, and using spreads sparingly) and choosing pre-prepared foods, and snacks that are low in fat, especially saturated fat.

If more New Zealanders followed the *Guidelines* diet, there would be a decrease in dietary SFA and cholesterol intake, an increase in soluble fibre and PUFA intake, and little or no increase in the prevalence of overweight and obesity. A reduction in SFA intake of one energy percentage point (eg, from 15 to 14 percent of energy, a relative decrease of 6.7 percent) would lower blood cholesterol by approximately 0.06 mmol/L (assuming SFA were replaced by a combination of carbohydrate and unsaturated fats). Reducing SFA intake to this extent is likely to also reduce dietary cholesterol intake by about 20 mg/day (6.3 percent), which would lower blood cholesterol by a further 0.014 mmol/L. Increasing intakes of foods rich in non-starch polysaccharide could lower blood cholesterol even further (ie, 0.015 mmol/L for every 1 g/day increase in soluble fibre). It is unlikely that the prevalence of overweight could be reduced in the short term, however. A more realistic aim would be to limit further increases in body weight. Assuming these changes are additive, the overall impact would be a reduction in blood cholesterol similar to that outlined under the intervention scenario. Improved diagnosis and management of people with 'clinically' high blood cholesterol may also contribute to a change in the population blood cholesterol distribution.

### **Butter option**

Another option for achieving the blood cholesterol reduction outlined under the intervention scenario would be to encourage more people to replace butter with margarine. Our use of butter as a spread is high compared to other Western countries. Almost all New Zealand adults (92 percent) use spreads on bread and crackers, and 50 percent of these use butter (Russell et al 1999). In Finland, the proportion of adults using butter on bread has decreased from approximately 60 to 10 percent between 1978 and 1998 (Lahti-Koski 2000). The decline in the use of butter as a spread in Finland is largely the result of community-based health promotion projects, the first of which was the North Karelia Project. The success of the North Karelia Project suggests that it is feasible to decrease butter consumption at a population level.

A key argument against substituting margarine for butter is that it may lead to increased consumption of trans fatty acids (TFA), a category of unsaturated fatty acids that have adverse effects on blood lipid profiles. Hydrogenation of unsaturated fatty acids to produce margarine is the main source of TFA. Margarines vary in their content of TFA: soft tub margarines tend to be lower in TFA than hard stick margarines used in confectionery and bakery products. Most tub margarines available in New Zealand are very low in TFA (Lake et al 1995). Based on a meta-analysis of 20 dietary intervention studies, replacing 1 percent of energy from butter with low TFA soft tub margarine lowers total cholesterol by 0.025 mmol/L (Zock and Katan 1997). Both butter and margarine contain a combination of SFA, MUFA and PUFA, but in varying proportions. As the exact composition of the butters and margarines in the Zock and Katan meta-analysis are not known, it is not possible to directly compare the reported decreases in total cholesterol with estimates obtained using the Clarke et al (1997) predictive equation. However, based on the composition of butter and soft tub margarine in New Zealand (Athar et al 2003), the predicted decrease in total cholesterol following replacement of 1 percent of energy from butter with margarine is approximately 0.035 mmol/L.

In New Zealand, 6 percent of total energy came from the butter and margarine food group in 1997 (LINZ® Activity and Health Research Unit 1999). However, the percentage energy from butter and margarine is likely to be higher than this estimate, given that these products are also ingredients in a number of other food groups, including cakes and muffins, bread-based dishes, biscuits, pies and pasties. Therefore, we estimate that butter *alone* is likely to provide approximately 6 percent of total energy in the typical New Zealand diet.

Based on the more conservative Zock and Katan (1997) estimate, to decrease mean population blood cholesterol concentrations by 0.1 mmol/L over and above the secular trend, we would need to replace 3 percent of energy from butter with low TFA soft tub margarine, which is equivalent to halving New Zealand's current butter intake. Such a change is feasible given that per capita butter consumption in New Zealand is considerably higher than in other similar countries, including Australia and England. According to food balance sheet data for 1996 collected by the FAO, per capita butter consumption in New Zealand was 8 kg, compared to only 3 kg in Australia (FAO 2002). So a reduction in butter consumption to near Australian levels would be sufficient to achieve the degree of reduction in total cholesterol outlined under the intervention scenario.

Alternatively, butter could be replaced with margarine containing plant sterols or stanols. Plant sterols and stanols reduce the absorption of cholesterol from the gut and alter LDL apolipoprotein B kinetics, so lowering blood cholesterol concentrations. Based on a quantitative review of randomised double blind trials, replacing butter with margarine containing plant sterols or stanols lowers LDL cholesterol more than twice as much as replacing butter with ordinary polyunsaturated margarine (Law 2000b). Therefore, to reduce total cholesterol as outlined under the intervention scenario it would only be necessary to replace one-fifth of butter with margarine containing plant sterols or stanols.

Plant sterol or stanol margarines are, however, expensive to produce and supplies are currently limited. Therefore, a more realistic option would be to replace butter with a combination of ordinary margarine and plant sterol or stanol-enriched margarine. For example, replacing approximately one-third of butter consumption with margarine comprising equal portions of ordinary margarine and plant sterol or stanol-enriched margarine would lower blood cholesterol by the target amount.

## 6. Vegetable and Fruit Intake and the Burden of Disease

### 6.1 Introduction

Vegetable and fruit consumption has been found to be protective against cardiovascular disease and some common cancers. Evidence exists for a protective effect on ischaemic heart disease, ischaemic stroke, as well as lung, oesophageal, stomach and colorectal cancer. In addition, vegetables and fruit may substitute for less healthy foods in the diet (eg, energy-dense foods), thus contributing indirectly to maintaining a healthy body weight and the health benefits ensuing from this.

### Cardiovascular disease

A number of possible mechanisms have been put forward to explain the protective benefit of vegetables and fruit against heart disease. Antioxidant vitamins such as vitamin C, vitamin E and beta-carotene may provide protection by reducing the oxidation of cholesterol in the arteries (Van Duyn and Pivonka 2001). Antioxidant minerals, such as selenium and zinc, and sulphur-containing compounds may play a similar role (Van Duyn and Pivonka 2001). Folate helps lower blood homocysteine, a risk factor for cardiovascular disease (Ford et al 2002). The soluble fibre in vegetables and fruit has been shown to improve blood lipid profiles (Brown et al 1999). Diets rich in vegetables and fruit lower blood pressure (Appel et al 1997). Some of the blood pressure-lowering effect of vegetables and fruit is likely to be mediated via potassium, which is present in most vegetables and fruit and has been shown to lower blood pressure (Whelton et al 1997).

Table 53 summarises the substances present in vegetables and fruit that are protective against cardiovascular disease.

**Table 53:** Substances present in vegetables and fruit protective for cardiovascular disease

Phytochemical	Vegetable/fruit
<b>Antioxidants</b>	
Vitamin C	Citrus fruit, kiwifruit, blackcurrants, strawberries
Vitamin E	Avocado
Carotenoids	Yellow and orange vegetables and fruit, green leafy vegetables, cruciferous vegetables
Flavonoids	Allium vegetables, legumes, cruciferous vegetables, green leafy vegetables
Phytoestrogens	Legumes
Saponins	Legumes
Resveritol	Grapes
Selenium	Most vegetables and fruit – levels proportional to selenium content of soil they are grown in
<b>Homocysteine lowering</b>	
Folate	Green leafy vegetables, dried beans, melons, oranges
<b>Blood pressure lowering</b>	
Potassium	Most vegetables and fruit

Adapted from Van Duyn and Pivonka 2001

## Cancer

Substances found in vegetables and fruit are also known to have anti-carcinogenic activities (Steinmetz and Potter 1996). These substances can be divided into those that inhibit the action of carcinogens, those that detoxify carcinogens, and antioxidants. Table 54 provides a summary of the anti-carcinogenic agents present in vegetables and fruit.

**Table 54:** Anti-carcinogenic substances present in vegetables and fruit

Phytochemical	Fruit/vegetable
<b>Antioxidant</b>	
Carotenoids	Yellow and orange vegetables and fruit, green leafy vegetables, cruciferous vegetables
Flavonoids	Allium vegetables, legumes, cruciferous vegetables, green leafy vegetables
Phytoestrogens	Legumes
Capsaicin	Hot chilli peppers
Anthocyanins	Radishes, grapes
Phenols	Allium vegetables, cruciferous vegetables, green leafy vegetables, berries, citrus
Vitamin C	Citrus fruit, kiwifruit, blackcurrants, strawberries
Vitamin A	Red and orange vegetables and fruit
<b>Carcinogen-detoxifying agents</b>	
Sulphides	Allium vegetables
Isothiocyanates	Legumes, cruciferous vegetables, citrus
<b>Carcinogen-inhibiting agents</b>	
Phytosterols	Legumes
Phytoestrogens	Legumes

Adapted from: Van Duyn and Pivonka 2001

Broccoli has recently been shown to inhibit the growth of *Helicobacter pylori*, which is a risk factor for stomach cancer (Fahey et al 2002). Other potentially anti-carcinogenic substances have been found in vegetables and fruit in general. For example, selenium is present in vegetables and fruit in amounts proportional to the selenium content of the soil in which they are grown. Selenium functions as a cofactor for an enzyme that protects against oxidative tissue damage (Steinmetz and Potter 1996) and may exert an anticarcinogenic effect.

When attempts have been made to identify which micronutrients present in vegetables and fruit protect against disease, unexpected results have been found. For example, while diets high in carotenoid-rich vegetables and fruits have been found to be protective against lung cancer, two randomised controlled trials have found that dietary supplementation with beta carotene had an adverse effect on the incidence of lung cancer (The Alpha-Tocopherol Beta Carotene Cancer Prevention Study Group 1994; Omenn et al 1996). It is therefore more likely that, while each of the nutrients present within fruit and vegetables may play a role in the prevention of disease, their effect in combination within whole foods may confer a greater health benefit.



Vegetables and fruit are also relatively low in fat and energy, and help maintain a healthy body weight, which will further reduce the risk of cardiovascular disease, as well as diabetes and obesity-related cancers.

## 6.2 Vegetable and fruit consumption

### Current distribution

The intake of vegetables and fruit in grams per day was not determined as part of the original 1997 NNS analyses, so additional analysis was undertaken to obtain this information. Vegetables and fruit were defined as all fresh, frozen, canned, dried or juiced vegetables and fruit, except potatoes and mature legumes (pulses). Due to methodological difficulties, the vegetable and fruit content of some mixed dishes could not be determined, which may result in a slight underestimation of vegetable and fruit intakes. Data are based on a single 24-hour recall and were not corrected for the high day-to-day variation in vegetable and fruit intake, as evidenced by the high standard deviations.

Vegetable and fruit intake ranged from 0 to 3135 g/day. The presence of some very high vegetable and fruit intakes had the effect of skewing the distribution, resulting in mean vegetable and fruit intake being approximately 60 g/day higher than median intakes (412 vs 349 g/day). As a result, although the mean intake was slightly over 400 g/day, 58 percent of the overall population had vegetable and fruit intakes less than 400 g/day, the level recommended by the World Health Organization (WHO 1990).

Overall, mean vegetable and fruit intakes were slightly higher in males (420 g/day) than in females (404 g/day) (Table 55). This higher intake in males occurs because intakes of vegetables and fruit are correlated with energy intake, and energy intakes tend to be higher in males than females due to their larger body size. Māori had slightly lower mean vegetable and fruit intakes compared to non-Māori (396 vs 415 g/day).

Although vegetable and fruit intakes tended to be slightly lower in the youngest and oldest age groups compared to those in the middle age groups, these differences were small, so for the purpose of our analysis vegetable and fruit intakes were averaged across all age groups.

**Table 55:** Mean vegetable and fruit intake (g/day), by ethnicity and sex, 1997

	Sex	Mean	Standard deviation
Non-Māori	Males	422	333
	Females	407	302
Māori	Males	403	376
	Females	389	390
Total	Males	420	338
	Females	404	314

## Trends and international comparisons

New Zealand trends in vegetable and fruit intake in grams per day cannot be assessed, as these data were not collated in national nutrition surveys prior to 1997. However, food balance sheet data suggest that vegetable and fruit availability increased in New Zealand between 1960 and 1996 (FAO 1996). More recently, surveys carried out by United Fresh as part of the 5+ A Day campaign show that between 1995 and 1999 the average number of daily servings of vegetables and fruit increased from 3.9 to 4.4, and the proportion of people consuming five or more servings of vegetables and fruit each day increased from 31 to 44 percent (5+ A Day 2000). A more recent survey found that by 2001 46 percent of people were consuming five or more servings of vegetables and fruit each day (5+ A Day 2001).

Although this increasing trend is encouraging, the 5+ A Day research shows that more than half of all New Zealanders do not consume the recommended number of servings of vegetables and fruit each day. The 1997 NNS results were slightly more encouraging (perhaps because the focus is on all vegetables and fruit rather than just fresh), with 67 percent of adults consuming three or more servings of vegetables each day and 46 percent consuming two or more servings of fruit each day. Re-extraction of the 1997 NNS data in grams per day shows that although mean vegetable and fruit intake is close to the recommended 400 g/day (WHO 1990), the majority (58 percent) of New Zealanders consume less than 400 grams of vegetables (excluding potatoes) and fruit each day.

International comparisons of vegetable and fruit intake are difficult due to differences in the definition of vegetables and fruit, and measurement of vegetable and fruit intakes between countries. In New Zealand, the mean intake of vegetables and fruit was 412 g/day in 1997 (Table 56). In Australia where similar data are available, mean vegetable and fruit intake (excluding potatoes, mature legumes and juice) was 313 g/day in 1995 (McLennan and Podger 1999). However, the Australian value does not include juice, which contributed almost 50 g/day to the mean vegetable and fruit intake in New Zealand in 1997.

The data available for European countries refer to estimated vegetable and fruit intake based on a series of surveys of vegetable and fruit availability conducted in and around 1990 (Naska et al 2000). It is not known whether the European estimates include juice, but they exclude potatoes and pulses. Overall, it appears that vegetable and fruit intakes in New Zealand are similar to those in Australia, higher than those in the UK and Ireland, but lower than those in Mediterranean countries such as Spain and Greece. In Greece, where mean vegetable and fruit intakes are almost 200 g/day higher than in New Zealand, there is still a significant portion of the population (37 percent) who consume less than 400 g/day.

**Table 56:** International comparisons of mean vegetable and fruit intake and prevalence of adequate consumption

Country, year	Mean vegetable and fruit intake (g/day)	Percent consuming $\geq$ 400 g/day
New Zealand, 1997	412	42
Australia, 1995	313	–
UK, 1993	290	24
Ireland, 1987	233	12
Germany, 1988	343	31
Greece, 1987/88	617	63
Spain, 1990/91	488	51

Sources: 1997 NNS dataset; McLennan and Podger 1999, Naska et al 2000.

There is evidence in the UK that vegetable intake has been declining in the past 50 years, while fruit intake has been increasing (Food Standards Agency 2002). In Australia, food supply data (a surrogate indicator of vegetable and fruit consumption at the population level) indicate that vegetable consumption decreased from 1989/90 to 1994/95, but increased again by 1998/99 (de Looper and Bhatia 2001). During the same period, fruit consumption in Australia increased by 15 percent (de Looper and Bhatia 2001). In the United States, vegetable and fruit consumption in servings per day remained stable from 1996–2000 (National Center for Chronic Disease Prevention and Health Promotion 2000).

### Theoretical minimum distribution

The theoretical minimum is the risk factor (ie, vegetable and fruit) distribution that would yield the lowest population risk of adverse health outcomes. In contrast to the other risk factors, the theoretical minimum for vegetables and fruit represents an increase over current intakes, as vegetable and fruit consumption is inversely associated with risk of disease. Therefore, for vegetables and fruit it is necessary to estimate the *maximum* consumption that would result in the theoretical minimum risk of disease.

While there is evidence suggesting that the relationship between vegetable and fruit intake and disease is continuous within the range of usual intakes, it is not known what level of vegetable and fruit intake is associated with the lowest risk of disease. Therefore, the theoretical minimum is based on the highest known mean population intake of vegetable and fruit, which is found in Mediterranean countries such as Greece and is estimated to be approximately 600 g/day. This maximal intake may turn out to be too low, meaning that the estimates of attributable and avoidable burden for vegetables and fruit may be conservative.

For this study, calculations of attributable and avoidable mortality for vegetable and fruit intake have been based on a theoretical minimum risk distribution of  $600 \pm 50$  g/day for all age, sex and ethnic groups. This theoretical minimum is based on that chosen by the WHO fruit and vegetable expert working group for the *World Health Report 2002* (Ezzati et al 2002; Lock et al in press).

## **Distributional transitions**

Two distributional transitions for vegetable and fruit intake were estimated: a business as usual (BAU) scenario (historical trend) and an intervention scenario (deviation from historical trend reflecting policy change). Distributional transitions were based on the limited data on trends in vegetable and fruit intakes in New Zealand and other similar countries.

Distributional transitions are expressed as a percentage shift from the current distribution towards the theoretical minimum. For vegetable and fruit intake, distributional transitions were set to be the same for everyone in relative terms. However, the absolute changes in intake vary by gender and ethnicity, depending on baseline intakes.

The avoidable burden is the difference between the projected (BAU) scenario and the intervention scenario, with the shift between the two scenarios being maintained over time.

### ***Business as usual scenario***

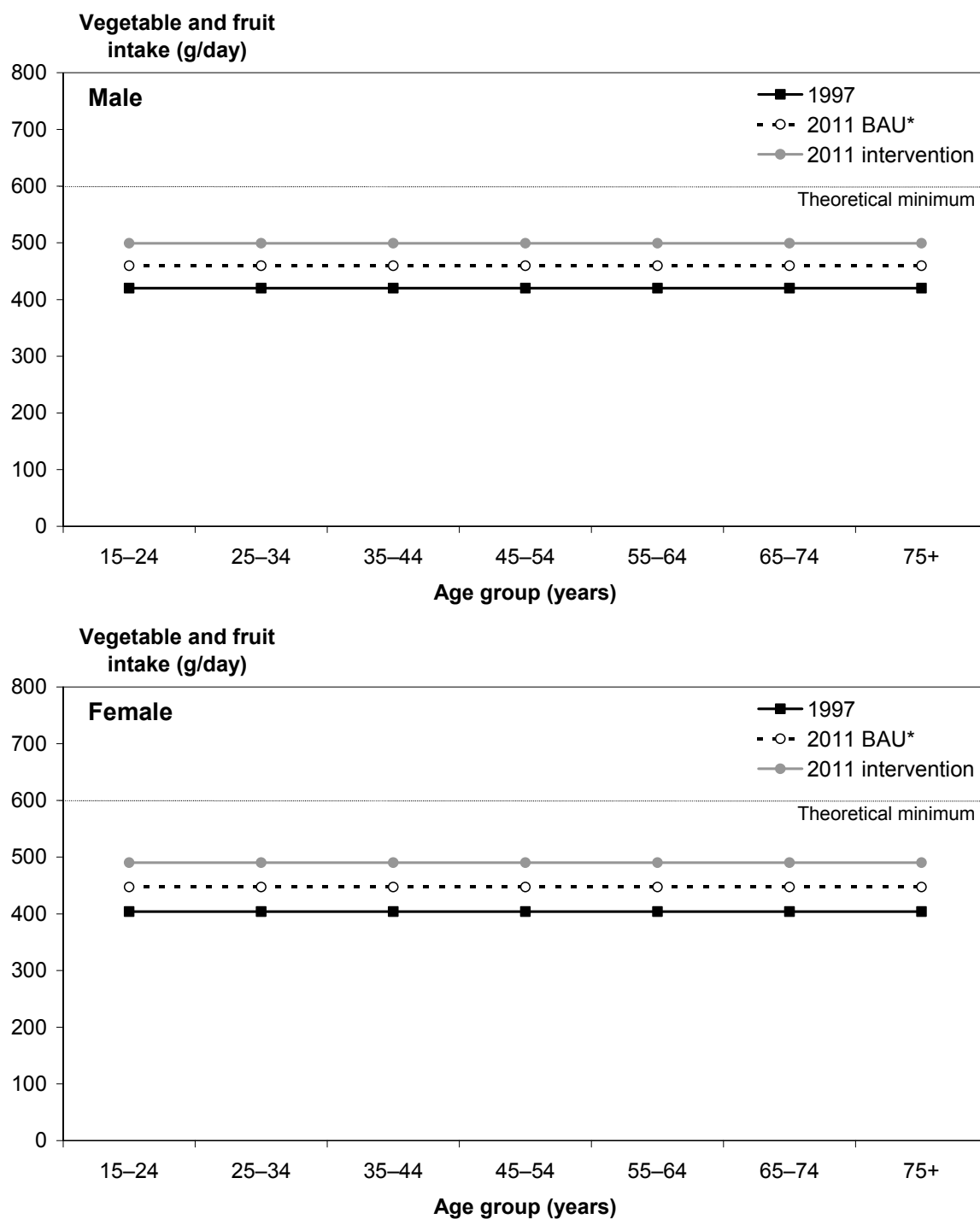
Given that current evidence suggests that vegetable and fruit consumption is gradually increasing in New Zealand, under the BAU scenario we estimated a 40 g/day (half a serving) increase in mean population vegetable and fruit intake by 2011. This increase in vegetable and fruit intakes would shift the current distribution 22 percent towards the theoretical maximal intake. In other words, under the business as usual scenario vegetable and fruit intakes would increase to approximately 460 g/day in males and 450 g/day in females by 2011.

### ***Intervention scenario***

Under the more optimistic intervention scenario, we estimated an 80 g/day (one serving) increase in mean population vegetable and fruit intake by 2011 (a 40 g increase over and above the BAU scenario). This increase in vegetable and fruit intakes would shift the current distribution 44 percent towards the theoretical maximal intake. In other words, under the intervention scenario, vegetable and fruit intakes would increase to approximately 500 g/day in males and 490 g/day in females by 2011.

Figure 22 shows the current (1997) vegetable and fruit intake, as well as projected vegetable and fruit intakes in 2011 under the BAU and intervention scenarios (separately for males and females) in relation to the theoretical maximum intake.

**Figure 22:** Current (1997) and projected future (2011) vegetable and fruit intakes



\* BAU = business as usual

## 6.3 Risk factor–disease relationships

### **Disease outcomes**

The outcomes assessed were based on those selected for the *World Health Report 2002* (WHO 2002) and include:

- ischaemic heart disease
- ischaemic stroke
- lung cancer
- oesophageal cancer
- stomach cancer
- colorectal cancer.

The selection of outcomes was based on strong evidence of a causal relationship and sufficient data to quantify the risk factor–disease relationship. Other disease outcomes linked to inadequate vegetable and fruit intake that did not meet selection criteria include cancers of the mouth, pharynx, larynx, pancreas and bladder, and type 2 diabetes.

### **Risk accumulation**

Risk accumulation refers to the nature and strength of the association between an exposure (ie, vegetable and fruit intake) and disease. Risk accumulation is expressed as the regression (risk) coefficient in the case of a continuous risk factor (ie, the increase in incidence or mortality of the disease per unit increase in exposure).

A systematic review of the literature was undertaken by Lock et al (in press) to identify cohort studies relevant to each disease endpoint. Where more than one study met the inclusion criteria for a particular disease, a meta-analysis was undertaken to obtain a summary risk coefficient estimate. Because there was no evidence that the strength of the association between vegetable and fruit intake and disease outcomes varied by sex, it was not necessary to have different risk accumulation estimates for males and females. It was assumed that the relationship between vegetable and fruit intake and disease outcomes was the same for all ethnic groups.

Vegetable and fruit intake is linked to other healthy behaviours. For example, people consuming vegetables and fruit frequently are likely to have other healthy habits: they are more likely to exercise regularly, be non-smokers, and have low intakes of saturated fat and cholesterol (Bazzano et al 2002). In order to account for potential confounders, one of the selection criteria for inclusion in a meta-analysis was that appropriate statistical adjustment had been made for potential confounders (except those on the causal chain between vegetable and fruit intake and disease). Most studies adjusted for age and sex, and the majority of recent studies also adjusted for a range of other variables such as cigarette smoking, energy intake, saturated fat intake, alcohol consumption, BMI and physical activity. However, as some potential confounders are not measured with great precision, some residual confounding may occur. Residual confounding could lead to an overestimation of the effect size.

### ***Ischaemic heart disease***

Details of the studies included and methods used to estimate risk coefficients for IHD have been published elsewhere (Lock et al, in press).

An 80 g/day (one serving) increase in vegetable and fruit intake was associated with a 9.9 percent lower risk of IHD in adults aged up to 64 years, dropping to 5.4 percent lower risk in adults aged 75 years and over (Table 57).

**Table 57:** Risk coefficients for ischaemic heart disease for an 80 g/day higher vegetable and fruit intake

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.901	9.9
35–44	0.901	9.9
45–54	0.901	9.9
55–64	0.901	9.9
65–74	0.916	8.4
75+	0.946	5.4

Source: WHO fruit and vegetable expert working group.

### ***Ischaemic stroke***

Details of the studies included and methods used to estimate risk coefficients for ischaemic stroke have been published elsewhere (Lock et al, in press).

An 80 g/day (one serving) increase in vegetable and fruit intake was associated with a 6.2 percent lower risk of ischaemic stroke among adults aged up to 54 years, dropping to a 3.7 percent lower risk in adults aged 75 years and over (Table 58).

**Table 58:** Risk coefficients for ischaemic stroke for an 80 g/day higher vegetable and fruit intake

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.938	6.2
35–44	0.938	6.2
45–54	0.938	6.2
55–64	0.946	5.4
65–74	0.961	3.9
75+	0.963	3.7

Source: WHO fruit and vegetable expert working group.

## ***Lung cancer***

Details of the studies included and methods used to estimate risk coefficients for lung cancer have been published elsewhere (Lock et al, in press).

An 80 g/day (one serving) increase in vegetable and fruit intake was associated with a 3.9 percent lower risk of lung cancer in adults aged up to 64 years, dropping to 2.6 percent lower risk in adults aged 75 years and over (Table 59).

**Table 59:** Risk coefficients for lung cancer for an 80 g/day higher vegetable and fruit intake

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.961	3.9
35–44	0.961	3.9
45–54	0.961	3.9
55–64	0.961	3.9
65–74	0.965	3.5
75+	0.974	2.6

Source: WHO fruit and vegetable expert working group.

## ***Oesophageal cancer***

Details of the studies included and methods used to estimate risk coefficients for oesophageal cancer have been published elsewhere (Lock et al, in press).

An 80 g/day (one serving) increase in vegetable and fruit intake was associated with a 6.2 percent lower risk of oesophageal cancer in adults aged up to 64 years, dropping to a 3.7 percent lower risk in adults aged 75 years and over (Table 60).

**Table 60:** Risk coefficients for oesophageal cancer for an 80 g/day higher vegetable and fruit intake

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.938	6.2
35–44	0.938	6.2
45–54	0.938	6.2
55–64	0.938	6.2
65–74	0.946	5.4
75+	0.963	3.7

Source: WHO fruit and vegetable expert working group.



### ***Stomach cancer***

Details of the studies included and methods used to estimate risk coefficients for stomach cancer have been published elsewhere (Lock et al, in press).

An 80 g/day (one serving) increase in vegetable and fruit intake was associated with a lower risk of stomach cancer in adults aged up to 64 years of 6.2 percent, dropping to a 3.7 percent lower risk in adults aged 75 years and over (Table 61).

**Table 61:** Risk coefficients for stomach cancer for an 80 g/day higher vegetable and fruit intake

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.938	6.2
35–44	0.938	6.2
45–54	0.938	6.2
55–64	0.938	6.2
65–74	0.946	5.4
75+	0.963	3.7

Source: WHO fruit and vegetable expert working group.

### ***Colorectal cancer***

Details of the studies included and methods used to estimate risk coefficients for colorectal cancer have been published elsewhere (Lock et al, in press).

An 80 g/day (one serving) increase in vegetable and fruit intake was associated with a 0.8 percent lower risk of colorectal cancer in adults aged up to 74 years, dropping to a 0.3 percent lower risk in adults aged 75 years and over (Table 62).

**Table 62:** Risk coefficients for colorectal cancer for an 80 g/day increase in vegetable and fruit intake

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.992	0.8
35–44	0.992	0.8
45–54	0.992	0.8
55–64	0.992	0.8
65–74	0.992	0.8
75+	0.997	0.3

Source: WHO fruit and vegetable expert working group.

## Risk reversal

Risk reversal refers to how quickly and how completely risk of disease is reversed following an increase in vegetable and fruit consumption. Randomised controlled trials provide information on risk reversal; however, very few studies have examined the effects of increasing vegetable and fruit intake on disease outcomes, largely due to the difficulty of conducting dietary intervention trials. Furthermore, most studies that have done so are of short duration and/or have included other dietary changes. Therefore, the risk reversibility for cardiovascular disease and cancer outcomes is based on those for other risk factors.

Estimates of risk reversibility used for this study were therefore as follows:

- 100 percent risk reversal for IHD and stroke within five years
- 84 percent risk reversal for cancer outcomes within 10 years.

These risk-reversal estimates are similar to those chosen by the WHO fruit and vegetable expert working group for the *World Health Report 2002*, details of which will be published in a forthcoming technical report (Lock et al, in press). It was assumed that risk reversal is the same for both sexes and all ethnic groups.

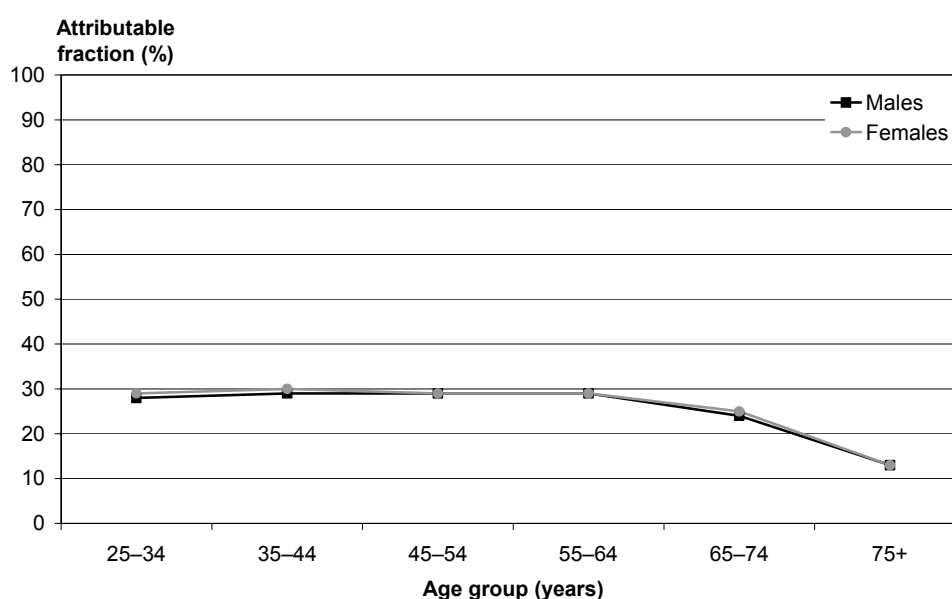
## 6.4 Attributable mortality

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### Ischaemic heart disease

Approximately 15–30 percent of IHD mortality in 1997 was due to ‘low’ vegetable and fruit intake (Figure 23). The lowest attributable fractions occurred in the older age groups.

**Figure 23:** Attributable fractions (%) for vegetable and fruit intake and ischaemic heart disease, 1997



Overall, ‘low’ vegetable and fruit intake contributed to 1171 IHD (95% CI 703–1915) deaths and 12,522 YLL in 1997 (Table 63). The majority (almost 75 percent) of IHD deaths attributable to ‘low’ vegetable and fruit intake occurred in adults aged 65 years and older.

The age-standardised IHD mortality rate attributable to ‘low’ vegetable and fruit intake was about twice as high in males as in females (Table 63). This difference reflects the higher IHD mortality rate in males, as attributable fractions were only slightly higher in males compared to females. The age-standardised IHD mortality rate attributable to ‘low’ vegetable and fruit intake was twice as high in Māori males and females as in non-Māori males and females. This difference reflects higher IHD mortality rates, and to a lesser extent a greater prevalence of ‘low’ vegetable and fruit intake in Māori compared to non-Māori.

**Table 63:** Attributable mortality for vegetable and fruit intake and ischaemic heart disease, 1997

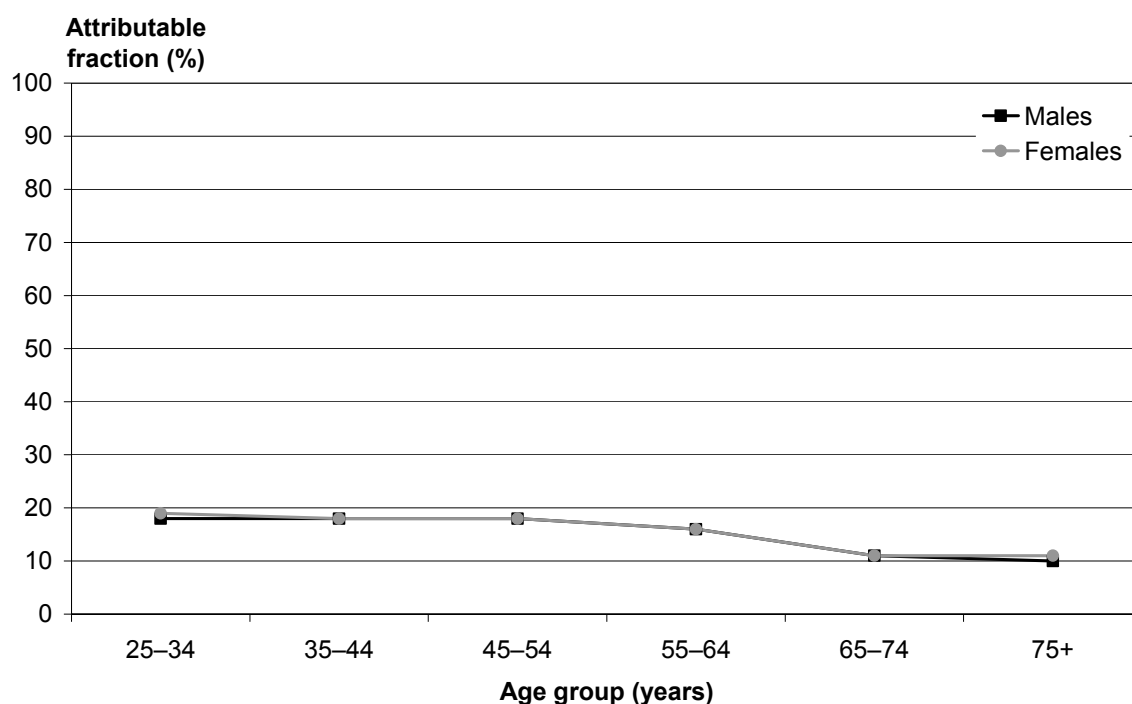
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	633	6,766	54.6	609
	Māori	79	1,197	106.4	1,399
	Total	712	7,963	58.4	673
Females	Non-Māori	416	3,946	24.5	259
	Māori	43	613	56.9	731
	Total	459	4,559	26.6	294

\* Rate per 100,000, age-standardised to WHO World population

## Ischaemic stroke

Approximately 10–20 percent of ischaemic stroke mortality in 1997 was attributable to ‘low’ vegetable and fruit intake (Figure 24). The lowest attributable fractions occurred in the older age groups.

**Figure 24:** Attributable fractions (%) for vegetable and fruit intake and ischaemic stroke, 1997



Applying the attributable fractions to the 1997 mortality data shows that ‘low’ vegetable and fruit intake contributed to 179 (95% CI 65–276) ischaemic stroke deaths and 1449 YLL in 1997 (Table 64). The majority (almost 80 percent) of ischaemic stroke deaths attributable to ‘low’ vegetable and fruit intake occurred in adults aged 75 years and older.

The age-standardised ischaemic stroke mortality rate attributable to ‘low’ vegetable and fruit intake was similar in males and females, but 20–25 percent higher in Māori males and females than in non-Māori males and females (Table 64). This difference reflects slightly higher ischaemic stroke mortality rates, as well as a greater prevalence of ‘low’ vegetable and fruit intake in Māori compared to non-Māori.

**Table 64:** Attributable mortality for vegetable and fruit intake and ischaemic stroke, 1997

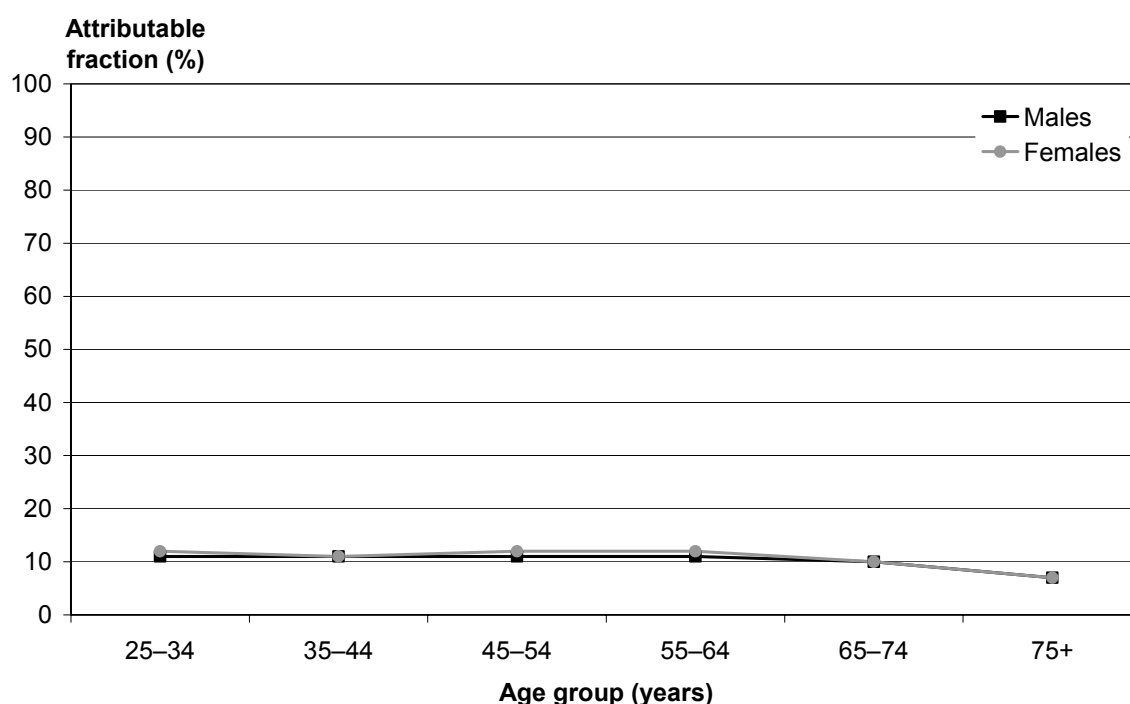
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	65	483	5.4	41
	Māori	4	41	6.5	59
	Total	69	524	5.5	43
Females	Non-Māori	105	863	5.5	49
	Māori	5	62	6.9	78
	Total	110	925	5.6	51

\* Rate per 100,000, age-standardised to WHO World population

## Lung cancer

Approximately 7–12 percent of lung cancer mortality in 1997 was due to ‘low’ vegetable and fruit intake (Figure 25). The lowest attributable fractions occurred in the older age groups.

**Figure 25:** Attributable fractions (%) for vegetable and fruit intake and lung cancer, 1997



Applying the attributable fractions to the 1997 mortality data shows that ‘low’ vegetable and fruit intake contributed to 131 (95% CI 69–246) lung cancer deaths and 1601 YLL in 1997 (Table 65).

The age-standardised lung cancer mortality rate attributable to ‘low’ vegetable and fruit intake was about twice as high in males as in females (Table 65). This difference primarily reflects the higher lung cancer mortality rates in males, as attributable fractions were actually slightly higher in females than in males. The age-standardised lung cancer mortality rate attributable to ‘low’ vegetable and fruit intake was four to five times higher in Māori than in non-Māori, primarily due to the much higher lung cancer mortality rate in Māori.

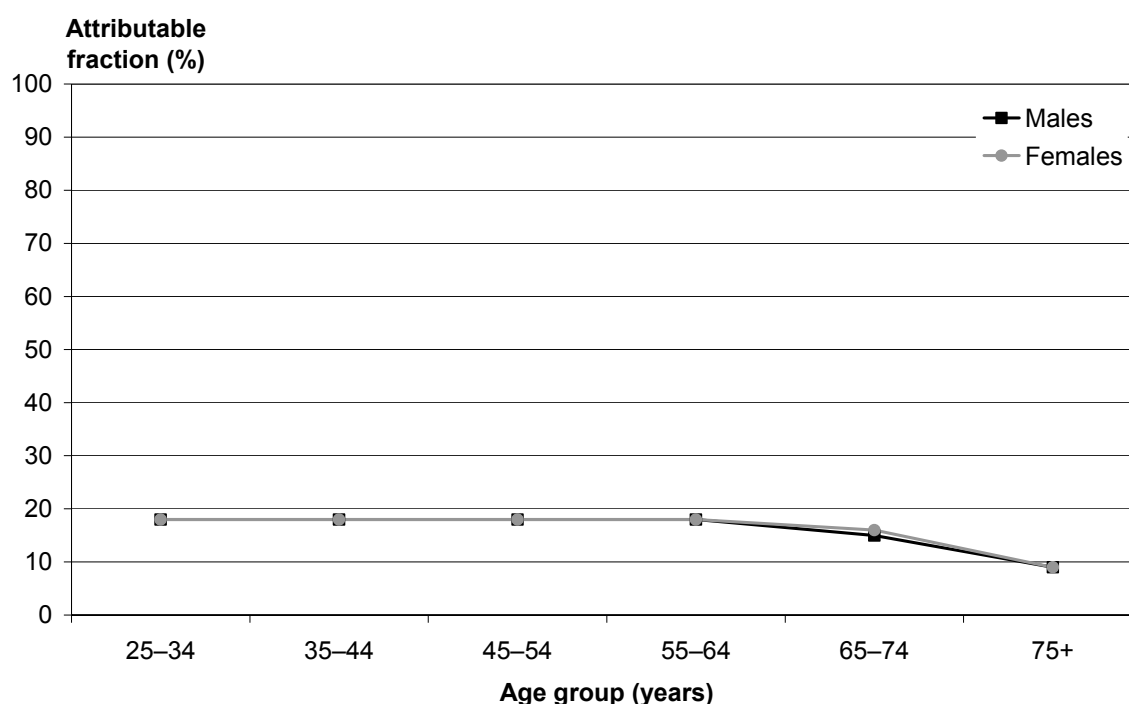
**Table 65:** Attributable mortality for vegetable and fruit intake and lung cancer, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	69	757	5.9	68
	Māori	12	170	17.1	215
	Total	81	927	6.7	78
Females	Non-Māori	39	507	2.9	42
	Māori	11	167	13.1	189
	Total	50	674	3.6	53

\* Rate per 100,000, age-standardised to WHO World population

## Oesophageal cancer

Approximately 10–20 percent of oesophageal cancer deaths in 1997 were due to ‘low’ vegetable and fruit intake (Figure 26). The lowest attributable fractions occurred in the older age groups.

**Figure 26:** Attributable fractions (%) for vegetable and fruit intake and oesophageal cancer, 1997

Applying the attributable fractions to the 1997 mortality data shows that ‘low’ vegetable and fruit intake contributed to 25 (95% CI 14–39) oesophageal cancer deaths and 297 YLL in 1997 (Table 66).

The age-standardised oesophageal cancer mortality rate attributable to ‘low’ vegetable and fruit intake was almost three times higher in males than in females (Table 66). This difference primarily reflects the higher oesophageal cancer mortality rates in males, as attributable fractions were only slightly higher in males compared to females. The age-standardised oesophageal cancer mortality rate attributable to ‘low’ vegetable and fruit intake was higher in Māori males and females than in non-Māori males and females. This difference reflects slightly higher oesophageal cancer mortality rates, as well as a greater prevalence of ‘low’ vegetable and fruit intake in Māori compared to non-Māori.

**Table 66:** Attributable mortality for vegetable and fruit intake and oesophageal cancer, 1997

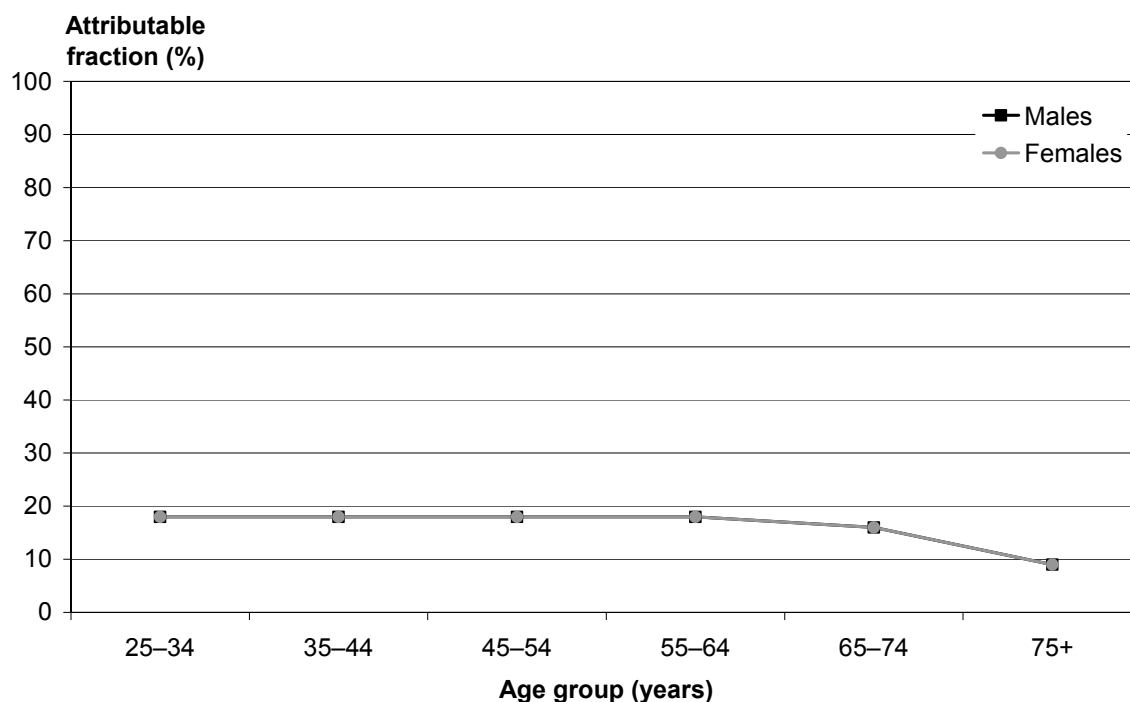
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	16	191	1.4	17
	Māori	1	20	1.7	23
	Total	17	211	1.4	18
Females	Non-Māori	7	76	0.5	6
	Māori	1	10	0.8	12
	Total	8	86	0.5	6

\* Rate per 100,000, age-standardised to WHO World population

## Stomach cancer

Approximately 10–20 percent of stomach cancer mortality in 1997 was due to ‘low’ vegetable and fruit intake (Figure 27).

**Figure 27:** Attributable fractions (%) for vegetable and fruit intake and stomach cancer, 1997



Applying the attributable fractions to the 1997 mortality data shows that ‘low’ vegetable and fruit intake contributed to 39 (95% CI 22–64) stomach cancer deaths and 473 YLL in 1997 (Table 67).

The age-standardised stomach cancer mortality rate attributable to ‘low’ vegetable and fruit intake was twice as high in males than in females (Table 67). This difference reflects the higher stomach cancer mortality rates in males, as attributable fractions were similar in males and females. The age-standardised stomach cancer mortality rate attributable to ‘low’ vegetable and fruit intake was three to four times higher in Māori males and females than in non-Māori males and females. This difference primarily reflects the much higher stomach cancer mortality in Māori compared to non-Māori.

**Table 67:** Attributable mortality for vegetable and fruit intake and stomach cancer, 1997

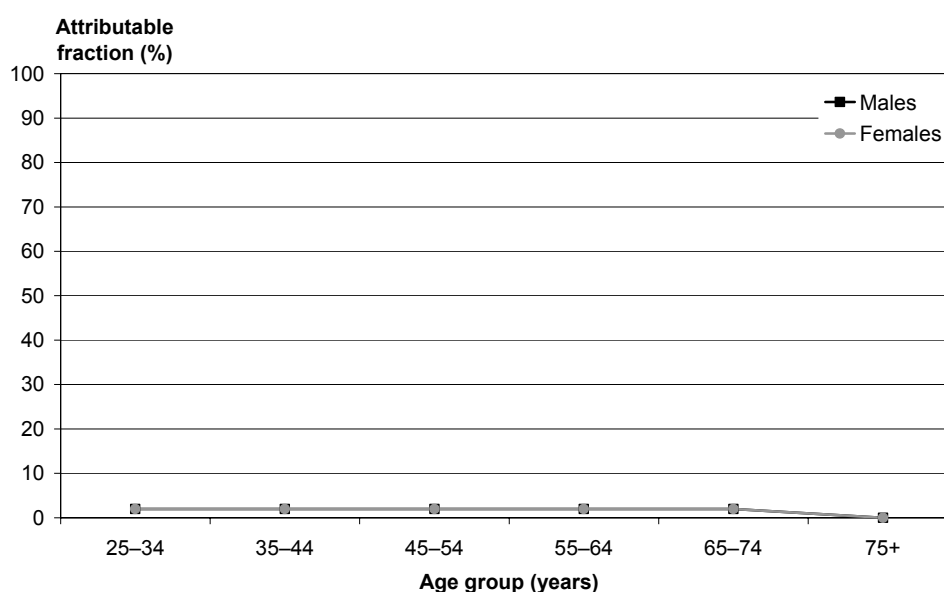
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)
Males	Non-Māori	19	210	1.6	19
	Māori	5	73	5.7	80
	Total	24	283	1.9	24
Females	Non-Māori	12	147	0.9	12
	Māori	3	43	3.5	47
	Total	15	190	1.0	14

\* Age-standardised to WHO world population

## Colorectal cancer

Approximately 2 percent of colorectal cancer mortality in 1997 was due to ‘low’ vegetable and fruit intake (Figure 28).

**Figure 28:** Attributable fractions (%) for vegetable and fruit intake and colorectal cancer, total, 1997





Applying the attributable fractions to the 1997 mortality data shows that ‘low’ vegetable and fruit intake contributed to 14 (95% CI 7–55) colorectal cancer deaths and 208 YLL in 1997 (Table 68).

The age-standardised colorectal cancer mortality rate attributable to ‘low’ vegetable and fruit intake was about 40 percent higher in males than in females (Table 68). This difference primarily reflects the higher colorectal cancer mortality rates in males, as attributable fractions were only slightly higher in males compared to females. The age-standardised colorectal cancer mortality rate attributable to ‘low’ vegetable and fruit intake was the same in Māori and non-Māori.

**Table 68:** Attributable mortality for vegetable and fruit intake and colorectal cancer, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	8	107	0.7	10
	Māori	0	6	0.6	8
	Total	8	113	0.7	10
Females	Non-Māori	6	90	0.5	8
	Māori	0	5	0.4	6
	Total	6	95	0.5	8

\* Rate per 100,000, age-standardised to WHO World population

### Total attributable mortality

Overall, ‘low’ vegetable and fruit consumption contributed to a total of 1559 deaths (6 percent of all deaths) and 16,550 YLL in 1997 (Table 69), including 18 percent of IHD deaths and 3 percent of all cancer deaths.

**Table 69:** Attributable mortality for vegetable and fruit intake and all diseases, 1997

Sex	Ethnicity	Deaths		Years of life lost	
		Count	%*	Count	%*
Males	Non-Māori	810	6	8,514	7
	Māori	101	7	1,507	6
	Total	911	6	10,021	6
Females	Non-Māori	585	5	5,629	5
	Māori	63	6	900	5
	Total	648	5	6,529	5

\* % = percentage of all deaths and years of life lost, 1997.

## 6.5 Avoidable mortality

Estimates of cardiovascular and cancer mortality in 2011 were projected based on trends since 1975 and 1960, respectively.

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### Ischaemic heart disease

With an increase in vegetable and fruit consumption as outlined under the intervention scenario, 4–20 percent of ischaemic heart disease deaths could be avoided depending on age and baseline intake. Although the highest proportion of avoidable deaths would occur in the younger age groups (20 percent in 25–34 years age group), the greatest number of deaths would be avoided in the older age groups. This shift in vegetable and fruit intake would mean that 235 IHD deaths and 2389 YLL could be avoided each year from 2011 (Table 70).

**Table 70:** Avoidable mortality\* for vegetable and fruit intake and ischaemic heart disease, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	132	1,320
	Māori	20	273
	Total	152	1,593
Females	Non-Māori	73	670
	Māori	10	126
	Total	83	796

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

### Ischaemic stroke

With an increase in vegetable and fruit consumption under the intervention scenario, 3–11 percent of ischaemic stroke deaths would be avoided depending on age and baseline intake. The greatest number of deaths would be avoided in the older age groups where ischaemic stroke is most likely to occur. This shift in vegetable and fruit intake would mean that 37 ischaemic stroke deaths and 306 YLL could be avoided each year from 2011 (Table 71).

**Table 71:** Avoidable mortality\* for vegetable and fruit intake and ischaemic stroke, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	14	104
	Māori	1	13
	Total	15	117
Females	Non-Māori	21	171
	Māori	1	18
	Total	22	189

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Lung cancer

With an increase in vegetable and fruit consumption as outlined under the intervention scenario, approximately 2–10 percent of lung cancer deaths would be avoided depending on age and baseline intake. The greatest number of deaths would be avoided in the older age groups. This shift in vegetable and fruit intake would mean that 39 lung cancer deaths and 469 YLL could be avoided each year by 2011 (Table 72).

**Table 72:** Avoidable mortality\* for vegetable and fruit intake and lung cancer, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	15	147
	Māori	3	41
	Total	18	188
Females	Non-Māori	15	192
	Māori	6	89
	Total	21	281

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Oesophageal cancer

With an increase in vegetable and fruit consumption as outlined under the intervention scenario, 3–17 percent of oesophageal cancer deaths would be avoided depending on age and baseline vegetable and fruit intake. If vegetable and fruit intake increased to this level, nine oesophageal cancer deaths and 107 YLL could be avoided each year from 2011 (Table 73).

**Table 73:** Avoidable mortality\* for vegetable and fruit intake and oesophageal cancer, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	6	67
	Māori	1	10
	Total	7	77
Females	Non-Māori	2	25
	Māori	0	5
	Total	2	30

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Stomach cancer

With an increase in vegetable and fruit consumption as outlined under the intervention scenario, 3–17 percent of stomach cancer deaths would be avoided depending on age and baseline intake. If vegetable and fruit intake increased to this level, 10 stomach cancer deaths and 126 YLL could be avoided each year from 2011 (Table 74).

**Table 74:** Avoidable mortality\* for vegetable and fruit intake and stomach cancer, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	4	51
	Māori	2	24
	Total	6	75
Females	Non-Māori	3	36
	Māori	1	15
	Total	4	51

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Colorectal cancer

With an increase in vegetable and fruit consumption as outlined under the intervention scenario, 1–2 percent of colorectal cancer deaths would be avoided depending on age and baseline intake. If vegetable and fruit intake increased to this level, four colorectal cancer deaths and 51 YLL could be avoided each year from 2011 (Table 75).

**Table 75:** Avoidable mortality\* for vegetable and fruit intake and colorectal cancer, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	2	25
	Māori	0	2
	Total	2	27
Females	Non-Māori	2	22
	Māori	0	2
	Total	2	24

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

### Total avoidable mortality

If vegetable and fruit intake increased as outlined under the intervention scenario, a total of 334 deaths and 3448 YLL could be avoided each year from 2011 (Table 76).

**Table 76:** Avoidable mortality\* for vegetable and fruit intake and all diseases, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	173	1,714
	Māori	27	363
	Total	200	2,077
Females	Non-Māori	116	1,116
	Māori	18	255
	Total	134	1,371

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## 6.6 Discussion

We estimate that in 1997 ‘low’ vegetable and fruit intake contributed to 1559 deaths (6 percent of all deaths) (Table 77). Although only a relatively modest incremental increase in mean population vegetable and fruit intake is proposed under the intervention scenario (40 g/day above the BAU scenario, which was itself 40 g/day above the average 1997 intake), this translates into significant decreases in mortality. If relevant policy initiatives were introduced now, such a change could prevent 334 deaths each year from 2011, three-quarters from reduced IHD mortality.

**Table 77:** Summary of attributable and avoidable mortality due to 'low' vegetable and fruit intake

	Attributable mortality (1997)		Avoidable mortality (2011)*	
	Deaths (count)	Years of life lost (count)	Deaths (count)	Years of life lost (count)
Ischaemic heart disease	1,171	12,522	235	2,389
Stroke	179	1,449	37	306
Lung cancer	131	1,601	39	469
Oesophageal cancer	25	297	9	107
Stomach cancer	39	473	10	126
Colorectal cancer	14	208	4	51
Total	1,559	16,550	334	3,448

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

Although a single intervention scenario has been modelled here, the effect of alternative scenarios can be readily estimated. The relationship between the distributional transition and the avoidable burden is approximately linear. Therefore doubling the shift in vegetable and fruit intake (from 22 percent to 44 percent beyond the BAU scenario) would double the avoidable burden (from 330 to 660 deaths in 2011).

## Policy implications

Many countries have recognised the importance of increasing vegetable and fruit consumption within their populations. As a result, many countries now have vegetable and fruit promotion campaigns, which are funded by either government or industry. Policies used to increase the consumption of vegetables and fruit include comprehensive multi-media social marketing strategies and setting-based approaches such as school canteens and community gardens. Because food pricing is an important factor influencing food consumption choices, lowering the price (eg, by subsidies for people on low incomes) and improving the availability of vegetables and fruit are likely to be more effective than health education or social marketing interventions alone (Joffe and Robertson 2001).

In New Zealand, 5+ A Day was launched in 1995 by United Fresh with the aim of encouraging New Zealanders to increase their intake of fresh fruit and vegetables to five or more servings each day. An evaluation completed in February 1999 found that, overall, 5+ A Day is proving successful in terms of increasing consumer awareness of the benefits of healthy eating. Nearly all primary schools nationwide now participate in the annual 5+ A Day week (P Dudley, personal communication, October 2000). In 1999 the 5+ A Day programme was launched into early education centres nationwide, with over 1600 centres participating.

In Western Australia, the Department of Health has been running a fruit and vegetable campaign over the past 10 years targeting adults, parents and children, the fruit and vegetable industry, catering industry, and health and education professionals (Miller 2001). This campaign has involved a comprehensive social marketing strategy and has used a range of state-wide interventions. Maintenance of the campaign has been supported by strengthening scientific evidence, identification as a priority in Departmental policy and planning documents, a high profile, demonstrated success, and the stability of organisational funding and staffing. Although other Australian states have conducted similar campaigns, with the exception of the Western Australia campaign none of these were sustained for more than two years.

Another example of an intersectoral campaign in Australia is the partnership formed in 1999 between the Dietitians Association of Australia and Coles Supermarkets, resulting in the 'Coles 7-a-day' programme. This programme aims to sustainably increase fruit and vegetable consumption by 0.25 serves per day each year. The results indicate that this goal has almost been achieved with an increase of 0.4 daily serves of fresh fruit and vegetables over the first two years of the programme.

In their 2000 National Health Service (NHS) Plan, the British government recognised that 'increasing fruit and vegetable consumption is considered the second most effective strategy for cancer prevention after reducing smoking' (Department of Health [London] 2000). As a result, a number of initiatives supported and, in some cases, funded by the British government are currently under way to increase vegetable and fruit consumption. Programmes include:

- National School Fruit Scheme
- Five-a-Day community projects
- working with retailers, producers, caterers and other key stakeholders
- a communications programme
- evaluation and monitoring.

Pilots for the National School Fruit Scheme and Five-a-Day community projects have been undertaken and evaluated to ascertain how a national programme would best be implemented. The results of both the evaluations have been positive (Department of Health [London] 2001; Caraher et al 2002; Chant et al 2002; Coady and O'Hara 2002; Durkin et al 2002; Rex 2002).

Aspects of the National School Fruit Scheme associated with greatest success were:

- using a whole-school approach
- eating fruit in class groups
- input into teaching and learning
- establishing a routine
- using staff as role models.

The Five-a-Day community pilots tended to focus on a settings approach, targeting schools, workplaces and communities in order to increase vegetable and fruit consumption. Innovative methods were used to improve accessibility and affordability of vegetables and fruit in the communities. Evaluation of the community pilots found that multi-sectoral collaboration and co-operation were important factors in the success of the pilot projects, as was having a dedicated project worker co-ordinating the project.

The national United States 5 A Day programme began in 1991 and is jointly sponsored by the National Cancer Institute (NCI) in the US Department of Health and Human Services, and the Produce for Better Health Foundation (PBHF), a non-profit consumer education foundation representing the fruit and vegetable industry. An evaluation of this programme was carried out in 2000 (Potter et al 2000). Findings of this evaluation were generally positive, particularly with respect to comments on NCI's collaboration with private industry. There have been increases in knowledge of the 5 A Day programme and its message. However, it is difficult to say whether there has been an increase in fruit and vegetable consumption resulting from the 5 A Day programme because there was no comparison group.

In summary, policy options to achieve the degree of change in vegetable and fruit consumption required by our modelled intervention scenario include a sustained, comprehensive social marketing strategy building on the existing but under-resourced 5+ A Day campaign; focused interventions in specific settings such as schools, workplaces and communities; and possibly fiscal interventions such as subsidising relevant products for low-income people and fostering community gardens and community fruit and vegetable purchasing co-ops.

## **Limitations**

A number of issues need to be considered in interpreting the results, including the measurement of vegetables and fruit consumption, the setting of the theoretical minimum, residual confounding, and limitations of the CRA methodology in general.

In regard to the last of these, the reality is that vegetables and fruit do not act in isolation in the pathogenesis of disease. For example, although vegetables and fruit have a role in reducing blood pressure, blood pressure itself is an independent risk factor for cardiovascular disease and is also influenced by other risk factors such as salt in the diet, BMI and physical activity. In addition, vegetable and fruit intake is part of a more global dietary or lifestyle pattern within both individuals and populations. For example, Serdula et al (1996) showed that the frequency of intake of fresh fruit and vegetables increased as the level of physical activity increased, whereas those who were sedentary, heavy smokers or heavy drinkers had lower consumption. The aetiology of disease is complex, and traditional control of confounding through multiple regression modelling cannot fully take into account the complex causal web leading to many disease states.



Data used to estimate current vegetable and fruit intake in New Zealand are based on one 24-hour dietary recall from a single national nutrition survey conducted in 1997. A single 24-hour recall results in a high intra-individual variation and so does not accurately represent an individual's usual intake. Using a single 24-hour recall may have resulted in a wider distribution of vegetable and fruit intake (with high standard deviations) with more people at the extremes of intake than the true distribution.

The fact that our standard deviations are high may have resulted in an underestimation of the attributable fractions. A sensitivity analysis was done using approximations to a skewed distribution that mimic the observed distribution more closely than a normal distribution, to identify the effect of skewing and high standard deviations on our results. This analysis confirmed that our results, based on a normal distribution, may have slightly underestimated the attributable fractions for the selected diseases.

The same theoretical minimum was used for all ethnicities, ages and genders. It is possible that a different theoretical minimum risk distribution (in this case maximum consumption for a protective effect) should have been used for males and females. Although mean vegetable and fruit intakes were slightly higher in males than in females (approximately 4 percent higher), this level of intake is lower as a proportion of energy intake for males given their higher energy intakes (11,942 vs 7969 kJ) (Russell et al 1999). On the other hand, there is no evidence that men need a higher absolute intake than females (corresponding to the same relative intake expressed as a percentage of energy) to obtain the same level of health benefit.

More generally, the chosen theoretical minimum distribution (the distribution conferring the minimum risk of disease) is based on limited evidence, and may prove to be too low for both genders – in which case the attributable burdens of disease associated with inadequate vegetable and fruit consumption will have been underestimated while the corresponding avoidable burdens will have been overestimated.

## 7. Energy Balance and the Burden of Disease

### 7.1 Introduction

Excess body fat is one of the most important modifiable risk factors for a number of important diseases, including type 2 diabetes mellitus, ischaemic heart disease (IHD), ischaemic stroke and several common cancers. The impact of overweight and obesity on these diseases operates, at least in part, through its effects on insulin resistance, blood glucose, blood lipids and blood pressure.

Body mass index (BMI) is the anthropometric measure that provides the most useful population-level indicator of overweight and obesity. BMI is a measure of weight, adjusted for height, and is calculated by dividing weight in kilograms by the square of height in metres ( $\text{kg/m}^2$ ). The normal range for BMI is 18.5–24.9. Adults with a BMI less than 18.5 are considered underweight, adults with a BMI of 25.0–29.9 are classified as overweight, and those with a BMI  $\geq 30$  are classified as obese. However, the association between BMI and type 2 diabetes and cardiovascular disease is continuous down to BMI values as low as 19 or 20  $\text{kg/m}^2$  (Willett et al 1999). Therefore, many individuals with BMI values in the normal range would benefit from weight loss.

### 7.2 Determinants of energy balance

Energy intake is determined by food and beverage consumption. Energy expenditure has three main components: basal metabolic rate (BMR), dietary thermogenesis (ie, energy expended converting food to nutrients), and physical activity (WHO 2000b). The most variable component of energy expenditure is physical activity, which contributes approximately 30 percent of energy expenditure in sedentary adults and approximately 50 percent in adults involved in heavy manual work. Dietary thermogenesis remains relatively constant at approximately 10 percent of energy expenditure, leaving BMR to account for approximately 40 percent of energy expenditure in adults involved in heavy manual work and 60 percent of energy expenditure in sedentary adults.

Overweight and obesity are the result of a positive energy balance, that is, a chronic excess of energy intake over energy expenditure. Currently it is not known whether a large positive energy balance on some days or a small positive energy balance on most days produces the larger increases in body weight.

Although some people are more genetically susceptible to weight gain than others, the rapid increase in the prevalence of overweight and obesity (especially the latter) during the last two decades has occurred too quickly to be explained by genetic changes (WHO 2000b). Most experts agree that the principal cause of the increase in the proportion of the population overweight or obese seen over recent decades has been a combination of sedentary lifestyles and energy-dense diets. However, current methods for measuring both energy intake and physical activity in free-living populations are neither precise nor sensitive enough to detect small changes in these complex behaviours. As a consequence, it has been impossible to determine whether diet or physical activity has been the more important contributor in bringing about this change in population BMI distributions – in any case it is the balance between energy intake and expenditure that matters.

## **Dietary intake**

### ***Energy intake***

Observational studies have not always found a positive relationship between energy intake and body weight or BMI. This is due to a combination of poor study design, failure to control for confounders (especially physical activity), and measurement error. Energy intake is difficult to measure precisely and is generally under-reported in dietary surveys. The degree of under-reporting varies by sex, age, socioeconomic status and BMI (Briefel et al 1995, 1997; Braam et al 1998; Kretsch et al 1999). In particular, overweight people are more likely to under-report their food intake than are their normal-weight counterparts (Gnardellis et al 1998; Kretsch et al 1999; Goris et al 2000). This explains why early studies of dietary intake in overweight people suggested that they ate less than others. Although under-reporting has always been a problem in dietary surveys, the degree of under-reporting in Western populations has increased in the last two decades (Hirvonen et al 1997; Heitmann et al 2000). This increase in under-reporting probably reflects the increasing prevalence of obesity combined with a greater awareness of a healthy diet (social desirability bias) among the general population.

The combination of changes in dietary assessment methodology and increased under-reporting makes it difficult to accurately monitor and interpret trends in energy intake. However, there is some evidence indicating that energy intake has increased in some developed countries during the past two to three decades. In Australia, energy intake increased by approximately 300–400 kJ in adults from 1983 to 1995 (AIHW 2002). In the United States, data from the National Health and Nutrition Examination Surveys (NHANES) suggest that energy intake increased by approximately 400–1200 kJ in adolescents and adults from 1976–80 to 1988–91 (McDowell et al 1994). Data from the 1977–78 Nationwide Food Consumption Survey and the 1989–91 and 1994–96 Continuing Surveys of Food Intake by Individuals indicate that total energy intake increased by approximately 800 kJ (11 percent) in Americans aged two years and over from 1977–78 to 1994–96 (Nielsen et al 2002). Food supply data from the United States Department of Agriculture indicate that per capita energy availability increased by 2100 kJ from 1970 to 1994 (Harnack et al 2000). The smaller increases in energy intake found in dietary surveys compared to food supply data could be due to problems associated with dietary assessment, including under-reporting.

## **Macronutrient intake**

Some macronutrients are more closely correlated with higher energy intakes and excess body weight than others. Observational studies of diet composition and BMI generally demonstrate a positive association between BMI and diets high in fat, whereas diets high in carbohydrates and low to moderate in fat are associated with lower energy intakes and lower BMI. Data from the National Diet and Nutrition Survey of British Adults show that high-fat consumers have higher energy intakes, and tend to have a higher BMI than low-fat consumers, although not all high-fat consumers are overweight (Macdiarmid et al 1996). Data from the United States Department of Agriculture National Food Consumption Surveys and the Continuing Survey of Food Intakes by Individuals indicate that, on average, people consuming low-fat diets ( $\leq 30$  percent of energy from fat) have lower energy intakes and lower BMI than people consuming high-fat diets ( $> 30$  percent of energy from fat) (Kennedy and Bowman 2001). Similarly, adults consuming high-carbohydrate diets (which are lower in energy density) are more likely to be in the normal weight range (Kennedy et al 2001; Bowman and Spence 2002). Dietary strategies associated with lower fat and energy intakes include the use of reduced-fat foods and choosing lean rather than higher fat meats (Lichtenstein et al 1998; Peterson et al 1999). Food items associated with low-fat diets include fruit, grains and skim milk (Lichtenstein et al 1998).

There are several reasons why dietary macronutrient composition may be important in the development of obesity. Macronutrients vary in their energy density, effect on satiety, storage capacity, metabolism and autoregulation (WHO 2000b). Dietary fat is the most energy-dense macronutrient, providing more than twice as much energy per gram (37 kJ) as carbohydrate and protein (16 and 17 kJ, respectively). However, relative to the amount of energy provided by dietary fat, its effect on satiety is weak. Furthermore, fat balance is not well regulated, as the capacity for fat storage (in adipose tissue) is virtually unlimited, and excess dietary fat does not markedly increase fat oxidation.

In contrast, protein and carbohydrate balance are well regulated, as they are preferentially oxidised due to their low storage capacity. The storage capacity for protein is limited, and as a result amino acid metabolism is tightly regulated to ensure oxidation of any excess. The capacity for carbohydrate storage (as glycogen) is small, and carbohydrate metabolism is also tightly regulated, with changes in carbohydrate intake being accompanied by changes in carbohydrate oxidation (autoregulation). Excess carbohydrate can be converted to fat, although this is uncommon unless a large excess of carbohydrate is consumed. However, when carbohydrate is oxidised, fat oxidation is reduced so that dietary fat is stored and endogenous fat retained (WHO 2000b).

These factors, together with the high palatability of foods containing fat, may explain why people exposed to high-fat foods are more likely to consume excess energy (passive overconsumption) and gain weight (Astrup 2001). In contrast, people exposed to low-fat foods tend to consume less energy and lose weight, even when there is no restriction on energy intake. For example, a meta-analysis of 18 controlled, ad libitum, low-fat dietary intervention trials (1728 normal and overweight subjects) lasting 2–12 months, found that a reduction in dietary energy from fat was associated with a net weight loss of 2.5 kg, which is equivalent to a 0.44 kg weight loss for a one percentage point decrease in energy consumed as fat in overweight subjects (BMI  $\sim 30$ ) (Astrup et al 2000). Although normal-

weight subjects consuming the low-fat diet did not necessarily lose weight, they did not experience the weight gain observed in the control group consuming usual-fat diets.

In another meta-analysis, based on 37 dietary intervention trials involving a reduction in dietary fat, the net weight loss in the intervention group was 2.8 kg (Yu-Poth et al 1999). The meta-analysis also found a highly significant relationship between reduction in dietary fat and weight loss, with a one percentage point-decrease in energy consumed as fat associated with a 0.28 kg decrease in body weight over a period of about two months.

A recent systematic review of 28 dietary intervention trials found that a reduction in dietary fat, without restriction of total food intake, was associated with weight loss in a dose-dependent fashion (Bray and Popkin 1998). Overall, a 10 percentage-point decrease in energy from fat was associated with a 16 g/day weight loss, which is equivalent to a 5.8 kg weight loss over one year. Based on these findings, the authors concluded that dietary fat does play an important role in the development of overweight, since diets low in fat result in weight loss (Bray and Popkin 1998).

Despite the body of evidence suggesting that high-fat diets are associated with higher energy intakes and greater body weight, some researchers argue that dietary fat cannot be a factor in the development of obesity because not all people consuming high-fat diets are overweight, and because dietary surveys indicate that the percentage of energy derived from fat has declined at the same time as the prevalence of obesity has increased (Willett 1998). Based on pooled data from a range of studies, dietary fat intake in individuals decreased in the United States from the mid-1960s to mid-1980s (Stephen and Wald 1990) and decreased in the United Kingdom from the mid-1970s to mid-1980s (Stephen and Sieber 1994). Data from the United States Continuing Survey of Food Intake of Individuals demonstrate a downward trend in fat intake as a percentage of total energy between 1965 and 1995, but an upward trend in absolute fat intake (grams/day) between 1989 and 1995 (Kennedy et al 1999). In the United States, by the mid-1990s the proportion of fat in the diet from mixed dishes, snack foods and fried potatoes had increased and started to offset reductions in fat from dairy, red meat and added fats (Popkin et al 2001).

Other researchers question whether dietary fat intake is really decreasing or is just reported to be decreasing (Heitmann et al 2000). Apparent declines in dietary fat intake may be due to selective under-reporting of certain foods. There is now considerable evidence indicating that both obese and non-obese individuals under-report fat intake more frequently than they under-report protein or carbohydrate intake (Poppitt et al 1998; Voss et al 1998; Samaras et al 1999). Under-reporting of fat intake probably occurs because people tend to under-report foods considered less healthy (eg, fast foods, confectionery, cakes and pastries, and snack foods) (Heitmann and Lissner 1995; Poppitt et al 1998; Lafay et al 2000). Trends in blood cholesterol levels do, however, suggest that intakes of saturated fat – and possibly total fat – have in fact decreased over recent decades, at least up until the mid-1990s.

### ***Dietary patterns***

Dietary patterns are the types and amounts of foods eaten, the settings in which foods are eaten, and the timing of eating. In recent years, dietary patterns have changed

considerably, with more people eating meals prepared outside the home and snacking between meals. In the United States, the proportion of total energy obtained from restaurants and fast-food establishments increased two- to three-fold between 1977 and 1996 (Nielsen et al 2002). A recent United States study found significant shifts away from meals to snacks in people of all ages: energy obtained from snacks increased from 11 to 18 percent between 1977 and 1996 (Nielsen et al 2002). These changes in dietary patterns are of concern as meals prepared outside the home and snack foods tend to be high in fat and/or sugar and low in nutrients.

Portion sizes also appear to be increasing, with larger portion sizes generally offering the best value for money. However, larger meals are higher in energy and have been linked to weight gain (Pearcey and de Castro 2002). The increased range of processed and convenience foods available is also a concern as many of these foods contain hidden fat and sugar, a technique used by food manufacturers to increase palatability (WHO 2000b).

These changing dietary patterns have led to an increase in the proportion of energy provided by foods considered energy-dense and nutrient-poor, such as cakes and biscuits, pizza and other fast foods, salty snacks, confectionery, and sugar-sweetened drinks. Consumption of these energy-dense nutrient-poor foods is positively correlated with both energy intake and the percentage of energy derived from fat (Kant 2000). Frequency of consumption of restaurant food has also been shown to be positively associated with body fatness (McCrory et al 1999).

Many factors have contributed to changes in dietary patterns in recent years. Advances in technology and globalisation have resulted in a wider range of highly palatable foods being readily available. There has been a rapid increase in the number of fast-foods outlets, restaurants and cafes, processed foods, and prepackaged meals. The percentage of females in the workforce has risen, resulting in more disposable income and increased demand for convenience foods. Many people are not learning how to cook and lack the skills to prepare a meal from scratch. Fast foods are aggressively advertised and are far more likely to be advertised than are healthy foods. For example, a recent New Zealand study found that two-thirds of food advertisements targeted at children were for food high in fat and/or sugar (Wilson et al 1999).

## **Physical activity**

Physical activity is the most variable and modifiable determinant of energy expenditure. An increase in energy intake does not result in weight gain provided that energy balance is maintained through increased energy expenditure (ie, increased physical activity). ‘Physical activity’ refers to all movement produced by skeletal muscles that increases energy expenditure, whether it is incidental (associated with everyday activities), occupational or recreational.

In most developed countries there has been a dramatic decrease in non-recreational physical activity in the last few decades. This decrease is due to a wide range of factors, including fewer manual jobs, greater reliance on motorised transport, and more labour-saving devices at home (WHO 2000b). There has also been an increase in sedentary pastimes, such as watching television or videos, and playing video or computer games. Television viewing is likely to contribute to the development of obesity through several pathways: displacement of physical activity, increased energy consumption while watching (ie, snacking), changed food purchasing behaviour due to exposure to food advertising, and reduced resting metabolism (Robinson 2001).

Many large cross-sectional studies have found that low levels of physical activity are associated with higher body weight or BMI. A study of 15,239 adults in the European Union found that leisure time physical activity was inversely associated with BMI, whereas sedentary behaviour was positively associated with BMI (Martinez-Gonzalez et al 1999). Compared with the most physically inactive quintile, those in the uppermost quintile of physical activity had a 50 percent lower risk of obesity (Martinez-Gonzalez et al 1999). The Seven Countries Study, which involved 12,763 middle-aged men, found that physical activity levels were inversely related to indices of body fatness (Kromhout et al 2001). In the Nurses' Health Study and Physicians' Health Study, BMI at baseline increased as the level of physical activity decreased (Lee et al 1999; Rockhill et al 2001).

Prospective studies confirm that low levels of physical activity are associated with greater weight gain. In the Health Professionals Follow-up Study, television and video viewing was associated with weight gain, whereas vigorous activity was associated with weight reduction over a four-year period (Coakley et al 1998). In the Nurses' Health Study, weight change since age 18 years was inversely associated with physical activity levels (Rockhill et al 2001). The Nurses' Health study has also shown that sedentary behaviours, especially television watching, were associated with a significantly increased risk of developing obesity and diabetes over a six-year period (Hu et al 2003). In a cohort of almost 80,000 Americans, 10-year changes in body mass index were inversely associated with walking four or more hours per week and performing vigorous activity (eg, jogging) one to three hours per week (Kahn et al 1997).

Numerous studies have examined the impact of increasing physical activity, either alone or in combination with diet, on body weight or BMI. These studies indicate that physical activity reduces body weight, and improves body composition by promoting fat loss and preserving or increasing lean body mass (Stefanick 1993). There is still some debate regarding the volume (ie, intensity, duration and frequency) of physical activity required to bring about and maintain weight loss, but, in general, the rate of weight loss is positively correlated with the frequency and duration of physical activity. Compared to diet, the effects of increased physical activity are less important in the short term. However, a recent meta-analysis suggests that in subjects who have lost weight, regular physical activity is an important predictor of long-term maintenance of weight loss (Anderson et al 2001). Increased physical activity may facilitate weight loss and maintenance of weight loss by increasing energy expenditure during and after exercise, stimulating fat (as opposed to carbohydrate) oxidation, preserving lean body mass, and possibly reversing diet-induced suppression of basal metabolic rate (Stefanick 1993).

## 7.3 BMI distributions

### Current distribution

Data on BMI by age group, sex and ethnicity were obtained from the 1997 NNS dataset. Mean and standard deviation for BMI, by ethnic group, age group and sex are shown below (Tables 78–79). BMI increases with age until age 45–64 years, then declines slightly in older age groups. BMI is considerably higher in Māori than in non-Māori New Zealanders.

**Table 78:** Mean body mass index (kg/m<sup>2</sup>), by ethnicity, sex and age group, 1997

	Sex	Age group (years)								
		15–24	25–34	35–44	45–54	55–64	65–74	75+	Total	Adjusted <sup>1</sup>
Non-Māori	Males	23.6	25.4	26.4	27.1	27.1	26.5	25.4	25.8	25.7
	Females	23.6	24.8	25.9	27.0	27.6	27.3	25.4	25.8	25.6
Māori	Males	25.0	28.6	31.0	31.9	31.5	28.4	25.8	28.7	28.9
	Females	26.3	28.1	29.8	31.1	31.4	30.4	28.6	28.7	29.0
Total	Males	23.9	25.8	26.9	27.5	27.4	26.7	25.3	26.2	26.1
	Females	24.1	25.3	26.4	27.4	27.9	27.4	25.5	26.1	26.0

<sup>1</sup> Age-standardised to WHO World population.

**Table 79:** Standard deviation body mass index (kg/m<sup>2</sup>), by ethnicity, sex and age group, 1997

	Sex	Age group (years)								
		15–24	25–34	35–44	45–54	55–64	65–74	75+	Total	Adjusted <sup>1</sup>
Non-Māori	Males	4.3	3.6	3.9	4.0	4.2	3.3	2.8	4.0	3.9
	Females	5.0	4.9	5.7	5.9	5.1	4.8	4.7	5.5	5.2
Māori	Males	5.8	6.4	5.4	6.3	4.5	6.0	4.0	6.3	5.6
	Females	5.8	5.6	6.4	6.1	7.1	6.0	5.0	6.3	6.0
Total	Males	4.7	4.1	4.3	4.4	4.4	3.4	2.8	4.4	4.2
	Females	5.2	5.2	6.0	6.0	5.5	4.9	4.8	5.6	5.5

<sup>1</sup> Age-standardised to WHO World population.

### Trends and international comparisons

Mean BMI and the prevalence of overweight and obesity have been increasing in developed countries for the last two decades. The speed and extent of the increase in obesity has led the WHO to describe it as an epidemic (WHO 2000b). In New Zealand adults aged 15 years and over, the prevalence of obesity increased from 9.5 to 14.7 percent in males and from 12.6 to 19.2 percent in females between 1989 and 1997, an increase of more than 50 percent for both males and females (Russell et al 1999). During the same eight-year period, mean BMI increased from 25.3 to 26.2 kg/m<sup>2</sup> in males and from 24.7 to 26.1 kg/m<sup>2</sup> in females.



In English adults aged 16 years and over, BMI increased from 25.9 to 27.0 kg/m<sup>2</sup> in males and from 25.7 to 26.7 kg/m<sup>2</sup> in females between 1993 and 2001 (Department of Health [London] 2003). During the same period, the prevalence of obesity increased from 13.2 to 21.0 percent in males and from 16.4 to 23.5 percent in females.

The prevalence of obesity has also increased in Australia and Scotland in recent years (The Scottish Executive Department of Health 2000; AIHW 2001). In Scottish adults aged 16–74 years, mean BMI was 26.5 kg/m<sup>2</sup> in both males and females in 1998, and the prevalence of obesity was 19.6 and 22.1 percent in males and female respectively (The Scottish Executive Department of Health 2000). In Australian adults aged 25 years and over, mean BMI was 26.9 kg/m<sup>2</sup> in males and 26.4 kg/m<sup>2</sup> in females in 1999/2000, and the prevalence of obesity was 19.3 and 22.2 percent in males and females respectively (Cameron et al 2003).

Information on mean BMI could not be located for the United States, but the prevalence of obesity (BMI ≥ 30) has increased in that country. In US adults aged 20–74 years, the prevalence of obesity increased from 20.2 to 27.5 percent in males and from 25.4 to 33.4 percent in females between 1988–94 and 1999–2000 (Flegal et al 2002).

The increases in mean BMI and in the prevalence of obesity in New Zealand and other similar countries are thought to be explained largely by a combination of sedentary lifestyles and relative over-consumption of energy-dense foods. Table 80 shows the most recent data on the prevalence of obesity and mean BMI (where available) in New Zealand compared with other similar countries.

**Table 80:** International comparisons of mean body mass index and the prevalence of obesity\*

Country, year	Body mass index (kg/m <sup>2</sup> )		Obesity (percent)	
	Males	Females	Males	Females
New Zealand, 1997	26.2	26.1	14.7	19.2
Australia, 1999/2000	26.9	26.4	19.3	22.2
England, 2001	27.0	26.7	21.0	23.5
Scotland, 1998	26.5	26.5	19.6	22.1
United States, 1999/2000	Not available	Not available	27.5	33.4

\* Body mass index ≥ 30 kg/m<sup>2</sup>, except for New Zealand Māori and Pacific ethnic groups, for whom BMI ≥ 32 kg/m<sup>2</sup>.

Source: Russell et al 1999; Cameron et al 2003; Department of Health [London] 2003; The Scottish Office Department of Health 1997; Flegal et al 2002.

## Theoretical minimum

The theoretical minimum is the risk factor (ie, BMI) distribution that would yield the lowest population risk of adverse health outcomes. In developed countries, the relationship between BMI and mortality is continuous down to BMI values of 19 to 21 kg/m<sup>2</sup> (Willett et al 1999). For this study, calculations of attributable and avoidable mortality were based on a theoretical minimum BMI of 21 ± 1 kg/m<sup>2</sup> for all age, sex and ethnic groups. This theoretical minimum is based on that chosen by the WHO overweight and obesity expert working group for the *World Health Report 2002* (Ezzati et al 2002; James et al, in press).

## Distributional transitions

Two distributional transitions for BMI were estimated: a business as usual (BAU) scenario (historical trend) and an intervention scenario (deviation from the historical trend reflecting policy change). Distributional transitions were based on trends in mean BMI and the prevalence of overweight and obesity in New Zealand and other developed countries. Unlike other risk factors, neither the BAU nor the intervention scenarios for BMI involve an improvement in the current situation, since most experts believe the obesity epidemic has yet to peak.

Distributional transitions were expressed as the percentage shift from the current distribution away from the theoretical minimum. For BMI, distributional transitions were assumed to be the same for all age, sex and ethnic groups. However, absolute changes in BMI vary by age, sex and ethnicity depending on baseline BMI values.

The avoidable burden is the difference between the projected BAU scenario and the intervention scenario, with the shift between the two scenarios being maintained over time.

### *Business as usual scenario*

Given that current evidence suggests that BMI will continue to increase, under the BAU scenario we estimated an increase in the population mean BMI of 1.3 kg/m<sup>2</sup> in both males and females by 2011. This increase in mean population BMI means that the current BMI distribution would shift 25 percent further away from the theoretical minimum in both males and females. Absolute changes in BMI vary by age, ranging from approximately 1.1 kg/m<sup>2</sup> in young adults to 1.6 kg/m<sup>2</sup> in middle-aged adults. Table 81 shows the projected BMI levels in 2011, by ethnicity, sex and age under the BAU scenario.

**Table 81:** Projected 2011 body mass index levels under the business as usual scenario

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Non-Māori	Males	26.5	27.8	28.6	28.6	27.9	26.5	27.0
	Females	25.8	27.1	28.5	29.3	28.9	26.5	27.0
Māori	Males	30.5	33.5	34.6	34.1	30.3	27.0	30.6
	Females	29.9	32.0	33.6	34.0	32.8	30.5	30.6
Total	Males	27.0	28.4	29.1	29.0	28.1	26.4	27.5
	Females	26.4	27.8	29.0	29.6	29.0	26.6	27.4

### Intervention scenario

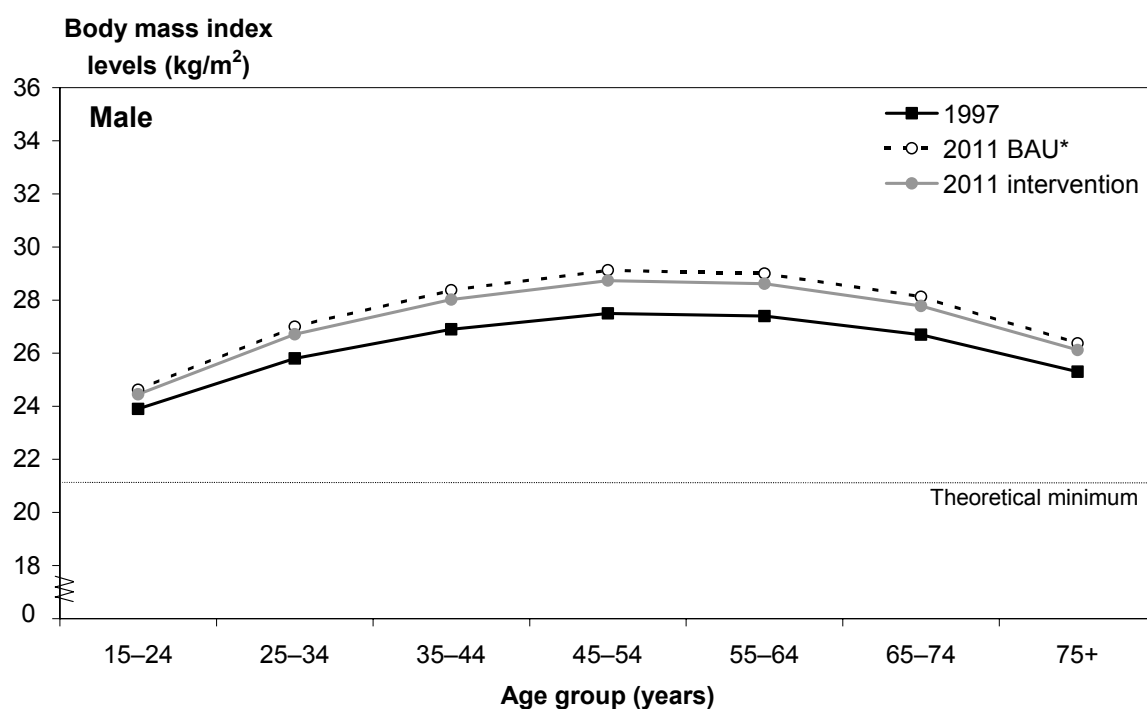
Under the more optimistic intervention scenario, we estimated that the increase in BMI by 2011 would be smaller, with an increase in the population mean BMI of 1.0 kg/m<sup>2</sup> in both males and females (0.3 kg/m<sup>2</sup> smaller increase in BMI in both males and females compared to the BAU scenario). This increase in mean population BMI implies that the current BMI distribution will shift 19 percent further away from the theoretical minimum in both males and females. Absolute changes in BMI vary by age, ranging from approximately 0.8 kg/m<sup>2</sup> in young adults to 1.2 kg/m<sup>2</sup> in middle-aged adults. Table 82 shows the projected BMI levels in 2011 by ethnicity, sex and age under the intervention scenario.

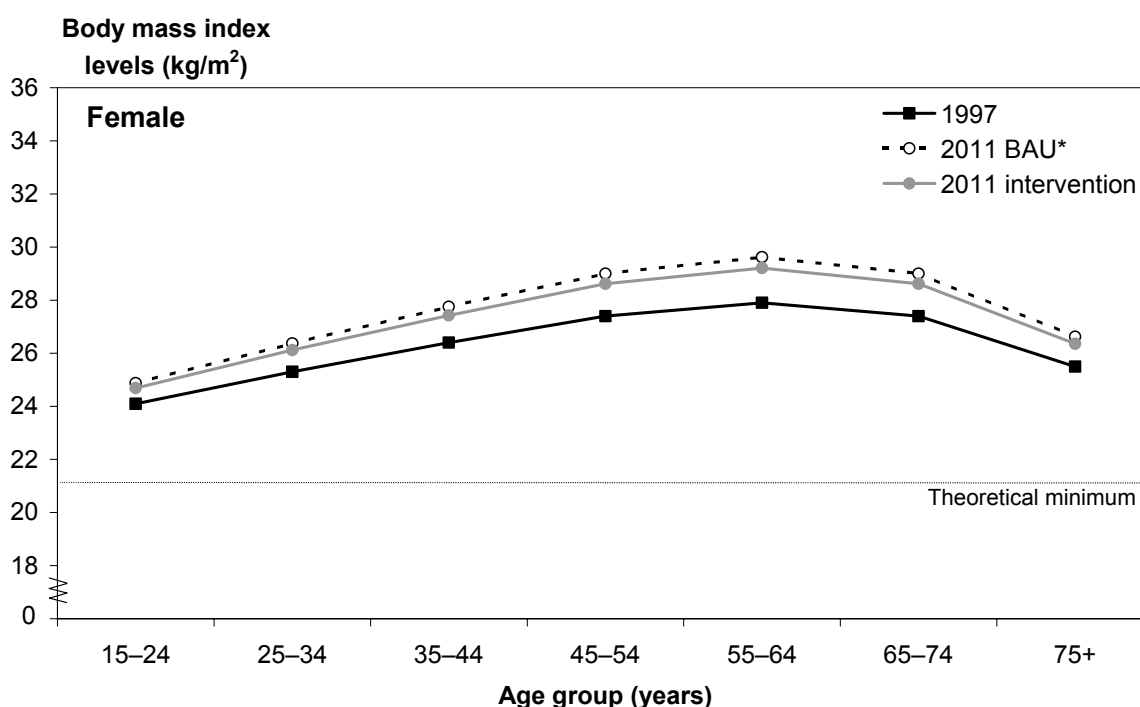
**Table 82:** Projected 2011 body mass index levels under the intervention scenario

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Non-Māori	Males	26.2	27.4	28.3	28.3	27.5	26.2	26.7
	Females	25.5	26.8	28.1	28.9	28.5	26.2	26.7
Māori	Males	30.0	32.9	34.0	33.5	29.8	26.7	30.2
	Females	29.4	31.5	33.0	33.4	32.2	30.0	30.2
Total	Males	26.7	28.0	28.7	28.6	27.8	26.1	27.2
	Females	26.1	27.4	28.6	29.2	28.6	26.4	27.1

Figure 29 shows the current (1997) BMI distribution, as well as projected 2011 levels under the BAU and intervention scenarios (separately for males and females) in relation to the theoretical minimum.

**Figure 29:** Current (1997) and future (2011) body mass index levels





\* BAU = business as usual

## 7.4 Risk factor–disease relationships

### Disease outcomes

The outcomes assessed were based on those selected for the *World Health Report 2002* (WHO 2002):

- ischaemic heart disease
- ischaemic stroke
- type 2 diabetes
- colorectal cancer
- post-menopausal breast cancer.

The selection of outcomes was based on strong evidence of a causal relationship and sufficient data to quantify the risk factor–disease relationship. The *World Health Report* also included hypertensive heart disease, osteoarthritis and endometrial cancer. Hypertensive disease and osteoporosis were not included in this study because it was limited to fatal outcomes and these conditions do not cause significant mortality (directly). Endometrial cancer was not included in our study because this disease makes only a negligible contribution to total mortality. Other outcomes likely to be causally associated with BMI, but not included in the *World Health Report* or our study because of a negligible contribution to total mortality or a lack of data to quantify the risk factor–disease association, include kidney cancer, back pain, menstrual disorders and infertility, dermatitis and gallstones (Ezzati et al 2002; James et al, in press).

## **Risk accumulation**

Risk accumulation refers to the nature and strength of the association between the exposure (BMI) and disease. Risk accumulation is expressed as the regression (risk) coefficient in the case of a continuous risk factor (ie, the increase in incidence or mortality of the disease per unit increase in exposure).

For cardiovascular disease endpoints (IHD and ischaemic stroke mortality), data from the Asia Pacific Cohort Studies Collaboration (APCSC) were used to estimate risk accumulation (James et al, in press). The APCSC included data from 33 cohorts in eight countries. The advantage of the APCSC study was that it included individual participant data (310,283 participants), and therefore it could more reliably control for confounding and provide estimates of risk accumulation for different age groups. Another advantage of the APCSC overview is that it includes data from Australia and New Zealand (ANZ). Approximately 20 percent of the sample was from ANZ, the remainder being from mainland China (15 percent), Japan (10 percent), South Korea (52 percent) and other Asian countries (4 percent). The mean age of participants at baseline was 47 years, and 60 percent were male. BMI measurements are based on measured height and weight. Data from participants recorded as having a BMI outside the feasible range (12–60 kg/m<sup>2</sup>) were excluded from analyses. The overall mean BMI at baseline was 23.6 kg/m<sup>2</sup>. Mean BMI was higher for the ANZ populations (26.4 kg/m<sup>2</sup>) than the Asian populations (22.9 kg/m<sup>2</sup>). The mean duration of follow-up was 6.9 years. As there was evidence of confounding due to disease at baseline, the first three years of follow-up were excluded from analyses. Smoking was included as a covariate in all analyses, since smoking reduces BMI while increasing mortality risk. Since cholesterol and blood pressure are intermediate variables on the causal pathway between BMI and CVD, they were not adjusted for in the analysis.

For diabetes, data from both unpublished and published sources were used to estimate risk accumulation (James et al, in press). These sources include unpublished Japanese data (from a nationally representative sample) of the prevalence of diabetes at different BMI levels by age and a similar Danish study (Drivsholm et al 2001) of middle-aged men and women. Further details on the derivation of risk accumulation estimates can be found in James et al (in press).

For colorectal cancer and post-menopausal breast cancer, data from a recent meta-analysis were used (Bergström et al 2001). Studies included in the meta-analysis were identified through a systematic search of the literature. BMI was used as the measure of excess weight and was measured on a continuous scale. For each cancer site, three meta-analyses were performed. The first analysis included all eligible studies suitable for meta-analysis, the second was restricted to studies with incident cases as outcome, and the third was further restricted to studies accounting for major confounders.

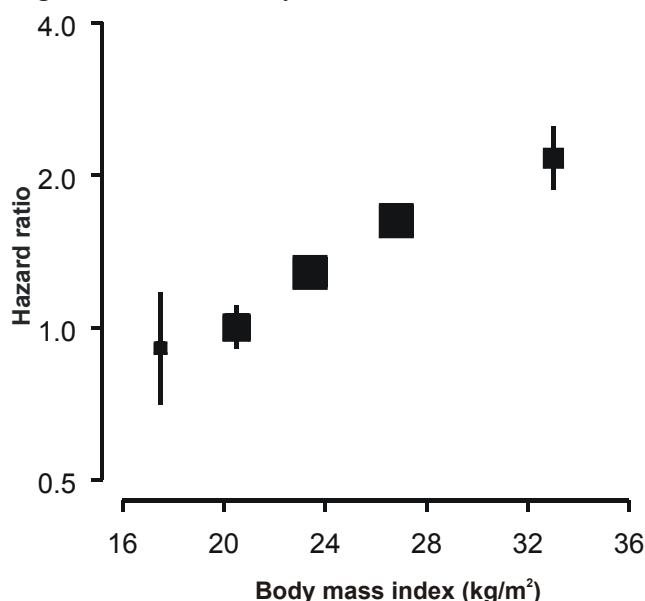
Data from the meta-analysis carried out by Bergström et al (2001) show a positive association between BMI and colorectal cancer. Six studies were included in the meta-analysis (four cohort and two case-control studies). When results were available by sex, these were abstracted separately and considered as independent observations in the meta-analysis. Age, sex, family history of colon cancer, ethnicity, social class, physical exercise and diet were considered as potential confounders. Most studies accounted for age and one other of these confounders. Restriction of the analysis to studies having incident cases as outcome did not affect the estimate.

A positive association was also found between BMI and post-menopausal breast cancer. Thirteen studies were included in the meta-analysis (nine case-control and four cohort studies). Limiting the meta-analysis to the three studies that adjusted for age, reproductive factors and alcohol or diet marginally affected the point estimate.

### ***Ischaemic heart disease***

Numerous observational studies have demonstrated a strong positive association between BMI and IHD. Data from the APCSC overview (James et al, in press) show that the relative risk of IHD increases with increasing BMI, and that the association is roughly linear when plotted on a log scale (Figure 30). The association is continuous, down to a BMI of about 21 kg/m<sup>2</sup>.

**Figure 30:** Mean body mass index and risk of ischaemic heart disease (ages pooled)



Source: Asia Pacific Cohort Studies Collaboration.

As shown in Table 83, the association between BMI and IHD varies with age: it is strongest in young adults and then attenuates with age. Since the APCSC data provide no evidence that the strength of the association between BMI and IHD varies by sex, it was not necessary to have different estimates for males and females. As the strength of the association between BMI and risk of IHD was similar in the ANZ and Asian cohorts within the APCSC, and in North American and European cohorts, it was assumed that the relationship between BMI and IHD was the same for all ethnic groups. The APCSC data provided no evidence of a differential association for fatal and non-fatal IHD endpoints.

The risk coefficients allow for partial deconfounding by covariates such as blood pressure and total cholesterol, which are both independent risk factors for IHD and also mediating variables on the pathway linking BMI to IHD outcomes. Adjustment is not made for diabetes, as much of the association between BMI and IHD operates through this pathway.

In summary, a 1 kg/m<sup>2</sup> lower BMI is associated with a lower risk of IHD of 12 percent in younger adults, dropping to a 4 percent lower risk in adults aged 75 years and over.

**Table 83:** Risk coefficients for ischaemic heart disease for a 1 kg/m<sup>2</sup> lower body mass index

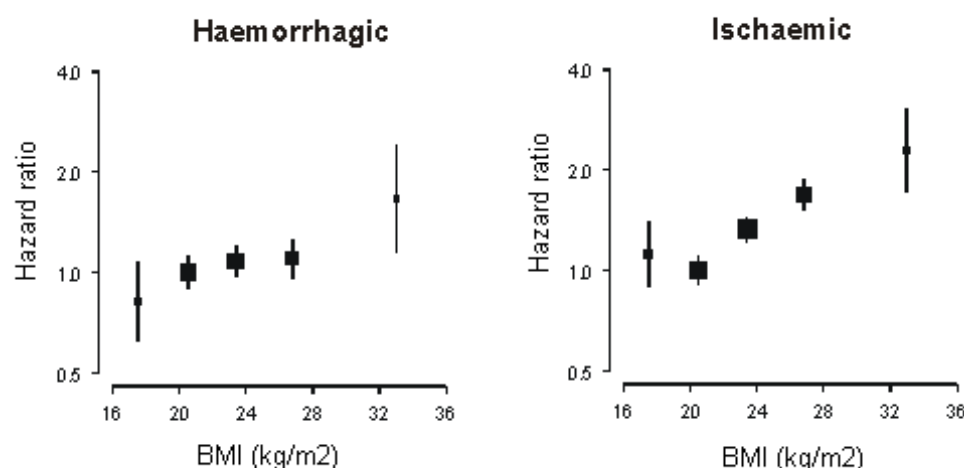
Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.88	12
35–44	0.89	11
45–54	0.91	9
55–64	0.93	7
65–74	0.95	5
75+	0.96	4

Source: Asia Pacific Cohort Studies Collaboration.

### ***Ischaemic stroke***

Data from the APCSC overview show a continuous relationship between increasing BMI and risk of ischaemic stroke, with a weaker association between BMI and risk of haemorrhagic stroke (Figure 31). Overall, a 1 kg/m<sup>2</sup> lower BMI was associated with a 6 percent lower risk of ischaemic stroke and a 3.5 percent lower risk of haemorrhagic stroke in the APCSC analyses (James et al, in press).

**Figure 31:** Mean body mass index and risk of ischaemic and haemorrhagic stroke (ages pooled)



Source: Asia Pacific Cohort Studies Collaboration.

As the APCSC data provided no evidence that the strength of association between BMI and ischaemic stroke varies by sex, it is not necessary to have different risk estimates for males and females. It was assumed that the relationship between BMI and ischaemic stroke was the same for all ethnic groups.

The risk coefficients allow for partial deconfounding by covariates such as blood pressure and total cholesterol, which are both independent risk factors for ischaemic stroke and also mediating variables on the pathway linking BMI to ischaemic stroke. No adjustment is made for diabetes which is considered to be fully a mediating variable.

In summary, a 1 kg/m<sup>2</sup> lower BMI is associated with a lower risk of ischaemic stroke of 13 percent in younger adults, dropping to a 4 percent lower risk in adults aged 75 years and over.

**Table 84:** Risk coefficients for ischaemic stroke for a 1 kg/m<sup>2</sup> lower body mass index

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.87	13
35–44	0.88	12
45–54	0.90	10
55–64	0.92	8
65–74	0.94	6
75+	0.96	4

Source: Asia Pacific Cohort Studies Collaboration.

## Diabetes

The association between BMI and diabetes was generally stronger in females than in males. In females, the association was strongest in young adults and then attenuated with age. A similar pattern was seen for males, with some fluctuations in age groups greater



than 55 years. It was assumed that the relationship between BMI and diabetes was the same for all ethnic groups.

In summary, a 1 kg/m<sup>2</sup> lower BMI is associated with a lower risk of type 2 diabetes of 32 percent in younger adults, dropping to a 17 percent lower risk in adults aged 75 years and over. These risk co-efficients are much larger than those for any other BMI-disease association.

**Table 85:** Risk coefficients for diabetes for a 1 kg/m<sup>2</sup> lower in body mass index

Age group (years)	Males		Females	
	Risk coefficient	Risk reduction (%)	Risk coefficient	Risk reduction (%)
25–34	0.74	26	0.68	32
35–44	0.74	26	0.68	32
45–54	0.81	19	0.75	25
55–64	0.83	17	0.79	21
65–74	0.82	18	0.83	17
75+	0.79	21	0.83	17

Source: WHO obesity and overweight expert working group.

### ***Colorectal cancer***

Overall, a 1 kg/m<sup>2</sup> lower BMI was associated with a 3 percent lower risk of colorectal cancer. There was no evidence that the association between BMI and colorectal cancer varies by age or sex (Table 86). It was assumed the relationship between BMI and colorectal cancer was the same for all ethnic groups.

**Table 86:** Risk coefficients for colorectal cancer for a 1 kg/m<sup>2</sup> lower body mass index

Age group (years)	Risk coefficient	Risk reduction (%)
25–34	0.97	3
35–44	0.97	3
45–54	0.97	3
55–64	0.97	3
65–74	0.97	3
75+	0.97	3

Source: WHO obesity and overweight expert working group.

### ***Post-menopausal breast cancer***

Overall, a 1 kg/m<sup>2</sup> lower BMI was associated with a 3 percent lower risk of breast cancer in post-menopausal women (aged 50 years and over) (Table 87). It was assumed that the relationship between BMI and post-menopausal breast cancer was the same for all ethnic groups.

**Table 87:** Risk coefficients for post-menopausal breast cancer for a 1 kg/m<sup>2</sup> lower body mass index

Age group (years)	Risk coefficient	Risk reduction (%)
45–54	0.97	3
55–64	0.97	3
65–74	0.97	3
75+	0.97	3

Source: WHO obesity and overweight expert working group.

## Risk reversal

Risk reversal refers to how quickly and how completely risk of disease is reversed following a reduction in BMI. Interventions involving individual behavioural changes (diet and/or physical activity) result in weight loss, a reduction in BMI and the prevalence of obesity, and improvements in cardiovascular and diabetes risk profiles in the short term, but have rarely been shown to be effective in the long term.

Nevertheless, based on limited evidence, mainly from short-term clinical trials, the estimates of risk reversibility used for this study were as follows:

- 100 percent risk reversibility for IHD and stroke within five years
- 100 percent risk reversibility for diabetes within two to three years
- 84 percent risk reversibility for cancer outcomes within 10 years.

These risk-reversal estimates are based on those selected by the WHO obesity and overweight expert working group for the *World Health Report 2002*, details of which will be published in a forthcoming technical report (James et al, in press). It was assumed that risk reversibility is the same for both sexes and all ethnic groups.

## 7.5 Attributable mortality

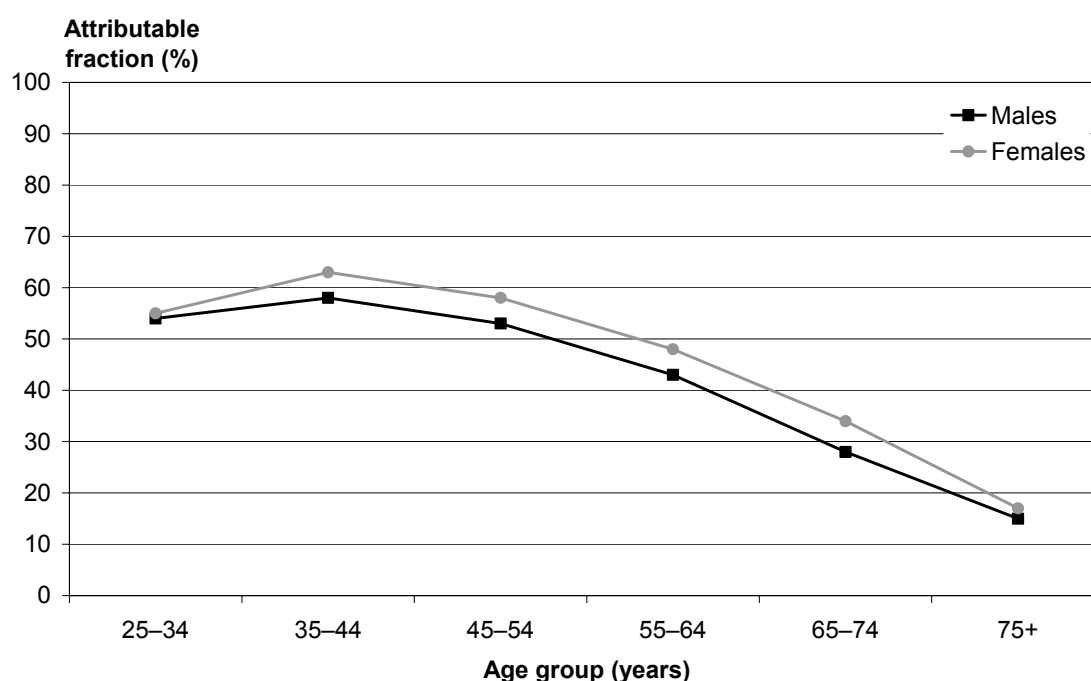
Combining the BMI distributional estimates with the risk accumulation and reversal estimates allows attributable and avoidable fractions to be calculated. These fractions can then be applied to estimates of current and projected burden to calculate attributable and avoidable burden respectively.

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### Ischaemic heart disease

In the two oldest age groups, where the majority of IHD deaths occur, approximately 20–30 percent of IHD was attributable to ‘high’ BMI (Figure 32).

**Figure 32:** Attributable fractions (%) for body mass index and ischaemic heart disease, 1997



Overall, ‘high’ BMI contributed to 1561 (95% CI 1243–1852) IHD deaths (6 percent of all deaths) and 17,910 YLL in 1997 (Table 88). The majority (almost 70 percent) of IHD deaths attributable to ‘high’ BMI occurred in those aged 65 years and older.

IHD mortality attributable to ‘high’ BMI was about two-fold higher in males than females (Table 88). This difference primarily reflects the higher IHD mortality in males, as attributable fractions were similar in males and females. The age-standardised IHD mortality rates attributable to ‘high’ BMI in Māori were about three times higher than the corresponding rates in non-Māori.

**Table 88:** Attributable mortality for body mass index and ischaemic heart disease, 1997

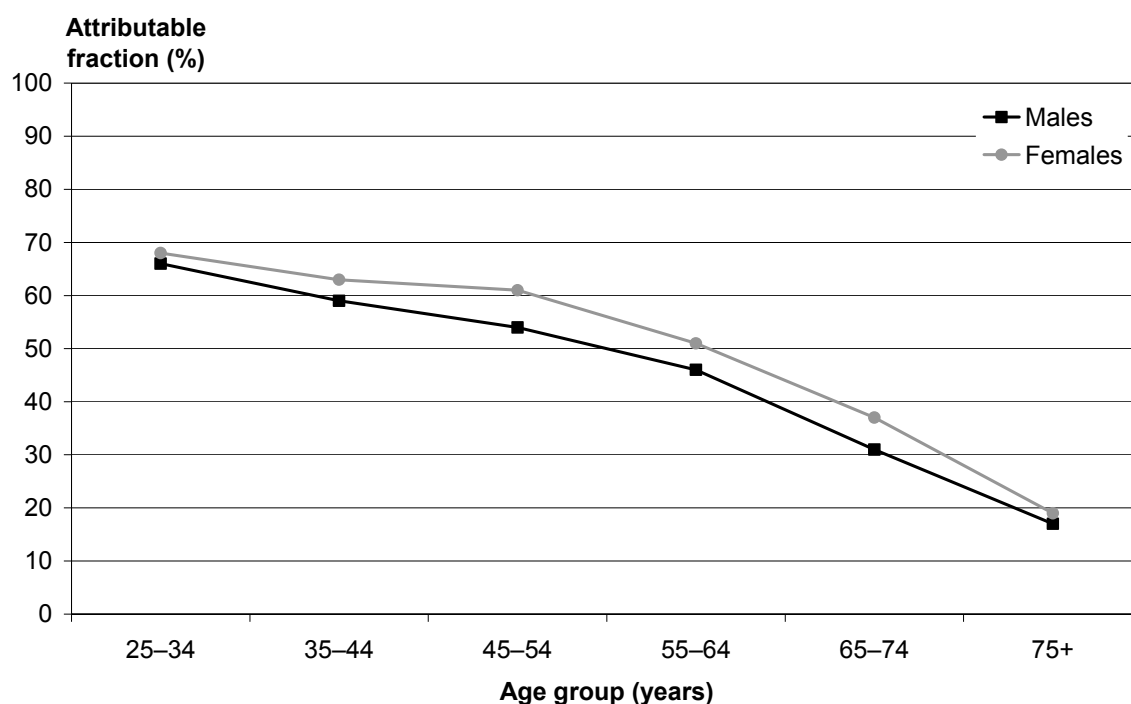
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	792	9,043	69.3	828
	Māori	148	2,385	183.0	2,633
	Total	940	11,428	78.2	979
Females	Non-Māori	541	5,309	32.6	359
	Māori	80	1,173	102.7	1,363
	Total	621	6,482	37.2	434

\* Rate per 100,000, age-standardised to WHO World population.

## Ischaemic stroke

In the two oldest age groups, where the majority of ischaemic stroke deaths occur, 20–30 percent of ischaemic stroke was attributable to ‘high’ BMI (Figure 33).

**Figure 33:** Attributable fractions (%) for body mass index and ischaemic stroke, 1997



Overall, ‘high’ BMI contributed to 367 (95% CI 174–517) ischaemic stroke deaths and 3284 YLL in 1997 (Table 89). The majority (almost 70 percent) of ischaemic stroke deaths attributable to ‘high’ BMI occurred in those aged 75 years and older.

The age-standardised ischaemic stroke mortality rate attributable to ‘high’ BMI was similar in males and females (Table 89). The age-standardised ischaemic stroke mortality rate attributable to ‘high’ BMI was about 1.5 times higher in Māori males than non-Māori males, and twice as high in Māori females compared to non-Māori females. This difference is primarily due to a greater prevalence of ‘high’ BMI in Māori compared to non-Māori, as ischaemic stroke mortality rates are similar for Māori and non-Māori.

**Table 89:** Attributable mortality for body mass index and ischaemic stroke, 1997

Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	130	1,091	10.8	94
	Māori	11	140	16.9	182
	Total	141	1,231	11.2	101
Females	Non-Māori	210	1,836	11.5	111
	Māori	16	217	21.6	263
	Total	226	2,053	12.2	123

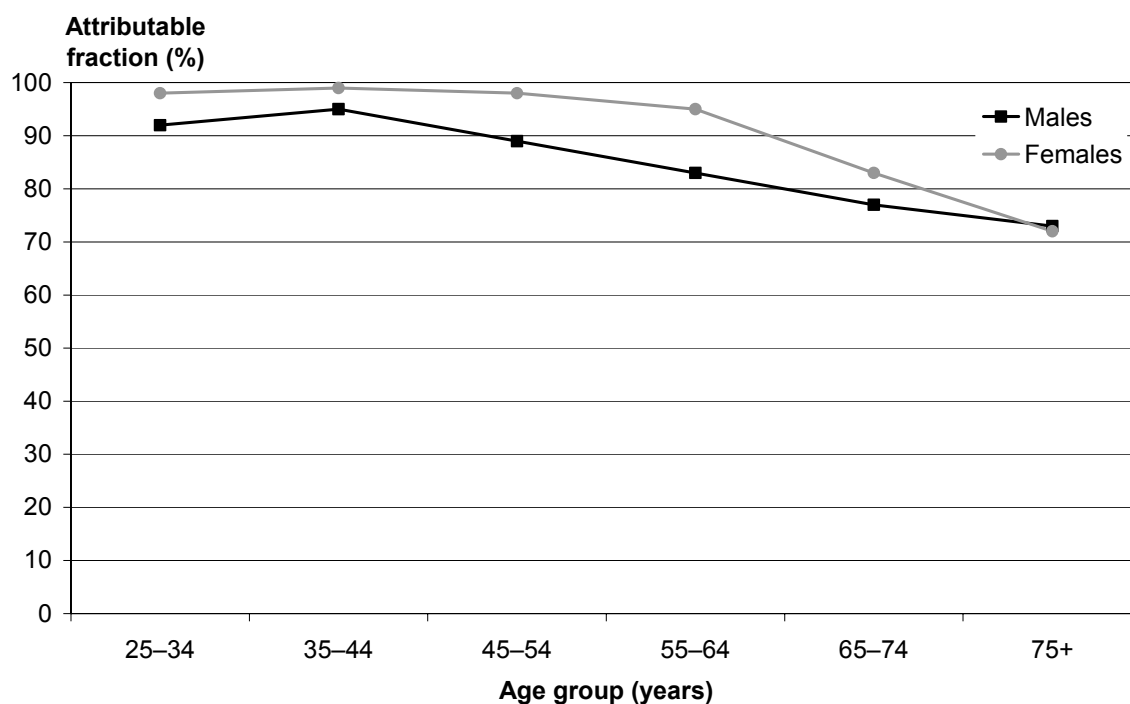
\* Rate per 100,000, age-standardised to WHO World population

Note that our analysis may overestimate the BMI-attributable stroke burden by using risk coefficients derived from pooled fatal and non-fatal ischaemic stroke outcomes, yet underestimate the burden by excluding haemorrhagic stroke from the model.

## Diabetes

In the older age groups, where the majority of diabetes deaths occur, approximately 80 percent of diabetes deaths were attributable to ‘high’ BMI (Figure 34).

**Figure 34:** Attributable fractions (%) for body mass index and diabetes, 1997



Overall, ‘high’ BMI contributed to 1231 (95% CI 1208–1247) diabetes deaths (5 percent of all deaths) and 16,535 YLL in 1997 (Table 90).

The age-standardised diabetes mortality rate attributable to ‘high’ BMI was slightly higher in males than in females (Table 90). The age-standardised diabetes mortality rate attributable to ‘high’ BMI was approximately 10 times higher in Māori males and females compared to non-Māori males and females. This difference is primarily due to the much higher diabetes mortality rate in Māori, and to a lesser extent a greater prevalence of ‘high’ BMI in Māori compared to non-Māori.

**Table 90:** Attributable mortality for body mass index and diabetes,<sup>1</sup> 1997

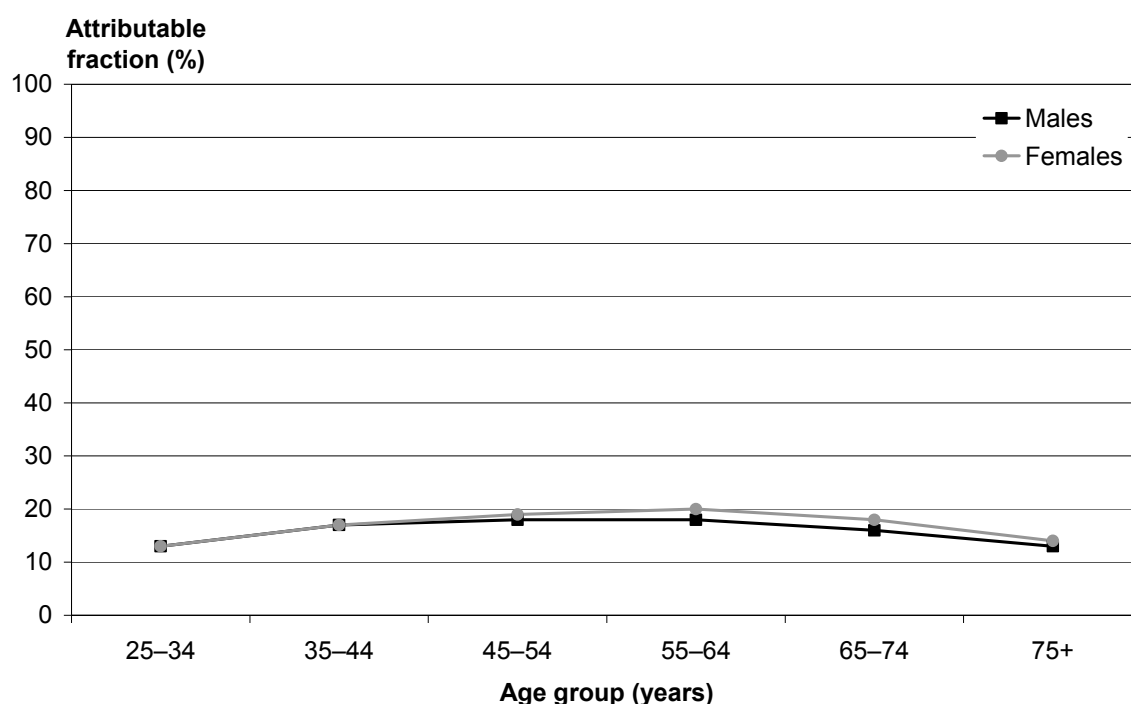
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate) <sup>2</sup>	Years of life lost (rate) <sup>2</sup>
Males	Non-Māori	415	4,875	36.3	442
	Māori	216	3,228	287.1	3,840
	Total	631	8,103	53.0	697
Females	Non-Māori	341	4,352	25.5	356
	Māori	259	4,080	311.1	4,565
	Total	600	8,432	44.9	674

1 Not adjusted for overlap with cardiovascular disease

2 Rate per 100,000, age-standardised to WHO World population

## Colorectal cancer

In the older age groups, where the most colorectal cancer deaths occur, approximately 15 percent of colorectal cancer was attributable to ‘high’ BMI (Figure 35).

**Figure 35:** Attributable fractions (%) for body mass index and colorectal cancer, 1997

Overall, ‘high’ BMI contributed to 177 (95% CI 156–197) colorectal cancer deaths and 2082 YLL in 1997 (Table 91). The majority (almost 70 percent) of colorectal cancer deaths attributable to ‘high’ BMI occurred in those aged 65 years and older.

The age-standardised colorectal cancer mortality rate attributable to ‘high’ BMI was slightly higher in males than in females (Table 91). Although Māori have higher BMIs than non-Māori, the age-standardised colorectal cancer mortality rate attributable to ‘high’

BMI was similar in Māori and non-Māori males and females because colorectal cancer mortality rates are apparently lower in Māori than in non-Māori.

**Table 91:** Attributable mortality for body mass index and colorectal cancer, 1997

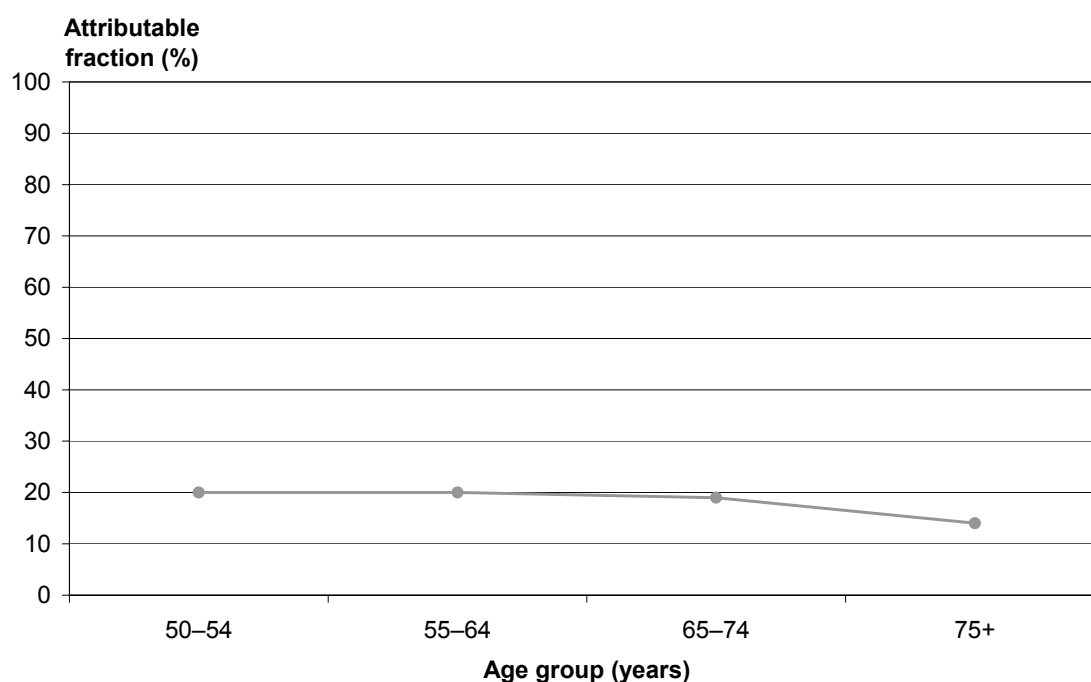
Sex	Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Males	Non-Māori	83	940	7.2	85
	Māori	5	74	7.5	93
	Total	88	1,014	7.3	86
Females	Non-Māori	84	998	5.9	79
	Māori	5	70	5.8	82
	Total	89	1,068	5.9	79

\* Rate per 100,000, age-standardised to WHO World population.

### Post-menopausal breast cancer

Approximately 15–20 percent of post-menopausal breast cancer deaths in 1997 were attributable to ‘high’ BMI (Figure 36).

**Figure 36:** Attributable fractions (%) for body mass index and post-menopausal breast cancer, 1997



Overall, ‘high’ BMI contributed to 91 (95% CI 76–106) post-menopausal breast cancer deaths and 1190 YLL in 1997 (Table 92).

The age-standardised post-menopausal breast cancer mortality rate attributable to ‘high’ BMI was twice as high in Māori females compared to non-Māori females (Table 92). This difference is primarily due to a greater prevalence of ‘high’ BMI in Māori compared to non-Māori, as the post-menopausal breast cancer rates are similar in Māori and non-Māori.

**Table 92:** Attributable mortality for body mass index and post-menopausal breast cancer, 1997

Ethnicity	Deaths (count)	Years of life lost (count)	Deaths (rate)*	Years of life lost (rate)*
Non-Māori	82	1,050	16.3	233
Māori	9	140	30.1	434
Total	91	1,190	17.2	247

\* Rate per 100,000, age-standardised to WHO World population.

### Total attributable mortality

Overall, 'high' BMI contributed to a total of 3154 deaths (11 percent of all deaths) and 37,373 YLL in 1997 (Table 93). This estimate adjusts for the estimated overlap between diabetes and cardiovascular disease mortality (only 78 percent of attributable diabetes deaths are included in this total).

**Table 93:** Attributable mortality for body mass index and all diseases, 1997

Sex	Ethnicity	Deaths		Years of life lost	
		Count	%*	Count	%*
Males	Non-Māori	1,328	10	14,877	11
	Māori	332	23	5,117	21
	Total	1,660	12	19,994	13
Females	Non-Māori	1,183	10	12,595	11
	Māori	311	27	4,784	25
	Total	1,494	11	17,379	13

\* % = percentage of all deaths in New Zealand in 1997.

## 7.6 Avoidable mortality

Although we assumed that BMI would increase under both the BAU and intervention scenarios, we projected a smaller increase in BMI under the intervention scenario and therefore some obesity-related mortality could potentially be avoided by 2011.

The distributional transitions modelled were an increase in the population mean BMI for both males and females of 1.3 kg/m<sup>2</sup> under the BAU scenario, and a smaller increase of 1.0 kg/m<sup>2</sup> under the intervention scenario.

Key results are summarised here; full age-, sex- and ethnic-specific results are provided in Appendix 3.

### Ischaemic heart disease

If increases in BMI were limited to those outlined under the intervention scenario, 2–10 percent of IHD deaths would be avoided depending on age and baseline BMI. This shift in BMI would mean that 85 IHD deaths and 957 YLL could be avoided from 2011 (Table 94), relative to the mortality expected under the BAU scenario.



**Table 94:** Avoidable mortality\* for body mass index and ischaemic heart disease, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	40	445
	Māori	12	181
	Total	52	626
Females	Non-Māori	28	262
	Māori	5	69
	Total	33	331

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Ischaemic stroke

If increases in BMI were limited to those outlined under the intervention scenario, 2–10 percent of ischaemic stroke deaths would be avoided depending on age and baseline BMI. This shift in BMI would mean that 25 ischaemic stroke deaths and 227 YLL could be avoided each year from 2011 (Table 95), relative to the mortality expected under the BAU scenario.

**Table 95:** Avoidable mortality\* for body mass index and ischaemic stroke, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	8	64
	Māori	1	16
	Total	9	80
Females	Non-Māori	15	127
	Māori	1	20
	Total	16	147

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Diabetes

If increases in BMI were limited to those outlined under the intervention scenario, between 10 and 40 percent of deaths due to diabetes would be avoided depending on age. This shift in BMI would mean that 323 diabetes deaths and 4535 YLL could be avoided each year from 2011 (Table 96), relative to the mortality expected under the BAU scenario.

**Table 96:** Avoidable mortality\* for body mass index and diabetes, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	64	759
	Māori	84	1,128
	Total	148	1,887
Females	Non-Māori	75	1,005
	Māori	100	1,643
	Total	175	2,648

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Colorectal cancer

If increases in BMI were limited to those outlined under the intervention scenario, approximately 2 percent of colorectal cancer deaths would be avoided. This shift in BMI would mean that 13 colorectal cancer deaths and 127 YLL could be avoided each year from 2011 (Table 97).

**Table 97:** Avoidable mortality\* for body mass index and colorectal cancer, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	5	49
	Māori	1	7
	Total	6	56
Females	Non-Māori	7	67
	Māori	0	4
	Total	7	71

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Post-menopausal breast cancer

If increases in BMI were limited to those outlined under the intervention scenario, approximately 2 percent of post-menopausal breast cancer deaths would be avoided corresponding to eight deaths and 100 YLL each year from 2011 (Table 98).

**Table 98:** Avoidable mortality\* for body mass index and post-menopausal breast cancer, 2011

Ethnicity	Deaths (count)	Years of life lost (count)
Non-Māori	7	86
Māori	1	14
Total	8	100

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

### Total avoidable mortality

If increases in BMI were limited to those outlined under the intervention scenario, a total of 385 deaths and 4951 YLL could be avoided each year from 2011 (Table 99), relative to the mortality expected under the BAU scenario. These estimates are adjusted for the reporting overlap between diabetes and cardiovascular deaths. Even after this adjustment, 254 of the 385 avoidable deaths per year (66 percent) represent avoided deaths from diabetes, reflecting the very strong association between ‘high’ BMI and type 2 diabetes.

**Table 99:** Avoidable mortality\* for body mass index and all diseases, 2011

Sex	Ethnicity	Deaths (count)	Years of life lost (count)
Males	Non-Māori	102	1,150
	Māori	80	1,085
	Total	182	2,235
Females	Non-Māori	117	1,326
	Māori	86	1,390
	Total	203	2,716

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## 7.7 Discussion

We estimate that in 1997 ‘high’ BMI contributed to 3154 deaths (11 percent of all deaths) (Table 100). It was not surprising to find that approximately 80 percent of type 2 diabetes was attributable to ‘high’ BMI. Obesity and overweight with abdominal fat distribution are known to account for the vast majority of type 2 diabetes. Diabetes has serious complications, is difficult to manage, and reduces life expectancy by about seven years in Europeans and 12 years in Māori and Pacific peoples (Ministry of Health 2002c). Despite the high prevalence of diabetes among overweight and obese individuals, diabetes is not usually the direct cause of excess mortality as cardiovascular disease accounts for approximately 60 percent of deaths in individuals with type 2 diabetes (Ministry of Health 2002c). Hence the estimates of attributable and avoidable burden had to be adjusted for reporting overlap between cardiovascular disease and diabetes as causes of death to avoid double counting some deaths.

Although only a reduction in the projected increase in mean population BMI is modelled under the intervention scenario, rather than an absolute decrease (0.3 kg/m<sup>2</sup> lesser increase in mean BMI than under the BAU scenario), this translates into significant decreases in expected mortality. If relevant policy initiatives were introduced now, such a change could prevent approximately 385 deaths each year from 2011 (Table 100), relative to the BAU scenario, of which approximately two-thirds represent avoided diabetes deaths.

**Table 100:** Summary of attributable and avoidable mortality due to 'high' body mass index

	Attributable mortality (1997)		Avoidable mortality (2011) <sup>2</sup>	
	Deaths (count)	Years of life lost (count)	Deaths (count)	Years of life lost (count)
Ischaemic heart disease	1,560	17,910	85	957
Stroke	367	3,284	25	227
Diabetes <sup>1</sup>	959	12,907	254	3,540
Colorectal cancer	177	2,082	13	127
Breast cancer	91	1,190	8	100
Total	3,154	37,373	385	4,951

1 Adjusted for overlap with cardiovascular disease.

2 Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

Although a single intervention scenario has been modelled here, the effect of alternative scenarios can be readily estimated. The relationship between the distributional transition and the avoidable burden is approximately linear. Therefore doubling the shift in BMI (from 6 percent to 12 percent less than the BAU scenario) would double the avoidable burden (from 385 to 770 deaths in 2011).

## Policy implications

There is no evidence indicating that the obesity epidemic has peaked. Therefore, under the BAU scenario we estimated that mean BMI would increase by 1.3 kg/m<sup>2</sup> in both males and females by 2011. Under the intervention scenario, which assumes that policy initiatives will be introduced to slow the rate of weight gain in the population, we estimated that increases in BMI would be smaller, at 1.0 kg/m<sup>2</sup> in males and females. In other words, under the intervention scenario, 0.3 kg/m<sup>2</sup> of the predicted increase in BMI would be avoided. Such a difference in BMI is roughly equivalent to preventing an average 1 kg per person weight gain in adults over the next decade (0.27 g per day). Therefore, although mean BMI is expected to be greater in 2011 than in 1997, it may still be possible to avoid about 385 incremental deaths each year from 2011, provided increases in BMI are limited to those outlined under the intervention scenario.

No single intervention is likely to slow the increase in BMI to this extent. Interventions involving individual behavioural changes (diet and/or physical activity) can result in weight loss, a reduction in the prevalence of obesity, and improvements in cardiovascular and diabetes risk profiles in the short term, but have rarely been shown to be effective in the long term (WHO 2000b). Because of this, the World Health Organization considers that the only effective means of tackling the obesity epidemic is through a combination of

environmental, social and behavioural interventions (WHO 2000b). Possible settings for behavioural interventions to improve diet and increase physical activity include schools and workplaces. However, such interventions are unlikely to be successful without broader environmental (regulatory) and community-based changes.

A range of national policies could be developed to influence food supply and purchase patterns, such as a ban on advertising high fat-high sugar foods (especially to children), taxing high fat-high sugar foods, providing subsidies or other incentives to purchase nutritious foods (especially fruit and vegetables), regulating portion sizes, and nutritional labelling of all foods, including meals at fast-food outlets and restaurants. Changes that could encourage physical activity include making stairs more accessible in buildings, increasing access to bicycle lanes, providing safe walking and recreational areas, and providing subsidies or other incentives to use public transport, join gyms or participate in organised sports or other active recreational pursuits.

Changing food consumption patterns is difficult. Many factors influence food choices including food supply and availability, food culture, individual preferences, and price. Similarly, the urban planning, public transport and sport and recreation policies needed to reduce motor vehicle dependence and encourage participation in physical activity will not be easily achieved. Hence even the relatively modest distributional transition modelled here as the intervention scenario may prove too ambitious.

## **Limitations**

While it can generally be assumed that individuals with a high BMI have excess body fat, BMI does not distinguish between weight associated with lean body mass (eg, muscle) and weight associated with fat (WHO 2000b). As a result, the relation between BMI and body fat varies according to body build and proportion, and often differs between population subgroups. For example, at any given BMI, Māori and Pacific peoples have a lower level of body fat than Europeans (Swinburn et al 1996; Rush et al 1997; Swinburn et al 1999). There is no clear evidence that this lower level of body fatness in Māori and Pacific peoples compared to Europeans with the same BMI is associated with a lower risk of disease. However, if it is, we may have overestimated the burden of disease attributable to 'high' BMI in Māori and Pacific peoples.

Also, BMI does not provide information on the distribution of body fat. There is good evidence that excess fat in the visceral (abdominal) compartment correlates most strongly with disease risk (including diabetes and cardiovascular disease) (Pi-Sunyer 2002). Waist circumference, a simple and convenient indicator of abdominal body fat, may provide a useful supplementary indicator to BMI when assessing disease risk, and could be incorporated into our model in the future.

Whichever indicator of body fat is used, the handling of mediating variables in the analysis of body fat-disease associations is problematic. The solution applied here is partial deconfounding for blood pressure and blood cholesterol but not for insulin resistance or diabetes. This solution attempts to account for the independent influences of body fat mass on blood pressure and cholesterol, while acknowledging that much of the metabolic impact of excess body fat on health may be mediated through diabetes.

It is likely that we have underestimated the impact of higher than optimal BMI on cancer mortality. A recent United States study found that approximately 14 percent of cancer deaths in men and 20 percent of those in women were attributable to the current overweight and obesity patterns in that country (Calle et al 2003). In comparison, we found that approximately 4 percent of all cancer mortality was attributable to higher than optimal BMI (see page 138). However, only post-menopausal breast cancer and colorectal cancer were included in our analyses, whereas the United States study included more cancer types. In addition, the risk coefficients we used were more conservative.

## 8. Estimating the Burden of Disease Attributable to Nutrition-related Risk Factors

### 8.1 Study limitations

Despite using high-quality data on risk factor prevalence and risk factor–disease relationships, as well as a comprehensive and standardised analytic approach, our study still has limitations of which users of the estimates reported here should be aware.

- Our study is limited to a consideration of only a single intervention scenario. A sensitivity analysis, in which a range of possible intervention scenarios is modelled, would be more comprehensive. However, it is unlikely that the key policy implications that emerge from the study would change as a result. Of course, once a possible course of action is decided upon through the *Healthy Eating – Healthy Action* strategy, a more specific intervention can readily be modelled using the study tools. Indeed, the study was specifically designed with such applications in mind.
- Our study is restricted to a limited number of nutrition-related risk factors. However, it is likely that most of the key risk factor–disease pairs have in fact been included. Consumption of whole-grain cereals/fibre is a protective factor which could be included in future updates.
- Our study is also restricted to fatal outcomes only. When data and methods to estimate non-fatal outcomes improve, the study could be extended to include such outcomes as well. Including non-fatal outcomes would provide a more comprehensive estimate of attributable and avoidable burdens – one that included the impact of disability as well as that of premature mortality – but would probably have little impact on the relative effect sizes or rank ordering of different nutritional risk factors.
- The CRA model as currently constructed does not fully capture all relevant dimensions of exposure. For example, the study estimates the burden of disease associated with each risk factor separately, acting in isolation from other risk factors. Very approximate estimates of the joint effects of risk factors acting together are, however, provided in this chapter.
- The model does not explicitly take into account the lag between exposure to a risk factor and disease outcome. Use of appropriately lagged exposure data would be preferable to current exposure data when estimating attributable fractions. However, correction for regression dilution bias, as done in this report, partially incorporates lagging by estimating *usual* rather than *current* levels of exposure distributions. Further correction for lagging may become possible in future as a time series of risk factor distribution data builds up.

- The risk factor–disease relationships had to be estimated largely from non-New Zealand studies. Often these studies did not coincide in time with the studies of risk factor exposure (1997). The assumption has to be made that the relative risk or risk coefficient estimates (and risk-reversal estimates) do not vary with population, time or place. These relative risks have been shown to be broadly consistent across disparate populations in North America, Europe and the Asia Pacific region but no direct data are available for Māori and Pacific peoples (particularly for the BMI-diabetes relationship).
- Estimates of avoidable burden are dependent on the projections of the total burden of the selected diseases. While more sophisticated statistical methods were applied to make these projections than have typically been used in the past, any forecast is uncertain and these projections may turn out to be overly optimistic. As a consequence, the avoidable burdens may have been underestimated – a conservative stance to take in the face of uncertainty.

## 8.2 Comparison with previous New Zealand estimates

This study updates previous work carried out by the Ministry of Health as part of the *Our Health, Our Future* population health monitoring report (Ministry of Health 1999b). Using the CRA methodology, the current estimates for the attributable burdens of the selected risk factors greatly exceed the earlier *Our Health, Our Future* estimates (avoidable burdens are not comparable because different scenarios were modelled and different total burden projections were used). There are three reasons for this difference in the attributable burden estimates between the two studies.

- This study is based on better estimates of risk factor exposure distributions and risk factor–disease relationships than were available for the *Our Health, Our Future* study.
- This study methodology reflects the continuous nature of exposure disease associations. It uses continuous risk factor distributions and relates these to a theoretical minimum risk factor distribution. By contrast, the *Our Health, Our Future* study compares clinical categories (eg, ‘hypertensive’, ‘hypercholesterolaemic’, ‘obese’) with the corresponding ‘normal’ categories (eg, ‘normotensive’, ‘normal cholesterol’, ‘non-obese’). *Our Health, Our Future* therefore considers far fewer people to be exposed than does the current study, and uses smaller relative risks because of the high threshold of ‘normality’ compared to the theoretical minimum risk factor distribution.
- The CRA methodology used in this study does not fully de-confound risk factor–disease relationships for covariates that mediate the effect of the risk factor of interest, whereas the *Our Health, Our Future* study focused solely on the independent effect of each risk factor.



It is therefore not surprising that the CRA methodology gives higher estimates for the attributable burdens than the *Our Health, Our Future* methodology. Which estimate is preferable depends on the purpose of the analysis. The current study adopts a whole-of-population perspective, which is more useful for health promotion policy. The *Our Health, Our Future* study is based on clinical categories and adopts more of a personal health care perspective; the latter estimates may therefore be preferable for health care policy. In general, however, we believe that the current estimates, as reported here, are to be preferred to the earlier estimates since they illustrate the full potential of population-based risk reduction strategies.

## 8.3 Attributable burden

### Ranking of risk factors

Table 101 summarises the total mortality burden estimated to be attributable to each risk factor included in our study. For the total New Zealand population in 1997, 4721 deaths were attributable to higher than optimal total blood cholesterol, 3699 deaths to higher than optimal systolic blood pressure, 3154 deaths to higher than optimal BMI, and 1559 deaths to inadequate vegetable and fruit intake.

**Table 101:** Total attributable burdens, all-cause mortality, 1997

Risk factor	Deaths (count)	Percentage of all New Zealand deaths (%)	Years of life lost (count)	Percentage of New Zealand years of life lost (%)
Total blood cholesterol	4,721	17	45,682	16
Systolic blood pressure	3,699	13	35,263	12
Body mass index <sup>1</sup>	3,154	11	37,373	13
Vegetables and fruit	1,559	6	16,550	6

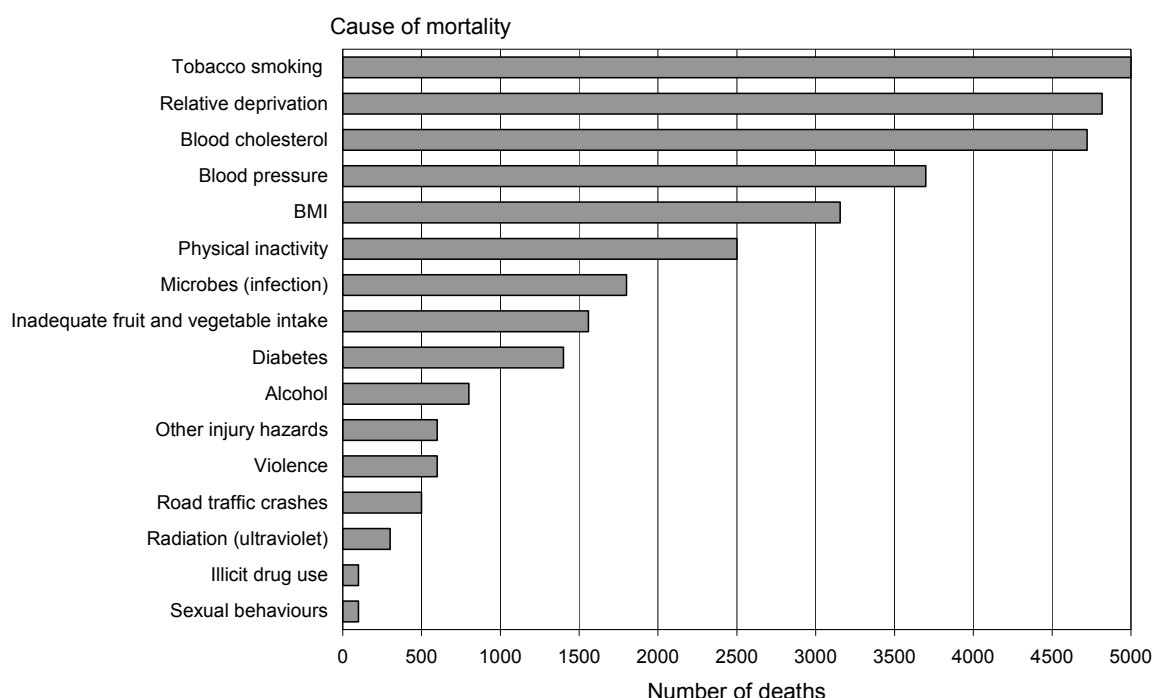
<sup>1</sup> Adjusted for cardiovascular disease/diabetes overlap.

Figure 37 shows the ranking of the nutrition-related risk factors included in this study relative to each other, and to other, non-nutrition-related risk factors. Estimates of total mortality attributable to the latter risk factors were derived by a similar process of counterfactual modelling as used here for the nutrition-related risk factors, or by categorical attribution (ie, some causes of death are assigned by definition to a particular risk factor), or by a combination of both methods.

Specifically, counterfactual modelling was used for tobacco smoking, physical inactivity, diabetes and relative deprivation (as well as the four risk factors considered in detail in this report). Categorical attribution was used to estimate burdens for infection, violence, road traffic crashes, other unintentional injury hazards, ultraviolet radiation, sexual behaviours and illicit drug use. Alcohol was modelled using both counterfactual modelling and categorical attribution for different outcomes.

The causes included in Figure 37 operate at three different levels of causation – from proximal (eg, diabetes) through intermediate (eg, physical inactivity) to distal (eg, deprivation). Because some of these causes are mediated, at least in part, by others their attributable burdens may not be comparable and cannot be added.

**Figure 37:** Attributable burdens, total mortality, for different causes, 1997



Adapted from: Ministry of Health 1999, Table 122; Howden-Chapman and Tobias 2000 (relative deprivation); Tobias and Roberts 2001 (physical inactivity).

**Explanatory notes:**

Alcohol – deaths caused only (not net of deaths prevented).

Diabetes – as a risk factor for other diseases as well as a disease in itself.

Other injury hazards – unintentional (eg, machinery, poisons, water hazards).

Relative deprivation – based on NZDep96 deciles (ie, counterfactual assigns decile 1 mortality rates to whole population). NZDep96 is a census-based small area measure of deprivation (Salmond et al 1998).

The estimate for the mortality burden attributable to physical inactivity shown in Figure 37 (2500 deaths in 1997) is almost certainly an underestimate relative to the estimates for the burdens for the nutritional risk factors included in this report. Physical activity could not be estimated on a continuous scale (as done for SBP, total blood cholesterol, vegetable and fruit intake and BMI) because of difficulties in measuring this complex behaviour. Instead this estimate could be based only on a discrete exposure distribution in which physical activity level was classified simply as ‘sufficient’ or ‘insufficient’ for health. Furthermore the threshold for ‘sufficient’ physical activity (30 minutes per day of moderate intensity activity) is relatively low and not analogous to the theoretical minimum distribution used in the comparative risk assessment methodology employed for the other nutrition-related risk factors included in this report.

Figure 37 shows that the nutrition-related risk factors rank highly in the league table of causes of death in New Zealand, far exceeding causes that attract much more policy attention, such as the road toll. Excluding social determinants of health such as deprivation, which act partially by shaping exposure to risk factors, only tobacco consumption ranks higher among risk factors than the causes selected for this study, with physical inactivity and infection also ranking higher than inadequate vegetable and fruit consumption. Furthermore, the gap between the tobacco and the cholesterol burdens is narrow and closing, as exposure to tobacco is declining faster than are population blood cholesterol levels. If these trends continue, cholesterol may shortly overtake tobacco as the leading cause of mortality, although not necessarily as the leading cause of *avoidable* mortality or of lost healthy life years. Also, while tobacco consumption, blood cholesterol level and systolic blood pressure have all been declining, BMI is rapidly increasing. Thus the ranking of the five leading risk factors (in terms of attributable mortality) may be very different in 10 or 20 years from what it is today.

The four risk factors assessed here rank differently for New Zealand compared to the estimates for economically developed regions reported in the *World Health Report 2002* (WHO 2002). In the *World Health Report*, systolic blood pressure ranked higher than total blood cholesterol, whereas in our analysis the rank order of these two risk factors is reversed. A possible reason for the difference between our ranking and the WHO ranking is that New Zealand may have a higher saturated fat intake in comparison to other developed countries thus explaining the higher burden attributable to total blood cholesterol. On the other hand, when correcting for calibration error we may have over-adjusted the SBP distribution downwards and thus underestimated the burden attributable to SBP. The most likely explanation of the difference in ranking is a combination of the above two factors.

In addition, the WHO used disability adjusted life years (DALYs) as the measure of burden whereas this report ranks the attributable burdens in relation to mortality only. Because not all events result in death, the use of a more integrated measure of population health such as DALYs may alter the risk factor rankings.

DALYs are calculated by combining YLLs with ‘equivalent’ healthy years lost due to disability (YLD). This calculation requires extensive epidemiological data as well as health state valuations, neither of which are available for New Zealand.

### **Combining risk factor burdens**

While burdens for the same risk factor can be added across mutually exclusive outcomes, the burdens attributable to different risk factors for any particular disease, or for all causes of death, are not additive. Non-additivity occurs because the same death could be attributed to more than one risk factor if the exposures overlap. It is therefore difficult to estimate what fraction of all-cause mortality, or of all IHD mortality, for instance, is attributable to the combined action of the four risk factors (or any subset of them) included in the study. Yet for policy purposes, estimates of the net impact of clusters of risk factors may sometimes be more useful (ie, when multiple interventions are involved). A very approximate joint effect estimate is provided below.

## Method

The joint effect of multiple risk factors acting on the same endpoint (eg, IHD mortality) is readily estimated if the risk factors (exposures) are independent and uncorrelated:

$$PAF = 1 - \prod_{i=1}^n (1 - PAF_i)$$

where  $PAF$  is the joint population-attributable fraction,  $PAF_i$  is the population-attributable fraction of individual risk factors,  $i$  is the individual risk factor, and  $n$  is the total number of risk factors (Miettinen 1974; Walter 1976; Ezzati et al, in press).

To see this, consider that if  $PAF_i$  is the fraction of disease attributable to risk factor  $i$  acting alone, then  $(1 - PAF_i)$  is the fraction not attributable to risk factor  $i$ . The product of all the  $(1 - PAF_i)$  terms is then the fraction of the disease burden not attributable to any of the risk factors. Therefore, 1 minus this term is the fraction attributable to all the risk factors combined.

However, the risk factors considered in this report are neither independent nor uncorrelated, and the above formula should ideally be adjusted to reflect their degree of non-independence (mediation and effect modification)<sup>1</sup> and the clustering of exposure (prevalence overlap or correlation).<sup>2</sup>

Insufficient New Zealand data were available for such adjustment; fortunately the joint PAF is not highly sensitive to dependency or clustering, provided risk coefficients are relatively large and exposures relatively prevalent (Ezzati et al, in press). For the risk factors and disease endpoints considered here, the net effect of non-independence and correlation in populations similar to New Zealand's is typically a small reduction in the joint PAF of less than 5 percent (Ezzati et al, in press).

## Estimates of combined burden

The results of the joint PAF calculations are summarised in Table 102. As these estimates have not been adjusted for non-independence and correlation, they may represent slight overestimates of the true joint effects. On the other hand, the individual PAFs on which they are based represent conservative estimates.

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<sup>1</sup> The biological effect of joint exposure to multiple risk factors may differ from the simple product of their independent effects when one risk factor alters the level of another (mediation – eg, BMI raises blood pressure) or alters the effect of another (effect modification – eg, high SBP is more atherogenic in the presence of high than low blood cholesterol).

<sup>2</sup> Depending on the sizes of the risk coefficients and whether they are multiplicative, positive correlation between risk factors typically implies a larger joint PAF than otherwise.

**Table 102:** Joint population-attributable fraction (PAF) estimates,<sup>1</sup> 1997

Contributing risk factors	Population-attributable fraction (%) <sup>2</sup>	Joint population-attributable fraction (%) <sup>3</sup>	Corresponding number of deaths
<b>Ischaemic heart disease mortality</b>			
Systolic blood pressure	40	87	5,542
Total cholesterol	64		
Body mass index	24		
Vegetables and fruit	18		
<b>Stroke mortality</b>			
Systolic blood pressure	48	70	1,687
Total cholesterol	26		
Body mass index	15		
Vegetables and fruit	7		
<b>Cancer mortality</b>			
Systolic blood pressure	0	6	469
Total cholesterol	0		
Body mass index	4		
Vegetables and fruit	3		
<b>Diabetes mortality</b>			
Systolic blood pressure	0	83	1,232
Total cholesterol	0		
Body mass index	83		
Vegetables and fruit	0		
<b>Total (all-cause) mortality</b>			
Systolic blood pressure	13	40	11,044
Total cholesterol	17		
Body mass index	11 <sup>4</sup>		
Vegetables and fruit	6		

1 Age groups and genders pooled.

2 Individual population-attributable fractions are the total attributable fractions derived from the tables in Appendix 3.

3 Obtained by applying the formula on page 137 to the individual population attributable fractions.

4 Adjusted for diabetes–cardiovascular disease overlap.

The mortality burden of ‘nutrition’ is estimated to be 11,044 deaths in 1997 (Table 102), which is equivalent to 113,753 YLL. This estimate is 84 percent of the number that would be obtained by simply summing each risk factor’s total attributable burden (13,133 deaths). The estimate of 11,044 deaths attributable to the combined effect of nutrition-related risk factors in 1997 corresponds to just under 40 percent of all deaths occurring in that year, but approximately 87 percent of all IHD deaths, 70 percent of all stroke deaths, 83 percent of all diabetes deaths and 6 percent of all cancer deaths. The proportion of cancer deaths attributable to nutrition-related factors is almost certainly an underestimate as only selected cancer types were included in the model.

**Table 103:** Population attributable fractions by sex, ethnicity and age group, 1997

	Deaths		Years of life lost	
	Joint population attributable fraction (%)	Count	Joint population attributable fraction (%)	Count
Males	40	5,735	40	61,883
Females	40	5,310	39	51,865
Māori	47	1,203	42	18,477
Non-Māori	39	9,852	39	95,489
25–34	6	33	6	960
35–44	29	232	29	5,433
45–54	45	687	44	13,894
55–64	52	1,417	51	22,926
65–74	46	2,702	45	30,979
75+	39	5,841	35	32,536

Table 103 shows the joint population attributable fraction by sex, ethnicity and age groups. Years of life lost are distributed evenly between males and females. Forty-seven percent of deaths among Māori are attributable to nutrition-related risk factors, compared with 39 percent among non-Māori. The proportion of deaths in each age group that is attributable to the joint effect of nutrition-related risk factors varies from only 6 percent in the 25–34 year age group to peak at 52 percent in the 55–64 year age group. The age distribution of nutrition attributable deaths is heavily concentrated in the older age groups. For example, as a proportion of the approximately 11,000 deaths attributed to nutrition, 53 percent occurred in the 75+ year age group (but only 31 percent of the 113,753 YLL attributable to nutrition).

Our estimate for the combined attributable burden of disease due to nutrition-related risk factors in New Zealand – approximately 11,000 deaths in 1997 – is thus over twice the corresponding tobacco attributable burden (including active and passive smoking – approximately 5000 deaths in 1997). However, because of our inability to adjust for non-independence and correlation of risk factors, the ‘nutrition’ burden may be slightly overestimated. This overestimation is probably not more than 5 percent if New Zealand resembles other developed countries in its risk profile (Ezzatti et al, in press)

More importantly, not all of the excess mortality resulting from elevated blood pressure, blood cholesterol and BMI can realistically be attributed to dietary exposures. In particular, most of the effect of physical inactivity on mortality (but not all of it) is mediated through these same risk factors. The estimate of approximately 11,000 attributable deaths should, therefore, be interpreted as reflecting the combined effect of dietary factors *and* physical inactivity (the latter at least in part). If the contribution of physical activity to the joint effect could be teased out, it is likely that the purely dietary effect would be substantially less – perhaps 2000–3000 less, allowing for underestimation of the burden of physical inactivity as shown in Figure 37. That is, approximately 8000–

9000 of the 11,000 deaths estimated to have been attributable to ‘nutrition’ in 1997 may reflect solely dietary exposures.

In summary, estimates of the total nutrition-related burden are inherently imprecise and comparisons with other risk factor burdens (such as tobacco) are best avoided. However, the method can still be used as a monitoring tool to assess *trends* in the total nutrition burden over time, providing the same assumptions are used consistently throughout. Such monitoring of joint effects can be useful if different nutrition-related risk factors are tracking in different directions (as appears to be currently the case).

## 8.4 Avoidable burden

Estimates of attributable burden, while interesting, are of limited policy relevance. This is because policy relevance depends on modifiability and inequality in distribution, as well as burden magnitude. Hence policy attention needs tend to focus on the estimates of avoidable as well as attributable burden.

The avoidable burden estimates for the nutrition-related risk factors, summed across all diseases and pooled across all population subgroups, are summarised in Table 104. It should be remembered that these avoidable burdens are based on a single intervention scenario only and refer to a single year (2011).

**Table 104:** Total avoidable burdens, all-cause mortality, 2011

Risk factor	Deaths (count)	Years of life lost (count)
Body mass index	385	4,951
Vegetables and fruit	334	3,448
Total blood cholesterol	300	2,670
Systolic blood pressure	282	2,613

These avoidable burden estimates may be thought of as indicating the number of deaths that could be avoided each year from 2011 if the exposure of the population to the risk factor of interest were to change as per the intervention scenario, while exposure to all other risk factors continue along their historical trends. Various policy options for achieving the intervention scenarios have been discussed in the text.

While only one intervention scenario has been modelled for each risk factor, as more specific intervention options are developed in the implementation phase of the *Healthy Eating – Healthy Action* Strategy, these can be simulated on the model and their likely impact assessed. Our intervention scenarios have tended to err on the conservative side, however, shifts towards the theoretical minimum distributions of greater (or lesser) magnitude can be readily approximated as the number of deaths avoided is proportional to the relative shift achieved.

Unlike attributable burdens where a formula is available to calculate joint effects (albeit without adjustment for non-independence and clustering of prevalence), it is difficult to estimate the joint avoidable burden across the selected risk factors. It is likely, however,

that the impact of multiple interventions acting simultaneously on multiple risk factors would differ from the simple addition of the separate avoidable burdens.

## 8.5 Policy implications

The estimates of attributable and avoidable burden reported here support the decision to include diet and body weight as priority objectives within the New Zealand Health Strategy, and to develop *Healthy Eating – Healthy Action* implementation plans to achieve these objectives.

Overall, policy attention is better focused on the individual attributable and avoidable burdens of the different nutrition-related risk factors separately, as these estimates are much more robust than those for ‘nutrition’ as a whole.

Our results illustrate that the well-established cardiovascular risk factors – cholesterol and blood pressure – remain by far the major modifiable causes of premature death (except for tobacco smoking), and are likely to continue to do so for at least several decades to come. Therefore, existing public health/nutrition messages and policies relating to fat intake (especially the ratio of saturated to poly- and mono-unsaturated fatty acids) and to sodium intake need to be maintained and indeed reinforced. If these messages and policies have failed to achieve substantive change in the past, then our results suggest that new policy approaches should be tried, not that the focus of policy attention should shift away from these issues.

At the same time, our results also suggest that the key public health/nutrition messages and policies need to be broadened to include a number of ‘newer’ issues as well. Foremost among these is BMI, not only because its attributable or avoidable burdens are currently large, but because of what they may become in the near future. Thus it is not enough merely to reduce saturated fat intake if this is simply going to be replaced with refined carbohydrates. As well as replacing saturated with poly/monounsaturated fatty acids and a variety of foods rich in non-starch polysaccharides for heart health, a key ancillary message must be to limit total energy intake and support this with sustained increases in energy expenditure by increasing physical activity in order to achieve and maintain healthy body weight.

Another key message relates to increasing vegetable and fruit consumption, because of what this would imply in terms of substitution for more energy-dense foods in the diet, consumption of fibre, improvement in the sodium : potassium ratio, and protection against cancer. While the latter remains important, this study for the first time quantifies vegetable and fruit intake as a major determinant of cardiovascular disease in New Zealand, as well as of cancer. This finding implies that much more policy attention needs to be focused on how to stimulate vegetable and fruit consumption (especially among disadvantaged communities), beyond the conventional 5+ A Day campaigns.

Indeed, the policy message is that a wider focus of attention covering vegetable and fruit intake and healthy weight maintenance in addition to blood pressure and cholesterol reduction would be more effective than focussing solely on the latter two risk factors. A combination of strategies, acting at multiple levels, on multiple risk factors, is likely to be



necessary to achieve sustainable change (WHO 1986). Furthermore, the optimal set of strategies will vary across population groups and over time.

Formulation of specific policy options to improve nutrition and physical activity levels, and maintain healthy body weight is beyond the scope of this report. However, a number of more general strategies have been modelled or implied in the relevant chapters (Table 105).

**Table 105:** Strategies to improve nutrition at local and national levels

Strategy	Example
<b>Local level</b>	
Primary health care	Statin (or possibly 'polypill' (Wald and Law 2003)) prescription, green prescription, counselling
Community and settings-based interventions	School canteens, workplace gyms
Urban planning and public transport	Community gardens, fruit and vegetable purchasing co-operatives, walking and cycling paths
<b>National level</b>	
Product engineering	Reduced saturated and trans fatty acid content of food products, reduced use of salt as a food additive, stanol fortification of margarines and salad dressings
Social marketing	Vegetables and fruit
Regulation	Portion size, nutritional labelling, advertising restrictions
Fiscal instruments	Subsidy on vegetables and fruit, tax on fast foods and soft drinks

As the Ministry of Health and other stakeholders develop specific policy options for improving diet and maintaining or regaining healthy body weight, these strategies can be analysed using the modelling tools reported here, so helping to identify 'best bets' and 'best buys'.

In developing these policy options, an important limitation of risk factor epidemiology should be borne in mind – namely that a focus on risk factors (nutritional or otherwise) can tend to decontextualise behaviour, leading to ineffective or even counterproductive interventions and to victim blaming (Krieger 1994; Pearce 1996). Rather, nutritional exposures need to be understood as being shaped by social structural and cultural determinants. For example, surveys of food security have shown that many low income New Zealanders may be unable to afford nutritious food choices (Russell et al 1999).

Interventions to improve nutrition, physical activity and body weight should include (simultaneously) strategies directly addressing the relevant risk or protective behaviours and strategies aimed at the underlying social inequalities in income, employment, housing and education. Policies that fail to take account of the sociocultural context within which individuals and families make lifestyle choices are unlikely to succeed.

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# Glossary and Abbreviations

APCSC	Asia Pacific Cohort Studies Collaboration.
Attributable burden	The proportion of current disease or injury burden that results from past risk factor exposure.
Avoidable burden	The proportion of future disease burden that could be avoided if risk factor exposure levels are reduced to those specified by some alternative, or counterfactual, distribution.
BMI	Body mass index ( $\text{kg/m}^2$ ).
Business as usual (BAU) scenario	A distributional transition in which the shift in exposure distribution reflects a continuation of the historical trend in exposure.
Counterfactual distribution	An alternative distribution of risk factor exposure from that currently experienced.
CRA	Comparative risk assessment – a systematic approach to estimating the burden of disease due to different risk factors.
CVD	Cardiovascular disease.
Distributional transition	A shift from the current risk factor exposure distribution towards an alternative distribution (in this study the distributional transition was a shift from the current exposure distribution towards the theoretical minimum distribution).
F&V	Fruit and vegetables (g/day).
HDL	High-density lipoprotein.
IHD	Ischaemic heart disease.
Intervention scenario	A distributional transition in which the shift in risk factor exposure distribution reflects policy interventions (over and above the BAU scenario).
LDL	Low-density lipoprotein.
MUFA	Monounsaturated fatty acids.
1997 NNS	1997 National Nutrition Survey.
PIF (potential impact fraction)	Proportional reduction in the total number of new (incident) cases of a certain disease or deaths from that disease, resulting from a specific change in the distribution of a risk factor in the population at risk.
PUFA	Polyunsaturated fatty acids.
Regression coefficient	A measure of risk accumulation in the case of a continuous risk factor. Also known as risk coefficient.



Risk accumulation	The nature and strength of the association between a risk factor exposure and disease. Risk accumulation is expressed as the regression coefficient for a continuous risk factor or as the relative risk for a discrete risk factor.
Risk coefficient	See <i>Regression coefficient</i> .
Risk factor distribution	The variation in level of the risk factor across the population.
Risk reversal	The extent and timing with which the risk of disease is reversed following a reduction in exposure of the population to the risk factor.
SBP	Systolic blood pressure (mmHg).
SFA	Saturated fatty acids.
TC	Total blood cholesterol (mmol/L).
Theoretical minimum risk distribution	The distribution of exposure that would have the lowest associated population health risk.
WHO	World Health Organization.
YLL	Years of life lost – a measure of the burden of premature mortality.

# **Appendix 1:**

## **Surveys Used for Current Distributions**

Data on the population distribution of selected risk factors were extracted from the following surveys.

### **National Nutrition Survey 1997**

The New Zealand Health Survey (NZHS) sample population was used to recruit participants for the 1997 National Nutrition Survey (NNS). The 1996/97 New Zealand Health Survey (NZHS) was the second nationally representative survey of the health status and health service utilisation of New Zealand adults and children. The NZHS was conducted over a one-year period (October 1996 to October 1997) by Statistics New Zealand and funded by the Ministry of Health and the former Central Regional Health Authority. At the conclusion of the NZHS, individuals were asked to participate in the 1997 NNS.

### **Survey sample**

The target population for the NZHS was defined as the total usually resident, non-institutionalised, civilian population of New Zealand of all ages residing in private households. A stratified cluster-sampling process was used to select a sample from this population. The sampling frame was area based. New Zealand is divided into 18,800 small geographic areas called primary sampling units (PSUs), which generally contain between 50 and 100 dwellings. The PSUs are divided into 122 groups (strata) according to various characteristics (eg, urban/rural, high/low Māori population density) derived from the 1991 Census data. PSUs were randomly selected from each of the 122 strata. A panel of households in each PSU was then randomly selected to participate in the NZHS.

In order to obtain more reliable estimates for Māori and Pacific people, these groups were oversampled. Each of the 1752 selected PSUs contained a panel of households that had recently taken part in the Household Labour Force Survey. A subset of these households that were known to contain Māori or Pacific people was added to the NZHS sample. The Central region was also oversampled to obtain reliable estimates at a sub-regional level.

Adult respondents were selected from each of the 1752 PSUs. One eligible adult (aged 15 years and over) was selected in each household and asked to participate. The sampling frame consisted of 11,921 households. The adult response rate was 73.8 percent. This represents the proportion of eligible households visited during the survey period that provided an adult respondent. The final sample comprised 7862 adults. Sample survey weights were calculated for each respondent based on individual probability of selection, differential non-response at a regional level, and the age, sex and Regional Health Authority distribution of the sample so that it was consistent with the March 1997 New Zealand population estimates.

A total of 4636 adults participated in the 1997 NNS, including an over-sample of Māori and Pacific people. It achieved a response rate of 59.0 percent. The 1997 NNS involved a 24-hour diet recall, food frequency questionnaire, food-related questions, physical measurements and a blood sample.

### **Current distribution data**

Data from the 1997 NNS were used to provide information on the current distributions of BMI, vegetable and fruit intake, total blood cholesterol, and blood pressure.

### ***Body mass index***

BMI was calculated by dividing weight (kilograms) by height (metres) squared. Height measurements were made without footwear using a portable stadiometer. Two height measurements were taken to the nearest 0.1 cm, and if these differed by more than 0.5 cm a third measurement was taken. The height measurement for each individual was the mean of the two closest measures. Weight measurements were made to the nearest 0.1 kg on a SECA Model 770 scale (capacity 200 kg) with the respondent in light clothing. Reported data have been corrected for a clothing weight of 1.2 kg. Scales were calibrated with standard weights each day. Two measurements were taken, and if these differed by more than 0.5 kg a third measurement was taken. The weight measurement for individuals was the mean of the two closest measures.

### ***Vegetable and fruit intake***

Vegetable and fruit intake was estimated based on data from the 24-hour diet recall. The interviewer administered the 24-hour recall in the home of the participant, and asked participants to recall all food and beverages consumed in the previous 24 hours (midnight to midnight), using the three-pass technique (quick list, specified probe question for each item on the quick list, and a review). Responses were entered directly onto a computer, which included logic checks. Manufactured foods with barcodes were scanned.

The intake of vegetables and fruit (g/day) was not determined as part of the original 1997 NNS analyses, so additional work was undertaken to obtain this information. Vegetables and fruit were defined as all fresh, frozen, canned, dried or juiced vegetables and fruit, except potatoes and mature legumes (pulses). Due to methodological difficulties the vegetable and fruit content of some mixed dishes could not be determined, which may result in a slight underestimation of vegetable and fruit intakes. Data are based on a single 24-hour recall and were not corrected for the high day-to-day variation in vegetable and fruit intake.

### ***Total blood cholesterol***

Total cholesterol measurements were made on a single non-fasting blood sample using enzymatic method for total cholesterol (Boehringer Mannheim, on a Hitachi 717 automated analyser). The methods were operated with International Accreditation New Zealand-approved quality-control procedures and underwent fortnightly peer review through the Royal College of Pathologists of Australasia Quality Assurance programme.

### ***Systolic blood pressure***

Blood pressure was measured with an Omron 706c smart-inflate blood pressure monitor. A large cuff was used for individuals with an upper arm circumference greater than 32 cm. The measurement was repeated up to three times and the measurements were averaged. Due to difficulty calibrating the instrument used to measure blood pressure, measurements were considered 'unreliable' by investigators (Noela Wilson, personal communication, November 2001). The difficulty in calibration is thought to be the explanation for the unexplained increase in blood pressure between the 1989 Life In New Zealand (LINZ) Survey and the 1997 NNS, an increase that is contrary to trends in blood pressure in Auckland during the late 1980s and early 1990s (Trye et al 1996), as well as trends observed in many similar populations internationally (Evans et al 2001). Blood pressure measurements for 1997 were therefore adjusted downward, to bring them in line with data from three cross-sectional surveys undertaken in Auckland during the 1980s and early 1990s as part of the WHO multinational Monitoring of Trends and Determinants in Cardiovascular Disease (MONICA) Project.

### **MONICA surveys**

Three cross-sectional surveys were undertaken in Auckland as part of the WHO MONICA Project in the 1980s and early 1990s: the 1982 Auckland Risk Factor Study (1568 subjects), the 1986–88 Auckland Heart Study (888 subjects), and the 1993–94 Auckland Heart and Health Study (1350 subjects). In each survey, age- and gender-stratified random samples were selected from Auckland general electoral roll. Response rates in the three studies were between 72 and 85 percent. The studies were limited to Europeans aged between 35 and 64 years.

## Appendix 2: Current and Projected Disease Burden Tables

### Ischaemic heart disease

**Table 106:** Ischaemic heart disease mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	9	54	185	387	921	1,746	3,303
	Females	3	8	32	105	385	2,082	2,614
Māori	Males	2	24	54	89	82	51	303
	Females	1	7	17	38	56	61	180
Total	Males	11	79	240	476	1,003	1,797	3,606
	Females	4	15	49	143	440	2,143	2,794
Years of life lost (count)								
Non-Māori	Males	233	1258	3,684	6,074	10,103	8,730	30,081
	Females	79	192	668	1,773	4,672	15,169	22,553
Māori	Males	52	559	1,075	1,397	899	255	4,237
	Females	26	168	355	642	680	444	2,315
Total	Males	285	1840	4,779	7,471	11,002	8,985	34,362
	Females	106	360	1,023	2,414	5,339	15,614	24,856
Deaths (rate per 100,000)								
Non-Māori	Males	3.9	22.2	90.6	276.9	815.7	2,622.1	278.6
	Females	1.4	3.0	15.5	74.6	311.7	1,853.2	142.4
Māori	Males	3.9	72.4	264.9	715.8	1,413.9	2,780.3	451.5
	Females	1.4	20.1	81.6	291.0	830.5	2,212.3	257.1
Total	Males	3.9	28.3	106.5	312.8	845.0	2,626.3	291.4
	Females	1.4	5.2	21.8	92.8	338.4	1,861.9	149.7
Years of life lost (rate per 100,000)								
Non-Māori	Males	101	518	1,805	4,345	8,947	13,110	2,652
	Females	36	73	325	1,259	3,782	13,502	1,340
Māori	Males	102	1687	5,275	11,234	15,509	13,902	5,256
	Females	38	482	1,703	4,914	10,077	16,119	2,951
Total	Males	101	659	2,121	4,910	9,269	13,132	2,860
	Females	36	125	455	1,567	4,107	13,565	1,455

\* Total rates are age-standardised to WHO World population.

**Table 107:** Ischaemic heart disease mortality, by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	2	14	88	309	645	1,366	2,424
	Females	0	1	9	59	240	1,243	1,552
Māori	Males	0	8	35	78	82	65	269
	Females	0	1	7	24	50	67	149
Total	Males	2	22	124	387	727	1,431	2,693
	Females	0	2	16	82	290	1,310	1,701
Years of life lost (count)								
Non-Māori	Males	52	326	1,752	4,850	7,075	6,830	20,885
	Females	0	24	188	996	2,912	9,056	13,177
Māori	Males	0	186	697	1,224	899	325	3332
	Females	0	24	146	405	607	488	1670
Total	Males	52	512	2,469	6,074	7,975	7,155	24,237
	Females	0	48	334	1,384	3,519	9,544	14,830
Deaths (rate per 100,000)								
Non-Māori	Males	0.7	5.6	33.7	140.4	472.5	1,419.9	147.3
	Females	0.1	0.4	3.4	25.9	162.5	893.3	67.0
Māori	Males	0.8	18.2	98.5	363.0	819.1	1,505.6	231.4
	Females	0.1	2.6	18.0	101.1	432.8	1,066.4	115.1
Total	Males	0.7	7.4	41.5	160.3	496.3	1,423.6	154.5
	Females	0.1	0.7	5.3	33.0	182.2	900.8	70.7
Years of life lost (rate per 100,000)								
Non-Māori	Males	19	130	671	2,204	5,184	7,100	1,338
	Females	2	9	71	438	1,971	6,508	607
Māori	Males	19	424	1,961	5,698	8,985	7,528	2,538
	Females	2	62	375	1,707	5,252	7,770	1,228
Total	Males	19	173	826	2,515	5,444	7,118	1,449
	Females	2	18	110	556	2,210	6,563	659

\* Total rates are age-standardised to WHO World population.

## Stroke

**Table 108:** Stroke mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	1	8	18	51	187	634	897
	Females	1	4	14	33	145	1230	1425
Māori	Males	1	2	3	10	12	16	44
	Females	1	1	6	11	15	23	57
Total	Males	1	9	21	61	199	650	941
	Females	2	5	20	43	159	1,253	1,483
Years of life lost (count)								
Non-Māori	Males	26	186	358	800	2,051	3,170	6,592
	Females	26	96	292	557	1,759	8,962	11,693
Māori	Males	26	47	60	157	132	80	501
	Females	26	24	125	186	182	168	711
Total	Males	26	210	418	957	2,183	3,250	7,044
	Females	53	120	418	726	1,929	9,129	12,375
Deaths (rate per 100,000)								
Non-Māori	Males	0.3	3.1	8.6	36.2	165.4	951.4	73.7
	Females	0.3	1.5	6.9	23.2	117.2	1094.6	74.3
Māori	Males	1.6	5.0	14.6	80.1	211.8	890.4	82.9
	Females	2.8	3.6	28.2	82.4	218.8	841.6	84.0
Total	Males	0.5	3.4	9.2	39.8	167.7	949.8	74.5
	Females	0.7	1.7	8.9	28.2	122.5	1088.5	75.8
Years of life lost (rate per 100,000)								
Non-Māori	Males	7	73	172	569	1,814	4,757	555
	Females	7	35	143	392	1,423	7,975	652
Māori	Males	41	116	291	1,258	2,323	4,452	727
	Females	75	88	590	1,391	2,655	6,132	929
Total	Males	12	78	183	625	1,839	4,749	570
	Females	18	42	186	476	1,486	7,930	680

\* Total rates are age-standardised to WHO World population.

**Table 109:** Stroke mortality, by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	1	5	12	45	127	582	773
	Females	1	2	8	22	86	1,001	1,120
Māori	Males	1	1	3	10	12	24	52
	Females	2	1	5	8	13	35	63
Total	Males	3	6	15	55	139	607	824
	Females	3	3	13	31	98	1,036	1,184
Years of life lost (count)								
Non-Māori	Males	26	116	239	706	1,393	2,910	5,391
	Females	26	48	167	371	1,044	7,293	8,950
Māori	Males	26	23	60	157	132	120	517
	Females	53	24	104	135	158	255	729
Total	Males	78	140	299	863	1,525	3,035	5,939
	Females	79	72	271	523	1,189	7,548	9,683
Deaths (rate per 100,000)								
Non-Māori	Males	0.5	1.9	4.6	20.6	93.1	605.0	45.3
	Females	0.4	0.9	2.9	10.0	58.1	719.4	46.1
Māori	Males	3.0	3.0	7.8	45.6	119.2	566.2	50.8
	Females	3.8	2.3	12.1	35.3	108.5	553.2	48.5
Total	Males	1.0	2.0	5.0	22.8	94.9	603.3	45.9
	Females	1.0	1.1	4.1	12.3	61.8	712.2	46.9
Years of life lost (rate per 100,000)								
Non-Māori	Males	14	44	92	323	1,021	3,025	334
	Females	10	22	61	168	705	5,241	389
Māori	Males	78	69	156	715	1,308	2,831	442
	Females	101	56	252	597	1,316	4,030	512
Total	Males	26	48	100	358	1,041	3,017	346
	Females	27	27	86	208	750	5,189	407

\* Total rates are age-standardised to WHO World population.



## Diabetes

**Table 110:** Diabetes mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	2	10	41	123	246	131	554
	Females	1	7	29	80	172	138	428
Māori	Males	2	12	42	94	70	18	238
	Females	2	14	46	98	86	28	274
Total	Males	5	22	83	216	317	149	791
	Females	4	21	75	178	257	167	702
Years of life lost (count)								
Non-Māori	Males	52	233	816	1,930	2,698	655	6,385
	Females	26	168	606	1,351	2,087	1,005	5,243
Māori	Males	52	279	836	1,475	768	90	3,501
	Females	53	336	961	1,655	1,044	204	4,252
Total	Males	129	512	1,653	3,390	3,477	745	9,907
	Females	106	505	1,566	3,005	3,119	1,217	9,517
Deaths (rate per 100,000)								
Non-Māori	Males	1.0	4.2	20.5	89.1	217.2	201.9	48.3
	Females	0.6	3.0	14.5	57.6	137.2	125.8	31.4
Māori	Males	5.6	36.0	210.0	763.8	1255.6	1010.4	331.3
	Females	4.6	38.5	226.9	774.7	1339.9	1059.5	348.1
Total	Males	1.7	8.0	37.5	144.3	266.0	223.1	66.5
	Females	1.2	7.4	34.2	117.6	195.7	147.7	52.0
Years of life lost (rate per 100,000)								
Non-Māori	Males	26	97	408	1,399	2,382	1,009	583
	Females	15	72	302	972	1,664	916	422
Māori	Males	144	839	4,182	11,987	13,773	5,052	4,324
	Females	121	926	4,737	13,080	16,259	7,719	4,975
Total	Males	43	185	747	2,264	2,918	1,116	856
	Females	32	178	714	1,986	2,375	1,076	753

\* Total rates are age-standardised to WHO World population.

**Table 111:** Diabetes mortality, by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	2	10	48	175	283	203	721
	Females	1	7	39	117	185	177	526
Māori	Males	2	16	62	141	126	55	403
	Females	2	15	80	146	134	77	454
Total	Males	4	26	111	317	409	257	1,124
	Females	3	22	119	262	320	254	980
Years of life lost (count)								
Non-Māori	Males	52	233	956	2,747	3,104	1,015	8,106
	Females	26	168	814	1,975	2,245	1,290	6,519
Māori	Males	52	373	1,235	2,213	1,382	275	5,529
	Females	53	360	1,670	2,465	1,626	561	6,736
Total	Males	103	606	2,210	4,975	4,487	1,285	13,666
	Females	79	529	2,485	4,423	3,883	1,851	13,250
Deaths (rate per 100,000)								
Non-Māori	Males	1.0	4.0	18.5	80.6	212.3	206.5	46.4
	Females	0.5	2.7	14.4	52.3	128.2	126.1	29.7
Māori	Males	3.9	38.8	176.6	676.7	1,274.6	1,300.9	326.7
	Females	3.8	32.2	205.1	647.7	1,179.0	1,257.6	316.1
Total	Males	1.5	9.0	37.3	132.9	285.7	251.4	68.1
	Females	1.2	7.2	38.4	106.9	204.9	173.1	53.3
Years of life lost (rate per 100,000)								
Non-Māori	Males	25	92	369	1,265	2,329	1,033	551
	Females	14	65	300	883	1,555	918	397
Māori	Males	100	904	3,516	10,621	13,982	6,504	4,094
	Females	101	774	4,283	10,936	14,306	9,163	4,433
Total	Males	39	210	742	2,085	3,134	1,257	861
	Females	31	172	802	1,805	2,487	1,261	763

\* Total rates are age-standardised to WHO World population.

## Cancer

### Post-menopausal breast cancer

**Table 112:** Post-menopausal breast cancer mortality, by ethnic and age group, 1997

	Age group (years)				
	50–54	55–64	65–74	75+	Total*
<b>Deaths (count)</b>					
Non-Māori	57	115	123	191	487
Māori	8	13	8	6	35
Total	65	129	131	197	521
<b>Years of life lost (count)</b>					
Non-Māori	1,190	1,947	1,493	1,394	6,023
Māori	160	225	97	41	523
Total	1,350	2,172	1,590	1,435	6,548
<b>Deaths (rate per 100,000)</b>					
Non-Māori	61.8	81.9	99.7	170.3	93.6
Māori	85.8	103.0	119.3	204.4	116.9
Total	63.9	83.7	100.7	171.1	95.1
<b>Years of life lost (rate per 100,000)</b>					
Non-Māori	1,291	1,383	1,209	1,241	1,300
Māori	1,791	1,739	1,448	1,489	1,648
Total	1,335	1,413	1,222	1,247	1,325

\* Total rates are age-standardised to WHO World population.

**Table 113:** Post-menopausal breast cancer mortality, by ethnic and age group, 2011

	Age group (years)				
	50–54	55–64	65–74	75+	Total*
<b>Deaths (count)</b>					
Non-Māori	69	150	142	225	586
Māori	14	20	13	13	60
Total	83	170	155	238	646
<b>Years of life lost (count)</b>					
Non-Māori	1,449	2,529	1,720	1,641	7,338
Māori	284	342	161	94	881
Total	1,733	2,870	1,881	1,734	8,218
<b>Deaths (rate per 100,000)</b>					
Non-Māori	51.1	65.5	91.0	159.9	81.2
Māori	71.0	82.7	109.0	192.3	101.4
Total	53.6	67.1	92.4	161.3	83.0
<b>Years of life lost (rate per 100,000)</b>					
Non-Māori	1,068	1,106	1,105	1,165	1,105
Māori	1,482	1,396	1,322	1,401	1,400
Total	1,119	1,134	1,121	1,175	1,133

\* Total rates are age-standardised to WHO World population.

## Colorectal cancer

**Table 114:** Colorectal cancer mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	3	8	45	103	202	192	553
	Females	3	5	37	81	138	256	520
Māori	Males	0	1	3	7	9	3	23
	Females	0	0	3	6	5	3	18
Total	Males	3	9	48	110	211	195	575
	Females	3	5	40	88	143	259	538
Years of life lost (count)								
Non-Māori	Males	78	186	896	1,617	2,216	960	5,952
	Females	79	120	773	1,368	1,675	1,865	5,879
Māori	Males	0	23	60	110	99	15	307
	Females	0	0	63	101	61	22	246
Total	Males	78	210	956	1,726	2,315	975	6,259
	Females	79	120	835	1,486	1,735	1,887	6,143
Deaths (rate per 100,000)								
Non-Māori	Males	1.1	3.3	22.0	73.4	179.3	288.3	47.7
	Females	1.2	1.9	18.2	57.8	112.1	227.9	35.1
Māori	Males	0.0	2.0	14.6	56.1	148.8	181.7	34.7
	Females	0.0	0.9	15.7	48.9	69.6	108.2	22.5
Total	Males	0.9	3.1	21.3	72.0	177.8	285.4	47.0
	Females	1.0	1.7	17.9	57.0	109.9	225.0	34.5
Years of life lost (rate per 100,000)								
Non-Māori	Males	28	77	438	1,152	1,967	1,442	535
	Females	32	46	380	976	1,360	1,660	448
Māori	Males	0	47	291	880	1,632	909	393
	Females	0	22	328	826	845	788	309
Total	Males	23	72	424	1,130	1,950	1,427	524
	Females	26	41	374	962	1,334	1,639	439

\* Total rates are age-standardised to WHO World population.

**Table 115:** Colorectal cancer mortality, by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	1	7	28	84	189	331	641
	Females	1	5	19	61	133	300	518
Māori	Males	0	1	3	7	11	10	31
	Females	0	0	2	5	6	7	22
Total	Males	1	8	31	91	200	341	672
	Females	1	6	21	66	139	307	540
Years of life lost (count)								
Non-Māori	Males	26	163	558	1,318	2,073	1,655	5,793
	Females	26	120	397	1,030	1,614	2,186	5,373
Māori	Males	0	23	60	110	121	50	364
	Females	0	0	42	84	73	51	250
Total	Males	26	186	617	1,428	2,194	1,705	6,157
	Females	26	144	438	1,114	1,687	2,237	5,647
Deaths (rate per 100,000)								
Non-Māori	Males	0.5	3.0	10.8	38.0	130.6	338.5	38.4
	Females	0.5	2.2	6.8	26.5	85.2	213.2	25.0
Māori	Males	0.0	1.8	7.2	29.0	108.4	213.4	27.3
	Females	0.0	1.1	5.9	22.4	52.9	101.2	14.9
Total	Males	0.4	2.8	10.4	37.2	129.1	332.9	37.7
	Females	0.4	2.0	6.7	26.1	82.8	208.1	24.3
Years of life lost (rate per 100,000)								
Non-Māori	Males	13	70	215	596	1,433	1,693	370
	Females	13	53	142	447	1,034	1,553	286
Māori	Males	0	42	143	455	1,189	1,067	269
	Females	0	26	123	378	642	737	183
Total	Males	10	65	207	584	1,416	1,665	361
	Females	11	48	140	441	1,005	1,516	278

\* Total rates are age-standardised to WHO World population.

## Lung cancer

**Table 116:** Lung cancer mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	0	7	38	131	304	287	767
	Females	1	11	31	71	158	149	421
Māori	Males	0	2	15	41	41	14	113
	Females	1	4	18	29	32	15	98
Total	Males	0	9	53	173	345	301	880
	Females	1	14	49	100	191	164	519
Years of life lost (count)								
Non-Māori	Males	0	163	757	2,056	3,335	1,435	7,745
	Females	26	264	647	1,199	1,917	1,086	5,140
Māori	Males	0	47	299	643	450	70	1,509
	Females	26	96	376	490	388	109	1,486
Total	Males	0	210	1,055	2,715	3,784	1,505	9,270
	Females	26	336	1,023	1,688	2,318	1,195	6,587
Deaths (rate per 100,000)								
Non-Māori	Males	0.1	2.9	18.4	94.0	269.3	430.4	65.4
	Females	0.3	4.2	15.2	50.7	128.3	132.9	30.2
Māori	Males	0.0	6.0	74.8	331.2	698.3	763.2	168.3
	Females	1.4	10.0	83.1	221.5	482.4	529.0	123.4
Total	Males	0.1	3.2	23.6	113.4	290.4	439.3	71.7
	Females	0.5	5.0	21.6	65.1	146.5	142.5	35.9
Years of life lost (rate per 100,000)								
Non-Māori	Males	4	67	367	1,475	2,954	2,152	686
	Females	7	102	318	856	1,557	968	406
Māori	Males	0	139	1,489	5,198	7,660	3,816	1,979
	Females	38	241	1,736	3,740	5,853	3,854	1,691
Total	Males	3	75	469	1,780	3,185	2,197	776
	Females	12	119	452	1,098	1,778	1,038	497

\* Total rates are age-standardised to WHO World population.

**Table 117:** Lung cancer mortality, by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	0	3	23	102	222	336	686
	Females	1	12	43	121	204	273	654
Māori	Males	0	1	13	37	42	28	121
	Females	1	5	35	56	60	52	209
Total	Males	0	4	36	139	264	364	807
	Females	2	17	78	177	264	325	863
Years of life lost (count)								
Non-Māori	Males	0	70	458	1,601	2,435	1,680	6,244
	Females	26	288	898	2,043	2,475	1,989	7,720
Māori	Males	0	23	259	581	461	140	1,464
	Females	26	120	731	945	728	379	2,930
Total	Males	0	93	717	2,182	2,896	1,820	7,708
	Females	53	408	1,629	2,988	3,203	2,368	10,650
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	1.2	8.8	46.1	153.7	343.5	41.0
	Females	0.4	4.7	15.8	52.7	131.1	194.1	34.3
Māori	Males	0.0	2.5	35.7	162.5	398.4	609.1	99.9
	Females	2.3	11.0	86.3	230.4	492.7	772.5	139.7
Total	Males	0.0	1.4	12.1	56.8	170.4	355.3	45.4
	Females	0.8	5.6	24.9	69.9	157.3	220.3	42.7
Years of life lost (rate per 100,000)								
Non-Māori	Males	0	28	175	724	1,686	1,718	391
	Females	11	113	330	890	1,591	1,414	444
Māori	Males	0	58	711	2,550	4,370	3,046	1,083
	Females	61	264	1,802	3,890	5,979	5,628	1,844
Total	Males	0	33	241	891	1,869	1,777	449
	Females	21	135	520	1,180	1,909	1,605	571

\* Total rates are age-standardised to WHO World population.



## Oesophageal cancer

**Table 118:** Oesophageal cancer mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	0	2	6	22	49	37	116
	Females	0	1	1	5	15	39	61
Māori	Males	0	1	1	2	3	1	8
	Females	0	0	0	2	1	1	4
Total	Males	0	3	7	25	52	37	124
	Females	0	1	2	6	16	40	65
Years of life lost (count)								
Non-Māori	Males	0	47	119	345	538	185	1,234
	Females	0	24	21	84	182	284	595
Māori	Males	0	23	20	31	33	5	113
	Females	0	0	0	34	12	7	53
Total	Males	0	70	139	392	570	185	1,357
	Females	0	24	42	101	194	291	653
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	1.0	2.8	16.0	43.7	55.1	10.0
	Females	0.0	0.3	0.7	3.3	12.2	34.7	3.6
Māori	Males	0.0	2.0	6.5	18.7	45.8	36.3	10.6
	Females	0.0	0.9	1.6	12.9	9.9	36.1	5.2
Total	Males	0.0	1.1	3.1	16.2	43.8	54.6	10.1
	Females	0.0	0.3	0.7	4.1	12.0	34.7	3.8
Years of life lost (rate per 100,000)								
Non-Māori	Males	0	22	55	251	479	275	111
	Females	0	6	14	56	147	253	39
Māori	Males	0	46	129	293	502	182	134
	Females	0	22	33	217	121	263	68
Total	Males	0	25	62	254	481	273	113
	Females	0	8	15	70	146	253	42

\* Total rates are age-standardised to WHO World population.

**Table 119:** Oesophageal cancer mortality, by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	0	2	10	34	55	69	170
	Females	0	1	3	6	17	48	74
Māori	Males	0	1	3	4	4	2	14
	Females	0	0	1	2	1	2	7
Total	Males	0	3	13	38	59	71	184
	Females	0	1	4	8	18	50	81
Years of life lost (count)								
Non-Māori	Males	0	47	199	534	603	345	1728
	Females	0	24	63	101	206	350	744
Māori	Males	0	23	60	63	44	10	200
	Females	0	0	21	34	12	15	81
Total	Males	0	70	259	596	647	355	1927
	Females	0	24	84	135	218	364	825
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	0.9	3.7	15.3	37.9	70.4	10.4
	Females	0.0	0.2	1.1	2.5	10.9	33.8	3.4
Māori	Males	0.0	1.9	8.8	17.9	39.8	46.5	10.9
	Females	0.0	0.8	2.6	9.6	8.9	35.2	4.8
Total	Males	0.0	1.0	4.4	15.5	38.1	69.3	10.5
	Females	0.0	0.3	1.3	3.2	10.7	33.9	3.6
Years of life lost (rate per 100,000)								
Non-Māori	Males	0	21	74	240	416	352	111
	Females	0	5	23	42	132	246	37
Māori	Males	0	44	175	281	437	233	138
	Females	0	19	54	162	108	256	62
Total	Males	0	23	88	243	418	347	115
	Females	0	7	27	54	130	247	40

\* Total rates are age-standardised to WHO World population.

## Stomach cancer

**Table 120:** Stomach cancer mortality, by ethnic group, age group and sex, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	2	2	9	17	51	63	144
	Females	2	2	4	10	23	58	100
Māori	Males	1	3	7	6	7	3	27
	Females	1	1	2	3	6	4	17
Total	Males	3	5	16	23	58	66	170
	Females	3	3	6	13	29	63	117
Years of life lost (count)								
Non-Māori	Males	52	47	179	267	559	315	1419
	Females	53	48	84	169	279	423	1055
Māori	Males	26	70	139	94	77	15	421
	Females	26	24	42	51	73	29	245
Total	Males	78	116	319	361	636	330	1,840
	Females	79	72	125	219	352	459	1,307
Deaths (rate per 100,000)								
Non-Māori	Males	0.7	0.8	4.6	12.4	44.9	94.1	12.2
	Females	0.8	0.8	2.1	7.3	18.6	51.9	6.4
Māori	Males	2.4	7.9	32.5	48.1	120.2	181.7	36.6
	Females	1.4	3.6	9.4	20.6	94.5	156.3	23.0
Total	Males	0.9	1.7	7.1	15.3	48.6	96.4	13.9
	Females	0.9	1.2	2.8	8.5	22.5	54.4	7.3
Years of life lost (rate per 100,000)								
Non-Māori	Males	18	19	91	195	492	470	125
	Females	22	19	44	124	226	378	78
Māori	Males	61	185	647	755	1,319	909	467
	Females	38	88	197	348	1,147	1,139	285
Total	Males	24	39	142	241	533	482	153
	Females	24	28	59	143	274	397	92

\* Total rates are age-standardised to WHO World population.

**Table 121:** Stomach cancer mortality by ethnic group, age group and sex, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (count)								
Non-Māori	Males	1	2	8	24	37	59	129
	Females	1	2	5	11	17	49	85
Māori	Males	0	3	7	9	7	5	33
	Females	0	1	4	3	7	7	22
Total	Males	1	5	15	33	44	64	162
	Females	1	3	9	14	24	56	107
Years of life lost (count)								
Non-Māori	Males	26	47	159	377	406	295	1,309
	Females	26	48	104	186	206	357	928
Māori	Males	0	70	139	141	77	25	452
	Females	0	24	84	51	85	51	294
Total	Males	26	116	299	518	483	320	1,762
	Females	26	72	188	236	291	408	1,222
Deaths (rate per 100,000)								
Non-Māori	Males	0.3	0.8	2.9	10.7	25.5	60.0	7.9
	Females	0.3	0.6	2.0	4.7	11.0	34.8	4.2
Māori	Males	0.9	7.3	20.5	41.4	68.2	115.8	24.4
	Females	0.6	2.9	8.8	13.2	56.0	104.7	15.2
Total	Males	0.4	1.7	5.0	13.5	28.4	62.5	9.4
	Females	0.4	1.0	2.9	5.5	14.3	38.0	5.0
Years of life lost (rate per 100,000)								
Non-Māori	Males	8	19	58	168	280	300	84
	Females	8	14	42	79	133	254	51
Māori	Males	23	170	408	650	748	579	321
	Females	16	70	184	223	680	763	192
Total	Males	10	40	100	212	312	313	108
	Females	11	24	61	93	174	277	65

\* Total rates are age-standardised to WHO World population.

## Appendix 3: Summary of Results

### Systolic blood pressure

### Ischaemic heart disease

#### *Attributable mortality*

**Table 122:** Attributable fractions (%) for systolic blood pressure and ischaemic heart disease, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	17	35	48	55	49	29
	Females	3	17	41	55	54	35
Māori	Males	24	51	62	65	57	29
	Females	5	39	67	76	59	30
Total	Males	18	40	51	57	49	29
	Females	3	28	50	60	54	35

**Table 123:** Attributable mortality (count) for systolic blood pressure and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	2	19	88	213	447	502	1,271
	Females	0	1	13	58	206	732	1,010
Māori	Males	0	12	34	58	47	15	166
	Females	0	3	12	28	33	19	95
Total	Males	2	31	122	271	495	517	1,438
	Females	0	4	25	86	239	750	1,104
Years of life lost (count)								
Non-Māori	Males	41	438	1,758	3,345	4,909	2,510	13,001
	Females	3	32	272	974	2,497	5,331	9,109
Māori	Males	10	290	669	908	516	74	2,467
	Females	1	69	241	480	400	136	1,327
Total	Males	51	728	2,427	4,253	5,425	2,584	15,468
	Females	4	101	513	1,454	2,897	5,467	10,436

**Table 124:** Attributable mortality (rate) for systolic blood pressure and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.7	7.7	43.2	152.5	396.5	753.7	109.0
	Females	0.0	0.5	6.4	41.0	166.8	651.3	57.3
Māori	Males	0.9	37.1	163.8	463.4	807.9	804.8	225.1
	Females	0.1	7.9	54.4	219.8	491.3	672.8	125.1
Total	Males	0.7	11.3	54.2	178.0	416.6	755.0	117.6
	Females	0.0	1.5	10.9	56.0	183.5	651.8	62.2
Years of life lost (rate per 100,000)								
Non-Māori	Males	17	179	860	2,393	4,349	3,768	1,161
	Females	1	13	134	692	2,024	4,745	567
Māori	Males	24	864	3,262	7,272	8,862	4,024	2,934
	Females	2	189	1,136	3,710	5,962	4,902	1,611
Total	Males	18	262	1,079	2,793	4,570	3,775	1,303
	Females	1	35	228	946	2,227	4,749	647

\* Age-standardised to WHO World population.

### Avoidable mortality

**Table 125:** Avoidable fractions (%)\* for systolic blood pressure and ischaemic heart disease, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	0	2	4	6	5	3
	Females	0	0	3	6	6	3
Māori	Males	1	5	7	8	7	3
	Females	0	3	8	11	7	4
Total	Males	0	3	5	6	5	3
	Females	0	1	6	7	6	3

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario.

**Table 126:** Avoidable mortality\* for systolic blood pressure and ischaemic heart disease, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	4	18	33	36	92
	Females	0	0	0	3	14	42	60
Māori	Males	0	0	2	6	5	2	17
	Females	0	0	1	2	4	3	10
Total	Males	0	1	6	25	38	39	109
	Females	0	0	1	6	18	45	70
Years of life lost (count)								
Non-Māori	Males	0	7	79	289	361	182	918
	Females	0	0	7	59	172	308	545
Māori	Males	0	8	49	96	59	11	225
	Females	0	1	12	42	45	20	121
Total	Males	0	15	128	386	421	193	1,142
	Females	0	1	19	101	217	328	666

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario.

## Stroke

### Attributable mortality

**Table 127:** Attributable fractions (%) for systolic blood pressure and stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	
Non-Māori	Males	15	31	42	50	47	42	
	Females	3	15	36	50	52	50	
Māori	Males	21	46	56	59	56	42	
	Females	5	35	60	70	58	44	
Total	Males	18	33	44	51	48	42	
	Females	4	20	43	55	53	50	

**Table 128:** Attributable mortality (count) for systolic blood pressure and stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	7	25	88	266	390
	Females	0	1	5	16	75	617	715
Māori	Males	0	1	2	6	7	7	22
	Females	0	0	4	8	8	10	30
Total	Males	0	3	9	31	95	273	412
	Females	0	1	9	24	84	628	745
Years of life lost (count)								
Non-Māori	Males	3	55	149	397	968	1,331	2,902
	Females	0	13	105	274	915	4,498	5,806
Māori	Males	4	18	33	93	75	34	258
	Females	2	11	76	127	103	75	393
Total	Males	6	73	182	490	1,043	1,365	3,160
	Females	2	24	181	401	1,017	4,573	6,199

**Table 129:** Attributable mortality (rate) for systolic blood pressure and stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	1.0	3.7	18.1	78.2	399.6	32.1
	Females	0.0	0.2	2.5	11.5	61.1	549.5	37.2
Māori	Males	0.3	2.3	8.2	47.6	117.9	376.2	40.0
	Females	0.1	1.3	17.0	58.0	126.4	371.1	43.5
Total	Males	0.1	1.1	4.1	20.5	80.1	399.0	32.7
	Females	0.0	0.4	3.9	15.5	64.4	545.2	38.2
Years of life lost (rate per 100,000)								
Non-Māori	Males	1	22	73	284	858	1,998	246
	Females	0	5	52	195	741	4,004	321
Māori	Males	9	53	163	747	1,294	1,881	374
	Females	3	30	355	980	1,534	2,704	505
Total	Males	2	26	81	322	879	1,995	256
	Females	1	8	80	261	782	3,972	340

\* Age-standardised to WHO World population.



## Avoidable mortality

**Table 130:** Avoidable fractions (%)\* for systolic blood pressure and stroke, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	0	2	4	5	5	4
	Females	0	0	3	5	6	5
Māori	Males	1	4	6	7	6	7
	Females	0	2	7	9	8	8
Total	Males	0	2	4	5	5	4
	Females	0	1	4	6	6	6

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario.

**Table 131:** Avoidable mortality\* for systolic blood pressure and stroke, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	2	6	25	34
	Females	0	0	0	1	5	54	61
Māori	Males	0	0	0	1	1	2	3
	Females	0	0	0	1	1	3	5
Total	Males	0	0	1	3	7	26	37
	Females	0	0	1	2	6	57	66
Years of life lost (count)								
Non-Māori	Males	0	2	9	37	69	123	240
	Females	0	0	5	20	59	396	479
Māori	Males	0	1	3	11	8	8	32
	Females	0	1	7	13	12	22	54
Total	Males	0	3	13	47	77	131	272
	Females	0	1	12	32	71	417	533

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario.

## Total

### Attributable mortality

**Table 132:** Attributable mortality (count) for systolic blood pressure and all diseases, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	2	21	96	238	536	768	1,661
	Females	0	2	18	74	281	1,349	1,724
Māori	Males	1	13	35	64	54	22	188
	Females	0	3	15	36	41	29	125
Total	Males	2	34	131	302	590	790	1,850
	Females	0	5	33	110	323	1,378	1,849
Years of life lost (count)								
Non-Māori	Males	43	493	1,907	3,742	5,877	3,840	15,903
	Females	3	45	377	1,249	3,412	9,829	14,915
Māori	Males	14	308	702	1,001	591	108	2,725
	Females	3	80	317	607	502	211	1,720
Total	Males	57	801	2,610	4,743	6,468	3,949	18,628
	Females	6	126	694	1,856	3,914	10,040	16,635

### Avoidable mortality

**Table 133:** Avoidable mortality\* for systolic blood pressure and all diseases, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	4	21	39	61	126
	Females	0	0	1	5	19	97	121
Māori	Males	0	0	3	7	6	4	20
	Females	0	0	1	3	5	6	15
Total	Males	0	1	7	28	45	65	146
	Females	0	0	1	8	24	102	136
Years of life lost (count)								
Non-Māori	Males	0	9	88	326	430	305	1,158
	Females	0	0	11	78	231	704	1,025
Māori	Males	0	10	53	107	68	19	256
	Females	0	1	19	55	57	42	175
Total	Males	0	18	141	433	498	324	1,414
	Females	0	1	31	133	288	746	1,199

\* Due to a 0.5 mmHg decrease in mean systolic blood pressure over and above the BAU scenario.

## Total blood cholesterol

### Ischaemic heart disease

#### *Attributable mortality*

**Table 134:** Attributable fractions (%) for total blood cholesterol and ischaemic heart disease, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	90	95	84	78	61	52
	Females	88	93	82	81	71	66
Māori	Males	98	98	89	80	58	40
	Females	83	91	80	79	61	51
Total	Males	91	96	85	78	61	52
	Females	87	92	82	81	69	66

**Table 135:** Attributable mortality (count) for total blood cholesterol and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	8	52	156	300	566	908	1,992
	Females	3	7	26	85	272	1,380	1,774
Māori	Males	2	24	48	72	48	21	214
	Females	1	7	14	30	34	31	116
Total	Males	10	76	205	372	614	929	2,206
	Females	3	14	40	115	306	1,412	1,890
Years of life lost (count)								
Non-Māori	Males	218	1,206	3,116	4,711	6,209	4,542	20,002
	Females	78	172	545	1,441	3,300	10,058	15,593
Māori	Males	42	555	964	1,123	525	103	3,312
	Females	15	161	289	503	410	227	1,604
Total	Males	260	1,761	4,080	5,835	6,734	4,645	23,314
	Females	92	332	834	1,944	3,710	10,284	17,197

**Table 136:** Attributable mortality (rate) for total blood cholesterol and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	3.5	21.2	76.5	214.7	501.5	1,363.9	170.8
	Females	1.2	2.8	12.8	60.6	220.4	1,228.8	98.1
Māori	Males	3.8	70.9	236.0	573.5	821.1	1,124.2	282.7
	Females	1.2	18.3	65.1	230.4	504.4	1,122.0	156.8
Total	Males	3.6	27.2	91.0	244.2	517.2	1,357.5	180.5
	Females	1.2	4.8	17.8	74.9	235.0	1,226.2	102.8
Years of life lost (rate per 100,000)								
Non-Māori	Males	91	494	1,523	3,370	5,501	6,820	1,798
	Females	32	68	268	1,024	2,674	8,953	948
Māori	Males	99	1,652	4,699	9,001	9,008	5,622	3,779
	Females	31	439	1,359	3,890	6,120	8,174	1,939
Total	Males	92	634	1,813	3,832	5,673	6,788	1,970
	Females	32	115	371	1,265	2,851	8,934	1,030

\* Age-standardised to WHO World population.

### Avoidable mortality

**Table 137:** Avoidable fractions (%)\* for total blood cholesterol and ischaemic heart disease, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	8	11	7	6	5	6
	Females	7	9	6	6	5	6
Māori	Males	13	14	8	10	8	8
	Females	6	8	6	6	7	6
Total	Males	9	12	7	7	6	6
	Females	7	9	6	6	5	6

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

**Table 138:** Avoidable mortality\* for total blood cholesterol and ischaemic heart disease, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	1	6	18	35	79	140
	Females	0	0	1	4	11	73	89
Māori	Males	0	1	3	8	6	5	23
	Females	0	0	0	1	4	4	10
Total	Males	0	3	9	26	41	85	163
	Females	0	0	1	5	15	78	98
Years of life lost (count)								
Non-Māori	Males	3	34	119	284	383	397	1,221
	Females	0	2	12	61	134	533	743
Māori	Males	1	24	57	121	68	26	297
	Females	0	2	9	23	44	31	109
Total	Males	4	59	177	405	451	423	1,518
	Females	0	5	21	84	178	565	853

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

## Ischaemic stroke

### Attributable mortality

**Table 139:** Attributable fractions (%) for total blood cholesterol and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	
Non-Māori	Males	45	55	65	59	36	30	
	Females	42	50	62	63	44	40	
Māori	Males	60	64	71	62	34	22	
	Females	37	48	60	61	36	29	
Total	Males	53	57	66	60	36	29	
	Females	39	50	62	63	43	40	

**Table 140:** Attributable mortality (count) for total blood cholesterol and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	6	17	42	131	197
	Females	0	1	4	12	39	348	404
Māori	Males	0	0	1	3	3	2	10
	Females	0	0	2	4	3	5	14
Total	Males	0	2	7	20	44	133	207
	Females	0	1	6	15	42	352	418
Years of life lost (count)								
Non-Māori	Males	3	44	113	264	458	655	1,537
	Females	3	20	91	195	476	2,533	3,318
Māori	Males	4	11	21	54	28	12	131
	Females	6	7	37	62	39	34	185
Total	Males	8	55	134	318	486	667	1,668
	Females	9	27	128	257	515	2,568	3,503

**Table 141:** Attributable mortality (rate) for total blood cholesterol and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.1	0.8	2.8	12.0	37.0	196.6	16.4
	Females	0.0	0.3	2.1	8.2	31.8	309.5	21.2
Māori	Males	0.4	1.4	5.1	27.6	43.9	135.3	16.7
	Females	0.5	0.8	8.4	28.2	48.4	169.0	19.5
Total	Males	0.1	0.8	3.0	13.3	37.3	194.9	16.6
	Females	0.1	0.4	2.7	9.9	32.7	306.1	21.5
Years of life lost (rate per 100,000)								
Non-Māori	Males	1	18	55	189	406	983	132
	Females	1	8	45	139	386	2,255	187
Māori	Males	10	33	102	433	482	676	174
	Females	12	19	175	476	588	1,231	231
Total	Males	3	20	60	209	410	975	137
	Females	3	9	57	167	396	2,230	194

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 142:** Avoidable fractions (%)\* for total blood cholesterol and ischaemic stroke, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	2	3	4	4	3	3
	Females	2	3	4	4	2	3
Māori	Males	3	4	5	6	4	4
	Females	2	2	3	4	3	3
Total	Males	3	3	4	4	3	3
	Females	2	3	4	4	2	3

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

**Table 143:** Avoidable mortality\* for total blood cholesterol and ischaemic stroke, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	1	2	11	15
	Females	0	0	0	0	1	20	22
Māori	Males	0	0	0	0	0	1	1
	Females	0	0	0	0	0	1	1
Total	Males	0	0	0	1	2	12	16
	Females	0	0	0	1	1	21	23
Years of life lost (count)								
Non-Māori	Males	0	1	5	14	22	57	100
	Females	0	1	3	8	14	146	172
Māori	Males	1	1	1	5	3	3	14
	Females	0	0	2	3	3	5	14
Total	Males	1	2	6	20	25	60	114
	Females	1	1	5	11	18	151	186

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.

## Total

### Attributable mortality

**Table 144:** Attributable mortality (count) for total blood cholesterol and all diseases, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	9	54	162	317	608	1,039	2,189
	Females	3	8	30	97	311	1,728	2,178
Māori	Males	2	24	49	75	50	23	224
	Females	1	7	16	33	37	36	130
Total	Males	10	78	212	392	658	1,062	2,414
	Females	4	15	46	130	348	1,764	2,308
Years of life lost (count)								
Non-Māori	Males	221	1,249	3,229	4,975	6,667	5,196	21,538
	Females	81	191	636	1,636	3,776	12,591	18,912
Māori	Males	47	566	985	1,177	553	116	3,443
	Females	20	167	326	565	450	261	1,789
Total	Males	268	1,815	4,214	6,152	7,220	5,312	24,981
	Females	101	359	962	2,201	4,225	12,852	20,701

### Avoidable mortality

**Table 145:** Avoidable mortality\* for total blood cholesterol and all diseases, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	6	19	37	91	155
	Females	0	0	1	4	12	93	110
Māori	Males	0	1	3	8	6	6	24
	Females	0	0	0	2	4	5	11
Total	Males	0	3	9	27	43	97	179
	Females	0	0	1	6	16	98	122
Years of life lost (count)								
Non-Māori	Males	3	36	124	298	406	454	1,321
	Females	0	3	15	69	148	679	915
Māori	Males	2	25	58	126	71	29	311
	Females	0	3	10	26	47	37	123
Total	Males	5	60	182	424	477	483	1,632
	Females	1	6	26	95	196	716	1,039

\* Due to a 0.1 mmol decrease in mean total blood cholesterol over and above the BAU scenario.



## Vegetables and fruit

### Ischaemic heart disease

#### Attributable mortality

**Table 146:** Attributable fractions (%) for vegetable and fruit intake and ischaemic heart disease, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	28	28	28	28	24	13
	Females	28	28	28	28	24	13
Māori	Males	30	30	30	30	26	14
	Females	31	31	31	31	26	15
Total	Males	28	29	29	29	24	13
	Females	29	30	29	29	25	13

**Table 147:** Attributable mortality (count) for vegetable and fruit intake and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	3	15	52	109	221	232	633
	Females	1	2	9	30	93	280	416
Māori	Males	1	7	16	27	21	7	79
	Females	0	2	5	12	15	9	43
Total	Males	3	23	69	136	242	239	712
	Females	1	4	14	42	108	289	459
Years of life lost (count)								
Non-Māori	Males	68	357	1,042	1,714	2,428	1,158	6,766
	Females	25	52	188	504	1,133	2,042	3,946
Māori	Males	13	170	325	421	231	36	1,197
	Females	5	55	112	197	179	65	613
Total	Males	81	527	1,367	2,135	2,659	1,194	7,963
	Females	30	107	300	701	1,312	2,108	4,558

\* Age-standardised to WHO World population.

**Table 148:** Attributable mortality (rate) for vegetable and fruit intake and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	1.1	6.3	25.6	78.1	196.1	347.6	54.6
	Females	0.4	0.9	4.4	21.2	75.7	249.5	24.5
Māori	Males	1.2	21.8	79.6	215.0	362.3	393.2	106.4
	Females	0.4	6.2	25.2	90.1	219.5	323.3	56.9
Total	Males	1.1	8.1	30.5	89.3	204.2	348.8	58.4
	Females	0.4	1.5	6.4	27.0	83.1	251.3	26.6
Years of life lost (rate per 100,000)								
Non-Māori	Males	28	146	509	1,226	2,151	1,738	609
	Females	10	21	92	358	918	1,818	259
Māori	Males	31	507	1,585	3,375	3,974	1,966	1,399
	Females	12	149	527	1,521	2,663	2,356	731
Total	Males	29	190	607	1,402	2,240	1,744	673
	Females	10	37	133	456	1,008	1,831	294

\* Age-standardised to WHO World population.

### Avoidable mortality

**Table 149:** Avoidable fractions (%)\* for vegetable and fruit intake and ischaemic heart disease, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	19	8	8	8	7	4
	Females	19	8	8	8	7	4
Māori	Males	19	9	9	9	7	5
	Females	20	9	9	9	8	5
Total	Males	19	8	8	8	7	4
	Females	19	9	9	9	7	4

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

**Table 150:** Avoidable mortality\* for vegetable and fruit intake and ischaemic heart disease, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	1	7	25	43	56	132
	Females	0	0	1	5	16	51	73
Māori	Males	0	1	3	7	6	3	20
	Females	0	0	1	2	4	3	10
Total	Males	0	2	10	32	49	59	152
	Females	0	0	1	7	20	55	83
Years of life lost (count)								
Non-Māori	Males	8	26	142	392	474	278	1,320
	Females	1	2	16	80	196	375	670
Māori	Males	2	16	63	110	67	15	273
	Females	0	3	14	38	48	23	126
Total	Males	10	42	205	502	541	293	1,593
	Females	1	5	30	118	244	398	795

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Ischaemic stroke

### Attributable mortality

**Table 151:** Attributable fractions (%) for vegetable and fruit intake and ischaemic stroke, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	18	18	18	15	11	10
	Females	18	18	18	16	11	11
Māori	Males	19	19	19	16	12	11
	Females	19	19	19	17	12	11
Total	Males	18	18	18	16	11	10
	Females	19	18	18	16	11	11

**Table 152:** Attributable mortality (count) for vegetable and fruit intake and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	1	2	4	13	46	65
	Females	0	0	1	3	10	90	105
Māori	Males	0	0	0	1	1	1	4
	Females	0	0	1	1	1	2	5
Total	Males	0	1	2	5	14	47	69
	Females	0	0	2	4	11	92	110
Years of life lost (count)								
Non-Māori	Males	1	14	31	69	139	229	483
	Females	1	7	26	48	121	659	863
Māori	Males	1	3	6	14	10	6	41
	Females	3	3	12	17	13	14	62
Total	Males	3	17	36	83	149	235	523
	Females	4	10	38	65	135	672	924

**Table 153:** Attributable mortality (rate) for vegetable and fruit intake and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	0.2	0.8	3.1	11.2	68.7	5.4
	Females	0.0	0.1	0.6	2.0	8.1	80.5	5.5
Māori	Males	0.1	0.4	1.4	7.4	15.3	68.5	6.5
	Females	0.2	0.3	2.7	7.8	16.4	66.9	6.9
Total	Males	0.0	0.3	0.8	3.5	11.4	68.7	5.5
	Females	0.1	0.1	0.8	2.5	8.5	80.1	5.6
Years of life lost (rate per 100,000)								
Non-Māori	Males	1	6	15	49	123	344	41
	Females	1	3	13	34	98	586	49
Māori	Males	3	10	27	116	168	343	59
	Females	6	8	57	132	199	488	78
Total	Males	1	6	16	54	126	344	43
	Females	1	3	17	42	104	584	51

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 154:** Avoidable fractions (%)\* for vegetable and fruit intake and ischaemic stroke, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	11	5	5	4	3	3
	Females	11	5	5	4	3	3
Māori	Males	11	5	5	5	3	3
	Females	11	6	6	5	3	3
Total	Males	11	5	5	4	3	3
	Females	11	5	5	4	3	3

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

**Table 155:** Avoidable mortality\* for vegetable and fruit intake and ischaemic stroke, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	1	2	11	14
	Females	0	0	0	1	2	19	21
Māori	Males	0	0	0	0	0	1	1
	Females	0	0	0	0	0	1	1
Total	Males	0	0	0	1	2	11	16
	Females	0	0	0	1	2	20	23
Years of life lost (count)								
Non-Māori	Males	1	2	6	16	24	54	104
	Females	1	1	4	9	19	137	171
Māori	Males	2	1	1	4	3	3	13
	Females	2	1	3	4	3	5	18
Total	Males	3	3	7	20	27	56	117
	Females	3	2	7	12	22	143	189

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Lung cancer

### Attributable mortality

**Table 156:** Attributable fractions (%) for vegetable and fruit intake and lung cancer, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	11	11	11	11	10	7
	Females	11	11	11	11	10	7
Māori	Males	12	12	12	12	11	7
	Females	12	12	12	12	11	7
Total	Males	11	11	11	11	10	7
	Females	12	11	12	12	10	7

**Table 157:** Attributable mortality (count) for vegetable and fruit intake and lung cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	1	4	15	30	19	69
	Females	0	1	3	8	16	10	39
Māori	Males	0	0	2	5	4	1	12
	Females	0	0	2	3	4	1	11
Total	Males	0	1	6	19	35	20	81
	Females	0	2	6	12	20	11	50
Years of life lost (count)								
Non-Māori	Males	1	18	83	228	332	95	757
	Females	2	29	73	135	195	74	507
Māori	Males	0	5	36	76	47	5	170
	Females	2	11	45	59	43	8	167
Total	Males	1	24	119	304	379	100	927
	Females	4	40	118	194	238	81	675

**Table 158:** Attributable mortality (rate) for vegetable and fruit intake and lung cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	0.3	2.0	10.4	26.8	28.5	5.9
	Females	0.0	0.5	1.7	5.7	13.0	9.0	2.9
Māori	Males	0.0	0.7	8.8	39.0	74.0	53.6	17.1
	Females	0.2	1.2	10.1	27.0	52.8	38.4	13.1
Total	Males	0.0	0.4	2.7	12.7	29.1	29.2	6.7
	Females	0.1	0.6	2.5	7.5	15.1	9.7	3.6
Years of life lost (rate per 100,000)								
Non-Māori	Males	0	7	41	163	294	143	68
	Females	1	11	36	96	158	66	42
Māori	Males	0	16	175	612	812	268	215
	Females	5	29	211	455	641	280	189
Total	Males	0	8	53	200	319	146	78
	Females	1	14	52	126	183	71	53

\* Age-standardised to WHO World population.

### Avoidable mortality

**Table 159:** Avoidable fractions (%)\* for vegetable and fruit intake and lung cancer, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	10	3	3	3	2	2
	Females	10	3	3	3	3	2
Māori	Males	10	3	3	3	3	2
	Females	10	3	3	3	3	2
Total	Males	10	3	3	3	2	2
	Females	10	3	3	3	3	2

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

**Table 160:** Avoidable mortality\* for vegetable and fruit intake and lung cancer, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	1	3	5	6	15
	Females	0	0	1	3	5	5	15
Māori	Males	0	0	0	1	1	1	3
	Females	0	0	1	2	2	1	6
Total	Males	0	0	1	4	7	7	18
	Females	0	0	2	5	7	6	21
Years of life lost (count)								
Non-Māori	Males	0	2	12	44	59	30	147
	Females	2	8	25	57	62	37	192
Māori	Males	0	1	8	17	12	3	41
	Females	3	4	23	30	21	8	89
Total	Males	0	3	20	61	72	33	188
	Females	5	12	49	88	83	45	281

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Oesophageal cancer

### Attributable mortality

**Table 161:** Attributable fractions (%) for vegetable and fruit intake and oesophageal cancer, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	18	18	18	18	15	9
	Females	18	18	18	18	16	9
Māori	Males	19	19	19	19	16	9
	Females	19	19	19	19	17	10
Total	Males	18	18	18	18	15	9
	Females	18	18	18	18	16	9



**Table 162:** Attributable mortality (count) for vegetable and fruit intake and oesophageal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	1	4	8	3	16
	Females	0	0	0	1	2	4	7
Māori	Males	0	0	0	0	0	0	1
	Females	0	0	0	0	0	0	1
Total	Males	0	1	1	4	8	3	18
	Females	0	0	0	1	2	4	8
Years of life lost (count)								
Non-Māori	Males	0	10	20	62	84	16	191
	Females	0	3	5	14	29	26	76
Māori	Males	0	3	5	7	5	0	20
	Females	0	2	1	5	1	1	10
Total	Males	0	12	25	69	88	17	211
	Females	0	4	6	20	30	26	86

**Table 163:** Attributable mortality (rate) for vegetable and fruit intake and oesophageal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	0.2	0.5	2.8	6.8	4.9	1.4
	Females	0.0	0.0	0.1	0.6	1.9	3.1	0.5
Māori	Males	0.0	0.4	1.2	3.5	7.5	3.4	1.7
	Females	0.0	0.2	0.3	2.5	1.7	3.5	0.8
Total	Males	0.0	0.2	0.6	2.9	6.8	4.8	1.4
	Females	0.0	0.1	0.1	0.8	1.9	3.1	0.5
Years of life lost (rate per 100,000)								
Non-Māori	Males	0	4	10	44	74	24	17
	Females	0	1	2	10	23	23	6
Māori	Males	0	9	24	55	83	17	23
	Females	0	4	6	42	21	26	12
Total	Males	0	4	11	45	74	24	18
	Females	0	2	3	13	23	23	6

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 164:** Avoidable fractions (%)\* for vegetable and fruit intake and oesophageal cancer, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	17	4	4	4	4	3
	Females	17	5	5	5	4	3
Māori	Males	17	5	5	5	4	3
	Females	16	5	5	5	5	3
Total	Males	17	5	5	5	4	3
	Females	17	5	5	5	4	3

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

**Table 165:** Avoidable mortality\* for vegetable and fruit intake and oesophageal cancer, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	2	2	2	6
	Females	0	0	0	0	1	1	2
Māori	Males	0	0	0	0	0	0	1
	Females	0	0	0	0	0	0	0
Total	Males	0	0	1	2	2	2	7
	Females	0	0	0	0	1	1	3
Years of life lost (count)								
Non-Māori	Males	0	2	9	24	23	9	67
	Females	0	1	3	4	8	9	25
Māori	Males	0	1	3	3	2	0	10
	Females	0	0	1	2	1	1	5
Total	Males	0	3	12	27	25	9	77
	Females	0	1	4	6	9	10	30

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Stomach cancer

### Attributable mortality

**Table 166:** Attributable fractions (%) for vegetable and fruit intake and stomach cancer, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	18	18	18	18	15	9
	Females	18	18	18	18	16	9
Māori	Males	19	19	19	19	16	9
	Females	19	19	19	19	17	10
Total	Males	18	18	18	18	16	9
	Females	18	18	18	18	16	9

**Table 167:** Attributable mortality (count) for vegetable and fruit intake and stomach cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	2	3	8	6	19
	Females	0	0	1	2	4	5	12
Māori	Males	0	1	1	1	1	0	5
	Females	0	0	0	1	1	0	3
Total	Males	0	1	3	4	9	6	23
	Females	0	1	1	2	5	6	15
Years of life lost (count)								
Non-Māori	Males	8	8	33	48	86	28	210
	Females	9	9	16	31	44	38	147
Māori	Males	5	12	25	18	13	2	73
	Females	3	6	8	9	13	3	43
Total	Males	12	20	58	66	98	29	283
	Females	13	15	24	40	57	41	190

**Table 168:** Attributable mortality (rate) for vegetable and fruit intake and stomach cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.1	0.1	0.8	2.2	6.9	8.3	1.6
	Females	0.1	0.1	0.4	1.3	2.9	4.7	0.9
Māori	Males	0.4	1.5	6.1	9.0	19.8	17.1	5.7
	Females	0.3	0.7	1.8	4.0	16.1	15.2	3.5
Total	Males	0.2	0.3	1.3	2.7	7.6	8.6	1.9
	Females	0.2	0.2	0.5	1.5	3.6	4.9	1.0
Years of life lost (rate per 100,000)								
Non-Māori	Males	3	3	16	34	76	42	19
	Females	4	3	8	22	35	34	12
Māori	Males	11	35	122	142	217	86	80
	Females	7	17	38	68	195	111	47
Total	Males	4	7	26	43	83	43	24
	Females	4	5	11	26	44	36	14

\* Age-standardised to WHO World population.

### Avoidable mortality

**Table 169:** Avoidable fractions (%)\* for vegetable and fruit intake and stomach cancer, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	17	4	4	4	4	3
	Females	17	5	5	5	4	3
Māori	Males	17	5	5	5	4	3
	Females	16	5	5	5	5	3
Total	Males	17	5	5	5	4	3
	Females	17	5	5	5	4	3

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

**Table 170:** Avoidable mortality\* for vegetable and fruit intake and stomach cancer, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	1	1	1	4
	Females	0	0	0	0	1	1	3
Māori	Males	0	0	0	0	0	0	2
	Females	0	0	0	0	0	0	1
Total	Males	0	0	1	2	2	2	6
	Females	0	0	0	1	1	1	4
Years of life lost (count)								
Non-Māori	Males	2	2	7	17	16	7	51
	Females	3	2	5	8	8	9	36
Māori	Males	2	4	7	7	3	1	24
	Females	1	2	4	3	4	2	15
Total	Males	4	6	14	24	19	8	75
	Females	4	4	9	11	12	11	51

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Colorectal cancer

### Attributable mortality

**Table 171:** Attributable fractions (%) for vegetable and fruit intake and colorectal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	
Non-Māori	Males	2	2	2	2	2	0	
	Females	2	2	2	2	2	0	
Māori	Males	2	2	2	2	2	0	
	Females	2	2	2	2	2	0	
Total	Males	2	2	2	2	2	0	
	Females	2	2	2	2	2	0	

**Table 172:** Attributable mortality (count) for vegetable and fruit intake and colorectal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	1	2	4	0	8
	Females	0	0	1	2	3	0	6
Māori	Males	0	0	0	0	0	0	0
	Females	0	0	0	0	0	0	0
Total	Males	0	0	1	2	5	0	8
	Females	0	0	1	2	3	0	6
Years of life lost (count)								
Non-Māori	Males	1	4	19	35	48	0	107
	Females	2	3	17	31	38	0	90
Māori	Males	0	0	1	2	2	0	6
	Females	0	0	2	2	1	0	5
Total	Males	1	4	21	37	50	0	114
	Females	2	3	19	33	39	0	95

**Table 173:** Attributable mortality (rate) for vegetable and fruit intake and colorectal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.0	0.1	0.5	1.6	3.9	0.0	0.7
	Females	0.0	0.0	0.4	1.3	2.5	0.0	0.5
Māori	Males	0.0	0.0	0.3	1.2	3.3	0.0	0.6
	Females	0.0	0.0	0.4	1.1	1.6	0.0	0.4
Total	Males	0.0	0.1	0.5	1.6	3.8	0.0	0.7
	Females	0.0	0.0	0.4	1.3	2.5	0.0	0.5
Years of life lost (rate per 100,000)								
Non-Māori	Males	1	2	9	25	42	0	10
	Females	1	1	8	22	30	0	8
Māori	Males	0	1	6	19	36	0	8
	Females	0	0	7	19	19	0	6
Total	Males	1	2	9	24	42	0	10
	Females	1	1	8	22	30	0	8

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 174:** Avoidable fractions (%)\* for vegetable and fruit intake and colorectal cancer, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	2	1	1	1	1	0
	Females	2	1	1	1	1	0
Māori	Males	2	1	1	1	1	0
	Females	2	1	1	1	1	0
Total	Males	2	1	1	1	1	0
	Females	2	1	1	1	1	0

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

**Table 175:** Avoidable mortality\* for vegetable and fruit intake and colorectal cancer, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	0	1	1	2
	Females	0	0	0	0	1	1	2
Māori	Males	0	0	0	0	0	0	0
	Females	0	0	0	0	0	0	0
Total	Males	0	0	0	0	1	1	2
	Females	0	0	0	0	1	1	2
Years of life lost (count)								
Non-Māori	Males	1	1	3	7	11	3	25
	Females	1	1	2	6	9	4	22
Māori	Males	0	0	0	1	1	0	2
	Females	0	0	0	1	0	0	2
Total	Males	1	1	3	7	11	3	27
	Females	1	1	2	6	9	4	23

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.

## Total

### Attributable mortality

**Table 176:** Attributable mortality (count) for vegetable and fruit intake and all diseases, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	3	18	62	137	284	305	809
	Females	1	4	16	45	128	390	585
Māori	Males	1	8	20	34	28	10	101
	Females	1	3	9	17	21	12	63
Total	Males	4	26	82	172	312	315	911
	Females	2	7	24	62	149	402	648
Years of life lost (count)								
Non-Māori	Males	79	411	1,227	2,155	3,116	1,525	8,514
	Females	40	102	325	764	1,559	2,839	5,629
Māori	Males	19	194	398	539	308	49	1,507
	Females	14	76	180	290	251	90	900
Total	Males	99	605	1,625	2,694	3,424	1,574	10,021
	Females	54	178	505	1,053	1,810	2,929	6,529

### Avoidable mortality

**Table 177:** Avoidable mortality\* for vegetable and fruit intake and all diseases, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	9	32	55	76	174
	Females	0	1	3	10	25	78	117
Māori	Males	0	1	4	9	8	4	27
	Females	0	0	2	5	6	5	19
Total	Males	1	2	13	41	63	80	201
	Females	1	1	5	14	31	84	136
Years of life lost (count)								
Non-Māori	Males	12	35	179	499	607	381	1,714
	Females	8	14	55	165	303	571	1,115
Māori	Males	6	22	83	142	88	21	363
	Females	7	10	45	77	76	39	254
Total	Males	18	58	262	642	695	402	2,076
	Females	15	24	101	242	379	610	1,370

\* Due to a 40 g/day increase in mean vegetable and fruit intake over and above the BAU scenario.



## Body mass index

### Ischaemic heart disease

#### *Attributable mortality*

**Table 178:** Attributable fractions (%) for body mass index and ischaemic heart disease, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	50	51	47	40	28	15
	Females	52	55	52	44	32	17
Māori	Males	75	73	70	57	38	17
	Females	71	72	67	60	44	25
Total	Males	54	58	53	43	28	15
	Females	55	63	58	48	34	17

**Table 179:** Attributable mortality (count) for body mass index and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	5	28	88	154	254	263	792
	Females	2	4	17	46	124	349	541
Māori	Males	1	18	38	51	31	9	148
	Females	0	5	12	23	24	15	80
Total	Males	6	46	126	205	285	272	939
	Females	2	9	28	68	148	365	621
Years of life lost (count)								
Non-Māori	Males	122	648	1,751	2,417	2,790	1,315	9,043
	Females	46	101	345	772	1,499	2,546	5,309
Māori	Males	32	416	756	797	341	43	2,385
	Females	12	126	243	382	296	112	1,173
Total	Males	154	1,065	2,507	3,214	3,131	1,358	11,428
	Females	59	227	589	1,154	1,795	2,658	6,482

**Table 180:** Attributable mortality (rate) for body mass index and ischaemic heart disease, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	2.0	11.4	43.0	110.2	225.3	394.8	69.3
	Females	0.7	1.7	8.1	32.5	100.1	311.0	32.6
Māori	Males	2.9	53.2	185.0	406.7	534.4	472.8	183.0
	Females	1.0	14.4	54.8	174.9	364.2	556.5	102.7
Total	Males	2.1	16.5	55.9	134.5	240.5	396.9	78.2
	Females	0.8	3.3	12.5	44.5	113.7	317.0	37.2
Years of life lost (rate per 100,000)								
Non-Māori	Males	51	265	856	1,729	2,472	1,974	828
	Females	19	40	170	548	1,215	2,266	359
Māori	Males	76	1,239	3,684	6,383	5,862	2,365	2,633
	Females	26	346	1,145	2,953	4,420	4,054	1,363
Total	Males	55	383	1,114	2,111	2,638	1,985	979
	Females	20	79	262	751	1,380	2,309	434

\* Age-standardised to WHO World population.

### Avoidable mortality

**Table 181:** Avoidable fractions (%)\* for body mass index and ischaemic heart disease, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	4	5	4	3	2	1
	Females	5	6	5	4	2	2
Māori	Males	10	9	8	5	4	2
	Females	8	9	7	6	4	2
Total	Males	5	6	5	4	2	1
	Females	6	7	6	4	3	2

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

**Table 182:** Avoidable mortality\* for body mass index and ischaemic heart disease, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	1	4	10	13	13	40
	Females	0	0	0	2	6	20	28
Māori	Males	0	1	3	4	4	1	12
	Females	0	0	1	1	2	1	5
Total	Males	0	1	6	14	16	15	52
	Females	0	0	1	4	8	21	33
Years of life lost (count)								
Non-Māori	Males	2	15	71	154	138	66	445
	Females	0	1	10	36	70	144	262
Māori	Males	1	16	55	63	39	7	181
	Females	0	3	11	24	22	9	69
Total	Males	3	30	126	217	177	73	626
	Females	0	4	21	60	93	153	331

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Ischaemic stroke

### Attributable mortality

**Table 183:** Attributable fractions (%) for body mass index and ischaemic stroke, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	54	55	51	43	30	17
	Females	56	59	56	47	35	19
Māori	Males	78	77	74	61	42	19
	Females	74	75	71	64	48	28
Total	Males	66	59	54	46	31	17
	Females	68	63	61	51	37	19

**Table 184:** Attributable mortality (count) for body mass index and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	4	12	35	76	130
	Females	0	1	4	9	31	165	210
Māori	Males	0	1	1	3	3	2	11
	Females	0	0	2	4	4	5	16
Total	Males	0	2	6	16	38	78	140
	Females	1	1	6	12	36	169	226
Years of life lost (count)								
Non-Māori	Males	4	43	89	192	384	380	1,091
	Females	4	23	81	145	382	1,200	1,836
Māori	Males	6	13	22	53	35	11	140
	Females	11	11	44	65	52	34	217
Total	Males	10	57	111	245	418	391	1,231
	Females	15	34	126	210	435	1,234	2,053

**Table 185:** Attributable mortality (rate) for body mass index and ischaemic stroke, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.1	0.8	2.2	8.7	31.0	114.0	10.8
	Females	0.1	0.4	1.9	6.1	25.5	146.6	11.5
Māori	Males	0.5	1.7	5.3	27.1	54.1	120.6	16.9
	Females	0.9	1.2	10.0	29.6	64.4	167.4	21.6
Total	Males	0.1	0.9	2.5	10.2	32.1	114.2	11.2
	Females	0.2	0.5	2.7	8.1	27.5	147.1	12.2
Years of life lost (rate per 100,000)								
Non-Māori	Males	2	18	44	137	340	570	94
	Females	2	9	40	103	310	1,068	111
Māori	Males	14	39	106	426	594	603	182
	Females	24	29	208	499	782	1,220	263
Total	Males	3	20	49	161	352	571	101
	Females	5	12	56	136	334	1,072	123

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 186:** Avoidable fractions (%)\* for body mass index and ischaemic stroke, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	5	5	4	4	2	1
	Females	6	6	6	4	3	2
Māori	Males	11	10	9	6	5	2
	Females	9	10	8	7	4	2
Total	Males	8	6	5	4	2	1
	Females	8	7	7	5	3	2

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

**Table 187:** Avoidable mortality\* for body mass index and ischaemic stroke, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
<b>Deaths (count)</b>								
Non-Māori	Males	0	0	0	1	2	5	8
	Females	0	0	0	1	1	13	15
Māori	Males	0	0	0	0	0	0	1
	Females	0	0	0	0	0	1	1
Total	Males	0	0	0	1	2	5	9
	Females	0	0	0	1	2	13	17
<b>Years of life lost (count)</b>								
Non-Māori	Males	1	2	5	14	19	23	64
	Females	0	2	5	9	18	94	127
Māori	Males	2	1	2	5	4	2	16
	Females	2	1	4	5	4	4	20
Total	Males	2	4	8	19	23	25	81
	Females	2	3	9	14	22	98	148

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Diabetes

### Attributable mortality

**Table 188:** Attributable fractions (%) for body mass index and diabetes, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	86	91	81	77	74	72
	Females	96	99	96	90	79	70
Māori	Males	99	99	96	91	90	80
	Females	99	100	99	98	91	83
Total	Males	92	95	89	83	77	73
	Females	98	99	98	95	83	72

**Table 189:** Attributable mortality (count) for body mass index and diabetes,\* 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	2	9	33	95	182	94	415
	Females	1	7	28	72	136	96	341
Māori	Males	2	12	40	85	63	14	216
	Females	2	14	46	96	78	23	259
Total	Males	4	21	74	180	244	109	632
	Females	3	21	73	169	215	119	600
Years of life lost (count)								
Non-Māori	Males	44	211	663	1,491	1,993	472	4,875
	Females	25	166	583	1,222	1,656	700	4,352
Māori	Males	51	276	804	1,337	688	72	3,228
	Females	52	336	950	1,624	948	170	4,080
Total	Males	95	488	1,467	2,828	2,681	544	8,103
	Females	78	502	1,533	2,846	2,604	870	8,433

\* Not adjusted for CVD–diabetes overlap.

**Table 190:** Attributable mortality (rate) for body mass index and diabetes,<sup>1</sup> 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total <sup>2</sup>
Deaths (rate per 100,000)								
Non-Māori	Males	0.7	3.7	16.3	67.9	161.0	141.8	36.3
	Females	0.4	2.7	13.7	51.4	110.6	85.6	25.5
Māori	Males	4.6	35.3	196.9	682.7	1,076.5	783.4	287.1
	Females	4.2	38.2	214.2	743.0	1,165.3	842.6	311.1
Total	Males	1.3	7.5	32.7	118.3	205.9	159.0	53.0
	Females	1.0	7.2	32.6	109.7	164.9	103.8	44.9
Years of life lost (rate per 100,000)								
Non-Māori	Males	19	87	324	1,066	1,766	709	442
	Females	10	66	286	868	1,342	623	356
Māori	Males	120	823	3,921	10,714	11,808	3,917	3,840
	Females	112	918	4,472	12,544	14,141	6,139	4,565
Total	Males	34	176	652	1,857	2,258	795	697
	Females	27	174	681	1,851	2,001	756	674

1 Not adjusted for CVD–diabetes overlap.

2 Age-standardised to WHO World population.

### ***Avoidable mortality***

**Table 191:** Avoidable fractions (%)<sup>\*</sup> for body mass index and diabetes, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	13	16	11	10	8	8
	Females	25	33	25	17	11	14
Māori	Males	30	29	22	15	25	24
	Females	35	41	30	26	16	12
Total	Males	21	24	17	12	13	11
	Females	31	38	28	22	13	13

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

**Table 192:** Avoidable mortality<sup>1</sup> for body mass index and diabetes, 2011<sup>2</sup>

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	5	17	24	16	64
	Females	0	2	10	19	20	24	75
Māori	Males	1	5	13	21	31	13	84
	Females	1	6	24	38	22	9	100
Total	Males	1	6	19	38	55	29	148
	Females	1	8	33	58	42	33	176
Years of life lost (count)								
Non-Māori	Males	7	36	106	268	262	80	759
	Females	7	55	199	325	244	175	1,005
Māori	Males	15	110	267	328	343	65	1,128
	Females	16	145	499	648	267	68	1,643
Total	Males	22	146	373	596	604	146	1,887
	Females	24	200	698	973	511	242	2,648

1 Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

2 Not adjusted for CVD–diabetes overlap.

## Colorectal cancer

### Attributable mortality

**Table 193:** Attributable fractions (%) for body mass index and colorectal cancer, 1997

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	13	16	17	17	16	13
	Females	13	16	18	19	18	14
Māori	Males	22	27	29	27	22	14
	Females	21	25	27	28	26	21
Total	Males	13	17	18	18	16	13
	Females	13	17	19	20	18	14



**Table 194:** Attributable mortality (count) for body mass index and colorectal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	1	8	18	32	24	83
	Females	0	1	7	16	25	36	84
Māori	Males	0	0	1	2	2	0	5
	Females	0	0	1	2	1	1	5
Total	Males	0	1	9	20	33	25	88
	Females	0	1	8	17	26	36	89
Years of life lost (count)								
Non-Māori	Males	9	29	154	280	347	121	940
	Females	10	18	143	262	305	260	998
Māori	Males	0	4	17	30	20	2	74
	Females	0	2	19	30	15	5	70
Total	Males	9	33	172	310	367	123	1,014
	Females	10	20	162	292	319	265	1,069

**Table 195:** Attributable mortality (rate) for body mass index and colorectal cancer, 1997

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total*
Deaths (rate per 100,000)								
Non-Māori	Males	0.1	0.5	3.8	12.8	28.0	36.2	7.2
	Females	0.2	0.3	3.4	11.0	20.3	31.8	5.9
Māori	Males	0.0	0.5	4.2	15.4	32.1	25.8	7.5
	Females	0.0	0.2	4.3	13.9	17.9	22.9	5.8
Total	Males	0.1	0.5	3.8	13.0	28.2	36.0	7.3
	Females	0.1	0.3	3.4	11.3	20.2	31.6	5.9
Years of life lost (rate per 100,000)								
Non-Māori	Males	4	12	75	200	307	181	85
	Females	4	7	70	186	247	232	79
Māori	Males	0	12	84	241	352	129	93
	Females	0	5	89	234	217	167	82
Total	Males	3	12	76	204	309	180	86
	Females	3	7	72	190	245	230	79

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 196:** Avoidable fractions (%) for body mass index and colorectal cancer, 2011

	Sex	Age group (years)					
		25–34	35–44	45–54	55–64	65–74	75+
Non-Māori	Males	1	1	1	1	1	1
	Females	1	1	1	1	1	2
Māori	Males	1	2	2	2	3	2
	Females	1	1	2	2	2	1
Total	Males	1	1	1	1	1	1
	Females	1	1	1	1	1	2

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

**Table 197:** Avoidable mortality\* for body mass index and colorectal cancer, 2011

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	0	0	1	2	2	5
	Females	0	0	0	1	1	5	7
Māori	Males	0	0	0	0	0	0	1
	Females	0	0	0	0	0	0	0
Total	Males	0	0	0	1	2	2	6
	Females	0	0	0	1	1	5	7
Years of life lost (count)								
Non-Māori	Males	0	1	5	13	18	11	49
	Females	0	1	4	11	16	34	67
Māori	Males	0	0	1	2	3	1	7
	Females	0	0	1	2	1	1	4
Total	Males	0	2	6	15	21	12	56
	Females	0	1	5	13	18	34	71

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Post-menopausal breast cancer

### Attributable mortality

**Table 198:** Attributable fractions (%) for body mass index and post-menopausal breast cancer, 1997

	Age group (years)			
	50–54	55–64	65–74	75+
Non-Māori	18	19	18	14
Māori	27	28	26	21
Total	20	20	19	14

**Table 199:** Attributable mortality (count) for body mass index and post-menopausal breast cancer, 1997

	Age group (years)				
	50–54	55–64	65–74	75+	Total
<b>Deaths (count)</b>					
Non-Māori	11	22	22	27	82
Māori	2	4	2	1	9
Total	13	26	24	28	91
<b>Years of life lost (count)</b>					
Non-Māori	213	372	271	195	1,050
Māori	42	64	25	9	140
Total	255	435	296	203	1,189

**Table 200:** Attributable mortality (rate) for body mass index and post-menopausal breast cancer, 1997

	Age group (years)				
	50–54	55–64	65–74	75+	Total*
<b>Deaths (rate per 100,000)</b>					
Non-Māori	11.4	15.6	18.1	23.8	16.3
Māori	23.4	29.2	30.7	43.2	30.1
Total	12.5	16.8	18.7	24.2	17.2
<b>Years of life lost (rate per 100,000)</b>					
Non-Māori	231	264	219	173	233
Māori	472	492	372	315	434
Total	252	283	227	177	247

\* Age-standardised to WHO World population.

## Avoidable mortality

**Table 201:** Avoidable fractions (%)\* for body mass index and post-menopausal breast cancer, 2011

	Age group (years)			
	50–54	55–64	65–74	75+
Non-Māori	1	1	1	2
Māori	2	2	2	1
Total	1	1	1	2

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

**Table 202:** Avoidable mortality for body mass index and post-menopausal breast cancer, 2011

	Age group (years)				
	50–54	55–64	65–74	75+	Total
<b>Deaths (count)</b>					
Non-Māori	1	2	1	3	7
Māori	0	0	0	0	1
Total	1	2	2	4	8
<b>Years of life lost (count)</b>					
Non-Māori	15	27	18	25	86
Māori	5	6	2	1	14
Total	20	33	20	27	100

\* Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

## Total

### Attributable mortality

**Table 203:** Attributable mortality (count) for body mass index and all diseases, 1997\*

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	7	38	126	258	463	437	1,328
	Females	3	11	60	148	309	652	1,183
Māori	Males	3	28	71	123	85	23	332
	Females	2	17	52	107	93	40	311
Total	Males	10	66	198	381	548	459	1,661
	Females	5	28	112	255	402	692	1,494
Years of life lost (count)								
Non-Māori	Males	169	886	2,512	4,052	5,075	2,183	14,877
	Females	80	271	1,245	2,504	3,748	4,747	12,595
Māori	Males	78	649	1,422	1,923	933	113	5,117
	Females	65	401	1,091	1,807	1,128	292	4,784
Total	Males	247	1,535	3,934	5,974	6,007	2,296	19,994
	Females	145	672	2,336	4,311	4,876	5,039	17,379

\* Adjusted for CVD–diabetes overlap.

### Avoidable mortality

**Table 204:** Avoidable mortality<sup>1</sup> for body mass index and all diseases, 2011<sup>2</sup>

	Sex	Age group (years)						
		25–34	35–44	45–54	55–64	65–74	75+	Total
Deaths (count)								
Non-Māori	Males	0	2	8	25	35	33	102
	Females	0	2	9	20	26	60	117
Māori	Males	1	4	13	21	29	12	80
	Females	1	5	20	32	20	9	86
Total	Males	1	6	22	46	63	45	182
	Females	1	7	29	52	45	69	203
Years of life lost (count)								
Non-Māori	Males	8	47	164	390	379	163	1,150
	Females	7	47	189	337	312	434	1,326
Māori	Males	14	103	267	326	314	61	1,085
	Females	15	117	410	543	238	67	1,390
Total	Males	22	150	431	716	693	224	2,235
	Females	21	164	599	880	550	501	2,716

<sup>1</sup> Due to a 1.0 kg/m<sup>2</sup> increase in mean body mass index over the current BMI distribution, rather than the 1.3 kg/m<sup>2</sup> increase under the BAU scenario.

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