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# Nutrition and the burden of disease in New Zealand: 1997–2011

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## Abstract

*Objective:* To estimate the burden of disease due to selected nutrition-related risk factors (high total blood cholesterol, high systolic blood pressure, high body mass index (BMI) and inadequate vegetable and fruit intake) in 1997, as well as the burden that could potentially be avoided in 2011 if small, favourable changes in the current risk factor distribution were to occur.
*Design:* Data on risk factor levels, disease burden and risk associations were combined using comparative risk assessment methodology, a systematic approach to estimating both attributable and avoidable burden of disease. Disease outcomes assessed varied according to risk factor and included ischaemic heart disease, stroke, type 2 diabetes mellitus and selected cancers.
*Setting:* New Zealand.
*Results:* Approximately 4500 deaths (17% of all deaths) in 1997 were attributable to high cholesterol, 3500 (13%) to high blood pressure, 3000 (11%) to high BMI and 1500 (6%) to inadequate vegetable and fruit intake. Taking prevalence overlap into account, these risk factors were estimated jointly to contribute to approximately 11 000 (40%) deaths annually in New Zealand. Approximately 300 deaths due to each risk factor could potentially be avoided in 2011 if modest changes were made to each risk factor distribution.
*Conclusions:* High cholesterol, blood pressure and BMI, as well as inadequate vegetable and fruit intake, are major modifiable causes of death in New Zealand. Small changes in the population distribution of these risk factors could have a major impact on population health within a decade.

**Keywords**
Nutrition
New Zealand
Mortality

Ischaemic heart disease, stroke, diabetes and cancer are leading causes of death in developed countries[1]. In 1997, these diseases resulted in approximately 17 500 deaths in adults aged over 25 years in New Zealand, 64% of all deaths in this age group[2].

It is well established that nutrition-related factors (such as blood pressure, cholesterol, vegetable and fruit intake, and body mass index (BMI)) play an important role in the development of ischaemic heart disease, stroke, diabetes and cancer[3]. However, the importance of sub-optimal nutrition on the health of the New Zealand population has never been fully quantified. Progress in recent years, both in the understanding of risk factor–disease relationships and in the estimation of population exposure to these risk factors, allows quantification of the impact of nutrition on mortality in New Zealand. Such information is relevant to policy-makers, health planners and health providers.

The relationship between some risk factors and disease outcomes exists on a continuous scale. This continuous relationship means that the level of disease risk is increased even amongst those with traditionally 'normal' risk factor exposure. As a consequence, a large number of people exposed to a small risk may generate more cases than a small number exposed to high risk[4]. Therefore, categorisation into 'exposed' and 'not exposed', as has been done in the past, can substantially underestimate the burden of disease due to a specific risk factor or group of risk factors. This study employed a modification of the 'comparative risk assessment' (CRA) methodology developed by the World Health Organization (WHO)[5] and used in the *World Health Report 2002* (WHR 2002)[1]. The CRA approach recognises that risk factor–disease relationships are continuous rather than categorical. By using full risk factor distributions, the method therefore provides a more accurate estimate of the disease burden attributable to selected risk factors than the traditional population-attributable risk methods, which use a categorical approach.

To date, traditional risk assessments have focused on the proportion of current burden caused by prior

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exposure to a risk factor – the population-attributable risk. However, of more policy relevance than attributable burden is the question of how many deaths could be avoided in the future. Taking the whole risk factor distribution into account, as can be done using CRA, also allows us to estimate how much disease could be potentially avoided if small, favourable changes in the current risk factor distributions were to occur.

The calculation of attributable and avoidable burden due to selected risk factors was carried out as part of the WHR 2002[1]. However, in the WHR 2002 New Zealand was included in the Western Pacific 'A' region, along with Australia, Japan, Singapore and Brunei, and country-specific burdens were not calculated. The objective of the present study was therefore to estimate, for selected nutrition-related risk factors, the New Zealand-specific attributable mortality in 1997 and the number of deaths that could potentially be avoided (avoidable burden) in 2011 if small, favourable changes in the current risk factor distributions were to occur.

## Methods

Full details of the CRA methodology utilised by the WHO and in this New Zealand project have been described elsewhere[1,5–7]. Using CRA methodology, attributable burden is calculated by comparing the burden of disease under the current risk factor distribution with that under an alternative risk factor distribution that would confer the lowest overall population risk of disease (the 'theoretical minimum' distribution)[8]. CRA also enables estimation of how much disease could potentially be avoided each year in the future if small favourable changes in the current risk factor distributions were to occur (the 'avoidable' burden). Figure 1 summarises the data inputs required for the estimation of attributable and avoidable burden.

Because we are considering a continuous distribution, there is no cut-off for 'high' risk. In this paper, high risk (e.g. high blood pressure) refers to the risk attributed to higher than optimal levels of the risk factor (optimal levels being those conferred by the theoretical minimum distribution). Similarly, 'inadequate' vegetable and fruit intake refers to lower than optimal intake.

### *Risk factor distributions*

Risk factors were chosen because of their impact and potential modifiability, as well as the availability of data on their distributions and disease relationships. The nutrition-related risk factors selected were high total blood cholesterol, high systolic blood pressure, high BMI and inadequate vegetable and fruit intake.

Data on the current population distribution of all the selected risk factors by 10-year age bands (from age 25 years) and sex were extracted from the 1997 National Nutrition Survey, a nationally representative survey of 4636 New Zealand adults living in private households[9]. Blood pressure measurements from this survey were considered 'unreliable' by investigators (N Wilson, personal communication, November 2001) due to calibration difficulties resulting in higher blood pressure measurements. Systolic blood pressure measurements from the 1997 National Nutrition Survey were therefore adjusted downwards, to bring them in line with data from three cross-sectional surveys undertaken in Auckland, New Zealand during the 1980s and early 1990s[10]. Further details on the correction of the blood pressure calibration error can be found elsewhere[6].

The theoretical minimum distribution for each risk factor (that which would yield the lowest population risk of disease) was defined by the expert working groups set up by the WHO for the WHR 2002 (Table 1). Levels were based on a variety of epidemiological data, and full details on how theoretical minimum distributions were defined can be found elsewhere[6,11–14].

### *Distributional transitions*

To calculate avoidable burden, 'distributional transitions' were modelled. These are relative shifts away from the current distribution of the risk factor towards the theoretical minimum distribution[5,15]. Avoidable burden was estimated from the difference in disease burden under two distributional transitions: a 'business as usual' (BAU)

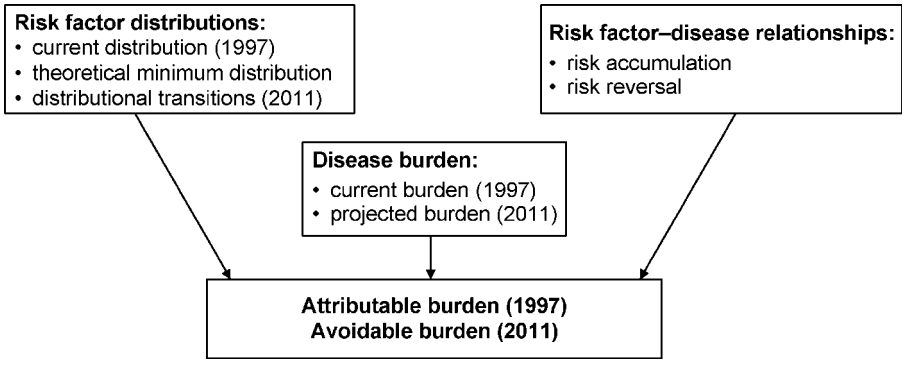


**Fig. 1** Data inputs for comparative risk assessment. Adapted from the *World Health Report 2002*[1]

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**Table 1** Risk factor–disease combinations, theoretical minimum and distributional transitions

| Risk factor | Disease outcome | Theoretical minimum distribution (mean) | Distributional shift towards the theoretical minimum | | Average change in risk factor after intervention scenario (range)* |
|---|---|---|---|---|---|
| | | | Business as usual (BAU) | Intervention | |
| Total blood cholesterol | Ischaemic heart disease<br>Stroke‡ | 3.8 mmol $l^{-1}$ | 7% | 10% | 0.1 (0.01–0.2) mmol $l^{-1}$ decrease |
| Systolic blood pressure | Ischaemic heart disease<br>Stroke | 115 mmHg | 8% | 16% | 0.5 (0.1–3) mmHg decrease |
| Body mass index† | Ischaemic heart disease<br>Stroke‡<br>Diabetes<br>Cancer§ | 21 kg $m^{-2}$ | 25% | 19% | 1.0 (0.8–1.2) kg $m^{-2}$ increase |
| Vegetable and fruit intake | Ischaemic heart disease<br>Stroke‡<br>Diabetes<br>Cancer¶ | 600 g $day^{-1}$ | 22% | 44% | 40 g $day^{-1}$ increase |

* Change over and above the BAU scenario.
† Unlike other risk factors, the intervention scenario for body mass index did not include an improvement in the current situation since there is no evidence that the obesity epidemic has peaked – the shift in distribution is *away* from the theoretical minimum. Consequently, the intervention scenario for body mass index involves a smaller increase in body mass index than that projected if current trends continue.
‡ Ischaemic stroke only.
§ Postmenopausal breast and colorectal cancers.
¶ Oesophageal, stomach, colorectal and lung cancers.

scenario and an 'intervention' scenario. BAU scenarios were intended to represent continuation over the next decade of historical trends in risk factor levels. The distributional transitions selected for the intervention scenarios were intended to be illustrative of feasible changes in the population exposure distribution to each risk factor over the next decade (Table 1).

### *Disease outcomes*

Selection of disease outcomes 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 reliable data on risk factor–disease associations. The disease outcomes assessed differed for each risk factor and included ischaemic heart disease, stroke, type 2 diabetes mellitus and selected cancers (Table 1).

The best measure of burden of disease is a summary of both fatal and non-fatal outcomes using an integrated measure of population health such as the disability-adjusted life year. However, in these analyses, burden of disease was restricted to fatal outcomes only because of limitations in the data available to estimate disease incidence and the lack of health-state valuations specific to New Zealand.

Data on current (1997) disease burden were extracted from the New Zealand Health Information Service mortality database. The number of deaths by 10-year age group (from age 25) and sex for each selected disease were extracted. Data were averaged for 1996–1998 to provide more reliable estimates.

The following codes from the WHO International Classification of Disease, Injuries, and Causes of Death, version 9 were used: 410–414 (ischaemic heart disease); 431–438 (stroke); 150 (oesophageal cancer); 151 (stomach cancer); 153–154 (colorectal cancer) 162 (lung cancer); and 174 (breast cancer).

To calculate future disease burden, projections of total cause-specific mortality through to 2011 were made. For non-cancer endpoints, classical age/period/cohort (APC) models were constructed[16]. For cancer endpoints, Bayesian and generalised non-linear modelling methods were used in addition to the APC models[17]. Further details on how future mortality was estimated are described elsewhere[6].

### *Risk factor–disease relationships*

Data on the magnitude of the risk factor–disease relationships (relative risk) for each nutritional risk factor and endpoint were based on those used in the WHR 2002[1,7]. Wherever possible, these estimates were based on overviews of cohort studies rather than individual studies and verified with data from randomised controlled trial overviews. Age-specific relative risk estimates were used, and where data were lacking for older age groups, a degree of age attenuation was modelled. Sex-specific estimates were necessary only for the BMI and diabetes associations.

Data on the magnitude and timing of the reduction in risk when the exposure distribution is shifted towards the theoretical minimum distribution (risk reversal) are also necessary to calculate avoidable burden. This information was obtained from overviews of clinical trials[11–14].

### *Calculation of attributable and avoidable burden*

Both attributable and avoidable burdens were calculated using potential impact fractions (PIFs) (Appendix A). PIFs are a multi-level extension of the conventional population-attributable risk measure widely used and extended

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to take into account continuous risk factor–disease relationships.

The attributable burden is estimated by applying the relevant PIF to the estimate of current burden. Further details of the methodology used to estimate attributable burden, with examples, can be found elsewhere[1,5–7,18]. An estimate of the joint effect of combining the four risk factors was obtained using a formula that adjusted for overlap between the risk factors (Appendix B)[19–21].

Avoidable burden was calculated by extending the methodology of the PIF formula described in Appendix A to include estimates of risk reversibility in the calculations, and applying the fractions calculated to the projected 2011 disease burden under both the BAU and intervention scenarios. The avoidable burden was the difference in mortality under these two scenarios.

Confidence intervals for the attributable burden were estimated by a simulation procedure incorporating sources of uncertainty from domains of the exposure distribution (i.e. mean and standard deviation) and the exposure–response relationships. Briefly, the method involved simultaneously varying all input parameters within their respective distributions and reiterating the calculation of the population-attributable fraction. An uncertainty distribution around each estimate of the population-attributable fraction was obtained after 500 iterations of the simulation and, from this, 95% confidence intervals were derived[1].

## Results

### *Attributable burden*

In 1997, 4721 (95% confidence interval: 4205–5116) deaths were attributable to high total blood cholesterol, 3699 (2843–4305) deaths to high systolic blood pressure, 3154 (2591–3645) deaths to high BMI and 1559 (880–2595) deaths to inadequate vegetable and fruit intake (Table 2). For all four nutrition-related risk factors selected, ischaemic heart disease made up the greatest proportion (ranging from 46% for BMI to 87% for total blood cholesterol) of the mortality burden attributable to each risk factor (Fig. 2). Almost one-third of all deaths caused by high BMI were due to diabetes.

The attributable mortality was split relatively equally between males and females for all risk factors except inadequate vegetable and fruit intake, where a greater proportion of the attributable deaths occurred in males than in females (60% vs. 40%). The distribution of attributable deaths by age varied by risk factor. The deaths attributable to high cholesterol and blood pressure tended to be in older age groups (about 60% of deaths occurred in those aged over 75 years) compared with those for high BMI and inadequate vegetable and fruit intake (35–45% of deaths occurred in those aged over 75 years).

Taking into account overlap between risk factors, it was estimated that approximately 11 000 deaths (40% of all deaths) in 1997 were due to the selected risk factors. Over half of these attributable deaths were due to ischaemic heart disease, with stroke the second leading cause of death. Overall, approximately 87% of all ischaemic heart disease deaths, 70% of all stroke deaths, 83% of all diabetes deaths and 6% of all cancer deaths could potentially be attributed to the combined effects of the selected nutrition-related risk factors.

### *Avoidable burden*

Table 1 summarises the intervention scenarios used to estimate avoidable burden. These changes in risk factor distribution are over and above what would be expected with continuation of current trends (BAU scenario). Although the average change in risk factor level is shown, a general shift in the whole distribution towards the theoretical minimum was modelled. This implies that greater improvements in risk factor levels will occur in the tail end of the distribution than the mean shifts shown in Table 1.

Under the specified intervention scenarios, approximately 300 deaths due to each risk factor would potentially be avoided in 2011 from feasible changes in each risk factor distribution. The greatest proportion of deaths avoided through improvements in total blood cholesterol, systolic blood pressure or vegetable and fruit intake would be from ischaemic heart disease (Fig. 3). The greatest proportion of deaths potentially avoided following a slowing down of the obesity epidemic would be due to diabetes.

Although the formula used to calculate joint effects for attributable burden is not directly applicable to avoidable burden, if all four risk factor distributions were changed as outlined and overlap was similar to that for attributable burden, then a total of over 1000 deaths would potentially be avoided.

**Table 2** Total attributable burden in 1997 and avoidable mortality in 2011, all-cause mortality for nutrition-related risk factors

| | Attributable burden, 1997 | | | Avoidable mortality, 2011 |
|---|---|---|---|---|
| Risk factor | Deaths (count) | Percentage of all deaths | Percentage of years of life lost | Deaths (count) |
| Total blood cholesterol | 4721 (4205–5116)† | 17% (15–19) | 16% (14–17) | 300 |
| Systolic blood pressure | 3699 (2843–4305) | 13% (10–16) | 12% (9–14) | 282 |
| Body mass index* | 3154 (2591–3645) | 11% (9–13) | 13% (11–15) | 385 |
| Vegetable and fruit intake | 1559 (880–2595) | 6% (3–9) | 6% (3–9) | 334 |

* Adjusted for cardiovascular disease/diabetes overlap.
† 95% confidence interval.

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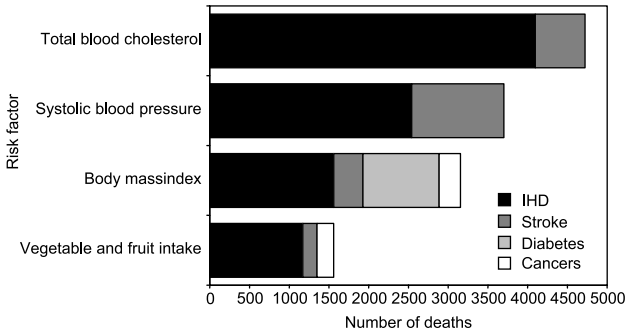


**Fig. 2** Attributable burden by risk factor and disease, 1997. IHD – ischaemic heart disease

## Discussion

This paper presents the first reliable estimates of the mortality burden due to nutrition-related risk factors in New Zealand. For the New Zealand population aged over 25 years in 1997, about 4500 deaths (17% of all deaths) were attributable to high total blood cholesterol, 3500 (13%) to high systolic blood pressure, 3000 (11%) to high BMI and 1500 (6%) to inadequate vegetable and fruit intake. The combined mortality burden of these four risk factors was estimated to be about 11 000 deaths. This represents just under 40% of all deaths occurring in that year, but approximately 87% of all ischaemic heart disease deaths, 83% of all diabetes deaths, 70% of all stroke deaths and 6% of all cancer deaths. The proportion of cancer deaths attributable to the selected risk factors is almost certainly an underestimate because only a limited number of cancer types were included in the model (those for which a causal link to the selected risk factors has been established and sufficient data to quantify the risk factor–disease relationship exist).

Comparison with previous estimates of the burden of disease caused by other risk factors indicates that cholesterol and blood pressure rank highly alongside tobacco smoking as important causes of death in New Zealand. In 1997, tobacco was the leading cause of death in New Zealand, responsible for about 5000 deaths[22].

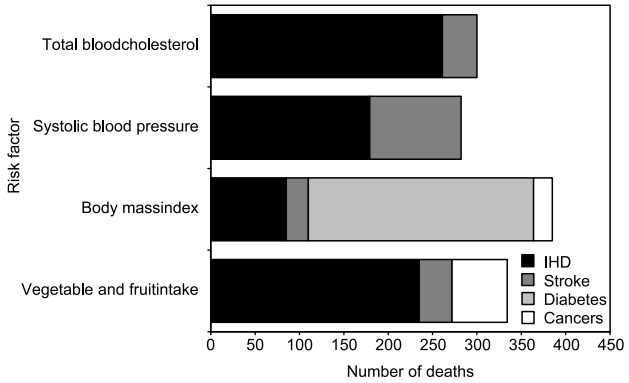


**Fig. 3** Avoidable burden by risk factor and disease, 2011. IHD – ischaemic heart disease

In the same year the road toll, which attracts much more policy attention than nutrition-related risk factors, resulted in 500 deaths[22]. If tobacco consumption is declining faster than are population blood cholesterol levels, in time high total blood cholesterol may overtake tobacco as the leading cause of mortality in New Zealand, although not necessarily as the leading cause of *avoidable* mortality or of potential years of life lost. Other changes that are likely to influence the ranking of the five leading risk factors (in terms of attributable mortality) over the next 10 or 20 years are the possible stabilisation of systolic blood pressure levels and the anticipated further increase in BMI.

The current analyses also estimated how many deaths could potentially be avoided in the future given realistic changes in risk factor distributions over the next decade. While more difficult to estimate accurately, avoidable burden is of more relevance to policy than attributable burden. Under the specified intervention scenarios, approximately 300 deaths due to each risk factor could potentially be avoided in 2011 from feasible changes in each risk factor distribution. Changes at least as great as those illustrated in the intervention scenarios have been observed in other similar countries[23–25]. In calculating avoidable burden the most up-to-date disease projections were used. These projections took into account trends of other risk factors as well as screening and treatment of disease. As a consequence, the avoidable burden estimated in this study is based only on changes in the risk factor concerned.

While this study provides the most accurate estimates of nutrition-related mortality to date, it has several limitations. Only fatal outcomes were included as a measure of the burden of disease. Including non-fatal outcomes would have provided a more comprehensive estimate of attributable and avoidable burdens through incorporating the impact of disability as well as that of premature mortality. Unfortunately, insufficient data were available to estimate disease incidence (as opposed to mortality). Furthermore, the lack of New Zealand-specific health-state valuations made the calculation of non-fatal burdens measured as disability-adjusted life years problematic. However, it is considered unlikely that including non-fatal outcomes would have a substantial impact on the relative effect sizes or rank ordering of different nutritional risk factors.

Other potential limitations relate to the input data utilised. Risk factor data were sourced from the 1997 National Nutrition Survey. Total blood cholesterol and BMI were measured in this survey using blood tests and height and weight measurements, respectively, and are considered the most reliable data. Data used to estimate vegetable and fruit intake were based on a 24-hour dietary recall, which may not accurately represent an individual's usual intake. As discussed earlier, the original blood pressure survey data were considered unreliable, and a correction factor was required to bring the

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estimates into line with other cross-sectional New Zealand surveys. Although the blood pressure and vegetable and fruit data are not ideal, they are the most recent, reliable, nationally representative estimates available for these risk factors.

The study results are also dependent on the theoretical minimum distribution. The same theoretical minimum was used for both sexes and all ages. It is possible that age- and sex-specific theoretical minimum risk distributions would be more appropriate. However, the theoretical minimum distributions were set according to the best available evidence, and age- and sex-specific distributions were not considered necessary. Instead of a theoretical minimum distribution, other counterfactual distributions such as a 'cost-effective minimum risk' distribution[8] could have been used. However, the use of another counterfactual distribution would have limited the comparability of our results with other studies.

While burdens for the same risk factor can be added across mutually exclusive outcomes, burdens attributable to different risk factors are not additive across risk factors. 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 cause-specific mortality, is attributable to the combined action of the four risk factors included in the study. In this study, an approximate joint effect estimate was calculated assuming the risk factor exposures were independent and uncorrelated. Since in reality these conditions will often not be met, our joint effect estimates will tend to be overestimates.

In spite of the limitations discussed, the current estimates were based on the most reliable data currently available for the New Zealand population, and the best risk factor–disease relationship estimates. In addition, we have incorporated the sources of uncertainty surrounding the input data into 95% confidence intervals around each of our point estimates.

Comparison of attributable mortality in the current study with the developed regions in the WHR 2002[1] demonstrates similar results for cholesterol (17% vs. 16%), BMI (11% vs. 11%) and vegetable and fruit intake (6% vs. 8%). However, the attributable burden for blood pressure was lower in New Zealand (13%) than in the WHR 2002 (22%). This suggests that we may have over-corrected for the calibration error in the blood pressure data, so underestimating the burden of high systolic blood pressure in New Zealand. On the other hand, running a national burden of disease study may have resulted in a more accurate picture of the attributable burden due to systolic blood pressure for New Zealand than a global project that looked at regions rather than specific countries.

Valid comparison with other international studies is not possible, as many studies have not included estimates of the burden of disease attributable to the same risk factors[26,27]. While a recent Australian study did include the same risk factors[28], it used categorical attribution to calculate the burden of disease and thus the attributable burden estimates were not comparable to the current New Zealand analyses.

This study demonstrates that the well-established nutritional risk factors remain major modifiable causes of premature death, and are likely to continue to be so for several decades. Therefore, existent public health/nutrition messages and policies relating to our selected risk factors need to be maintained, reinforced and extended.

In developing any interventions, it must be acknowledged that nutritional exposures are 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[29]. Therefore, interventions to improve nutrition, physical activity and body weight should include strategies directly addressing the relevant risk or protective behaviours as well as 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. Intersectoral collaboration, involving sectors other than health, is needed. As specific policy options are developed for improving diet and maintaining or regaining healthy body weight, the strategies can be analysed and evaluated using the modelling tools reported here, so helping to identify 'best bets' and 'best buys'.

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## Disclaimer

Niki Stefanogiannis, Maria Turley and Martin Tobias are employees of the New Zealand Ministry of Health. The views expressed in this paper are the authors' own and do not represent the views or policies of the Ministry of Health. The paper was submitted for publication with the permission of the New Zealand Director General of Health.

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## Appendix A – Calculation of potential impact fraction

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

where $RR(x)$ is the relative risk at exposure level $x$, $P(x)$ is the population distribution of exposure, $P'(x)$ is the alternative or counterfactual distribution of exposure and $m$ is the maximum exposure level. (*Source: World Health Report 2002*[1].)

## Appendix B – Calculation of joint population-attributable fraction

$$PAF = 1 - \prod_{i=1}^{n} (1 - PAF_i),$$

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.

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