# Scenario Modelling of Potential Health Benefits Subsequent to the Introduction of the Proposed Standard for Nutrition, Health and Related Claims

Report developed for Food Standards Australia New Zealand

Centre for Health Economics Research and Evaluation

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## About CHERE

CHERE is an independent research unit affiliated with the University of Technology, Sydney. It has been established since 1991, and in that time has developed a strong reputation for excellence in research and teaching in health economics and public health and for providing timely and high quality policy advice and support. Its research program is policy-relevant and concerned with issues at the forefront of the sub-discipline.

CHERE has extensive experience in evaluating health services and programs, and in assessing the effectiveness of policy initiatives. The Centre provides policy support to all levels of the health care system, through both formal and informal involvement in working parties, committees, and by undertaking commissioned projects. For further details on our work, see www.chere.uts.edu.au.

# Table of Contents

# 1 Executive Summary

As part of Proposal P293, Food Standards Australia New Zealand (FSANZ) commissioned the Centre for Health Economics Research and Evaluation (CHERE), University of Technology, Sydney, to model the potential health benefits that might be possible for the populations of Australia and New Zealand, ten years after the introduction of the proposed standard for nutrition, health and related claims (Proposal P293).

By undertaking a scenario modelling exercise, the report models the potential health benefits that might be possible, ten years after the introduction of the proposed nutrition, health and related claims standard. For illustrative purposed we have focussed on the reductions in the intakes of two nutrients; 1) sodium from processed foods, and 2) saturated fats.

For sodium, this report focuses on sodium derived from salt added to food during processing because this is the sodium source that is most likely to be influenced by the health claims standard. Naturally occurring sodium, discretionary salt and non-salt sodium containing additives will not be discussed in this report, because their consumption or levels in food is unlikely to be affected by the proposed Standard.

It is important to note that the health benefits provided in this report are purely hypothetical, and based upon high and low case scenarios provided by FSANZ. The actual contribution to these scenarios from food manufacturers specifically reacting to the new health claims standard is unknown. Therefore any health benefits proposed by this report are not posed as being due to the new standard, but rather the new standard would play a part and incentivise manufacturers to change formulations.

## 1.1 Sodium reduction in Australia and New Zealand

We illustrated the potential health gains that may be possible based on pragmatic estimates in the reduction of sodium intake via processed foods in Australia and New Zealand. The estimates reflect the assumption that intake of sodium from processed food will reduce by 15% (low scenario) and 25% (high scenario) by 2018. For clarity and illustrative purposes, we have focussed our findings on the reduction in myocardial infarction (MI) and stroke, and then extended the analysis to included Disability Adjusted Life Years (DALYs).

Based on current incidence of MI and stroke, we extrapolated an expected incidence in ten years, whilst adjusting for changes in the population demographics, “ageing population”. We linked sodium consumption under the two scenarios with reduced incidence and mortality using large population trial data. Finally, we estimated the DALY improvement associated with reduced incidences in MI and stroke.

Without reduced sodium consumption, the incidence of both MI and stroke are expected to increase due to the ageing population (certis paribus). Reduced sodium consumption, modelled in this study, counteracts much of the expected increase in incidence associated with population ageing. As a proportion of total DALYs lost, these figures represent up to 10% of the total burden of disease from stroke, and 12% of the total burden of disease from MI (both under the high sodium reduction target).

**Table 1: Summary of DALYs averted through sodium reduction from salt in processed food (2018 only)**

| | Scenario | Australia | New Zealand | Total |
|---|---|---|---|---|
| **MI** | Low | 11,013 | 3,296 | **14,309** |
| | High | 18,278 | 5,509 | **23,787** |
| **Stroke** | Low | 9,773 | 2,782 | **12,555** |
| | High | 14,661 | 4,647 | **19,308** |
| **Total** | Low | **20,786** | **6,078** | **26,864** |
| | High | **32,939** | **10,156** | **43,095** |

## 1.2 Saturated fat reduction in Australia and New Zealand

This section describes a modelling exercise in which the total reduction in saturated fat intake is reduced by 15% (low scenario) and 25% (high scenario) by 2018. For clarity and illustrative purposes, we have focussed our findings on the reduction in myocardial infarction (MI). To avoid missing other benefits, we extended the analysis to included DALYs.

Based on the current incidence of MI, we extrapolated an expected incidence in ten years, whilst adjusting for the "ageing population" in both countries. We link saturated fat consumption under the low and high scenarios with reduced MI incidence and mortality using large population trial data. Finally, we estimated the DALY improvement associated with reduced incidences in MI.

It should be noted that we only identified tentative evidence regarding the scale of the risk reduction in a female population. Therefore, we looked only at this effect, aware it probably will underestimate the true benefit.

Without reduced saturated fat consumption, the incidence of MI is expected to increase due to the ageing population (ceteris paribus). Relative to this base case, both low and high reduction scenarios lead to a substantial improvement in the burden of disease. These estimates are likely to under represent the true effect since we have limited our analysis to MI and women.

**Table 2: Summary of DALYs averted through saturated fat reduction**

| | Scenario | Australia | New Zealand | Total |
|---|---|---|---|---|
| **MI** | Low | 9,811 | 2,689 | **12,500** |
| | High | 16,409 | 4,429 | **20,838** |

# 2 Introduction

## 2.1 Background and purpose for this report

As part of Proposal P293, Food Standards Australia New Zealand (FSANZ) commissioned the Centre for Health Economics Research and Evaluation (CHERE), University of Technology, Sydney, to model the potential health benefits that might be possible for the populations of Australia and New Zealand, ten years after the introduction of the proposed standard for nutrition, health and related claims (Proposal P293). The report will model the health impact of reductions in the intakes of two nutrients; 1) sodium from processed foods, and 2) saturated fats.

> It is important to note that the health benefits provided in this report are purely hypothetical, and based upon high and low case scenarios provided by FSANZ. The actual contribution to these scenarios from food manufacturers specifically reacting to the new health claims standard is unknown. Therefore any health benefits proposed by this report are not posed as being due to the new standard, but rather the new standard would play a part and incentivise manufacturers to change formulations.

## 2.2 Nutrition, Health and Related Claims Standard

FSANZ issued a Draft Assessment Report in November 2005 setting out a proposed approach to the regulation of Nutrition, Health and Related claims together with the proposed new Standard 1.2.7 – Nutrition, Health and Related claims (Food Standards Australia New Zealand, 2005). The proposed draft Standard set out the criteria and conditions for making content claims, health claims and related claims, and included composition of foods able to make claims, wording conditions and exemptions from the general approach, and incorporated substantiation requirements. Following consultation, FSANZ issued a Preliminary Final Assessment Report in April 2007 which recommended some elements of the initial report be modified (Food Standards Australia New Zealand, 2007a).

In summary, the proposed Nutrition, Health and Related Claims Standard comprises two types or claims; nutrition content claims and health claims. A nutrition content claim is a claim about the presence or absence of a property of a food. A health claim is a claim that refers to a relationship between a food or a property of a food and a health effect. There are two levels of health claims; general level health claims and high level health claims. High level health claims differ from general level health claims in that they make reference to a serious disease or a biomarker of a serious disease. Consequently high level health claims require pre-market assessment and approval by FSANZ.

For more details regarding Proposal 293, please refer to the two FSANZ assessment reports and the consultation paper (Food Standards Australia New Zealand, 2007a, Food Standards Australia New Zealand, 2007b, Food Standards Australia New Zealand, 2005).

## 2.3 Scope of the analysis

The aim of this report is to:

a) undertake a scenario modelling exercise that illustrates the potential health benefits that might be possible for the populations of Australia and New Zealand, ten years after the introduction of the proposed standard for nutrition, health and related claims;

b) model the potential reduction in the burden of disease, expressed in measures such as DALYs where possible;

c) model separately the impacts in Australia and New Zealand;

d) focus on reductions in the intakes of two nutrients: sodium and saturated fats;

   I. for sodium: focus on the potential reduction in the sodium content of processed foods, including high case and low case scenarios

   II. for saturated fats: provide an indicative assessment of how the proposed food standard could similarly generate health benefits due to reductions in intakes in saturated fats

e) prepare a narrative to accompany the modelling, indicating that the modelling is purely illustrative of the health benefits that could be realised due to the incentives provided by the proposed food standard. Noting that:

   I. Sodium and saturated fats were selected because food manufacturers could focus on reducing the quantity of these components in a range of products that are not currently eligible under the Nutrition Profiling Scoring Criteria. Hence enabling the manufacturers to make general level health claims about a range of components (Presuming the qualifying and other criteria are met).

   II. Pre-approved high level health claims relating to reductions in sodium, and saturated fat intakes have been substantiated as part of the development of the health claims standard. Manufacturers would be able to use these claims as soon as the Standard is gazetted, on products which meet the criteria.

   III. The ten year period was chosen for the scenario because it would allow industry time to fully consider and respond to the possibilities and opportunities under the proposed food standard.

   IV. It is recognised that the proposed standard plays a role in realising these health outcomes, but would not be expected to directly deliver these health outcomes on its own.

# 3 The potential impact of dietary sodium reduction following the introduction of the health claims standard

## 3.1 Dietary sodium

Sodium in the diet is derived from four sources: sodium naturally present in food; salt which is added to food during processing; other non-salt sodium containing compounds which are added to food during processing[1]; and salt which may be added to food during cooking and eating (discretionary salt). The major dietary source of sodium is salt (i.e. sodium chloride). It is estimated that the vast majority (90%) of dietary sodium consumed in countries like Australia and New Zealand comes from salt – either added during processing or as discretionary salt. (National Health and Medical Research Council and the New Zealand Ministry of Health, 2006) This report focuses on sodium derived from salt added to food during processing because this is the sodium source that is most likely to be influenced by the health claims standard. Naturally occurring sodium, discretionary salt and non-salt sodium containing additives will not be discussed in this report, because their consumption or levels in food is unlikely to be affected by the proposed Standard.

The National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (NZMoH) recommend an Upper Level (UL) of 2300 mg of sodium (100mmol per day) for the adult population[2] but have also Suggested Dietary Target of 1600 mg for adults (National Health and Medical Research Council and the New Zealand Ministry of Health, 2006).

## 3.2 High blood pressure

Blood pressure is regulated by the renin-angiotensin-aldosterone (RAA) system. In summary, sodium causes water to be retained, thus increasing blood volume and blood pressure (Altun and Arici, 2006).

The Heart Foundation of Australia defines high blood pressure (hypertension) as: systolic blood pressure (SBP) greater than or equal to 140 mm Hg; or a diastolic blood pressure (DBP) greater than or equal to 90 mm Hg; or receiving medication for high blood pressure, Table 3 (National Heart Foundation, 2004).

[1] For example sodium bicarbonate is used as a raising agent in cakes and biscuits.

[2] The following are the conversion factors for the units used to express the sodium content of food:
1mmol = 23 milligrams
1 gram = 43mmol
One gram of sodium chloride (NaCl) contains 17 mmol, or 391 milligrams, of sodium.

**Table 3 Definitions and classification of blood pressure levels (mmHg) according to the 2004 Heart Foundation of Australia guidelines. (National Heart Foundation, 2004)**

| Category | Systolic | Diastolic | Action |
|---|---|---|---|
| Normal | <120 | <80 | Recheck in 2 years |
| High-normal | 120-139 | 80-89 | Recheck in 1 year – lifestyle advice |
| Grade 1 (mild) | 140-159 | 90-99 | Confirm within 2 months – lifestyle advice* |
| Grade 2 (moderate) | 160-179 | 100-109 | Evaluate or refer within 1 month lifestyle advice* |
| Grade 3 (severe) | ≥ 180 | ≥ 110 | Further evaluate and refer within 1 week* (or immediately depending on clinical situation) |

*Early initiation of drug therapy may be indicated in patients with high risk

High blood pressure can be classified as primary or secondary. Patients with primary (sometimes known as essential) hypertension have no definable cause. Lifestyle factors such as obesity, excessive alcohol consumption, physical inactivity and diet, can contribute to primary hypertension (Australian Institute of Health and Welfare, 2006a). Secondary hypertension, which accounts for approximately 10% of all cases, is caused by an underlying condition such as a hormonal disorders or renal disease.

The 1999/2000 Australian Diabetes, Obesity and Lifestyle study (AusDiab), which is a cross sectional survey, highlighted the prevalence of hypertension, (defined as BP ≥ 140/90 mm Hg or self report used of antihypertensive medication) in the Australian population, to be 28.6% (95% CI, 25.0–32.3) (Briganti, et al., 2003). Five year follow up data demonstrated that, of those classified with hypertension at baseline, 18% were classified as having normal blood pressure at follow up. For people aged 25-34 years the risk of developing high blood pressure was 1.0%, which increased to 8.4% per year for people aged 65-74 years. Interestingly 13.9% of the individuals considered ‘normal’ at baseline had subsequently developed hypertension (Barr, et al., 2006).

Since 1980 the prevalence of high blood pressure in Australia has decreased. The proportion of females aged 25-64 years with hypertension more than halved (32% in 1980 to 16% in 1999-2000). In males, the proportion went from 47% to 21%, during the same period (Australian Institute of Health and Welfare, 2006a). In spite of these trends, high blood pressure is one of the largest contributors to poor health outcomes from cardiovascular disease in Australia. It causes the third greatest burden of disease, second only to physical inactivity and tobacco smoking (Begg, et al., 2007). It is also the most frequently managed problem in general practice, accounting for 9.4% of consultations in Australia (Britt, et al., 2007).

Self reported data collected from the National Aboriginal and Torres Strait Islander 2004-05 Health Survey indicated that 15% of Aboriginal and Torres Strait Islanders reported high blood pressure compared with 10% of non-Indigenous Australians. (Australian Institute of Health and Welfare, 2006a) However, it is worth noting that self reported health surveys often give lower prevalence of hypertension than comparable studies that measure blood pressure.

## 3.3 Cardiovascular disease

Cardiovascular disease covers all diseases and conditions of the heart and blood vessels. The major contributors to the cardiovascular disease burden in Australia include: coronary heart disease (CHD), stroke, heart failure and peripheral vascular disease. (Australian Institute of Health and Welfare, 2006a) Ischaemic heart disease is the most common form of heart disease. There are two major clinical conditions: acute myocardial infarction (AMI) and angina.

The most important manifestation of cerebrovascular disease is stroke. A stroke occurs when a blood vessel to the brain occludes (ischaemic stroke – 85% of cases) or haemorrhages (haemorrhagic stroke – 15% of cases). This may result in localised brain cell death due to the lack of blood, leading to a loss of brain function, including; movement and cognitive impairment, and may lead to death (Australian Institute of Health and Welfare, 2006a).

Ischaemic heart diseases and cerebrovascular diseases are leading causes of mortality in Australia. Despite declines in mortality rates during the last 30 years, cardiovascular disease accounts for 47,637 or 36% of all deaths (2004). Ischaemic heart disease accounted for 19% of all deaths, and cerebrovascular diseases a further 9% (Australian Bureau of Statistics, 2007a).

It is important to note that mortality related to cardiovascular disease is higher among people living in rural areas, socioeconomically disadvantage groups and Indigenous Australians (Bennett, 1996). In 2002, it was reported that adults from the most disadvantaged areas of Australia have between 1.6 and 1.9 higher death rates from cardiovascular disease, CHD and stroke compared to those from the least disadvantaged areas (Australian Institute of Health and Welfare, 2006b).

### 3.3.1 Cardiovascular disease and health care utilisation

In 2003-04 there were 448,859 hospital separations including a principal diagnosis of cardiovascular disease. These separations increase by age, accounting for 77% of separations in those aged 55 or over (Australian Institute of Health and Welfare, 2006a).

Total health expenditure attributable to cardiovascular disease in 2000-01 in Australia was $5.5b, or 11% of total health care expenditure (Australian Bureau of Statistics, 2007b). In 2005, health care costs on medicines commonly used to prevent or treat cardiovascular disease amounted to $2 billion (Senes, et al., 2007).

Stroke was the principal diagnosis for 68,866 hospital separations in 2002-03, its sequelae taking up 1,073,645 patient days (Australian Institute of Health and Welfare, 2006c). The ageing of the Australian population is expected to increase the number of strokes in the future (Australian Institute of Health and Welfare, 2006c). The average cost per case of stroke, treated during the first 12 months, was A$44,428 (1997 Australian dollars) (Dewey, et al., 2001).

Health expenditure for cerebrovascular disease in 2000-01 was $896 million, which is 1.8% of total health system expenditure (Australian Institute of Health and Welfare, 2006c).

After heart disease and cancer, stroke is the third major cause of death in women in New Zealand. Even though the proportion of women who have experienced a stroke has not changed markedly over 1981-2003 there are ethnic differences. Stroke rates for women categorised as European have decreased (Rate ratio 0.84, 95% CI 0.73-0.96) whilst rates in Pacific women have increased (Rate ratio 2.71 95% CI 1.00-7.29).

### 3.3.2 High blood pressure and cardiovascular risk

Epidemiologic studies have shown a direct association between blood pressure and the risk of cardiovascular disease (Eastern Stroke and Coronary Heart Disease Collaborative Research Group, 1998, Kannel, 1996, Kshirsagar, et al., 2006, MacMahon, et al., 1990, Neaton and Wentworth, 1992, Sagie, et al., 1993, Selmer, et al., 2000, van den Hoogen, et al., 2000, Vasan, et al., 2001). The association between cardiovascular risk and blood pressure highlighted in these studies is across a broad spectrum of blood pressures. Conen *et al*. noted that women with "high" normal blood pressure have a higher cardiovascular risk than women with normal blood pressure (Conen, et al., 2007). Krum *et al*. reported that in Australia about two-thirds of chronic heart failure (CHF) cases are caused by hypertension (Krum, et al., 2006).

The relationship between blood pressure and risk of cardiovascular disease has been described as "continuous, consistent and independent of other risk factors". The higher the blood pressure, the greater the chance of myocardial infarction, stroke, heart failure and kidney disease (Chobanian, et al., 2007). A meta-analysis of 61 observational studies reported that for individuals aged 60-69, each difference of 20 mm Hg in SBP or 10 mm Hg in DBP doubles the risk of cardiovascular disease (Lewington, et al., 2002).

In addition, factors such as: personal history, age, gender, family history, smoking status, cholesterol, diabetes, weight, alcohol intake, physical activity, ethnicity influence the absolute cardiovascular risk and prognosis (Heart Foundation, 2007).

## 3.4 Sodium and blood pressure

Trial and meta-analysis data demonstrate that reduction in the consumption of dietary sodium lowers blood pressure in hypertensive patients (Alam and Johnson AG, 1999, Cutler, et al., 1997, Graudal, et al., 1998, He and MacGregor, 2002, Midgley, et al., 1996).

A FSANZ commissioned report, investigating the relationship between dietary sodium intake (alone or in combination with potassium) and the risk of hypertension in adults, highlighted the relationship between sodium consumption and blood pressure. This review critically assessed the Health Canada scientific summary (on sodium and hypertension) and supplemented the evidence published between 2000 and March 2005 (Samman, 2006). The report concluded that there is strong evidence between high sodium intake and increased risk of cardiovascular disease.

The United Kingdom Scientific Advisory Committee on Nutrition (SACN) *"Salt and Health"* report and the World Health Organisation/Food and Agriculture organisation *"Diet, Nutrition and The Prevention of Chronic Disease"* report; concluded that there is compelling evidence that high sodium intake increased

the risk of developing cardiovascular disease (2003, World Health Organisation, 2002).

The effect of sodium restriction on blood pressure is greater in older and middle aged people, as well as individuals with diabetes or chronic kidney disease (Mancia, et al., 2007). There is a heterogeneous response of BP to dietary salt intake. Individuals, both hypertensive and normotensive, vary in their blood pressure responses to changes in dietary salt intake (Altun and Arici, 2006).

Long term interventions promoting low sodium intake have shown difficulty in maintaining significant sodium restrictions (Altun and Arici, 2006). Routine dietary advice in primary care may not be effective in achieving a reduction in sodium intake (Little, et al., 2004). Some commentators have noted that a better way of reducing sodium intake could be better achieved by a general reduction in the content of sodium in manufactured food products (Altun and Arici, 2006, Hooper, et al., 2002, Melander, et al., 2007, Sacks, et al., 2001).

Despite the wealth of evidence, large randomised controlled trials on the effects of long term sodium reduction on cardiovascular disease are scarce. Cook *et al*. conducted a follow up study of individuals who took part in two large randomized controlled trials, the Trial of Hypertension prevention (TOHP) I and II (study results and critical evaluation of these have been presented elsewhere see footnote)[3]. Once the trials were completed no further advice was given to participants. A 10-15 year post trial follow up showed that individuals who were initially allocated to the reduced sodium group had a 30% reduction risk of developing cardiovascular disease (Cook, et al., 2007). However the main limitation of the study is the assumption that people continue having a low sodium intake. It is unknown if trial participants were able to achieve and maintain this dietary change for 10 years.

[3] For study details see (Samman, 2006)

**Table 4. Summary of key studies. Detailed description as well as limitations of these studies is provided elsewhere. (Samman, 2006)**

| Author | Studies | Summary of evidence |
|---|---|---|
| (Intersalt Cooperative Research Group, 1988) | Epidemiological - INTERSALT | Significant positive association between 24 h urinary sodium and BP. Rise in BP related with age. |
| (Law, et al., 1991) | Meta-analysis | A mean reduction in sodium intake of 50 mmol of sodium (1150 mg) per day predicted fall of SBP by an average of 5 mm Hg (in normotensives). |
| (Midgley, et al., 1996) | Meta-analysis | Decreased in BP for a 100 mmol of sodium (2300 mg)/day reduction in daily sodium excretion was :<br>Hypertensive 3.7 mm Hg (2.35-5.05) for systolic and 0.9 (-0.13-1.85) for diastolic<br>Normotensive 1.0 mm Hg (0.51-1.56) for systolic and 0.9 (-0.32-0.51) for diastolic |
| (Cutler, et al., 1997) | Meta-analysis | The effects of reducing sodium intake on mean blood pressure effects were all substantial and significant and larger for hypertensive than normotensive subjects. |
| (Sacks, et al., 2001) | Randomised control Trial - Dietary Approach to Stop Hypertension (DASH) | The DASH Diet (a diet rich in fruits, vegetables and low-fat dairy foods and with reduced saturated and total fat) with low sodium level (100 mmol sodium (2300 mg)/day) decreased mean systolic BP by:<br>Hypertensive 11.5 mm Hg<br>Normotensive 7.1 mm Hg |
| (Hooper, et al., 2007, Hooper, et al., 2002) | Meta-analysis/ Systematic review | Small but significant fall in BP. Lower sodium diets seem to allow people with elevated blood pressure to reduce medication. |
| (He and MacGregor, 2004, He and MacGregor, 2002) | Meta-analysis/ Systematic review | Hypertensive Median reduction in sodium excretion of 78 mmol sodium (1800 mg)/day mean reduction in BP 4.96/2.73 ± 0.40/0.24 mm Hg<br>Normotensive Median reduction in sodium excretion of 74 mmol sodium (1700 mg)/day mean reduction in BP 2.03/0.97 ± 0.27/0.21 mm Hg<br>Hypertensive 100 mmol of sodium (2300 mg)/day predicted a fall of BP of 7.11/3.88 mm Hg<br>Normotensive 100 mmol of sodium (2300 mg)/day predicted a fall of BP of 3.57/1.66 mm Hg |
| (Jurgens and Graudal, 2004) | Systematic review | Short term (4-52 weeks) sodium reduction in Caucasians with high blood pressure decreased SBP by 4 mm Hg |
| (Dickinson, et al., 2006) | Systematic review | Reducing sodium intake by 80-100 mmol sodium (1840-2300 mg)/day from 180 mmol sodium (4140 mg)/day reduced BP by an average of 4-6 mm Hg (Although with a large patient variability) |

# 4 The evaluation of sodium reduction in Australia and New Zealand

## 4.1 Summary

In this section we illustrate the potential health gains that may be possible following implementation of the health claims standard. The gains are based on pragmatic estimates in the reduction of sodium chloride intake via processed foods in Australia and New Zealand. This section assumes that the sodium reduction in food will be due to reduction of salt (sodium chloride) rather than to the reduction of non-salt sodium-containing compounds. The estimates reflect the assumption that intake of sodium from processed food will reduce by 15% (low scenario) and 25% (high scenario) by 2018. This section describes a modelling exercise in which the total reduction in sodium intake meets these scenarios. For clarity and illustrative purposes, we have focussed our findings on the reduction in myocardial infarction (MI) and stroke. Realising that these are primary endpoints and using these endpoint solely would miss important benefits associated with sodium reduction, we have extended the analysis to included Disability Adjusted Life Years (DALYs).

Based on current incidence rates, we extrapolated an expected incidence in ten years, whilst adjusting for changes in the population demographics "ageing population" in both countries. We link sodium consumption under the two scenarios with reduced incidence and mortality using large population trial data. Finally, we estimated the DALY improvement associated with reduced incidences in MI and stroke.

Without reduced sodium consumption, the incidence of both MI and stroke are expected to increase due to the ageing population (certis paribus). Reduced sodium consumption, modelled in this study, counteracts much of the expected increase in incidence associated with population ageing. This is particularly the case in New Zealand where the baseline consumption of sodium through processed food is relatively high (and therefore the absolute reduction in consumption associated with a percentage reduction is higher). The results are summarised in Table 5.

As a proportion of total DALYs lost, these figures represent up to 10% of the total burden of disease from stroke, and 12% of the total burden of disease from MI (both under the high sodium reduction target).

**Table 5: Summary of DALYs averted through sodium reduction from salt in processed food (2018 only)**

| | Scenario | Australia | New Zealand | Total |
|---|---|---|---|---|
| **MI** | Low | 11,013 | 3,296 | **14,309** |
| | High | 18,278 | 5,509 | **23,787** |
| **Stroke** | Low | 9,773 | 2,782 | **12,555** |
| | High | 14,661 | 4,647 | **19,308** |
| **Total** | Low | **20,786** | **6,078** | **26,864** |
| | High | **32,939** | **10,156** | **43,095** |

## 4.2 Introduction

In this section we identify the hypothetical effect of changing sodium intake over a ten year period (2008-2018) in Australia and New Zealand. The estimated outcomes are: reductions in systolic and diastolic blood pressure; myocardial infarction (MI) events and deaths; stroke events and deaths; and lost DALYs averted through reductions in these episodes. The argument that is presented follows this basic structure.

- Step 1: Identify current consumption of sodium. Focussing on the proportion obtained from processed food (rather than discretionary use).
- Step 2: Estimate realistic sodium reduction scenarios in processed food, and its effects on total sodium consumption across genders and age groups
- Step 3: Collect information regarding the relationship between consumption, blood pressure, and clinical events
- Step 4: Describe the current incidence of MI and stroke (fatal and non-fatal) in Australia and New Zealand
- Step 5: Consider the effect of an ageing population on the expected number of events in ten years
- Step 6: Estimate the reduction of events in the 2018 population based on the changing levels of sodium consumption from processed sources
- Step 7: Illustrate the DALY impact of reducing morbidity and mortality
- Step 8: Illustrate limitations of the analysis

## 4.3 Step 1: Identify current consumption of sodium

The recent estimates of salt consumption for Australia and New Zealand were provided by FSANZ. These data also show the amount of sodium (and salt) derived from processed food (Table 6). Due to lack of availability of data, intake for children in New Zealand was extrapolated from Australian data, adjusting for the higher level of sodium consumption.

**Table 6: Sodium (and salt) consumption (adapted from FSANZ documentation)**

| Age | Mean salt (sodium) from processed food & total (salt: g/day, sodium: mg/day) | | | |
|---|---|---|---|---|
| | Australia | | New Zealand | |
| | processed | total | processed | total |
| 2-3 | 3.50 (1369) | 3.78 (1478) | 3.93 (1537)# | 4.54 (1775)# |
| 4-8 | 4.04 (1580) | 4.46 (1744) | 4.53 (1771)# | 5.35 (2092)# |
| 9-13 | 4.74 (1853) | 5.30 (2072) | 5.32 (2080)# | 6.36 (2487)# |
| 14-18 | 5.64 (2205) | 6.40 (2502) | 6.33 (2475)* | 7.68 (3003)* |
| 19-29 | 5.62 (2197) | 6.33 (2475) | 5.95 (2326) | 7.23 (2827) |
| 30-49 | 5.00 (1955) | 5.64 (2205) | 5.78 (2260) | 7.02 (2745) |
| 50-69 | 4.52 (1767) | 5.19 (2029) | 5.36 (2096) | 6.51 (2545) |
| 70+ | 4.16 (1627) | 4.83 (1889) | 4.82 (1885) | 5.86 (2291) |

Processed: equals the amount of sodium derived from processed foods, Total: equals the amount of sodium derived from all salt (processed and discretionary)
* Figure based on 15-18 year olds
# Due to lack of availability of data, intake for children in New Zealand was extrapolated from Australian data accounting for the relatively higher overall level of consumption.

## 4.4 Step 2: Estimate realistic sodium reduction scenarios in processed food, and its effects on total sodium consumption across genders and age groups

The United Kingdom has proposed a target reduction in mean salt consumption to 6g/day (2300mg sodium) which is approximately a 33% reduction from the intake previously estimated to be 9.5g salt equivalents per day. The corresponding study estimates a 13% reduction in stroke and 10% reduction in coronary heart disease (Jebb, et al.). However, the 6g salt target proposed in the UK appears to refer to the total quantity of sodium in the diet from all sources, expressed as “salt equivalents”. Therefore it would be incorrect to infer that Table 6 indicates that the target has been largely met in Australia. This is because Table 6 excludes naturally occurring sodium and non-salt sodium-containing compounds that are added by manufacturers.

As noted above, the NHMRC and NZMoH (2006) have set an Upper Level of 2300mg sodium and a ‘suggested dietary target’ of 1600mg sodium for adults. The adequate intake for adults is much lower: 460-920mg sodium. As 6g salt contains 2300mg sodium, it is clear that sodium derived from the salt content of the diet is near the Upper Level for total sodium.

Reducing the salt content of food by 25% would lower the sodium intake from salt to approximately the ‘suggested dietary target’. The total sodium intake would be higher owing to the other sources of sodium in the diet. Therefore, FSANZ provided high and low scenarios for the predicted reduction in sodium

consumption in Australia and New Zealand following the introduction of the Health Claims standard, Table 5. In that the high and low scenarios refer to salt from manufactured foods rather than total "salt equivalents", the high scenario is more conservative than the UK target and so we believe the following scenarios are plausible. The purpose of these targets is to illustrate the effect of reducing sodium on the health and life expectancy of the population.

The validity of these scenarios is outside the scope of this report. Achieving any reduction in the sodium content of processed foods is based upon the value that manufacturers place on meeting the new health claims standard. However, it is believed that 15% reduction is a realistic scenario, whilst the 25% reduction is a best case scenario. Based on the UK data, we believe the following scenarios are achievable.

**Table 7: Scenarios**

| | % reduction in intake of sodium from salt from processed food in Australia and New Zealand |
|---|---|
| Low scenario | 15% |
| High scenario | 25% |

This report considers only sodium from salt added to processed food. We assume the intake from other sources (discretionary salt and naturally occurring sodium, and non-salt sodium-containing compounds added in processing) remains constant.

**Table 8: Reduction in sodium intake from processed foods required to meet the low and high scenarios**

| | **Sodium (salt) intake reduction (mg/day) under 2 scenarios** | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **Australia** | | | | **New Zealand** | | | |
| Age | low scenario | | High scenario | | Low scenario | | High scenario | |
| | Na (mg) | Salt (g) | Na (mg) | Salt (g) | Na (mg) | Salt (g) | Na (mg) | Salt (g) |
| 19-29 | 328 | (0.84) | 551 | (1.41) | 348 | (0.89) | 583 | (1.49) |
| 30-49 | 293 | (0.75) | 489 | (1.25) | 340 | (0.87) | 567 | (1.45) |
| 50-69 | 266 | (0.68) | 442 | (1.13) | 313 | (0.80) | 524 | (1.34) |
| 70+ | 242 | (0.62) | 407 | (1.04) | 282 | (0.72) | 473 | (1.21) |

## 4.5 Step 3: Collect information regarding the relationship between intake, blood pressure, and events

### 4.5.1 Reduction in blood pressure associated with sodium intake reduction

He and McGregor undertook a meta-analysis on the relationship between sodium consumption and systolic / diastolic blood pressure in normotensive and hypertensive patients. They searched MEDLINE, EMBASE, Cochrane library and CINAHL, identifying 28 studies. The conclusions were; for an intake reduction of 1800mg sodium/day (1700mg sodium/day) for hypertensive (normotensive) patients (He and MacGregor, 2002).

*"The pooled estimates of blood pressure fall were 4.96/2.73 +/- 0.40/0.24mmHg in hypertensives (P<0.001 for both systolic and diastolic) and 2.03/0.97 +/- 0.27/0.21mmHg in normotensives (P<0.001 for both systolic and diastolic)".*

The importance of this finding is illustrated by the recent AusDiab report, which demonstrated that nearly a third of Australians are hypertensive (AusDiab, 2005).

### 4.5.2 Reduction in cardiovascular events associated with reduction in sodium consumption

Despite many studies investigating sodium reduction and blood pressure, there are few studies that look at the effect of sodium reduction and clinical endpoints such as cardiovascular disease or death. Cook and colleagues investigated the relationship between dietary sodium reduction and cardiovascular disease outcomes. Data from two large clinical trials were used (total n=3126), which had a weighted mean reduction in sodium intake of 35.618mmol/24h (equivalent to 820mg sodium/day). Once the results were adjusted for trial, clinic, age, race, sex, baseline sodium excretion and weight, the relative risk of a cardiovascular event over the subsequent ten to fifteen years in the intervention group was 0.70 (0.53 to 0.94)[4]. The relative risk of death (all cause mortality) in the intervention group was 0.80 (0.51 to 1.26, P=0.34) (Cook, et al., 2007).

Since the reduction in sodium consumption reported by Cook *et al*. is greater than the estimates of sodium intake reduction for Australia and New Zealand presented scenarios in Table 8, we adjusted the relative risks. To make this adjustment we assumed a linear relationship between sodium intake reduction and cardiovascular events and death[5]. The relative risks of events and death under each of the scenarios are given in Table 9.

**Table 9: Relative risks of cardiovascular events under reduction scenarios**

| | Relative Risk (95% CI) | | | |
|---|---|---|---|---|
| | Australia* | | New Zealand* | |
| Age | Low scenario | High scenario | Low scenario | High scenario |
| 19-29 | 0.88 (0.81-0.98) | 0.80 (0.68-0.96) | 0.87 (0.80-0.98) | 0.79 (0.67-0.96) |
| 30-49 | 0.89 (0.83-0.98) | 0.82 (0.72-0.96) | 0.88 (0.81-0.98) | 0.79 (0.68-0.96) |
| 50-69 | 0.90 (0.85-0.98) | 0.84 (0.75-0.97) | 0.89 (0.82-0.98) | 0.81 (0.70-0.96) |
| 70+ | 0.91 (0.86-0.98) | 0.85 (0.77-0.97) | 0.90 (0.84-0.98) | 0.83 (0.73-0.97) |

* Relative risk is <1, therefore the likelihood of a cardiovascular event is less under the reduced sodium scenarios.

Applying the results in this way suggests that the most significant proportional reduction in events will be in younger age groups, since the percentage targets lead to a relatively greater reduction in sodium intake (through processed food) in those with higher baseline consumption. However, since events are rare in these age groups, the majority of the cases averted will be in the older age groups.

[4] The relative risk of a cardiovascular event is a ratio of the probability of the event occurring under the low sodium diet compared to the high sodium diet. If the ratio is <1 the event is less likely to occur, if >1 the event is more likely to occur.

[5] The relationship was assumed linear because of the lack of data to support an alternative

The figures for mortality are calculated using the same approach, and are shown below in Table 10.

**Table 10: Relative risk of all cause mortality under reduction scenarios**

| | Australia* | | New Zealand* | |
|---|---|---|---|---|
| Age | Low scenario | High scenario | Low scenario | High scenario |
| 19-29 | 0.92 (0.80-1.00) | 0.87 (0.67-1.00) | 0.92 (0.79-1.00) | 0.86 (0.65-1.00) |
| 30-49 | 0.93 (0.83-1.00) | 0.88 (0.71-1.00) | 0.92 (0.80-1.00) | 0.86 (0.67-1.00) |
| 50-69 | 0.94 (0.84-1.00) | 0.89 (0.74-1.00) | 0.92 (0.81-1.00) | 0.87 (0.69-1.00) |
| 70+ | 0.94 (0.86-1.00) | 0.90 (0.76-1.00) | 0.93 (0.83-1.00) | 0.89 (0.72-1.00) |

* Relative risk is <1, therefore the likelihood of a cardiovascular event is less under the reduced sodium scenarios

## 4.6 Step 4: Describe current incidence of MI and stroke (fatal and total) in Australia and New Zealand

To estimate the cases averted, it is first necessary to identify current levels of fatal and total stroke and MI in Australia and New Zealand. Both Australian and New Zealand data are reported using ICD-10 categories. For stroke, we used I60-I69. The categories chosen to represent MI were I21 and I22. These are 'Acute Myocardial Infarction' and 'Subsequent Myocardial Infarction'. The Ischaemic Heart Disease categories not included are I20 (Angina Pectoris), I23 (Certain current complications following acute myocardial infarction), I24 (Other Acute Ischaemic Heart Disease) and I25 (Chronic Ischaemic Heart Disease). The effect of this exclusion is to underestimate the true effect of sodium reduction on events since high blood pressure is a risk factor for other ischaemic heart disease. We chose this more limited description of MI since it had the strongest and most convincing evidence base.

### 4.6.1 New Zealand

The most contemporary data in New Zealand on causes of death is the 2003 Mortality and Demographic Data Report 2003. This identifies 3,229 deaths from MI (defined as ICD-10 AM codes I21 and I22), divided by age and gender. This is summarised in Table 11. The mortality figures for stroke are shown in Table 13. The figures for stroke separations are given in Table 14.

**Table 11: New Zealand deaths from myocardial infarction (Total population, 2003)**

| Age | Male | Female | Total |
|---|---|---|---|
| 25-29 | 1 | 0 | 1 |
| 30-34 | 3 | 0 | 3 |
| 35-39 | 9 | 0 | 9 |
| 40-44 | 18 | 4 | 22 |
| 45-49 | 39 | 7 | 46 |
| 50-54 | 51 | 15 | 66 |
| 55-59 | 77 | 21 | 98 |
| 60-64 | 91 | 35 | 126 |
| 65-69 | 150 | 52 | 202 |
| 70-74 | 213 | 127 | 340 |
| 75-79 | 306 | 231 | 537 |
| 80-84 | 356 | 315 | 671 |
| 85+ | 380 | 728 | 1,108 |
| Total | **1,694** | **1,535** | **3,229** |

* There were no recorded deaths in these categories for those aged younger than 25 years.

**Table 12: New Zealand myocardial infarction separations (Total population, 2002/3)**

| Age | Male | Female | Total |
|---|---|---|---|
| 25-29 | 10 | 3 | 13 |
| 30-34 | 40 | 9 | 49 |
| 35-39 | 119 | 24 | 143 |
| 40-44 | 333 | 65 | 398 |
| 45-49 | 425 | 104 | 529 |
| 50-54 | 665 | 188 | 853 |
| 55-59 | 795 | 272 | 1,067 |
| 60-64 | 845 | 301 | 1,146 |
| 65-69 | 898 | 430 | 1,328 |
| 70-74 | 926 | 555 | 1,481 |
| 75-79 | 1,039 | 692 | 1,731 |
| 80-84 | 792 | 826 | 1,618 |
| 85+ | 562 | 936 | 1,498 |
| Total | 7,449 | 4,405 | 11,854 |

Source: NZHIS website (combining private and public separations for I21/I22)

**Table 13: New Zealand deaths from stroke (I60-I69) (Total population, 2003)**

| Age | Male | Female | Total |
|---|---|---|---|
| 30-34 | 5 | 2 | 7 |
| 35-39 | 2 | 4 | 6 |
| 40-44 | 12 | 14 | 26 |
| 45-49 | 12 | 14 | 26 |
| 50-54 | 18 | 27 | 45 |
| 55-59 | 26 | 20 | 46 |
| 60-64 | 42 | 30 | 72 |
| 65-69 | 69 | 58 | 127 |
| 70-74 | 103 | 101 | 204 |
| 75-79 | 154 | 203 | 357 |
| 80-84 | 219 | 362 | 581 |
| 85+ | 305 | 884 | 1,189 |
| Total | **969** | **1,723** | **2,692** |

* 6 individuals aged younger than 30 years were identified as dying from cerebrovascular disease. However, since these are not likely to be a result of high blood pressure, they were excluded from this analysis.

**Table 14: Strokes (I60-I69) in New Zealand (Total population, 2002/3)**

| Age | Male | Female | Total |
|---|---|---|---|
| 30-34 | 28 | 46 | 74 |
| 35-39 | 46 | 69 | 115 |
| 40-44 | 90 | 105 | 195 |
| 45-49 | 126 | 115 | 241 |
| 50-54 | 208 | 175 | 383 |
| 55-59 | 284 | 206 | 490 |
| 60-64 | 364 | 292 | 656 |
| 65-69 | 511 | 430 | 941 |
| 70-74 | 684 | 516 | 1,200 |
| 75-79 | 739 | 732 | 1,471 |
| 80-84 | 648 | 871 | 1,519 |
| 85+ | 477 | 1,070 | 1,547 |
| Total | **4,205** | **4,627** | **8,832** |

Source: NZHIS website

### 4.6.2 Australia

**Table 15: Myocardial infarction (I21) mortality in Australia (average over 2003-5)**

| Age | Male | Female | Total |
|---|---|---|---|
| 25-29 | 6 | 2 | 8 |
| 30-34 | 14 | 5 | 18 |
| 35-39 | 31 | 9 | 40 |
| 40-44 | 57 | 16 | 74 |
| 45-49 | 115 | 30 | 145 |
| 50-54 | 167 | 35 | 202 |
| 55-59 | 265 | 64 | 329 |
| 60-64 | 368 | 107 | 475 |
| 65-69 | 504 | 195 | 699 |
| 70-74 | 755 | 375 | 1,130 |
| 75-79 | 1,136 | 752 | 1,888 |
| 80-84 | 1,326 | 1,304 | 2,630 |
| 85+ | 1,789 | 3,258 | 5,047 |
| Total | **6,536** | **6,153** | **12,689** |

Source: ABS Data request
* Total may not sum due to rounding.
^ When the total number of deaths in an age group is small, ABS deem numbers not publishable to avoid release of confidential data. For I21 in this age group, this only occurs for the male and female figures in the 25-29 age group. In this case, we assumed the subdivision between genders occurred in the same ratio as in the 30-34 age group.

**Table 16: Myocardial infarction in Australia (2004/5)**

| Age | Male | Female | Total |
|---|---|---|---|
| 25-29 | 69 | 13 | 82 |
| 30-34 | 258 | 48 | 306 |
| 35-39 | 573 | 157 | 730 |
| 40-44 | 1,267 | 338 | 1,605 |
| 45-49 | 2,184 | 483 | 2,667 |
| 50-54 | 3,081 | 672 | 3,753 |
| 55-59 | 3,863 | 1,030 | 4,893 |
| 60-64 | 3,644 | 1,207 | 4,851 |
| 65-69 | 3,747 | 1,502 | 5,249 |
| 70-74 | 3,648 | 1,982 | 5,630 |
| 75-79 | 3,732 | 2,646 | 6,378 |
| 80-84 | 3,010 | 2,941 | 5,951 |
| 85+ | 2,262 | 3,521 | 5,783 |
| Total | **31,338** | **16,540** | **47,878** |

Source: AIHW Data Cubes (ICD 10 AM Codes I21/I22)

We were unable to identify the source of stroke mortality, stratified by age and gender. The best identified source was the Australian Bureau of Statistics publication 'Causes of Death, Australia, 2005' (Australian Bureau of Statistics, 2007a) which stratified by gender. We extrapolated the numbers in each age group by applying the New Zealand distribution to the Australian totals, as shown in Table 17.

**Table 17: Stroke (I60-I69) mortality in Australia (2005)**

| Age | Male | Female | Total |
|---|---|---|---|
| 30-34 | 24 | 8 | 30 |
| 35-39 | 10 | 16 | 26 |
| 40-44 | 58 | 56 | 111 |
| 45-49 | 58 | 56 | 111 |
| 50-54 | 87 | 107 | 192 |
| 55-59 | 125 | 79 | 197 |
| 60-64 | 202 | 119 | 308 |
| 65-69 | 332 | 230 | 543 |
| 70-74 | 496 | 401 | 872 |
| 75-79 | 742 | 806 | 1,527 |
| 80-84 | 1,055 | 1,438 | 2,485 |
| 85+ | 1,469 | 3,512 | 5,085 |
| Total | **4,668** | **6,845** | **11,513** |

Source: Causes of Death Australia 2005 (Australian Bureau of Statistics, 2007a) (Distribution by age extrapolated from NZ data)
* Total may not sum due to rounding.

**Table 18: Stroke separations in Australia (2004-5)**

| Age | Male | Female | Total |
|---|---|---|---|
| 30-34 | 184 | 159 | 343 |
| 35-39 | 246 | 275 | 521 |
| 40-44 | 426 | 432 | 858 |
| 45-49 | 654 | 580 | 1,234 |
| 50-54 | 953 | 731 | 1,684 |
| 55-59 | 1,523 | 888 | 2,411 |
| 60-64 | 1,870 | 1,057 | 2,927 |
| 65-69 | 2,415 | 1,441 | 3,856 |
| 70-74 | 3,029 | 2,030 | 5,060 |
| 75-79 | 3,814 | 3,056 | 6,871 |
| 80-84 | 3,306 | 3,750 | 7,059 |
| 85+ | 2,527 | 4,697 | 7,224 |
| Total | **20,947** | **19,096** | **40,048** |

Source: AIHW Data Cubes (I60-I69)

## 4.7 Step 5: Consider the effect of an ageing population on the expected number of events in ten years

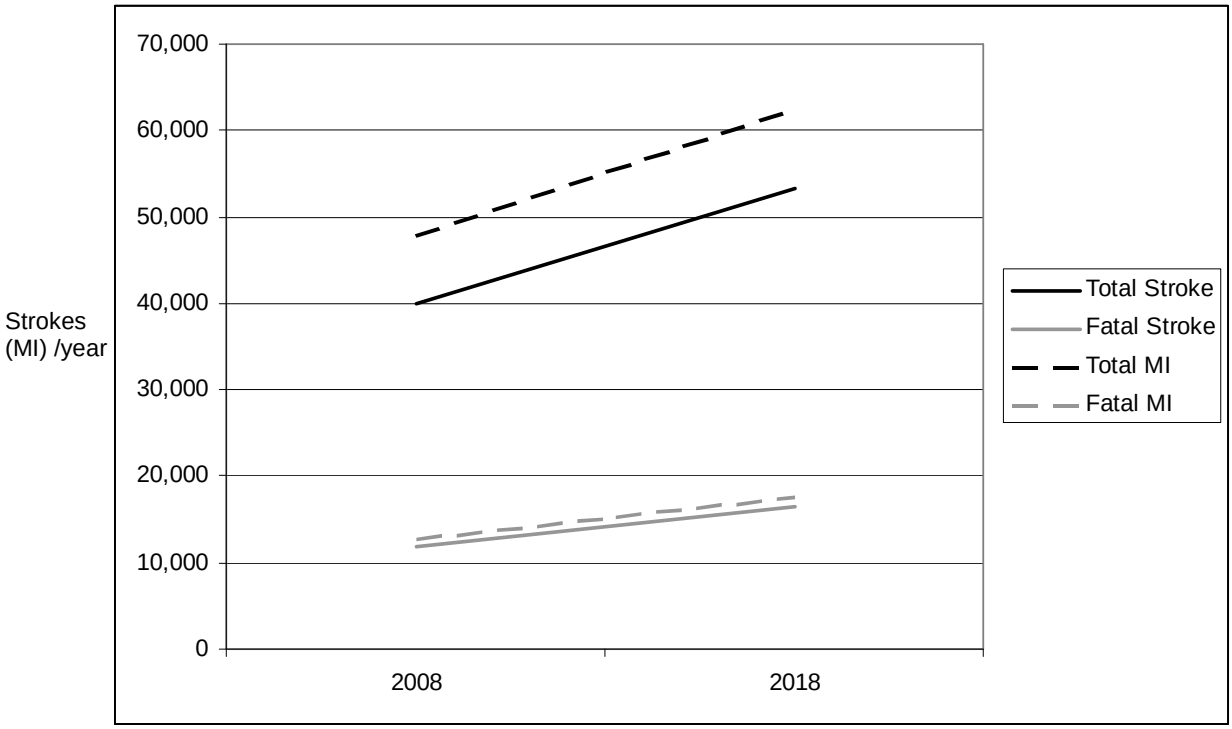
The reduced mortality and morbidity of stroke and MI has been estimated by combining the incidences of stroke and MI data with the relative risk data. However, to extend the data over ten years, we have to consider the 'ageing populations' of Australia and New Zealand. Both the Australian Bureau of Statistics and Statistics New Zealand publish projections taking into account these trends. Using the current population breakdown and the risk of events and death by age, as described in Table 11 through to Table 18, it is possible to estimate the trends into the future as the population ages. If nothing other than the age of the population changes, between 2008 and 2018 (as shown in the Appendix), the level of total (fatal) stroke and MI will change as shown in Table 19.

It should be noted that the incidence and death from MI and stroke is declining steadily due to various factors such as reduction in smoking and more effective medical interventions. For this analysis we ignored this trend and only allowed for the ageing population effect.

**Table 19: MI and Stroke in 2008 and 2018, adjusting for population ageing**

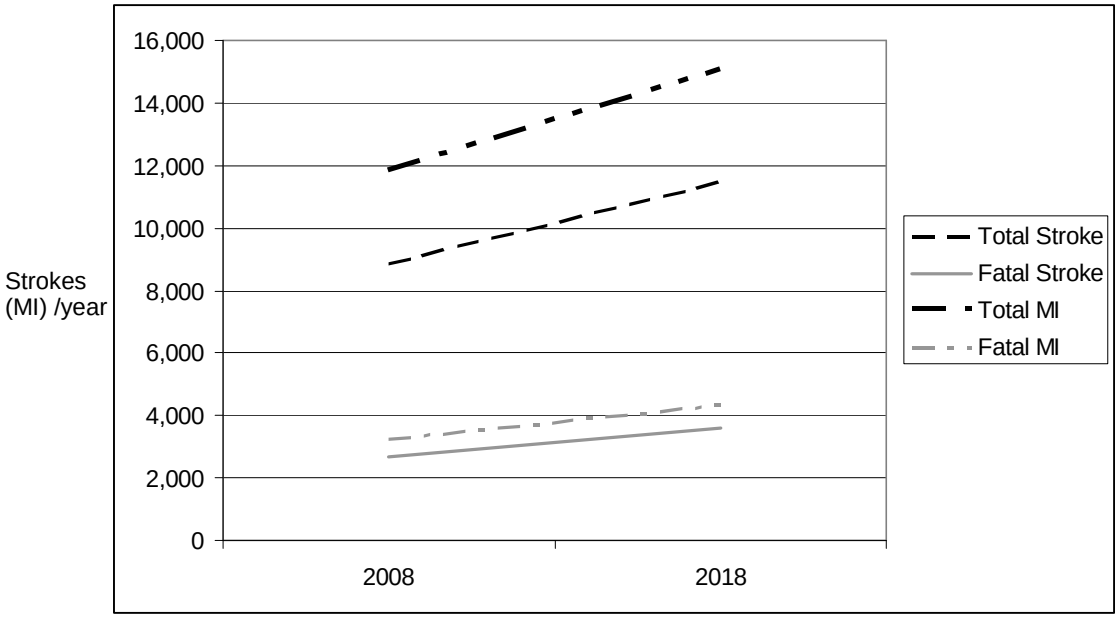
| | | Australia | | New Zealand | |
|---|---|---|---|---|---|
| | | **2008** | **2018** | **2008** | **2018** |
| Stroke | Total | 40,048 | 53,368 | 8,832 | 11,480 |
| | Fatal | 11,895 | 16,525 | 2,686 | 3,625 |
| MI | Total | 47,878 | 62,295 | 11,854 | 15,077 |
| | Fatal | 12,685 | 17,491 | 3,229 | 4,317 |

**Figure 1: Projected stroke / MI in Australia allowing for the ageing population (no change in sodium intake)**




* Ignoring the second trend of reduced smoking, new medical interventions etc.

**Figure 2: Projected stroke / MI in New Zealand allowing for the ageing population (no change in sodium intake)**




* Ignoring the second trend of reduced smoking, new medical interventions etc.

Figure 1 and Figure 2 both illustrate the effect of the ageing population in Australia and New Zealand. They demonstrate that the numbers of MI and stroke events are likely to increase across the population. These findings rely on there being no other significant changes in factors, such as: smoking, fat intake, exercise uptake or pharmaceutical interventions, influencing stroke and MI.

## 4.8 Step 6: Estimate the reduction of events in the 2018 population based on the changing levels of sodium consumption from salt in processed sources

To this point, we have identified the current clinical events and deaths in both countries, identified the link between sodium consumption and reduced events and deaths, and illustrated the reduction in sodium consumption associated with the introduction of the Health Claims standard, as per *a priori* in Table 7. With this information, it is possible to estimate the reduced cases and mortality of stroke and MI under the two scenarios.

We have assumed that the relative risk ratios presented in the Cook et al study, can be applied equally to all subcomponents of cardiovascular disease (i.e. stroke and MI). In the absence of any other information, we believe this is a reasonable assumption.

**Table 20: Mortality effects in Australia through MI and stroke reduction under low and high sodium intake projections in 2018 (both genders)**

| Age | Total deaths per annum (no sodium reduction) | Low scenario # | High scenario # |
|---|---|---|---|
| 25-29 | 9 | 8 (Min:7) | 8 (Min:6) |
| 30-34 | 39 | 37 (Min:33) | 35 (Min:28) |
| 35-39 | 78 | 72 (Min:64) | 68 (Min:55) |
| 40-44 | 142 | 132 (Min:117) | 125 (Min:100) |
| 45-49 | 257 | 239 (Min:212) | 226 (Min:182) |
| 50-54 | 354 | 331 (Min:298) | 316 (Min:261) |
| 55-59 | 594 | 555 (Min:499) | 530 (Min:437) |
| 60-64 | 874 | 817 (Min:735) | 780 (Min:643) |
| 65-69 | 1,676 | 1,567 (Min:1,410) | 1,495 (Min:1,234) |
| 70-74 | 3,015 | 2,837 (Min:2,578) | 2,717 (Min:2,283) |
| 75-79 | 4,649 | 4,374 (Min:3,975) | 4,188 (Min:3,519) |
| 80-84 | 6,277 | 5,907 (Min:5,367) | 5,656 (Min:4,752) |
| 85+ | 16,052 | 15,105 (Min:13,725) | 14,463 (Min:12,151) |
| **Total*** | **34,016** | **31,982 (Min:29,019)** | **30,606 (Min:25,651)** |

* May not sum due to rounding

# Note that, since the upper bound of the relative risk exceeded one, it is considered better to present only the lower bound since reduced sodium intake at the level investigated here is unlikely to lead to increased mortality. Therefore, the worst case scenario would be no reduction in MI or stroke.

**Figure 3: Australian MI and stroke mortality – The effect of the low scenario**

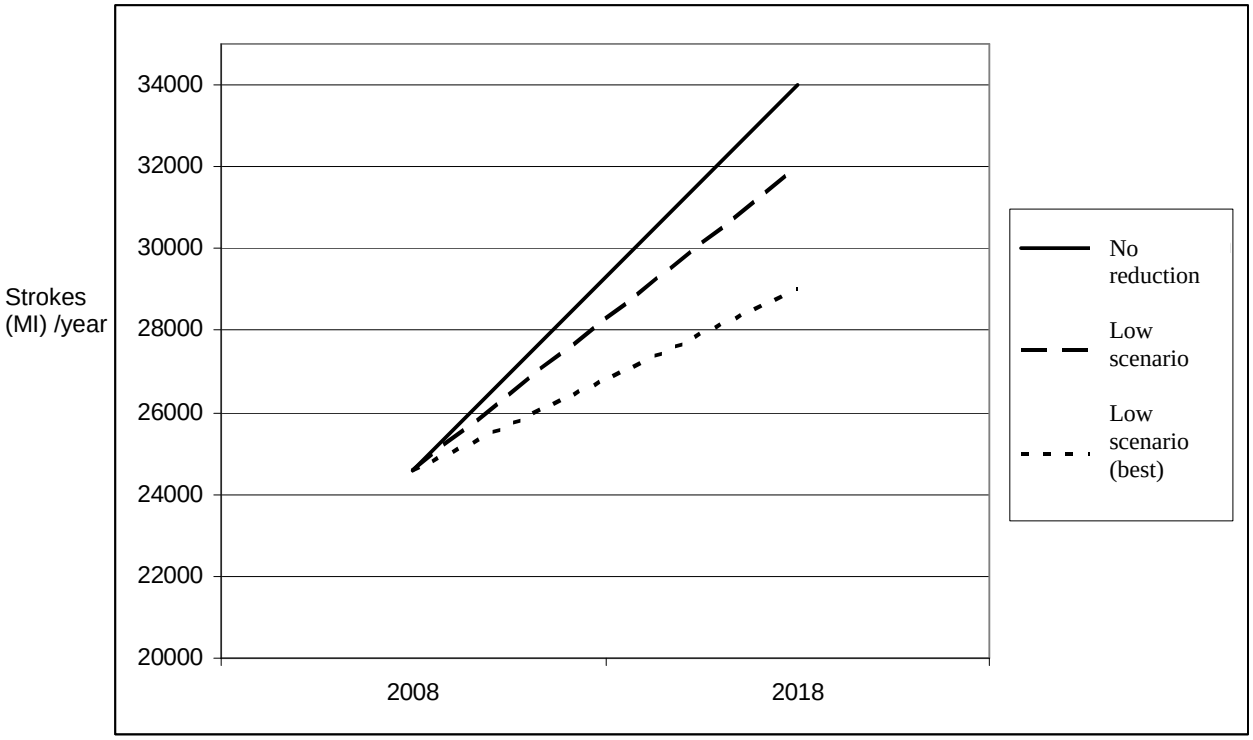


**Figure 4: Australian MI and stroke mortality – The effect of the high scenario**

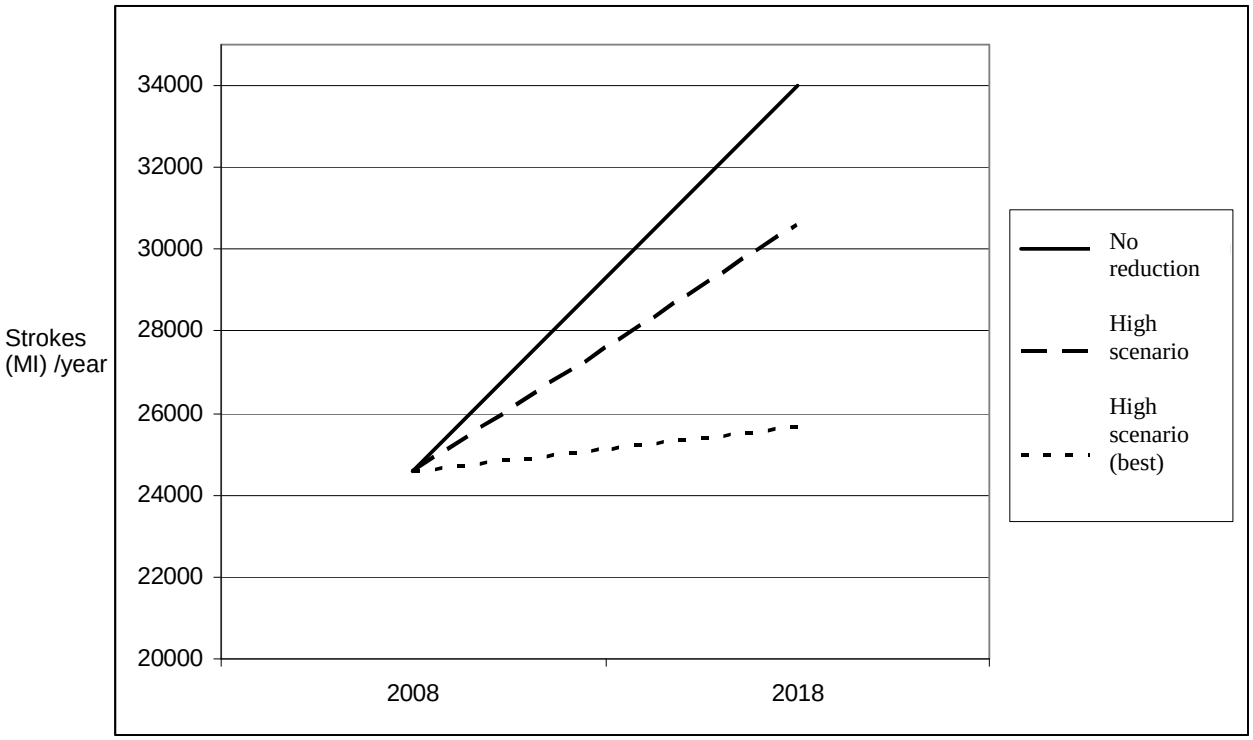



**Table 21: Incidence of stroke under low and high sodium reduction scenarios in Australia in 2018 (both genders)**

| Age | Strokes (baseline) per annum | Low scenario | High scenario |
|---|---|---|---|
| 30-34 | 386 | 345 (321-378) | 317 (278-372) |
| 35-39 | 539 | 481 (448-528) | 442 (388-520) |
| 40-44 | 901 | 805 (750-883) | 740 (649-869) |
| 45-49 | 1,315 | 1,175 (1,094-1,288) | 1,080 (947-1,268) |
| 50-54 | 1,836 | 1,658 (1,557-1,801) | 1,540 (1,371-1,777) |
| 55-59 | 2,858 | 2,581 (2,424-2,804) | 2,398 (2,135-2,767) |
| 60-64 | 3,539 | 3,196 (3,001-3,472) | 2,969 (2,644-3,426) |
| 65-69 | 5,734 | 5,178 (4,863-5,626) | 4,811 (4,284-5,551) |
| 70-74 | 7,918 | 7,213 (6,817-7,775) | 6,738 (6,073-7,680) |
| 75-79 | 9,041 | 8,236 (7,784-8,878) | 7,694 (6,934-8,770) |
| 80-84 | 8,500 | 7,743 (7,318-8,347) | 7,233 (6,519-8,245) |
| 85+ | 10,800 | 9,839 (9,299-10,606) | 9,191 (8,284-10,476) |
| **Total*** | **53,368** | **48,450 (45,677-52,384)** | **45,154 (40,506-51,720)** |

* May not sum due to rounding

**Figure 5: Stroke in Australia – The effect of the low scenario**

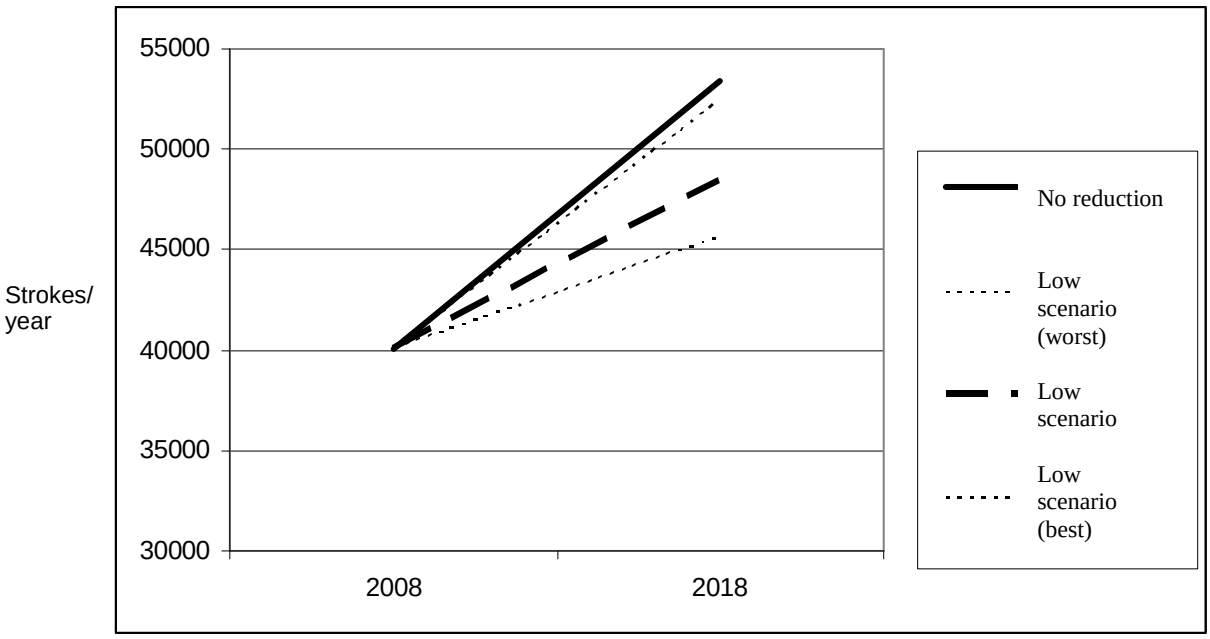



**Figure 6: Stroke in Australia – The effect of the high scenario**

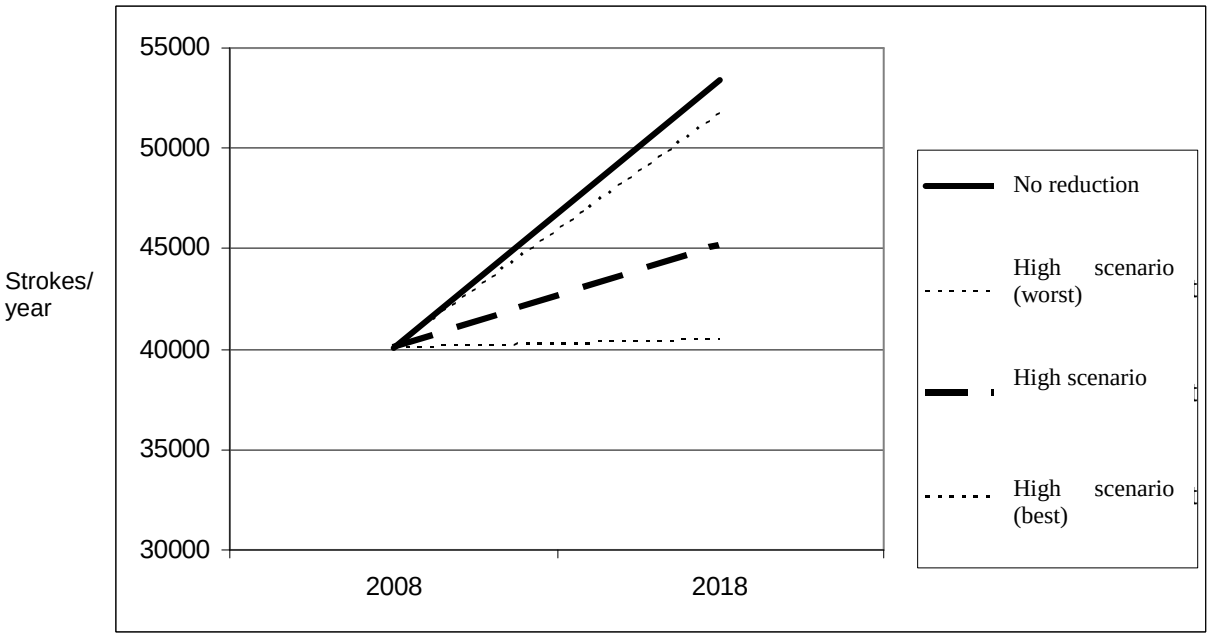


**Table 22: Incidence of MI under low and high sodium reduction scenarios in Australia in 2018 (both genders)**

| Age | MI (baseline) per annum | Low scenario | High scenario |
|---|---|---|---|
| 25-29 | 90 | 79 (73-88) | 72 (62-86) |
| 30-34 | 345 | 308 (287-337) | 283 (248-332) |
| 35-39 | 755 | 674 (628-739) | 620 (544-728) |
| 40-44 | 1,686 | 1,506 (1,403-1,651) | 1,384 (1,214-1,626) |
| 45-49 | 2,843 | 2,539 (2,365-2,783) | 2,334 (2,047-2,740) |
| 50-54 | 4,092 | 3,695 (3,470-4,014) | 3,433 (3,056-3,961) |
| 55-59 | 5,800 | 5,238 (4,919-5,690) | 4,866 (4,333-5,615) |
| 60-64 | 5,866 | 5,297 (4,974-5,754) | 4,921 (4,382-5,678) |
| 65-69 | 7,806 | 7,049 (6,620-7,658) | 6,549 (5,831-7,556) |
| 70-74 | 8,809 | 8.025 (7,585-8,651) | 7,497 (6,757-8,545) |
| 75-79 | 8,392 | 7,645 (7,226-8,241) | 7,142 (6,437-8,140) |
| 80-84 | 7,166 | 6,528 (6,170-7,037) | 6,098 (5,496-6,951) |
| 85+ | 8,646 | 7,876 (7,444-8,490) | 7,358 (6,631-8,386) |
| **Total*** | **62,295** | **56,458 (53,162-61,133)** | **52,557 (47,037-60,344)** |

* May not sum due to rounding

**Figure 7: MI in Australia – The effect of the low scenario**

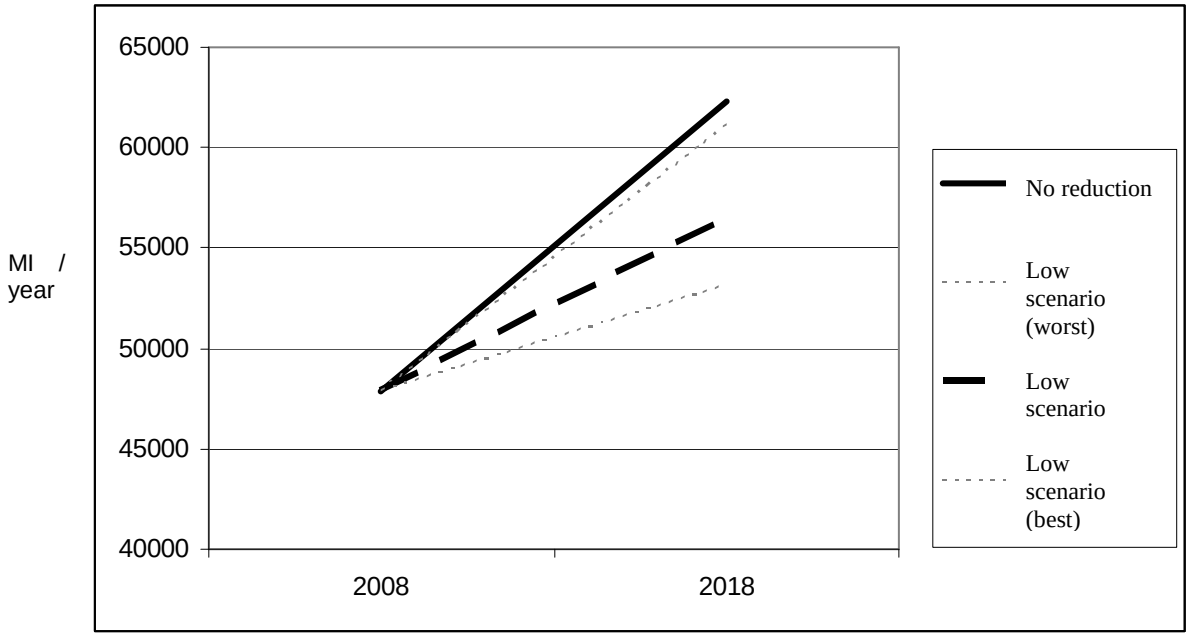


**Figure 8: MI in Australia – The effect of the high scenario**

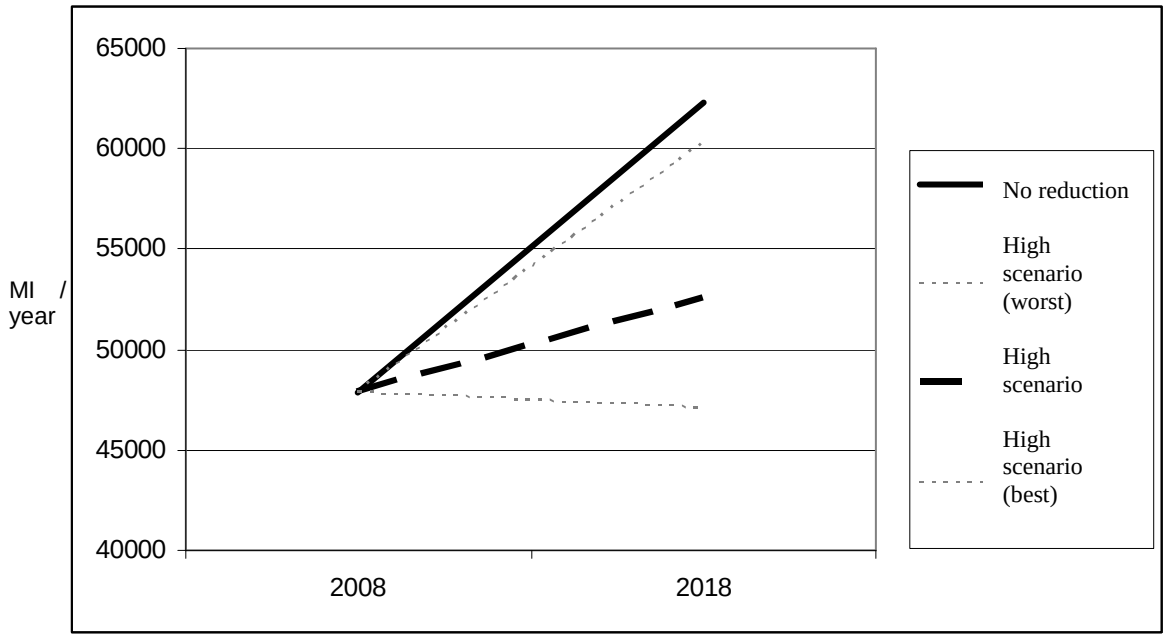



**Table 23: Mortality effects in New Zealand through MI and stroke reduction under low and high sodium intake projections in 2018 (both genders)**

| Age | MI (baseline) per annum | Low scenario | High scenario |
|---|---|---|---|
| 25-29 | 1 | 1 (Min:1) | 1 (Min:1) |
| 30-34 | 10 | 9 (Min:8) | 9 (Min:7) |
| 35-39 | 15 | 14 (Min:12) | 13 (Min:10) |
| 40-44 | 52 | 48 (Min:42) | 45 (Min:35) |
| 45-49 | 78 | 72 (Min:62) | 68 (Min:52) |
| 50-54 | 121 | 112 (Min:98) | 105 (Min:83) |
| 55-59 | 157 | 145 (Min:127) | 137 (Min:108) |
| 60-64 | 216 | 199 (Min:175) | 188 (Min:148) |
| 65-69 | 478 | 442 (Min:389) | 417 (Min:329) |
| 70-74 | 832 | 775 (Min:692) | 736 (Min:597) |
| 75-79 | 1,150 | 1,071 (Min:957) | 1,018 (Min:826) |
| 80-84 | 1,474 | 1,372 (Min:1,226) | 1,304 (Min:1,058) |
| 85+ | 3,357 | 3,125 (Min:2,793) | 2,971 (Min:2,410) |
| **Total*** | **7,942** | **7,385 (Min:6,584)** | **7,012 (Min:5,663)** |

* May not sum due to rounding

**Figure 9: New Zealand MI and stroke mortality – The effect of the low scenario**

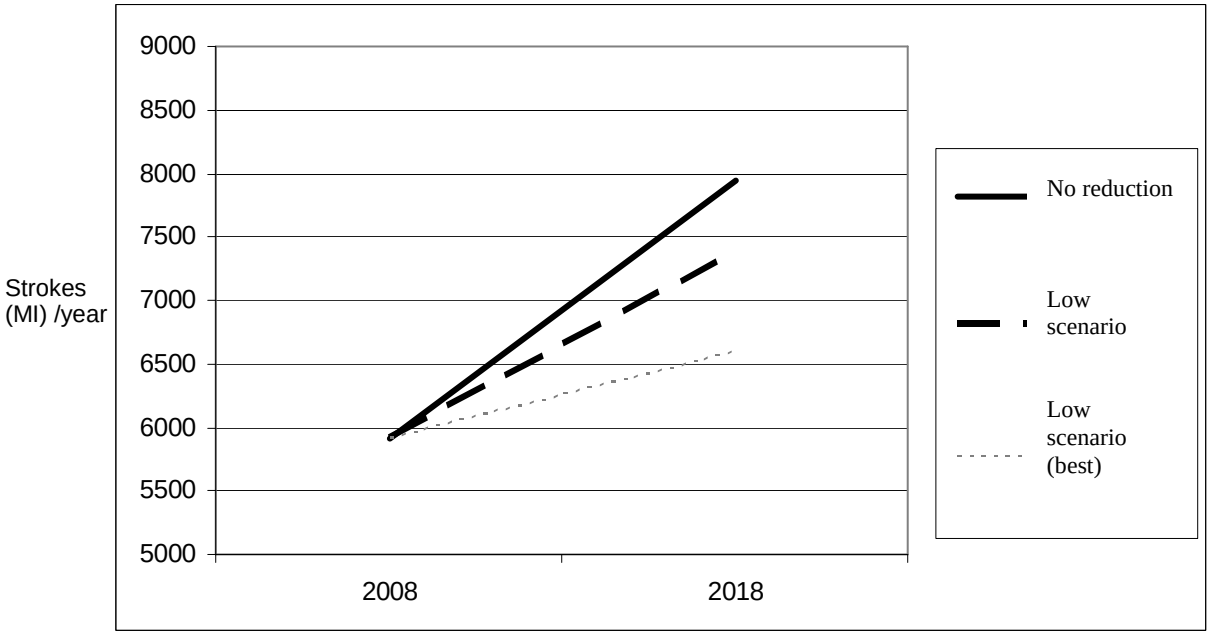



**Figure 10: New Zealand MI and stroke mortality – The effect of the high scenario**

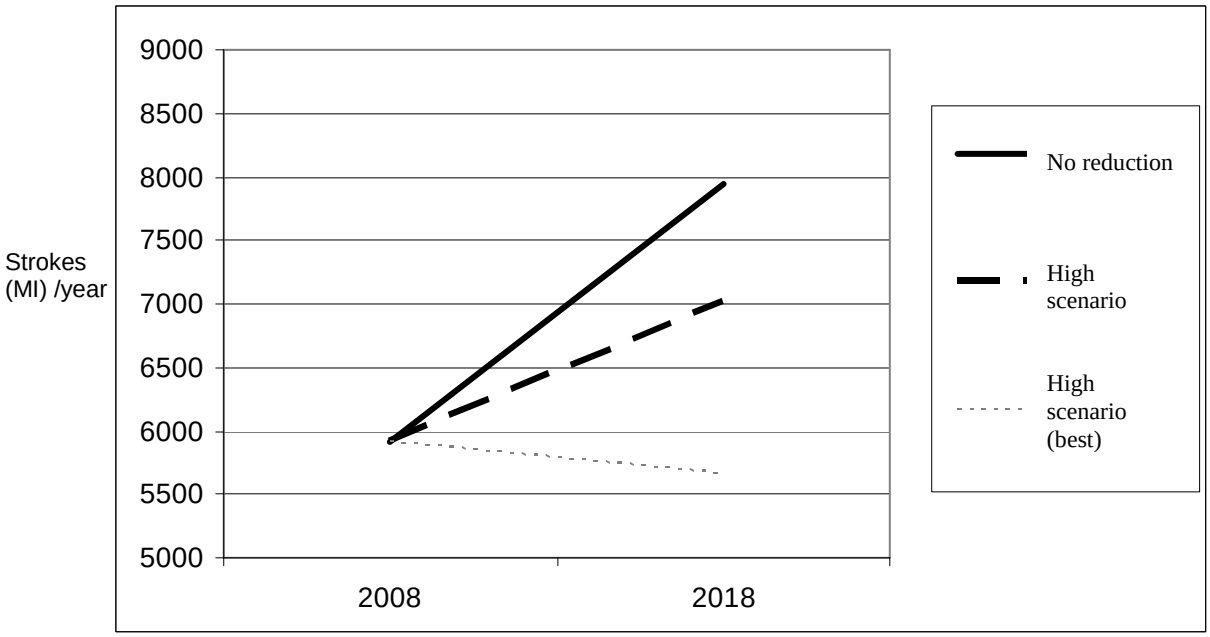


**Table 24: Stroke reduction under low and high reductions in sodium consumption in New Zealand in 2018 (both genders)**

| Age | Stroke (baseline) per annum | Low scenario | High scenario |
|---|---|---|---|
| 30-34 | 76 | 67 (62-75) | 61 (52-73) |
| 35-39 | 119 | 104 (96-116) | 94 (80-114) |
| 40-44 | 212 | 186 (171-207) | 168 (143-204) |
| 45-49 | 262 | 230 (211-256) | 208 (177-252) |
| 50-54 | 417 | 369 (342-408) | 337 (292-401) |
| 55-59 | 534 | 472 (438-521) | 432 (374-513) |
| 60-64 | 714 | 632 (587-698) | 578 (500-687) |
| 65-69 | 1,368 | 1,211 (1,123-1,337) | 1,107 (958-1,316) |
| 70-74 | 1,836 | 1,647 (1,540-1,797) | 1,518 (1,338-1,771) |
| 75-79 | 1,892 | 1,697 (1,587-1,852) | 1,565 (1,379-1,826) |
| 80-84 | 1,788 | 1,604 (1,500-1,750) | 1,479 (1,303-1,725) |
| 85+ | 2,261 | 2,028 (1,897-2,213) | 1,870 (1,648-2,182) |
| **Total*** | **11,480** | **10,247 (9,554-11,230)** | **9,416 (8,245-11,065)** |

* May not sum due to rounding

**Figure 11 Stroke in New Zealand – The effect of the low scenario**

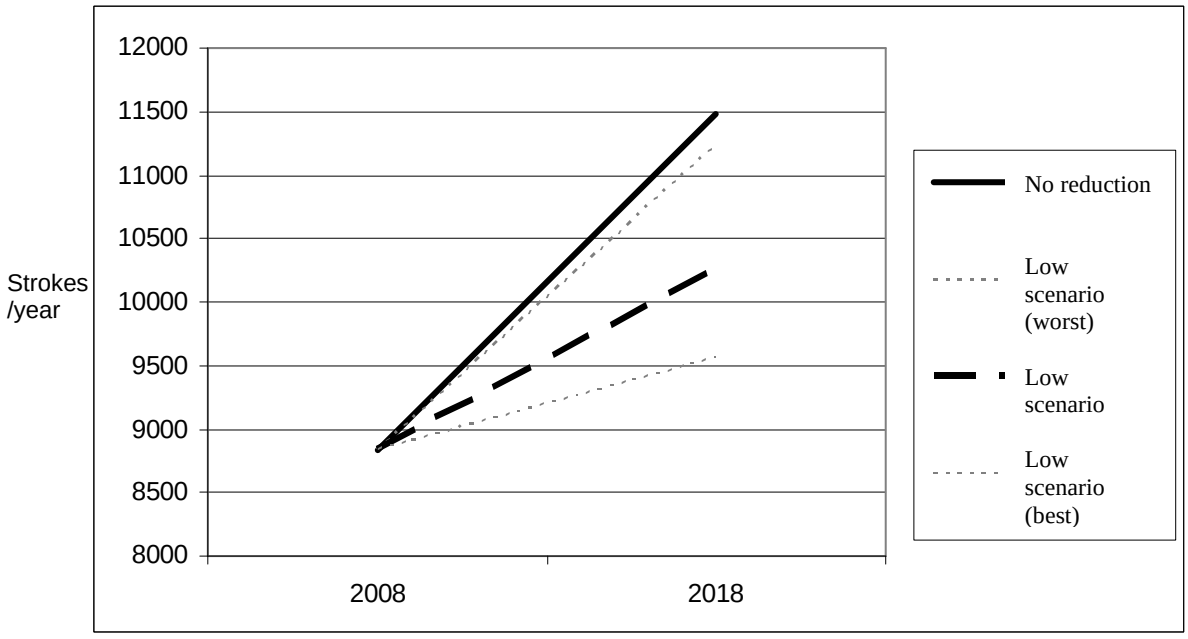


**Figure 12 Stroke in New Zealand – The effect of the high scenario**

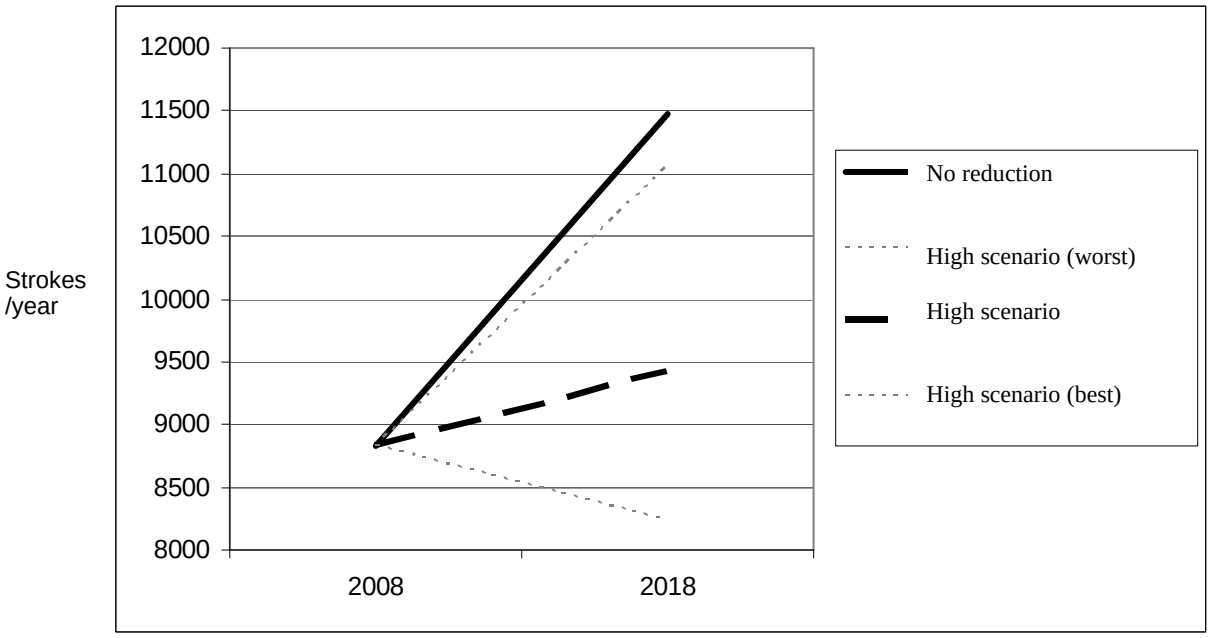



**Table 25: MI reduction under low and high reductions in sodium consumption in New Zealand in 2018 (both genders)**

| Age | MI (baseline) per annum | Low scenario | High scenario |
|---|---|---|---|
| 25-29 | 13 | 12 (11-13) | 11 (9-13) |
| 30-34 | 51 | 44 (41-49) | 40 (34-49) |
| 35-39 | 148 | 129 (119-144) | 117 (100-142) |
| 40-44 | 433 | 380 (349-423) | 344 (293-416) |
| 45-49 | 576 | 505 (464-562) | 457 (389-552) |
| 50-54 | 929 | 822 (763-908) | 752 (650-894) |
| 55-59 | 1,162 | 1,028 (954-1,135) | 940 (813-1,118) |
| 60-64 | 1,248 | 1,105 (1,025-1,219) | 1,010 (874-1,201) |
| 65-69 | 1,931 | 1,709 (1,585-1,886) | 1,562 (1,351-1,857) |
| 70-74 | 2,265 | 2,032 (1,901-2,218) | 1,874 (1,651-2,186) |
| 75-79 | 2,227 | 1,997 (1,868-2,180) | 1,841 (1,623-2,149) |
| 80-84 | 1,905 | 1,708 (1,598-1,865) | 1,575 (1,388-1,838) |
| 85+ | 2,189 | 1,964 (1,837-2,143) | 1,811 (1,596-2,113) |
| **Total*** | **15,077** | **13,435 (12,513-14,745)** | **12,332 (10,772-14,526)** |

* May not sum due to rounding

**Figure 13: MI in New Zealand – The effect of the low scenario**

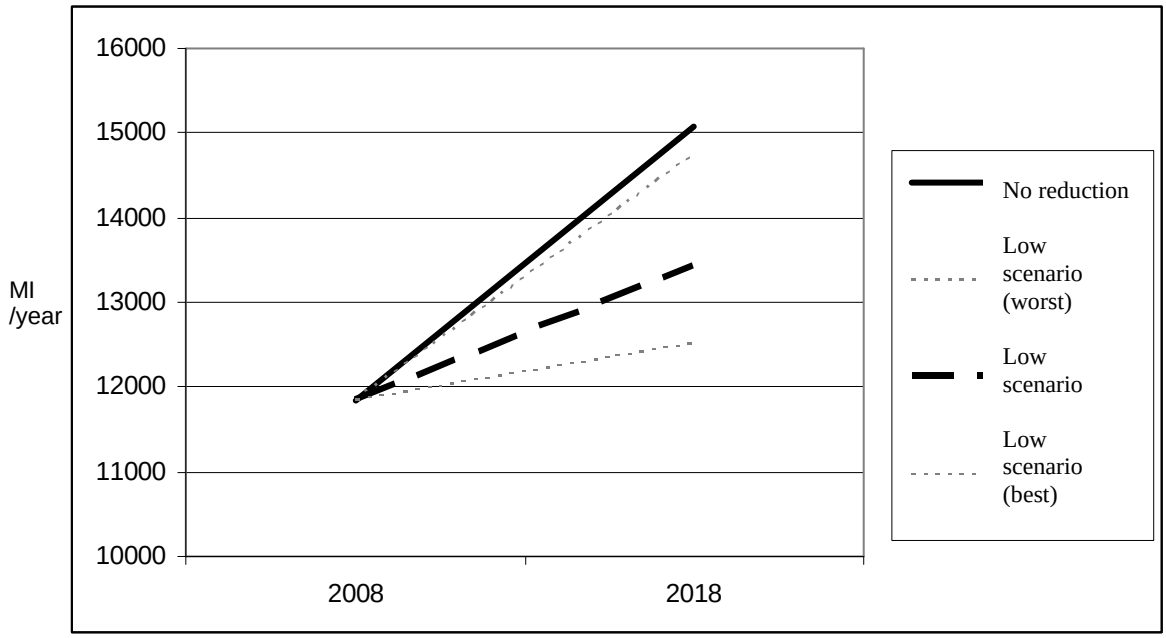



**Figure 14: MI in New Zealand – The effect of the high scenario**

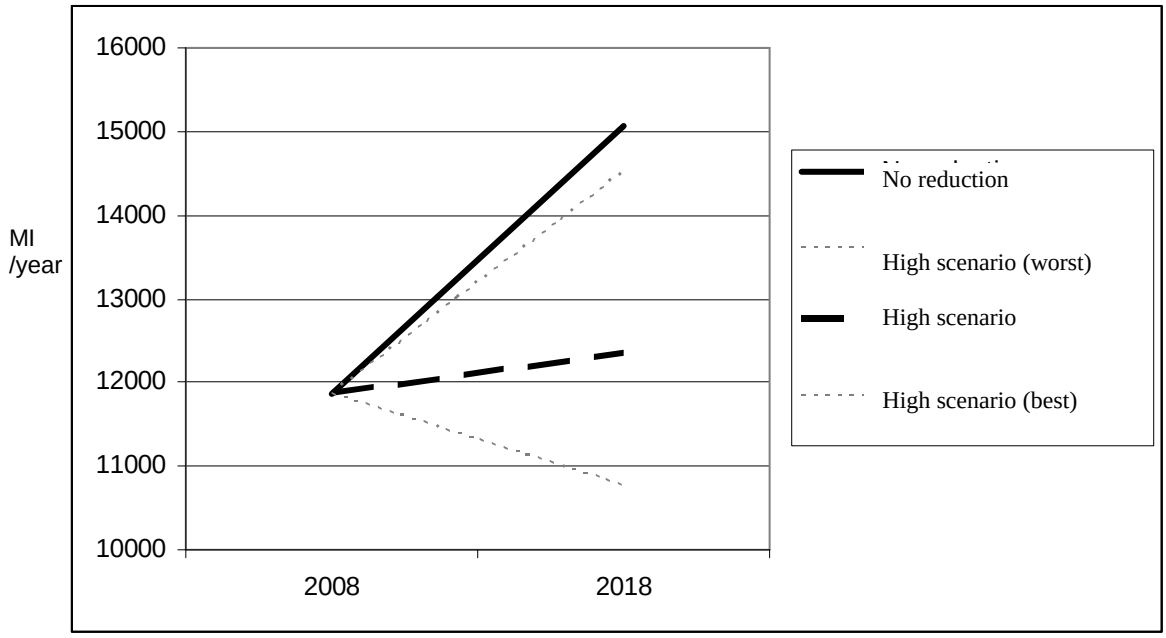



**Table 26: Summary of the MI and stroke events avoided in 2018 under each scenario in Australia**

| | | | | 2018 (low scenario) | | 2018 (high scenario) | |
|---|---|---|---|---|---|---|---|
| | | 2008 | 2018 | Predicted | Events avoided | Predicted | Events avoided |
| Stroke | Total | 40,048 | 53,368 | 48,450 | **4,918** | 45,154 | **8,214** |
| | Fatal | 11,487 | 15,886 | 14,936 | **950** | 14,293 | **1,593** |
| MI | Total | 47,878 | 62,295 | 56,458 | **5,837** | 52,557 | **9,738** |
| | Fatal | 12,685 | 17,491 | 16,442 | **1,049** | 15,733 | **1,758** |

**Table 27: Summary of the MI and stroke events avoided in 2018 under each scenario in New Zealand**

| | | | | 2018 (low scenario) | | 2018 (high scenario) | |
|---|---|---|---|---|---|---|---|
| | | 2008 | 2018 | Predicted | Events avoided | Predicted | Events avoided |
| Stroke | Total | 8,832 | 11,480 | 10,247 | **1,233** | 9,416 | **2,064** |
| | Fatal | 2,686 | 3,625 | 3,371 | **254** | 3,202 | **423** |
| MI | Total | 11,854 | 15,077 | 13,435 | **1,642** | 12,332 | **2,745** |
| | Fatal | 3,229 | 4,317 | 4,014 | **303** | 3,811 | **506** |

## 4.9 Step 7: Illustrate the DALY impact of reducing morbidity and mortality

### 4.9.1 Disability adjusted life years (DALYs)

Disability adjusted life year (DALY) is a health measurement that takes into account the potential years of life lost due to premature death (PYLL) and equivalent years of 'healthy' life lost by virtue of being in poor health (disability). One DALY represents the loss of one year of equivalent full health.

The DALYs for a disease are the sum of years of life lost, across the population, due to premature mortality (years of life lost, or YLL) and the years lost due to disability (YLD) for incident cases of the health condition.

Therefore; DALY = YLL + YLD

Where; YLL for a given disease, age and sex, corresponds to the number of deaths multiplied by the standard life expectancy at the age at which death occurs. YLD in a given time period, is the number of incident cases in the period multiplied by the average duration of the disease, multiplied by a weight factor that reflects the severity of the disease. The weight scale ranges from 0 (perfect health) to 1 (dead).

### 4.9.2 Applying DALYs

So far we have illustrated the cases of stroke and MI potentially averted, after the introduction of the Health Claims standard, if both the high and low sodium reduction scenarios are met. With regard to the burden of these conditions in Australia, we have extrapolated the findings using the report produced by Begg and colleagues (Begg, et al., 2007). Focusing first on stroke, they have identified a burden of disease for 2003 equal to 118,462 DALYs, constituting twenty-five percent of the burden associated with all cardiovascular disease (or 4.5% of the total burden of disease). This figure is divided between burden associated with fatal events (71%) and that associated with non-fatal events (29%).

We have estimated that, allowing for an ageing population, the fatal and total events in 2018 will be 38.3% and 31.2%, respectively, higher than in 2008. Therefore, we can estimate the baseline burden of disease for stroke in 2018,

and identify the reduction associated with the high and low sodium consumption scenarios.

**Table 28: The DALY impact of reduced sodium from salt in processed food on stroke in Australia**

| | 2008 DALYs | 2018 DALYs | Sodium reduction | % averted | DALYs saved* |
|---|---|---|---|---|---|
| Fatal events | 84,108 | 116,321 | Low scenario | 6.0 | 6,979 |
| | | | High scenario | 9.0 | 10,469 |
| Total events | 34,354 | 45,072 | Low scenario | 6.2 | 2,794 |
| | | | High scenario | 9.3 | 4,192 |
| **Total** | | | | **Low scenario** | **9,773** |
| | | | | **High scenario** | **14,661** |

We did not identify a DALY burden of stroke in New Zealand. Since prognosis following stroke is likely to be similar in the two countries, we applied the Australian data. For example, if we expect the number of strokes in New Zealand to be 20% of the Australian figure, we would assume that the DALY burden to be 20% of the Australian number. The New Zealand figures are presented in Table 29.

**Table 29: The DALY impact of reduced sodium from salt in processed food on stroke in New Zealand**

| | 2008 DALYs | 2018 DALYs | Sodium reduction | % averted | DALYs saved* |
|---|---|---|---|---|---|
| Fatal events | 18,549 | 25,022 | Low scenario | 7.0 | 1,752 |
| | | | High scenario | 11.7 | 2,928 |
| Total events | 7,393 | 9,446 | Low scenario | 10.9 | 1,030 |
| | | | High scenario | 18.2 | 1,719 |
| **Total** | | | | **Low scenario** | **2,782** |
| | | | | **High scenario** | **4,647** |

DALYs saved, refers to the number of DALYs saved in 2018 only.

Begg and colleagues also provide a total burden of disease figure for ischaemic heart disease. They estimated the total burden is equal to 263,497 DALYs, approximately 10% of the total burden of disease. 218,143 DALYS are due to mortality burden. They do not break this figure down into categories such as myocardial infarction or angina pectoris (Begg, et al., 2007). Therefore, no definitive total burden of disease could be found. However, it was possible to construct DALY estimates based on the methodology used by Begg *et al*. Firstly, for years of disability, the assumption they used (i.e. 3 months following discharge after acute MI weighted at 0.395) can be replicated. For life years lost, Year Book Australia 2007 (Australian Bureau of Statistics, 2007b) provides life expectancy at particular ages. Since we have previously generated the number of deaths under the baseline, low and high projected scenarios, we can multiply to produce an estimate of years lost through death. The results of this are shown in Table 30.

**Table 30: The DALY impact of reduced sodium from salt in processed food on MI in Australia**

| | 2008 DALYs | 2018 DALYs | Sodium reduction | % averted | DALYs saved* |
|---|---|---|---|---|---|
| Fatal events | 131,650 | 175,599 | Low scenario | 6.0 | 10,536 |
| | | | High scenario | 10.0 | 17,560 |
| Total events | 3,557 | 4,544 | Low scenario | 10.5 | 477 |
| | | | High scenario | 15.8 | 718 |
| **Total** | | | | **Low scenario** | **11,013** |
| | | | | **High scenario** | **18,278** |

DALYs saved, refers to the number of DALYs saved in 2018 only.

For New Zealand (as in Australia) we used the same assumption taken from Begg *et al*. regarding disability following a non-fatal MI. The results are shown in Table 31.

**Table 31: The DALY impact of reduced sodium from salt in processed food on MI in New Zealand**

| | 2008 DALYs | 2018 DALYs | Sodium reduction | % averted | DALYs saved* |
|---|---|---|---|---|---|
| Fatal events | 35,068 | 45,410 | Low scenario | 7.0 | 3,179 |
| | | | High scenario | 11.7 | 5,313 |
| Total events | 852 | 1,063 | Low scenario | 11.0 | 117 |
| | | | High scenario | 18.4 | 196 |
| **Total** | | | | **Low scenario** | **3,296** |
| | | | | **High scenario** | **5,509** |

DALYs saved, refers to the number of DALYs saved in 2018 only.

**Table 32: Summary of DALYs averted through sodium reduction from salt in processed food (2018 only)**

| | **Scenario** | **Australia** | **New Zealand** | **Total** |
|---|---|---|---|---|
| **MI** | Low | 11,013 | 3,296 | **14,309** |
| | High | 18,278 | 5,509 | **23,787** |
| **Stroke** | Low | 9,773 | 2,782 | **12,555** |
| | High | 14,661 | 4,647 | **19,308** |
| **Total** | Low | **20,786** | **6,078** | **26,864** |
| | High | **32,939** | **10,156** | **43,095** |

Table 32 summarises the potential DALY improvements after the introduction of the Health Claims standard, if both the high and low sodium reduction scenarios are met. The effect across the two conditions is comparable, although MI represents a slightly larger DALY improvement.

**Figure 15: Burden of disease in Australia**

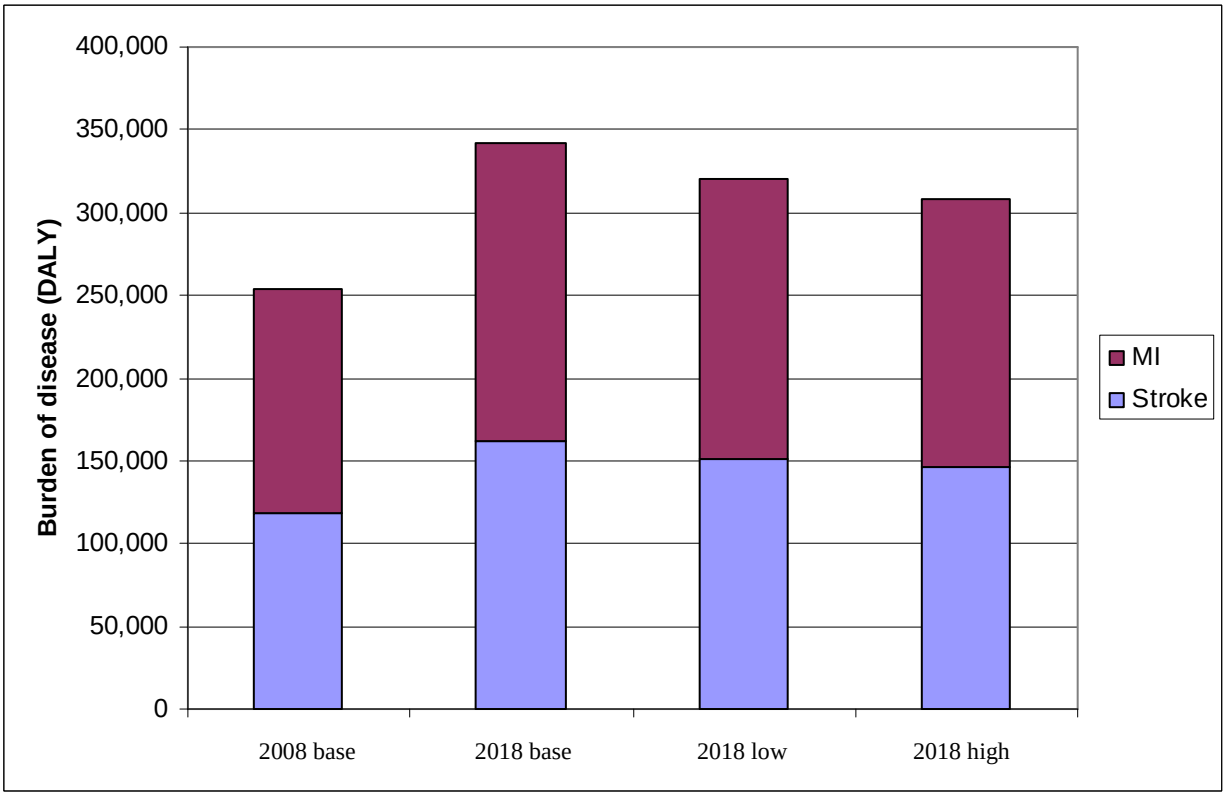
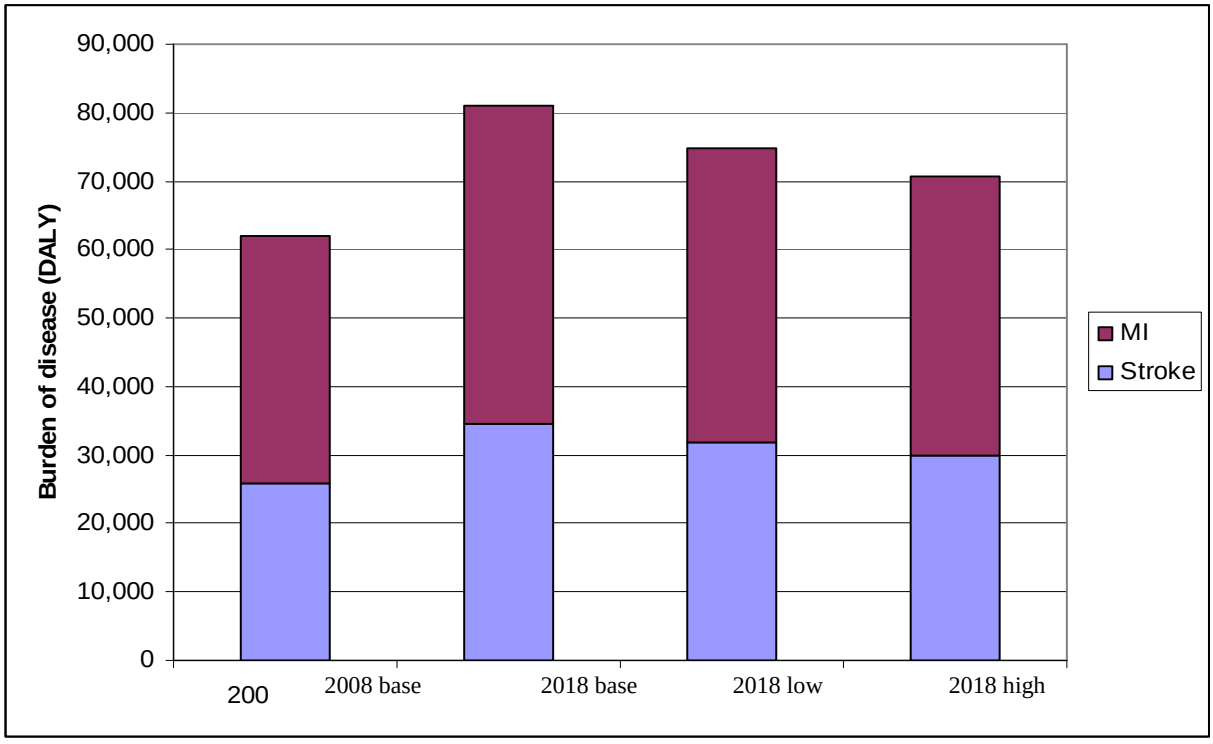



**Figure 16: Burden of disease in New Zealand**




## 4.10 Step 8: limitations of the analysis

There are a number of limitations to these figures. Firstly, there is considerable uncertainty regarding the future distribution of the Australian and New Zealand populations. In extrapolating current events to future populations, a number of unknown variables (such as migration, birth and death rates) affect the numbers of people in each age band. Where a range of options was available, we selected the middle option.

Secondly, the other significant point about the baseline extrapolation of incidence data is that it only accounts for the ageing of the population. In reality, we might expect the long-run trend in declining cardiovascular events to continue. This trend exists despite population ageing. The effect of including the expected reduction in events would be small since the absolute reduction in sodium consumption under both projections would be similar to that considered without these other exogenous factors.

Finally, a further issue is whether reduced amounts of sodium in processed foods may lead to a substitution effect towards discretionary salt use or increased consumption of processed foods made with lower levels of added sodium.

It should also be considered that reducing incidence of stroke and MI has the potential to lead to substantial cost savings to society. Firstly, the burden on the healthcare service is reduced with fewer presentations and therefore interventions. Additionally, there is a cost saving to society at large through reducing the numbers of individuals not in the productive workforce, either through death or disability. The estimation of this effect is difficult to do with any precision, but is important in conditions which affect people prior to retirement age.

# 5 The evaluation of saturated fat reduction in Australia and New Zealand

## 5.1 Summary

In this section we illustrate more potential health gains that may be possible following implementation of the health claims standard. The gains are based on pragmatic estimates in the reduction of saturated fat intake in Australia and New Zealand. The estimates reflect the assumption that the intake reduces by 15% (low scenario) and 25% (high scenario) by 2018. This section describes a modelling exercise in which the total reduction in saturated fat intake meets these scenarios. For clarity and illustrative purposes, we have focussed our findings on the reduction in myocardial infarction (MI). To avoid missing other benefits, we have extended the analysis to include DALYs.

Based on the current incidence of MI, we extrapolated an expected incidence in ten years, whilst adjusting for changes in the population demographics "ageing population" in both countries. We link saturated fat consumption under the low and high scenarios with reduced MI incidence and mortality using large population trial data. Finally, we estimated the DALY improvement associated with reduced incidences in MI.

It should be noted that we only identified tentative evidence regarding the scale of the risk reduction in a female population. Therefore, we looked only at this effect, aware it probably will underestimate the true benefit.

Without reduced saturated fat consumption, the incidence of MI is expected to increase due to the ageing population (ceteris paribus). Relative to this base case, both low and high reduction scenarios lead to a substantial improvement in the burden of disease. These estimates are likely to under represent the true effect since we have limited our analysis to MI and women. The results are summarised in Table 33.

**Table 33: Summary of DALY averted through saturated fat reduction**

| | Target | Australia | New Zealand | Total |
|---|---|---|---|---|
| **MI** | Low scenario | 9,811 | 2,689 | **12,500** |
| | High scenario | 16,409 | 4,429 | **20,838** |

## 5.2 Introduction

### 5.2.1 Saturated fat

Fatty acids can be categorised as: saturated fatty acids (SFAs), mono-unsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs). (Minihane and Harland, 2007) The World Health Organization (WHO) recommends that SFAs intake should be less than 10% of daily energy intake and less than 7% for high risk groups. (World Health Organisation, 2002)

The National Heart Foundation of Australia (NHFA) and the Cardiac Society of Australia and New Zealand (CSANZ) dietary advice includes a "low saturated fat eating plan" which incorporates moderate amounts of polyunsaturated and monounsaturated fats and oils. Physical activity and weight reduction are also recommended for lipid reduction. (Tonkin, et al., 2005) The National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (NZMoH) Suggested Dietary Target for saturated fat and *trans* fatty acids (combined) should be limited to 8-10% of total energy intake. (National Health and Medical Research Council and the New Zealand Ministry of Health, 2006)

Despite the gaps in the evidence, the NHFA, the American Heart Association (AHA) and the Canadian Cardiovascular Society among others strongly endorse lifestyle modification, including low SFA intake, for individuals with high blood cholesterol. (Lichtenstein, et al., 2006, McPherson, et al., 2006, Tonkin, et al., 2005) As suggested by Ketola's *et al*, multifactorial lifestyle interventions (including smoking cessation, diet and physical activity) are more effective than single dietary interventions. (Ketola, et al., 2000)

### 5.2.2 Saturated fat, blood cholesterol and coronary heart disease

In epidemiological studies, populations with low saturated fat diets have less cardiovascular events. These populations also tend to have lower plasma lipid concentrations. Low density lipoprotein (LDL) levels are sensitive to the amount of SFA and cholesterol in diet. (Keys, 1980, Robertson, et al., 1977) SFAs raise total and LDL-C (Hu, et al., 1997). However, the potency of individual fatty acid varies. SFAs with 12-16 carbons in the fatty acid chain; for example, myristic (C:14) and palmitic (C16) have greater effect on raising LDL-C. (Minihane and Harland, 2007, World Health Organisation) Mensink *et al* (1992), determined that replacement of saturated by unsaturated fatty acids raised the HDL to LDL cholesterol ratio[6]. Thus, under isocaloric conditions the most favourable lipoprotein risk profile for coronary heart disease was achieved if saturated fatty acids were replaced by unsaturated fatty acids. (Mensink and Katan, 1992)

A comprehensive literature review was conducted in 1999 by the NHFA. This report identified that there was a positive association between SFA intakes and coronary endpoints (non-fatal MI and fatal CHD) and an inverse association with

[6] Elevated concentrations of LDL cholesterol, total cholesterol, triglycerides or low concentrations of HDL cholesterol are risk factors for cardiovascular disease. Plasma lipid profile is used as a surrogate marker for cardiovascular. (Dzau, et al., 2006) Epidemiological evidence indicates that the risk for cardiovascular disease is continuous (log-linear) with increasing cholesterol levels. LDL elevation and/or mild to moderate hypertriglyceridaemia, especially if associated with reduced HDL, increases the risk of cardiovascular disease. (Law and Wald, 2002, Law and Wald, 1994)

PUFAs. In addition, SFAs have been shown to have an adverse effect on serum total cholesterol and LDL-C (National Heart Foundation of Australia, 1999a).

A FSANZ commissioned report investigated the relationship between SFAs, *trans* unsaturated fatty acids, LDL-cholesterol and CHD. The report concluded that there is convincing evidence for a "causal" link between saturated and *trans* unsaturated fatty acids and LDL cholesterol. It also concluded that there is a "probable" direct association between SFAs and CHD. The authors only suggest a "probable" rather than "convincing" association because of observed heterogeneity. This review critically assessed the Health Canada scientific summary and supplemented the evidence with literature published between 1999 and June 2005 (Booker and Mann, 2007).

Many studies include different preventative strategies, including SFA intake, to examine the effect on coronary endpoints. This makes it difficult to isolate the effect of dietary fat/SFAs intake on the incidence of CHD. However reduction or modification of dietary fat intake may be sufficient to reduce cardiovascular events in certain patients as shown in the following studies: 1) The Lyon Diet Heart Study[7], which demonstrated that the Mediterranean style diet[8] can significantly reduce non fatal and fatal MIs when used in addition to standard post MI pharmacotherapy (de Lorgeril, et al., 1999); 2) The Healthy Ageing: a Longitudinal study in Europe (HALE) [9], demonstrated that adherence to a Mediterranean diet and healthy lifestyle among individuals 70 to 90 years, was associated with a lower rate of all-cause and cause-specific mortality, including death due to CHD and cardiovascular disease (Knoops, et al., 2004).

Up to this point, most of the evidence has centred on establishing an indirect link between SFAs, LDL cholesterol and cardiovascular events. The majority of studies suggest a positive association between SFA intakes and coronary end points, without establishing a direct link.

The Health and Lifestyle Survey was conducted in the United Kingdom between 1984 and 1985. This survey included among other measures a dietary questionnaire. After the study period, deaths were monitored for the subsequent 16 years. The study identified that a level of saturated fat 100g higher per week corresponds to a relative risk for CHD death of 1.00 (0.86-1.18) in men and 1.40 (1.09-1.79) in women. In this cohort there was strong evidence of SFAs and CHD death in women (Boniface and Tefft, 2002).

Finally, there is little evidence that reducing SFA intakes alters the incidence of stroke (National Heart Foundation of Australia, 1999b). Due to the limited number of studies, the effect of SFA intakes on other risk factors such as blood pressure, insulin resistance and overweight as well as thrombosis and arrhythmia is unclear. Consequently these conditions are not included in this report

## 5.3 Illustration of potential benefits

We identify the hypothetical effect of changing saturated fat intake over a ten year period (2008-2018) in Australia and New Zealand. The estimated outcomes are: myocardial infarction events and deaths (as with sodium, MI is defined as ICD-10 I21/I22); and lost DALYs averted through reductions in these episodes. The argument that is presented follows this basic structure:

[7] For study details see Brooker et al FSANZ commissioned report.
[8] Mediterranean style diet is high in fruits and vegetables, fish, poultry, cereal grains, nuts
[9] For study details see Brooker et al FSANZ commissioned report.

- Step 1: Identify current saturated fat intake in both Australia and New Zealand, stratified by gender
- Step 2: Identify realistic scenarios for the reduction of saturated fat, and estimate the new intake profile in the two countries if these scenarios are met
- Step 3: Establish the link between saturated fat consumption and Coronary Heart Disease; events and deaths
- Step 4: Estimate the reduction in MI in the 2018 population based on changing levels of saturated fats, accounting for population ageing
- Step 5: Estimate the DALY averted under the low and high saturated fat reduction scenarios, comparing the magnitude of the effect with that generated in the analysis of sodium reduction
- Step 6: Discuss the limitations of the modelling

## 5.4 Step 1: Identify current saturated fat intake in both Australia and New Zealand, stratified by gender

Estimates of saturated fat consumption were supplied by Food Standards Australia and New Zealand. These data are presented in Table 34. The estimates are applicable to the adult populations of Australia and New Zealand, subdivided by gender.

**Table 34: Saturated fat intake in Australian and New Zealander adults (aged 19 years and older)**

| | Estimated intake of saturated fat (g/day) | | | |
|---|---|---|---|---|
| | Australia | | New Zealand | |
| | **Mean** | **95$^{th}$ Percentile** | **Mean** | **95$^{th}$ Percentile** |
| Males | 38.8 | 68.5 | 46.9 | 76.6 |
| Females | 26.8 | 48.2 | 32.0 | 54.2 |
| All | 32.4 | 60.7 | 38.1 | 67.4 |

In general, males have a greater intake of saturated fat, but in terms of saturated fat intake per kilogram of body weight, the figures are much closer. In New Zealand the level of saturated fat consumed is higher across both genders. With this in mind, the scenarios presented below, which are specified in percentage reduction, will accordingly lead to a greater absolute reduction in saturated fat intake per person in New Zealand.

## 5.5 Step 2: Identify realistic scenarios for the reduction of saturated fat, and estimate the new intake profile in the two countries if these scenarios are met

To illustrate the potential health gains that may be possible following reduced saturated fat consumption, FSANZ provided low and high reduction scenarios for saturated fat consumption in Australia and New Zealand. These are pragmatic estimates and reflect the assumption that intake of saturated fat will reduce by 15% ('low estimate') and 25% ('high estimate') by 2018. The purpose of these scenarios is to illustrate the effect of reducing saturated fat on the health and life expectancy of the population.

The validity of these scenarios is outside the scope of this report. Achieving any reduction in the saturated fat is based upon the value that manufacturers place on meeting the new health claims standard. However, it is believed that 15% reduction is a realistic scenario, whilst the 25% reduction is a best case scenario.

The implications of these targets on current levels of saturated fat consumption are shown in Table 35. As discussed previously, the reduction figures are considerably higher in New Zealand due to higher consumption of saturated fat in 2008.

**Table 35: Saturated fat consumption scenarios**

| | | Intake of saturated fat (g/day) (reduction) | | |
|---|---|---|---|---|
| | | 2008 | 2018 | |
| | | Current mean | Low scenario | High scenario |
| Australia | Males | 38.8 | 32.98 (5.82) | 29.1 (9.70) |
| | Females | 26.8 | 22.78 (4.02) | 20.1 (6.70) |
| | All | 32.4 | 27.54 (4.86) | 24.3 (9.10) |
| New Zealand | Males | 46.9 | 39.86 (7.04) | 35.17 (11.73) |
| | Females | 32.0 | 27.20 (4.80) | 24.00 (8.00) |
| | All | 67.4 | 57.29 (10.11) | 50.55 (16.85) |

## 5.6 Step 3: Establish the link between saturated fat consumption and Coronary Heart Disease; events and deaths

The link between saturated fat intake and CHD events and mortality is difficult to quantify. Most of the existing studies involve a mixed intervention approach to CHD-avoidance thus making it difficult to disentangle particular effects. Boniface *et al*, identified that an increase in saturated fat of 100g per week corresponds to a relative risk for CHD death of 1.00 (0.86-1.18) in men and 1.40 (1.09-1.79) in women (Boniface and Tefft, 2002). Based on these data, we can use the estimated reductions in saturated fat associated with the two scenarios and the expected number of CHD deaths in Australia and New Zealand in 2018 (previous section), to calculate the reduction in deaths (or events) associated with achieving the two scenarios. Note that the relative risks given in Table 36 are less than or equal to one. This is because they are inverted to represent the reduction

in risk associated with a lower consumption of saturated fat. For example, a 100g/week reduction in saturated fat leads to a relative risk of 1/1.4=0.714.

**Table 36: Mortality relative risks under low and high saturated fat reduction scenarios in Australia and New Zealand**

| | | Mortality relative risk | |
|---|---|---|---|
| | | **Low scenario** | **High scenario** |
| Australia | Males | 1 | 1 |
| | Females | 0.8874 | 0.8124 |
| New Zealand | Males | 1 | 1 |
| | Females | 0.8656 | 0.7760 |

* Note that we have assumed a linear relationship between saturated fat reduction and mortality (e.g. In Boniface, a 100g increase in saturated fat consumption per week leads to a 40% increase in CHD mortality risk, a 50g increase leads to a 20% increase).

With regard to morbidity effects of high saturated fat intake, we were unable to identify a quantifiable association between high saturated fat and MI event rates. For men, we made the conservative assumption that, since the mortality RR associated with increased saturated fat intake was 1, the event RR would also be 1. For women, we assumed a relative risk range of between 1 - 1.40, which is the RR associated with increased weekly saturated fat intake of 100g. The rationale being that the lower extreme (RR=1) assumes that the only effect of reducing saturated fat is reduced mortality (therefore relying only on the available evidence) and the upper extreme (RR=1.4) assumes that the rate of events follows the mortality rates. It is arguable that factors such as statins would make the risk of death contingent on having an MI would differ but, in the absence of good quality data, we have assumed this is not a factor. The relative risks for events are given in Table 37.

**Table 37: Event relative risks under low and high saturated fat reduction scenarios in Australia and New Zealand**

| | | Event relative risk | |
|---|---|---|---|
| | | **Low scenario** | **High scenario** |
| Australia | Males | 1 | 1 |
| | Females | 0.8874-1 | 0.8124-1 |
| New Zealand | Males | 1 | 1 |
| | Females | 0.8656-1 | 0.7760-1 |

## 5.7 Step 4: Estimate the reduction in MI in the 2018 population based on changing levels of saturated fats, accounting for population ageing

In the previous section, we identified the natural increase in MI between 2008 and 2018, accounting for the expected ageing population (Table 19). This table is replicated below. Since the expected effect of reduced saturated fat consumption affects males and females differently, we have reported events in both aggregate form, and by gender.

**Table 38: Expected MI events and death in 2008 and 2018 (no change in saturated fat intake)**

| | | Australia | | New Zealand | |
|---|---|---|---|---|---|
| | | **2008** | **2018** | **2008** | **2018** |
| MI total | (Male) | 31,338 | 40,736 | 7,449 | 9,475 |
| | (Female) | 16,540 | 21,559 | 4,405 | 5,602 |
| | Total | **47,878** | **62,295** | **11,854** | **15,077** |
| MI Fatal | (Male) | 6,536 | 9,009 | 1,694 | 2,317 |
| | (Female) | 6,153 | 8,482 | 1,535 | 2,000 |
| | Fatal | **12,685** | **17,491** | **3,229** | **4,317** |

* One issue with this result is that New Zealand population projections do not separate for males and females. Therefore, in constructing this table, we had to assume a comparable male/female divide in both countries.

We can now estimate the reduction in both fatal and total MI under the low and high saturated fat intake scenarios. This is done by combining the expected cases in the above table with the relative risks given in Table 36 and Table 37.

**Table 39: Reduction in MI events and mortality under both saturated fat intake scenarios in Australia and New Zealand**

| | | Total per annum (2018) | | |
|---|---|---|---|---|
| | | **(no saturated fat reduction)** | **Low scenario** | **High scenario** |
| Australia | Total | 62,295 | 59,868-62,295 | 58,251-62,295 |
| | Fatal | 17,491 | 16,536 | 15,890 |
| New Zealand | Total | 15,077 | 14,324-15,077 | 13,822-15,077 |
| | Fatal | 4,317 | 4,048 | 3,869 |

## 5.8 Step 5: Estimate the DALYs averted under the low and high saturated fat reduction scenarios, comparing the magnitude of the effect with that generated in the analysis of sodium reduction

Begg and colleagues consider the burden of disease associated with ischaemic heart disease as a result of high blood cholesterol[10] (Begg, et al., 2007). In 2003, they attribute 13,371 deaths (10.1% of the total) and 138,605 DALYs (5.3% of the total) to this. However, as with high sodium intake, they do not break this down to allow figures to be derived for MI. Therefore, we replicated the approach used in the sodium section, by estimating the number of years lost through death, and adding that to the years lost through disability.

[10] High blood cholesterol has been used as a proxy for high levels of saturated fats.

## Table 40: The DALY impact of reduced saturated fat on MI in Australia

| | 2018 DALYs | Saturated fat reduction | % averted | DALYs saved |
|---|---|---|---|---|
| Fatal events | 175,599 | Low scenario | 5.5% | 9,658 |
| | | High scenario | 9.2% | 16,155 |
| Total events | 4,560 | Low scenario | 3.4% | 153 |
| | | High scenario | 5.6% | 254 |
| **Total** | | | **Low target** | **9,811** |
| | | | **High target** | **16,409** |

* Note that the range implicit in Table 37 is not given here since the upper range will simply be 0 DALYs saved for non-fatal MI events.

## Table 41: The DALY impact of reduced saturated fat on MI in New Zealand

| | 2018 DALYs | Saturated fat reduction | % averted | DALYs saved |
|---|---|---|---|---|
| Fatal events | 48,426 | Low target | 5.4% | 2,609 |
| | | High target | 9.0% | 4,349 |
| Total events | 1,120 | Low target | 4.3% | 48 |
| | | High target | 7.1% | 80 |
| **Total** | | | **Low target** | **2,689** |
| | | | **High target** | **4,429** |

* Note that the range implicit in Table 37 is not given here since the upper range will simply be 0 DALYs saved for non-fatal MI events.

**Figure 17: MI burden of disease associated with low and high saturated fat scenarios (Australia)**

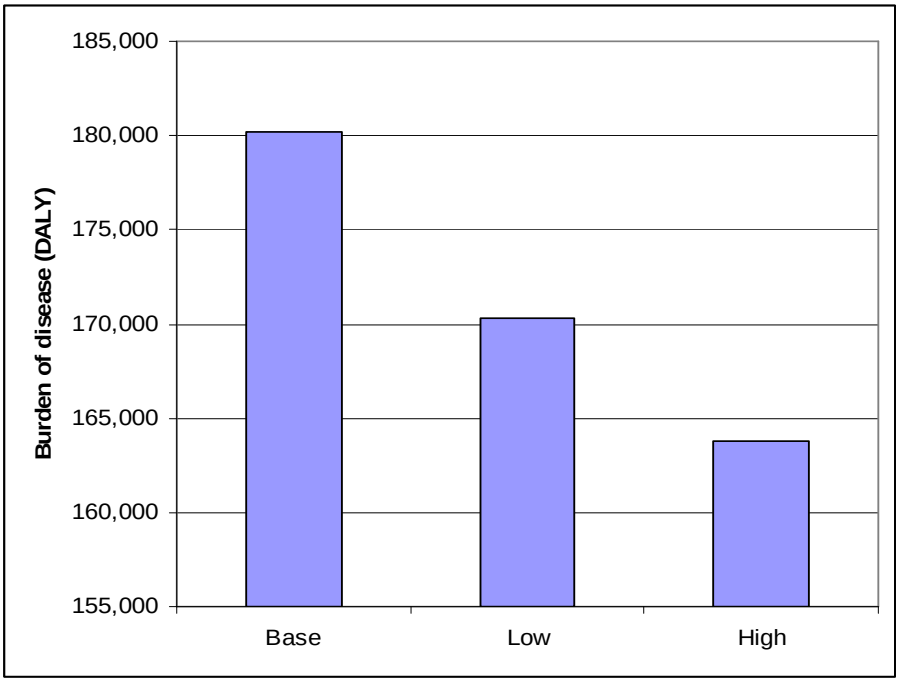
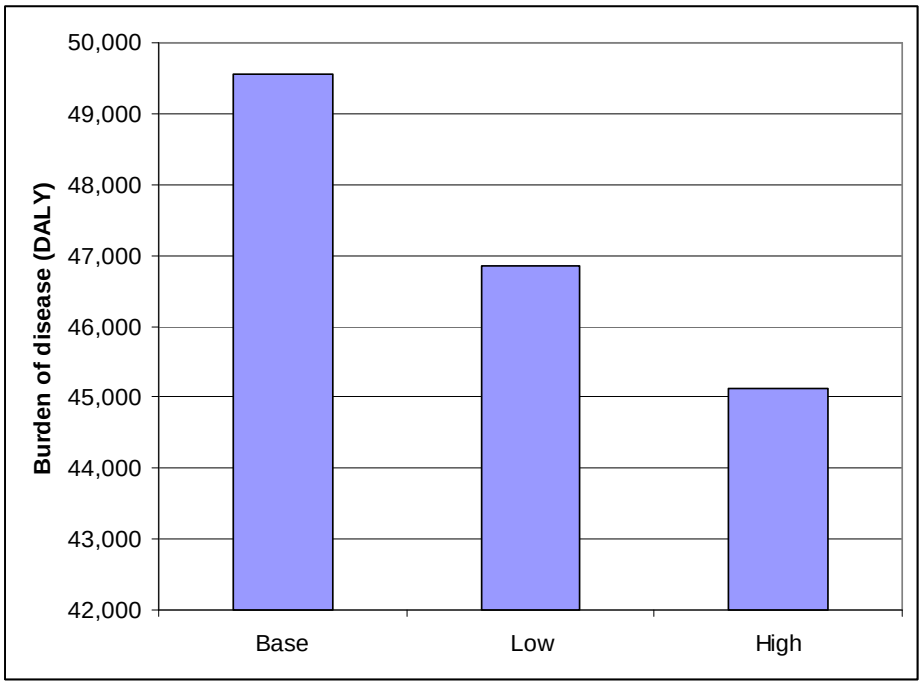



**Figure 18: MI burden of disease associated with low and high saturated fat scenarios (New Zealand)**



## 5.9 Step 6: Discuss the limitations of the modelling

A number of limitations which applied to the previous discussion on sodium intake apply in this case. Firstly, there is considerable uncertainty regarding the future distribution of the Australian and New Zealand populations. In extrapolating current events to future populations, a number of unknown variables (such as migration, birth and death rates) affect the numbers of people in each age band. Where a range of options were available, we selected the middle option.

Secondly, the other significant point about the baseline extrapolation of incidence data is that it only accounts for the ageing of the population. In reality, we might expect the long-run trend in declining cardiovascular events to continue. This trend exists despite population ageing. The effect of including the expected reduction in events would be small since the absolute reduction in saturated fat consumption under both projections would be similar to that considered without these other exogenous factors.

Thirdly, it should also be considered that reducing incidence of MI has the potential to lead to substantial cost savings to society. Firstly, the burden on the healthcare service is reduced with fewer presentations and therefore interventions. While quantifying the link between saturated fat consumption and MI is difficult, the link between saturated fat and cholesterol is convincing, which means any reduction in saturated fat consumption is likely to have an important impact on the use of cholesterol lowering drugs. Additionally, there is a cost saving to society at large through reducing the numbers of individuals not in the productive workforce, either through death or disability. The estimation of this effect is difficult to do with any precision, but is important in conditions which affect people prior to retirement age.

A limitation specific to saturated fat consumption is that the evidence of the scale of the association between saturated fat consumption and MI is tentative a best, and limited to women. Therefore, the results presented here are limited to this population group and are likely to be an underestimate of the true effect of reduced saturated fat intake.

# 6 Appendix

**Table 42: Australian and New Zealand* Populations (2008 and 2018)**

| | 2008 | | 2018 | |
|---|---|---|---|---|
| **Age** | **Australia** | **New Zealand** | **Australia** | **New Zealand** |
| 25-29 | 1,460,672 | 294,160 | 1,602,916 | 303,720 |
| 30-34 | 1,456,999 | 294,160 | 1,640,616 | 303,720 |
| 35-39 | 1,584,226 | 294,160 | 1,638,659 | 303,720 |
| 40-44 | 1,502,700 | 273,160 | 1,578,782 | 297,480 |
| 45-49 | 1,550,956 | 273,160 | 1,653,162 | 297,480 |
| 50-54 | 1,396,959 | 273,160 | 1,523,026 | 297,480 |
| 55-59 | 1,296,725 | 273,160 | 1,537,132 | 297,480 |
| 60-64 | 1,133,700 | 273,160 | 1,370,821 | 297,480 |
| 65-69 | 842,190 | 158,270 | 1,252,471 | 230,095 |
| 70-74 | 677,107 | 127,247 | 1,059,497 | 194,643 |
| 75-79 | 560,154 | 105,268 | 737,046 | 135,405 |
| 80-84 | 433,011 | 81,375 | 521,387 | 95,786 |
| 85+ | 371,634 | 69,840 | 555,601 | 102,071 |

*Due to wider categories being used in New Zealand, we assumed a uniform distribution in the 25-39 and 40-64 year old group. In the 65 year and over groups, we adopted the Australian distribution of ages as a good estimate of the New Zealand population.

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