Public Health Nutrition: 8(4), 402–408 DOI: 10.1079/PHN2004704

Mortality attributable to higher-than-optimal body mass index in New Zealand

Cliona Ni Mhurchu[1,*], Maria Turley[2], Niki Stefanogiannis[2], Carlene MM Lawes[1], Anthony Rodgers[1], Stephen Vander Hoorn[1] and Martin Tobias[2]

[1]Clinical Trials Research Unit, University of Auckland, Private Bag 92 019, Auckland, New Zealand: [2]Public Health Intelligence Unit, Ministry of Health, Wellington, New Zealand

Submitted 17 May 2004: Accepted 17 November 2004

Abstract

Objectives: To estimate the burden of mortality in New Zealand due to higher-than-optimal body mass index (BMI) in 1997, as well as mortality that could be avoided in 2011 with feasible changes in mean population BMI.

Setting: New Zealand.

Design: Comparative risk assessment methodology was used to estimate the attributable and avoidable mortality due to high BMI. Outcomes assessed were ischaemic heart disease (IHD), ischaemic stroke, type 2 diabetes mellitus, colorectal cancer and postmenopausal breast cancer.

Results: In 1997, 3154 deaths (11% of all deaths) in New Zealand were due to higher-than-optimal BMI ($>21\,kg\,m^{-2}$). This amounted to 83% of diabetes deaths, 24% of IHD deaths, 15% of ischaemic stroke deaths and 4% of all cancer deaths. If the projected increase in mean population BMI by 2011 was limited to $1.0\,kg\,m^{-2}$ rather than $1.3\,kg\,m^{-2}$, approximately 385 deaths could be prevented annually, mainly from diabetes.

Conclusions: These results quantify the importance of higher-than-optimal BMI as a major modifiable cause of premature death in New Zealand. Intervention policies that would have only modest effects on slowing the rate of increase in mean population BMI by 2011 could still prevent hundreds of deaths annually.

Keywords
Body mass index
Obesity
Mortality
New Zealand

Overweight and obesity are becoming increasingly common in both developed and developing populations[1]. Body mass index (BMI) is the anthropometric measure that provides the most useful population-level indicator of excess body weight. The World Health Organization (WHO) guidelines define a BMI of 18.50 to $24.99\,kg\,m^{-2}$ as normal and $\geq 25\,kg\,m^{-2}$ as overweight[2]. The recent WHO Global Burden of Disease report[3] estimated that obesity rates (BMI $\geq 30\,kg\,m^{-2}$) vary regionally from 2–3% in some Asian countries to 75% in several Pacific Island nations. Currently, there are over 300 million obese individuals in the world and more than 750 million are overweight[3].

Secular trends indicate that the prevalence of overweight and obesity is increasing rapidly. In the USA, the prevalence of obesity increased from 20% of men and 25% of women in 1988–1994 to 28% of men and 33% of women in 1999–2000[4], and in England the prevalence increased from 13% of men and 16% of women in 1993 to 21% of men and 24% of women in 2001[5]. Similar trends are being seen in many other countries including countries such as China, where rates of obesity are relatively low[6]. In New Zealand, the prevalence of obesity increased by more than 50% between 1989 and 1997, from 11% to 17%[7].

High BMI is an important contributor to cardiovascular disease[8,9], operating in part through effects on blood pressure[10], blood lipids[11,12] and blood glucose[13]. High BMI is also associated with increased incidence of type 2 diabetes mellitus[14,15] and several common cancers[16,17]. Current evidence[18] suggests that the association between BMI and some diseases such as type 2 diabetes and coronary heart disease is continuous from BMI values as low as $21\,kg\,m^{-2}$, which is well below the accepted cut-off values for overweight and obesity[2]. Estimations of the global burden of disease indicate that high BMI is a leading cause of loss of healthy life: 33 million disability-adjusted life years (DALYs) world-wide[19].

Despite increasing secular trends in overweight and obesity, the full impact of high BMI on burden of disease has never been accurately quantified in New Zealand. Therefore, the aims of the present study were to estimate the burden of mortality in New Zealand due to higher-than-optimal BMI ($>21\,kg\,m^{-2}$) in 1997, as well as the burden of mortality that could be avoided in 2011

*Corresponding author: Email c.nimhurchu@ctru.auckland.ac.nz

if a smaller increase in BMI than that projected (based on current trends) was to occur.

Methods

The analyses described in this paper were conducted as part of a larger study examining the impact of four nutrition-related risk factors on the burden of disease in New Zealand: blood pressure, total blood cholesterol, fruit and vegetable intake, and BMI[20]. The study methods have been described in detail elsewhere[20] but a brief summary is provided below.

To estimate the attributable and avoidable mortality due to excess body weight, the comparative risk assessment (CRA) methodology developed by the WHO for the 2002 World Health Report[21] was used. CRA is a systematic approach to estimating the current burden of disease attributable to various risk factors, as well as the future burden of disease that could be avoided if exposure to these risk factors were reduced. It uses standardised methods to obtain best estimates of risk factor distributions, risk factor–disease relationships, disease burden and estimates of parameter uncertainty. The methodology takes into account the entire population risk factor distribution by focusing on continuous (rather than categorical) risk factor–disease relationships. Attributable mortality is calculated by comparing the current risk factor distribution with the one that confers minimal risk: the theoretical minimum distribution. Avoidable mortality is calculated by estimating the number of deaths potentially avoidable in the future if there were changes to the risk factor distribution.

Data on current measured population BMI levels by age group and sex were obtained from the 1997 National Nutrition Survey, a nationally representative survey of 4636 adult New Zealanders[22]. Calculations of attributable and avoidable mortality were based on a theoretical minimum BMI of $21 \pm 1\,\mathrm{kg\,m^{-2}}$, which was chosen by the WHO for their global burden of disease study[3,19] because available evidence suggests that this BMI distribution would yield the lowest population risk of adverse health outcomes[8,18].

To estimate avoidable burden, two scenarios for projected BMI were estimated: a business as usual (BAU) scenario based on historical trends, and an intervention scenario (deviation from the historical trend reflecting policy change). Distributional transitions for the BAU and intervention scenarios were calculated as a percentage shift away from the theoretical minimum from the current BMI distribution. Because BMI distribution varied by age and sex, absolute shifts depended on current BMI. The avoidable burden was the difference between the projected BAU scenario and the intervention scenario.

The five mortality outcomes assessed were based on strong evidence of a causal relationship and sufficient data to quantify the risk–factor disease relationship[21]: ischaemic heart disease (IHD), ischaemic stroke, type 2 diabetes, colorectal cancer and postmenopausal breast cancer. To estimate the nature and strength of the association between BMI and cardiovascular disease endpoints (IHD and ischaemic stroke mortality) data from the Asia Pacific Cohort Studies Collaboration were used[8], which incorporated individual participant data from 33 cohorts in Asia (80% of participants), and Australia and New Zealand (20% of participants), involving 310 000 participants. There were no significant differences in the size or shape of the BMI and cardiovascular disease associations between Asian and Australian/New Zealand cohorts, so the combined data from all cohorts were used. To estimate the nature and strength of the associations between BMI and diabetes mortality, data from both published and unpublished sources were used[3]. For colorectal cancer and postmenopausal breast cancer mortality, data from a recent meta-analysis were used[17].

The number of deaths in New Zealand in 1997 for each selected disease was extracted from the New Zealand Health Information Service (NZHIS) mortality database. Projections by the Ministry of Health were used to estimate the predicted number of deaths for each disease in 2011. For non-cancer endpoints, classical age–period–cohort (APC) models were constructed[23]. For cancer endpoints, Bayesian and generalised non-linear modelling methods were used in addition to the APC models[24]. Mortality counts were translated into years of life lost (YLL) counts using the 'remaining life expectancy' method[25]. YLL counts were discounted to the present using a 3% per annum discount rate.

Simple extraction from the NZHIS mortality database could not be used for diabetes because many deaths caused by diabetes are not coded as such but rather to cardiovascular or renal codes, reflecting the immediate cause of death. A multi-state life table model[26] estimated that the number of deaths attributable to diabetes was approximately 120% greater than the number coded to diabetes overall, so appropriate adjustments were made for this overlap in our analyses.

Confidence intervals for attributable burden were estimated by a simulation procedure incorporating sources of uncertainty from domains of the exposure distribution (i.e. mean and standard deviation) and the exposure–response relationships. Briefly, the method involved simultaneously varying all input parameters within their respective distributions and reiterating the calculation of the population-attributable fraction. An uncertainty distribution around each estimate of the population-attributable fraction was obtained after 500 iterations of the simulation and, from this, 95% confidence intervals were derived[21]. The added complexity of taking into account the uncertainty in projected disease rates, as well as uncertainty within the estimates of risk reversibility, made it too difficult to reliably quantify uncertainty around avoidable burden.

https://doi.org/10.1079/PHN2004704 Published online by Cambridge University Press

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

Results

Current distribution

Mean (standard deviation) population BMI was 26.2 (4.4) kg m^{-2} for males and 26.1 (5.6) kg m^{-2} for females. Mean BMI increased steadily with age and peaked in the 55–64 year age group, after which it declined slightly in older age groups. There was little difference in mean BMI between males and females (Fig. 1).

Distributional transitions

The BAU scenario was based upon historical trends of population BMI in New Zealand from 1977 to 1997[22,27,28] and was estimated as an increase in mean population BMI of 1.3 kg m^{-2} (1.1–1.6 kg m^{-2} depending on age) in both males and females by 2011. This increase in BMI would mean an average 25% further shift away from the theoretical minimum (Fig. 1) and would lead to a mean population BMI of 27.5 kg m^{-2} in males and 27.4 kg m^{-2} in females.

Under the more optimistic intervention scenario, it was estimated that realistic policy intervention could reduce the projected rate of increase in mean population BMI

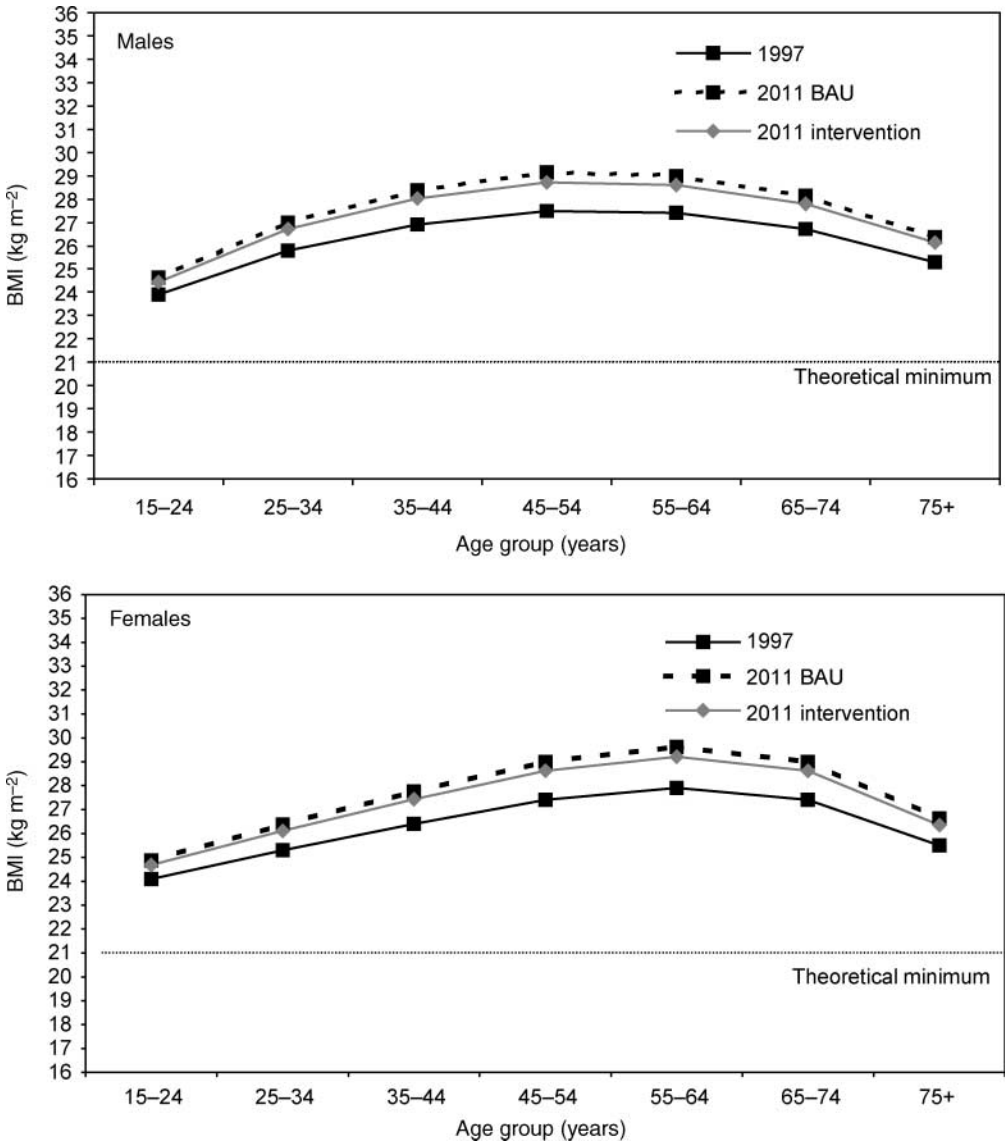

Fig. 1 Current and projected body mass index (BMI) distribution (including business as usual (BAU) and intervention scenarios, and theoretical minimum)

https://doi.org/10.1079/PHN2004704 Published online by Cambridge University Press

by approximately a quarter, corresponding to an increase of 1.0 kg m^{-2} (0.8–1.2 kg m^{-2} depending on age) in both males and females by 2011. This increase in BMI would mean an average 19% further shift away from the theoretical minimum (Fig. 1) and would lead to a mean population BMI of 27.2 kg m^{-2} in males and 27.1 kg m^{-2} in females.

Disease outcomes

Age-specific risk reductions associated with a 1 kg m^{-2} reduction in BMI ranged from 4 to 13% for IHD and stroke, from 17 to 32% for diabetes, and averaged 3% for cancer (Table 1). For IHD, stroke and diabetes, steeper associations with BMI were apparent in younger age groups (25–44 years) but there was no apparent effect of age on the association between BMI and cancer.

Attributable mortality

Higher-than-optimal BMI (>21 kg m^{-2}) contributed to 1561 (95% confidence interval 1243–1852) IHD deaths, 367 (174–517) ischaemic stroke deaths, 1231 (1208–1247) diabetes deaths, 177 (156–197) colorectal cancer deaths and 91 (76–106) postmenopausal breast cancer deaths in 1997 (Table 2). Overall, 'high' BMI contributed to a total of 3154 deaths (11% of all deaths) and 37 373 YLL (13% of all YLL). Proportionally, this amounted to 83% of diabetes deaths, 24% of IHD deaths, 15% of ischaemic stroke deaths and 4% of all cancer deaths (Fig. 2).

The majority (70–80%) of IHD, ischaemic stroke, diabetes and cancer deaths attributable to higher-than-optimal BMI occurred in those aged 65 years and older. Age-standardised mortality rates attributable to higher-than-optimal BMI were about twofold higher in males than in females for IHD (78/100 000 vs. 37/100 000 deaths), slightly higher in males than in females for diabetes (53/100 000 vs. 45/100 000), and similar for males and females for ischaemic stroke (11/100 000 vs. 12/100 000 deaths) and colorectal cancer (7/100 000 vs. 6/100 000) (Fig. 3).

Table 1 Risk reduction (%) associated with a 1 kg m^{-2} reduction in body mass index

	Age group (years)					
Disease	25–34	35–44	45–54	55–64	65–74	75+
Ischaemic heart disease	12	11	9	7	5	4
Ischaemic stroke	13	12	10	8	6	4
Diabetes (males)	26	26	19	17	18	21
Diabetes (females)	32	32	25	21	17	17
Colorectal cancer	3	3	3	3	3	3
Postmenopausal breast cancer			3	3	3	3

Table 2 Attributable mortality for body mass index, 1997

Disease outcome	Deaths (count)	YLL (count)
Ischaemic heart disease	1561	17 910
Ischaemic stroke	367	3284
Diabetes	1231	16 535
Colorectal cancer	177	2082
Postmenopausal breast cancer	91	1190
Total*	3154 (880–2595)†	37 373 (30 702–43 191)†

YLL – years of life lost.
* Adjusts for estimated overlap between diabetes and cardiovascular disease mortality.
† 95% confidence interval.

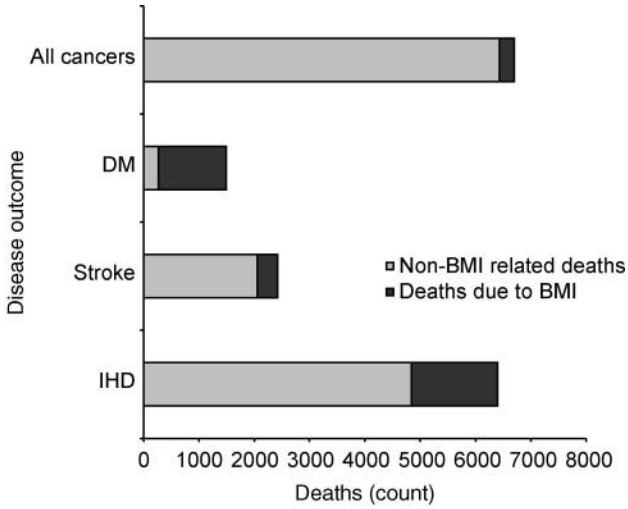

Fig. 2 Proportion of deaths due to higher-than-optimal body mass index (BMI) in 1997. DM – type 2 diabetes mellitus; Stroke – ischaemic stroke; IHD – ischaemic heart disease

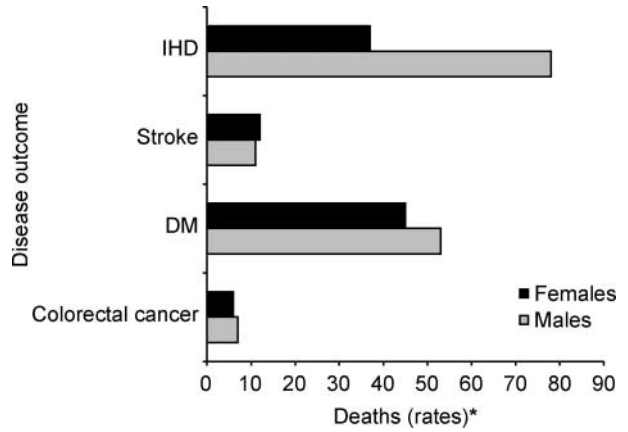

Fig. 3 Mortality rates due to higher-than-optimal body mass index, by sex, in 1997. IHD – ischaemic heart disease; Stroke – ischaemic stroke; DM – type 2 diabetes mellitus. *Rate per 100 000, age-standardised to World Health Organization world population

https://doi.org/10.1079/PHN2004704 Published online by Cambridge University Press

Avoidable mortality

If projected increases in BMI were limited to those outlined under the intervention scenario, it was estimated that 85 IHD deaths, 25 ischaemic stroke deaths, 323 diabetes deaths, 13 colorectal deaths and eight postmenopausal breast cancer deaths could be avoided each year from 2011 relative to the number anticipated under the BAU scenario (Table 3). Overall, a total of 385 deaths and 4951 YLL could be avoided each year. As would be expected, more mortality would be avoided in the older age groups and in men.

Discussion

We estimate that in 1997 higher-than-optimal BMI ($>21\,kg\,m^{-2}$) contributed to 3154 deaths or 11% of all deaths in New Zealand. Eighty-three per cent of diabetes deaths were attributable to excess body weight. A small reduction in the projected increase in mean population BMI modelled under the intervention scenario ($0.3\,kg\,m^{-2}$ lesser increase) indicated that effective policy changes could prevent approximately 385 deaths each year in New Zealand from 2011, of which approximately two-thirds represent diabetes deaths.

This is the first study to provide reliable estimates of the mortality burden due to higher-than-optimal BMI in New Zealand. However, the study does have some limitations, including the restriction to fatal outcomes. Inclusion of both fatal and non-fatal outcomes in an integrated measure such as the DALY would more adequately represent the population's level of health by recognising the importance of widely prevalent, severely disabling conditions. Additionally, BMI as an anthropometric measure does not distinguish between weight associated with lean body mass and that associated with fat. This may lead to an overestimation of the burden of disease attributable to high BMI in some population subgroups in New Zealand who have a higher proportion of lean body mass[29].

It has been estimated that globally the proportion of disease burden attributable to higher-than-optimal BMI is 21% for IHD, 23% for ischaemic stroke, 58% for type 2 diabetes, 12% for colon cancer and 8% for postmenopausal breast cancer[3]. The Australian burden of disease study[30,31] found that the burden of disease attributable to overweight and obesity was 40% of cardiovascular diseases (IHD, ischaemic stroke) and hypertension together, 28% of diabetes and 14% of cancers. In comparison, our New Zealand estimates found a lower proportion of stroke attributable to high BMI (15%) and a higher proportion of diabetes (83%). However, there were important differences between the various studies that may account for these differences. The global and Australian estimates were based upon both fatal and non-fatal outcomes, whereas the New Zealand estimates were based only on mortality data, indicating that the inclusion of non-fatal outcomes reduces the relative burden of disease attributable to type 2 diabetes but increases the relative burden attributable to other outcomes such as ischaemic stroke and cancers. In addition, the Australian study used a higher theoretical minimum for BMI ($25\,kg\,m^{-2}$) than the New Zealand study ($21\,kg\,m^{-2}$), and also arbitrarily halved published relative risks for overweight and obesity to account for confounding by other risk factors[30]. The overall burden of disease attributable to high BMI has been estimated to be 2–4% of total DALYs[19,31,32]. It is notable, however, that across developed regions high BMI has been estimated to account for up to 7.4% of DALYs[19].

The theoretical minimum BMI used in this study was $21\,kg\,m^{-2}$, which is substantially lower than traditional cut-offs used for overweight and obesity[2]. Had a higher BMI threshold been used, estimates of attributable mortality would have been lower and estimates of avoidable mortality would have been higher. However, current evidence provides no clear epidemiological basis for specific cut-points to define overweight. Categorisation of overweight suggests that those in the 'normal' BMI category do not have any increased risk of disease, whereas to date several analyses[8,18] have shown that a considerable proportion of BMI-attributable events occur well below $25\,kg\,m^{-2}$. As is generally seen, many more events arise from the 'moderate' middle of the distribution than the 'high-risk' tail[33], and therefore, at a population level, the most appropriate focus should be on mean BMI rather than on proportions of the population above arbitrary thresholds.

Nutrition-related risk factors rank highly in causes of death in New Zealand. Only tobacco consumption, responsible for 5000 (18%) deaths in New Zealand in 1997[34], ranks higher than high blood cholesterol (17% deaths), high systolic blood pressure (13% deaths) and high BMI (11% of deaths)[20]. While tobacco consumption, blood cholesterol levels and systolic blood pressure levels have all been declining in the population, BMI is increasing rapidly. Thus, the ranking may be very different in the future.

Table 3 Avoidable mortality* for body mass index, 2011

Disease outcome	Deaths (count)	YLL (count)
Ischaemic heart disease	85	957
Ischaemic stroke	25	227
Diabetes	323	4535
Colorectal cancer	13	127
Postmenopausal breast cancer	8	100
Total†	385	4951

YLL – years of life lost.
* Due to a projected $1.0\,kg\,m^{-2}$ increase in mean body mass index, rather than the projected $1.3\,kg\,m^{-2}$ increase under the business as usual scenario.
† Adjusted for the reporting overlap between diabetes and cardiovascular deaths.

https://doi.org/10.1079/PHN2004704 Published online by Cambridge University Press

Excess body weight is caused by an energy imbalance where energy intake in the form of food and beverages is greater than energy expenditure. Traditional methods of weight loss focus on reducing energy intake through low-fat or calorie-controlled diets, increasing energy expenditure via an increase in physical activity, or a combination of these. Epidemiological surveys suggest that intentional weight loss can reduce the mortality of overweight individuals towards that of the general population[35,36], while studies of surgical treatment have shown a dramatic reduction in the incidence of diabetes, hypertension, hyperinsulinaemia and hypertriglyceridaemia[37]. However, the long-term effectiveness of these weight-reduction methods is limited[38,39], with an overall pattern of moderate weight loss followed by gradual weight regain.

It is unclear whether the current epidemic of obesity is due primarily to population increases in food consumption or decreases in exercise or a combination of both[40]. Modification of food consumption patterns is difficult. Barriers to healthy eating include social, environmental and behavioural issues[2] and major influences on food consumption include taste, cost, convenience, nutrition, and weight-control concerns[41]. Single interventions are therefore unlikely to have a large impact on population food consumption and, as such, a range of national policies and approaches is necessary to influence food supply and purchase patterns. Such policies could include subsidies or other incentives to produce and purchase more nutritious foods, guidelines for the nutrient content of foods including fast food and takeaway meals, or working with industry to achieve more appropriate portion sizes and advertising practices of certain foods. Changes to encourage increased physical activity could include provision of safe activity and recreational areas, and incentives to walk, cycle, use active forms of public transport or participate in active recreational pursuits. Such potential interventions could be implemented as part of the New Zealand strategy for a more integrated approach to addressing nutrition, physical activity and obesity[42].

In conclusion, our results quantify the importance of higher-than-optimal BMI as a major cause of premature death in New Zealand. Intervention policies that would have only modest effects on slowing the rate of increase in population BMI by 2011 could still prevent hundreds of deaths annually.

Disclaimer

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

References

1 James PT, Leach R, Kalamara E, Shayeghi M. The worldwide obesity epidemic. *Obesity Research* 2001; **9**(Suppl. 4): 228S–33S.

2 World Health Organization (WHO). *Obesity: Preventing and Managing the Global Epidemic*. Geneva: WHO, 2000.

3 James WPT, Jackson-Leach R, Ni Mhurchu C, Kalamara E, Shayeghi M, Rigby NJ, *et al*. Overweight and obesity (high body mass index). In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors*. Geneva: World Health Organization, 2004; 497–596.

4 Flegal KM, Carroll MD, Ogden LG, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *Journal of the American Medical Association* 2002; **288**: 1723–7.

5 Department of Health. *Health Survey for England: Trend Data for Adults 1993–2001*. London: Department of Health, 2003.

6 Du S, Lu B, Zhai F, Popkin BM. A new stage of the nutrition transition in China. *Public Health Nutrition* 2002; **5**(1A): 169–74.

7 Wilson BD, Wilson NC, Russell DG. Obesity and body fat distribution in the New Zealand population. *New Zealand Medical Journal* 2001; **114**: 127–30.

8 Ni Mhurchu C, Rodgers A, Pan WH, Gu DF, Woodward M, on behalf of the Asia Pacific Cohort Studies Collaboration. Body mass index and cardiovascular disease in the Asia–Pacific region: an overview of 33 cohorts involving 310,000 participants. *International Journal of Epidemiology* 2004; **33**: 751–8.

9 Whitlock G, Lewington S, Ni Mhurchu C. Coronary heart disease and body mass index: a systematic review of the evidence from large prospective cohort studies. *Seminars in Vascular Medicine* 2002; **2**(4): 369–81.

10 Dyer AR, Elliott P, Shipley M, Stamler R, Stamler J, on behalf of the INTERSALT Cooperative Research Group. Body mass index and associations of sodium and potassium with blood pressure in INTERSALT. *Hypertension* 1994; **23**(1): 729–36.

11 Denke MA, Sempos CT, Grundy SM. Excess body weight. An underrecognized contributor to high blood cholesterol levels in white American men. *Archives of Internal Medicine* 1993; **153**: 1093–103.

12 Denke MA, Sempos CT, Grundy SM. Excess body weight. An under-recognized contributor to dyslipidemia in white American women. *Archives of Internal Medicine* 1994; **154**: 401–10.

13 Grinker JA, Tucker KL, Vokonas PS, Rush D. Changes in patterns of fatness in adult men in relation to serum indices of cardiovascular risk: the Normative Aging Study. *International Journal of Obesity and Related Metabolic Disorders* 2000; **24**: 1369–78.

14 Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care* 1994; **17**(9): 961–9.

15 Carey VJ, Walters EE, Colditz GA, Solomon CG, Willett WC, Rosner BA, *et al*. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. The Nurses' Health Study. *American Journal of Epidemiology* 1997; **145**: 614–9.

16 Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of US adults. *New England Journal of Medicine* 2003; **348**(17): 1625–38.

17 Bergstrom A, Pisani P, Tenet V, Wolk A, Adami HO. Overweight as an avoidable cause of cancer in Europe. *International Journal of Cancer* 2001; **91**: 421–30.

18 Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. *New England Journal of Medicine* 1999; **341**(6): 427–34.

19 Ezzati MLA, Rodgers A, Vander Hoorn S, Murray CJL, and the Comparative Risk Assessment Collaborative Group. Selected major risk factors and global and regional burden of disease. *Lancet* 2002; **360**: 1347–60.
20 Ministry of Health and University of Auckland. *Nutrition and the Burden of Disease: New Zealand 1997–2011*. Wellington: Ministry of Health, 2003.
21 World Health Organization (WHO). *Reducing Risks, Promoting Healthy Life. World Health Report 2002*. Geneva: WHO, 2002.
22 Russell DG, Parnell WR, Wilson NC, Faed J, Ferguson E, Herbison P, *et al. NZ Food: NZ People. Key Results of the 1997 National Nutrition Survey*. Wellington: Ministry of Health, 1999.
23 Clayton D, Schifflers E. Models for temporal variation in cancer rates. II: Age–period–cohort models. *Statistics in Medicine* 1987; **6**: 469–81.
24 Ministry of Health. *Cancer in New Zealand: Trends and Projections*. Wellington: Ministry of Health, 2002.
25 Murray CJ, Lopez AD. *The Global Burden of Disease*. Boston, MA: Harvard University Press, 1996.
26 Ministry of Health. *Modelling Stroke: A Multi-state Life Table Model*. Wellington: Ministry of Health, 2002.
27 Birkbeck JA. *New Zealanders and Their Diet: A Report to the National Heart Foundation of New Zealand on the National Diet Survey 1977*. Auckland: National Heart Foundation, 1979.
28 Russell DG, Wilson NC. *Life in New Zealand Survey.Vol. V: Health*. Dunedin: University of Otago, 1991.
29 Swinburn B. Using the body mass index: weigh then weigh up. *New Zealand Medical Journal* 1998; **111**(1075): 377–9.
30 Mathers CD, Vos T, Stevenson C. *The Burden of Disease and Injury in Australia*. Canberra: Australian Institute of Health and Welfare, 1999.
31 Mathers CD, Vos ET, Stevenson CE, Begg SJ. The Australian burden of disease study: measuring the loss of health from diseases, injuries and risk factors. *Medical Journal of Australia* 2000; **172**: 592–6.
32 Diderichsen F, Dahlgren G, Vagero D. *Determinants of the Burden of Disease in the European Union*. Stockholm: National Institute of Public Health, 1997.
33 Rose G. Sick individuals and sick populations. *International Journal of Epidemiology* 1985; **14**: 32–8.
34 Ministry of Health. *The Burden of Disease and Injury in New Zealand*. Wellington: Ministry of Health, 2001; 1.
35 Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40–64 years. *American Journal of Epidemiology* 1995; **141**: 1128–41.
36 Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in overweight white men aged 40–64 years. *American Journal of Epidemiology* 1999; **149**: 491–503.
37 Sjostrom CD, Lissner L, Wedel H, Sjostrom L. Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS intervention study. *Obesity Research* 1999; **7**(5): 477–84.
38 Glenny AM, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature. *International Journal of Obesity and Related Metabolic Disorders* 1997; **21**: 715–37.
39 Douketis JD, Feightner JW, Attia J, Feldman WF, with the Canadian Task Force on Preventive Health Care. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. *Canadian Medical Association Journal* 1999; **160**(4): 513–25.
40 Cutler DM, Glaeser EL, Shapiro JM. *Why Have Americans Become More Obese?* Cambridge, MA: Harvard University, 2003.
41 Glanz K, Basil M, Maibach E, Goldberg J, Snyder D. Why Americans eat what they do: taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. *Journal of the American Dietetic Association* 1998; **98**(10): 1118–26.
42 Ministry of Health. *Healthy Eating – Healthy Action: oranga kai – oranga pumau: A Strategic Framework*. Wellington: Ministry of Health, 2003.

https://doi.org/10.1079/PHN2004704 Published online by Cambridge University Press