Health gains, health inequality impacts, and health system cost savings – associated with modelled reductions in type 2 diabetes incidence

July 2020

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Contents

Abstract

Background: People with type 2 diabetes have increased morbidity and substantially decreased life expectancy. Approximately 5.5% of New Zealand (NZ) adults have been diagnosed with diabetes.

Methods: Theoretical reductions in type 2 diabetes, in 10% increments, compared to business-as-usual were modelled through to health-adjusted life years (HALYs) gained, health system costs in NZ$ 2011 and increases in life expectancy using an established multi-state life-table model.

Results: Health-adjusted life years gained, health system cost savings and average life expectancy all increase linearly with increasing reductions in type 2 diabetes incidence. The majority of this gain comes from the impacts of type 2 diabetes directly, but a proportion also comes from the impact of type 2 diabetes on coronary heart disease and stroke incidence. For a theoretical reduction of 10% (quite plausible to achieve with known interventions) average life expectancy was estimated to increase by 0.37 years, 150,000 HALYs were gained and over NZ$ 3 billion dollars were saved in health system costs (all over the remaining lives of the population alive in 2011 and undiscounted). Potential benefits for reducing health inequities were suggested by the per capita health gains being 1.71 times higher in Māori than non-Māori or 2.22 times higher when an equity analysis was applied.

Conclusions: This modelling provides additional justification from a health gain, health inequities, and health cost savings perspectives, for the NZ Government to further invest in effective interventions to reduce the incidence of type 2 diabetes in NZ.

Acknowledgements

The authors thank the Ministry of Business, Innovation and Employment (MBIE) for funding support for this work (Grant UOOX1406). They also thank the Health Research Council of New Zealand for supporting the BODE^3 DIET Model development (Grants 10/248 and 16/443). They also thank Counties Manukau DHB for providing support at the early stages of this work in terms of highlighting the importance of diabetes control from a DHB perspective.

Introduction

Type 2 diabetes mellitus is the most common type of diabetes. It is estimated that worldwide in 2014, 422 million people had diabetes (an age-standardised prevalence of 8.5% of adults), the majority of whom had type 2 diabetes (World Health Organization, 2016). In 2013-2014 it has been estimated that 198,000 to 241,381 New Zealand adults (around 5.5% of those aged 15 years and over) had diagnosed diabetes (Ministry of Health, 2014). However, diabetes prevalence is not distributed evenly across the population. Men are more likely than women to have diabetes. Māori and Asian New Zealanders are approximately twice as likely as non-Māori, or non-Asian New Zealanders (respectively) to be diagnosed with diabetes, while Pacific New Zealanders are almost three times as likely as non-Pacific New Zealanders to have been diagnosed with diabetes (after adjustment for age and sex) (Ministry of Health, 2014). Furthermore, people living in more deprived neighbourhoods are almost twice as likely to be diagnosed with diabetes as those living in less deprived neighbourhoods (after adjustment for age, sex and ethnic group) (Ministry of Health, 2014).

Several risk factors for type 2 diabetes have been identified, including: obesity, poor diet, physical inactivity, advancing age, family history of diabetes, ethnicity, and high blood glucose during pregnancy affecting the foetus (International Diabetes Federation, 2013). “In 2012 there were 1.5 million deaths worldwide directly caused by diabetes” (p.21)(World Health Organization, 2016). “The total burden of deaths from high blood glucose in 2012 has been estimated to amount to 3.7 million. This number includes 1.5 million diabetes deaths, and an additional 2.2 million deaths from cardiovascular diseases, chronic kidney disease, and tuberculosis related to higher-than-optimal blood glucose” (pp.21 & 23)(World Health Organization, 2016). People with type 2 diabetes have a substantially decreased life expectancy (UK Prospective Diabetes Group, 1991, World Health Organization, 2016). Of this 3.7 million deaths, 43% are in people aged <70 years (World Health Organization, 2016). People with type 2 diabetes also have a high incidence of complications, for example they have an approximately two-fold risk of coronary heart disease and ischaemic stroke (after adjustment for age, sex, smoking status, body-mass index (BMI), and systolic blood pressure (The Emerging Risk Factors Collaboration, 2010). In New Zealand, diabetes accounted for 3% of all illness, disability and premature mortality in 2006 (Ministry of Health, 2013). Diabetes is one of the main causes of blindness, kidney failure and lower extremity amputation, all of which have substantial healthcare costs (UK Prospective Diabetes Group, 1991). It has been estimated that globally in 2013 more than US$ 548 billion was spent treating diabetes and its complications (International Diabetes Federation, 2013). In New Zealand it has been estimated that healthcare expenditure for people with type 2 diabetes was NZ$ 526 million in 2006 (Lal et al., 2012).

Research has shown that it is possible to reduce type 2 diabetes incidence rates through health sector interventions such as the diabetes prevention programme (DPP). The DPP showed a 34% reduction in type 2 diabetes incidence in the lifestyle intervention group and 18% reduction in the metformin group compared to the placebo group, 10 years after randomisation (Diabetes Prevention Program Research Group, 2009). Large health gains and health system cost savings are likely with this kind of reduction in population incidence rates. This report outlines these potential

impacts for a range of theoretical reductions in type 2 diabetes incidence in the New Zealand population alive in 2011.

Methods

OVERVIEW

Main outputs from this modelling were incremental health gain in HALYs and health system costs in 2011 New Zealand dollars (NZ$) between the theoretical reductions in type 2 diabetes incidence modelled and business-as-usual (BAU). Both health gain and costs were undiscounted. This modelling takes a health system perspective, and involved a theoretical intervention so no intervention costs were included. Benefits and costs were modelled over a lifetime horizon for the whole New Zealand adult population alive in 2011.

A DIET multi-state life-table model (MSLT) was built from an established tobacco control MSLT model (using many of the same diseases), from which we have published work previously (Blakely et al., 2015, Pearson et al., 2016, Van der Deen FS, 2017, Cleghorn et al., 2018). This BODE^3 DIET MSLT model has itself already being used for studying a number of dietary interventions (Cleghorn C et al., 2018, Cleghorn et al., 2019, Cleghorn et al., 2020, Drew et al., 2020). The DIET MSLT model is described further in an online technical report (Cleghorn et al., 2017).

The DIET MSLT model that was used for this modelling is structured as a main life-table with projected all-cause mortality and morbidity rates by sex and age for Māori and non-Māori. The model has 17 diet-related diseases running in parallel (i.e., type 2 diabetes, coronary heart disease (CHD), stroke, osteoarthritis and multiple cancers: endometrial, kidney, liver, oesophageal, pancreatic, thyroid, colorectal, breast, ovarian and gallbladder). The disease state of type 2 diabetes itself, was also included as an independent risk factor for increased risk of CHD and stroke. For this project all diseases except type 2 diabetes, CHD and stroke were 'switched off' in the model.

BUSINESS-AS-USUAL (BAU) INPUT PARAMETERS

All input parameters, specified by sex, age and ethnicity unless stated differently, are shown in Table 1. Incidence, prevalence and case-fatality rates in 2011 are included for each disease. Morbidity was quantified for each disease. This was calculated as prevalent years of life lived with disability (YLDs) from the New Zealand Burden of Disease Study (BDS), divided by the population count.

Individually-linked data for publicly-funded (and some privately-funded) health events occurring in 2006-10 was used to calculate sex and age specific health system costs in 2011 NZ$. These costs included hospitalisations, inpatient procedures, outpatients, pharmaceuticals, laboratories and expected primary care usage. Costs that were assigned in the model fell into the following three categories. Firstly, sex and age-specific annual cost of a citizen who does not have a diet-related disease and is not in the last six months of their life. Secondly, disease-specific excess costs for people in the first year of diagnosis, last six months of life if dying of the given disease, and otherwise prevalent cases of each disease in the model. Lastly, the costs associated with the last six months of life if dying from a disease not in the model.

MODEL STRUCTURE

Life-table analysis

Life-tables are at the centre of the BODE^3 DIET MSLT model, both an overall life-table and multiple disease 'state' life-tables that are mathematically linked to the main life-table. In the baseline or BAU model, the New Zealand population is projected out into the future through all-cause and disease-specific expected trends in incidence, case-fatality and mortality. The contribution of the New Zealand diet to these trends is not *explicitly* modelled in the BAU model.

The population is divided into five-year age group cohorts (from age 0-4 to age 105-109), modelled as four separate sex by ethnic (Māori, non-Māori) populations, and simulated in the life-table until death (or age 110).

The model is a *proportional multi-state life-table model(Blakely T, 2020 (in press))*. This basically means that:

- Everyone still alive in each cycle of the model (more specifically, the alive proportion for whichever five-year cohort is currently being modelled) is represented in the main life-table. In this main life-table, age-specific all-cause mortality and morbidity rates are applied in each cycle to the 'alive cohort', until the age of 110 years when all remaining alive people are assumed to die. As such, the sum of HALYs can be tallied.

- In parallel, proportions of the cohort can simultaneously reside in one or more parallel disease-specific life-tables or states. Or put more correctly, multiple disease states are modelled independently.[1] Within these disease-specific life-tables, disease incidence rates, remission and case-fatality rates, and disease-specific morbidity (disability weights from the New Zealand Burden of Disease Study (BDS) (Ministry of Health, 2013) and the Global Burden of Disease (GBD) Study (Salomon et al., 2012)), and disease-specific costs, are modelled.

- The disease-specific life-tables have both a BAU and intervention model. The latter intervention model differs from the BAU model, in that incidence rates are changed (usually lowered) based on population impact fractions (PIFs; a 'merging' of changes in risk factor distributions and relative risks). This allows a calculation of differences in disease-specific mortality and morbidity rates, and differences in disease-costs per capita.

- These differences are then summed across all parallel disease states, and added or subtracted to the all-cause mortality and morbidity rates in the main life-table and captured as cost differences between BAU and intervention, allowing estimation of HALYs gained (or lost) and health system cost change between the BAU and intervention scenarios for the population overall – the main objective of the modelling.

Diet-related disease models

Diseases are modelled, within each disease process or parallel disease state as above, using a set of differential equations that describe the transition of people between four states (healthy, diseased, dead from a disease in the model, and dead from all other causes), with transition of people

[1] With the exception of diabetes, which has been 'linked' to coronary heart disease and stroke states (see page 7 for details).

between the four states based on rates of background mortality, incidence, case-fatality and remission (Figure 1).

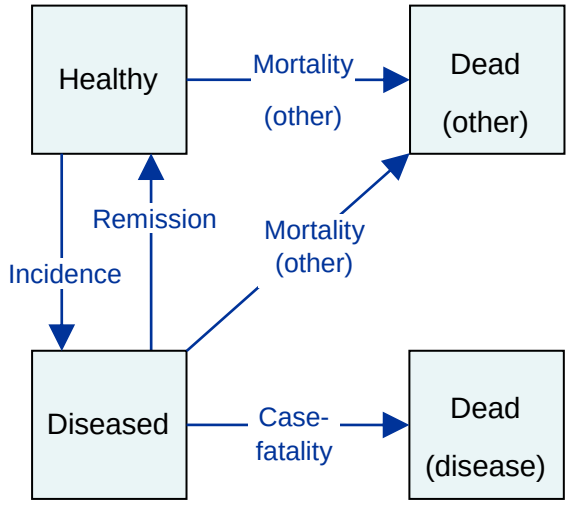

Figure 1: Each disease is modelled with four states (healthy, diseased, dead from the disease, and dead from all other causes) and transition probabilities between states of incidence, remission, case-fatality and mortality from all other causes.

The default model structure was that diseases were modelled independently. Specifically, the sex-, age- and ethnic-specific incidence, remission, and case-fatality rates for each disease were modelled independently. However, we include dependency for type 2 diabetes as a disease state, essentially treating it both as a disease state and a risk factor itself for coronary heart disease and stroke.

Type 2 diabetes: both a disease and a risk factor

The DIET MSLT model typically has key independence assumptions, including:

1. *Risk factor distribution:* the distributions of each risk factor can be treated as though independent of other risk factors.
2. *Disease incidence rates:* the incidence rate for a given disease (e.g. CHD) is independent of other diseases (e.g. the presence of type 2 diabetes).
3. *Disease case-fatality and remission rates*: the rates for a given disease (e.g. CHD) are independent of those for other diseases (e.g. type 2 diabetes).

The second assumption is the focus here, for type 2 diabetes. Type 2 diabetes is associated with increased rates of CHD and stroke, be it by shared common causes (i.e. confounding) or cause and effect (the concern here). Whether to address such 'dependency' depends on what one is doing with the model, through what risk factors. In the case of this modelling which directly changes incidence of diabetes without going through a change in risk factor the links between risk factor and disease are not directly relevant but the following description illustrates the justification for the model structure.

For the BODE^3 DIET MSLT model, interventions that change BMI and thence disease incidence are important. Figure 2 gives the standard structure. BMI is independently associated with each of CHD and type 2 diabetes, and change (Δ) in the BMI distribution combined with the relative risk for the BMI→CHD and BMI→ type 2 diabetes association to give PIF results in a change in both disease incidence rates. The change in mortality, morbidity and cost rates that result are then 'added' to the overall mortality, morbidity and cost rates in the main life-table.

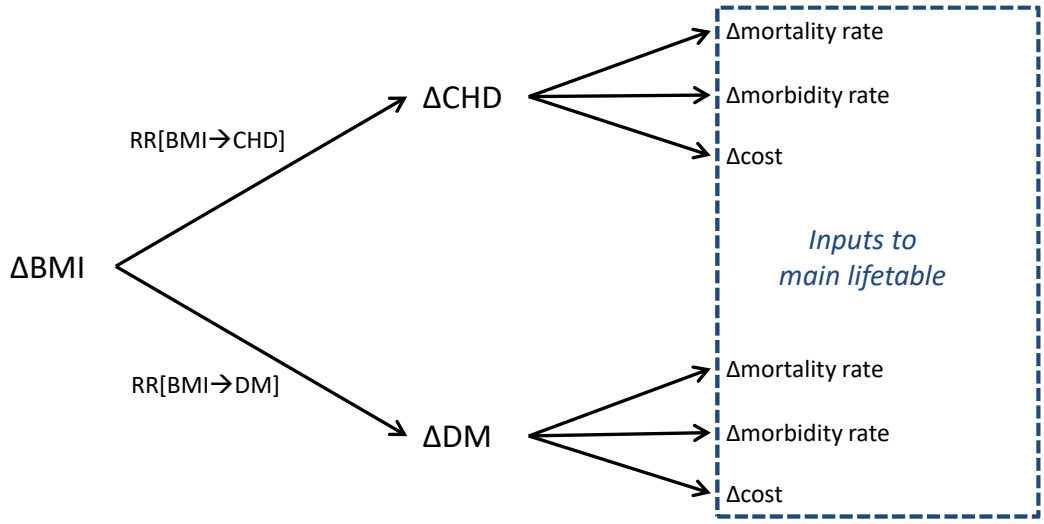

Figure 2: Standard structure in the BODE^3 DIET MSLT for BMI as risk factors and CHD and type 2 diabetes states (RR = relative risk; DM: type 2 diabetes)

A modelled intervention that lowers BMI may result in an overestimation of health gain (in HALYs) if the reduction in type 2 diabetes and CHD 'double-count' the gains when considered independently. But if only the 'pure type 2 diabetes' mortality rate (e.g. based on the deaths coded as type 2 diabetes) is estimated in the type 2 diabetes state, and the higher than average population mortality rate otherwise (e.g. due to people with type 2 diabetes having higher CHD and stroke mortality) is not allowed for, the prevalence of type 2 diabetes will drift too high over time as the total mortality rate modelled for people with diabetes is not high enough. This over-estimated morbidity rate, in turn, may lead to an overestimate of morbidity gains due to a BMI lowering intervention. (And likewise an overestimate of costs savings as costs are a function of prevalence.)

One solution to this dependency problem is a microsimulation model, where each individual's other disease status is 'known'. But for the BODE^3 DIET MSLT model, the partial solution we use is to restructure and re-parameterise the model.

Figure 3 below gives that structure. The changes are:

- To 'link' the type 2 diabetes state to the CHD state (and stroke state; not shown), such that:
    - type 2 diabetes becomes a risk factor for CHD, linked through a RR that is adjusted for BMI (which is now a confounder of the type 2 diabetes →CHD association). Specifically, a change in the type 2 diabetes prevalence changes CHD incidence through a PIF link.
    - The RR for the BMI → CHD association is now the 'direct effect' (VanderWeele, 2015), i.e. that not through type 2 diabetes.

    - The outputs from the CHD state that input to the MSLT remain unchanged in structure.
- The mortality rate output from the type 2 diabetes to the main life-table in the MSLT is:
    - 'just' the mortality rate due to deaths coded as type 2 diabetes in the default model. This use of a case-fatality rate due to type 2 diabetes-coded deaths only is likely an underestimate of the death due to type 2 diabetes. However, the CHD and stroke excess deaths are explicitly modelled through the PIF link from the type 2 diabetes prevalence to CHD and stroke incidence.
    - given the uncertainty above in the default model, as a scenario analysis we model excess mortality among people with type 2 diabetes from having type 2 diabetes, excluding CHD and stroke mortality as that is quantified in, and outputted from, the CHD (and stroke) states instead of the type 2 diabetes-only case-fatality rate above. This will probably overestimate the mortality due to type 2 diabetes, but does give an upper limit.
- But to 'allow' for the higher mortality rate among people with diabetes, a 'total excess' mortality rate (mort[all-cause| type 2 diabetes] – mort[all-cause], where the former is the all-cause mortality rate among people with diabetes, and the latter is the all-cause mortality rate in the general population without type 2 diabetes) is applied within the type 2 diabetes state as an absorbing state. This mortality is only used to 'allow simulated people to die' in the model to allow for dependent mortality risk; without this higher mortality rate taking people out of the alive type 2 diabetes population, the prevalence would drift too high (impacting on costs and morbidity).

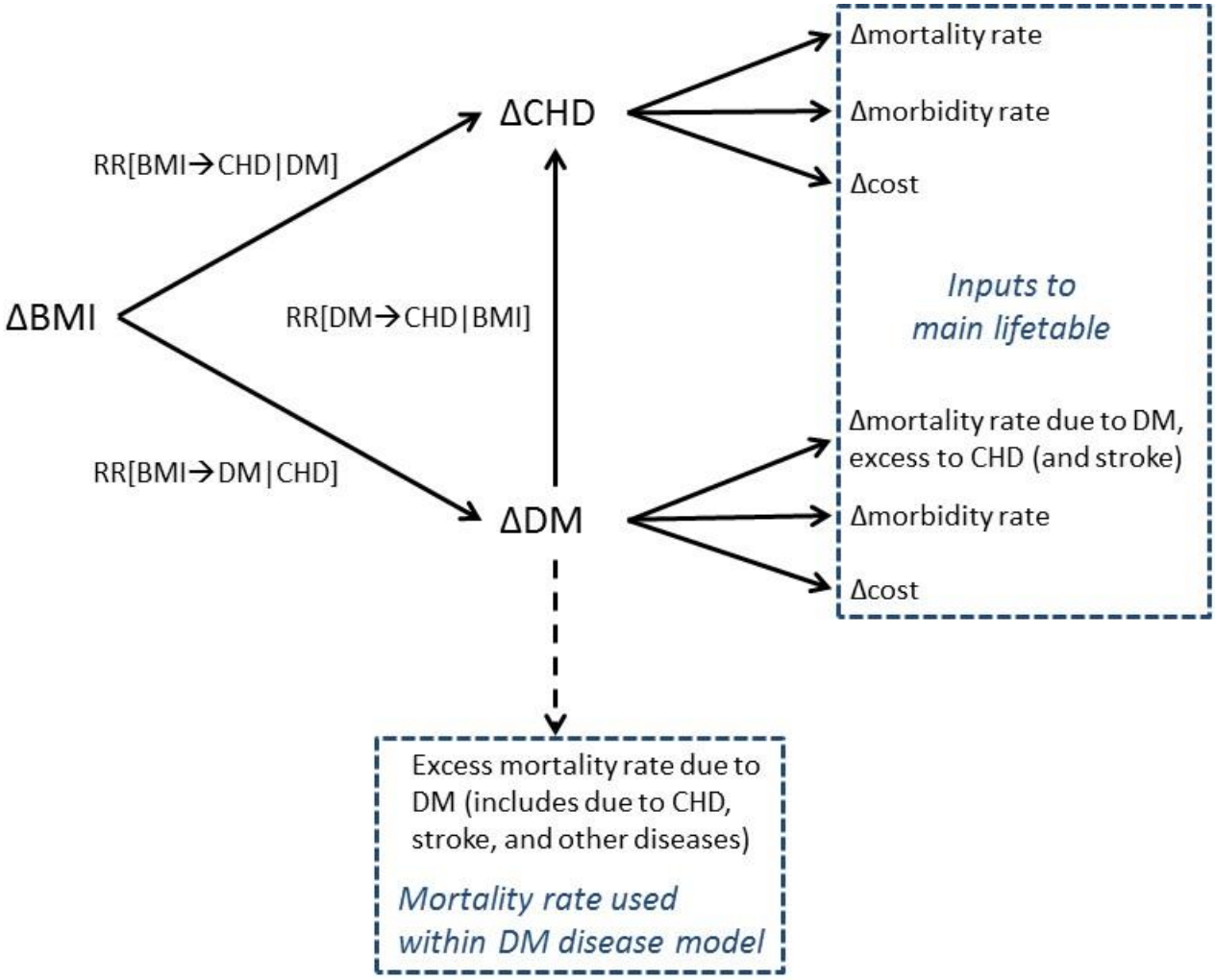

Figure 3: Altered structure in the BODE^3 DIET MSLT to allow for dependency of CHD (and stroke – not shown) on type 2 diabetes (DM)

Figure 4 gives an alternate depiction of the mortality 'stack'. The total of all components is the total mortality rate among people with diabetes. 'C', 'S' and 'O' are, respectively, the excess CHD, excess stroke, and excess non-CHD non-stroke mortality among people with type 2 diabetes compared to the general population, and 'O' is partitioned again into type 2 diabetes coded deaths and non- type 2 diabetes coded deaths.

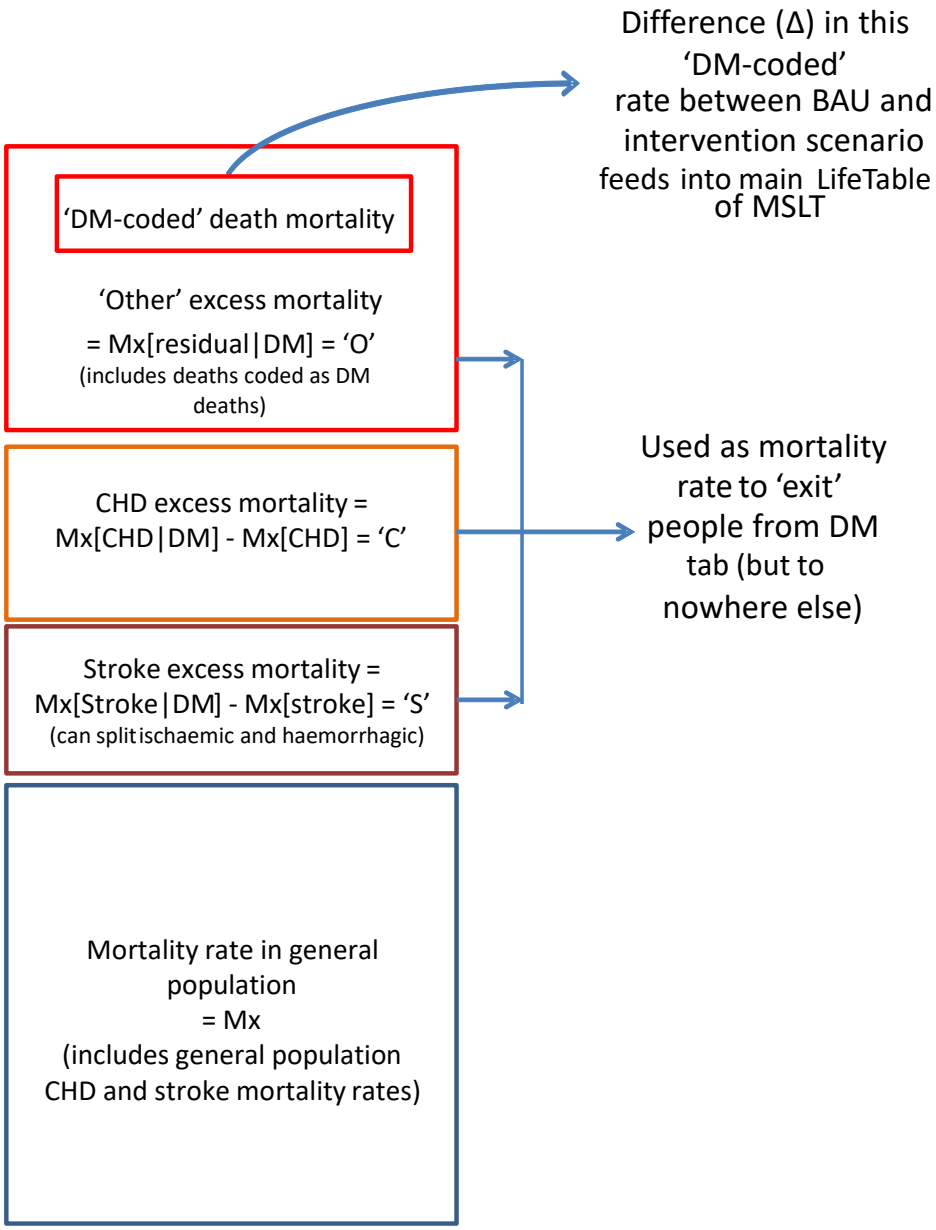

Figure 4: Partitioning of total mortality rate among people with type 2 diabetes (DM) into components relevant to BODE^3 DIET MSLT structure

The above structure (Figure 3 and Figure 4) and parametrisation is an improvement for a disease like type 2 diabetes. However, it is still not optimal modelling (which would require a much more resource-intensive micro-simulation model).

Table 1 Baseline input parameter table used in modelling theoretical changes in type 2 diabetes incidence

Baseline input Parameter	Source and application to model	Expected Value and 95% UI	Distribution
Baseline population count	Statistics NZ (SNZ) population estimates for 2011.	Nil uncertainty.	
All-cause mortality rates	SNZ mortality rates for 2011.	Nil uncertainty.	
Disease-specific incidence, prevalence, and case-fatality rates (and remission rates)	For type 2 diabetes, coherent sets of incidence rates, prevalence, case-fatality rates (CFR), (remission rates set to zero) were estimated using DISMOD II using data from NZBDS, HealthTracker and the Ministry of Health.	Uncertainty: rates all +/- 5% standard deviation (SD).	Log-normal
Disease trends	Trends are applied to incidence (CHD and Stroke: -2%, type 2 diabetes: +3%) and case-fatality (CHD and Stroke: -2%, type 2 diabetes: -3%). These are switched on until 2026 and then kept constant for the remainder of the lifetime.	Uncertainty: +/- 0.5% absolute change. Type 2 diabetes: Uncertainty +/- 1.5% absolute change.	Normal
Total morbidity per capita in 2011	The per capita rate of years of life lived with disability (YLD) from the NZBDS.	Uncertainty: +/- 10% SD.	Log-normal
Disease morbidity rate per capita	Type 2 diabetes was assigned a disability rate (DR; by sex and age) equal to the YLDs (scaled down to adjust for comorbidities) from the 2006 NZBDS projected forward to 2011, divided by the disease prevalence. This DR was assigned to the proportion of the cohort in the type 2 diabetes tab.	Uncertainty: +/- 10% SD.	Normal
Health system costs	Linked health data (hospitalisations, inpatient procedures, outpatients, pharmaceuticals, laboratories, and expected primary care usage) for each individual in NZ for the period 2006–2010 had unit costs assigned to each event, and then five health system costs (2011 NZ$) were estimated.	Estimated at SD = ±10% of the point estimate.	Gamma

MODELLING AND ANALYSIS

An Ersatz add-in (Barendregt, 2012) to Microsoft Excel was used to incorporate parameter uncertainty and run the multiple sex by age by ethnic group cohorts through the model 2000 times each. Each iteration involved a random draw from the probability density function about the Table 1 parameters, specified with uncertainty. The main results produced by the model were incremental HALYs gained and net health system costs. Results for the base case are presented for the total population and by sex and ethnicity (Māori and non-Māori).

SCENARIO AND SENSITIVITY ANALYSES

Māori have higher background mortality and morbidity, resulting in a lesser 'envelope' for potential health gains which disadvantages Māori in the analysis. Therefore, an additional equity analysis whereby non-Māori all-cause mortality and population morbidity rates were used for Māori (McLeod et al., 2014) (Table 3).

A scenario analysis for a 10% decrease in type 2 diabetes incidence was modelled which used the excess mortality among people with type 2 diabetes from having type 2 diabetes. This differs from the base case modelling which uses the case-fatality rate due to type 2 diabetes coded deaths only (see page 8 for more detail).

Results

The modelling results indicated that health-adjusted life years gained (HALYs; Figure 5), health system cost savings (Figure 6) and average life expectancy in the whole population (Figure 7) all increased linearly with increasing reductions in type 2 diabetes incidence being modelled. The majority of this gain came from the impacts of type 2 diabetes directly but a modest proportion (approximately 14% for life expectancy, 6% for HALYs and 1% for health system costs) came from the impact of type 2 diabetes on CHD and stroke incidence.

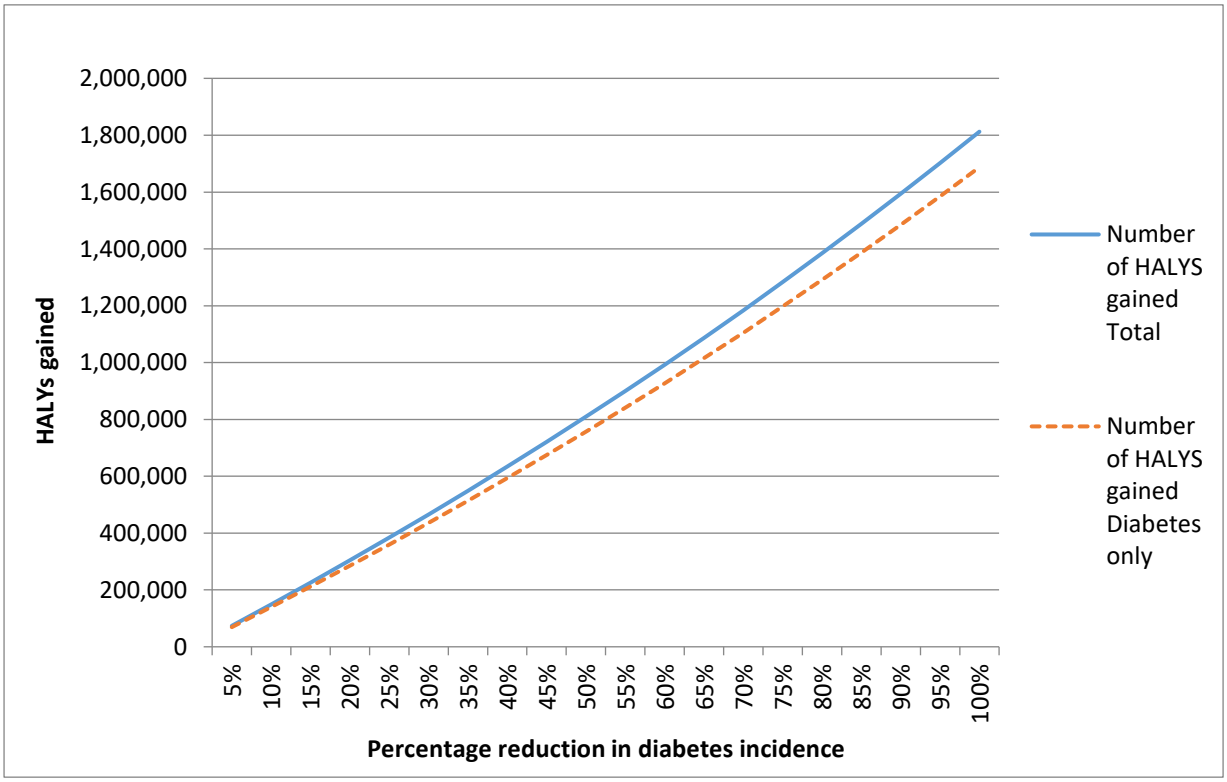

Figure 5: Total number of HALYs gained over the lifetime of the cohort with percentage reductions in type 2 diabetes incidence

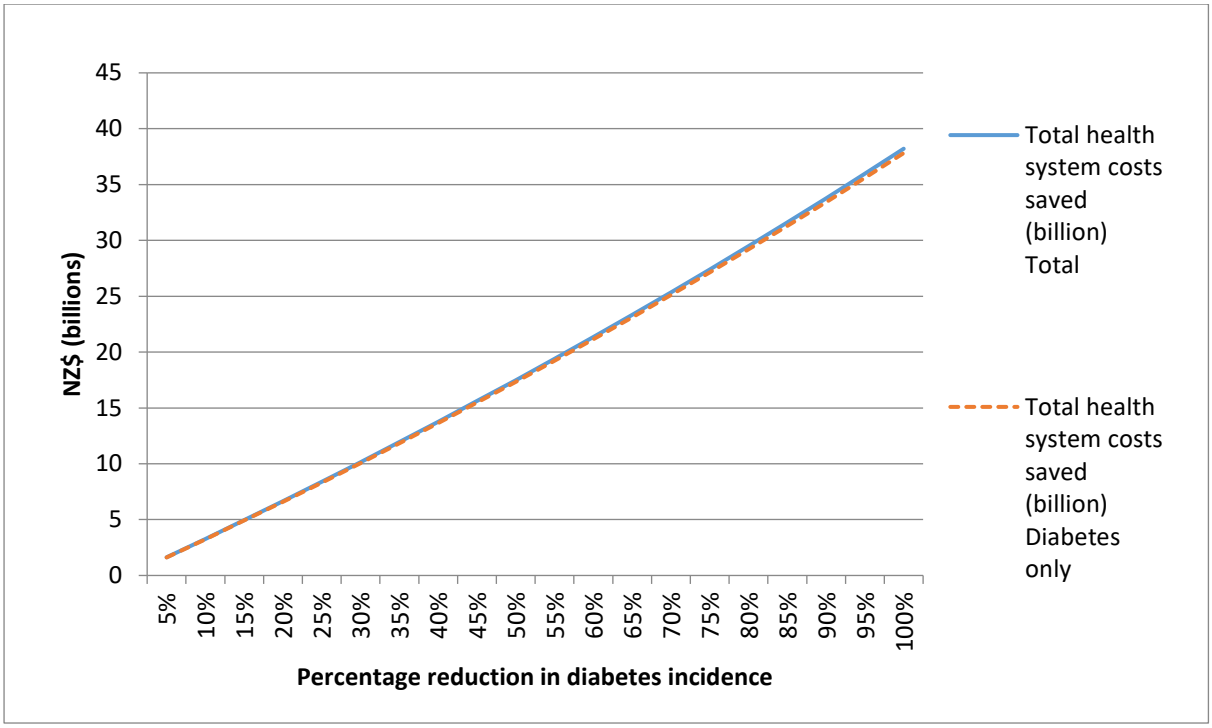

Figure 6: Total health system cost savings over the lifetime of the cohort with percentage reductions in type 2 diabetes incidence

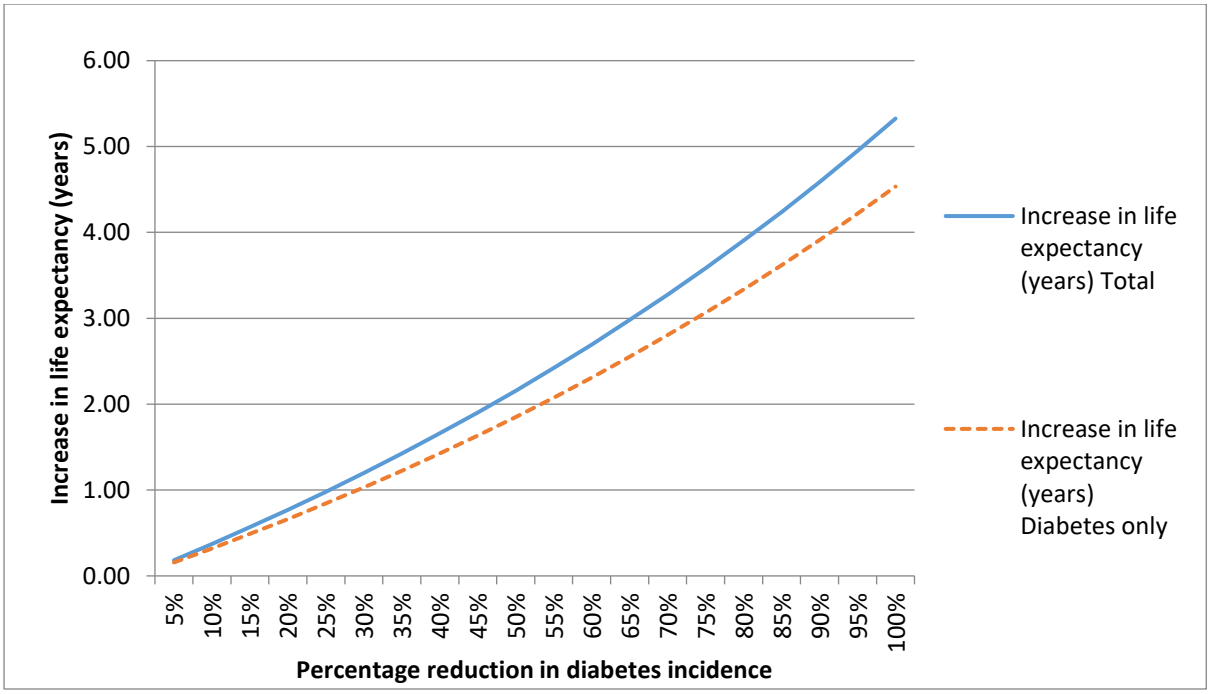

Figure 7: Increases in average life expectancy per New Zealander over the lifetime of the cohort with percentage reductions in type 2 diabetes incidence

If new cases of type 2 diabetes were completely eliminated from New Zealand in 2011 (e.g., via a theoretical new treatment or a vaccine), there would be an increase of over 5 years in average life expectancy, 1.8 million HALYs gained and over $38 billion saved in health system costs over the lifetime of the cohort (Table 2). For a more realistic reduction of 10% in incidence these numbers would be 0.37 years in average life expectancy (4.4 months), 150,000 HALYs gained and over $3 billion saved in health system costs.

HALYs gained were similar between males and females with the majority of the gain in 25 to 64 year old non-Māori and 25 to 44 years in Māori (Table 3). Health system cost savings were highest in those who were 25 to 64 year olds in 2011.

Per capita HALYs gained were 1.71 times higher in Māori (52.3 per 1000 people) than non-Māori (30.5). The 'equity analysis' presented in Table 3 shows an 29% increase in HALYs gained when non-Māori background mortality and morbidity were used (so as to "value" potential health gains from preventing diseases similarly between Māori and non-Māori (McLeod et al., 2014)). Health gains per capita were 2.22 times greater for Māori than non-Māori when this equity analysis was applied.

In the scenario analysis for a 10% decrease in type 2 diabetes incidence, when the case fatality rate used for type 2 diabetes was changed from type 2 diabetes coded deaths only to excess mortality among people with type 2 diabetes from having type 2 diabetes HALYs increased to 177,000, and cost savings to the health system to 3.1 billion.

Table 2 Increase in life expectancy, total number of HALYs gained and health system cost savings over the lifetime of the cohort with percentage reductions in type 2 diabetes incidence

Diabetes incidence reduction (%):	**5%**	**10%**	**15%**	**20%**	**25%**	**30%**	**35%**	**40%**	**45%**	**50%**
Increase in life expectancy (years) per average person										
Total	0.18	0.37	0.57	0.77	0.98	1.20	1.42	1.66	1.90	2.16
Diabetes only	0.16	0.32	0.49	0.66	0.84	1.03	1.22	1.43	1.63	1.85
Number of HALYS gained (whole population)										
Total	73,800	149,000	226,000	304,000	384,000	466,000	549,000	634,000	721,000	809,000
Diabetes only	69,200	139,700	212,000	285,000	360,000	436,000	514,000	593,000	674,000	757,000
Total health system costs saved (NZ$ billion) (whole population)										
Total	$ 1.63	$ 3.28	$ 4.96	$ 6.66	$ 8.39	$ 10.20	$ 11.90	$ 13.80	$ 15.60	$ 17.50
Diabetes only	$ 1.61	$ 3.25	$ 4.91	$ 6.6	$ 8.32	$ 10.06	$ 11.84	$ 13.64	$ 15.47	$ 17.33
Diabetes incidence reduction (%):	**55%**	**60%**	**65%**	**70%**	**75%**	**80%**	**85%**	**90%**	**95%**	**100%**
Increase in life expectancy (years) per average person										
Total	2.42	2.69	2.98	3.27	3.58	3.90	4.24	4.59	4.95	5.33
Diabetes only	2.08	2.31	2.55	2.80	3.06	3.34	3.62	3.91	4.22	4.53
Number of HALYS gained (whole population)										
Total	900,000	993,000	1,090,000	1,180,000	1,280,000	1,380,000	1,490,000	1,590,000	1,700,000	1,810,000
Diabetes only	841,000	928,000	1,020,000	1,110,000	1,200,000	1,290,000	1,390,000	1,480,000	1,590,000	1,690,000
Total health system costs saved (NZ$ billion) (whole population)										
Total	$ 19.40	$ 21.30	$ 23.30	$ 25.30	$ 27.40	$ 29.50	$ 31.60	$ 33.80	$ 36.00	$ 38.20
Diabetes only	$ 19.22	$ 21.15	$ 23.11	$ 25.1	$ 27.13	$ 29.19	$ 31.29	$ 33.43	$ 35.6	$ 37.82

Table 3 Health gain (in HALYs gained) and health system costs saved from a 10% decrease in type 2 diabetes incidence among the New Zealand population alive in 2011

	Non-Maori	Māori		Ethnic groups combined	
Sex and age (in 2011)	**HALYs**	**HALYs**	**HALYs – equity†**	**HALYs**	**Net cost savings (NZ$ billion)**
Sex and age groups combined	114,000 (93,000 to 138,000)	35,300 (30,100 to 40,800)	45,600 (39,000 to 52,700)	149,000 (123,000 to 179,000)	$3.27 ($2.61 to $4.08)
Males					
0-14 year olds	7,320 (5,910 to 8,970)	4,360 (3,720 to 5,060)	5,500 (4,720 to 6,370)	11,700 (9,600 to 14,000)	$0.28 ($0.22 to $0.35)
15-24 year olds	8,140 (6,580 to 9,960)	3,430 (2,930 to 3,960)	4,330 (3,710 to 5,000)	11,600 (9,500 to 13,900)	$0.28 ($0.22 to $0.35)
25-44 year olds	19,600 (16,000 to 23,700)	5,520 (4,750 to 6,340)	7,110 (6,140 to 8,140)	25,100 (20,700 to 30,000)	$0.60 ($0.48 to $0.74)
45-64 year olds	18,200 (15,000 to 21,800)	3,350 (2,910 to 3,800)	4,600 (4,010 to 5,250)	21,500 (17,900 to 25,500)	$0.48 ($0.38 to $0.59)
65+ year olds	3,170 (2,640 to 3,750)	300 (260 to 340)	450 (400 to 520)	3,470 (2,900 to 4,090)	$0.07 ($0.05 to $0.08)
All ages	56,400 (46,100 to 68,000)	17,000 (14,600 to 19,500)	22,000 (19,000 to 25,300)	73,300 (60,700 to 87,500)	$1.7 ($1.36 to $2.11)
Females					
0-14 year olds	7,000 (5,620 to 8,620)	4,360 (3,650 to 5,150)	5,410 (4,560 to 6,370)	11,400 (9,300 to 13,800)	$0.26 ($0.21 to $0.33)
15-24 year olds	7,510 (6,050 to 9,210)	3,440 (2,890 to 4,040)	4,280 (3,620 to 5,020)	10,900 (8,900 to 13,300)	$0.25 ($0.20 to $0.31)
25-44 year olds	19,500 (15,800 to 23,700)	6,170 (5,220 to 7,200)	7,910 (6,730 to 9,210)	25,700 (21,000 to 30,900)	$0.55 ($0.44 to $0.69)
45-64 year olds	19,600 (16,000 to 23,600)	3,940 (3,360 to 4,560)	5,420 (4,640 to 6,290)	23,600 (19,400 to 28,100)	$0.44 ($0.36 to $0.55)
65+ year olds	3,870 (3,220 to 4,590)	390 (330 to 450)	580 (500 to 680)	4,260 (3,550 to 5,030)	$0.07 ($0.05 to $0.08)
All ages	57,500 (46,700 to 69,700)	18,300 (15,500 to 21,400)	23,600 (20,100 to 27,600)	75,800 (62,300 to 91,200)	$1.57 ($1.25 to $1.96)
Per capita (HALYs/1000 people & $)	30.5 (24.9 to 36.9)	52.3 (44.6 to 60.5)	67.6 (57.9 to 78.2)	33.9 (28. to 40.5)	$0.74 (0.59 to 0.93)

† Māori "HALYs—Equity" are calculated using non-Māori background mortality and morbidity rates so as not to "penalise" Māori because of worse background mortality and morbidity.

Discussion

MAIN FINDINGS AND INTERPRETATION

This modelling work suggests that increasing reductions in the incidence of type 2 diabetes results in a linear increase in HALYs gained, health system cost savings and average life expectancy. The majority of this gain comes from the impacts of type 2 diabetes directly but a modest proportion of the health gains also come from a reduction in CHD and stroke incidence. Very large health gain and health system cost savings are possible through a reduction in type 2 diabetes incidence – up to a 5 year average increase in life expectancy, 1.8 million HALYs gained and over 38 billion dollars saved in health system costs over the lifetime of the cohort. But smaller reductions in diabetes incidence, of the magnitude likely to be seen by an effective national diabetes prevention programme, are still substantial and cost saving to the New Zealand health system. For example, the internationally famous Diabetes Prevention Programme (see *Introduction*) found a reduction of 34% in the incidence of type 2 diabetes in the lifestyle intervention group 10 years after randomisation compared to the placebo group (Diabetes Prevention Program Research Group, 2009). When applying this to New Zealand, a 35% reduction in incidence was estimated to increase average life expectancy by 1.42 years, gain approximately 550,000 HALYs and produce NZ$ 12 billion cost savings to the New Zealand health system.

Per capita health gains were higher for Māori than non-Māori, especially when the 'equity analysis' was applied. This suggests that if an effective intervention was equally effective in reducing diabetes incidence for Māori then this intervention could contribute to a reduction in ethnic health inequities in New Zealand.

STUDY STRENGTHS AND LIMITATIONS

This Report outlines the modelling of a theoretical reduction in type 2 diabetes incidence and the impact that it might have. It therefore does not include any specific practical ways of decreasing incidence rates and does not include any costs of policies or interventions. As such, the Report is intended to be used in conjunction with evidence of interventions that reduce diabetes incidence, to illustrate their potential in New Zealand in terms of potential health gains and cost savings.

The base year for demographic, epidemiological and costing specification is 2011, with trends out to 2026 – as per other evaluations in the Burden of Disease Epidemiology, Equity and Cost-Effectiveness (BODE^3) Programme from which this evaluation arises. This allows useful comparisons with other interventions. It was beyond the scope of this evaluation to update the entire model to a more recent base-year such as 2018. Had this been done, we anticipate the total health gain in HALYs would have increased slightly due to population growth and ongoing high obesity rates, but the general pattern of findings would change little.

POTENTIAL IMPLICATIONS FOR RESEARCH AND HEALTH AGENCIES

This Report clearly indicates the potentially large health gains, favourable impacts on health inequities, and health system cost savings that are possible through reducing the incidence of type 2 diabetes in the New Zealand population. This provides a strong justification to establish effective programmes for reducing type 2 diabetes and shows the maximum cost the programme can be for overall cost savings to remain. For example, if the evidence of a particular intervention shows that it will reduce the incidence of type 2 diabetes by 10% and will cost 100 million NZ$ to implement, the New Zealand Government could be confident that overall long-term cost savings would occur as a reduction of this size is estimated to save over NZ$ 3 billion in health system costs (albeit spread out over the life-time of the cohort and without discounting). Nevertheless, there are also other diabetes prevention interventions that would cost relatively little (e.g., the few million dollars to pass a law in New Zealand(Wilson et al., 2012) such as a sugary drinks tax or a junk food tax(Blakely et al., 2020). Increasing physical activity levels will also assist with preventing diabetes and there is evidence for this activity levels being modifiable according to a systematic review e.g., "improving neighbourhood walkability, quality of parks and playgrounds, and providing adequate active transport infrastructure is likely to generate positive impacts on activity in children and adults"(Smith et al., 2017). Such interventions can also have favourable benefit to cost ratios according to another systematic review(Brown et al., 2016).

CONCLUSIONS

This modelling provides additional justification from a health gain, health inequities, and health cost savings perspectives, for the New Zealand Government to further invest in effective interventions to reduce the incidence of type 2 diabetes in the country. Fortunately, a range of proven interventions exist which include the specific (e.g., the Diabetes Prevention Programme) and also those which change the obesogenic environment (e.g., sugary drink taxes and walking/cycling infrastructure).

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