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SUMMARIES

Health and equity impacts of climate change in Aotearoa-New Zealand, and health gains from climate action [Special Article]

Hayley Bennett, Rhys Jones, Gay Keating, Alistair Woodward, Simon Hales, Scott Metcalfe

Climate change and related environmental changes (like hotter temperatures, heavy rainfall and drought) create many risks to health. These risks are recognised by health experts and leading health organisations all around the world. If climate change continues unchecked, parts of the world will be unsuitable to live in by the end of this century. New Zealanders will face serious health threats, especially those people who already experience poorer health. However well planned action to address climate change can reduce these threats, and can also improve health and save money in the health sector.

Alcohol intake, marijuana use, and sleep deprivation on the risk of falls occurring at home among young and middle-aged adults: a case-crossover study

Simon Thornley, Bridget Kool, Roger J Marshall, Shanthi Ameratunga

This study investigated whether hospitalised fall-related injuries among young and middle-aged adults were associated with short term effects of alcohol intake, marijuana use and sleep deprivation. The study found that the estimated risk of injury was substantially higher after consuming alcohol within the preceding 6 hours. There was no significantly elevated risk of fall-related injury associated with sleep deprivation (<6 hours sleep in the preceding 24 hours), or marijuana use in the preceding 3 hours.

Relationship between fructose and lactose intakes and functional gastrointestinal symptoms in a sample of 50-year-old Cantabrians in New Zealand

Robin Spencer, Richard Gearry, John Pearson, Paula Skidmore

The role that sugars play in human health is an area of considerable research interest. Therefore we were interested in the role that fructose and lactose play in irritable bowel syndrome symptoms. In a study of 227 fifty year olds from Canterbury we found that the main food sources of fructose were soft drinks, fruit juice and fruit, and for lactose the main food sources were milk and yoghurt. We found that those people who consumed more lactose and fructose experienced less pain from IBS symptoms. It is possible that those who experience IBS pain may reduce their intake of fructose and lactose rich foods, in order to reduce pain but further research is needed to see if this is the case.

Self-monitoring blood glucose test strip use with diabetes medicines in people with types 1 and 2 diabetes in New Zealand

Scott Metcalfe, Peter Moodie, Hew Norris, Dilky Rasiah

Self-Monitoring of Blood Glucose (SMBG) test strips help people with diabetes measure and maintain the concentration of glucose in their blood when using diabetes medicines. Should a patient's glucose levels drop too low from diabetes treatment, it could result in a medical emergency. Likewise, if their glucose levels remain too high despite diabetes treatments, they risk long-term harm from their diabetes. Glucose test strips provide people using diabetes medicines a quick and easy way to test their blood sugar levels themselves at any time. The study compared patients' uptake of publicly funded test strips in New Zealand, compared with published guidance on appropriate rates of usage. The study indicated that in the 2011 year, there was both under and over-dispensing of test strips to different groups of patients. Whilst some under and over-use of test strips may be appropriate to patients' own clinical circumstances, persisting patterns of unsuitable use are cause for concern.

Better adherence to published guidelines for SMBG may improve health outcomes and reduce pain, anxiety and disruption to patients.

Evaluation of a rural primary referred cardiac exercise tolerance test service
Katharina Blattner, Garry Nixon, Carol Horgan, Jayne Coutts, Marara Rogers, Brandon Wong, John Wigglesworth, Gerard Wilkins

Rural residents have inferior access to diagnostic tests and usually have to travel long distances to the test site. We provided a local clinician-led Exercise Tolerance Test (ETT) service at two different rural hospitals (Rawene in Northland and Dunstan in Central Otago) over 12 months. ETT was no more costly to the system overall when provided rurally and significantly less costly for the patient. A shift in resource for ETT from large institutions in cities to local rural health providers may help overcome some of the disparities in access to cardiac investigations faced by rural patients . This is particularly relevant for rural Maori.

Projecting future smoking prevalence to 2025 and beyond in New Zealand using smoking prevalence data from the 2013 Census
Frederieke S van der Deen, Takayoshi Ikeda, Linda Cobiac, Nick Wilson, Tony Blakely

The New Zealand Government has a smokefree nation goal for 2025. In this study, we have explored what the smoking prevalence amongst Māori and non-Māori men and women would be in the year 2025, assuming that the current level of tobacco control activities would not change (including no further tax increases after January 2014). We have defined this as a business-as-usual (BAU) scenario in the paper. Under these assumptions, it appears that the smokefree 2025 goal is not achieved by any population group. Further strengthening of current tobacco control measures (for example continued scheduling of tobacco tax increases each year) or introducing entire new policies (for example ones that would permit only very low levels of nicotine per cigarette) may be needed to achieve very low levels of smoking prevalence for all population groups in New Zealand.

Is New Zealand water fluoridation justified? [viewpoint]
Yindi Jiang, Lyndie A Foster Page, John McMillan, Karl Lyons, Jonathan Broadbent, Kate C Morgaine

CWF (community water fluoridation) reduces the risk of ill health; it reduces health inequalities; it does not coerce ordinary adults into leading healthy lives; it addresses the health of children; and it does not fully achieve the public health aim of not intervening without the consent of those affected.

Profits or people? The informative case of alcohol marketing [viewpoint]
Sally Casswell

Sophisticated marketing of alcohol recruits young people to drinking earlier and encourages heavier drinking. Marketing is effectively unregulated in New Zealand and, despite the evidence of an adverse impact on young people and a strong popular sentiment in favour of regulation, no significant policy change has been forthcoming. It is hard not to conclude the profits of the corporate producers of alcohol are given more weight than the health and wellbeing of the New Zealand population when these policy decisions are made.

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1406/6361

EDITORIAL

Tackling obesity: a call to action

Rachael McLean, Jim Mann

The prevalence of obesity continues to increase in New Zealand.The most recent New Zealand Health Survey indicates that 31% of all adults (48% of Māori and 68% of Pacific adults) and 11% of all children aged 2–14 years (19% of Māori and 27% of Pacific children) are obese.[1]

Two major reports outlining evidence based recommendations for interventions have been released in the past few months. In May 2014, the New Zealand Medical Association (NZMA) issued its policy briefing *Tackling Obesity*: an evidence based and highly readable summary of recommended measures to improve health and decrease obesity through environmental change and improvements in health literacy.[2]

In July 2014, Swinburn and colleagues from the University of Auckland published a report which contained recommendations for reducing obesity by improving food environments.The recommendations were based on ratings by an Expert Panel of independent public health experts and representatives from medical associations and non-governmental organisations (NGOs) who identified and prioritised interventions needed to improve food environments for health.[3] Their report suggests that New Zealand performs well in some areas (e.g. food labelling and monitoring of health status) but also identifies 34 further actions that should be undertaken.

Both these reports add to the growing stable of reports internationally which consistently call on governments to do more. It is generally accepted that governments around the world have not been sufficiently diligent with regard to the adoption of anti-obesity programmes.In particular, they have been reluctant to introduce measures which might be expected to influence the obesogenic environment, considered to be essential components in attempts to stem the tide of the obesity epidemic.[4] This failure has been associated with increasing obesity rates in most countries, and warnings of subsequent increases in non-communicable disease rates into the future.

Chronic non-communicable diseases are responsible for a major burden of morbidity around the world.The Global Burden of Disease Study indicates that non-communicable disease caused 34.5 million or 65.5% of deaths in 2010, with ischaemic heart disease and stroke ranked first and second, and diabetes 9th, (up from 15th in 1990).[5]

The four major groups of chronic disease (heart disease, cancer, diabetes and chronic lung disease) have common risk factors, notably tobacco use, unhealthy diets, physical inactivity and harmful use of alcohol.[6] The risks of coronary heart disease, diabetes, and some cancers (including postmenopausal breast and colorectal cancer) associated with obesity, inappropriate nutrition and physical inactivity appear to be mediated via common mechanistic pathways: growth factors (mainly insulin and IGF1) inflammation, and hormones (mainly oestrogen).

Despite these strong common aetiological relationships these chronic diseases do not show similar trends over time in all countries, nor do they follow dietary changes particularly closely.The involvement of different polygenic and environmental determinants involved in the complex aetiology of chronic diseases probably explains the different time lag between exposure to these risk factors and the emergence of clinical manifestations of these diseases.

The relatively short time frame between the emergence of the epidemic of obesity and the escalating rates of diabetes which followed, as well as the speed at which diabetes risk reduction occurs with weight loss and associated lifestyle changes, confirm the pivotal role of obesity and its associated abnormalities as a cause of type 2 diabetes. This has been clearly demonstrated in intervention studies such as the Diabetes Prevention Study (DPS) and the Diabetes Prevention Program (DPP) which found that within 2 years, modest weight loss associated with regular physical activity and dietary

changes aimed at reducing total and saturated fat and increasing dietary fibre, can appreciably reduce the risk of progression from impaired glucose tolerance to diabetes.[7,8] The relatively rapid remission of type 2 diabetes and weight loss observed following bariatric surgery provides further confirmation.[9]

Coronary heart disease on the other hand appears to be associated with a more complex interaction of risk factors and measures aimed at reducing atherogenesis (e.g. cholesterol reduction and obesity related risk factors) may require an appreciably longer time frame before benefit is apparent.[10] Evidence from intervention trials of dietary and lifestyle improvement suggests that it takes at least 2 years of intervention and follow up to demonstrate improvements in cardiovascular outcomes.[10]

It is generally assumed that many years elapse between the initiation of the carcinogenic process following exposure of susceptible cells to carcinogens and the development of truly neoplastic cells and clinical manifestations of cancer.[11] Thus any benefit of weight reduction in terms of reducing the risk of obesity related cancers is unlikely to accrue without a prolonged period of weight loss maintenance.Support for this proposal comes from an intervention trial of morbidly obesity patients who underwent bariatric surgery.After a median follow up period of more than ten years a significant reduction in risk of developing cancer emerged in women who had undergone surgery compared with controls.[12]

There is widespread recognition among health professionals of the need for individual and public health measures to halt and reverse the escalating rates of obesity. Increasingly developing countries which have neither the capacity nor the budget to manage epidemics of diabetes, coronary heart disease and cancer will also need to pay attention to developing public health programmes to combat a growing prevalence of obesity and obesity related disorders.However, monitoring obesity rates appears to have had relatively little impact in terms of influencing governments of the need for urgent action, and continues to be challenged in both the popular and peer reviewed literature.[13,14] This may be due to the notion that obesity is a ‘risk factor’ rather than a disease state.

Rather alarmingly the recent economic recession and election of more conservative governments have led to the withdrawal of some promising public health initiatives aimed at reducing rates of obesity.[15] Furthermore, obesity, inappropriate nutrition and physical inactivity are often painted as lifestyle choices and subject to individual responsibility rather than major chronic disease risk factors requiring a substantial public health response.

In New Zealand several public health interventions to decrease and prevent obesity have been labelled “nanny state”, and withdrawn on these grounds,[16]and in the United Kingdom many nutrition and physical activity related public health initiatives have been discontinued, while representatives from the food industry have been appointed to key advisory roles to develop a new approach to public health.[17]

The imminent launch of the Healthy Families New Zealand initiative in 10 communities around the country[18]will go some way to promote healthy lifestyles at a community level, but a more comprehensive nation-wide strategy is required if substantial change is to be achieved.

An impressive evidence base supports the case for the environmental and societal changes, which need to underpin policy aimed at reducing obesity and its associated co-morbidities.These require implementation, which transcends political ideology and is sustainable beyond electoral cycles.

The NZMA report recommends a range of measures: from individual interactions between health professionals and their patients through to government interventions targeted at improving nutrition environments in schools and communities, as well as changes to the way food is taxed and marketed.

Swinburn et al emphasise the importance of strong political leadership and a comprehensive plan underpinning specific initiatives.While the introduction of a broad range of interventions is justified, a number of specific interventions are highlighted in both reports, and should form the basis of immediate policy response to obesity in New Zealand.These include:

- Restricting the marketing and promotion of unhealthy food to children and adolescents
- Introducing (or re-introducing[15]) food and nutrition guidelines for provision of healthy foods to children in schools and early childhood education settings
- Improvements in food labelling so individuals are better able to improve their own nutrition.
- Fiscal measures to improve nutrition such as a tax on sugar sweetened beverages.

The first two of these measures aimed at children and adolescents should be implemented as a matter of priority.The introduction of the Health Star rating system has the potential to improve food labelling and enhance consumer understanding of the healthfulness of food products, but needs formal evaluation once implemented.Fiscal measures such as changes in taxation are controversial, but worthy of investigation into how effective they would be in a New Zealand context.

Health professionals have a unique and potentially powerful influence on public opinion and government policy. These reports are a call to action to all health professionals to consider the implications of the increasing prevalence of obesity into the medium to long-term future for the health of New Zealanders. We should all take up this challenge, and communicate more emphatically and effectively with government about the importance of implementing change at the environmental level to improve health.

Competing interests: Nil.

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EDITORIAL

Reducing childhood overweight and obesity in New Zealand through setting a clear and achievable target

Stefanie Vandevijvere, Boyd Swinburn

A government target to reduce New Zealand's very high and increasing rates of childhood overweight and obesity[1] is timely since rates in several other OECD countries are flattening or decreasing.[2–4] About one-third of New Zealand children are now overweight or obese[1] compared to about one in four in Australia.[5,6]

The purpose of this analysis is to consider the plausibility of options for New Zealand to reach Australia's current childhood prevalence rates by 2025 and, importantly, to reduce or not increase disparities in the process.

The most recent prevalence of childhood (2–14 years) overweight and obesity is 33% with it being significantly higher among Māori (44%) and Pacific (55%), than New Zealand (NZ) European (28%) and Asian (27%) children.[1] In order to achieve the target of the current Australian prevalence (25%) over the next 10 years 2015–2025, an average reduction in childhood overweight and obesity of 0.8% points per year will be needed. If all childhood ethnic groups were to achieve the target prevalence of 25% by 2025, in practice this would mean a reduction of 0.2–0.3% points per year for NZ European and Asian children, 1.9% points per year for Māori children and 3.0% points per year for Pacific children (scenario 1, Table 1).

Although this scenario is very desirable in order to reduce (even eliminate) both absolute and relative inequalities across ethnic groups, it does not seem feasible. Community-based programs focused on environmental and capacity building interventions have shown that reductions in overweight and obesity of 1.3% points per year are possible among disadvantaged white children in Australia.[7] However, the same approach taken with Pacific and Māori children in South Auckland showed no effect.[8]

If the rates of obesity and overweight among NZ European and Asian children would reduce by 1.3% points per year over the next 10 years, then the yearly reductions needed in overweight and obesity rates among Māori and Pacific children in order to achieve the overall target by 2025, would be between 0.2% and 0.4% points (scenario 2a and 2b, Table 1). However in view of the importance of reducing inequalities, this would not be the preferred scenario either. Other scenarios to achieve the 2025 target, without increasing absolute inequalities (scenario 3), or slightly decreasing absolute inequalities (scenarios 4 and 5) are shown in Table 1.

In order to not increase or reduce absolute inequalities, the %-point yearly reductions in overweight and obesity prevalence among Māori/Pacific children need to be at least as high as the %-pointy early reductions among NZ European/Asian children.

The New Zealand auditor general's 2013 report (Our future needs – is the public sector ready?[9]) revealed a clear decline in the Government's focus on obesity compared to the past. Improving unhealthy diets (New Zealand's number one risk factor[10]) or reducing childhood obesity currently do not figure as priorities in the latest Statement of Intent of the Minister for Health[11] or as part of the New Zealand annual Health targets[12] or 5-year public sector targets.[13]

Internationally, a wide range of countries, even emerging economies such as Brazil[14] and South Africa,[15] have included targets to reduce childhood obesity as part of their non-communicable diseases (NCDs) action plans.

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Table 1. Different scenarios to achieve the overall target of a prevalence of childhood (2–14 years) overweight and obesity of 25% by 2025 in New Zealand

			Scenario 1		Scenario 2a		Scenario 2b		Scenario 3		Scenario 4		Scenario 5	
population group	% overweight and obese 2013	% of total population of children	%point reduction py	2025 target	%point reduction py	2025 target	%point reduction py	2025 target	%point reduction py	2025 target	%point reduction py	2025 target	%point reduction py	2025 target
Māori	44.4	21.6	1.9	25.0	0.2	42.4	0.0	44.4	1.0	34.4	0.9	35.4	1.0	34.4
Pacific	55.3	11.1	3.0	25.0	0.0	55.3	0.4	51.3	1.0	45.3	1.0	45.3	0.9	46.3
Asian	27.0	8.4	0.2	25.0	1.3	14.0	1.3	14.0	1.0	17.0	0.9	18.0	0.9	18.0
NZ European/other	27.5	58.9	0.3	25.0	1.3	14.5	1.3	14.5	1.0	17.5	0.9	18.5	0.9	18.5
All				**25**		**25**		**25**		**24**		**25**		**25**

Scenario 1: All population groups reach the target of a childhood overweight and obesity prevalence of 25% by 2025 (out of the 5 scenarios the only scenario that reduces relative inequalities in overweight and obesity).
Scenario 2: NZ European and Asian children reduce overweight and obesity prevalence by 1.3% points per year (shown feasible in local communities in Australia), and either the childhood overweight and obesity rate among Pacific children (scenario 2a) or among Maori children (scenario 2b) is held constant.
Scenario 3: All population groups reduce the overweight and obesity prevalence among children by the same yearly rate in order not to increase absolute inequalities among ethnic groups. This gives an overall prevalence of overweight and obesity of 24% in 2025, which is a bit lower than the target of 25%.
Scenario 4-5: NZ European and Asian children reduce overweight and obesity prevalence by 0.9% points. If one of the other ethnic groups also reduces obesity and overweight prevalence by 0.9% points a year, the other group has to reduce obesity and overweight prevalence by 1.0% points a year to achieve the target.
py = per year.

The medical and health communities are also calling for a renewed focus on obesity. The New Zealand Medical Association's report on Tackling Obesity[16] sets out the top 10 priorities and a policy brief launched by the New Zealand Beverage Guidance Panel[17] identified the priorities for reducing sugar-sweetened beverage consumption. In addition, the healthy Food Environment Policy Index (Food-EPI),[18] recently launched by an international network of research groups and NGOs[19] was applied first in New Zealand in April 2014.

The Food-EPI Expert Panel of 52 New Zealand based public health and medical experts, performed a detailed review of the evidence of the extent of implementation of food policies by the New Zealand Government.[20] Major implementation gaps were found, clearly outweighing the good performance of the Government in some areas, such as regulation on health claims or monitoring of NCDs and their risk factors, including obesity.[20] The seven priority actions recommended by the Expert Panel included the need to set a target to reduce childhood overweight and obesity.

The analyses presented in Table 1 show the enormous challenge it will be for New Zealand to reach by 2025 where Australia is now and to reduce or at least not increase inequalities across ethnic groups at the same time. This suggests two broad approaches are needed: community-based interventions which prioritise those at risk populations and broader policy/regulatory approaches. Healthy Families NZ,[21] which is a systems-based approach to prevention at the community level, will be implemented in 10 high-need areas.

This will be an excellent opportunity to focus on Māori and Pacific populations and address some of the socio-cultural factors which appear to be barriers to obesity prevention.[22] However, given the lack of success in previous community prevention approaches in Pacific and Māori adolescents,[8,23,24]they should not be considered to be the single solution. Indeed there is a risk that educational interventions focused on information and knowledge will be more beneficial for individuals from higher socioeconomic backgrounds.[25] This might apply to the Health Star Rating system, recently approved by the Government for implementation in New Zealand, as well.

On the other hand, there are several policy/regulatory interventions aimed at making food environments healthier which have been shown to be cost-effective[26–30] and which are highly recommended by World Health Organization (WHO) and the New Zealand medical and public health community.

These approaches, which also have greater potential for producing equal or greater benefit among lower socioeconomic groups, are: restrictions of unhealthy foods in schools and early childhood settings, restrictions of unhealthy food marketing to children, government procurement policies which favour healthy rather than unhealthy foods, taxes on unhealthy foods, and food product reformulations. Several Australian studies[31,32] have shown that there is strong public support for the implementation of healthy food policies, such as restriction of food marketing to children and the reformulation of food products.

The WHO has established a high-level Commission on Ending Childhood Obesity[33] which is chaired by Professor Sir Peter Gluckman, New Zealand's Chief Science Advisor to the Prime Minister, with former Prime Minister Helen Clark as another of the distinguished members of the Commission.

This excellent international leadership would be strengthened by a serious attempt to address childhood obesity in New Zealand. The analyses presented here show that reducing overweight and obesity among Māori and Pacific children will be the greatest challenge and that both targeted community interventions and strong policy/regulatory actions on food environments will be needed to reach even very modest targets.

Competing interests: Nil.

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SPECIAL ARTICLE

Health and equity impacts of climate change in Aotearoa-New Zealand, and health gains from climate action

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Abstract

Human-caused climate change poses an increasingly serious and urgent threat to health and health equity. Under all the climate projections reported in the recent Intergovernmental Panel on Climate Change assessment, New Zealand will experience direct impacts, biologically mediated impacts, and socially mediated impacts on health. These will disproportionately affect populations that already experience disadvantage and poorer health.

Without rapid global action to reduce greenhouse gas emissions (particularly from fossil fuels), the world will breach its carbon budget and may experience high levels of warming (land temperatures on average 4–7°C higher by 2100). This level of climate change would threaten the habitability of some parts of the world because of extreme weather, limits on working outdoors, and severely reduced food production.

However, well-planned action to reduce greenhouse gas emissions could bring about substantial benefits to health, and help New Zealand tackle its costly burden of health inequity and chronic disease.

Human-caused climate change is a serious and increasingly urgent threat to human health and wellbeing.[1–5] Climate change will cause higher temperatures, extreme weather such as heatwaves, heavy rainfall events and/or drought, intense tropical storms and sea-level rise. It is projected that rising levels of carbon dioxide (CO_2) in the atmosphere will increase the acidity of the oceans by 150–200% by 2100. These changes result in many risks to human health that are recognised by world health and science authorities, New Zealand health bodies, and leading medical journals alike.[2–11]

Globally and in New Zealand, leading health threats include high temperatures and extreme events (direct impacts), changing patterns of infectious diseases and water/food shortages or price changes (biologically mediated impacts), and risks related to economic change, loss of livelihoods and forced migration (socially mediated impacts).[3,12–16]

Without rapid global action to reduce greenhouse gas emissions (particularly from fossil fuels), the world will breach its carbon budget and may experience high levels of warming (4–7°C or higher by 2100).[1,6,17,18] At such levels of warming the Intergovernmental Panel on Climate Change (IPCC) warns that normal human activities (e.g. growing food, working outdoors) will be increasingly compromised in some parts of the world during parts of the year; there will be large risks to global and regional food security; and higher risk of crossing 'tipping points' (thresholds for abrupt and irreversible change) in the earth and interlinked human systems.[6]

However, if well-planned action to reduce greenhouse gas (GHG) emissions were undertaken globally and in New Zealand, there could be substantial positive impacts not only for limiting future climate change, but also for health, equity, and wellbeing.[2–4,19]

This paper reflects the recent Fifth Assessment Report of the IPCC (AR5), and the increased urgency indicated for action to avoid worsening human health impacts from climate change. It also updates both Metcalfe et al's Special Article on climate change and health in the *Journal* in 2009,[12] and Phipps et al's paper on the climate change challenge for General Practice in New Zealand in the *Journal* in 2011.[13]

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1406/6366

Global health impact of climate change

Climate change is already contributing to global disease, disability and premature death—most seriously affecting people in poor countries, and the most disadvantaged and vulnerable within all countries.[2,6,7,20]

By the 2050s, the projected health impacts are extensive (summarised in Box 1).[2] Levels of risk will be influenced by population vulnerability (population health status, age, gender, health infrastructure) as well as the degree of social and economic development within populations during this timeframe.[2]

Box 1. Expected global health impacts with projected climate change to 2050[2]

Health Impact	IPCC Level of Confidence*
Higher risk of injury, disease and death from more intense heat waves and fires	Very high confidence
Higher risk of food- and water-borne diseases	Very high confidence
Higher risk of under-nutrition from lower food production in poor regions	High confidence
Health impacts related to lost work capacity/lower labour productivity in vulnerable populations	High confidence
Higher risk of vector-borne diseases in some areas	Medium confidence
Modest improvements in cold-related mortality and morbidity in some areas	Low confidence
Reduced capacity of disease-carrying vectors (from exceedance of thermal thresholds) in some areas	Medium confidence

* Confidence: IPCC qualitative assessment of evidence (type, amount, quality, consistency) and the agreement of evidence.

Box 1 includes some possible health gains from climate change (e.g. reduction in cold-related morbidity and mortality), but the IPCC has concluded that any positive effects from climate change will be outweighed globally by negative effects.[2,3]

It is important to note that many climate-health risk assessments to date remain conservative (based on lower-range warming scenarios of around 2°C) and consider relatively near-future timeframes (e.g. by 2030 or 2050).[20] However it is becoming increasingly likely that higher levels of warming may occur by 2100.[1,2,17,18] This would lead to environmental conditions (e.g. periods of extreme high temperatures; inability to raise food crops) that threaten human health and wellbeing in large parts of the world.[2,21] Under such scenarios, resources would become scarce and populations may be forced to migrate to other regions, creating risk factors for violence and conflict.[2,22]

Health impacts of climate change in Aotearoa-New Zealand

New Zealand is already experiencing climate change, and more change is expected.[23] According to the projections reported in the AR5, New Zealand will continue to warm over coming decades, and will be wetter in the west and drier in the east and north. Heavier and more frequent extreme rainfalls are expected (with increased flood risk), along with more drought, the duration of drought in the north and east is projected to at least double by 2040.[16,23,24]

There is expected to be more extreme heat (up to 60 more days >25°C in the north by 2090), with increased wild-fire risk. Some of these trends (e.g. increases in heavy precipitation) have already been observed.[16,23,24]

Table 1. Expected health impacts of climate change in New Zealand

Food security and nutrition: Increased global food prices, affecting a large number of locally produced and imported food staples in New Zealand, are likely to reduce the ability of some groups to afford a variety of nutritious foods, further compromising nutritional outcomes for those groups.[2,30–32]
Mental health and suicide: Increased stress and mental health issues (e.g. farmers with drought, victims of extreme weather). Young people may suffer anxieties about catastrophic climate change, not unlike those experienced by children growing up with the fear of nuclear war.[2,33–36]
Housing and health: Healthiness of some housing will be affected by extreme weather, for example, indoor moisture (with heavy rainfall, flooding), high indoor temperatures (during heatwaves in poorly insulated houses).[37] It is also likely that people will arrive in New Zealand from climate-change affected areas. This may put further pressure on availability of low income-larger family homes, potentially impacting household overcrowding and the incidence of some infectious diseases.[14,38,39]
Injury and illness from extreme weather events (e.g. flooding, storms, landslides, storm surges, drought): Immediate trauma, and indirect health impacts in weeks to months after extreme events (e.g. mental health problems, exacerbation of pre-existing medical conditions).[2,40–42]
Heat-related deaths and illness: Increases in heat-related deaths and illness, particularly for those with chronic illness and those aged over 65 years. Heat stress for outdoor workers. Winter deaths may decline, but this is uncertain as winter deaths may be influenced by seasonal factors that are unrelated to climate.[2,43–50]
Vector-borne and zoonotic (animal to human) disease: Increased likelihood that mosquito vectors could establish in New Zealand, which could lead to local transmission of mosquito-borne diseases (e.g. dengue, Ross River virus). Also possible impacts on other vector-borne diseases (e.g. tick-borne) and zoonotic diseases.[2,51–56]
Food- and water-borne disease: Heavy rainfall can lead to contamination of drinking and recreational water/shellfish with faecal pathogens from animals and humans. Both high and low rainfall, and higher temperatures may impact on bacterial and parasitic diseases causing gastroenteritis (e.g. giardiasis, salmonellosis). Dry conditions could affect continuity of household water supplies, impacting diseases influenced by hygiene.[2,56–59]
Ultraviolet (UV) radiation: Climate change may delay recovery of stratospheric ozone. Warmer temperatures could promote increased or decreased outdoor time, affecting exposure to solar ultraviolent (UV) radiation—with possible impacts on rates of skin cancer, eye disease, and vitamin D levels.[2,60–63]
Physical activity: Warmer temperatures, and either increases or decreases in outdoor time, may impact on levels of recreational physical activity—an important determinant of health.[64]
Cardio-respiratory disease from air pollution: High temperatures can exacerbate photo-chemical air pollution with impacts on respiratory disease. Hot, dry conditions increase potential for bush/forest fires, where smoke impacts on people with cardiorespiratory disease.[2,65–68]
Allergic diseases, including asthma: Possible impacts on allergic conditions with changes in plant distribution, flowering, and pollen production.[2,69]
Indoor environment: Climate change may affect the healthiness of indoor environments (e.g. overheating of buildings, changes in indoor air pollutants, flood damage and indoor moisture).[37,70]

Sea-level rise is expected to continue, with an increase in the frequency of extreme high tides and their associated risks, including coastal flooding, inundation, and erosion.[16,23,24]

These climate and related environmental changes have multiple implications for health and wellbeing in New Zealand (Table 1). The magnitude of health impacts will depend on the existing burden of climate-sensitive diseases, the extent and rate of climate change in New Zealand, the capacity of individuals and society to adapt, and the policies chosen to reduce and adapt to climate change.[25]

New Zealand is already affected by a range of diseases that are sensitive to climatic factors,[26–29] and climate trends may well be affecting New Zealanders' health and wellbeing, although such effects are not yet well quantified.[25]

Furthermore, given that global greenhouse gas emissions are continuing to track near the upper end of projections, it will be important to gain a better understanding of the health impacts in New Zealand under high-end scenarios of climate change.[2]

Effects on the determinants of health in Aotearoa-New Zealand

In addition to the health issues listed in table 1, climate change will impact on the broader socioeconomic determinants of health in New Zealand.[14–16]

The economy will be influenced by global climate change.[15] Reduced export income due to, for example, effects on agricultural production (or overseas markets) could lead to higher unemployment, less household money to secure the basics for good health, and a reduced tax-base for health and social spending. An analysis prepared for the Ministry of Primary Industries in 2013 showed that under a high end warming scenario (4.4°C average temperature increase by 2100) there would be a significant decline in dairy pasture production, along with increased dairy cow heat stress in many dairying areas of New Zealand.[71]

However, some positive effects on agriculture/horticulture in New Zealand are also possible.[16,23,71] Thus forward planning and adaptability within the sector will be required to safeguard the economic output of climate sensitive primary industries,[72] which many New Zealanders rely on for good health and wellbeing.

Furthermore, responses to mitigate climate change also have the potential to adversely impact on health. For example, mitigation policies that raise costs for fuel and energy (and therefore increase costs of goods and services) without compensatory measures, could place extra financial burden on people, particularly for low income families, thus affecting ability to afford the basics for good health.[73]

Risks of climate change to health equity and Māori health in Aotearoa-New Zealand

Climate change will cause different impacts for different population groups depending on geographic location, age, ethnicity, health status, and socioeconomic circumstances.[2,25] Māori, Pacific, and low-income groups in New Zealand are at risk of greater adverse health impacts from climate change.[10,14,74]

Māori are at risk of greater impacts (compared with NZ European people) because of a disproportionate burden of disease across many of the health conditions affected by climate change: infectious diseases (e.g. gastrointestinal infection),[75,76] chronic conditions (e.g. cardio-respiratory disease),[75,77,78] and mental ill-health.[75,79,80]

The disproportionately high number of Māori living in deprived circumstances[78,81] means that climate change effects on food security[30,82] and vulnerable infrastructure and housing[25,83,84] will be more difficult to prepare for and recover from–meaning that important determinants of health (such as healthy nutrition, safe drinking water, healthy homes) are undermined.

Any additional pressure on the availability of low income and/or larger family homes resulting from arrival of climate migrants in areas with existing housing pressures (e.g. Auckland region)[14,85,86] would also disproportionately affect Māori who have higher levels of household overcrowding and crowding-related infectious diseases.[87,88] Previous experience in New Zealand has shown that factors that affect the ability of low income families to buy or rent adequately sized houses can lead to families co-habiting, with resultant household overcrowding.[89]

Additional factors which increase climate-health risks for Māori include indigenous relationships with the environment, greater exposure to food-borne disease risk through customary practices such as collection of kaimoana (seafood),[90] greater exposure to outdoor heat whilst undertaking outdoor labour (Māori are overrepresented in semi-skilled/unskilled workforces),[91,92] and poorer access to and through health and social services.[93–100]

Perhaps even more significant are the implications for the economic determinants of health for Māori. The Māori economy is heavily invested in climate-sensitive primary industries;[23,84] and policy responses that place extra financial burden on low income families (disproportionately Māori), without counter-balancing measures, would exacerbate Māori experience of poverty and poverty-related diseases.[73,76]

It is important to note that while this section has focussed on the equity impacts for Māori, many of these issues are also relevant to Pacific peoples in New Zealand and to low income New Zealanders.[10,14]

Health benefits of climate action

The other important link between climate change and health is the substantial opportunity to improve current population health and wellbeing through well-designed policies to reduce greenhouse gas (GHG) emissions.[2–4,19] Knowledge in this area has increased substantially in the last five years, and the health chapter in the recent Fifth Assessment Report of the IPCC included, for the first time, a dedicated section about the health co-benefits of climate action.[2]

Health and health equity gains are possible for heart disease, cancer, obesity, musculoskeletal disease, Type 2 diabetes, respiratory disease, motor vehicle injuries, and mental health, with resultant cost savings for the health system.[2–4,19.]

These co-benefits arise because some emission reductions measures impact on important determinants of health, especially energy intake (nutrition) and expenditure (physical exercise). For example:

- Active transport (walking, cycling, public transport) in addition to reducing CO_2 emissions, improves physical activity and can reduce air pollution and road traffic injuries.[2,101–107] Walking and cycling are inexpensive, and public transport is used proportionately more by people with lower incomes. Thus improved active and public transport infrastructure has the potential to benefit health, climate and equity.[101]
- In New Zealand healthy eating, including increased plant and less red meat and animal fat consumption, would reduce agricultural GHG emissions, and likely lead to reduced rates of bowel cancer and heart disease.[2,108–111]
- Improving indoor environments (e.g. energy efficiency measures such as home insulation) can reduce illnesses associated with cold, damp housing (e.g. childhood asthma and chest infections which are leading causes of hospital admissions, particularly for Māori and Pacific children).[112–114]
- Increasing energy efficiency and/or moving away from fossil fuels would reduce health-damaging air pollution (e.g. particulates) from fuel combustion, in both indoor and outdoor environments, with health gains.[2]

Thus well planned climate action could contribute to significant reductions in the large burden of chronic disease and health inequity in New Zealand, leading to large cost savings for the health sector and society as a whole. This could offset a great deal of the early costs associated with climate change mitigation measures.[2,3]

The New Zealand research community continues to make a strong contribution to the body of knowledge on the health co-benefits of climate action. The housing and health programme (University

of Otago, Wellington) has led the way in quantifying the costs and benefits (including health) of insulation and clean heating.[112,113]

Research at the University of Auckland, using novel modelling techniques, has indicated that transport policy that enables safe commuter bicycling in Auckland has the potential to yield benefits (with respect to injury, physical activity, fuel costs, air pollution, and carbon emissions) that are 10–25 times greater than costs.[107]

The way forward

Rapid and sustained global action to reduce GHG emissions is required to avoid the worst health effects of climate change.[2,115] It is possible to limit the degree of future climate change and to improve health, if the world rapidly upscales carbon-neutral energy production to replace energy production from fossil fuels, along with reducing energy usage, increasing carbon dioxide sinks (e.g. forests) and curbing rising levels of methane and nitrous oxide by modifying our waste management and agricultural/food systems.[4,115]

All individuals, groups, businesses and organisations have a role in reducing emissions, reducing investment in fossil fuels, and demanding that local and central governments act to reduce climate risks in ways that improve health and equity.[4]

Some New Zealand health organisations are beginning to take a lead in addressing their climate-health responsibilities, with action to measure and reduce organisational carbon footprint (Counties-Manukau District Health Board, Canterbury District Health Board), and employment of Sustainability Officers (Counties-Manukau, Waitemata, Auckland and Canterbury District Health Boards). A national network of health professionals interested in collaborating to improve the environmental sustainability of the New Zealand health sector was established in early 2014.[116]

There is much untapped willingness amongst health professionals to improve environmental sustainability within their workplaces (with large potential for operational cost savings),[117,118] but as yet no national framework or mandate to support this, despite a growing international movement and ample international expertise.[119]

There is also a need for the health sector to plan for the inevitable health impacts of climate change in coming decades. Health services should plan for more climate-sensitive diseases, extreme weather events and their casualties, and climate migrants with new and challenging health issues.[10,14,120]

Public Health Services should be strengthened to enable planning and response capability for impacts on drinking water, sewage systems, and civil defence emergencies. Public health surveillance systems need to be in place to detect new and emerging illnesses.[10,120]

It is essential that planning prioritises those population groups most in need of health support in the face of climate change—Māori, Pacific, people on low incomes, migrants, rural people, children, and the elderly.[10] Other events (e.g. Christchurch earthquakes, Hurricane Katrina) have shown that planning is required to avoid an inverse equity pattern in post-disaster responses and outcomes.[121–123]

Outside the health sector, effective public policies are required that both lessen climate risk, and improve population health and health equity. These policies should include an effective carbon pricing system (to replace the largely ineffective Emissions Trading Scheme),[124] while ensuring that financial costs do not adversely affect those on low incomes.[4,73]

Greater investment is required in programmes that both decrease GHG emissions and improve health, such as healthy housing modifications (insulation and clean/efficient heating), active transport infrastructure, and interventions that encourage increased plant and less red meat and animal fat consumption.

One way to encourage this is to ensure that public policy decisions include a health impact analysis, so that potential adverse health impacts can be avoided and positive effects maximised.[125] It is also

critical that any such decisions incorporate an equity analysis, to ensure that the resulting interventions contribute to reducing social and health inequities.

New Zealand must also consider its role in international climate change negotiations and responses. As a high (and growing) per-capita greenhouse gas emitter,[126–128] New Zealand has a responsibility to both increase its own ambitions with respect to greenhouse gas emission reductions, and to promote fair and equitable approaches to emissions reductions globally that take into account historical responsibility and capacity to mitigate.[129,130]

New Zealand, as part of the Pacific, will also need to play a role in supporting the health, wellbeing and adaptation of Pacific Island and other developing nation populations who will face many of the worst health effects of climate change.[131,132]

Conclusion

Climate change poses an urgent threat to human health, wellbeing, and health equity globally, and in Aotearoa-New Zealand.

On the other hand, well-planned action to reduce greenhouse gas emissions offers opportunities to improve population health, equity, and reduce chronic disease burden. This could result in large cost savings for the health sector and society as a whole, which would offset a great deal of the early costs associated with climate change mitigation measures.

As health professionals, we have a responsibility to raise awareness of the health implications of climate change, and to press for urgent action. If we act quickly, we have an opportunity to turn one of our greatest health threats into positive action to significantly improve the health, equity, and resilience of our patients and population.

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ORIGINAL ARTICLE

Alcohol intake, marijuana use, and sleep deprivation on the risk of falls occurring at home among young and middle-aged adults: a case-crossover study

Simon Thornley, Bridget Kool, Roger J Marshall, Shanthi Ameratunga

Abstract

Aim This study investigated whether hospitalised fall-related injuries among young and middle-aged adults were associated with short term effects of alcohol intake, marijuana use and sleep deprivation.

Method A case-crossover design was used to study 690 adults (aged 20 to 64 years) admitted to public hospitals within 48 hours of a fall-related injury, occurring at home, in three regions of New Zealand during August 2008 to December 2009. A matched-pair interval method of analysis was used to compare alcohol intake, marijuana use and sleep deprivation before the event with similar information in two control periods: 24 hours-before and 1 week-before the time of injury.

Results After adjustment for other paired exposures, the estimated risk of injury was substantially higher after consuming alcohol within the preceding 6 hours, with a dose response gradient. After adjusting for confounding variables, the data did not support a significantly elevated risk of fall-related injury associated with sleep deprivation (<6 hours sleep in the preceding 24 hours), or marijuana use in the preceding 3 hours.

Conclusion The findings support the expansion of efforts to reduce the harmful effects of alcohol intake in the home environment.

Unintentional injuries at home impose a major burden of potentially preventable morbidity, disability, lost productivity and healthcare costs in New Zealand.[1] Falls which lead to hospital admission are the leading cause of unintentional injury at home among young and middle-aged adults (aged 20 and 64 years) in New Zealand.[2]

Well-established evidence has linked alcohol, marijuana use, and driver sleepiness with road traffic injury.[3–9] However, the immediate effects (sometimes referred to as acute effects) of these factors on the risk of fall injuries in the home among young and middle-aged adults has received comparatively little research attention.[10]

Case-crossover studies can provide useful insights about associations between transient exposures with short-term effects and outcomes that have a defined onset. They obviate the need for controls (a potential source of bias in case-control studies) as each case provides exposure information during the period immediately before the injury, and during pre-defined control or reference periods, or as typically exposed. This design has been used extensively to investigate the causes of injuries.[11–14]

We conducted a population-based case-crossover study, the primary focus of which was to investigate the immediate effects of alcohol intake, marijuana use and sleep deprivation on the risk of hospitalised unintentional fall-related injury at home among young and middle-aged adults. This work was conducted as part of a larger project that included a case-control study.[15]

Method

Participants were recruited from admission registers at all trauma-admitting hospitals in three regions (Auckland, Waikato, and Otago) in New Zealand, from August 2008 to December 2009. Cases were defined as people aged 20 to 64 years who were admitted to hospital within 48 hours of an injury due to an unintentional fall at home. Individuals whose injury occurred in the context of paid work or in a residential institution and those unable to complete the questionnaire in English were excluded.

A research nurse interviewed cases using a structured questionnaire which lasted about 30 minutes. Most interviews were conducted face-to-face during the hospital stay, and, if this was not possible, via telephone after discharge. Information was collected about participants' sociodemographic characteristics, circumstances before the injury, medical conditions, smoking status, prescription drug use, and usual alcohol use. Information was also collected about the exposures immediately before (the "index" period)preceding the time of injury, and two previous "control" periods: *24 hours-before* and *1 week-before* the time the injury occurred.

Data about the quantity and type of alcoholic drink consumed, as recorded by the interviewer, during the preceding 6 hours at each time period were coded into standard units (10g of ethanol), while marijuana and other recreational drug use was collected for the 3 hours preceding each time period. The total number of hours spent asleep during the preceding 24 hours was also ascertained, and less than 6 hours sleep was considered as acute deprivation. Case medical records were reviewed to extract the International Classification of Disease (ICD-10) external cause of injury codes.

We examined the association between exposures (as categorical variables) and fall injury using the case-crossover matched-pair interval method. Separate analyses were done for the two control periods and, for each, crude effects for binary exposure variables were calculated as ratios of discordant pairs, and using the exact binomial test.[18] The effects of binary and continuous exposures, adjusting for other matched covariates, were assessed by conditional logistic regression, assuming 2:1 control period: case event matching. For this analysis the regression models accounted for both control periods. Other covariates, besides the exposure, were retained in the models if the inclusion of each resulted in an incremental change in the beta-coefficient for the exposure of interest by >10%.[19]

A previous case-control study which investigated the effect of alcohol intake on any injury, from the United States,[20] found the proportion of discordant exposures (between case and control exposure periods) within individuals was 12% when 'none' and 'any' alcohol use were compared. Based on this information, we estimated that a sample of 690 participants (our final sample size, after exclusions) would allow us to estimate a relative risk of 1.9 with 90% power, using a two-sided 5% level for statistical significance.[21]

The study was approved by the Multi-regional Ethics Committee (MEC/08/13/EXP), and the research boards of the hospitals from which cases were recruited.

Results

Of the 765 cases deemed eligible for study, 75 (9.8%) were unable to be contacted or refused to take part in the study, leaving 690 for analysis.

Table 1 show the characteristics of these cases. The median age of participants was 48 years and 45% were male. One-third of participants reported they did not have the equivalent of 3 years of secondary school.

Most injuries occurred as a result of falls on the same level (slips, trips or stumbles). Almost a third of cases (30%; 195/666) reported hazardous or harmful patterns of usual alcohol consumption (AUDIT score $\geq$ 8). Weekly, or more frequent, marijuana use among this population was rare (5%; 31/682).

Table 2 shows the distribution of alcohol use among participants in the index and control periods. Due to missing information, the number of complete paired entries was less than 690.The discordant pairs odds ratio for one or more drinks was 124/16=7.75 using the day before control period, and 75/12=6.25 for the week before control period.

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Table 1. Participant characteristics

Characteristic	*n* (%)
Gender	
Male	308 (44.6)
Age category	
20 to 29	97 (14.1)
30 to 39	103 (14.9)
40 to 49	171 (24.8)
50 to 59	165 (23.9)
60 to 65	154 (22.3)
Ethnic group	
European (New Zealand & Other)	495 (71.7)
Maori	87 (12.6)
Other	62 (9.0)
Pacific	45 (6.5)
Missing/Refused	1 (0.1)
Highest qualification	
None	225 (32.6)
School Certificate	58 (8.4)
Overseas secondary school	22 (3.2)
Sixth Form Certificate/Bursary	64 (9.3)
Trade certificate	200 (29.0)
Tertiary degree	105 (15.2)
Missing/Refused	16 (2.3)
Employed	
Yes	428 (62.0)
No	260 (37.7)
Missing/Refused	2 (0.3)
Day of week injury occurred	
Mon to Thurs	315 (45.7)
Fri to Sun	375 (54.3)
Fall injury mechanism	
On same level (W01)	305 (44.2)
Ladder (W10)	212 (30.7)
Stairs or steps (W11)	77 (11.2)
From or out of building (W13)	48 (7.0)
Others	48 (7.0)
AUDIT[a] total score (alcohol use)	
(0–7) low risk	471 (68.3)
(8–15) risky or hazardous	152 (22.0)
(16–19) high-risk or harmful	18 (2.6)
(over 20) high-risk/dependent	25 (3.6)
Missing/Refused	24 (3.5)
Marijuana use (at least weekly)	
Yes	31 (4.5)
No	651 (94.3)
Missing/Refused	8 (1.2)
Current smoker	
No	473 (68.6)
Yes	216 (31.3)
Missing/Refused	1 (0.1)
Other recreational drug use (any during preceding year)	
Yes	31 (4.5)
No	649 (94.1)
Missing/Refused	10 (1.4)

[a]AUDIT - Alcohol Use Disorders Identification Test[16]

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Table 2. Alcohol intake during the 6-hour period before the injury, compared to the control period (day before or week before)

Alcohol intake during 6-hour control period	Alcohol intake in the index period 6 hours before injury		
	0 drinks	1 to 3 drinks	4 or more drinks
Day before injury (*n*=652)			
0 drinks	440	62	62
1 to 3 drinks	14	29	16
4 or more drinks	2	4	23
Week before injury (*n*=606)			
0 drinks	434	38	37
1 to 3 drinks	8	33	13
4 or more drinks	4	7	32

After adjusting for other covariates (Table 3), drinking any alcohol in the preceding 6 hours (compared with none) had an almost eight-fold increase in the odds of injury. The increasing odds ratios for '1 to 3 drinks' and '4 or more drinks', compared to none, suggested a dose-response effect. Table 3 also shows the effects for sleep deprivation and marijuana use. Neither of the adjusted estimates is statistically significant.

Table 3. Summary of crude and adjusted measures of association

Exposure	Control period 1 "Day before"		Control period 2 "Week before"			
	crude OR	95% CI	crude OR	95% CI	Adjusted* OR	95% CI
Alcohol (≥1 vs. 0 drinks)	7.75	4.97–13.2	6.25	3.80– 11.7	7.81	4.74–12.8
Alcohol (1 to 3 vs. 0 drinks)	4.43	2.86–7.73	4.75	2.68–10.5	5.21	3.02–8.99
Alcohol (≥4 vs. 0 drinks)	31.0	8.95–254	9.25	3.93–33.1	15.2	7.60–30.4
Sleep deprivation (< 6 hours vs. ≥ 6 hours in last 24 hour period)	2.09	1.44–3.73	1.17	1.02–1.75	1.41	0.69–2.89
Marijuana (any use in 3-hour period, compared to none)	1.38	1.06–2.56	1.83	1.20–4.27	0.35	0.11–1.11

*Alcohol, sleep deprivation, and marijuana estimates adjusted for all these exposures (using conditional logistic regression) and accounting for both control periods.

OR – odds ratio; CI – confidence interval.

No evidence of effect modification was apparent in the associations between alcohol use and fall-related injury by the following binary coded variables: smoking status, sleep deprivation, ethnic group, age, gender, weekend injury, or recreational drug use. Similarly, level of alcohol use, divided into four categories by AUDIT score, did not influence the acute alcohol-injury association.

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Discussion

Acute intake of alcohol was associated with an 8-fold increase in the risk of unintentional fall injury occurring at home among young and middle-aged adults. Further, a dose-response effect is likely. The findings are consistent with a case-control analysis which drew on participants from a narrower age group (25 to 60 years) and from only one region of New Zealand (Auckland) and which found a three-fold increase in risk.[10]Given the larger sample size, the present study provided more precise estimates about the association between alcohol and falls of this nature. Crude associations suggesting increased risks of fall-related injury associated with marijuana use and sleep deprivation were attenuated in models adjusted for confounding variables, including alcohol.

Despite a good response rate (90%), several potential biases may exist. The exposure information was self-reported and "social desirability bias"[26] may have led to under-reporting of substance use. Previous research, however, suggests good correlation between self-report of alcohol use and objective measures. One study, for example, showed that self-report of acute alcohol intake among people who presented to an emergency department, when compared to serum alcohol levels, showed reasonable levels of agreement.[27]

Another threat to the validity of this study is period-dependent misclassification of exposure.[28] Recall of alcohol preceding an injury may be more reliable than during the control periods. This is supported by the observation that more people had missing information for the 'week before', compared with the 'day before' period. However, the small amount of missing information (less than 15%) for crude or multivariate analyses suggests recall bias is unlikely to affect our results.[29]

If control period alcohol exposure were under-reported, the association between alcohol intake and injury risk would be inflated, but the comparative accuracy of the exposure information and resulting bias in the measure of association is difficult to assess. In a previous case-crossover study of the antecedent causes of Meniere's disease, the researchers found that the risk of misclassification of exposures at the proximal and distal reference periods did not pose a major threat to the validity of the study findings.[28]

The effect size relating to alcohol use in our study is somewhat higher than the pooled estimates of the contribution of acute alcohol use to different classes of injury in meta-analyses by Taylor and colleagues[9,23]who found that for motor vehicle crashes, the odds ratio increased by 1.24 (95% CI: 1.18–1.31) per drink (10g pure alcohol), and for non-motor vehicle-related injury by 1.30 (95% CI: 1.26–1.34) per 10g. A case-crossover study conducted by our research team to examine risk factors for cutting and piercing injuries at home, in the same study region, found that drinking in the previous 6 hours (compared with not drinking) was associated with a three-fold risk in injury.[25]

The epidemiological evidence of the injury risks apparent in this study is consistent with well-recognised influences of alcohol on cognitive and psychomotor performance. Clinical studies demonstrate that consuming alcohol can impair reaction time, cognitive processing, coordination and vigilance. These effects are most commonly observed after consuming three to four standard alcoholic drinks (~30 to 40g of alcohol).[30]

As a case-crossover study, we have only examined the immediate effects of alcohol and marijuana. It does not address the issue of longer term effects, for example, whether someone who habitually and continually drinks alcohol, or smokes marijuana, is generally at higher risk of injury, irrespective of excess short term effects.

In New Zealand, alcohol is widely available and relatively inexpensive.[31] The study findings reinforce the need for broader public health policies which address problem drinking.[32] Screening and early intervention in primary care,[33] limiting access to alcohol,[34-36] and the introduction of alcohol taxes[37] are some examples of effective strategies that may reduce the risk of injury from falls in this relatively young and economically productive age group.

Competing interests: Nil.

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ORIGINAL ARTICLE

Relationship between fructose and lactose intakes and functional gastrointestinal symptoms in a sample of 50-year-old Cantabrians in New Zealand

Robin Spencer, Richard Gearry, John Pearson, Paula Skidmore

Abstract

Aims To examine the relationship between fructose and lactose consumption and irritable bowel syndrome (IBS) symptoms in 50-year-old adults residing in Canterbury, New Zealand.

Methods The Canterbury Health Ageing and Life Course (CHALICE) study is a study of 50-year-old Cantabrians. A 4-day estimated food and beverage diary (FBD) was completed by 227 participants, 75.7% of those recruited. The Birmingham IBS symptom questionnaire was administered and individual participant scores were calculated for constipation, diarrhoea, pain score, and total symptom score. Associations between symptoms and the intake of fructose and lactose were examined using binary logistic regression.

Results Greater mean daily intakes of fructose (P=0.05) and lactose (P=0.04) were associated with a lower prevalence of IBS pain symptoms after adjusting for demographics and social economic status. However there was no evidence of an association with constipation, diarrhoea or total IBS score.

Conclusions Although our data show inverse relationships between fructose and lactose intakes and IBS pain symptoms, the use of cross-sectional data do not allow us to determine causality in these relationships. However it is possible that participants may have reduced their intake of fructose and lactose in response to IBS related pain. Follow up of this cohort would allow us to determine if this is the case. Future research could also investigate whether people with IBS could benefit from guidance from a dietitian around consumption of high lactose or fructose-containing foods.

Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal syndrome characterised by recurrent abdominal pain, altered bowel habits, bloating, distension, flatulence, relief of pain with defecation and incomplete defecation.[1] The estimated worldwide prevalence of IBS varies from 8–23% depending on the population studied and the diagnostic criteria used.[2] Results from the Dunedin Multidisciplinary Health and Development Study in New Zealand (NZ), using the Manning criteria, estimated an IBS prevalence of 18% when participants were 26 years old.[3]

IBS is the most common gut disorder in the general population and imposes a substantial financial burden on society. Annual direct costs associated with IBS have been estimated to cost 15–30 billion US dollars per year for all IBS patients in the United States of America (USA).[4,5] New Zealand has an ageing population that is anticipated to substantially increase healthcare costs in the future.[6] IBS patients have a substantially reduced quality of life andon average, IBS patients miss 13.4 days of work or school per year.[9]

There are multiple possible aetiologies for IBS and no consistently effective therapies. Most IBS patients believe that diet plays a role in their symptoms.[11] It may, therefore, be expected that IBS patients are selective with their dietary choices. However, studies comparing the diet of IBS patients and controls have found mixed results.[9,12–14]The reduction of Fermentable Oligo-, Di- and Mono-saccharides And Polyols (FODMAPs) in the diet has been proposed as an effective way to manage IBS symptoms.[15]

When lactose and fructose are poorly absorbed in the small intestine they exert an osmotic effect increasing water delivery to the large intestine where they are rapidly fermented by luminal bacteria producing gas, luminal distension and pain.[5,15] Many studies have shown that when fructose and/ or

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lactose is limited or excluded from the diet and patients with IBS are compliant, IBS symptoms significantly improve; however one study found controls had greater improvement than IBS patients.[12,16–23]

Although international studies have found that limiting fructose intake improves IBS symptoms, their results cannot necessarily be generalised to the New Zealand population. Furthermore, the patterns of dietary manipulation have not been examined in specific age groups, a New Zealand population, and particularly those who have established dietary patterns. Therefore, we aimed to examine the relationship between fructose and lactose consumption and functional gastrointestinal symptoms in 50 year olds residing in Canterbury, New Zealand.

Methods

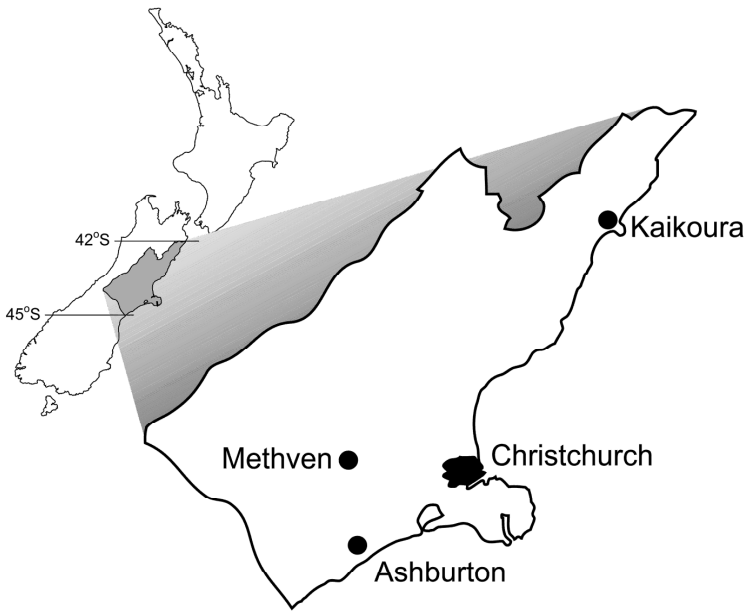
Study design—The CHALICE study comprises a random sample of 50-year-old adults selected at random from the electoral roll who live within the Canterbury District Health Board (CDHB) catchment in NZ (Figure 1). Participants were recruited in a ratio of 4:1 non-Māori to Māori.

The fullCHALICE methods are described in detail elsewhere.[26] In brief, assessment data were collected during a 4–6 hour face-to-face interview, with self-completed questionnaires, lifestyle diaries, and diagnostic tests.

The interview comprised seven modules although for the purposes of this study, only data from modules two (personal health history questionnaire) and seven (lifestyle questionnaire including a 4-day estimated food and beverage diary [FBD]) and the Birmingham IBS symptom questionnaire was used.

Ethical approval was obtained from the Upper South A Regional Ethics Committee and all participants provided written informed consent.

Figure 1. The geographic boundaries of Canterbury as defined by the Canterbury District Health Board and specific Territorial Local Authorities



Food and beverage diaries—The 4-day FBD was completed by participants in their own time after their initial assessments and returned to CHALICE by post. Participants were shown how to complete the FBD and shown examples by a trained CHALICE interviewer; written instructions for completion were also included with the

diary. When the diaries were returned, study nutritionists checked the FBD for any missing information. FBD data were converted to nutrients using the dietary assessment software; Kai-culator.[27] The following nutrients were used: free fructose (g), lactose (g), total energy (Kcal), and total sugars (g).

Irritable bowel syndrome symptoms—The Birmingham IBS symptom questionnaire is a validated assessment tool that incorporates a symptom frequency scale measuring IBS symptoms over the past 4-week period.[28] The questionnaire comprises 11 IBS symptom questions and each question has a standard 6-point response scale ranging from 1 = "all of the time" to 6 = "none of the time". Raw data from the Birmingham IBS symptom questionnaire was used to calculate individual participant scores for constipation-predominant IBS, diarrhoea-predominant IBS, pain score, and total symptom score. Participants who reported any IBS symptoms during the last 4 weeks were categorised into the IBS symptom group and those who reported no symptoms were categorised into the no symptoms group.

Standard of living—Participants also completed the Economic Living Standard Index short form ($ELSI_{SF}$), which takes into account home ownership, social participation, economising, self- rated standard of living, satisfaction with standard of living, and adequacy of income. The $ELSI_{SF}$ score is calculated as a continuous score ranging from 0 to 31. Those with scores from 0 to 24 (hardship and comfortable categories) were categorised as having a lower standard of living. Participants with scores from 25 to 31 (good and very good categories) were categorised as having a high standard of living.

Statistical methods—All statistical analyses were performed using R Studio version 0.97.551 (R Studio, inc, 2009–2012). Means, standard deviations, and frequencies were calculated to describe each variable. The criterion for statistical significance was set at $P<0.05$. The Shapiro-Wilk test was used to test for normality. In a random population such as CHALICE, most people will not report IBS symptoms. Two binary logistic regression models were used to predict relationships between the mean daily intake of fructose and lactose (independent variables) and each of the four IBS dimensions (dependent variables). Model 1 was a univariate model and the following predictors were adjusted for in model 2: sex, ethnicity, and $ELSI_{SF}$.

Results

As of 20 June 2013, 300 CHALICE participants had completed all seven modules. 227 (75.7%) of the 300 participants completed and returned the Birmingham IBS symptom questionnaire and completed at least 3 days of the FBD and were included in this study.

Fifty-three percent of participants were female, 15.9% identified as NZ Māori and the majority of participants (63%) were in the higher $ELSI_{SF}$category (Table 1).

Table 1. Demographic characteristics and prevalence of participants who experienced any irritable bowel syndrome(IBS) symptoms

Variable	Category description	Number of participants n=227 (%)
Sex	Male	107 (47)
	Female	120 (53)
Ethnicity [a]	Māori	36 (16)
	Non-Māori	191 (84)
$ELSI_{SF}$	Hardship / Comfortable	83 (37)
	Good / Very good	144 (63)
IBS	IBS Symptoms reported	172 (76)
	No Symptoms	55 (24)
Pain [b]		106(47)
Constipation [b]		117 (52)
Diarrhoea [b]		122 (54)

[a] Participants who identified as both NZ European and NZ Māori were counted as Māori and all other participants were counted as Non-Māori.

[b]Data represents participants who experienced at least one symptom over the prior 4-week period.

Table 1also shows the number of participants who scored at least one in each of the four dimensions of IBS symptoms (i.e. described any functional symptoms as opposed to none). Twenty-four percent of the participants had experienced no IBS symptoms in the prior 4-week period.

Table 2. Participants' estimated mean daily energy and nutrient intakes

Variable	Females (n=120) Mean (SD)	Males (n=107) Mean (SD)	All (n=227) Mean (SD)
Energy (kcal/day)	1835 (460)	2478 (633)	2141 (621)
Fructose (g/day)	20.6 (10.2)	25.5 (14.4)	22.9 (12.6)
IBS Symptoms	20.6 (10.4)	23.8 (10.6)	20.0 (10.6)
No Symptoms	20.6 (9.4)	29.9 (20.8)	25.7 (17.4)
Lactose (g/day)	14.2 (8.9)	15.1 (9.2)	14.6 (9.0)
IBS Symptoms	13.4 (9.3)	15.5 (9.3)	14.3 (9.3)
No Symptoms	17.6 (6.4)	14.0 (9.1)	15.6 (8.1)
Total Sugars (g/day)	98.6 (40.0)	125.4 (59.3)	111.2 (51.6)
Fructose mg/1000 kcal	11.1 (4.9)	10.3 (5.0)	10.7 (4.9)
Lactose mg/1000 kcal	7.8 (4.6)	6.1 (3.6)	7.0 (4.2)

Table 2 shows participants' mean daily intake of energy, fructose, lactose and total sugars. The main food groups that participants obtained their fructose intake from were soft drinks, fruit juice and fruit, and for lactose the main food groups were milk and yoghurt.

Table 3. Fructose and lactose intake and four irritable bowel syndrome dimensions

			Adjusted	
Independent Variable	**Odds Ratio (CI)**	**P-value**	**Odds Ratio (CI)**	**P-value**
Fructose Intake				
Total IBS Symptoms				
Fructose	0.98 (0.95–1.00)	0.07	0.98 (0.95–1.00)	0.11
Sex			0.72 (0.38–1.36)	0.31
Ethnicity			0.89 (0.39–2.17)	0.78
$ELSI_{SF}$			0.34 (0.16–0.68)	0.004*
Pain				
Fructose	0.98 (0.95–1.00)	0.04*	0.98 (0.95–1.00)	0.05
Sex			0.82 (0.48–1.41)	0.48
Ethnicity			0.65 (0.30–1.35)	0.25
$ELSI_{SF}$			0.66 (0.38–1.14)	0.14
Constipation				
Fructose	0.99 (0.97–1.01)	0.35	0.99 (0.97–1.01)	0.45
Sex			0.74 (0.42–1.29)	0.29
Ethnicity			0.76 (0.36–1.60)	0.47
$ELSI_{SF}$			0.32 (0.18–0.56)	<0.001*
Diarrhoea				
Fructose	0.99 (0.97–1.02)	0.61	0.99 (0.97–1.02)	0.60
Sex			1.06 (0.62–1.82)	0.84

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Ethnicity			1.07 (0.52–2.23)	0.86
$ELSI_{SF}$			0.52 (0.30–0.90)	0.02*
Lactose Intake				
Total IBS Symptoms				
Lactose	0.99 (0.95–1.02)	0.37	0.99 (0.95–1.02)	0.47
Sex			0.66 (0.35–1.23)	0.19
Ethnicity			0.89 (0.39–2.16)	0.78
$ELSI_{SF}$			0.34 (0.16–0.69)	0.004*
Pain				
Lactose	0.97 (0.94–1.00)	0.04*	0.97 (0.94–1.00)	0.04*
Sex			0.76 (0.44–1.29)	0.31
Ethnicity			0.64 (0.30–1.32)	0.23
$ELSI_{SF}$			0.68 (0.39–1.17)	0.17
Constipation				
Lactose	1.00 (0.97–1.03)	0.73	1.00 (0.97–1.03)	0.90
Sex			0.71 (0.41–1.23)	0.22
Ethnicity			0.76 (0.36–1.61)	0.48
$ELSI_{SF}$			0.32 (0.18–0.56)	<0.001*
Diarrhoea				
Lactose	0.99 (096–1.02)	0.60	0.99 (0.96–1.02)	0.69
Sex			1.03 (0.61–1.75)	0.91
Ethnicity			1.07 (0.52–2.23)	0.87
$ELSI_{SF}$			0.52 (0.30–0.91)	0.02*

* P-value of <0.05 is considered statistically significant

First columns are OR (CI) and p value from univariate logistic model; Second columns are OR (CI) and p value from logistic regression with covariates for sex, ethnicity and socioeconomic status.

Total IBS score, constipation, and diarrhoea were not associated with fructose intake (Table 3). A higher fructose intake was negatively associated with the pain score. For every 10 g increase in mean daily fructose intake, the probability of experiencing any pain decreased by 21% (P=0.04, CI 0.95, 1.00). After adjustment for covariates (model 2), the effect size was unchanged (P=0.05, CI 0.95, 1.00).

A higher mean daily lactose intake was negatively associated with the pain score (Table 3). For every 10 g increase in mean daily lactose intake, the probability of experiencing any pain decreased by 28% (P=0.04, CI 0.94, 1.00). This association remained significant after adjusting for covariates (P=0.04, CI 0.94, 1.00).

Discussion

This cross-sectional population-based study of 50 year olds from Canterbury, New Zealand found that a higher mean daily intake of fructose and lactose was associated with a lower abdominal pain score in the preceding 4 weeks. Results from previous similar cross-sectional studies in other countries have been conflicting.

A cross-sectional study of 187 women from the USA found that participants with more severe IBS symptoms had lower intakes of fructose,[13] however, the same group published another study of 55 women in which there was no difference in fructose intake between women with and without IBS-like

symptoms.[14] The participants in both of these studies were all female and younger (aged 18–48) than the CHALICE participants although other study procedures were similar.

This association shown between fructose intake and pain score does not mean that there is a causal relationship involved. However, there is strong evidence from intervention studies in other populations that a low fructose diet improves IBS symptoms.[16–18,29,30] Alternatively, this association may reflect alterations in food choice that have been driven by participants' previous experiences with high fructose foods. While it is possible that the participants may have knowledge of low FODMAP dietary interventions, patient knowledge of food choice was not specifically assessed in the present study.

A higher mean daily intake of lactose was associated with a lower pain score. A similar trend was found in a USA study where women with IBS-like symptoms consumed significantly less lactose than women without symptoms.[14] This small study of 18 women aged 19–45 analysed lactose intake with 3-day diet FBDs. Another study found that IBS patients who had received no dietary advice, consumed significantly less milk, milk products, and calcium compared to controls.[9] However the data are not consistent with another study finding no difference in lactose intake between IBS patients and controls, albeit the controls used were staff members at which the clinic the study was conducted.[12]

While the results from the present study show inverse associations between lactose intake and pain symptoms, the cross-sectional nature of the data means that we cannot determine if subjects reduce lactose intakes because they experience pain. While it is possible that lactose protects against IBS symptoms, it may be that participants exclude certain foods to avoid symptoms as there is moderate evidence from intervention studies that shows that limiting lactose intake improves IBS symptoms.[12,20–22]Unlike fructose, lactose intake is more widely known to be associated with IBS symptoms.

Seventy six percent and 47% of the participants reported that they experienced at least one IBS symptom and one pain symptom in the prior 4 weeks, respectively. Within NZ, there is only one other study that has examined the prevalence of IBS symptoms, the Dunedin Multidisciplinary Health and Development Study—a birth cohort of around 1000 participants born in Dunedin.When participants were 26 years old, 64% experienced at least one IBS symptom in the previous year, and 46.5% reported abdominal pain.[3]

CHALICE participants may have a higher prevalence of IBS symptoms than those in the Dunedin Multidisciplinary Health and DevelopmentStudy due to age differences between cohorts as the peak age of IBS presentation is between 30–50 years of age.[8] This is supported by a USA study, which found that the incidence of IBS increased with age where those older than 55-years had the highest IBS incidence.[7]

As CHALICE participants are 50 years old, they are also more likely to have developed gastrointestinal diseases other than IBS that cause gastrointestinal symptoms such as diverticular disease. The difference in IBS symptom prevalence between the present study and theDunedin Multidisciplinary Health and Development Study, may also be due to different criteria used to assess IBS symptoms.

The Dunedin Multidisciplinary Health and Development Study used the Manning criteria to assess IBS symptoms over the past year whilst CHALICE used the Birmingham questionnaire to assess the past 4 weeks. Therefore, the constipation and diarrhoea dimensions of IBS may not be directly comparable between the studies.

The mean daily fructose intake of 25.5g for males and 20.6g for females is comparable to that of similar age groups of participants in the 1997 national nutrition survey (NNS97) and the 2008/09 NZANS.[24,31] This suggests NZ adult fructose intake has remained stable since 1997 in contrast to the USA where a mean daily fructose intake of 49g increased from 37g/day over two decades.[25]

The mean daily lactose intake was 15.1g for males and 14.2g for females, which is comparable to a similar age group in the NNS97 and the 2008/09 NZANS.[31,32] These intakes are comparable to Swedish (12g/day) and USA (9–15g/day) intake data.[33,34] The CHALICE study recruitment method provides a wide participant pool and allowed Māori to be over-sampled. A 4-day FBD allowed sufficient data to estimate usual intake without causing a high respondent burden,[35] demonstrated by the high return rate of the FBD (75.7%).

There may be a number of weaknesses in the design of the present study. This is a cross-sectional study and causality cannot be determined. The results of this study may not be generalisable to the NZ or worldwide population as the participants were all 50 years old and living in the CDHB catchment area, which may differ from other regions.

Severe earthquakes in the CDHB catchment area in the previous 4 years may have increased junk food consumption due to stress. However, it is important to perform such studies in different regions as the mean intake and food sources of fructose and lactose may differ significantly between populations. Limitations arise when using estimated FBD as estimating the volume of food consumed is difficult and as with any form of dietary assessment, people may alter their eating patterns or under-report food intake.

The Birmingham IBS symptom questionnaire was not designed to diagnose IBS but to measure the prevalence of IBS symptoms in those with IBS.However, the questionnaire has been validated and was found to be reliable for use in cross-sectional studies.[28] Overall there are two results that are significant at the 5% P-value threshold. Further studies of this longitudinal cohort will provide a larger pool of participants, increasing the statistical power of analyses. Further questioning of CHALICE participants regarding IBS symptoms in the future may also confirm those with long-term functional symptoms.

The majority of previous studies in the area of fructose and lactose intake and IBS symptoms comprise experimental studies where fructose and lactose are restricted from the diet. There are few studies assessing the dietary intake of both fructose and lactose in IBS patients in the general population and in the New Zealand population. Therefore, this study complements the current literature by examining the relationship between fructose and lactose consumption and IBS symptoms in a 50 year olds residing in Canterbury New Zealand.

The current study found that 50-year-old Cantabrians who have higher fructose and lactose intakes are less likely to experience IBS pain symptoms. Further longitudinal research in this cohort would allow us to determine if participants reduce their lactose and fructose intakes because of IBS pain. Although we cannot ascertain if this is the case in this study, it may be worthwhilefor dietitians who counsel patients with IBS to be aware of this possibility so they can help guide them to achieve a well-balanced diet.

Competing interests: Nil.

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ORIGINAL ARTICLE

Self-monitoring blood glucose test strip use with diabetes medicines in people with types 1 and 2 diabetes in New Zealand

Scott Metcalfe, Peter Moodie, Hew Norris, Dilky Rasiah

Abstract

Aims (1) To identify actual dispensings of publicly funded blood glucose test strips (SMBG) in New Zealand according to severity of disease, as proxied by the type of medicines prescribed; and (2) To compare these rates with published consensus guidelines on SMBG usage.

Method All dispensings of diabetes medicines and blood glucose test strips (SMBG) in 2011 were identified and matched to patients, using encrypted National Health Index numbers (NHIs).

Five hierarchical treatment groups were identified, as the use of:

• Insulins without oral hypoglycaemic agents (OHs);

• Insulins with OHs;

• Sulphonylurea-containing OH regimens without insulins (with or without other diabetes medicines);

• Metformin alone, with or without glitazones or acarbose; and

• No diabetes medication but accessing SMBGs.

The average SMBG dispensings to patients in each of these groups was then calculated. The calculation was performed only for 'steady-state' patients, i.e. patients assumed stabilised on the same medication regimen for at least one year. Differences between actual and expected dispensings were calculated from expected daily strip use for each group.

Results An estimated 183,000 patients were dispensed diabetes medicines and/or SMBG during 2011. Of these, 122,000 were identified as 'steady-state' patients. Patient numbers and median ages varied widely across treatment groups and by gender and ethnicity. Dispensing rates for SMBG varied by treatment group, with probable over-dispensing in some groups and under-dispensing in others when compared with published guidelines.

In particular there appeared to be relatively large under-dispensing of SMBG in patients requiring insulin (especially the 25–44 age-group or Māori and Pacific peoples) and a high over-dispensing in those using metformin alone or on no diabetic medication.

Conclusion There are appreciable variations in the use of SMBG between treatment groups. Adherence to published guidelines may improve efficacy and health outcomes for those using insulin and reduce pain, anxiety and disruption for those using metformin or diet alone for control of their diabetes.

Blood glucose testing for patients with type 1 diabetes, and those with type 2 diabetes using insulin, is a mainstay of clinical management. For those on treatment with sulphonylurea medicines but not using insulin, the main long-term benefit of self-monitoring blood glucose (SMBG) usage is to detect hypoglycaemia; however it is acknowledged that there will be greater usage in this group if they are transitioning to an insulin-based regimen.[1–4]

For patients with diabetes on metformin alone or other non-hypoglycaemia causing diabetes medication(s), or indeed on no diabetes medication at all, SMBG testing should not be a routine occurrence—as regular measurement of glycated haemoglobin (HbA1c) levels is the main means of

managing dosage changes.[1–4]This is where clinical evidence[5–8] and guidelines[1–4] suggest that there is usually little need for patients maintained on diet and exercise or metformin alone to routinely self-monitor their blood glucose, but the intermittent use of SMBG may be encouraged as an educational and clinical management tool to detect patterns of glycaemia.[1–4]

Despite national and international clinical guidelines on the optimal use of SMBG monitoring,[1–4] to date there has been little information on how, at a national level, actual testing rates for different groups have compared with the guidance. Such information is useful, as under-testing can suggest suboptimal care (expected from first principles to contribute to poor diabetes outcomes); conversely, over-testing can be inconvenient, disruptive to patients, possibly harmful,[9] and is an opportunity cost to public health system in New Zealand—where $23 million for test strips was publicly funded via the Pharmaceutical Schedule during the 2011/12 financial year[10] (this spending predating PHARMAC's funding decision in 2012 on strips and meters[11]). Understanding real-world practice compared with optimal practice can therefore inform discussion and focus on the educational messages given to patients. It can also help ensure funding priorities in the health budget are appropriate.

Once a funded prescription is dispensed in New Zealand, information is collected in a national repository and available for analysis. In addition to prescriber details, the medication name, strength, quantity and dosage are recorded, along with an encrypted National Health Index (NHI) number where this is available.

The NHI number is a unique identifier for nearly everybody in New Zealand who has ever had contact with the health service. This number can be linked anonymously to New Zealand census data and contains information about the individual's date of birth, ethnicity and socio-economic status. Most general practices in New Zealand have computerised prescribing systems, and over 95% of all prescriptions recorded in the New Zealand Health Information Service (NZHIS) database now have an NHI number attached.

This brief analysis compares publicly funded blood glucose test strip uptake in New Zealand against published guidance on appropriate rates of usage. This helped inform the thinking behind PHARMAC's 2012 funding decision for meters and strips.[11]

Methods

This observational audit (see endnote *)[12] identified all patients who were prescribed diabetes medicines and/or blood glucose test strips between 1 January and 31 December 2011. These anonymised data[12] were extracted from the Pharmaceuticals Collection (previously PharmHouse) administrative claims database,[13]and were then categorised into five therapeutic-wide groupings (with categories within) consisting of patients using:

- Insulins without oral hypoglycaemic agents (OHs) (which we divided into various subgroups, labelled as categories A, D and E). These groups were regarded as surrogate markers for type 1 diabetes;
- Insulins with OHs (category C), being a surrogate marker type 2 diabetes on insulin;
- Sulphonylurea-containing OH regimens without insulins, with or without other diabetes medicines (category F)—a surrogate for more severe type 2 diabetes;
- Metformin alone, with or without glitazones (thiazolidinediones) or acarbose (category G)—a surrogate for less severe type 2 diabetes;
- No diabetes medicines and presumably managed by diet alone but using SMBG (category H)—a surrogate for early type 2 diabetes.

Note that the glucagon-like peptide-1 (GLP-1) agonist and dipeptidyl peptidase-4 (DPP-4) inhibitor classes of diabetes medicines (incretin mimetics, gliptins) are not funded in New Zealand.

Those patients who had received funded diabetes medicines or SMBG in 2011 were identified and then censored to comprise only those patients who had received the same script in the first and the last quarter of the year ('steady-state' patients); this was essentially a surrogate marker for those who had been on treatment for at least a year and likely to have been consistently accessing specific medicines and test combinations sustained

over 12 months (see endnote ῖ). After these steady-state patients were identified, they were further categorised according to type of medication regimen they were dispensed, alongside counts of test strips dispensed.

For these steady-state patients, we inferred current test strip usage (access) compared with expected, by comparing counts of dispensings to specific patient subgroups with expected use (i.e. clinical recommendations as to the number of times per day or week patients should be self-testing and therefore how many test strips should be dispensed to them).This analysis updated earlier analysis (2004)[14]—which had included epidemiological assumptions around patient mix and best-practice guidance for expected strip use—then in general applied those assumptions and methods[14] to the recent data on steady-state patients.

These definitions of expected SMBG use, originally based on advice during the mid-2000s (international guidelines at the time regarding insulin use, the expert focus group on SMBG convened by PHARMAC in 2004, the Diabetes Subcommittee of the Pharmacology and Therapeutics Advisory Committee (PTAC), the 2003 NZGG diabetes guidelines, and the BNF),[15–21]were since reiterated by the Canadian Agency for Drugs and Technologies in Health (CADTH) in 2009, the National Institute for Health and Clinical Excellence (NICE) and the Scottish Intercollegiate Guidelines Network (SIGN) in 2010, and the New Zealand Guidelines Group (NZGG) in 2011.[1–4]

Appendix 1, at the end of this article, summarises the workings and rationales for these groupings and provides expected test strip usages rates per patient/year for each group, where ideally in effect:

- At least 4 strips/day for patients stabilised on insulins without oral hypoglycaemic agents (OHs) (categories A,D,E);
- At least 2 strips/day for those on insulin with OHs (category C);
- 4 strips/week on average for those stabilised on sulphonylurea-containing OH regimens without insulins, with or without other diabetes medicines (category F);
- 3–4 strips/month on average for those stabilised on metformin alone, with or without glitazones (thiazolidinediones) or acarbose (category G); and
- 5 strips/month on average spread over the year for those stabilised on diet alone but using SMBG (category H).

Information included age, gender and ethnic group (see endnote §).We calculated median and mean dispensings and units for each patient group, both those dispensed SMBG and all (SMBG and no SMBG dispensings), with corresponding standard deviations and interquartile ranges across all strata. All remained anonymised.[12]

Results

A total of 2,624,405 dispensings of diabetes medicines and/or blood glucose test strips were recorded during the year 2011, in which 2,606,179 (99.3%) occurred for 181,342 known patients (i.e. dispensed to patients whose prescriptions contained their NHI number). With linear scaling (see endnote ‡), this means that approximately 183,000 patients were dispensed diabetes medicines and/or blood glucose test strips at some stage during the year 2011.

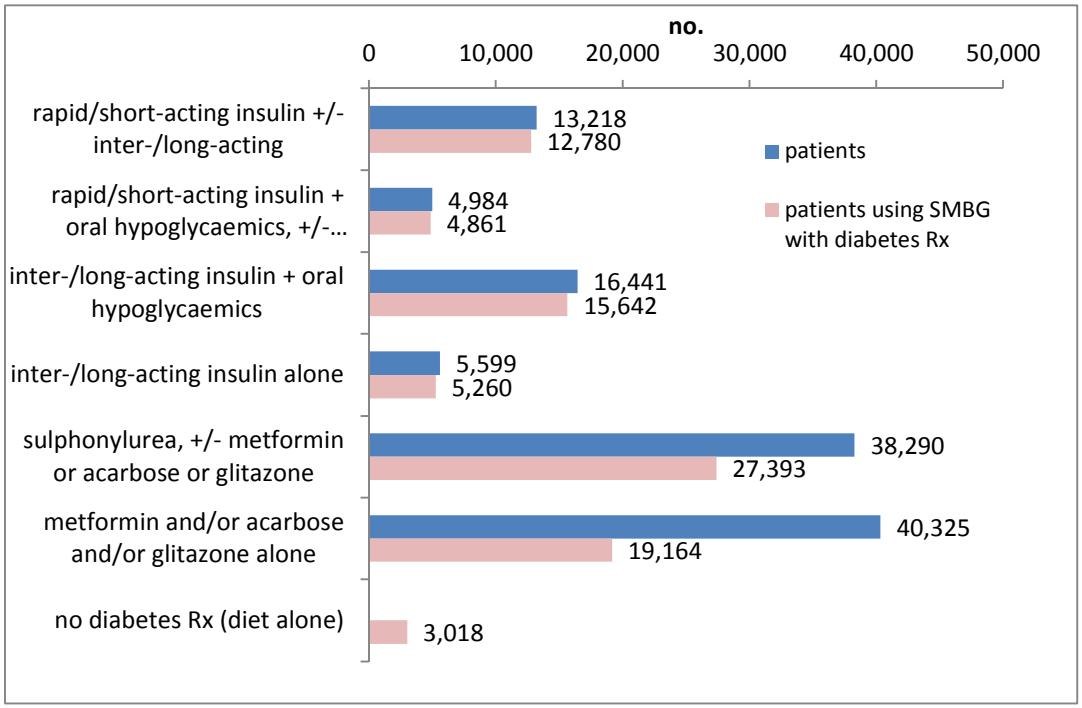
Of the 181,342 patients identified, about 122,000 received the same script combination in the first and fourth quarter of the 2011 year. Patients dispensed insulins and/or sulphonylureas (categories A-F) were likely to be such steady-state, but more than 2/5^{ths} and 4/5^{ths} of patients dispensed metformin (category G) or SMBG alone (category H), respectively, had different script combinations (endnote ῖ).

Patient numbers and median ages varied widely across treatment groups. Of the ~122,000 steady-state patients, 11% were dispensed category A (rapid/short-acting insulin ± inter-/long-acting insulin), compared with 33% dispensed category G (metformin and/or acarbose and/or glitazone alone), for example. Median ages varied between 48 years for category A (interquartile range 30–63 years) and 71 years for category E (intermediate-/long-acting insulin alone)(61–78 years).

Further details are provided in Figure 1 below, endnote § and in Supplementary results to this paper (at http://www.nzma.org.nz/__data/assets/pdf_file/0004/38290/SupplResults.pdf)

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1406/6371

Figure 1. Numbers of censored steady-state patients accessing diabetes medicines and/or blood glucose test strips during 2011



For those steady-state patients dispensed test strips, we observed (amongst other things):

- Patients who had no identified diabetes medicines who were dispensed test strips (category H) received on average 18.8 test strips per patient per month (median 17, interquartile range 13-21);
- Those on metformin alone (category G) who were dispensed test strips received 12.7 test strips per patient-month (median 8 [4–17])—where nearly half (19164/40325) of steady-state metformin alone patients were dispensed test strips (see Table 1).

Table 1. Dispensings of blood glucose test strips in 2011 to steady-state patients

SMBG users, steady state:	patients		strips/month	
	no.	% SMBG	median (interqrtle range)	mean (+/- 1 SD)
[A] rapid/short-acting insulin +/- inter-/long-acting	12,780	97%	100.0 (54-150)	112.0 (77.0)
[C] rapid/short-acting insulin +/- inter-/long-acting + oh	4,861	98%	58.3 (33-100)	72.0 (50.5)
[D] oral hypoglycaemics + inter-/long-acting insulin only	15,642	95%	37.5 (25-63)	45.5 (31.5)
[E] inter-/long-acting insulin alone	5,260	94%	50.0 (25-75)	55.1 (40.0)
[F] sulphonylurea +/- metformin or acarbose or glitazone	27,394	72%	16.7 (8-29)	21.9 (19.1)
[G] metformin and/or acarbose and/or glitazone alone	19,164	48%	8.3 (4-17)	12.7 (10.1)
[H] no diab Rx	3,018	91%	16.7 (13-21)	18.8 (13.0)
subtotal, SMBG users	88,119	72%	34.9	41.8
no SMBG	33,761			
total	121,879			

- For those on rapid/short-acting insulin, with or without intermediate/long-acting insulins (category A) there were 19.3 million strips dispensed, being 2.1 million strips (-11%) less than expected from 4 strips/day ideal (where 3.3% (438/13218) of these patients were not dispensed test strips)(see Table 2).

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Overall test strip dispensing in steady-state patients was ostensibly 8% less than what it should have been (see Figures 1 and 2 and Table 2 below):

- Stabilised patients using insulin ± oral hypoglycaemics (categories A–E) ostensibly under-used test strips, dispensings being 14% less than expected;
- Likewise dispensings were 9% less than expected for sulphonylurea ± metformin or acarbose or glitazone (category F);
- At the same time, steady-state actual test strip use was three times that expected for metformin and/or acarbose and/or glitazone alone (category G); and possibly up to two to five times higher for diet alone (category H).

Table 2. Actual vs expected use of blood glucose test strips in 2011in steady-state patients, based on clinical recommendations

category		units test strips	no. patients no strips	strips	total	Actual test strips /user /month	avg, all pts/ mnth
1. insulin needing >2 SMBG/day							
rapid/short-acting insulin +/- inter-/long-acting	A	17,180,892	438	12,780	13,218	112.0	108.3
2. inter-/long-acting insulin +/- oh needing 2 SMBG/day							
rapid/short-acting insulin +/- inter-/long-acting + oh	C	4,199,276	123	4,861	4,984	72.0	70.2
oral hypoglycaemics + inter-/long-acting insulin only	D	8,539,838	799	15,642	16,441	45.5	43.3
inter-/long-acting insulin alone	E	3,478,962	339	5,260	5,599	55.1	51.8
3. oral hypoglycaemics w/o insulin							
sulphonylurea +/- metformin or acarbose or glitazone	F	7,213,167	10,897	27,393	38,290	21.9	15.7
metformin and/or acarbose and/or glitazone alone	G	2,918,271	21,161	19,164	40,325	12.7	6.0
4. no diabetes Rx	H	679,285		3,018		18.8	
total		44,209,692	33,757	88,118	121,875	41.8	30.2
- subtotal, diabetes Rx		43,530,406	33,757	85,100	118,857	42.6	30.5

(continued)

category		Expected test strips no./pt/day	avg /pt/month	total strips/yr	Actual vs expected % variation	no. strips/yr
1. insulin needing >2 SMBG/day						
rapid/short-acting insulin +/- inter-/long-acting	A	4.0	122.0	19,298,591	-11%	-2,117,698
2. inter-/long-acting insulin +/- oh needing 2 SMBG/day						
rapid/short-acting insulin +/- inter-/long-acting + oh	C	2.0	61.0	3,638,440	15%	560,836
oral hypoglycaemics + inter-/long-acting insulin only	D	2.0	61.0	12,001,849	-29%	-3,462,011
inter-/long-acting insulin alone	E	2.0	61.0	4,087,176	-15%	-608,214
3. oral hypoglycaemics w/o insulin						
sulphonylurea +/- metformin or acarbose or glitazone	F	0.56	17.2	7,886,412	-9%	-673,245
metformin and/or acarbose and/or glitazone alone	G	0.07	2.2	1,051,324	178%	1,866,947
4. no diabetes Rx	H	0.12	3.7	133,027	411%	546,258
total		2.52	76.9	48,096,818	-8%	-3,887,127
- subtotal, diabetes Rx		2.53	77.1	47,963,791	-9%	-4,433,385

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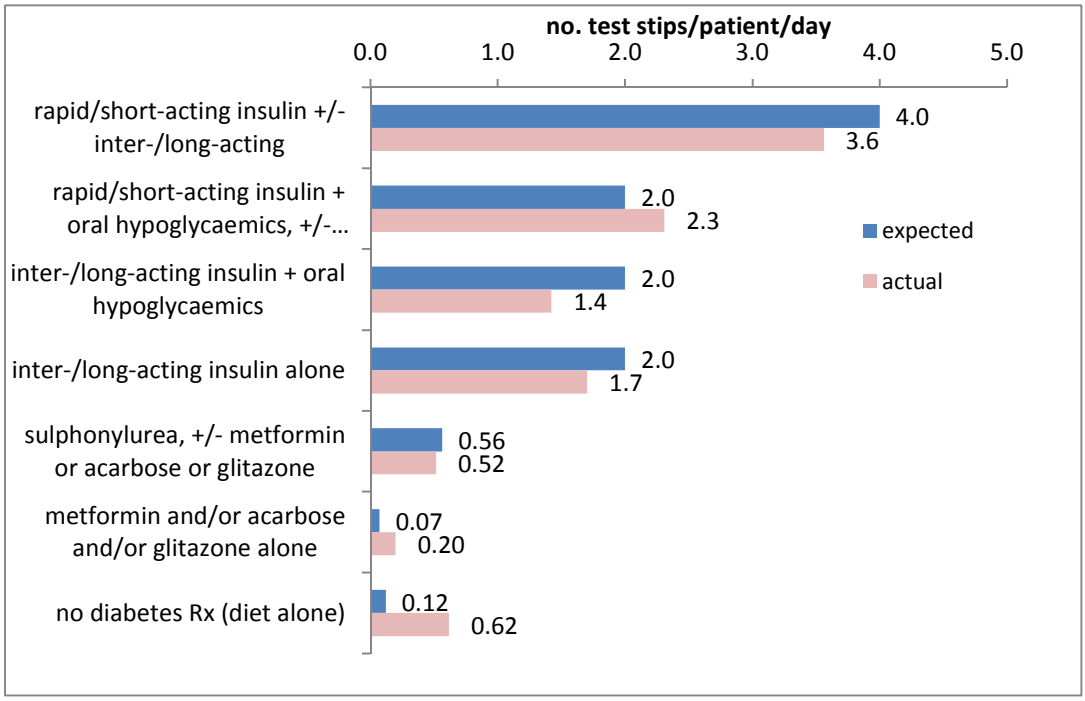
Figure 2. Actual vs expected use of blood glucose test strips in 2011 (steady-state patients), based on clinical recommendations



Beyond these overall patterns by group (categories A-H), Māori and Pacific peoples in the 25-44 age-group were appreciably under-dispensed in some insulin-containing regimens (categories A, D).

Further information is available in Supplementary results to this paper.

Discussion

This study was designed to examine which patient groups actually received SMBGs and whether this ostensible usage was consistent with guidelines, such as they are.

During 2011 there appeared to be both under-dispensing of test strips among some patient groups as well as over-dispensing in other groups.

New Zealand is fortunate to have its pharmaceutical databases reliably providing dispensing information at a national level. National and international clinical guidelines and advice indicate that there is usually little need for patients maintained on diet/exercise or metformin alone to be routinely self-monitoring their blood glucose;[1–8,22–24] however, the expenditure on test strips in these two patient groups has been at approximately $2.6 million per year. Unfortunately it is not possible to estimate from the dispensing data what proportion of non-medicated people with diabetes use test strips.

Limitations—The study's main strength is that it links patterns of dispensing of diabetes medicines with the related use of SMBG, at a national level.

Limitations that affect validity include:

- The study is confined to patients with diabetes identified from diabetes medicines and/or blood glucose test strip dispensings. This method omits diabetes patients who are not currently treated with diabetes medicines or dispensed test strips, treated instead by diet alone and clinically monitored by periodic HbA1c testing (not SMBG).

- The data are dispensing-based, not based on prescription-at-doctor-visit nor patient end-use (see endnote **).
- Diagnoses are by inference and will include other conditions where diabetes medicines are used, including polycystic ovary syndrome;[25] however the numbers of these cases are likely to be relatively small.
- The steady-state grouping reflects patients being dispensed any diabetes medication and/or test strips in the first three and last three months of the year; this omits where patients' medication regimens may have changed during the year.
- Patients were grouped hierarchically according to all medicines received over the year, rather than their specific regimen at any one point in time (or their weighted average regimen over the entire 12 months).
- The analysis has not attempted to link SMBG usage with laboratory data such as HbA1c measurements of glycaemic control, nor has it considered the full range of demographic and clinical information—socioeconomic deprivation, region, type of diabetes, renal function, other macrovascular and microvascular complications, neuropathies, use of other medicines (e.g. ACE inhibitors, statins), etc.—to better elucidate key patterns and gaps in the treatment of patients with diabetes.

Note that the audit period (2011) was when conceivably patients may have been aware of oncoming changes in the meter/strip supplier,[11] with potential stockpiling of strips and thus seemingly higher SMBG dispensings. Most patients however would have been unaware of the possible changes until early 2012 (i.e. after the audit period), when consultation began.[26]

Comparisons with usage rates elsewhere including for SMBG—This analysis provides a nationwide perspective of diabetes medication and SMBG dispensing patterns in New Zealand. Dispensing rates and patterns appear to be largely comparable with that of recently published series overseas:

- Reports of Tasmanian data (1995–97, 2001) indicate similarly persistently high rates of SMBG usage amongst respondents with insulin-treated diabetes, with 98% reporting any self-monitoring (c.f. 95% usage in this analysis—40230/46816 for insulin alone in New Zealand 2011) and 74% of respondents stating they self-monitored daily.[27,28]
- A postal survey in Scotland (60% response rate) reported 87% of patients using diabetes medications used SMBG (c.f. 64% in NZ 2011), with higher rates in insulin users.[29]
- Western Australia data (1993-96) reported 71% of patients with type 2 diabetes using SMBG (c.f. NZ 2011 68% for patients with possible type 2 diabetes).[30,31]
- Canadian data from Ontario (for all ages[32,33] and for those aged 65+[34]) and the elderly in Nova Scotia[35] suggest similar proportions of diabetes medicines use as occurs in New Zealand (Ontario[32,33]), increasing use of SMG over time (Ontario elderly[34]) and more marked variation from expected for oral hypoglycaemics and diet alone than occurs in New Zealand; endnote ††provides further information.

Implications—This analysis provides population level commentary that will not necessarily reflect individual clinical or personal circumstances. Advice from clinicians in the field is that patients may over- or under-use due to their personal preference with respect to their diabetes.[36] In addition clinicians often initiate newly diagnosed type 2 patients on test strips so that patients can see what effect diet and exercise have on their blood glucose.[36]Our calculations have taken this into account, allowing 3 months of daily use in the 62% of patients expected to dose-escalate in any year; however dispensings were still at least double expected despite this adjustment. Numbers can be substantial when converting daily use (strip counts) to bottles per year.

Clinicians have also noted that patients currently have ample opportunity to access test strips and that it is up to the clinician and patient to agree to the appropriate level of testing; however long-term testing may be damaging, as increased testing may lead to increased anxiety, unnecessarily.[9,36,37]Information on prescriber and patient consumer perspectives would be helpful (see endnote ‡‡).

Given the view that those people on insulin have appropriate information and access to test strips, it may be that there is no need to have specific targeting campaigns. General education on the appropriateness of ongoing testing is being undertaken, for instance by the Best Practice Advocacy Centre (bpac^{nz})[22] and such information can reinforce these education messages. One such message for patients with type 2 diabetes could be only to test if they are then likely to change what they do, this advice being consistent with that of CADTH, NICE, SIGN, NZGG, and the American Diabetes Association.[1–4,38]

Conclusions

This study compared publicly funded blood glucose test strip uptake in steady-state (stabilised) patients against published guidance on appropriate rates of usage. There was continuing both under-accessing of test strips among some patient groups and over-accessing in other groups.

While some of the ‘under-use’ may be appropriate to patients’ circumstances, persisting patterns of ostensible under-use (especially in younger Māori and Pacific peoples on insulin) are cause for concern and require further research, in the context of measures of diabetes control and long-term clinical outcomes.

Conversely, while again some ‘over-use’ may prove clinically acceptable, the continued accessing of expensive testing regimens with little evidence of benefit but good evidence of cost represents possible unintended harm.[9,24]

Competing interests: Scott Metcalfe is a member of the NZMA Services Board.

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Endnotes

* The study was an observational study (being an audit observing outcomes without controlling study variables nor having an intervention) with the secondary use of data for quality assurance/outcome analysis/resource review undertaken by people employed by the service provider holding the information (PHARMAC) and where participants remain anonymous. It did not meet criteria for requiring ethics committee review.[12]

î Patients were groupable into two groups. These comprised:

1. stable treatment (steady-state) patients, being those patients who likely consistently accessed the same specific medicines and test combinations sustained over 12 months (proxied by the same combination in the first and last quarters of the year), and
2. changing treatment (dynamic) patients, being those who started, stopped or changed specific medicines and test combinations during the year (death; medication changes; adherence; etc.) or who otherwise were not dispensed the same specific medicines and test combinations during both the first and last 3 months of the 12 month period.

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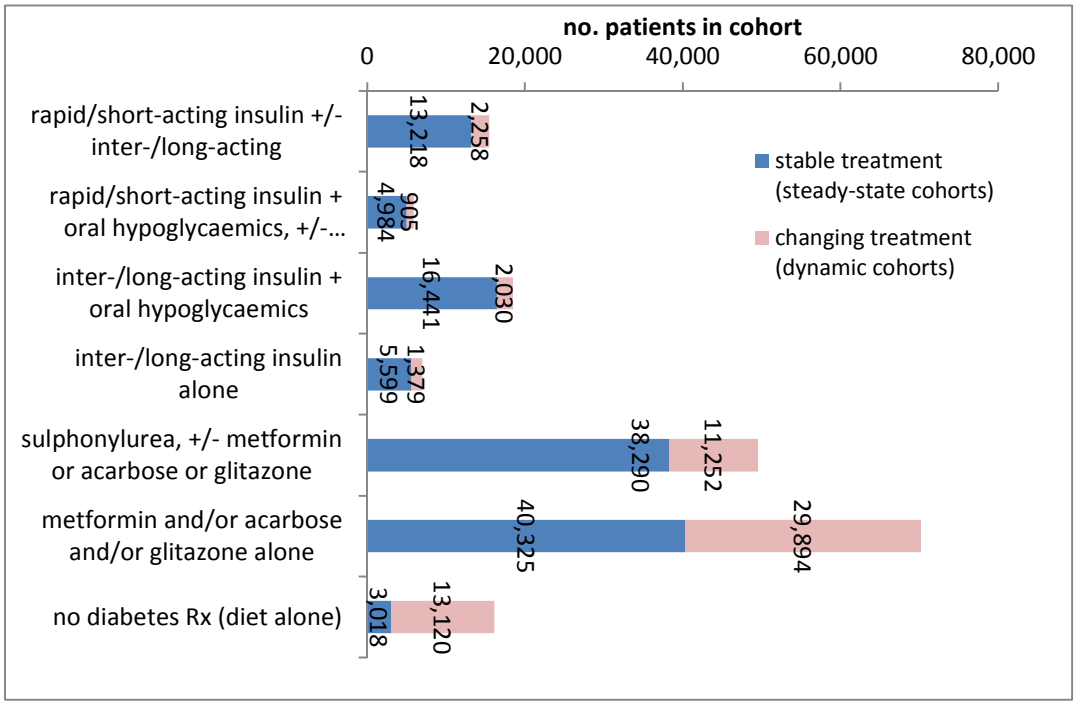
This analysis was confined to censored stable treatment (steady-state) patients. There were some 122,000 steady-state patients and 61,000 changing treatment (dynamic) patients. Of 16,200 total patients on diet &exercise alone, 3,000 of whom accessed test strips throughout the year (steady-state), the other 14,000 (dynamic) accessing test strips for just part of the year (consistent with use when establishing new regimens or starting late or ending before the year's end); some patients on diet & exercise alone (of indeterminate number) would have been monitored alone by HbA1c, not using SMBG.

Steady-state (stabilised) vs dynamic patients accessing diabetes medicines and/or blood glucose test strips 2011



‡ Scaling occurred to adjust patient numbers to account for scripts with missing NHI numbers, based on total units dispensed / units dispensed on scripts with known NHIs. Scaling therefore used linear interpolation, where the scaled no. scripts for a medicine for an age/sex/ethnic group = no. scripts with NHI numbers for that medicine for an age/sex/ethnic group × total scripts for medicine ÷ total scripts for medicine with NHI numbers.

§ Patient numbers and median ages of treatment groups varied widely by ethnicity. By prioritised ethnicity,[39]Europeans comprised 52 to 82% of all patients (category D vs category A; median ages 49-74 years). This compared with Māori whose range was 6-19% (category H vs. D, median 41-67 years). Pacific peoples ranged 3-18% (category A vs D, median 45-64 years), and Asian patients ranged 3-14% (A vs H, median 49-61 years).

In common with many analyses undertaken within the health sector, this analysis used prioritised ethnicity. With this 'prioritised output' system, adopted in earlier years by Statistics New Zealand, each person is identified as belong to just one ethnic group, prioritised in a hierarchy by Māori first, etc. (i.e. all individuals identifying as Māori (including those also identifying with other ethnic groups) are coded as Māori; all those identifying as Pacific peoples, other than those also identifying as Māori, are coded as Pacific peoples; etc.), and so on.[39] Problems with prioritised ethnicity, which Statistics NZ no longer supports (recommending since 2004 against its use) nor provides publicly, have been summarised previously in the *Journal*.[39] Alternatives include the use of sole ethnic groups.

** Dispensing based data (rather than prescription-at-doctor-visit or patient end-use) do not capture end-use (i.e. whether medicines dispensed are actually taken by the patient—wastage and suboptimal treatment), nor prescriber intent (since not all prescriptions are necessarily dispensed and captured in the data).

ïï Table and figures comparing diabetes medicines and SMBG use in New Zealand 2011 with Ontario2006.[33]

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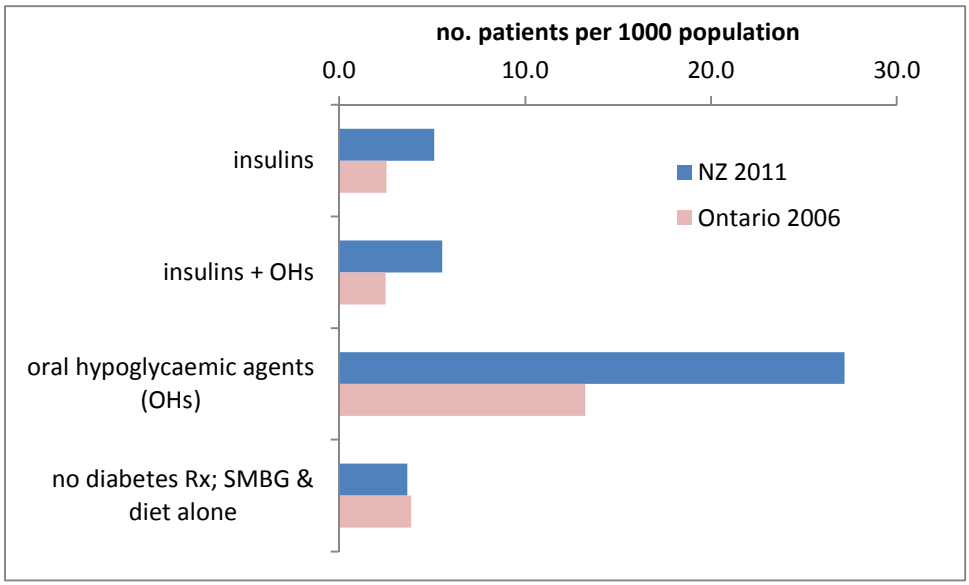
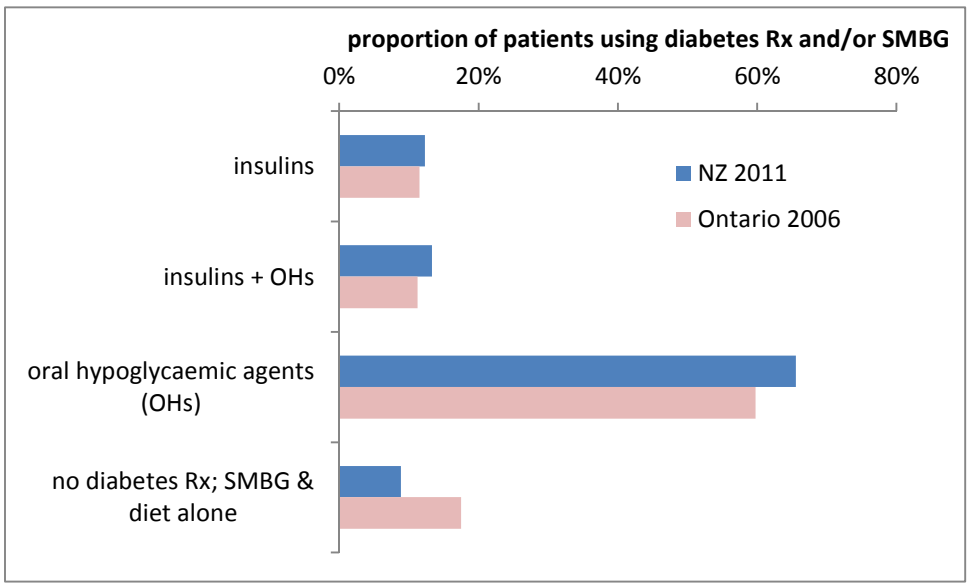
category	patients no.		prvl per 1000		distribution		strips no.		prvl per million		strips/pt/day expected	actual		% variation from ideal	
	NZ 2011	Ontario 2006	NZ 2011	Ontario 2006	NZ 2011	Ontario 2006	NZ 2011	Ontario 2006	NZ 2011	Ontario 2006	(guidelines)	NZ 2011	Ontario 2006	NZ 2011	Ontario 2006
insulin alone	22,457	30,959	5.1	2.5	12%	11%	22,526,657	31,198,044	5.1	2.6	3.0	2.7	2.8	-8%	-7%
insulin + OHs	24,360	30,214	5.5	2.5	13%	11%	13,793,164	25,589,681	3.1	2.1	1.8	1.6	2.3	-13%	30%
OHs alone	119,786	160,938	27.2	13.2	66%	60%	12,329,643	79,151,388	2.8	6.5	0.2	0.3	1.3	74%	732%
no diabetes Rx; diet alone	16,139	47,124	3.7	3.9	9%	18%	2,057,136	17,079,794	0.5	1.4	0.1	0.3	1.0	182%	701%
total	182,741	269,235	41.5	22.1	100%	100%	50,706,600	153,018,907	11.5	12.6	0.7	0.8	1.6	5%	116%





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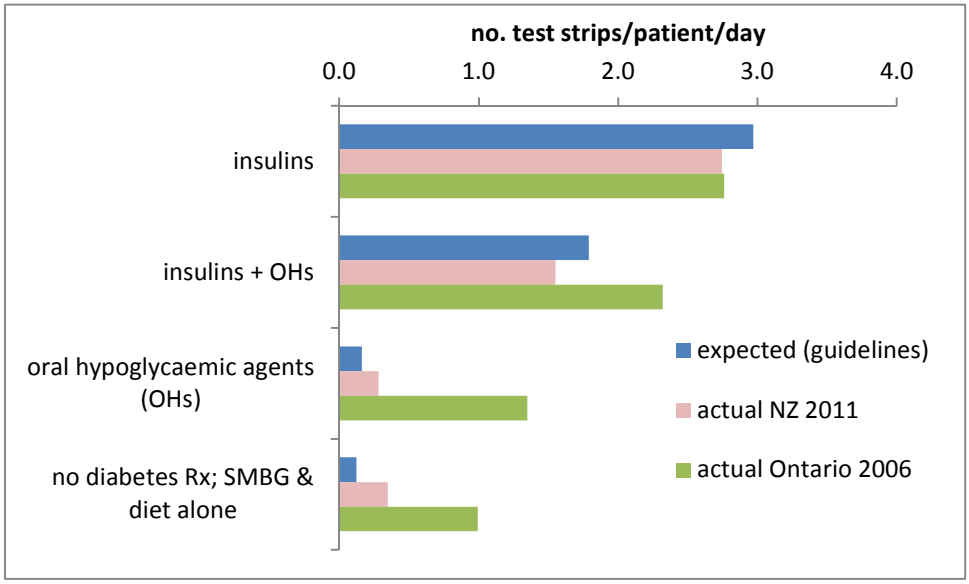

‡‡ Patients on metformin or diet alone, part of the group that is arguably over-testing, did not have funded access to blood glucose meters, so they would have been receiving meters from their doctor (who would in turn have received them from the pharmaceutical supplier) or by paying for them privately.

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Appendix 1. Expected SMBG usage for medicines-related groups

To examine actual versus expected use of SMBG, five mutually-exclusive hierarchical groups of steady-state (stabilised) patients were defined according to the following treatment-based hierarchy, with associated assumptions and calculations for average SMBG test strip use per patient-year:

Groups:

1. rapid/short-acting insulin ± intermediate-/long-acting insulin (categories A,D,E*);#1

2. oral hypoglycaemics (OHs) with insulin and intermediate-/long-acting insulin (± rapid/short-acting insulin) (category C*);#2

3. sulphonylurea-containing oral hypoglycaemic regimens without insulin—i.e sulphonylurea ± metformin or acarbose or a glitazone) (category F);#3

4. residual non-sulphonylurea oral hypoglycaemics w/o insulin—i.e metformin and/ or acarbose alone and/or glitazones (category G);#4 and

5. no diabetes Rx (diet/exercise alone) (category H).#5

* note there is no category 'B'.

Assumptions/calculations for average SMBG test strip use in steady-state (stabilised) patients per patient-year:

#1. rapid/short-acting insulin ± intermediate-/long-acting insulin (categories A,D,E)
= using rapid/short-acting insulin ± intermediate-/long-acting insulin but not when combined with oral hypoglycaemics (suggesting less brittle control):
ideally x4/day

#2. oral hypoglycaemics (OHs) with insulin and intermediate-/long-acting insulin (+/-rapid/short-acting insulin) (category C):
ideally x2/day

#3. sulphonylurea-containing oral hypoglycaemic regimens without insulin (category F):
ideally 4- to 8- per week (based on BNF 47 and NZGG diabetes guidelines) for patients with HbA1c ≥7.0% (59% in "Get Checked" for 2003),
perhaps once a week if HbA1c <7.0% (41%) (higher if HbA1c >8.0%, contemplating insulins),
= weighted average of 0.56 per day

#4. residual non-sulphonylurea oral hypoglycaemics w/o insulin (category G):
ideally this would be perhaps once a fortnight (broadly based on SIGN, NZGG diabetes guidelines and PTAC subcommittee advice);
however, perhaps 15% of patients need to escalate their treatment regimens, hence for these patients ideally one per day (in 15% of patients) whilst contemplating regimen escalation (broadly based on BNF 47 and NZGG diabetes guidelines and previous PHARMAC analysis), occurring over a 3-month period;
hence overall weighted average of 0.11 per day (15% *3/12 * daily + [85%+ (15%*9/12)] * every 14th day), i.e. ~0.8/week on average

#5. no diabetes Rx (diet/exercise alone) (category H):
for patients well controlled on diet alone, nil strips (i.e. HbA1c monitoring alone) aside from perhaps daily testing for 3 months following diagnosis (awareness raising);
for patients with poorer control contemplating regimen escalation, ~25% of all diet alone, ideally daily testing for the 3 months (broadly based NZGG diabetes guidelines),
hence = ~1.2/week on average

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ORIGINAL ARTICLE

Evaluation of a rural primary-referred cardiac exercise tolerance test service

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Abstract

Aim To describe the feasibility, clinical impact and cost-effectiveness of a rural generalist-led cardiac exercise tolerance test (ETT) service for primary care patients in two different rural communities.

Method For 12 months, from Sept 2011, a generalist-led ETT service was provided in two rural hospitals in New Zealand: Dunstan in Central Otago and Rawene in Northland. Data was collected to describe the patient outcomes of this service. An audit of ETT reports and financial and rural-urban analyses were undertaken.

Results The cost per test of the local ETT service at Dunstan, ($132.50), and Rawene, ($200.00), was less than the national price ($281.13 in 2012). The majority of patients (83% at Dunstan and 70% at Rawene) were not referred to specialist services; the ETT result allowing the GP to continue to manage the patient in primary care. Where the ETT indicated specialist treatment, this was subsequently provided in a timely manner.

Conclusion : ETT can be provided cost-effectively in a variety of rural settings. Improved access to ETT for rural communities may help address inequities across New Zealand in terms of access to cardiac investigations and early and appropriate treatment.

Cardiac exercise tolerance test (ETT) is often the first step when investigating a patient with suspected ischaemic heart disease (IHD) or risk stratifying a patient with known IHD. The results of ETT often determine whether or not a patient proceeds to more complex cardiology investigations and treatment.

In New Zealand public ETT services are generally provided in urban and provincial hospitals with specialist oversight. Few rural hospitals (only five in New Zealand at the time of this study, including the two in this study), offer ETT services.

There is little information on the impact of distance on the utilisation of urban based secondary services in NZ. This is in contrast to primary care where inequalities are acknowledged and there is both research and policy aimed at improving access for rural communities.[1]

Published evidence (both NZ and international), suggests rural patients have inferior access to cardiovascular diagnostic investigations including ETT compared with urban patients and have poorer outcomes as a result.[2–9] Despite a significantly higher prevalence of IHD in Māori vs non Māori, intervention rates for Māori are low.[10,11] Combinations of rurality, Māori ethnicity, and socioeconomic deprivation reflect groups with increasingly poor access to services including coronary angiography.[1,12–15]

ETT is a relatively low-cost, non-invasive diagnostic test that has the potential to be widely available at a community level. A recent Australian study looking at the utility of ETT in a remote setting concluded that ETT is a particularly useful tool for the diagnosis of IHD in areas where onsite specialist cardiology services are limited.[16]

Using Ministry of Health (MOH) Rural Innovation Funding we provided an ETT service for two different rural communities (Central Otago and Hokianga), from their respective rural hospitals for 12 months.

The tests were conducted by specifically trained local medical and nursing staff for patients referred directly by their GP.

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The aim of the MoH-funded ETT service project was to improve access to ETT for these communities in a way that was cost-effective and did not compromise standards of care.

Methods

Background

The catchment population of Dunstan Hospital is approximately 25,000, encompassing Central Otago District and the Wanaka part of the Queenstown Lakes District. It is classified as rural/remote, (Rural Ranking Score (RRS) between 55 and 90). Six percent of the population is Māori. It has deprivation indices ranging from 2 to 7. Dunstan Hospital is operated by Central Otago Health Services Ltd (COHSL). Services include a generalist inpatient unit and a range of visiting specialist outpatient clinics. Base hospital and interventional cardiology services are provided 200 km away at Dunedin Public Hospital, Southern District Health Board (SDHB).

Prior to this project, patients needing an ETT were referred to the visiting cardiology clinic or, if necessary, admitted acutely to Dunstan Hospital. ETTs were conducted as part of the cardiology clinic and occasionally for in-patients. Local staff supervised the tests, all of which were then read by a Dunedin based visiting cardiologist.

The Hokianga area is rural/remote with a RRS of 65. The population is 6500, 74% of the population is Māori and the deprivation index is 10. The Hokianga Health Enterprise Trust (HHET) provides integrated health services for the area, including the hospital in Rawene. Base hospital services are provided 130 km away at Whangarei Hospital, Northland District Health Board (NDHB). The nearest interventional cardiology centre is 280 km away in Auckland. With the exception of emergency transfers, there is no direct referral pathway from Rawene Hospital to Auckland.

Visiting medical clinics (every second month) are part of the small range of outpatient clinics provided at Rawene Hospital by specialists from Whangarei Hospital. There is no on-site ETT equipment at Rawene Hospital. Prior to this project, Hokianga patients needing an ETT were first referred to a specialist outpatient clinic either at Rawene or Whangarei and would then have to travel to Whangarei Hospital, (or occasionally to Kaitaia), for the ETT. Those requiring an ETT acutely were transferred to Whangarei Hospital.

Description of the service

Clinical protocols were drawn up by the rural hospitals with input from the participating specialists, according to established guidelines.[17] General Practitioners were advised of the referral process by letter. A standardised clinical record form was completed by the generalist for each ETT.

ETT clinics were held once a week at Dunstan Hospital using on site equipment.

ETT clinics were held every 6–8 weeks in Rawene coinciding with the visiting general physician's outpatient clinics. The same physician bought portable ETT equipment from his practice and made it available to Hokianga Health to use on this project, free of charge, in conjunction with an onsite treadmill. At both sites the tests were supervised by local generalist doctors and nurses.

Testing commenced in September 2011 and continued through to November 2012 (Dunstan) and September 2012 (Rawene).

At Dunstan Hospital tests were read by a rural hospital generalist. The generalist provided a report for those tests where he felt confidently able to do so. Equivocal tests were sent on to the cardiologist for reporting.

At Rawene Hospital, all reporting was done by the rural hospital generalist and each report verified by the physician on the day.

All reports were sent to the patient's GP and to the referring doctor, (if these were not the same.)

In both Dunstan and Rawene, the majority of patients were referred back to their GP to discuss their results and for ongoing management. On occasions, where clinically indicated, the patient was referred directly for specialist care from the ETT clinic.

With clinics only possible every 6–8 weeks in Rawene, any referrals which were deemed more urgent were referred to Whangarei Hospital.

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Study design

In this study we documented the patient outcomes [ongoing GP management, referral to cardiologist, percutaneous intervention (PCI), Coronary Artery Bypass Grafting (CABG)]; audited the reporting of ETT by generalist doctors; determined the tangible costs and attempted to determine whether or not there was any rural - urban difference in the utilisation of ETT.

Ethics committee approval was obtained from the Lower South Regional Ethics committee with respect to the reading of the tests LRS/11/EXP/002.

Data was collected from the standardised clinical record form including the reason for referral, test result, patient disposition post-ETT and basic demographic data. We categorised each test result using conventional Criteria.[17,18] These data were then collated, reviewed and tabulated.

An audit was undertaken of the ETT reports generated at the Dunstan site. In cases where the rural generalist did not feel confident about finalising the report, a provisional local report and the test were sent to the cardiologist for formal reporting. The provisional report and the cardiologist's report were compared.

An additional 25 tests where only a local report had been generated were sent to the cardiologist for formal reporting at the end of the study. The tests were ordered according to the day and time they were performed and every sixth test was selected. The cardiologist report was then compared to the locally generated one. The cardiologist was blinded to the local report.

A simple financial analysis was undertaken to determine the tangible costs of the service.

We planned to collect data from the relevant DHB's on the utilisation of ETT one year prior to and during the project. The intention was to identify any rural urban disparities and the impact this study had on these disparities.

Results

Patient demographic, referral and outcome data

Over a period of 12 months, 202 ETTs were carried out at Dunstan Hospital and 33 at Rawene Hospital. Three (1.4%) of the Dunstan patients and 22 (67%) of the Rawene patients identified as Māori.

The most common reason for the ETT was for diagnosis in patients with suspected IHD. A smaller number were referred because of arrhythmia or risk assessment in a patient with known IHD.

At Dunstan, 31 patients (15%) had a positive test result. Eighteen out of these 31 patients were referred directly to the public cardiology clinic (one was admitted acutely to Dunstan Hospital first), and another 7 were referred to a private cardiologist. The remaining 6 were returned to GP care following a discussion between the rural hospital doctor and the cardiologist.

Seventeen of the patients with positive tests proceeded to angiography and of these five had PCI, four had CABG and eight had angiograms with no flow-limiting stenoses (NFLS). Four patients (3%) with negative tests were also subsequently seen by cardiologists and one went on to have an angiogram with NFLS. Five of the 12 patients with equivocal tests were referred to cardiologists (two public and three private). Only one of these patients went on to have angiography (the result showed NFLS). All the remaining patients returned to their GP for follow up.

At Rawene, two patients (6%) had positive tests; both were seen by the visiting physician on the same day as the ETT and referred directly to Auckland for angiography. One was referred urgently and subsequently underwent CABG. The other was referred semi-urgently; the angiogram showed NFLS.

Of the 21 negative tests, (63%), all but one were returned to GP care. One patient with a negative ETT at full workload was referred to cardiology in view of a suspicious history despite the negative ETT, was referred on for angiography and went on to have PCI .

Two patients had an arrhythmia during the ETT, were seen by the physician on the same day and referred for further investigations. Six tests (18%) were suboptimal, where the patient was unable to

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exercise long enough to get to their target heart rate. Three of these were referred on for further testing after discussion with the physician. Please refer to Table 1 below.

Table 1. ETT and patient outcomes in Rawene and Dunstan Hospitals

Dunstan Hospital			Referred to:			Angiography / Intervention				
Test result	N	GP	Private cardiologist	Public cardiologist	Admit ward	Urgent	Routine	No FLS	PCI	CABG
Negative	153	149		4		1		1		
Positive	28	3	7	18	1	1	16	8	5	4
Symptom positive	3	3								
Equivocal	12	7	3	2			1	1		
Arrhythmia	2	1			1					
Suboptimal	4	4								
Total	**202**	**167**	**10**	**24**	**2**	**2**	**17**	**10**	**5**	**4**

Rawene Hospital			Referred to:		Angiography / Intervention			
Test result	N	GP	Physician	Urgent/ semi-urgent	Routine	No FLS	PCI	CABG
Negative	21	20	1		1		1	
Positive	1		1	1				1
Symptom positive	1		1	1		1		
Equivocal	1		1					
Arrhythmia	2		2					
Suboptimal	6	3	3					
Exercise capacity	1		1					
Total	**33**	**23**	**10**	**2**			**1**	**1**

Audit – reporting of test results

The generalist doctor reporting the Dunstan tests felt confident in providing a final report for 149 (74%) of the tests. For 53 tests (26%), (which were predominantly equivocal tests), the generalist wrote a preliminary report before the test was sent on to the cardiologist for definitive reporting. For the majority of these 53 tests (39 tests, 73%), there was no variation between the rural hospital generalist's preliminary report and the cardiologist's final one. For 11 tests (23%) the cardiologist downgraded the test (changed the result from equivocal to negative or positive to negative). For three tests (6%) the cardiologist upgraded the report.

There was 100% agreement between the local report and the cardiologists review for the 25 locally generated final reports that were randomly selected for audit at the end of study.

Rural – urban differences in ETT utilisation

We asked for domicile data on all patients receiving publicly funded ETTs in the year preceding the project and for the 12 months of the project. The SDHB was unable to retrieve this data from their systems because the ETT data is not collected in their main patient management system. Limited data was obtained from the NDHB from which an estimated ETT rate for Hokianga residents and urban Whangarei residents for 2010/11 and 2011/12 was extracted (see Table 2). This does not include the ETTs done at Rawene during this project. The utilisation rate for the urban Whangarei population was more than 1.5 times that of the rural Hokianga population.

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Table 2. Accessibility

District	Distance to ETT (km)	Closest ETT	ETT/1000 pop (2010–11)	ETT/1000 pop (2011–12)
Whangarei central	0–15 km	Whangarei Hospital	5.9	7.1
Hokianga	60–160 km	Whangarei or Kaitaia Hospital	3.8	4.4

Financial analysis

We calculated the direct costs of the service including doctor and nurse time, equipment costs, consumables, and cardiologist/physician oversight. The cost per test at both Dunstan ($132.50) and Rawene ($200) was considerably less than the national price of $281.13 (community referred tests-cardiology; purchase unit code MS00045 in the National Non Admitted Patient Collection [NNPAC] 2015).

The estimated real cost at Rawene was higher than at Dunstan due to the lower volume, the cost of loaning portable ETT equipment, and the specialist's reading of every ETT test . The actual cost during the project was lower in Rawene because the specialist provided his services free of charge.

Nearly all of the patients referred to these rural services would otherwise have been referred to specialist cardiology services. The national price for a cardiology First Specialist Assessment (FSA) in 2012 was $429.49.

Comparing the cost the system would have incurred for these patients had they all been referred to a specialist cardiology service and undergone ETT (using the national prices), we can demonstrate a potential savings of over $125,000 for this small cohort of patients. Please see Tables 3 and 4 below for a summary of this information.

Table 3. Estimated cost – rural generalist ETT followed by FSA if required

Rural generalist ETT followed by FSA if required				
No. of patients	**Dunstan ETT**	**Rawene ETT**	**Referred to FSA**	
202	$132.50 per patient			$26,765.00
33		$200.00 per patient		$6,600.00
20	(18 patients)	(2 patients)	$429.49 per patient	$8,589.80
Total cost to system				**$41,954.80**

Table 4. Direct referral for ETT/FSA estimated cost

Direct referral to FSA for ETT using national prices				
No. of patients	**Dunstan ETT**	**Rawene ETT**	**Referred to FSA**	
202	$281.13 per patient		$429.49 per patient	$143,545.24
33		$281.13 per patient	$429.49 per patient	$23,450.46
Total cost to system				**$166,995.70**

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Discussion

Over a period of 12 months we provided a cost effective local generalist ETT service for two rural communities. Though the organisation of the service at the two hospitals necessarily differed to fit existing local services and available resources, the essence of both was an easily accessible service provided by local clinicians.

Central to both was collaboration between the DHB and the local trust-operated rural hospital, and integration between primary and secondary care. This, along with the option to refer to private cardiology services (Dunstan), and an innovative link-in from private practice (Rawene), resulted in a more seamless patient journey and shifted care 'closer to home'.

Testing at both sites showed that the majority of patients did not require onward referral to specialist services, providing instead, useful information for the GP to continue to manage these patients in the community. For example the information from the ETT report could support intensified medical management of IHD or a non-cardiac cause.

Clinical judgment about a patient's ongoing symptoms over-rules an ETT result because of the test's limited sensitivity as was the case here for one Rawene patient.

Our negative ETT rate is consistent with that reported elsewhere.[18]

Our outcomes agree with previous findings that an appropriately trained and experienced generalist doctor is capable of safely reporting the majority of ETTs. They are also able to recognise those tests where the reporting is less straightforward and send them on to a cardiologist for further assessment and reporting.[19]

Cost

ETT is no more costly to the health system overall when run in a rural area, and significantly less costly for the patient.

Travel to a distant hospital for the patient (and their support networks), means not only direct travel and often accommodation costs but also time off work and other duties. Further, public transport is sparse or absent in rural areas. For the Hokianga community there is added meaning to cost of travel from a cultural perspective: Māori have intrinsic relationships with whenua (land and identity), their whanau (family contribution to their health), and tupuna (elders who provide security, wisdom and guidance) which are generationally driven and is how Māori value their living environment which itself sustains life. Maintaining these values through kaitiakitanga (caring) sustains the people, therefore being moved from a trusted health service to distant health services causes anxiety in regard to Kaitiaki o te kainga (caring for home and land) and can further impact on health outcomes.

Our financial comparison of specialist delivered ETT vs. generalist delivered ETT is subjective. When DHBs fund providers they may use the purchase unit price, or the actual cost of delivering the test. Whilst this may be subject to negotiation with the provider, the difference is not material. We can identify no additional costs to the District Health Boards by providing this service rurally.

Rural vs urban differences in ETT utilisation

We were not able to obtain data to assess the geographic variation in the utilisation of ETT in the SDHB region or the impact that this study may have had on this. The data obtained from the NDHB was limited. We also noted that the way rural residents' addresses are recorded in hospital systems is frequently not a good indicator of where the person actually lives, but instead often is their postal address—e.g. the closest store which could be several tens of kilometers away.

A recent NZ study identified large 'rural versus urban' disparities in the utilisation of computed tomography.[20] Good data is needed if the urban vs rural disparities in access to services are to be identified and corrected; DHBs should be encouraged to collect this data.

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Implications for rural services

Though we looked at only two rural areas and access to a single diagnostic test, this project illustrates the complexity and variation in systems within NZ in which rural clinicians and patients are required to operate. It suggests that the degree to which services are integrated across primary, secondary and tertiary care at the presenting rural facility has an important role in the delivery of downstream health care for that specific community.

Not only do these services and pathways seem fragmented and poorly co-ordinated, as has been noted elsewhere,[21,22] they do not appear to be related in any way to patient need.

An integrated regional approach to the provision of ETT services is needed if disparities in access are to be remedied.[2,17,18] A collaborative approach that involves local health service providers in clinical pathway development from the earliest stages will help ensure the service is locally appropriate and sustainable.[23, 24]

Transferability

We believe this is a sustainable way of delivering ETT services to rural communities that provides savings to the health system, improves access for rural patients and may help overcome some of the disparities in access to cardiac investigations faced by rural patients.

This service is capable of being replicated at other rural sites pending small investments in rural generalist training and equipment.

Competing interests: Nil.

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ORIGINAL ARTICLE

Projecting future smoking prevalence to 2025 and beyond in New Zealand using smoking prevalence data from the 2013 Census

Frederieke S van der Deen, Takayoshi Ikeda, Linda Cobiac, Nick Wilson, Tony Blakely

Abstract

Background We have previously published a forecasting model of future smoking prevalence in New Zealand (NZ). Under business-as-usual (BAU) assumptions NZ's smokefree 2025 goal was not attained by any demographic group. However, the 2013 Census (which included a question on smoking) showed a greater than expected fall in prevalence, especially for Māori. We therefore aimed to provide upgraded projections to inform policy around tobacco endgame planning.

Method The previously developed dynamic forecasting model was re-specified using smoking prevalence data from the 2006 and 2013 censuses from NZ. Calculations included changes in initiation by age 20 years, and net annual cessation rates, by sex, age, and ethnicity (Māori vs non-Māori). Projections under 2006–2013 trends (adjusted for no tax rises since 2010), by sex and ethnicity were made out to 2025 and beyond.

Results Between the 2006 and 2013 censuses (adjusted for no tax rises since 2010), initiation of daily smoking by age 20 years decreased annually by 3.4% (95% uncertainty interval 3.2% to 3.6%) and 2.7% (2.5% to 2.8%) for non-Māori men and women, and by 2.9% (2.6% to 3.2%) and 3.2% (2.9% to 3.5%) for Māori respectively. Annual net smoking cessation rates ranged from 3.7% to 7.7% across demographic groups. The revised projected smoking prevalence in 2025 (allowing for tax increases that have occurred from 2010 to 2014) was 8.3% and 6.4% for non-Māori, and 18.7% and 19.3% for Māori men and women, respectively.

Conclusions The upgraded smoking prevalence projections still suggests that the NZ Government's smokefree 2025 goal would not be attained by any demographic group. It is likely that more intensive existing interventions, or entirely novel ones, will be needed to achieve the 2025 endgame goal.

Internationally, there seems to be a growing interest in setting tobacco endgame goals – i.e. planning towards achieving close to zero prevalence of tobacco use within a specific time frame with the ultimate goal of eliminating the tobacco-related health burden in the foreseeable future.[1,2]

As of mid-2014, New Zealand is one of four countries that has a government with a smokefree national goal, to be achieved within the next 10 to 25 years.[3–6] The New Zealand goal is to (further) reduce the prevalence of smoking and the availability of tobacco products to minimum levels by 2025,[5] which is often interpreted as reaching a smoking prevalence of below 5%.[7]

In light of New Zealand's smokefree goal, and the persistently higher observed smoking rates in Māori compared to the general population,[8] it is important to explore if the 2025 target is achievable under current trends in smoking uptake and cessation. Exploring what it would take to get under 5% smoking prevalence for all New Zealanders by 2025, was therefore the main rationale for our analysisthat was published in 2013.[9]This modelling work suggested that under business-as-usual (BAU) assumptions, smoking prevalence in 2025 would be 11% and 9% for non-Māori men and women, and 30% and 37% for Māori respectively. However, at the time of this modelling work, 2013 Census data were not available.

Three data series on smoking prevalence from the more regularly conducted New Zealand Health Survey (NZHS) from 2002 to 2011 were therefore used to provide information on recent annual trends in smoking uptake and cessation as input for future BAU smoking prevalence projections.

These data, however, often had wide 95% confidence intervals around smoking prevalence estimates, especially for Māori.

Since this previous modelling work, smoking prevalence data from the New Zealand 2013 Census has become available,[10] and this reported a lower than expected smoking prevalence, especially for Māori. Between the 2006 and 2013 censuses, daily smoking rates fell from 20.7% to 15.1% in adults aged 15 years and over, and from 42.2% to 32.7% for Māori[11] (compared to a daily smoking prevalence of 17.0% for the general adult population, and 38.4% for Māori in the 2011/2012 NZHS).[12]

With these population-wide data (and the 2006 Census which also included a question on smoking), much more accurate information is now available to estimate changes over time in initiation and cessation rates. Therefore, in this new study we aimed to provide new BAU smoking prevalence projections to 2025 and beyond to inform tobacco endgame planning in New Zealand.

Methods

As detailed in the previous work,[9] the modelling approach of the dynamic tobacco forecasting model for New Zealand closely followed the approach for an Australian model by Gartner et al.[13]

In this paper, we describe the general modelling approach, but mainly focus on the latest iterations to the previous model. The tobacco forecasting model is a dynamic model built in Microsoft Excel including a base and a forecasting model. In the base model, recent trends in smoking initiation and cessation are established, which are then used as inputs for future BAU smoking prevalence projections in the forecasting model. Changes in smoking initiation in this model are reflected as the annual percentage change in smoking prevalence in 20 year olds, whereas cessation is the annual net cessation rate which reflects the net effect of current smokers quitting and former smokers relapsing in a given year.

The previous dynamic forecasting model[9]was built to project future smoking prevalence from the baseline year 2011. For the current analysis, wherein we provide new BAU smoking prevalence projections to 2025 and beyond using smoking prevalence data from the 2013 Census, the same baseline year of 2011 was maintained for consistency with other tobacco modelling work.[14,15]

Thus, to update smoking prevalence projections from 2011 to 2025 and beyond, recent annual trends in initiation and cessation in New Zealand were estimated in the base model by using the following inputs: a) current, former, and never smoking prevalence data from the 2006 and 2013 censuses; (b) number of births by sex from 1981 to 2012; (c) annual 'year of age'-specificprobabilities of dying from life-tables; and (d) estimates of relative risks of mortality for both current and former smokers from the New Zealand Census Mortality Study.

However, tobacco tax has increased each year by at least 10% since mid-2010 in New Zealand (but not from 2006 to 2009). It is very likely that these tobacco tax increases have played a role in the reduction in smoking rates that has been observed in the general adult population (e.g., from 20.7% to 15.1%) and in Māori (e.g., from 42.2% to 32.7%) between the 2006 and 2013 censuses.[11] However, in a BAU scenario we cannot assume that tax rises will continue indefinitely.

We thereforeestimatedannual changes in initiation rates and cessation rates between 2006 and 2013 *as though there had been no tax rises*, by simply adjusting the 2013 Census data to what we estimate it would have been in the absence of tax rises from 2010 using price elasticities for smoking prevalence of 0.38 (for ≤20 year olds), 0.29 (for 21-24 year olds), 0.19 (for 25-34 year olds), and 0.10 (for 35+ year olds) from New Zealand tobacco tax modelling work.[14] The observed 2006 and 'tax rises removed' 2013 Census prevalence data was then used to estimate changes in initiation and net cessation during this period as previously explained.

The outputs from the base model (annual trends in initiation and cessation between 2006 and 2013 adjusted for no tax rises since 2010) were then used as inputs for the future BAU smoking prevalence projections from 2011 to 2060 in the forecasting model. As a starting smoking prevalence for the baseline year 2011, we calculated what the smoking prevalence in 2011 would have been by using the actual smoking prevalence data from 2013 and removing the effect of tax rises that occurred in 2012 and 2013 (using the same age-specific price elasticities as mentioned above).

For our BAU smoking prevalence projections by sex and ethnicity from 2011 to 2025 and beyond, we then applied the above initiation and cessation rates in the dynamic model from 2006 to 2013 with the slight modification of adding in the estimated effects of annual 10% tax increases in 2012, 2013, and 2014 that have

actually occurred. From 2014 onwards, we assumed no further tax increases, and trends in annual initiation and cessation rates to fall back to the 2006-2013 pattern had there been no tax increases since 2010.

Using population-wide census data to estimate annual initiation and cessation rates means that the uncertainty interval around these estimates is narrow (i.e., the census is an extremely large data base essentially meaning little random error). However, in reality, there is likely to be more uncertainty around the annual initiation and cessation parameters (for example caused by changing cultural norms around smoking ['denormalisation']).

At present, we are unable to quantify this structural uncertainty accurately. Therefore, to give a sense of this potential source of uncertainty in the initiation and cessation rates, additional modelling by sex and ethnicity was done. In the "optimistic scenario", the annual percentage reduction in smoking prevalence in 20 year olds, and net annual cessation rates were multiplied by 1.5 (an arbitrary amount but one which is likely to capture a plausible amount of the uncertainty). In the "pessimistic scenario", these rates were divided by 1.5. For example, if the BAU annual net cessation rate was 4.0%, this would increase to 6.0% in the optimistic scenario, whereas it would reduce to 2.7% in the pessimistic scenario.

Results

Table 1 shows the initiation and cessation results for 2002 to 2011 (from the previous model[9]) and for 2006 to 2013 (from the updated model with 2013 Census smoking prevalence adjusted for no tax increases since 2010). For 2006 to 2013, it indicates that daily smoking prevalence at age 20 years decreased annually by 3.4% (95% uncertainty interval [UI]: 3.2% to 3.6%) and 2.7% (95% UI: 2.5% to 2.8%) for non-Māori men and women, and by 2.9% (95% UI: 2.6% to 3.2%) and 3.2% (95% UI: 2.9% to 3.5%) for Māori respectively. Annual net cessation rates ranged from 3.7% to 7.7% and 4.5% to 6.9% in the different age groups (20–34, 35–54, 55+ years) in Māori men and women, and from 3.8% to 7.2% and 4.3% to 7.1% for non-Māori respectively.

Compared to the initiation and cessation results for the 2002 to 2011 period from the previous model,[9] annual net cessation rates were found to be higher from 2006 to 2013 for Māori for all age groups, whereas annual net cessation rates were higher in non-Māori over 55 years of age in 2006 to 2013, but varying from lower to higher for the other age groups. Of note is that the uncertainty intervals were considerably less for the 2006 to 2013 estimates, due to very small standard errors in census data prevalence estimates (compared to prevalence estimates from the NZHS data series).

Also shown in Table 1 are the low and high values of these parameters (divided or multiplied by 1.5) for the pessimistic and optimistic projections.

Figures 1 and 2 show the new projections of future smoking prevalence by sex and ethnicity using the (adjusted) annual initiation and net cessation rates from 2006 to 2013. In these the slope of the future BAU smoking prevalence projections by sex and ethnicity is steep from 2011 to 2014 as these are the years where we allowed for the effect of the 10% tax rises that actually occurred.

Under a BAU scenario, the projected smoking prevalences in 2025 are 8.3% and 6.4% for non-Māori men and women, and 18.7% and 19.3% for Māori respectively. Under this BAU forecasting scenario, a below 5% smoking prevalence will be achieved by non-Māori women in the year 2032 and men in 2040, and sometime after 2060 for Māori men and women.

Only the optimistic scenario for non-Māori women is projected to reach below 5% smoking prevalence in 2025.However, this would require a substantial 50% increase in the annual percentage decrease smoking prevalence amongst 20 year olds from 2.7% to 4.1 %, and in annual net cessation rates across the age groups (e.g., from 5.5% to 8.3% in 20–34 year olds).

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Table 1. Outputs from the baseline tobacco model for various demographic groups in New Zealand over time—net annual cessation rates and annual percentage changes in 20–24 year old smoking prevalence (reflecting smoking initiation levels) for non-Māori and Māori between 2002 and 2011 (original model) and 2006 and 2013 (updated model*)

	2002 to 2011 (used in previous projections)[9] Best estimates		2006 Census to 2013 Census Best estimates Scenarios for projections					
	Men	Women	Men	Women	Men		Women	
					Pessimistic scenario	Optimistic scenario	Pessimistic scenario	Optimistic scenario
Non-Māori								
Net cessation rates†								
20–34 years	3.2 (1.8 to 4.6)	4.4 (3.0 to 5.8)	4.1 (4.0 to 4.3)	5.5 (5.3 to 5.8)	2.8	6.2	3.7	8.3
35–54 years	3.8 (2.8 to 4.9)	5.9 (5.0 to 6.9)	3.8 (3.7 to 4.0)	4.3 (4.1 to 4.5)	2.6	5.8	2.9	6.5
55+ years	4.5 (3.2 to 6.0)	6.1 (4.7 to 7.2)	7.2 (7.0 to 7.5)	7.1 (6.9 to 7.4)	4.8	10.8	4.8	10.7
Smoking prevalence in 20-year olds‡	3.1 (0.8 to 5.7)	1.1 (-1.2 to 3.2)	3.4 (3.2 to 3.6)	2.7 (2.5 to 2.8)	2.3	5.1	1.8	4.1
Māori								
Net cessation rates†								
20–34 years	0.6 (-0.9 to 2.0)	1.4 (0.3 to 2.5)	3.9 (3.7 to 4.2)	4.5 (4.2 to 4.8)	2.6	5.9	3.0	6.8
35–54 years	2.8 (1.4 to 4.2)	3.5 (2.6 to 4.6)	3.7 (3.5 to 3.9)	4.7 (4.5 to 5.0)	2.5	5.5	3.1	7.1
55+ years	0.4 (-1.7 to 2.4)	5.8 (4.2 to 7.3)	7.7 (7.1 to 8.3)	6.9 (6.4 to 7.6)	5.1	11.5	4.7	10.5
Smoking prevalence in 20-year olds‡	4.7 (2.2 to 7.1)	0.0 (-2.2 to 1.8)	2.9 (2.6 to 3.2)	3.2 (2.9 to 3.5)	1.9	4.3	2.1	4.8

*The 2013 smoking prevalence data was first adjusted to what we estimate it would have been in the absence of tax rises from 2010. Changes in initiation rates and cessation rates between 2006 and 2013 were calculated using the adjusted 2013 Census data. †Although the pattern in annual cessation rates by age appears to be different from 2002 to 2011 (steadily increasing by age) than 2006 to 2013 (U-shaped pattern; higher cessation rates for 20-34 year olds and 55+ year olds, and somewhat lower cessation rates for 35-54 year olds), uncertainty intervals around the estimates for 2002-2011 are wide and overlapping for the different age groups. Some other countries with relatively advanced levels of tobacco control (for example United States and Canada) have recently reported higher abstinence rates in the younger and older age groups (albeit no statistically difference was found between the age groups).[16,17] ‡Percentage decline, not absolute percentage change. Thus if 20 year old smoking prevalence is 20% at t_0 and the 'percentage decline in initiation rate' per annum is 3%, then the 20 year old smoking prevalence in year t_1 is 20% × (100%-3%) = 19.4%. Furthermore, the smoking prevalence in year t_{10} is 20% × $97\%^{10}$ = 14.7%.

Note: 95% uncertainty intervals in parentheses.

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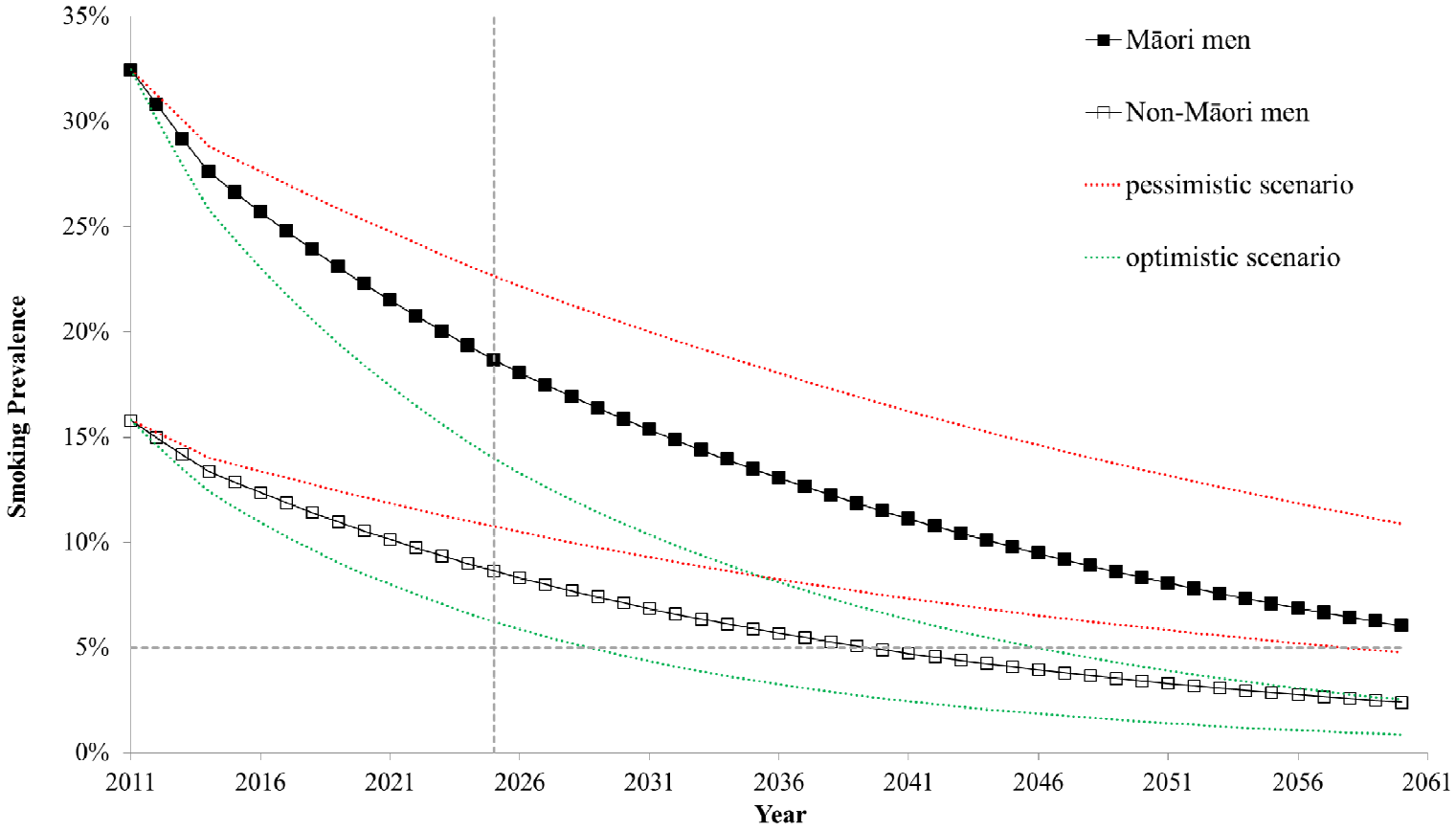
Figure 1. Business-as-usual, optimistic scenario, and pessimistic scenario* projections of daily smoking prevalence for Māori and non-Māori men in New Zealand using change in initiation and net cessation parameters from 2006 to 2013 (adjusted for no tax rises since 2010)



*In the optimistic scenario, annual percentage reductions in smoking prevalence in 20 year olds and annual net cessation rates were multiplied by 1.5, whereas these rates were divided by 1.5 in the pessimistic scenario. For example, if the BAU annual net cessation rate was 4.0%, this would increase to 6.0% in the optimistic scenario, whereas in the pessimisticscenario it would reduce to 2.7%.

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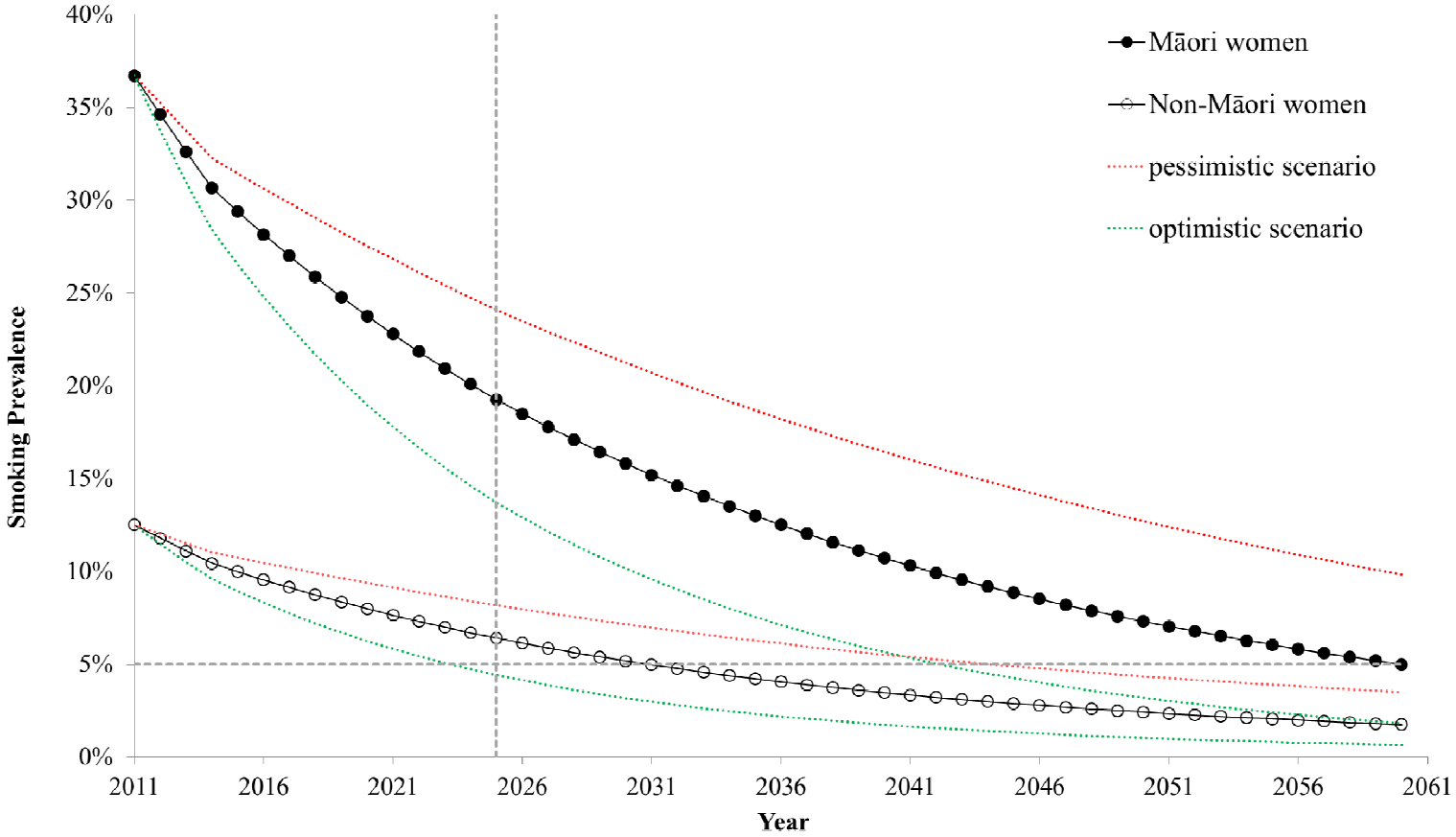
Figure 2 Business-as-usual, optimistic scenario, and pessimistic scenario projections of daily smoking prevalence for Māori and non-Māori women in New Zealand using change in initiation and net cessation parameters from 2006 to 2013 (adjusted for no tax rises since 2010)



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Discussion

Projecting future smoking prevalence, using the most recent trends in initiation and cessation from full population census data on smoking and allowing fortax increasesuntil 2014, resulted in an estimated prevalence in 2025 of 19% for both Māori men and women, and 8% and 6% for non-Māori respectively.

Although these projections are more favourable from a public health perspective (especially for Māori) than our previous modelling results using NZHS smoking data,[9]a smoking prevalence below 5% by 2025 is still not attained by any demographic group. Furthermore, although the projected decline in smoking for Māori will be a welcomed development in the New Zealand health sector, large inequalities in smoking prevalence between non-Māori and Māori are projected to remain(under this BAU scenario).

Attaining the 2025 goal for all New Zealanders may therefore require substantially larger increases in annual net cessation rates, and decreases in initiation than our optimistic scenario, especially for Māori.

Given the uncertainty around future tobacco control policy, we cannot assume that tax rises will continue indefinitely (in a BAU scenario). We therefore used price elasticities of demand for tobacco (varying by age) to estimate annual changes in initiation and cessation rates between 2006 and 2013 as though there had been no tax increases since 2010. These adjusted annual initiation and cessation rates were then applied in the model from 2014 onwards. That is, our BAU scenario projections are for a future world with no further tax increases, other than the routine annual inflation-adjustments (projections with tax increases are provided elsewhere[14]).

Although price elasticities may also vary by other demographics than age, such as sex, ethnicity, and level of deprivation,[18,19] the adjusted 2013 prevalence only marginally changed after using price elasticities to remove the effect of tax rises that occurred since 2010 (e.g., from 30.5% to 30.9% for Māori men, from 34.7% to 35.1% for Māori women, from 15.5% to 15.7% for non-Māori men, and from 12.5% to 12.7% for non-Māori women). This means that, compared to the large reduction in the observed smoking prevalence between the 2006 and 2013 censuses, annual initiation and cessation rates in the model are not that sensitive to how much tax effect is removed (i.e., variations in price elasticities).

Using smoking prevalence data from the 2006 and 2013 censuses potentially has some limitations. First of all, the census captures the prevalence of daily smoking for those aged 15 years and over, whereas the NZHS also reports the prevalence of current smoking which includes both daily and non-daily smoking behaviour across the population.[20] According to the results of the 2012/2013 NZHS, the prevalence of non-daily smoking is approximately 2% in New Zealand.[20]

The census might therefore underestimate the prevalence of any smoking behaviour across the population. Furthermore, about 6.7% of the New Zealand population did not answer the smoking question in the 2013 census.[10]However, for the census prevalence to be notably biased would require the 6.7% of people not responding to the smoking question to have very different smoking behaviour to the other respondents.

The smoking prevalence results from the 2013 census seem credible when looking at the daily smoking rate reported in the 2012/2013 NZHS for the totaladult population (15.5%).[20]Smoking prevalence data from the 2006 and 2013 censuses proved to be more stable, especially for Māori, than using data from multiple NZHS series.

Projecting (and updating) future BAU smoking prevalence may be useful for assisting policy makers in New Zealand in planning how much more intense tobacco control policy has to be (or if new major strategies are required such as a tobacco outlet phase down[15]) to achieve the endgame goal. It proved to be relatively easy to update the previously published forecasting model based on new prevalence data from the 2013 census, and by demographic group to estimate how inequalities may track into the

future under a BAU scenario. It also forms a possible foundation for future modelling of the impacts of a variety of novel tobacco control interventions (e.g., phasing down the level of nicotine in smoked tobacco) or changes in the future nicotine market (e.g., through increased availability of electronic cigarettes).

E-cigarettes appear to be increasingly popular in New Zealand owing to legal importation for personal use and potentially some level of illegal sales. It is possible that in the future there may be electronic cigarette brands that meet regulatory standards that allow them to be legally sold in New Zealand or that current New Zealand law is revised to permit sales of brands that meet minimal standards. If so, such changes in the nicotine market may impact on future trends in tobacco cigarette initiation and cessation rates.

Some recent signs of progress in tobacco policy planning are that the New Zealand Government has announced that the pattern of annual tax increases of 10% above annual inflation will be extended to 2016,[21] and the Tobacco Plain Packaging Bill (as per Australia) has passed its first reading in Parliament with a majority of votes.[22]Nevertheless, attaining the smokefree goal, and reducing large inequalities in smoking prevalence between Māori and non-Māori, will probably require implementation of more intense or entirely novel tobacco endgame strategies such as a more permanent approach to tobacco tax increases, a gradual phase-down in nicotine levels of tobacco products until a non-addictive level is reached, or major reductions in the number of current tobacco retail outlets (possibly limiting these to one outlet type only, for example pharmacies[23]).

Competing interests: Nil.

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VIEWPOINT

Is New Zealand water fluoridation justified?

Yindi Jiang, Lyndie A Foster Page, John McMillan, Karl Lyons, Jonathan Broadbent, Kate C Morgaine

Abstract

Public health programmes extend beyond the clinical context and focus on measures that affect the lives of large subgroups or the population as a whole. An example of this is community water fluoridation (CWF), the altering of fluoride levels in the water supply with the aim of preventing the initiation and slowing the progression of dental caries lesions for the benefit of entire populations. Despite the unfeasibility of randomised controlled trials of CWF, a large volume of evidence is available on the topic. However, CWF remains a polarising and keenly contested issue. CWF is also an intervention where it is difficult to provide everyone affected with a choice. The Nuffield Council on Bioethics is an independent body that examines and reports on ethical questions, and they have provided a useful ethical framework for considering CWF via the 'stewardship' model. This commentary aims to discuss each of the public health aims and how they can be applied and weighed to reach a justified position about CWF.

The ethical principles that are relevant to public health are distinct from those that have been developed for other issues within health care. This commentary will explain the ethical principles that are relevant to public health and how they can be balanced and applied to public health dentistry. Public health programmes often affect the lives of large subgroups or the population as a whole and extend beyond the clinical context.[1] Because of the population focus of public health and its emphasis upon prevention, it can often have implications for those who would not consider themselves to be ill or do not agree with a particular public health measure.

Fluorides are naturally-occurring compounds found in soil, air and water.[2] It is present naturally in water with varying concentrations from less than 0.5 parts per million (ppm) to 25ppm.[1]Fluoride is effective in preventing the initiation and slowing the progression of dental carious lesions and it works best via frequent applications at low concentration with community water fluoridation (CWF) being its optimal system of delivery.[2] This benefit was discovered when researchers in the USA compared areas with different levels of naturally-occurring fluoride in water and found that at approximately 1 ppm, the caries preventive benefit was maximised while the prevalence and severity of dental fluorosis (a type of developmental defect of enamel which appears as diffuse white patches or streaks on the teeth) was low.[2] The current New Zealand Ministry of Health guideline for CWF is between 0.7 and 1.0ppm.[3]

Adjusting fluoride levels in the water supply is keenly contested by some. Although CWF has been implemented in some areas for several decades and there is a large volume of evidence available on the topic, ethical and technical issues mean high level research such as randomised controlled trials are virtually impossible to perform.[4] CWF directly affects whole populations and it is not straightforward to provide each affected individual with a choice. Because of this, CWF raises ethical and policy issues.

Individuals or groups may object to and question the responsibilities and authority of the state and other agents to affect people's lives in such ways. For example, a libertarian might argue that consent is required for every public health measure.[5] While that perspective might be consistent and theoretically appealing to some, it would have the consequence that universal public health measures which could have a profound impact upon health and carry no risk of harm, would become impermissible without universal consent.

Strictly speaking, a libertarian view would also require individual consent in order to remove excess fluoride in regions where very high levels of fluoride occurs naturally in water. At the other end of the spectrum, a utilitarian approach might focus upon achieving the greatest possible collective benefit.

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This means that the interests of some people may be 'sacrificed' if this were to lead to an increase in overall welfare.[5]

Between the libertarian and utilitarian there are highly divergent ways of viewing public health. To adjudicate and balance those divergent views, a principlist approach can be used, and the principles developed by the Nuffield Council on Bioethics provide a useful framework for considering CWF.

The Nuffield Council on Bioethics is an independent body that examines and reports on ethical questions. In 2007, the Nuffield Council published a report which aimed to address the key ethical considerations that are relevant to public health, including CWF, and developed a 'stewardship' model of public health.[1] The "stewardship" model includes six prima facie public health aims of the state or district council that can be applied and weighed to reach a justified position about CWF.[5-7]

The stewardship model states that public health measures should aim to:

- Reduce the of risk of ill health;
- Address the health of children;
- Reduce health inequalities;
- Not intervene without the consent of those affected;
- Minimise interventions that affect important areas of personal life; and
- Not coerce ordinary adults to lead healthy lives.

Reducing the risk of ill health

Oral health is fundamental to general health and well-being.[8] Poor oral health has significant effects on the quality of life as a result of pain, discomfort and impaired oral functioning.[8]Teeth also make a substantial contribution to physical appearance,and oral health problems can have negative impact on earningsand employment opportunities.[9]

Just as water is treated in several ways to improve safety, fluoride levels can be adjusted to promote and improve health for the population. The level to which CWF reduces ill-health is keenly debated. The Nuffield Council's view is that the fluoridation of water does reduce the prevalence of caries, but the degree to which it is reduced is not clear from the evidence.[1]Others have argued that the role of fluoridated water in preventing caries has decreased significantly following the introduction and widespread use of other fluoridated products such as fluoridated toothpaste.[10]

It should be noted however, that a halo effect may be functioning,[11]in that the benefits from a fluoridated public water supply might be weakened because beverages and food products processed in fluoridated communities are exported to surrounding non-fluoridated communities.Studies measuring the effectiveness of CWF that consider only its direct benefit may therefore have underestimated the total contribution of CWF to caries reduction.[12]

Despite this, existing evidence indicates the CWF remains one of the most effective and socially equitable means of achieving community wide exposure to the caries prevention effects of fluoride.[12]The York Review acknowledged that the beneficial effects of CWF were still evident in spite of the assumed exposure to non-water fluoride in the populations studied.[4]They also found that after adjustment for potential confounding variables, introducing CWF into an area significantly increased the proportion of caries-free children, and decreased mean decayed, missing and filled primary/permanent teeth (dmft/DMFT) compared to areas that were non-fluoridated over the same time period.[4]Studies have found the reduction in caries to range from 15% to as high as 50%,[13,14]whilst others have demonstrated that the prevalence of caries increased when fluoride was removed from the water.[15]

To date, the only established adverse effect of CWF is a dose-response relationship between fluoride levels and the risk of dental fluorosis.[12]Dental fluorosis is one of several different types of defects of the tooth enamel that causes visible markings on the teeth. It can vary from mild speckling which is imperceptible to the naked eye through to more severe staining and mottling of teeth.[4] At concentrations below 1 ppm, which has commonly been used for CWF, most incidences of fluorosis are mild and only 3-12.5% of fluorosis are considered to be of aesthetic concern.[4,16,17]

Other than fluorosis, there is no evidence of an increase in risk of other harms, such as bone fractures, cancer, or any other adverse effects.[12] In fact, some studies have identified lower rates of hip fractures among those exposed to optimally fluoridated water.[18] It is important to note that it may be difficult to determine whether particular harms are caused by CWF because of the presence of confounding factors, difficulties in estimating people's total fluoride exposure and whether there is a long lag between exposure and occurrence of harm.[1]

Despite this, a large reduction in caries incidence for a small increase in the incidence of visible fluorosis may be considered a worthwhile trade off.Furthermore, it has been reported that children with mild fluorosis tend to rate their teeth as healthier and more attractive than those that do not have dental fluorosis.[16]

Reducing health inequalities

Despite significant gains in oral health in recent decades across the developed world, social inequalities in oral health remain.[19] The bulk of the scientific literature indicates that the oral health of lower socioeconomic status (SES) groups does not match that of their higher SES counterparts.[19]

Disadvantaged people often have unhealthy habits or knowledge about oral health and are less likely to visit a dentist if available.[8] As an extreme, "poor oral health could threaten job security and economic productivity that in turn may exacerbate adverse social, psychological and economic circumstances, resulting in a downward spiral that further damages health."[8]

It is clear from the New Zealand Oral Health survey that significant inequalities in oral health still exist within New Zealand.[20] The reduction of health inequalities should be a central goal of any public health programme and, according to Petersen and Kwan, CWF is one of the most cost-effective public health measures to improve oral health and reduce inequalities.[8,21,22] While it provides some benefit to all social groupings, the effects are greater among the most deprived populations; this is particularly the case where access to oral health services is limited.[8,23] The Nuffield Council states "prioritarian programmes that address inequalities can, in principle, be ethically justified".[1] Therefore the potential for CWF to reduce inequalities is an important argument in favour of the intervention.

One objection to the oral health inequalities argument for CWF is that there are other methods of preventing dental caries. Instead of adjusting fluoride levels in the water we should be attending to the social determinants of poor oral health and in addition improve dental hygiene to reduce the incidence of caries. However, changing social determinants would require a broad range of government initiatives and time for such initiatives to make a difference. Evaluations of oral health education campaigns in Scotland, found significant improvements in oral health in children from schools located in non-deprived areas compared to higher deprivation areas.[24] This suggests that oral health education in some cases, may increase inequalities in oral health.

Systematic reviews of the health education literature have found that dental health education interventions have no discernible effect on rates of tooth decay.[25,26] Educational approaches aimed at reducing health inequalities in disadvantaged social and economic groups are unlikely to be effective unless they are sensitive to the environment in which such people live and are backed by wider policies to create a supportive community.[13]

In contrast, CWF is an intervention that can be provided directly to everyone, meaning it is accessible to all. The ability of CWF to reduce health inequalities may also be a function of its passive mode of delivery. CWF has the advantage over toothpaste and mouth rinse of ensuring complete uptake of the

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measure at no added cost to the individual.[1] Changes to lifestyle or behaviour is also not required, which can be difficult to achieve.[27]

Special attention to the health of children

The public health aim to provide an adequate level of care for all children is also a good argument in favour of CWF. Children are born into a defined social stratum and are consequently exposed to the oral health benefits or vulnerabilities that come with it.[28] Children represent a particular vulnerable group in many public health contexts and this is also true for oral health. They are less able to make informed choices about their oral health, and are dependent on parents and caregivers to assist with, or promote preventive measures such as tooth brushing.[1]

While other methods of delivering fluoride such as the fluoridation of salt, milk, and toothpaste have the advantage in that it is easier for adults to opt out of being exposed to fluoride, they have the disadvantage of reaching fewer children. Research has shown that given a choice, those from higher SES groups that are more likely to choose fluoridated salt and supplements.[27] This indicates that these measures do not necessarily reach the whole population and may miss those who are most likely to develop caries, especially children from lower SES groups.[27] The social origins hypothesis proposes that early childhood disadvantages, exposures, and oral health status ultimately determines oral health in adulthood.[29]

Life course analysis has shown how social advantage and disadvantage accumulates or clusters at critical periods, particularly in early life, thus contributing to the creation of health inequalities. Results from the Dunedin Multidisciplinary Study provided strong evidence for this theory.[29,30]

Not coercing ordinary adults to lead healthy lives:

Not being overweight or obese, not drinking more units of alcohol per week than is recommended and having 30 minutes of exercise a day are things that are good for heath. While there is no doubt that the majority of people would agree that these public health aims would be good for them, there are people who choose to not live healthy lives and we should not coerce or pressure them into living a healthier way. This is because some people are content with a lifestyle that might contribute to a shorter life but is one where they can eat what they wish and or drink more than is recommended if they choose.

It could be argued that these public health aims are so important that they outweigh worries about people being coerced into living healthy lives, but if we are serious about respecting the right of all people to decide how they will live, coercive public health measures are ethically dubious. Even though drinking to excess, over-eating and smoking are unhealthy; many people prefer to do these things even though they are unhealthy. However, CWF is different in that it does not involve forgoing anything; drinking tap water is something that people would do in any case. Unlike many other public health interventions, CWF does not require any change in a person's lifestyle, so it does not coerce people into leading healthy lives.

Not intervening without the consent of those affected and minimising interventions that affect important areas of personal life

When fluoride concentration is adjusted in the water supply, there is no doubt that for some members of the community the public health aim of not intervening without the consent of those affected will not be achieved.

Consent is important for medical interventions, and this could be used to argue that public health interventions should not be introduced where some individuals, however few, are opposed to it. However, it could be argued that the removal of fluoride from the drinking supply should also require universal consent. Both of these implications would be problematic because they would give a great deal of weight to the importance of choice and consent and allow a few people to override the collective good that might be achieved through such public health interventions.

Another argument that might be made against CWF is that it restricts the choices of individuals in a significant way, as individuals are less able to exercise choice over the water they consume. Likewise, some would claim that CWF is an intervention that impacts upon important areas of personal life.

It is hard to dispute that people who wish to avoid fluoridated water have to go to some lengths to do so, however, it is less clear CWF coerces people into drinking fluoridated water. Someone who does not want to drink water that has had the fluoride concentration adjusted could drink tap water that has been filtered so as to remove the fluoride, collect and drink rain water, access bore water or purchase bottled water. However it does have to be conceded that the degree of inconvenience involved in sourcing non-fluoridated water may be significant for some, and this frustration to a person's interests is a cogent argument against CWF. This frustration, however, would be more than counterbalanced by the potential inconvenience of accessing fluoridated water should there be no CWF.

Conclusion

Ultimately, how the public health aims, identified by the Nuffield Council, should be applied and weighed up will be contested. There are some who will argue that the evidence that CWF reduces ill health and the lack of evidence of there being significant risks is not compelling enough to outweigh concerns about consent. There will be others who contest the claim that CWF is especially beneficial for children or that it is the best option for reducing oral health inequalities.

Debate about those issues is healthy and should be welcomed. Given how controversial CWF is for some, it is appropriate that we are vigilant about the evidence base for CWF. However, given the oral health of many children, that poor oral health is disproportionately high among lower social economic groups, and that CWF appears to be an effective way of addressing these public health aims, it seems reasonable for city and district councils to adjust the level of fluoride in the water supply, given our current state of knowledge about CWF.

Not being coerced into living a health life is an important consideration that needs to be factored in when considering public health initiatives. We showed that this is not an issue for CWF because it does not require any change in lifestyle. The absence of individual consent to being provided with fluoridated water might be taken to imply that one public health objective of the Council is not met, although as we showed, consent is also applicable to decisions to cease CWF.

Given that there are strong and divergent views about CWF this can be mitigated by finding other ways in which a state, or local authority, can demonstrate "deliberative democracy." The Nuffield Council's suggestion of a "transparent decision making processes", with involvement of stakeholders in the decision making process, and the opportunity to challenge such interventions, can counter balance concerns about CWF and also provide an opportunity for decision makers to be transparent about the considerations they are acting on. The Nuffield Council concluded that the most appropriate way of deciding whether fluoride should be added to a water supply is to rely on democratic decision-making procedures. This is the status quo in New Zealand at present where elected local authorities are charged with making decisions about public health in their region.

Doubts have been expressed about whether district and city councils are best placed to make evidenced-based decisions about CWF, despite them being well placed to engage in deliberative democracy. However, ourview is that regional, evidence-based decision making, be it via councils or DHBs, is an appropriate way for decisions to be made about CWF.

Competing interests: Dr McMillan reports personal fees from South Taranaki District Council, during the conduct of the study.

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VIEWPOINT

Profits or people? The informative case of alcohol marketing

Sally Casswell

Abstract

Aim To analyse influence on alcohol marketing policy in New Zealand.

Method Document and literature review.

Results There is a powerful argument and popular support for restricting alcohol marketing but no significant policy action taken.

Conclusion Greater priority has been placed on the profits of influential corporations compared with protecting the health of future generations of New Zealanders.

The exposure of young New Zealanders to commercial messages to promote drinking has been a controversial issue since the government first opened up the broadcast media to brand advertising in 1992. There have been numerous (unsuccessful) private members' bills proposed[1,2] and numerous enquiries held under the auspices of the vested interests[3] involved; these have recommended minor changes to the self-regulation system.

More recently marketing was under consideration in the Law Commission's review of alcohol legislation. Their first report in 2009[4] did not propose major changes in alcohol marketing policy. In our meeting with the Commission it was apparent the processes developed by the ASA (Advertising Standards Authority - the industry body), with a focus on the content of advertising, had impressed the chair, Sir Geoffrey Palmer. However, by the time of the final report[5] the Commission recommended a three stage reform which, if carried through, would result in some effective restrictions on exposure to alcohol marketing. What had happened?

Some of the change probably reflected increased understanding of the research evidence; the Commission were provided with a proof copy of the about to be published review of evidence on alcohol policy, Alcohol: No Ordinary Commodity.[6] The alcohol industry had made much of New Zealand's focus on evidence-based policy to argue against change.[7] However, the evidence reviewed in Alcohol: No Ordinary Commodity,[6] established both that exposure to alcohol advertising recruits young people to drink earlier and to drink larger quantities and that voluntary codes such as that operated by the ASA are ineffective.

A more likely influence on the Law Commission's change of heart was the level of popular support for change. An unprecedented number of submissions were made to the Law Commission (almost 3000) and almost all of them commented on alcohol marketing and 86% of these supported banning or restricting alcohol advertising of all alcohol in all media.[7]The same pattern emerged in the submissions made to the Select Committee which subsequently considered the Sale and Supply of Alcohol Act.

Yet, all that occurred was an extension to pre-existing regulation, focussed on the retail sector, which prohibits promotions promoting excessive drinking, having special appeal to minors, offering free alcohol or discounts of 25% or more or offering free goods or services with the purchase of alcohol. The promotion which is so powerful in building brand relationships and normalising drinking was left untouched.

Yet another committee, the Advertising Forum, was established by government in lieu of further policy change (and has recently heard submissions[8] to investigate the issue). So why was the response so feeble?

Some clues for this can be found in the influence of the several powerful vested interests concerned with marketing. The CEO of the ASA was actually given a seat at the table of the most recent committee, the Advertising Forum[8] despite the obvious conflict of interest.

Stepping back and looking at the harm alcohol does in our society, the evidence on marketing's effect on young people's consumption and the popular support evinced for change, the only reasons to maintain alcohol marketing in its current largely unrestricted state are to: first, protect the profits of the transnational corporate producers by allowing them to appeal to new cohorts of young people with marketingwhich recruits them as consumers as early as possible and encourages drinking of larger amounts and second, to protect the financial interests of the advertising and media industries.

It is hard to avoid drawing a conclusion that government's failure to act was based on a decision to protect the interests of these large corporations at the expense of protecting the health and wellbeing of future generations of New Zealanders.

A restriction on alcohol marketing similar to that adopted more than 20 years ago in relation to tobacco (Smoke-free Environments Act, 1990) or specific to alcohol similar to that in France (LoiEvin, 1991) will not impact in any meaningful way on adult consumers' knowledge of the availability of alcohol.

Significant restrictions on alcohol marketing will, however, likely effect the normalisation of alcohol. Normalisation, the acceptance of ubiquitous and perception of unproblematic use, makes it more difficult for health promotion and social marketing to affect consumption among heavy drinking social networks or for family and whanau to place limitations on access to alcohol by vulnerable young people.

A strong move to restrict alcohol marketing will send a message that the New Zealand community acknowledges alcohol, like tobacco, is no ordinary commodity. In turn this may increase the acceptability of other effective policies, such as price increases. This is a major reason why the alcohol industry is so focused on preventing any form of regulation of alcohol marketing.

Need for comprehensive restrictions

Alcohol marketing is broader than alcohol advertising and includes sponsorship (branding of events and teams/people), branded merchandise, price promotions, competitions and more. All aspects of marketing need to be subject to restriction. It is necessary for regulation to focus on preventing all exposure to alcohol marketing rather than attempting to affect the content of advertising. The extensive peer-reviewed research on tobacco[9] and food[10]as well as alcohol promotion shows it is advertising per se that encourages young people into the market, not particular channels of advertising or types of message.

In order to protect young people from alcohol advertising we have to reduce their exposure to any form of promotion; not just adjust the message and channel mix.

Sponsorship, funding to allow for branding of positive events and experiences, is a major part of marketing in New Zealand and deserves urgent attention. The purpose of sponsorship is two-fold: first, to provide opportunities to promote alcohol products in contexts in which people are having positive and exciting experiences, and second, to promote the brand/corporation as a good corporate citizen, to legitimise their position as part of the 'solution' and thereby help ward off effective policy change. Neither of these are positive reasons from a public health perspective.

Recent research from New Zealand[11] and Australia has found heavier drinking among sportspeople in teams sponsored by the alcohol industry and "receipt of alcohol industry sponsorship [was] associated with alcohol-related aggression/antisocial behaviours in university sportspeople" (p. 241).[12]

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Alcohol industry sponsorship can easily be banned in all sporting, cultural, and community activities. Funding can be replaced by public funds, either from the consolidated fund, as was done for tobacco sponsorship, or by increasing the very small Health Promotion Association (HPA) levy on alcohol sales to allow for buy out. The HPA has the necessary experience and expertise to carry out such a process.

Social media

New developments in alcohol marketing increase the urgency for restrictions on alcohol marketing. Use of the social media has expanded exponentially since 2012 and provided extensive opportunities for the dissemination of alcohol marketing materials.

Social media case studies of a selection of alcohol brands by RAND Europe in 2012 showed that these all had considerable online media presence featuring both marketer-generated and user generated content. In the analysis carried out by RAND Europe[13] Facebook, YouTube and Twitter were the three social media sites most used by young people. RAND analysed five alcohol brands, which all maintained a Facebook page, YouTube channel and Twitter account.

Facebook features included profile pages for comments by marketers and users, and additional content such as competitions, videos, recipes or applications such as games, inviting users to engage with marketer content. Similarly, marketer-generated YouTube sites contained a variety of videos related to the product, including adverts, and in one case comedy videos.

Twitter accounts contained tweets by the marketer and others, relating to the product, but also tweets on a variety of other subjects including comedy, fashion and recipes. New Zealand research has shown the use of these SNS (Social Networking Sites) provides young people with the opportunity to create and share ‘intoxigenic social identities’ and digital spaces which further contribute to the normalisation of youth consumption of alcohol.[14]

The response on sites such as Facebook has dramatically increased since 2012 and underage users can access alcohol material[15] while this remains largely under the parental radar. In a new development transnational corporations such as Diageo and Heineken have commercial partnerships with Facebook which they describe as a ‘social university’. Heineken’s global head of digital media explains: "One of the criteria we have for agencies when they come up with a proposal is: 'why would I share this?' We aren't just developing campaigns for the people we send them to – we are developing them for the friends of those friends."[16]

In a letter to the FTC in 2011, the attorneys general (the leading law enforcement officers) of 24 US states and territories warned of ‘a “brave new world” of marketing that will expose millions of American youth to alcohol advertising messages on their cell phones and computers while at the same time taxing regulators’ capacity and understanding’. They urged the FTC not to rely solely on industry assurances of responsibility, but rather to gather the facts necessary for an independent assessment of what regulatory oversight is appropriate (cited in Jernigan & Rushman, 2014[15]).

Drinking by young people

While the young are by no means the only New Zealanders exposed to health hazards due to drinking alcohol the confluence of their propensity to drink to intoxication, risk taking behaviour, increased risk of neurophysiological impacts and risk of developing dependence over time all suggest reasons for concern.

In New Zealand we have seen a marked increase in drinking by young people since the first introduction of brand advertising in the broadcast media in 1992. The reduction in the minimum purchase age in 2000 further stimulated increased consumption, particularly in the 16–17 year-old age group.[17]

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We still have large amounts of alcohol being consumed by the young. In 2013 the Alcohol Policy in New Zealand (APINZ) survey carried out as part of the International Alcohol Control (IAC) study found among the groups aged 16–17 and 18–19 years more than one in three reported drinking at least eight drinks on a typical drinking occasion (unpublished data).

Such high levels of heavy quantity drinking (often described as binge drinking) are predictive of later drinking problems and injury[18]and a range of other adverse consequences.[19]Furthermore the earlier onset and larger amounts consumed contribute to lifetime consumption which is a causal factor in cancer along with other alcohol diseases.[20]

Lower socioeconomic populations are most at risk from heavy occasion drinking (and this, of course is relevant for Maori and Pasifika to the extent they remain disproportionately represented among heavier consumers). Alcohol therefore contributes to the health inequalities we experience in New Zealand and restrictions on alcohol marketing will contribute to reducing these inequalities by: first, removing the adverse influence of marketing, and second, allowing a better chance for health promotion and social marketing to have a positive impact

What should be done?

The ideal is a complete ban on alcohol marketing. This is feasible and a useful model is available in the Smoke-free Environments Act 1990. It has the advantage of thorough coverage of all marketing and sponsorship and includes internet marketing.[21]

A less restrictive model is the LoiEvin, the French legislation.[22] The New Zealand Law Commission,[5] following a lengthy evidence review and consultation process, made recommendations which reflect the LoiEvin and suggested a three stage process of implementation. The LoiEvin has withstood legal challenges, as has restrictive legislation in Sweden.

It must be noted the LoiEvin does not address internet marketing, an increasingly crucial issue and recent data suggest some increases in young people's consumption in France.[23]Around the world, governments are trying to figure out how to regulate internet marketing.

Russia has banned all alcohol marketing originating from sites inside Russia. Finland has recently passed legislated restrictions on alcohol advertising which include restrictions on alcohol marketing in social media based on contents produced or shared by consumers themselves or games, raffles or contests.[24]These illustrate first attempts to deal with what is an increasingly important issue.

Whichever approach is taken it is essential this is done in the form of legislation and not left to the ineffective self-regulation by the industries.

The tobacco precedent

The parallel with restrictions on marketing of tobacco is an important one. While at first many people will think it is not a useful parallel because the substances are different and the end game is different (there is no expectation alcohol will not continue to be consumed and enjoyed in New Zealand) the parallel is nevertheless a real one; marketing is a driver of the harm associated with alcohol use just as it is with tobacco use.

Some 20 years ago it seemed a very big step to ban tobacco marketing, including sponsorship. The Smoke-free Environments Act has, however, contributed to the ongoing reduction in tobacco-related harm and the same is possible for alcohol.

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LETTER

Dog bites, treatment and prevention in New Zealand

James A Oxley, June Cheng

There currently appears to be a lack of recent research in New Zealand relating to the occurrence of dog bites, treatment received within the hospital and possible methods of prevention of such incidents.

According to the New Zealand Department of Internal Affairs,[1] the ACC (Accident Compensation Corporation), in the financial year between 2012 and 2013, recorded 12,406 new dog bite claims received, a rise of 13.4% from 2008/9 (10,748).

Langley[2] previously carried out a study recording dog bites in New Zealand between 1979–1988 and found that 182 incidents occurred which needed treatment within the Emergency Department. The findings from the latter research appear to be consistent with more recent research in other countries, which is that male children between the ages of 5 and 9 are the category most frequently bitten.[3–5]

Interestingly, Schalamon et al[6] found that in 75% of dog bite cases the child interfered with a dog (e.g. playing, cuddling, pulling the tail, feeding). However, the two highest rates of bites occurred involved playing with the dog (28%), whereas 26% of bites the circumstances were unknown. Parent and dog owner education is therefore highly important in the prevention of dog bites.

The treatment of dog bite wounds and their management has recently been noted as controversial and the time when treatment occurs and the treatment type (e.g. irrigation) have been noted to be important factors in the prevention of wound infection and resulting aesthetic appearance.[7]

A variety of additional factors have been noted to determine the rate of infection. These include the location of the wound, the type of wound, and whether antibiotics are used.[7-8](Chen et al, 2010; Paschos et al, 2014).

The use of antibiotics is controversial. Consideration of whether to routinely use antibiotics requires, not only a study of the above factors, but estimates of the proportion of dog bites that become infected, and the proportion of these for which antibiotics is an effective treatment. With a thorough analysis, a decision on whether to routinely use antibiotics can be argued on the basis of cost. (See Quinn et al[9]).

The authors note that further research needs to be conducted on dog bite victims, which may help indicate the incident rate and associated treatments within New Zealand. Various information could be collected per dog bite incident including: age, gender, relation to dog, location of bite (e.g. owner's home), situation in which the bite occurred (e.g. playing, approaching dog whilst eating), dog behaviour before the bite, dog breed involved, severity of bite/wound, location of bite (e.g. face, hands), type/method/timing of treatment (surgery, dressings, antibiotics etc.), length of stay in hospital and post care advice and information.

Furthermore, whether the dog is a pet or working dog (e.g. police dog) is likely to be influential on wound type and management. For example, Meade[10]found that individuals bitten by police dogs, in comparison to pet dogs, were seen to be bitten multiple times, have more hospitalisation episodes and were operated on more often. Dog bite records within hospitals should be compared to data from external sources such as the ACC to help identify an accurate rate of dog bites victims having to attend hospitals.

This information would not only provide an insight into the occurrence of bites, and the severity and treatment received within New Zealand hospitals, but would help to identify the need for dog bite prevention methods and identify the frequency and types of bites that result in hospital attendance. It

is important to state that parent and dog owner education is key in the prevention of dog bites and therefore any dog bite research should take this into account.

Overall, it is clear that dog bite treatments are controversial and multifactorial. An understanding of the causes, the treatments used, and the steps used in prevention and education would be gained from further research.

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LETTER

Levels of dog control and dog fouling in a large public park: methods issues and survey results

Nick Wilson

Companion dog ownership is a highly valued aspect of human society and one that is likely to provide both mental health benefits[1] and prevent cardiovascular disease[2](with the latter likely driven by increased walking levels by dog owners[3]). Although these benefits probably substantially outweigh the downsides of dog ownership, those problems can still be significant. For example, one estimate is that there are 240 hospitalisations per year from dog bite injuries in New Zealand,[4] with occasional deaths.[5]

One New Zealand study reported that the most common location for humans being bitten by dogs was public streets/walkways at 26% of the total, followed by various private settings (other locations were 4% in parks and 6% in other public areas).[6]

Other problems with dogs include: nuisance and anxiety when unleashed dogs approach people; nuisance impacts from barking and howling[7]; disturbance of wildlife[8]; and nuisance and disease risk from fouling with faecal material in public places. The relevant zoonotic diseases that could occur from contact with dog faeces include: campylobacteriosis, salmonellosis, cryptosporidiosis, *E. coli* infection, and toxocariasis (albeit with limited risk information in the NZ setting for some of these, such as for toxocariasis[9–11]).

Various observational studies have provided data on dog-related issues in public places (e.g., in the UK[12] and France[13]), but no study of dogs in New Zealand parks appears to have been published to date. Therefore, this study aimed to start addressing this knowledge gap.

Methods: Observations of park users walking their dogs were conducted in a single large urban park (Karori Park) in the suburb of Karori in Wellington City. The visits were all part of convenience visits to the park as part of routine fitness activities by the observers during weekends and evenings in the five months of May to September 2014. This park has sports fields, a children's play area, a defined dog exercise area, and a paved perimeter walking track. Observations were conducted with discretion (with a distance of at least 20 metres being typically maintained between the researcher/s and the people/dogs), so that dog walker behaviour would not change due to a sense of being observed. Data were entered into a smartphone in the field which contributed to the normalcy of the research process, given how common smartphone use is. This study had ethical review and approval (University of Otago process).

Results: The method of this observational study was found to be feasible in this New Zealand setting. It involved a total of 17.3 hours of observer time for60 park visits on 35 separate days (i.e., averaging 14.5different dogs observed per hour). Dogs were present on most park visits (80%) and were in view 58% of the time on average. There was no evidence that any park users were aware that the observational data were being collected and observers felt safe at all times.

Of the 250 dogs observed, the majority (90%) were kept on a leash, as required by the City Council bylaws (Table 1). However, bylaws were broken when dogs were off the leash (10% of dogs), when dogs were on the sports fields (8%), and when in the children's play area (5%). Dog defecation was observed on 23 occasions (Table 1). Faeces were picked up and removed, as required by the bylaw, the majority of the time (87%).

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Table 1. Observed dog behaviours, and human dog-walker behaviours in a large public park (Karori Park, Wellington 2014, n=250 dogs unless otherwise stated)

Dog behaviours and related human behaviours around dog management	Number	Percentage
Dog always on a lead (when outside the designated dog exercise area)	225	90.0
Dog off lead at least for some of the time (contrary to the bylaw)	25	10.0
Dog on the sports field (contrary to the bylaw)	20	8.0
Dog in the children's play area (contrary to the bylaw)	12	4.8
Dog observed to defecate	23	9.2
Dog faeces removed	20/23	87.0
Dog faeces not removed* (contrary to the bylaw)	3/23	13.0

* Includes a case where the person with the dog attempted to push the faeces off the grass and under a fenced tree. In contrast one dog owner followed their dog into a shrubbery area to remove the faeces.

Discussion: This study suggests it is feasible to do such observational studies of dog and human behaviour in at least some public parks in New Zealand. But as it was a convenience sample (fitting with observer preferences for outdoor exercise times),the data collection could probably have been more efficiently collected if times of high park use only were studied (e.g., only weekend afternoons on fine weather days).Yet a problem with focusing on popular park use times is that this would probably further bias the results towards detecting more law-abiding behaviours due to social pressures of more other people being around at such times.

The small size of this study and focus on just one (albeit large and popular) park means that it has limited generalisability, so ideally, further studies in a range of New Zealand parks seems desirable. Nevertheless, it does provide some limited evidence to suggest that people walking dogs are typically responsible and follow dog control bylaws. Indeed, these results for leash use and removing dog faeces were generally higher than those found in studies elsewhere (e.g., results for cleaning up faeces elsewhere were: 54% in a UK study[12]; 43% in a French study[13] and 56% and 63% in two other studies as cited by Gaunet et al[13]).

Yet it is still desirable that there is improved compliance with bylaws in the New Zealand setting to further minimise injury risk from bites from unleashed dogs and to reduce the risk of zoonotic disease transmission from dog faeces. Options for city councils might include:

Installing larger signage at park entrances – with the signs mentioning the word "bylaw" and possibly the size of the fine and the reasons for it ("dog fouling can spread disease"; "unleashed dogs can cause nuisance and injury to other park users"; "follow nearly all other dog owners in cleaning up after your dog").

Occasionally enhancing enforcement efforts with actual prosecution of offenders. Media publicity of prosecutions might also generate compliance improvements.

Possibly expand the provision of free dog waste bags at park entrances (along with dog-shaped waste receptacles that are used in some New Zealand parks).

But given the public health benefits of dog ownership (see *Introduction*), there may also be a case for expanding urban access to dog exercise areas in parks along with other interventions to promote dog walking in urban areas. These could include improved quality of street connecting walkways[14] and improved night-time lighting of park walkways to facilitate dog walking in the evenings.

Competing interests: Nil. (The author has enjoyed having pet dogs previously but is not a current dog owner. There was no funding for this study.)

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LETTER

The potential of citizen engagement and empowerment for obesity prevention in New Zealand

Stefanie Vandevijvere, Erica D'Souza, Boyd Swinburn

About one-third of children are now overweight or obese in New Zealand.[1] A range of New Zealand health professionals recently brought the problem of childhood obesity under the attention of policymakers.

The New Zealand Medical Association's report on Tackling Obesity[2] set out the top 10 priorities and a policy brief launched by the New Zealand Beverage Guidance Panel[3] identified the priorities for reducing fizzy drink consumption.

Additionally, a large New Zealand-based Expert Panel rated Government performance to create healthy food environments against international best practice and formulated 7 key priorities for immediate implementation4. However, the implementation of a comprehensive package of strong policies at the national level to reduce obesity typically takes time and is particularly difficult when the political climate for it is not optimal.

To date, the pressure for action on reducing obesity and diet-related non-communicable diseases (NCDs) has been predominantly elite (i.e. health professional driven), not grass-roots (i.e. public driven), and this is part of the reason that there has been little policy action. While most of the public are highly supportive of various policies to improve the healthiness of food environments, as shown in multiple studies in Australia.[5,6] it is a quiet support, and strategies, tools and processes are needed to effectively convert that support into vocal demands for increased and stronger actions on food environments. Government action is more likely with strong, visible public pressure.

Therefore, initiatives should be taken to engage and empower citizens and consumers to generate local actions to improve the healthiness of their community food environments. Local action could potentially be stimulated through encouraging and inspiring citizens to collect data through smartphone applications on the healthiness of their local food environment (e.g. outdoor food advertising, advertising through local sport clubs and events, healthiness of foods in schools and supermarkets, density of fast food outlets around schools)and feeding it back to local stakeholders, such as Members of Parliament, Council representatives, local NGO branches, and school and retailer representatives. An additional feature could be the benchmarking of local communities, schools and retailers according to the healthiness of their food environment.

Local, direct feedback to decision-makers and fostering relationships with them is arguably a more powerful way to convert evidence into action for healthier food environments than a national approach only (e.g. local parents providing feedback to schools versus a national survey written up in journals and reported in the media). Citizen engagement and empowerment might also strengthen and create social movements for healthy foods. The proposed approach holds promise as well for stimulating improvements in local physical activity environments.

Data on food environments, generated for local benefit of New Zealand communities, schools and retailers, could also feed into national monitoring of the healthiness of food environments and reduce the costs associated with it.

The first national survey on food environments and policies in the world is currently being organized in New Zealand[7] by the International Network for Food and Obesity/NCDs Research, Monitoring and Action Support (INFORMAS).[8] While national surveys and statistics have an important role to generate policy action, local data may be even more powerful. Examples from other disciplines

include smoking deaths by electorate, hospital data by District Health Board, and road deaths by locality.

To get such level of local data, novel data collection methods, such as crowdsourcing, will be needed, and their feasibility and validity tested. INFORMAS aims to test approaches to crowdsourcing data on the healthiness of community food environments and using the data to generate feedback for local stakeholders, in New Zealand in 2015–2016.

In general, the use of crowdsourcing has benefited many sectors of society, but it has yet to be fully realized as a method for improving public health.[9] A crowdsourcing approach through the FoodSwitch smartphone application,[10] has recently been successfully developed, tested and implemented in New Zealand to help consumers make healthier food choices. The app also allows consumers to contribute new products to the *Nutritrack* database of packaged food products.

The New Zealand *Nutritrack* database currently contains the nutritional content of more than 16000 packaged and fast foods (from major fast food outlets with ≥20 stores nationwide).[11,12] The app has been downloaded more than 55,000 times and users crowd sourced about 8000 new products in New Zealand. Another crowdsourcing example is *NatureWatch New Zealand,*[13] where nature-watchers record and share information online. In 18 months, 85,000 pictures have been uploaded with 50,000 observations from 785 observers.

Approaches to engaging and empowering citizens and consumers to improve the healthiness of their local food environments through crowdsourcing and local feedback loops will be tested in New Zealand, and might show potential for reducing childhood obesity rates, at times when implementation of strong policies at the national level proves to be challenging and slow. Ultimately, public pressure, in addition to the current pressure of health professionals, might accelerate the implementation of strong national policies to reduce childhood obesity in New Zealand.

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LETTER

What does the 2025 Smokefree Goal mean to the New Zealand public?

Philip Gendall, Janet Hoek, Richard Edwards

In 2010, the Māori Affairs Select Committee conducted an inquiry into the tobacco industry and the consequences of tobacco use for Māori. Following a comprehensive submission and hearings process, the Committee recommended that the government aim to halve tobacco consumption and smoking prevalence by 2015 across all demographics, with the ultimate goal of "making New Zealand a smokefree nation by 2025."[1] The National Party-led Government supported this recommendation and set out its commitment to the "longer-term goal of reducing smoking prevalence and tobacco availability to minimal levels, thereby making New Zealand essentially a smokefree nation by 2025".[2] (The tobacco control sector has defined "minimal levels" as less than 5%.)

New Zealand's world-leading goal has strong public support,[3–5] though comprehensive endorsement by politicians remains elusive.[6] However, while studies have tested public support for endgame components and new policy measures,[4,7] they have not explored public understanding of what the 2025 goal might entail. As a result, we know little about how the general public interpret either the goal or its implications. Such knowledge is important as misperceptions may reduce public support for the goal and exacerbate politicians' apparent reluctance to introduce proportionate tobacco control policies.

Using an online survey of 833 smokers and non-smokers sourced from the ResearchNow internet panel, we tested the New Zealand public's support for the 2025 goal when this was presented as an unexplained concept and when presented with a definition. The sample was stratified by smoking status (daily n=335; intermittent n=73; former n=160; never n=265), gender and age and weighted to correspond to the age-gender distribution of smokers and non-smokers reported in the 2013 Census.[8]

First, we asked respondents: "Do you support or oppose the Smokefree 2025 goal, or do you have no preference either way?", with no explanation of the 2025 goal. In the second question, asked at the end of the survey and separated from the first by 27 other questions, we defined the goal as: "To reduce smoking prevalence and tobacco availability to minimal levels, thereby making New Zealand essentially a smokefree nation by 2025. (This is often assumed to mean reducing smoking to less than 5% of people in all population groups.)" before asking: "Do you support or oppose this Smokefree 2025 policy goal, or do you have no preference either way?"

Table 1 below contrasts responses to these two questions and shows several patterns. Just over 30% of respondents reported either having not heard of the 2025 goal or having no preference one way or the other. Once provided with a definition of the goal, support increased significantly overall and among three of the four smoking status groups examined (most likely only small numbers in the sub-sample of social smokers prevented the change in support from reaching statistical significance for this group). Predictably, support for the goal shows a clear and consistent pattern by smoking status: non-smokers were consistently more likely, and daily smokers less likely, to support the 2025 goal, regardless of whether it had been explained.

Perceptions of what the 2025 goal encompassed also varied significantly by smoking status (see Table 2). A majority in all groups thought the goal meant smoking would be disallowed in public places (80%) and reduced to very low levels (61%); never smokers were significantly more likely to hold this view than daily smokers. By contrast, daily smokers were more likely than other groups to view the goal as banning the sales of tobacco (53%) and smoking itself (52%), and aiming at a country where no-one smokes (56%). These perceptions of the 2025 goal as a ban on smoking may account for the relatively high level of opposition to it among daily smokers.

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Table 1. Support for the 2025 Smokefree Goal when *unexplained* and *explained*

	Support when goal was *unexplained*				
	Daily Smoker (n=335) %	**Social Smoker (n=73) %**	**Former Smoker (n=160) %**	**Non-smoker (n=265) %**	**Weighted sample (n=833) %**
Support	19[a]	44	49[b]	71[c]	56[d]
Oppose	48	30	13	2	12
No preference	24	21	24	9	16
Never heard of goal	10	6	15	18	16
Total	**100**	**100**	**100**	**100**	**100**
	Support when goal was *explained*				
Support	28[a]	53	69[b]	90[c]	74[d]
Oppose	48	29	11	2	11
No preference	24	18	20	8	14
Total	**100**	**100**	**100**	**100**	**100**

Note: Superscripts (a,b,c,d) denote statistically significant ($p<0.05$) changes in support within each group (column) between unexplained and explained questions.

Table 2. Perceptions of the Smokefree2025 Smokefree Goal

By 2025	**Yes, part of goal**					**p-value for χ^2 test**
	Daily Smoker (n= 335) %	**Social Smoker (n= 73) %**	**Former Smoker (n= 160) %**	**Non-smoker (n=265) %**	**Weighted sample (n=833) %**	
No smoking is allowed in any public place in New Zealand	77	70	76	83	80	.010
Smoking in New Zealand is reduced to a very low level	50	58	57	67	61	.000
Less than 5% of people in New Zealand smoke	39	53	47	53	49	.001
Sales of cigarettes and tobacco in New Zealand are restricted to very few outlets	40	48	38	52	46	.000
No retail sales of cigarettes or tobacco in New Zealand	53	45	46	43	45	.008
No-one in New Zealand smokes	56	43	41	35	40	.000
Smoking is banned in New Zealand	52	43	39	33	37	.000

Our findings have two important implications; first, they suggest greater knowledge of the 2025 goal translates into stronger support. It should thus be of concern to tobacco control advocates that nearly 20% of non-smokers (the group with the strongest support for the goal) reported being unaware of it. Second, they reveal considerable misunderstanding of the goal itself, which is likely to stimulate reactance and opposition, particularly among smokers, half of whom interpreted the goal as amounting to a total ban on smoking or the sale of tobacco. Furthermore, the most common interpretation was that smoking would not be allowed in any public place by 2025, a perception that is also not set out in the goal.

We suggest three responses to our findings. First, we urgently need a comprehensive mass media campaign promoting understanding and uptake of the 2025 goal.[9] Second, because mass media

advertising will support quitting, cessation support must become more widely available so the vast majority of smokers who regret smoking will be more easily able to act on their desire to be smokefree.[10] Finally, the government must articulate and endorse the goal it set, and should give it a high political priority, for example by making it a Better Public Service target.

The 2025 goal is truly world-leading; its achievement would herald enormous health, social and economic benefits and act as an exemplar the rest of the world could follow.

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LETTER

"Enjoy the future of smoking"

Brian R McAvoy

The development and promotion of electronic cigarettes (e-cigarettes) has polarised medical opinion over policy, with advocates for tougher legislation pitched against those promoting harm minimisation.[1]

Currently there is no robust evidence on the long-term effects, efficacy in harm reduction or smoking cessation, or safety of e-cigarettes. In the UK, marketing of e-cigarettes is extensive, and growing rapidly. They are promoted as lifestyle products and are available in a wide range of flavours and packaging that is likely to appeal to children and young people.[2] They are available in an extensive range of venues, including some pharmacies, shopping malls, supermarkets and petrol stations. Spending on e-cigarette promotion in the UK increased from £1.7 million in 2010 to £13.1 million in 2012.[3]

The tobacco industry is taking an increasingly dominant role in this market.[3] Although the e-cigarette industry claims that it is promoting its products to encourage tobacco smoking cessation, its marketing techniques, embracing aesthetic appeal, internet and social media tools, celebrity endorsement and sports sponsorship, suggest otherwise.

On a recent visit to Scotland I noticed this prominent advertisement on top of a petrol station bowser (Figure 1). The tagline "Enjoy the future of smoking" reveals the true motivation of the manufacturers. Such advertising directly challenges the unambiguous "say no to smoking" message, and undermines hard-won tobacco control policies such as smokefree public places, age restricted sales and point of sale restrictions.

While awaiting clear evidence to emerge on the role, if any, of e-cigarettes in smoking cessation, it is vital that governments and public health authorities act rapidly to counter unfettered commercial exploitation of vulnerable populations, especially young non-smokers, pregnant women and adults with heart disease.

The New Zealand Ministry of Health should continue to support the World Health Organization advice on electronic nicotine delivery systems,[4] and should be vigorous in monitoring and enforcing the restriction of their sales to pharmacies.

The New Zealand Government needs to resist undermining of its tobacco control laws and endeavour to limit marketing of e-cigarettes until the research evidence is available to establish their place (if any) as a safe and effective component of tobacco control at the individual and population levels.[5]

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Figure 1. Advertisement at a petrol station in Scotland

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100 YEARS AGO

Letter: Treatment of the Insane (part 2)

End of a letter written by "Physician" and published in NZMJ 1914 March;13(49):60–6 (continued from part 1 in the 7 Nov 2014 edition)

In making these strictures on the conduct of asylums and in commenting on the hopelessly bad treatment to which the insane are subjected I do not wish it to be understood that I blame the staffs of these institutions. They do their best, and that it is a very poor best is not their fault. To enable them to deal effectively with the situation would mean the expenditure of large sums of money. Probably half a million would be required to bring things up to date.

It is rather more doubtful whether the head of the asylum service and his senior assistants can be so completely exonerated. They one and all must study know how far their service falls short in common humanity to the insane, and that it is hopelessly behind the English system. Is it not their duty to bring much more vigorous pressure to bear to secure the necessary funds. Are they facing the situation as courageously as they might?

The time is opportune in that the present Government is not responsible for the present state of affairs, and can remedy it without condemning its own past. One thing is certain, if the public generally knew, as some of us know, how the insane are treated in New Zealand, there would be such an outcry that the money necessary for reform would soon be forthcoming.

I enclose my card, and regret that for various reasons it is necessary to shelter behind the nom de plume

PHYSICIAN.

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It is rather more doubtful whether the head of the asylum service and his senior assistants can be so completely exonerated. They one and all must surely know how far their service falls short in common humanity to the insane, and that it is hopelessly behind the English system. Is it not their duty to bring much more vigorous pressure to bear to secure the necessary funds? Are they facing the situation as courageously as they might? The time is opportune in that the present Government is not responsible for the present state of affairs, and can remedy it without condemning its own past. One thing is certain, if the public generally knew, as some of us know, how the insane are treated in New Zealand, there would be such an outcry that the money necessary for reform would soon be forthcoming.

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METHUSELAH

Accuracy of urinary human papillomavirus (HPV) testing for presence of cervical HPV

The authors of this study note that there has been a downward trend in the uptake of cervical cancer screening in women aged less than 35 years, as sampling is invasive, time consuming, and requires a clinician. Cervical testing for HPV is being piloted as a more accurate method for cervical cancer screening, but it is invasive and time consuming. They speculate that testing the urine for HPV DNA may be feasible alternative.

Their review and meta-analysis involved 16 articles reporting on 14 studies involving 1443 women. Most used commercial polymerase chain reaction methods on first void urine samples. Detection of high risk HPV had a pooled sensitivity of 77% and specificity of 88%.

The researchers conclude that detection of HPV in urine seems to have good accuracy for the detection of cervical HPV, and using first void samples was more accurate than random or midstream sampling. When cervical testing is considered difficult, urine testing should be regarded as an acceptable alternative.

BMJ 2014;349:g5264.

Ultrasound vs CT for suspected nephrolithiasis

This issue is investigated in this multicentre trial in the USA. 2759 patients with suspected nephrolithiasis underwent randomisation. 908 had diagnostic ultrasonography performed by an emergency physician. 893 had sonography by a radiologist and 958 had CT.

The sensitivity and specificity for the diagnosis of nephrolithiasis were similar in the three study groups. There were no significant differences between the three groups in terms of serious adverse events, pain scores, return emergency department visits or hospitalisations. The mean 6-month cumulative radiation exposure was significantly lower in the ultrasonography groups.

N Engl J Med 2014;371:1100–1110.

Guideline-based antibiotic treatment for acute exacerbations of chronic obstructive pulmonary disease (COPD)

The Lung Foundation of Australia has formulated guidelines for the treatment of acute exacerbations of COPD. They include the prescription of bronchodilators, a short course of systemic corticosteroids and oral antibiotics. The recommended antibiotic is either amoxicillin or doxycycline.

This study has reviewed adherence to the guidelines in patients admitted to a tertiary hospital with acute exacerbation of their COPD. The case records of 84 such patients have been reviewed. 72 of the 84 (85.7%) received guideline-discordant antibiotics, of whom 76% received intravenous antibiotics. The mean length of stay in hospital was significantly lower among patients receiving guideline-concordant therapy compared with those receiving guideline-discordant therapy (mean 1.6 days vs 3.7 days; $P = 0.002$). There was no significant difference between groups in rates of readmission.

The researchers estimate an excess cost per patient associated with guideline-discordant treatment to be $Aus 2642.

Internal Medicine Journal 2014;44:903–910.

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OBITUARY

Ronald James (Jim) Methven

Ronald James Methven, the first full-time Consultant Child Psychiatrist to the Auckland Hospital died aged 86 on 23 August 2014. He was the founding father of child mental health services in Auckland.

Jim, as everyone called him, and the name he preferred, was born in Nelson the older of the two children of Ronald Henley Methven MA (1897–1944) and Ethel Mary Methven née Peck MA (1901–1985).

His father, Senior French master at Nelson College and connected to the well-known South Island Methven family, had been born in New Zealand. Jim's mother was born in England and came to New Zealand in her teens with her own father, Frank Peck FRIBA, the English architect who designed Nelson Cathedral.

Jim attended Nelson College and distinguished himself at swimming being Junior (1943), Intermediate (1944) and Senior (1945) School Champion and winning School "blues" for swimming in 1944 and 1945.

He represented Nelson at the New Zealand Amateur Swimming Championships in 1944. Swimming was a lifelong interest and he coached his grandson, Nathan Smith, to victory in the national junior 100 metres backstroke in 1996.

Early in 1945 Jim, now in the sixth form, left school because his father had died in 1944 and he felt the need to support his mother and sister financially. He became apprenticed to Hinchcliffe's, a Nelson chemist. He took the Diploma in Pharmacy in 1949 and worked in Palmerston North and Nelson Public Hospitals. Not entirely happy at the prospect of a career in pharmacy Jim switched to medicine, graduating MB ChB (NZ) from Otago University in 1957.

While a junior doctor at Nelson Hospital (1958–59) Jim decided to specialise in psychiatry. In 1960 he entered the training programme of the Victoria State Mental Health Authority with medical officer posts at Ararat Hospital (1960) and Larundel Hospital (1961–3). He took the University of Melbourne Diploma in Psychological Medicine in 1963.

Deciding to become a child psychiatrist he took further postgraduate training at the Royal Melbourne Children's Hospital (1963–65). His last post in Australia was Consultant Psychiatrist at the Observatory Child Psychiatry Clinic in Melbourne.

Jim returned to Auckland in 1966 to be Director of the New Zealand Department of Health's Child Health Clinic at Marinoto and to provide outpatient and liaison services to Princess Mary Hospital. In 1970 the Child and Family Unit (CFU) opened to provide the inpatient service. This was based in Ward 12, an aged standalone building in the grounds of Auckland Hospital near the Princess Mary Hospital. The upper admission age of 14 years was soon raised to 15 then to 17. Adolescents with schizophrenia, bipolar illness and anorexia nervosa could then be treated. The CFU provided a national inpatient service for some 10 years until child psychiatry services developed in the other

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1406/6376

main centres. In 1993 the CFU moved into the new Starship Children's Hospital. Jim retired from full-time work in 1993 but continued part-time consulting work for five more years.

In 1970 Jim was joined by John Werry as a part-time consultant. John had been appointed Professor of Psychiatry in the University of Auckland in 1970. He brought the academic and research outlook into child psychiatry. The CFU taught child psychiatry to medical students and to trainees in paediatrics and in child psychiatry. Jim and John maintained a close, harmonious working relationship based on mutual respect and clear domains of responsibility.

Jim did no personal research, preferring to develop the service and to participate in the educational efforts of the Royal Australian and New Zealand College of Psychiatrists. This resulted in a training programme in Auckland leading to a postgraduate qualification in child psychiatry. For this work Jim received an RANZCPsych citation in 1973.

Jim believed nuclear war to be the pre-eminent threat to the survival of humankind and he opposed the proliferation of nuclear weapons. To further his opposition he joined the New Zealand Affiliate of International Physicians for the Prevention of Nuclear War (IPPNW) and was Chairman from 1993 to l999. With Erich Geiringer he represented New Zealand at IPPNW conferences in Moscow (1987), Hiroshima (1989) and Mexico City (1993).

The writings of the great philosophers exercised Jim in his later years extending a life-long interest in philosophy. He shared his reflections with his friends by leading monthly seminars of the West Auckland U3A called Great Ideas.

Jim was twice married. In 1957 he married Jeanette (Jan) McKenzie; they had three children. They divorced in 1994. In 1995 Jim married Kate Hyde-Smith. Jim is survived by Kate; by Jeanette and the three children of their marriage; by three step-children from Kate's former marriage; nine grand-children, two great grand-children and his sister Dorothy Carmody.

Jim's legacy is the child mental health service in Auckland which today employs over 500 staff. Jim was a pioneer in Auckland and his influence extended to the rest of New Zealand and to Australia.

Dip Pharm 1949; MB ChB (NZ) 1957; DPM (Melb) 1963; FRANCP 1979. Born Nelson, 5 August 1928. Died Auckland, 23 August 2014.

Still are thy pleasant voices, thy nightingales, awake;
For Death, he taketh all away, but them he cannot take.

John Werry and Brian Barraclough wrote this obiuary.

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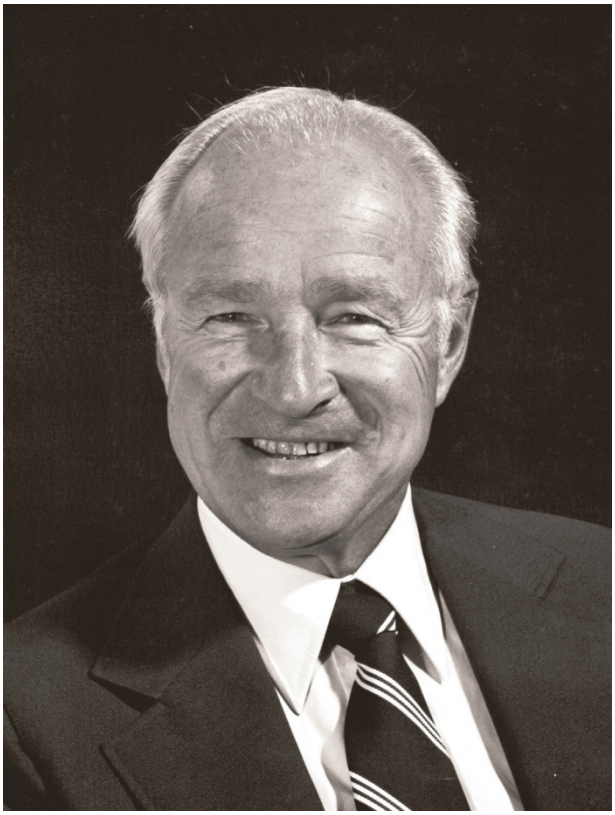
OBITUARY

James Blaul Kriechbaum

(10 June 1918 – 26 October 2014)

Jim Kriechbaum was born and raised in Burlington, Iowa. Both his parents were of German descent, and his father was a general surgeon. Jim graduated BA in 1940, and MD from the University of Iowa in 1943.



In the Second World War Jim served as a captain in the United States Medical Corp. After the war, Jim trained in ophthalmology at St Luke's Hospital in Chicago, and subsequently passed the American Boards in Ophthalmology.

During his ophthalmology training, Jim had the good fortune to meet Dr June Reid, an Otago medical graduate who was gaining overseas experience in cardiology. Jim and June were married in 1949.

They lived and practised in Chicago, but June persuaded Jim that they would enjoy a better life in New Zealand. However Jim's American Board Certification in Ophthalmology was not accepted by the New Zealand Medical Council for specialist registration.

They lived in England for a year in order for Jim to pass the Diploma in Ophthalmology which was acceptable, despite being a lesser qualification. They finally settled in New Zealand in 1954.

Jim was highly regarded and consequently was invited to join the practice of Dr Cecil Pittar, who at the time was New Zealand's leading ophthalmic surgeon. Later they were joined by Lindsay Poole, and in 1978 by Bruce Hadden. Jim was also appointed as a part-time visiting ophthalmologist at Auckland Hospital, with his special interest and expertise in glaucoma.

Jim was of the old school, in that he did not delegate the routine tasks to technicians. He did everything himself for his patients, from prescribing glasses through to surgery. Jim's patients were in knowledgeable, intelligent, and competent hands. He freely sought opinions from colleagues with difficult cases. "The patients' interests are paramount" was Jim's modus operandi, long before this maxim came to be written into a code of conduct.

The three of us practised together in Symonds Street, until the people renting the floor below us were arrested for growing marijuana plants under fluorescent light tubes. We figured it was not a good look for our professional practice, and purchased a building in nearby Mount Street, where we worked happily for many years.

In all our years together, there was never a cross word between us. Jim was a gentleman. He had great integrity, and he was frank. He had a sense of fun with an extraordinary supply of jokes. He was the ideal business partner- communicative, trustworthy, and no hidden agenda.

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Jim and June were happily married for 65 years. They were caring and intellectually stimulating parents to their five children. Anthony and Jim were both dux of Auckland Grammar, a record almost unique to the Kriechbaum family. Both are general practitioners, Anthony in Palmerston North and Jim in Auckland. Caroline is a veterinary surgeon, Christopher an Air New Zealand pilot, and Sara is with the St Johns Ambulance. Together they have provided Jim and June with the joys of fifteen grandchildren.

New Zealand has lost an ophthalmologist who was greatly liked and respected by both his colleagues and his patients. On their behalf, our sincere sympathy to June, and to their children and grandchildren.

Bruce Hadden (Associate Professor, Department of Ophthalmology, University of Auckland) wrote this obituary.

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PROCEEDINGS

Proceedings of the 226th Scientific Meeting of the Otago Medical School Research Society, Wednesday 5 November, 2014

Effect of myocardial ischemia-reperfusion on cardiac sympathetic nerve activity in the rat. T Hall, D Schwenke. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

Acute myocardial infarction (MI) is characterised by occlusion of a coronary artery. Early mortality following MI is associated with a significant and sustained increase in cardiac sympathetic nerve activity (cSNA), which enhances the risk of potentially fatal ventricular arrhythmias. Surgical restoration of blood flow to the ischemic myocardium undoubtedly improves patient prognosis, however, it remains unknown whether this reperfusion therapy provides the additional benefit of normalising the dangerously elevated cardiac sympathetic tone. Accordingly, the aim of this study was to investigate the effect of coronary reperfusion therapy on cSNA in a rat model of myocardial ischemia.

Twenty-five male Sprague-Dawley rats were anaesthetised. A left thoracotomy exposed the heart, enabling the cardiac sympathetic nerve to be isolated for electrophysiological recording of cSNA using platinum recording electrodes. MI induction was achieved via ligation of the left anterior descending coronary artery. Following 45 minutes of cardiac ischemia, the ligature was released allowing full restoration of coronary blood flow. Sham controls (n = 8) underwent the identical surgery but did not receive MI. cSNA was quantified by integrating the raw nerve signal and calculating nerve firing frequency and spike amplitude before, during 45 minutes of ischemia, and then for two hours of coronary reperfusion.

Forty-five minutes of cardiac ischemia elicited a substantial rise in the integrated SNA compared to pre-MI (61 ± 11 %, mean ± SEM) which further increased following reperfusion (188 ± 28 % at two hours reperfusion, $P < 0.0001$, 1-way ANOVA). At two hours reperfusion, firing rate (189 ± 30 %, $P < 0.0001$) and spike amplitude (90 ± 21 %, $P < 0.0001$) were also significantly elevated.

These findings indicate that reperfusion treatment following a 45 minute period of myocardial ischemia may not attenuate the elevated cSNA, and could in fact exacerbate adverse sympathetic hyper-activation and risk of arrhythmias.

Administration of secreted amyloid precursor protein-α rescues spatial memory and long-term potentiation deficits in aged rats. M Xiong[1], O Jones[1], K Peppercorn[2], W Tate[2], W Abraham[1]. Brain Health Research Centre, [1]Department of Psychology, [2]Department of Biochemistry, Otago School of Medical Sciences, University of Otago, Dunedin.

The senescent brain is highly susceptible to cognitive impairment. Spatial memory deficits, in particular, arise from cellular changes in the hippocampus, a critical structure for memory of events

and places. Long-term potentiation (LTP) is a persistent strengthening of synaptic connections between neurons which underlies memory formation, and is also impaired during aging. We hypothesised that an acute application of secreted amyloid precursor protein-α (sAPPα), a neuroprotective and memory-enhancing molecule, would ameliorate these aging-related deficits.

Male Long-Evans rats were tested for spatial memory using the novel object-location task. Aged rats (24-mo) injected with sAPPα (300 nM, 2 µL, n = 8) and young rats injected with a vehicle solution (5-mo, n = 9) preferentially explored a familiar object displaced to a novel location while vehicle-treated aged rats did not (n = 6, $P < 0.05$, 1-way ANOVA, post-hoc LSD test). This result indicates that transient sAPPα exposure is capable of facilitating spatial memory in cognitively impaired animals.

One week after memory testing, the relation between behavioural performance and LTP was investigated. The same animals were euthanised and hippocampi were dissected for *in vitro* extracellular electrophysiological recordings. Transverse hippocampal slices (400 µm) were superfused with artificial cerebrospinal fluid, and then treated with either a control solution or sAPPα (10 nM) for 30 minutes before the induction of LTP via theta-burst stimulation (TBS). Control slices from aged animals showed no LTP one hour post-TBS (102 ± 5% of baseline, n = 8) whereas similar slices treated with sAPPα (125 ± 6%, n = 5) and slices from young control animals (133 ± 7%, n = 4) displayed robust LTP ($P < 0.05$).

Combined, these results indicate that sAPPα can rescue physiological processes necessary for spatial memory in aged rats and consequently may have potential as a therapeutic agent in normal and pathological aging.

Detection of a single prostate cancer derived cell transcriptome in a background population. C Harris, R Day, T Godwin, D Zhou, P Guilford. Department of Biochemistry, Otago School of Medical Sciences, University of Otago, Dunedin.

Rare populations of prostate cancer cells have been found in the urinary *milieu* of patients with early stage prostate cancer. This study aimed to design a method capable of detecting the transcriptome of single prostate cancer derived cells (PC3 cells) in a simulated *milieu*. The eventual goal is to detect prognostic urinary mRNA biomarkers for prostate cancer.

We analysed the transcriptomes of single PC3 cells and homogenous populations of 30 PC3, 30 HeLa and 5 HeLa cells. We simulated a scarce cell type in a background *milieu* by micro-pipetting single PC3 cells into different background populations of the readily available HeLa cell type (4 cells or 29 cells). All transcriptomes were amplified and sequenced by next generation sequencing.

The mRNA transcripts from the *MAGED1* gene were capable of indicating the presence of a single PC3 cell in background populations of HeLa cells. The *MAGED1* gene was significantly highly expressed in a mixture (1 PC3 cell in 29 HeLa cells) compared to HeLa homogenous populations (30 cells), (log fold change = 5.8, n = 12, $P < 0.01$; empirical Bayes statistics with false discovery rate control). The average transcriptome correlation between populations of 30 PC3 cells (n = 3) and an averaged population of 30 HeLa cells was high (n = 4), (R = 0.92 ± 0.003, CI = 0.95). Interestingly, the average transcriptome correlation between single PC3 cells (n = 8) and an averaged population of 30 PC3 cells (n = 3) was lower (R = 0.83 ± 0.03, CI = 0.95).

The low correlation between PC3 single cells and PC3 populations indicates the challenge of biological and/or technical stochasticity in single cell approaches. Our mixture experiment demonstrates that single cells can be detected in a background population, supporting a multi-cell sampling approach to highly sensitive diagnostics and prognostics.

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1406/6377

The pathophysiological role of microRNAs in diabetic cardiac stem cells. N Purvis, A Bahn, R Katare. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

Recently, a population of resident cardiac stem cells (CSCs) have been found in the heart. These cells are known to re-constitute dead cardiomyocytes during ischaemic injury by undergoing cell proliferation and differentiation. Several studies have demonstrated the ability of transplanted healthy CSCs to regenerate damaged tissue in infarcted mice hearts. Diabetes mellitus leads to a progressive loss in the number and functional efficacy of CSCs and transplanted CSCs from diabetic heart failed to improve the recovery of damaged heart tissue. However, the mechanism behind this loss is not clear. MicroRNAs (miRs) are small, non-coding RNA molecules that regulate gene expression at the post-transcriptional level. This study aims to demonstrate the effect of diabetes on miR expression in CSCs, to further understand their pathophysiological role within these cells.

CSCs were isolated from type-2 diabetic (n = 24) and age-matched non-diabetic mice hearts (n = 24) using collagenase digestion, followed by purification of the cells using stem cell specific antigen-1 (Sca-1) magnetic labelling. RNA was extracted from the CSC samples, and miR expression profiles were analysed using an nCounter miR expression assay (Nanostring Technologies). Among 601 miRs evaluated, 16 miRs were significantly altered (14 up-regulated and 2 down-regulated) in diabetic CSCs. The online tool, 'MiRPath', was used to identify signalling pathways and common target genes which are regulated by these miRs, revealing 7 miRs which are involved in regulating cell proliferation/differentiation, the predominant function of CSCs. The expression profiles of 4 of these 7 miRs were validated by a miR-specific RT-PCR, narrowing the target miRs to miR-376c, miR-329, miR-495, and miR-30c. The functional efficacy of these miRs in diabetic CSCs was also investigated by Western blot analysis, which showed a slight increase in the expression of Glycogen synthase kinase 3 beta, a key gene regulated by the 4 target miRs.

Our results thus far suggests that diabetes alters the normal expression pattern of miRs in CSCs, and that certain miRs may regulate the functional efficacy of CSCs.

Combination intervention with atenolol and diazepam, but not diazepam alone, is cardioprotective against seizure-induced cardiomyopathy. R Millen, M Read, D McCann, J Harrison, D Kerr, I Sammut. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

Seizures have become frequently associated with an increased risk of cardiomyopathy, with electrocardiograph (ECG) and pathological structural changes commonly reported in both clinical and animal studies. This study examined whether intervention with the peripheral β_1-adrenergic antagonist, atenolol, in combination with the anticonvulsant, diazepam, would prevent cardiac injury in an animal seizure model.

Male Sprague-Dawley rats (n = 6 – 8 per group) were instrumented with ECG/electroencephalograph transmitters and seizures induced by intrahippocampal kainic acid (KA; 2 nmol). Saline, diazepam (5 mg/kg initial then 1 mg/kg bid., subcutaneous), or diazepam + atenolol (5 mg/kg, subcutaneous) interventions were administered at 1 hour post-KA and continued daily for 7 days. Plasma cardiac troponin I levels were analysed by an enzyme-linked immunosorbent assay, and cardiac immunohistochemistry was performed examining apoptosis (terminal deoxynucleotidyl transferase dUTP nick end labelling assay), collagen I deposition, and macrophage (CD68) infiltration. Apoptosis and macrophage infiltration were quantified by cell counting, and collagen I by digital positive-pixel quantification.

KA induced high-level seizure behaviours (19-fold increase in cumulative score; $P< 0.05$ vs. baseline) with simultaneous ECG data demonstrating increased heart rate (495 ± 20 beats per minute; mean ± SEM; $P< 0.05$ vs. baseline; 2-way ANOVA, Bonferonni *post-hoc*) and pronged QT intervals (1.31-

fold increase above baseline; $P< 0.05$). Myocardial injury was evident in saline-treated animals, with increased troponins (5-fold above baseline at 24 hours, $P< 0.05$), apoptosis (15-fold increase), macrophage infiltration (84.0 ± 8 cells/mm^2), and collagen deposition (1.4-fold increase) (all immunohistochemistry $P< 0.05$ vs. naïve controls). Diazepam intervention alone effectively attenuated seizure behaviours (45% reduction, $P < 0.05$ *vs*. saline-treated) but failed to reduce heart rate, QT intervals, and cardiac injury. Conversely, atenolol + diazepam treatment successfully attenuated seizure behaviours, tachycardia, QT prolongation, and prevented all cardiac injury (all $P< 0.05$ *vs*. saline-group).

This study clearly demonstrated that combination therapy with diazepam and atenolol, but not diazepam monotherapy, is cardioprotective in KA-induced seizures, supporting the adjunct use of atenolol in seizure management.

Antiviral properties of natural extracts against Herpes Simplex Virus Type 1. A Menorca, S Mros, H Brooks, L Wise, M McConnell. Department of Microbiology and Immunology, Otago School of Medical Sciences, University of Otago, Dunedin.

Human Herpes Simplex Virus type 1 (HSV-1) is present in 40-80% of the world's population and infection leads to recurrent oral lesions. HSV-1 infections are currently treated with nucleoside analogues such as acyclovir. However, there is increasing resistance by HSV-1 strains to nucleoside analogues and their use is associated with cytotoxicity, particularly in immunocompromised individuals. Extracts derived from natural sources are widely utilised in traditional medicine for treating skin infections. The aim of this study was to investigate whether natural extracts possess anti-HSV-1 activity.

Human keratinocytes (HaCat) and green monkey kidney epithelial (Vero) cells were infected with HSV-1. Cell death was detected using the crystal violet (CV) assay. Extracts were added to the cells concurrently with the virus, or at select time-points prior to or after infection and the ability of the extracts to reduce cell death was assessed. A NHS ester-activated fluorescent dye was conjugated to primary amine residues on HSV-1 virions so as to enable cell infection to be visualised using a fluorescent microscope.

Concurrent treatment with wine waste extract (1.25 mg/mL) or chitosan (12.5 mg/mL), a polysaccharide from crustacean shells, was associated with a 3-fold reduction in cell death compared with the HSV-1 control, with cell viability restored to that of uninfected cells ($n = 18$, $P< 0.05$, ANOVA with Dunnett's multiple comparisons test). Pre-incubation of cells with chitosan, prior to infection also prevented HSV-1-induced cell death (2-fold reduction compared with HSV-1 control, $n = 18$, $P< 0.05$). Visualisation of infection with fluorescently-labelled HSV-1 confirmed that chitosan inhibited viral attachment and/or entry into cells.

This study is the first to identify natural extracts that can prevent HSV-1 infections of cells. These findings suggest that natural compounds such as chitosan and extracts from wine waste may be viable treatment options for HSV-1 infections.