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This Issue in the Journal

Ethnic disparities in nutrition-related mortality in New Zealand: 1997–2011
C Lawes, N Stefanogiannis, M Tobias, N Paki Paki, C Ni Mhurchu, M Turley, S Vander Hoorn, A Rodgers

This article estimates the number of deaths due to four nutrition-related risk factors—high blood pressure, high cholesterol, high body mass index (BMI), and inadequate vegetable and fruit intake in 1997—with a close look at Māori/non-Māori disparities. The burden of disease that could be potentially avoided in 2011 if exposure to these risk factors were reduced is also estimated. About 47% of deaths among Māori and 39% of deaths among non-Māori were estimated to be due to the selected risk factors. Small changes in risk factor levels could have a major impact on population health within a decade, with relatively greater gains for Māori, particularly with improvements in BMI.

Flies, fingers, fomites, and food. Campylobacteriosis in New Zealand—food-associated rather than food-borne
W Nelson, B Harris

Diarrhoea caused by Campylobacter bacteria occurs following eating or drinking contaminated products. Chicken meat is known to be highly contaminated with Campylobacter, and this is commonly assumed to be the major source of the disease in humans. Eating raw chicken, or raw foods that have been cross-contaminated by chicken, is thought to be the most likely transmission route. This paper suggests an alternate route, taking into account both the highly seasonal nature of the disease and the strong association with eating chicken that is commonly reported.

Dietary intakes by different markers of socioeconomic status: results of a New Zealand workforce survey
P Metcalf, R Scragg, P Davis

Dietary nutrient intakes and food group servings were compared across various measures of socioeconomic status in 5517 Maori, Pacific, and Other workers aged 40 to 78. The socioeconomic measures were income, education, and the occupation-based New Zealand Socioeconomic Index (NZSEI). In general, there were trends across socioeconomic status levels—i.e. lower NZSEI occupation classes, lower family income, and secondary school- or trades education-only groups had lower intakes of dietary fibre, calcium, and alcohol as well as higher intakes of dietary cholesterol (reflected by their lower intakes of fruit, vegetables, milk, cheese, and wine; and higher intakes of eggs). However, these findings were not consistent across all measures of socioeconomic status. Lower socioeconomic (poorer) classes were associated with a less healthy pattern of food group selections (and dietary nutrient intakes) compared to the highest socioeconomic (richer) classes.
Metabolic characteristics of patients with apparently normal fasting plasma glucose
G Braatvedt, G Gamble, C Kyle

The limits for defining normal fasting glucose are under debate. In this study, 310 patients (not known to have diabetes) underwent oral glucose tolerance testing (OGTT) and were classified according to their fasting glucose as <5.5 (normal), 5.5–6.0 (“high fives”), 6.1–6.9 (impaired fasting glucose), and ≥7.0 mmol/L (diabetes). Almost one-third (111) of the patients had a normal fasting glucose (of <5.5 mmol/L), but of these, 23 had impaired glucose tolerance (IGT) and 2 diabetes after OGTT; 85 patients had a fasting glucose 5.5–6.0 mmol/L, and 18 of these had IGT and 11 diabetes after OGTT; 75 patients had a fasting glucose of 6.1–6.9 mmol/L, and of these, 33 had IGT and 21 diabetes after OGTT; and 39 patients had a fasting glucose ≥7 mmol/L, and 38 were confirmed as having diabetes after OGTT. This study suggests that the upper limit of normal fasting glucose be lowered from the current 6 mmol/L to <5.5 mmol/L (in line with Australian and American Diabetes Society guidelines).

Atherogenic lipid profiles in rheumatoid arthritis
D White, S Fayez, A Doube

This study confirms the association of active rheumatoid arthritis with abnormal lipid profiles which may be partly responsible for the increased cardiovascular mortality of this condition. We demonstrated a high level of untreated lipid abnormalities in our cohort and argue that as well as good disease control, attention to traditional cardiovascular risk factors is important in rheumatoid arthritis patients.

Botulinum toxin type-A (Botox-A) injections for treatment of sialorrhoea in adults: a New Zealand study
S Shetty, P Dawes, D Ruske, M Al-qudah, B Lyons

Sialorrhoea (drooling or excessive salivation) is a common problem for a considerable number of persons with cerebral palsy, intellectual disability, and other neurological conditions. The injection of Botox into the parotid and submandibular glands is safe and effective in controlling drooling; provides the most effective treatment of significant sialorrhoea; and can greatly improve the quality of life of patients and their families or caregivers. We discuss our experience in treating eight adult patients with intraglandular Botox injection and recommend the appropriate dosing and technique for the safe management of sialorrhoea.
Organ donation and legislation

John McCall

New Zealanders’ access to life-saving transplant operations is limited by availability of donor organs. The donor rate in New Zealand, about 10 per million per annum, is similar to Australia but less than the United Kingdom (14), USA (20), and European countries such as Spain (>30).

The idea that organ donation may be prevented from occurring because family members object, even if the deceased person may not have objected, is viewed by some as a problem that needs fixing. A Private Member’s Bill introduced to Parliament recently by National Party List MP Jackie Blue (Human Tissue Amendment Bill) proposes the establishment of an organ donor registry that is intended to enable people to register a legally binding wish that could not be overridden by family. The Bill is obviously well intentioned, and to many people it seems like common sense, but would it have the desired outcome?

A major weakness of all donor registries is that they struggle to enlist more than a fraction of the population, despite expensive public campaigns. For example, in the UK and Australia, which have medical systems similar to our own, organ donor registries have failed to enlist more than about 20% of the population, and there has been no increase in donor rates following their inception.

In the USA, 35 states have donor registers in one form or another but most of these have only been initiated in the last 5 years. There is, as yet, no evidence to determine whether these have had an impact on donation rates. Donor registries appeal to policy-makers because they show that ‘something is being done’, but there is little evidence that registries achieve their primary objective. On the down side, they are costly to administer and divert resources away from other initiatives that could be more successful.

So, would legislating to ensure that donor wishes are respected improve organ donation rates? Intensive care specialists and donor co-coordinators, who are the ones directly involved in obtaining consent for organ donation from grieving families, say it is rare for the known wishes of a donor to be overridden by family members. This assertion is backed up by several overseas studies. In those three studies, families provided new information showing that the deceased did not want to donate, or overrode the deceased person’s decision, in only 3.5%, less than 1%, and between 2 and 4% of cases respectively. Translating this to the New Zealand context, making donor wishes legally-binding would only result in between 0 and 1 additional donors per annum.

In fact, organ procurement without next of kin agreement does not occur anywhere in the world, except for countries that permit the use of organs from executed prisoners. In New Zealand, organ donation is a gift, not an obligation. Spain has the highest organ donation rate of any country and even there organs are not taken without consent from family. Indeed, I know of no intensive care specialist or transplant
surgeon in New Zealand who would proceed with organ donation if the family expressed clear objections, irrespective of legislation. The reasons for this are obvious to those regularly working in situations of tragedy and loss, but are less clear to those who see only the need for more organs. Furthermore, public attitudes to organ donation are highly sensitive to negative publicity, and overriding the family at a time of intense grief would risk generating an outcry that could harden public attitudes and prejudice many future organ-donation decisions.

Currently, New Zealanders can indicate their wish to donate on their driver licence. Only 42% of driver licence holders are listed as donors whereas 55% of families consent to organ donation when asked. Several studies have shown that individuals and families that are poorly informed are more likely to withhold consent for organ donation. Not being adequately informed when applying for a driver licence may explain the discrepancy between the driver licence ‘consent’ rate (42%) and the actual consent rate (55%). It follows that mandating poorly informed choices would lead to a fall in donor numbers.

Although New Zealand has a comparatively low organ donor rate, this is not due to unwillingness to donate. The consent rate in New Zealand is 55%. In Spain it is 59% on initial request (increased by 25% after making up to 5 further requests!), in the UK 55%, and the USA 58%.

What then can be done to address the shortage of organs for transplantation?

Organ donation takes place in intensive care units so this is where we first need to look. An audit of deaths in New Zealand ICUs, carried out in 1999–2000, found that organ donation was pursued in most but not all cases where it could have been. New Zealand has relatively few intensive care beds (70 per million) compared to Australia (88), Spain (150), USA (310), and France (320). Consequently, our facilities, and those working in them, are under greater pressure. ICUs need adequate reimbursement to cover the cost to supporting organ donors, expertise in the physiological maintenance of organ function after brain death and, perhaps more than anything else, support and education for the sensitive task of discussing the option of organ donation with grieving relatives.

Proscriptive legislation will provide none of these things, and risks alienating the people we rely on to bring organ donation to fruition. There has been recent progress with the creation of Organ Donation New Zealand, including the appointment of a Medical Director. However, there is still more to do.

Finally, what are transplant services themselves doing to alleviate the pressure on their waiting lists?

Laparoscopic donor nephrectomy was introduced in New Zealand in 2000, and more than one-third of kidney transplants are now performed using live donors. A quarter of New Zealand paediatric liver transplants have also been performed using live donors, and most other paediatric grafts come from ‘splitting’ an adult deceased donor organ for two recipients. The New Zealand Liver Transplant Unit is the only centre in Australasia that currently offers adult-to-adult live donor liver transplantation. Whilst Māori may be under-represented in deceased donor numbers, in our experience, they
are more likely than non-Māori to volunteer to be live donors (definitely not a soft option).

Transplant units are extending criteria in terms of deceased donor organs by accepting older donors with more comorbidities. A protocol is also being developed in New Zealand for donation after cardiac death. This has the potential to increase kidney donor numbers by about 10% and provide some additional livers and lungs for transplantation.

The challenge for transplant units is to improve access to live-saving treatments without compromising either the safety of live donors or the outcome for transplant recipients. The challenge for policy-makers is to provide a milieu where opportunities can be maximised. The proposed new legislation is not the way to achieve this.

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The Law Reform (Epidemic Preparedness) Bill—a proper response to the pandemic threat?

Brendon Gray, Cheryl Brunton, Pauline Barnett

Abstract

The ethical frameworks for public health practice are not as clearly articulated as those used in clinical medicine. This poses problems when Medical Officers of Health are required to exercise coercive powers over individuals in the context of communicable disease control. Proposed legislation exacerbates this problem. The Law Reform (Epidemic Preparedness) Bill, recently introduced into Parliament, extends coercive powers but does not provide legal protection for individuals subject to such powers.

A framework exists to critique the legitimacy of coercive public health powers and includes the principles of necessity, effectiveness, proportionality, and fairness. The fairness principle is under threat in the draft Bill, which fails to provide “due process” procedures to protect the rights of individuals.

Experience in managing the SARS epidemic suggests that coercive powers alone will not enhance the response to public health crises. Respect for individual rights, a positive relationship between government and the community, and the scope for review of decisions are more consistent with modern public health practice and are likely to lead to improved outcomes. In our view, the Bill should be amended to include legal protection for individuals before it is passed into law.

In clinical medicine, doctors have the support of a well-developed and articulated set of ethical principles to inform their decision-making, complemented by legislation that protects patients’ rights and ensures standards of practice. For doctors working in public health, the ethical framework for their practice is less clear, and current public health legislation does not provide essential protection for people in the community who may be subject to coercive public health action.

This situation is likely to become worse unless important amendments are made to the Law Reform (Epidemic Preparedness) Bill, recently introduced into Parliament. The purpose of the Bill is to provide Government with powers to ensure a “proper response” if the threat to human health materialises from the now global outbreak of avian influenza. The Bill also aims to address gaps in statutory public health powers under the Health Act 1956. Some of these gaps relate to coercive administrative public health powers that authorise the isolation, detention, examination, and treatment of communicable disease sufferers.

Protecting public health through the use of coercive public health legislation of the type envisaged by the draft Bill dictates that competing individual interests (such as autonomy, privacy, liberty, and freedom of association) must be curtailed in certain circumstances. Balancing the competing claims of human rights and the protection of the public poses an ethical dilemma for public health practitioners.
This is a dilemma for which there is no easy resolution and analysis using a traditional bioethical framework is not always helpful, as others have found.\(^1\) Bioethics has articulated clear principles that include beneficence, non-maleficence, autonomy, and justice in relation to **individuals** receiving treatment and care. For public health, the focus of intervention is based on outcomes for **populations**. Furthermore, according to Childress et al, ‘the health of the public is the primary end that is sought and the primary outcome for measuring success.’\(^2\)

Public health outcomes may be justified on the basis of communitarian principles or utilitarian values where the “greatest happiness of the greatest number” is sought.\(^3\) Using these philosophical paradigms is necessary because a bioethical framework, in which patient autonomy is given ‘pre-eminent moral status,’ is ‘arguably a poor fit for public health practitioners seeking ethics guidance for their community-oriented work.’\(^4\)

The absence of an appropriate public health ethical framework has been highlighted by the avian influenza threat. However, groups have been working to fill this gap. In 2005, the University of Toronto Joint Centre for Bioethics\(^5\) published an ethical framework for pandemic planning and we are aware that the New Zealand National Ethics Advisory Committee is currently working on a similar project.

There is, of course, a difference between law and ethics. The law is a body of rules and principles, governing the affairs of people, which is enforced by a political authority. Ethics, on the other hand, is a philosophical understanding of moral values and rules. While ethics may influence the content and elaboration of a legal rule, ethical principles are not in themselves legally binding.

Developing an ethical framework that can be used to critique the legitimacy of public health action (based on the scope of existing laws) is both important and necessary. However, in discussions about the state of influenza pandemic preparedness, debate about the character of the laws themselves has been notably absent. The introduction of the Law Reform (Epidemic Preparedness) Bill to Parliament is an opportunity to address this omission.

**The Law Reform (Epidemic Preparedness) Bill**

New Zealand’s public health laws are currently contained in disparate and antiquated legislation. This legislation includes the Tuberculosis Act 1948, the Health Act 1956, the Health (Quarantine) Regulations 1983, and other subordinate secondary legislation. Since the early 1990s, there have been various attempts to amend and consolidate these laws. The most recent proposals for reform were contained in a Ministry of Health discussion paper, published in 2002, which was expected to culminate in a new Public Health Act. However, this has not yet happened.

In the absence of a new Public Health Act, the Law Reform (Epidemic Preparedness) Bill offers wider powers for public health officials (backed up by new police enforcement powers) to meet threats from infectious diseases. In addition, commendably, the Bill also amends a range of other laws that deal with matters such as death certification, notification of death to the coroner, immigration, taxation, welfare provision, parole hearings, and the sentencing of prisoners (which could be adversely affected by an influenza pandemic or any similar disease).
The Bill will insert a number of new provisions into a wide range of legislation. These provisions will become operative if the Prime Minister issues an “epidemic notice”. The “trigger” for issuing such a notice is a requirement that the Prime Minister is satisfied that the effects of an outbreak of a stated infectious disease are ‘likely to disrupt essential governmental and business activity in New Zealand (or stated parts of New Zealand) significantly’ (Clause 5). The disease need not yet have reached New Zealand for a notice to be issued.

The current definition of a quarantinable disease (a term which at present applies to the three diseases covered by the International Health Regulations 1969—cholera, plague, and yellow fever) will be extended by adding avian influenza to the list in a new Part 3 to the 1st Schedule of the Health Act 1956. Quarantinable disease will also include any disease stated in an epidemic notice that is in force (Clause 17).

Emergency powers available to a Medical Officer of Health acting under Sections 70 and 71 of the Health Act, applicable to persons residing in New Zealand, will be expanded. The Bill leaves essentially untouched powers of compulsory medical examination [Section 70 (1) (e) of the Health Act] and detention powers through the use of isolation and quarantine [Section 70 (1) (f)].

Police are to be granted new powers to do anything reasonably necessary (including the use of force) to assist Medical Officers of Health acting under Sections 70 and 71 of the Health Act. Police will also have immunity from personal liability (providing they act in good faith), and people failing to comply with orders made under Sections 70 or 71 of the Health Act will face imprisonment for up to 6 months, a fine of up to $4,000, or both (Clause 20).

The Bill does not amend the existing power under Section 77 of the Health Act, enabling a Medical Officer of Health to enter any private residence and examine anyone whom he or she suspects to be suffering from a notifiable infectious disease for the purpose of the Act. While suspected criminals have some protection against entry without warrant in New Zealand, a person who may be suffering from a notifiable infectious disease does not.

The Bill also clarifies and extends the public health powers available to officials at the border. A person is liable to quarantine upon arrival in New Zealand if a Medical Officer of Health believes or suspects (on reasonable grounds) that the person is suffering from, or has been exposed within the previous 14 days to, a quarantinable disease (regardless of whether or not the disease was so classified at the time of suspected exposure). Persons liable to quarantine are required to give information, submit themselves for examination, and are liable to detention for up to 28 days (Clause 23).

The detention of people suspected of suffering from a communicable disease involves questions of both law and ethics. As Bernheim points out, officials must first ask if there is legal authority to act, and then decide how to do so ethically in the particular situation they face. However, the idea that such ethical analysis can provide additional justification and legitimacy for public health authority presupposes that the law upon which such action is based is itself legitimate.

Of course, under New Zealand’s unwritten constitution, Parliament can pass any law it likes, including laws that contravene international human rights conventions, and
not even the New Zealand Bill of Rights Act 1990 affects the supremacy of Parliament in this regard. However, today’s society is very different from that of 1956, when the Health Act was passed, and principles of human rights protection have developed significantly. Therefore, legal legitimacy must also incorporate social expectations about protecting such rights. In our view, this is the crucial “missing debate” and we question whether New Zealand’s communicable disease laws (whether current or proposed) reflect a modern understanding of human rights protection.

**Justifying coercive legislation**

How are we to critique the legitimacy of our laws by the standards of these expectations? Professor Lawrence Gostin offers a framework to assess the legitimacy of coercive public health powers based on four principles that are consistent with human rights norms.

**Necessity**—Necessity implies that the risk to public health must be significant before public health powers are invoked. While there may be sceptics who view the emergence of avian influenza A H5/N1 as “Y2K with feathers,” balanced opinion acknowledges that the current outbreak is unprecedented and that there is significant potential for a devastating impact upon human health.

**Effectiveness**—Gostin’s second principle demands that the proposed intervention envisaged by the legislation is effective. There is little robust international evidence to demonstrate the effectiveness of coercive public health interventions for pandemic influenza and “proving” effectiveness is problematic. However, it would be reasonable to invoke the precautionary principle and argue that having coercive powers is a sensible and prudent measure in the context of the risk posed by avian influenza A H5/N1. We support such a view.

**Proportionality**—Proportionality means that the least restrictive alternative should be employed to protect the public health and that the intervention should not exceed what is necessary to address the level of risk posed to the public. For example, there has been recent debate in Scotland about compulsory HIV testing of suspects following criminal incidents where there has been an assessed risk of infection to police officers. The National AIDS Trust, in response, cited evidence showing that such orders would provide no treatment benefit and would have only a marginal impact on society. Furthermore, they argued that mandatory testing would be a disproportionate measure, and that less coercive approaches would be equally as effective. Proportionality however, is not fixed. As Harrington points out, also in relation to HIV/AIDS, the emergence of more effective interventions may in fact encourage a more coercive, but yet more proportionate, response. In the case of pandemic influenza it is possible to argue that depriving individuals of their liberty is a proportionate response if the level of harm to the public is on the scale of the 1918 pandemic.

**Fairness**—Fairness is said to be a feature of good government that comprises three features: equity, natural justice, and transparency. Equity in this context means that the benefits and burdens of public health law are applied equally across society. While there is nothing inherently unequal in either existing or proposed public health legislation in New Zealand, there are indications that such laws were applied
unequally in the past. For example, in past smallpox and influenza epidemics, travel restrictions were imposed solely on Māori.\textsuperscript{13,14}

Respect for the rules of natural justice suggests that coercive powers be tempered by procedures that allow for the review of an individual’s rights in a timely fashion by an independent and impartial judicial body. New Zealand public health laws fall well short of this ideal, and the draft Bill does little to address this deficiency. In terms of transparency, administrative and police public health powers in the Bill are strengthened without providing adequate checks or balances on their use.

Detention in the context of infectious disease does not involve a question of establishing guilt. However, it has been argued that administrative orders for detention in this context are punitive in character (because people are deprived of their liberty), and such orders should only be made by the judiciary.\textsuperscript{15}

Nevertheless, a cumbersome judicial process should not impede effective and urgent public health action where such action is needed. It is important that public health officials have emergency powers available that can be used without application to the courts. However, an independent judicial body should review the use of such powers within a timely period; review by a magistrate within 24 hours has been recommended in Canada.\textsuperscript{16}

The absence of any review and appeal procedure in the new Bill is remarkable, given that it extends coercive powers that are now at least 50 years old and society has changed greatly in the interim. Furthermore, a lack of provision for “due process” in public health legislation has been a “gap” in our laws that has been recognised previously by the Ministry of Health. The 2002 Ministry discussion paper observed that current public health legislation fails to reflect a modern understanding of rights protection for individuals enshrined by more recent legislation such as the New Zealand Bill of Rights Act 1990, the Human Rights Act 1993 or the Privacy Act 1993.\textsuperscript{17}

This is not just a theoretical issue. Section 79 of the Health Act 1956, a provision devoid of any inbuilt right to appeal or review, has been used on several occasions to detain people suffering from infectious disease. The most notable cases were of two HIV-positive men detained under this Section in 1999. One of these men is still detained and no court has ever considered the appropriateness of his detention. Balancing the rights of this man with the need to protect the public is a heavy responsibility. Some public health officials have indicated to us that this is a responsibility they would prefer to share with the judiciary.

The 2002 discussion paper recognised that providing effective regulation for communicable disease control may create tensions with human rights values; it proposed finding a balance between protecting the public health on the one hand and the rights of people suffering from an infectious disease on the other. Despite this recognition by the Ministry of Health, the new Law Reform (Epidemic Preparedness) Bill does little to address the need for this balance.

**Public health or public order—does this matter?**

The World Health Organization recognises the importance of legal preparedness by calling on national governments to ensure that laws or regulations necessary to deal
with a public health emergency are in place in advance of any pandemic. The international public health community largely supports this call.

In 2005, the SARS Commission in Canada recommended strengthening relevant laws. In the same year, the Centers for Disease Control and Prevention in the United States proposed regulations that would empower the Federal Government to isolate and quarantine an expanded category of unwell arriving passengers to the United States.

However, unlike Canada or the United States, the New Zealand Government plans to strengthen coercive administrative public health powers while providing only minimal checks or balances in the legislation itself to prevent the abuse of administrative power. We are not aware of any similar initiative in jurisdictions whose legal traditions are similar to our own. Does this matter?

In our view, what is exemplified in the Bill is an over reliance on a “public order” approach to communicable disease control. This approach assumes that a recalcitrant population is likely and that a coercive response (where individual rights are subjugated) will best protect the public health. In the United States, the Model State Emergency Health Powers Act, a post-9/11 response to bioterrorism threats, takes a similar approach, and some commentators have suggested this unnecessarily erodes civil liberties.

During SARS, the World Health Organization leadership endorsed strong public health measures to control the epidemic. The way different countries managed these measures provides evidence against the effectiveness of a public order approach. During a 3-day period in China, almost a quarter of a million people are estimated to have fled Beijing following rumours of quarantine and martial law, and there were reports of rioting in response to government measures. In Taiwan, officials now believe that aggressive use of quarantine contributed to public panic and was counterproductive.

By contrast, in Toronto, Canada, almost everyone who was asked to submit to quarantine did so, with authorities having to seek a written order in only 27 cases. Both Federal and Provincial Governments provided special insurance to protect the financial wellbeing of those quarantined, thus increasing the acceptance of public health measures taken. Civic duty and confidence in being treated fairly, and not the fear of legal consequences, were the main motivating factors for observing quarantine in Canada.

These are important lessons for New Zealand, where success at mitigating the potentially devastating effects of an influenza pandemic will depend on public confidence and trust in our public health officials. Over-reliance on unfair coercive measures will only undermine that trust and ultimately have adverse effects on the public health.

A positive public health approach that respects individual rights, based on trust between the community and government, will be more effective in controlling epidemics than coercive powers alone. While such powers must be available to public health officials as a precaution, the laws upon which such powers are based must be “fair” and they must be applied within an appropriate ethical framework.
With respect to Medical Officer of Health powers, the Law Reform (Epidemic Preparedness) Bill (as written) contains few inbuilt mechanisms to protect the rights of individuals; and an appropriate pandemic response relies upon officials adopting strict ethical public health practice. However, exactly what would constitute this practice is not yet well articulated.

Not only does this Bill provide little support for Medical Officers of Health who may be required to deprive people of their liberty, it also leaves wide open the potential for the abuse of power, however well-intended. As such, this Bill does not ensure a proper response if the pandemic threat materialises and does not protect public health.

We consider that legal protection for individuals must be included in the Bill before it is enacted into law.

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Ethnic disparities in nutrition-related mortality in New Zealand: 1997–2011

Carlene Lawes, Niki Stefanogiannis, Martin Tobias, Natalie Paki Paki, Cliona Ni Mhurchu, Maria Turley, Stephen Vander Hoorn, Anthony Rodgers

Abstract

Aims To estimate the mortality due to non-optimal levels of systolic blood pressure, total blood cholesterol, body mass index (BMI), and vegetable and fruit intake amongst Māori and non-Māori in New Zealand in 1997. In addition, to estimate the ethnic-specific burden of disease that could potentially be avoided in 2011 if exposure to these risk factors were reduced.

Methods The study uses comparative risk assessment methodology, a systematic approach to estimating both attributable and avoidable burden of disease developed by the World Health Organization.

Results About 47% of deaths among Māori and 39% of deaths among non-Māori were estimated to be due to the selected risk factors. Age-standardised mortality rates for attributable ischaemic heart disease burden were consistently higher in Māori for individual risk factors. Age standardised mortality attributable to BMI was relatively higher for Māori, especially diabetes mortality.

Estimates of avoidable mortality suggest that the health gains for Māori would be relatively greater than for non-Māori across all risk factors, but particularly with improvements in BMI.

Conclusions Non-optimal levels of systolic blood pressure, cholesterol, BMI, and to a lesser extent vegetable and fruit intake are major modifiable causes of death in New Zealand. Small changes in risk factor levels could have a major impact on population health within a decade, with relatively greater health gains for Māori.

The impact of nutrition on health is considerable. Numerous studies have demonstrated that nutrition-related risk factors such as elevated blood pressure, cholesterol, and bodyweight; and inadequate vegetable and fruit intake; are established risk factors for cardiovascular disease (CVD), such as ischaemic heart disease (IHD) and stroke.1

Among developed countries worldwide (including New Zealand), CVD remains the leading cause of premature mortality and disability.2,3 Elevated body weight and inadequate vegetable and fruit intake are also important aetiological factors for other diseases such as diabetes and certain cancers.3 In 1997, CVD, diabetes mellitus, and cancer accounted for 64% of all deaths in adults over 25 years of age in New Zealand.4

In New Zealand, significant ethnic disparities exist for these nutrition-related risk factors. Non-Māori have (on average) lower levels of blood pressure, cholesterol, and obesity than Māori.5
Current mortality patterns also differ between the groups. Non-Māori have a lower overall rate of death than Māori, and lower death rates from diabetes mellitus and IHD. CVD deaths also occur at relatively older ages in non-Māori where over 85% of IHD deaths and over 90% of stroke deaths occurred in those aged ≥65 years, compared to only about half of IHD deaths and about two-thirds of stroke deaths in Māori. There is evidence of declining rates of CVD between 1980–1999 in all ethnic groups, however this decline has been more rapid in non-Māori.

The World Health Organization (WHO) Global Burden of Disease (GBD) study estimated that elevated blood pressure, cholesterol, and bodyweight and inadequate fruit and vegetable intake—together with tobacco and alcohol use, and inadequate physical activity—accounted for about half of all non-communicable disease mortality in developed world regions.

The current study employed a modification of the ‘comparative risk assessment’ methodology developed by the WHO for the GBD to assess the impact of these aetiological factors on New Zealanders. It estimates the burden of disease attributable to nutrition-related risk factors in 1997, and the number of deaths that could potentially be avoided in 2011 if small, favourable changes in current risk-factor distributions were to occur.

The overall results for the study have been reported elsewhere. The aim of these analyses is to describe the mortality attributable to these nutrition-related causes with particular reference to Māori and non-Māori. Exploration of any differences found can contribute to planning and implementing appropriate policies to address ethnic inequalities in health related to the selected nutrition-related risk factors.

Methods

Study design—The analyses described in this paper were conducted as part of a larger study examining the impact of systolic blood pressure (SBP), total blood cholesterol (TBC), body mass index (BMI), and vegetable and fruit intake on the burden of disease in New Zealand. Full details of comparative risk assessment (CRA) methodology developed by the WHO and modified for this project have been described elsewhere, but a brief summary is provided below.

CRA is a systematic approach to estimating the current burden of disease attributable to various risk factors, as well as the future burden of disease that could be avoided if exposure to these risk factors were reduced. The methodology takes into account the entire population risk factor distribution by focusing on continuous (rather than categorical) risk factor-disease relationships. It uses standardised methods to obtain best estimates of risk factor distributions, risk factor disease relationships, and disease burden.

Current risk factor distributions and the theoretical minimum distribution—The nutrition-related risk factors selected were TBC (as a marker for saturated fat intake); SBP (as a marker for several nutritional factors, primarily sodium intake); inadequate vegetable and fruit intake; and BMI (as a marker of energy balance). Data on the current population distribution of all the selected risk factors by age, sex, and ethnicity (Māori/non-Māori) were extracted from the 1997 National Nutrition Survey. The most recent available national survey. To obtain more reliable estimates, the National Nutrition Survey included a higher proportion of Māori than are found in the general population (704 Māori and 3932 non-Māori).

There were concerns over the reliability of blood pressure measurements due to calibration difficulties with equipment used in the survey. Therefore measurements were adjusted downwards based on other survey data. Data from the survey demonstrated that overall mean SBP levels were lower in non-Māori; and Māori females and non-Māori males had the lowest TBC levels (Table 1). Vegetable and fruit intake was higher overall in males and in non-Māori. The greatest ethnic differences were apparent in BMI where non-Māori levels were consistently lower across all age groups.
<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Disease outcomes*</th>
<th>Theoretical minimum distribution (mean)</th>
<th>Average change in risk factor after intervention scenario (range)#</th>
<th>Mean risk factor levels¶</th>
<th>Māori</th>
<th>Non-Māori</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>Ischaemic heart disease, stroke</td>
<td>115 mmHg</td>
<td>0.5 (0.1–3.0) mmHg decrease</td>
<td>Males: 126</td>
<td>122</td>
<td>118</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Females: 122</td>
<td>118</td>
<td>118</td>
</tr>
<tr>
<td>Total blood cholesterol (mmol/L)</td>
<td>Ischaemic heart disease, stroke</td>
<td>3.8 mmol/L</td>
<td>0.1 (0.08–0.2) mmol/L decrease</td>
<td>Males: 6.0</td>
<td>5.6</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Females: 5.4</td>
<td>5.7</td>
<td></td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>Ischaemic heart disease, stroke, diabetes, cancer§</td>
<td>21 kg/m²</td>
<td>1.0 (0.8–1.2) kg/m² increase†</td>
<td>Males: 28.9</td>
<td>25.7</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Females: 29.0</td>
<td>25.6</td>
<td></td>
</tr>
<tr>
<td>Vegetable and fruit intake (g/day)</td>
<td>Ischaemic heart disease, stroke, diabetes, cancer¥</td>
<td>600 g/day</td>
<td>40 g/day increase</td>
<td>Males: 403</td>
<td>422</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Females: 389</td>
<td>407</td>
<td></td>
</tr>
</tbody>
</table>

*ICD-9 codes: Ischaemic heart disease – 410–414; Total stroke – 431-438; Cancers: Oesophageal – 150, Stomach – 151, Colorectal – 153-154, Lung – 162, Breast – 174; #The change is over and above the business as usual (BAU) scenario and the range represents the range over different age and sex subgroups; † Unlike other risk factors, the intervention scenario for body mass index did not include an improvement in the current situation since there is no evidence that the obesity epidemic has peaked. The shift in the distribution for the intervention scenario is therefore away from the theoretical minimum, but it represents a smaller increase in body mass index than was projected under the BAU scenario; ‡ Ischaemic stroke only; § Post-menopausal breast and colorectal cancers; ¥ Oesophageal, stomach, colorectal and lung cancers; ¶ Mean SBP, TBC and BMI levels are age standardised to the WHO World population.

To calculate attributable burden, the current risk factor distribution was compared with the one that confers minimal risk. This alternative ‘ideal’ risk factor distribution or theoretical minimum distribution (TMD) for each risk factor was defined by the expert working groups set up by the WHO for the GBD (Table 1). The same TMD was applied to all age, sex, and ethnic groups, and full details on how TMDs were defined can be found elsewhere.2,14–19

**Distributional transitions**—To calculate avoidable burden ‘distributional transitions’ or relative shifts away from the current distribution of the risk factor towards its theoretical minimum distribution were modelled. Avoidable burden was estimated from the difference between two distributional transitions: a ‘business as usual’ (BAU) scenario representing continuation of historical trends in risk factor levels, and an ‘intervention’ scenario which was intended to reflect feasible changes in the population exposure distribution to each risk factor over the next decade. Both scenarios are projections only, modelled on historical and current trends in risk factors.

The ‘business as usual’ (BAU) scenario indicated that overall population mean SBP and TBC levels are expected to decline and vegetable and fruit intake and BMI levels are expected to continue to increase by 2011. Under a more optimistic intervention scenario, it was estimated that there would be greater decreases in population mean SBP and TBC and a greater increase in vegetable and fruit intake. The intervention scenario for BMI involved a slowing of the trend for increasing BMI, as there is no evidence that current trends will reverse over the next decade. The distributional transitions used for each risk factor are summarised in Table 1.

**Disease outcomes and disease burden**—The mortality outcomes assessed were based on strong evidence of a causal relationship and sufficient data to quantify the risk-factor disease relationship (Table 1). The number of deaths in New Zealand in 1997 for each selected disease was extracted from the New Zealand Health Information Service mortality database. Projections by the Ministry of Health were used to estimate the predicted number of deaths for each disease in 2011.5,10 Ethnic-specific models of mortality were not possible due to the changing concepts and classifications of ethnicity over time. Therefore an overall projection was made, and then split into Māori and non-Māori estimates assuming that the ratio of ethnic-specific mortality rates remained constant.5 Mortality counts were also
translated into ‘years of life lost’ (YLL) counts, and discounted to the present using a 3% per annum discount rate.

Risk factor–disease relationships—To estimate the nature and strength of the association between risk factors and disease endpoints, estimates of relative risk were required. Estimates of relative risk were based on those used by the expert working groups set up by the WHO.\textsuperscript{2,14-16} Wherever possible, these estimates were based on overviews of cohort studies and randomised controlled trials rather than individual studies. Age-specific relative risk estimates were used, and where data were lacking for older age groups, a degree of age attenuation was modelled. The same relative risk estimates were used for both ethnic groups, and gender-specific estimates were only necessary for the BMI-diabetes association.

Calculating attributable and avoidable burden—Attributable and avoidable burden was calculated using potential impact fractions (PIFs) which are extensions of the widely used population attributable risk measure. Attributable burden was estimated by applying the relevant PIF to the estimate of current burden. Avoidable mortality was calculated by extending the methodology of the PIF formula to include estimates of risk reversibility in the calculations, and by applying the fractions calculated to the projected 2011 disease burden under both the BAU and intervention scenarios. Further details of the methodology used to estimate attributable and avoidable burden, with examples, can be found elsewhere.\textsuperscript{9,26}

An estimate of the joint effect of combining the four risk factors was obtained using a formula that adjusted for overlap between the risk factors and considered sources of uncertainty relating to the risk factor distribution, the risk factor–disease relationship, and the projected total disease burden.\textsuperscript{9,10}

Results

Here we primarily report results of the Māori and non-Māori analyses. (The overall results for the study have been reported elsewhere.\textsuperscript{9-13})

Attributable mortality due to nutrition-related risk factors—The proportion of disease specific deaths attributable to the individual risk factors provides an indication of the impact that each risk factor had on the selected causes of death in Māori and non-Māori.

These proportions (attributable fractions) were usually highest in those aged 55–74 years where about 50–65% of both stroke and IHD deaths were attributable to higher than optimal SBP, and the proportions tended to be higher in Māori. Attributable fractions in this age group were of similar magnitude for higher than optimal TBC but lower for inadequate vegetable and fruit intake (20–30%), with proportions similar for Māori and non-Māori. In those aged 55–74 years, 30–45% of both IHD and ischaemic stroke deaths were attributable to higher than optimal BMI in non-Māori compared to 40–60% in Māori.

For BMI, attributable fractions were higher in those aged <55 years (50–55% and 70–80% for non-Māori and Māori respectively) and also higher overall for diabetic deaths (75–99% and 90–100% respectively). Due to multi-causality, attributable fractions for different risk factors cannot be added together for a given cause of death—as there is overlap in causality between these risk factors.

Table 2 presents the total number of deaths and years of life lost (YLL) attributable to the selected risk factors by sex and ethnicity in 1997. The ratio of all attributable deaths and YLL for males:females ranged from 50:50 to 60:40 for most risk factors in both ethnic groups. Overall, 11–14% of all deaths and 9–14% of all YLL were attributable to higher than optimal SBP; 11–18% of deaths and 9–16% of YLL were attributable to higher than optimal TBC; and 5–7% of deaths and YLL were attributable to inadequate vegetable and fruit intake. Among non-Māori, 10% of
deaths and 11% of YLL were attributable to higher than optimal BMI, while in Māori it was 23–27% of deaths and 21–25% of YLL.

Table 2: Attributable mortality burden in 1997 and avoidable mortality in 2011 for risk factors and all selected diseases (by ethnicity and sex)

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<thead>
<tr>
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<tbody>
<tr>
<td></td>
<td>Deaths (count)</td>
<td>Years of life lost (count)</td>
</tr>
<tr>
<td>------------------------------</td>
<td>----------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td></td>
<td>Māori</td>
<td>Non-Māori</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>188</td>
<td>1,661</td>
</tr>
<tr>
<td>Females</td>
<td>125</td>
<td>1,724</td>
</tr>
<tr>
<td>Total blood cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>224</td>
<td>2,189</td>
</tr>
<tr>
<td>Females</td>
<td>130</td>
<td>2,178</td>
</tr>
<tr>
<td>Body mass index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>332</td>
<td>1,328</td>
</tr>
<tr>
<td>Females</td>
<td>311</td>
<td>1,183</td>
</tr>
<tr>
<td>Vegetable and fruit intake</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>101</td>
<td>809</td>
</tr>
<tr>
<td>Females</td>
<td>63</td>
<td>585</td>
</tr>
</tbody>
</table>

Since deaths can be caused by the joint action of more than one risk factor, the number of deaths or years of life lost cannot be added across risk factors.

The above percentages give an overall picture of the total number of deaths and YLL attributable to individual risk factors in 1997. However they only give a limited picture of the relative burden of disease experienced by Māori and non-Māori due to the different age structures of the two groups.

Figure 1 controls for differences in age structure by presenting the age-standardised mortality rates of the attributable mortality burden for both Māori and non-Māori. The age-standardised mortality rates for IHD attributable to each of the four risk factors were all higher for Māori compared with non-Māori. Overall, the rates for IHD were about twice as high for higher than optimal SBP; about 50% higher for higher than optimal TBC; almost three times as high for higher than optimal BMI; and about twice as high for inadequate vegetable and fruit intake in Māori.

Age-standardised mortality rates for stroke deaths attributable to higher than optimal SBP and TBC, and inadequate vegetable and fruit intake were similar for Māori and non-Māori. In contrast, age-standardised mortality rates for stroke attributable to high BMI were about twice as high in Māori, and mortality rates for diabetes attributable to high BMI were about 10 times higher in Māori.

Age-standardised mortality rates for cancer (attributable to high BMI and inadequate vegetable and fruit intake) varied by cancer outcome (Data not shown). Rates were similar for colorectal, oesophageal, and stomach cancer, but there were higher rates in Māori for lung cancer and breast cancer.
When the overlap between the nutrition-related risk factors was taken into account, it was estimated that (overall) approximately 11,000 deaths (40% of all deaths in 1997) were due to the selected risk factors. In Māori, this equates to 47% of deaths (1,203) and 42% of YLL (18,477)—and in non-Māori, 39% of deaths (9,852) and 39% of YLL (95,489).

Avoidable mortality due to nutrition-related risk factors—Estimates of avoidable mortality in 2011 were calculated under intervention scenarios of feasible changes in risk factor distribution.
Results indicated that (overall) approximately 250 deaths in non-Māori, and 35 deaths in Māori, could potentially be avoided in 2011 with small changes in SBP levels (Table 2). Potentially avoidable deaths with improvements in TBC and vegetable and fruit intake were similar to SBP estimates. The number of deaths potentially avoided with the modelled change in BMI were similar in Māori (approximately 165 deaths) and non-Māori (approximately 220 deaths).

Figure 2. Avoidable mortality by risk factor, disease, and ethnicity (rates per 100,000; 2011)
Figure 2 presents avoidable burden in 2011 in terms of age-standardised mortality rates; it demonstrates that the potential health gains are greater for males than females. This difference is most striking for IHD where rates in males are about twice as high as those in females for both Māori and non-Māori. The health gains were also greater for Māori than non-Māori with age-standardised mortality rates for IHD deaths being about 2–3 times higher in Māori for each risk factor. Rates for potentially avoidable stroke deaths were about 1.5–2 times higher for Māori. The greatest health gains overall are predicted to come from a slowing of current trends in BMI in Māori.

**Discussion**

This paper demonstrates that a substantial proportion of the mortality burden for Māori and non-Māori in New Zealand in 1997 was due to nutrition-related risk factors. Overall, approximately 10–15% of all deaths and all YLL were attributable to high SBP, 10–20% of all deaths and YLL were attributable to high TBC and 5–7% of all deaths and YLL were attributable to inadequate vegetable and fruit intake for both Māori and non-Māori. Among non-Māori, about 10% of all deaths (11% YLL) were attributable to high BMI, while in Māori it was 23–27% of all deaths (21–25% YLL).

While the proportion of deaths due to individual risk factors (apart from BMI) was similar for Māori and non-Māori, age-standardised attributable mortality rates show that (in many cases) Māori carry a higher burden of disease due to the selected risk factors. For example, the IHD-attributable mortality rates were approximately twice as high for higher than optimal SBP and inadequate vegetable and fruit intake, and 50% higher for higher than optimal TBC for Māori than non-Māori.

Most striking were the age-standardised attributable mortality rates for higher than optimal BMI which were 10 times higher for diabetes, three times as high for IHD and twice as high for stroke for Māori. These ethnic differences are due to higher IHD mortality rates in Māori, but most importantly they are contributed to by differences in current risk factor levels, in particular BMI levels, between the groups.

When the overlap between the nutrition-related risk factors was taken into account, it was estimated that approximately 47% of all deaths (1203) in Māori in 1997, and 39% of all deaths (9,852) in non-Māori, were due to the selected risk factors.

The current analyses also estimated how many deaths could potentially be avoided in the future given realistic changes in risk factor distributions over the next decade. While more difficult to estimate accurately, avoidable mortality is of more relevance to policy than attributable burden as it gives an indication of areas for potential health gain.

Under the specified intervention scenarios, avoidable mortality, or potential health gains were greater for Māori than non-Māori with reasonably small population-wide improvements in risk factor levels. Such greater gains for Māori could contribute to narrowing relative health disparity between Māori and non-Māori. Age-standardised avoidable mortality rates for each risk factor were higher in Māori for IHD and for stroke. The greatest relative health gains overall were predicted to come from a slowing of current trends in BMI among Māori.

For both Māori and non-Māori potential health gains were relatively greater for males, with the difference being most marked for IHD where rates in males were about twice as high as those in females.
While this study provides the most accurate estimates of nutrition-related mortality among Māori and non-Māori in New Zealand to date, it has several limitations. The best measure of burden of disease is a summary of both fatal and non-fatal outcomes using an integrated measure of population health such as the disability-adjusted life year (DALY). However, in these analyses, burden of disease was restricted to fatal outcomes only, i.e. deaths and years of life lost (YLL), because of limitations in the data available to estimate disease incidence and lack of New Zealand-specific health state valuations.

Including non-fatal outcomes may have had an effect on the relative effect sizes of different nutritional risk factors when comparing ethnic groups as there is clear evidence that Māori have a younger age distribution in mortality rates compared to non-Māori. Developing disease at a younger age is likely to result in both a greater number of ‘years of life lost due to premature mortality’ and ‘years of life lived with disability or non-fatal conditions’ in Māori. There is also considerable non-fatal disease burden associated with diseases such as diabetes.

Other potential limitations relate to the input data utilised: the limitations surrounding the risk factor data used; setting the theoretical minimum distributions; projecting future trends in risk factors and disease outcomes; and calculating joint effects. These limitations are discussed elsewhere and apply to both ethnic groups.

Potential limitations for this ethnic comparison relate to the small sample size of Māori (704 respondents) in the National Nutrition Survey. The small sample size may influence the robustness of the risk factor data for Māori—particularly amongst the older age groups where the numbers of Māori were low.

In addition, the same BMI TMD mean (21 kg/m²) and the same relative risk estimates for both Māori and non-Māori were used. There are some studies suggesting that (for a given BMI) Māori and Pacific people have lower body fat than Europeans, suggesting a higher BMI threshold may be appropriate. However the evidence is not conclusive in relation to risk of disease, and ethnic specific cut-offs for BMI remain controversial.

For the risk factor-disease relationships studied, expert working groups for the WHO GBD study found very little variation between ethnic groups internationally. However, one study has suggested that there may be differences in disease risk for Māori and non-Māori. For example, for smoking-related mortality, a New Zealand study demonstrated that all-cause relative risks appeared to be significantly higher among non-Māori non-Pacific people than Māori. However, these relative risks were for all-cause mortality, while all the data collated for the WHO study, and utilised here, related to disease-specific relative risk.

In spite of the limitations discussed here, the current estimates were based on the most reliable data available for the New Zealand population, and the best risk factor-disease relationships available at the time.

Overall the current estimates of attributable mortality for Māori and non-Māori were of similar magnitude to those for ‘developed’ regions in the WHO GBD study for high cholesterol and inadequate vegetable and fruit intake. Our estimates of attributable burden were lower for high SBP than in the WHO study (11–14% vs 22%), possibly due to an overcorrection of mean blood pressure levels in the current
study. In contrast, attributable burden was higher for high BMI in Māori compared to the WHO study (>20% vs 11%), due to relatively high BMI levels in Māori.

In designing and implementing the most effective combination of interventions to address these risk factors, it is important to acknowledge that these exposures are shaped by a complex interaction of social, structural, and other factors, including historical influences and discrimination.

Interventions to improve nutrition, physical activity, and body weight should therefore include strategies directly addressing the relevant risk or protective behaviours as well as strategies aimed at underlying social inequalities in food security, income, employment, housing, and the built environment.

Single interventions are unlikely to have a major impact on population wide levels of nutrition-related risk factors, and inter-sectorial collaboration is necessary. This needs to involve various health, educational, industry, and community groups developing ways to make healthy food choices and increased physical activity accessible, affordable, safe, and easy.

One component is Healthy Eating – Healthy Action (HEHA) which is the New Zealand Ministry of Health’s strategic approach to improving nutrition, increasing physical activity and achieving healthy weight for all New Zealanders; Māori are a priority group targeted in the HEHA implementation plan. In addition, evaluation of interventions and policies is also important to ensure that health gains are occurring.

In conclusion, this study has quantified ethnic disparities in nutrition-related mortality in New Zealand. The analyses indicate that small changes in risk factor levels could have a major impact on population health within a decade, and there is potential for relatively greater health gains in Māori compared to non-Māori, particularly with strategies to decrease BMI.

To achieve maximal health gain, interventions that are appropriate for Māori are important to ensure that disparities between the two groups are reduced.

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

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**References**


Flies, fingers, fomites, and food. Campylobacteriosis in New Zealand—food-associated rather than food-borne

Warrick Nelson, Ben Harris

Abstract

Aims New Zealand has a very high rate of seasonal, sporadic campylobacteriosis compared to other OECD countries. Can the seasonality of New Zealand cases fit with an explanation of food-borne transmission (especially by chicken meat), and where does the fly-transmission hypothesis fit?

Methods Analysis of seasonal campylobacteriosis reports at the District Health Board level compared to regional ambient temperatures, and chicken consumption data. Literature review particularly of food-associated disease risks and transmission routes.

Results Campylobacteriosis rates in New Zealand show a national annual increase at a rate similar to chicken consumption. A drastic reduction in chicken consumption was associated with significantly reduced campylobacteriosis cases in two European countries, further strengthening the link between disease risk and chicken consumption. However, serotype analysis of the Campylobacter isolates is ambiguous regarding chicken meat itself as the major source of infection. The seasonal colonisation pattern in live chickens follows the seasonal increase in human cases. Flies are a plausible vector, associated with increasing overwintered fly foraging activity, rather than a summer increase in fly numbers.

Conclusion The typically sporadic rather than outbreak nature of campylobacteriosis, the disjoint between seasonal patterns of human and chicken infection, the seasonal pattern itself, and inconclusive serotype evidence indicates against chicken meat itself as the major source of infection. However, chicken consumption is a significant risk factor.

Sporadic contamination by flies of individual portions of food is plausible, but does not account for the clear chicken-consumption association.

Cows are the most likely source of high environmental Campylobacter contamination in New Zealand. It is proposed that flies are indeed the link between environmental sources and food. Increased fly foraging activity as temperatures rise into summer increase the opportunity for finger contamination by contact with fly faecal and regurgitated matter deposited on commonly touched surfaces.

Takeaway meals are a particular risk because of the almost universal lack of hand hygiene prior to eating, especially chicken meals because of the high moist food contact and licking of fingers during consumption. Thus flies, fomites, fingers, and food account for both the observed seasonal pattern and chicken consumption associations with campylobacteriosis.

New Zealand has a particularly high rate of campylobacteriosis compared to other OECD countries, with 12,000 to 14,000 notified cases per annum (or 300 cases per 100,000 population per annum).
Campylobacteriosis is the dominant cause of the 15,000 bacterial food-borne illnesses reported each year in New Zealand.\textsuperscript{1,2} Actual prevalence for these diseases is commonly estimated to be 10–20 times the reported rates. As well as gastroenteritis, \textit{Campylobacter} can also cause other illnesses including Guillain-Barré syndrome.

**Food-borne**

Campylobacteriosis is typically a sporadic illness (as distinct from outbreak clustering). It is reported throughout the year, with marked summer seasonal peaks (Figure 1).

**Figure 1. New Zealand-reported sporadic campylobacteriosis cases per month with least squares linear regression fitted (grey bars and dotted line). Chicken meat production in tonnes per annum with least squares linear regression shown (black bars and line)**

![Campylobacteriosis and Chicken Production Graph](image)

Campylobacter data is from Environmental and Scientific Research (www.esr.cri.nz), and chicken data is from Poultry Industry Association of New Zealand (www.pianz.co.nz)

The New Zealand trends, apart from being higher per capita, are similar to other temperate OECD countries, except the peak month in the Northern Hemisphere shifts to June or July. About 80% of campylobacteriosis cases are thought to be by food-borne transmission, but unusually with fewer than 1% of cases occurring as outbreaks.

Chicken consumption alone has been implicated as the source in about 40% of cases, and \textit{Campylobacter} colonisation of chickens themselves (both living and prepared in retail packs) is high while also showing a seasonal pattern of colonisation/contamination.\textsuperscript{2-9}

The association between chicken consumption and human campylobacteriosis cases appears conclusive. The association is even more specific to chicken eaten outside the home. Figure 1 suggests that New Zealand campylobacteriosis cases are increasing at about the same rate as New Zealand chicken consumption (chicken production in New Zealand is almost entirely for local consumption, and chickens represent almost all poultry meat consumption).
During the Belgian dioxin crisis of 1999, a significant decline in campylobacteriosis cases occurred at the same time as poultry meat consumption dropped leading to the conclusion that about 40% of campylobacteriosis is associated with chicken consumption. A similar marked reduction in campylobacteriosis was reported in the Netherlands following mass culling of chickens to eradicate an avian influenza outbreak.

The lack of outbreak patterns of disease strongly suggests *Campylobacter* is not commonly transmitted by raw food or during food preparation alone. Sporadic cases suggest a sporadic source or sporadic exposure.

While chicken meat prior to cooking is commonly contaminated, the sporadic incidence of human cases suggests this is not the source of most infections. Furthermore, analysis at the serotype strain level is ambivalent for chicken as the primary source of human campylobacteriosis.

In New Zealand, the primary source of infection is suggested to be dairy and beef cattle. New Zealand has high dairy and beef cattle numbers—rising from 8.8 to 9.6 million in the period 2000–2004 (www.nzmeatstatistics.co.nz accessed 30/11/2005)—or 2.3 animals per capita human population.

**Fly transmission**

Flies have been suggested as a transmission vector to account for the striking seasonal incidence of campylobacteriosis. There is no doubt that flies are physically able to transmit the low infective dose required for *Campylobacter*. Flies have already been directly implicated as vectors in broiler chicken infection, but no confirmation for human cases yet exists.

Nichols suggests that increased fly breeding rate, and therefore an increased risk of fly faecal contamination of foods, explains the seasonal nature of infection. This is not supported by New Zealand data where the seasonal peak occurs directly during the rise in temperatures into summer (Figure 2) and not with a delay for fly gestation and breeding.

This is more likely to relate to increased existing fly foraging activity of adult flies that have survived over winter, in contrast to chickens (where contamination occurs following consumption of the flies), and their infection peak occurs later when fly numbers have increased.

Increased fly foraging activity with consequent increased opportunity for direct and sporadic food contamination fits the observed seasonal pattern of campylobacteriosis. However, this does not explain the very strong association with chicken consumption, and particularly chicken eaten away from home. Flies are unlikely to contaminate chicken meals specifically.

The opportunity for flies to contaminate takeaway meals is relatively low compared to barbecued foods, yet these are the meals commonly implicated following illness.
Figure 2. Sporadic campylobacteriosis for (top to bottom) Taranaki, Canterbury and Southland District Health Boards

Raw numbers of cases have been converted to cases per 100,000 based on the 2001 national Census for normally resident population in each District. Average monthly temperature for New Plymouth, Christchurch, and Invercargill are plotted on the right hand side. Temperature data per National Institute for Water and Atmospheric Research (www.niwa.cri.nz)
Fomites and fingers

There is therefore another step in the route for environmental Campylobacter to cause human disease. Increased fly activity will result in increased fly contamination on common surfaces through faecal deposits and extracorporeal digestion (fly spots). Some of these deposits will be on hand rails, door handles, and other surfaces commonly touched by people.

Campylobacter deposited on human fingertips in an organic medium has been demonstrated to remain viable for at least 1 hour, and it can be recovered from dry surfaces 24 hours after contamination.

Fast food meals are typically eaten without eating utensils, even when eaten in restaurant facilities. Personal observation of diners at such outlets shows very few diners wash their hands prior to touching food, as noted elsewhere.

The specific association with chicken meals may be because chicken portions eaten with the fingers involve more moist-and-fatty food contact compared with most other fast foods. Contamination on fingers therefore has more opportunity to be transferred from the fingers to the mouth compared to foods eaten with less finger-licking. It is also possible that survey responses are biased against chicken through prior expectation of chicken as the culprit food.

Contrary to the findings of Hearnden et al., this fly-mediated food-associated transmission route could prove to be the single dominant transmission route in New Zealand.

Regional differences in seasonal incidence must take into account environmental sources of Campylobacter, fly foraging activity, and longevity of Campylobacter in fly deposits on touchable surfaces. These sources are likely to be affected by temperature, humidity, and rainfall variation, affecting both the environmental availability of bacteria, and through weather impacts on fly and human activities.

Discussion

Further research to elucidate a food-association source of campylobacteriosis will be necessary. Direct sampling of diners’ fingers at fast food outlets may indicate the validity of fingers as a source of environmental Campylobacter, although difficulties in culture may mask it.

Since New Zealand already has a reporting procedure in place for campylobacteriosis, intervention studies, such as an intensive fly control programme and hand washing education at fast food outlets, could also be used to determine the role of this mode of transmission.

This suggested food-associated role via flies and diners’ fingers is not intended to negate efforts to reduce campylobacteriosis by other means, especially the risk of outbreak disease through contamination of foods at source or during preparation. Furthermore, while 40% of cases are thought to be associated with chicken consumption, the other 60% of cases must therefore be from non-poultry sources.
New Zealand has the worst per capita statistics for campylobacteriosis amongst OECD countries. A high level of environmental *Campylobacter* availability because of intensive farming practices (linked with a ready transmission route from rural to urban areas), coupled with poor food hygiene practices, can go a long way to explaining this high statistic.

Understanding the source of the disease is key to preventive practices. Programmes in place to reduce the risk of direct food-borne transmission must not be reduced. However, additional steps to reduce food-associated transmission, resulting in the sporadic pattern of disease, can be undertaken relatively easily.

**Conclusion**

Campylobacteriosis rates in New Zealand follow similar patterns as those in other temperate OECD countries. This is a marked seasonal pattern peaking in early summer, sporadic rather than outbreak pattern of incidence, and increasing cases per capita with increasing chicken meat consumption.

The marked seasonal pattern does fit with an explanation as flies as vectors, and flies are implicated directly as transmission agents for *Campylobacter* to chickens. The seasonal and sporadic pattern does not suggest illness arising directly from contaminated meat.

The empirical data suggests a more complex ecology. This is proposed as involving seasonal increased overwintered fly foraging activity accounting for the seasonal component of disease. The flies bring *Campylobacter* organisms from the likely environmental source (cow faeces) to contaminate fomites by defecation and extracorporeal digestion.

The diners’ fingers become contaminated by touching these fomites and an infectious dose is transferred during eating, particularly as it is common not to wash hands prior to eating a takeaway meal. Chicken meals are particularly implicated because of the intensive finger contact with moist food, and finger licking, during consumption.

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**Correspondence:** Warrick Nelson, 888 Management Ltd, PO Box 6393, Upper Riccarton, Christchurch. Email: warrick.nelson@gmail.com

**References:**


Dietary intakes by different markers of socioeconomic status: results of a New Zealand workforce survey

Patricia Metcalf, Robert Scragg, Peter Davis

Abstract

Aim To compare dietary nutrient and food group intakes of men and women in a work force with various measures of socioeconomic status.

Methods Daily nutrient intakes were calculated from a self-administered food frequency questionnaire from participants in a cross-sectional health screening survey of a multiracial workforce carried out between May 1988 and April 1990. Participants comprised 5517 Maori, Pacific Island and Other workers (3997 men, 1520 women) aged 40 to 78 years. Socioeconomic measures included the New Zealand Socioeconomic Index (NZSEI), gross household income and level of education.

Results In general, there were trends across socioeconomic status levels with lower NZSEI occupational classes, lower family income, and non-tertiary education groups having lower intakes of dietary fibre, calcium, and alcohol and higher intakes of dietary cholesterol. These were reflected by their lower intakes of fruit, vegetables, milk, cheese and wine, and higher intakes of eggs. However, associations were not consistent across all measures of socioeconomic status.

Conclusions Dietary intakes showed a generally more adverse pattern in the lower socioeconomic strata. NZSEI and education were associated with food group selections, whereas nutrient intakes were associated with income. More money available for food could improve nutrition. Public health programmes to improve nutrition need to be targeted at these groups and be coupled with personal support and structural changes that make “healthy choices the easy choices”.

Studies concerning the relationships between diet and risk of chronic diseases suggest that dietary habits may play an important role in the pathogenesis of some diseases. Food intake during childhood and early adulthood is thought to influence the risk of several chronic diseases and conditions including coronary heart disease, high blood pressure, some cancers, diabetes mellitus, alcohol-related diseases, osteoporosis, and obesity in later life.

Previous overseas and local studies have reported clear socioeconomic differentials in the dietary patterns of various population subgroups. Measures of socioeconomic status that have been shown to have the greatest influence on health in New Zealand include income and poverty, employment and occupation, education, housing, population-based services, social cohesion and culture, and ethnicity.

Previous large New Zealand nutrition surveys include the 1977 Nutrition Survey, the 1988/89 Life in New Zealand survey, and the 1997 National Nutrition Survey. These surveys used the 24-hour dietary recall for estimating nutrient intakes and a food frequency questionnaire for reporting frequency of intake of the various foods,
but used different measures of socioeconomic status when reporting their results, namely the Registrar General Socioeconomic Scale of occupations (for alcohol only), the Elley-Irving classification of occupations, and a proxy measure of socioeconomic status based on deprivation (NZDEP96), respectively.

The aim of this study was to compare daily dietary nutrient intakes and food group servings per month across New Zealand Socioeconomic Index (NZSEI) occupational classes, income, and levels of tertiary education groups to determine whether there were important independent gradient differentials after adjusting for other measures of socioeconomic status.

**Subjects and Methods**

5677 individuals (4108 men and 1569 women) aged 40–64 years participated in a health screening survey of a local workforce between May 1988 and April 1990. Food frequency questionnaires (FFQ) were completed by 5614 individuals (4067 men, 1547 women) comprising 79.2% European, 7.6% Maori, 11.4% Pacific, and 1.8% Asian workers. Pacific participants comprised 53.2% Samoan, 10.8% Tongan, 7.0% Niuean, 26.7% Cook Islander, and 2.3% from other Pacific Islands. Ethnicity was self-defined and all participants gave informed consent. Ethical approval was obtained from the University of Auckland Human Ethics Committee.

Food intake over the previous 3 months was estimated by a 142-item food frequency questionnaire (FFQ) that included commonly eaten Maori and Pacific Islands foods (e.g. mutton flaps, povi masima, pipis, mussels, oysters, coconut cream, green bananas, puha, kumara, yam, taro tuber, and Maori bread). The FFQ was filled in by participants at their home, and checked for errors and omissions at their interview the following morning.

Serving sizes of vegetables, meat, fish, and cake were assessed using colour photographs of foods which participants used to rank themselves into three portion size groups (more, same, less). These were scaled as less 0.5, same 1.0, and more 1.6. Otherwise, natural serving sizes, such as the average weight of a piece of fruit, or slice of bread were used or published serve sizes.

The comprehensive version of the food composition tables was used to calculate nutrient intake. We have previously reported that this FFQ was valid and reproducible in European, and Maori and Pacific Islands participants.

Occupational class was assigned as the highest of the participant or their spouse using the New Zealand Socioeconomic Index (NZSEI). NZSEI was then transformed into discrete ‘occupational classes’ as proposed by Davis et al.

These classes are comprised of:

- Class 1 – legislators and administrators;
- Class 2 – various professionals;
- Class 3 – corporate managers, associate professionals, and the armed forces;
- Class 4 – trade workers, plant operators, and office clerks;
- Class 5 – other trade workers, machine operators and labourers; and
- Class 6 – market-orientated agricultural and fishery workers.

Classes 1 and 2 and Classes 5 and 6 were combined due to their small numbers. Education was classified as ‘no tertiary education’, trade school, or technical institute (e.g. carpenter, electrician, or secretary-typing certificate), professional institution (e.g. teacher, registered nurse, accountant or naval officer), or university attendance. Gross annual household income categories were <$20,000, $20,000 to < $30,000, $30,000 to $40,000; and >$40,000.

Resting metabolic rate, the total minimal activity of all tissue cells of the body under steady state conditions, is expressed as the rate of heat production or oxygen consumption related to some unit of body size. Resting metabolic rate (RMR) was calculated using standard equations.

Minimal requirements for the ratio of total energy intake (MJ) to resting metabolic rate (EI/RMR) is 1.55 by WHO criteria and 1.38 according to Goldberg et al. This assesses the degree of any under-
reporting and was included to determine whether there was differential under-reporting between any of the socioeconomic status groups.

Because of the positively skewed frequency distribution of nutrient intakes, these were converted to log values for calculations; the results presented are geometric means (the exponential of the log-transformed data) and associated 95% tolerance factor\{exponential[loge(1.96*standard error)]\}. A 95% confidence interval can be obtained as mean± tolerance factor to mean*tolerance factor.

Analysis of covariance was used to calculate age, gender, and ethnicity adjusted nutrients and age, gender, ethnicity, and total energy-adjusted food servings in the different groups. A two-tailed P value <0.01 was the criterion for statistical significance because of multiple nutrient comparisons. Analyses were carried out using SAS software.16

Results

Higher correlations were observed between NZSEI occupational class and education \(r=0.49\), NZSEI, and income \(r=0.35\) than between income and education \(r=0.28\).

Geometric mean daily nutrient intakes are reported in Table 1 by NZSEI occupational classes after adjusting for age, gender, and ethnicity. Compared to class 1&2, daily calcium intakes were lower in classes 3 to 6, cholesterol intakes were higher in classes 4 and 5&6, and sucrose intakes were higher and fibre intakes lower in class 5&6.

There was no evidence of differential under-reporting between the occupational classes as the energy intakes divided by resting metabolic rate (EI/RMR) were not significantly different. After further adjusting for family income and education, the only significant differences from occupational class 1&2 were dietary cholesterol intakes in classes 4 to 6.

The significant differences for dietary fibre and calcium intakes were no longer significant after adjusting for level of education, and sucrose was no longer significant after adjusting for income.

When nutrients were expressed as their percentage contribution to total energy intakes and adjusted for age, gender, ethnicity, family income, and education (individual data not shown), only cholesterol intakes remained significantly higher in NZSEI classes 4 and 5&6 compared to class 1&2.

Table 2 shows food group intakes by NZSEI occupational group after adjusting for age, gender, ethnicity, and total energy intakes. There were trends towards lower vegetable, fruit, cheese, milk, and wine servings; and higher egg and beer servings; in the lower occupational classes compared to class 1&2. However, after further adjusting for education, only the higher egg and beer intakes and lower cheese, milk, and wine intakes remained statistically significant.

The results remained unchanged after further adjustment for family income. It should be noted that the size of the servings may differ as some people may eat more in lesser servings.

Geometric mean daily nutrient intakes by gross household income, adjusted for age, gender, and ethnicity, are shown in Table 3. Compared to incomes ≥$40,000, incomes of <$20,000 and $20≤$30,000 had significantly less under-reporting as measured by the total energy intake divided by the resting metabolic rate.

Compared to the highest income group, the lowest income group had significantly higher total energy, carbohydrate, starch, sucrose, protein, fat, saturated fat, and cholesterol intakes and significantly lower fibre and alcohol intakes.
Table 1. Geometric mean (95% tolerance factor) daily nutrient intakes by NZSEI occupational class (1&2=highest, 5&6=lowest) adjusted for age, gender, and ethnicity

<table>
<thead>
<tr>
<th>NZSEI occupational class</th>
<th>1&amp;2</th>
<th>3</th>
<th>4</th>
<th>5&amp;6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>825</td>
<td>882</td>
<td>2276</td>
<td>1631</td>
</tr>
<tr>
<td>Energy (MJ)</td>
<td>9.1 (1.02)</td>
<td>8.9 (1.02)</td>
<td>9.2 (1.01)</td>
<td>9.4 (1.02)</td>
</tr>
<tr>
<td>EI/RMR [mean(SE)]</td>
<td>1.35 (0.02)</td>
<td>1.32 (0.02)</td>
<td>1.38 (0.01)</td>
<td>1.39 (0.01)</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>239 (1.03)</td>
<td>230 (1.03)</td>
<td>241 (1.02)</td>
<td>243 (1.02)</td>
</tr>
<tr>
<td>Starch (g)</td>
<td>122 (1.03)</td>
<td>116 (1.03)</td>
<td>124 (1.02)</td>
<td>124 (1.04)</td>
</tr>
<tr>
<td>Sucrose (g)</td>
<td>51 (1.04)</td>
<td>51 (1.04)</td>
<td>53 (1.03)</td>
<td>55 (1.03)</td>
</tr>
<tr>
<td>Fibre (g)</td>
<td>27 (1.03)</td>
<td>25 (1.03)</td>
<td>26 (1.02)</td>
<td>25 (1.02)</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>87 (1.02)</td>
<td>84 (1.02)</td>
<td>87 (1.01)</td>
<td>89 (1.02)</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>93 (1.03)</td>
<td>92 (1.03)</td>
<td>94 (1.02)</td>
<td>95 (1.02)</td>
</tr>
<tr>
<td>SFA (g)</td>
<td>38 (1.03)</td>
<td>37 (1.02)</td>
<td>39 (1.02)</td>
<td>39 (1.02)</td>
</tr>
<tr>
<td>PUFA (g)</td>
<td>11 (1.03)</td>
<td>11 (1.03)</td>
<td>11 (1.02)</td>
<td>11 (1.02)</td>
</tr>
<tr>
<td>MUFA (g)</td>
<td>29 (1.03)</td>
<td>28 (1.03)</td>
<td>29 (1.02)</td>
<td>29 (1.02)</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>314 (1.03)</td>
<td>317 (1.03)</td>
<td>334 (1.02)</td>
<td>347 (1.02)</td>
</tr>
<tr>
<td>Alcohol (g)</td>
<td>4.2 (1.08)</td>
<td>4.8 (1.08)</td>
<td>4.5 (1.05)</td>
<td>4.0 (1.06)</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>795 (1.03)</td>
<td>750 (1.03)</td>
<td>759 (1.02)</td>
<td>741 (1.02)</td>
</tr>
</tbody>
</table>

*P<0.01; **P<0.001 compared to NZSEI Class 1&2. EI/RMR=total energy intake divided by resting metabolic rate; SFA=saturated fatty acids; PUFA=polyunsaturated fatty acids; MUFA=monounsaturated fatty acids. †No longer significant after adjusting for age and gender.

Table 2. Mean (SE) servings of food groups per month adjusted for age, gender, ethnicity, and total energy intake by NZSEI occupational class (1&2=highest, 5&6=lowest)

<table>
<thead>
<tr>
<th>NZSEI occupational class</th>
<th>1&amp;2</th>
<th>3</th>
<th>4</th>
<th>5&amp;6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>825</td>
<td>882</td>
<td>2276</td>
<td>1631</td>
</tr>
<tr>
<td>Red meat</td>
<td>34.0 (0.65)</td>
<td>34.4 (0.63)</td>
<td>35.0 (0.38)</td>
<td>35.5 (0.47)</td>
</tr>
<tr>
<td>Chicken</td>
<td>5.3 (0.21)</td>
<td>5.4 (0.21)</td>
<td>5.3 (0.13)</td>
<td>5.9 (0.15)</td>
</tr>
<tr>
<td>Fish</td>
<td>8.4 (0.28)</td>
<td>8.1 (0.27)</td>
<td>8.0 (0.17)</td>
<td>8.6 (0.20)</td>
</tr>
<tr>
<td>Vegetables</td>
<td>130.7 (2.07)</td>
<td>128.5 (2.01)</td>
<td>122.5 (1.23)</td>
<td>123.5 (1.50)</td>
</tr>
<tr>
<td>Fruit</td>
<td>59.5 (1.59)</td>
<td>56.0 (1.54)</td>
<td>51.9 (0.94)</td>
<td>51.5 (1.15)</td>
</tr>
<tr>
<td>Eggs (number)</td>
<td>9.1 (0.37)</td>
<td>10.4 (0.36)</td>
<td>11.6 (0.22)</td>
<td>11.9 (0.27)</td>
</tr>
<tr>
<td>Cheese(^2^)</td>
<td>12.4 (0.42)</td>
<td>11.5 (0.41)</td>
<td>10.8 (0.25)</td>
<td>8.9 (0.31)</td>
</tr>
<tr>
<td>Milk (cups per month)</td>
<td>71.4 (2.29)</td>
<td>65.5 (2.21)</td>
<td>64.4 (1.37)</td>
<td>58.3 (1.67)</td>
</tr>
<tr>
<td>Bread (slices per month)</td>
<td>28.0 (0.55)</td>
<td>26.7 (0.53)</td>
<td>30.0 (0.33)</td>
<td>29.6 (0.40)</td>
</tr>
<tr>
<td>Cereal</td>
<td>31.5 (2.50)</td>
<td>30.0 (2.42)</td>
<td>28.9 (1.49)</td>
<td>29.0 (1.81)</td>
</tr>
<tr>
<td>Wine</td>
<td>9.6 (0.62)</td>
<td>10.2 (0.60)</td>
<td>7.6 (0.37)</td>
<td>5.1 (0.45)</td>
</tr>
<tr>
<td>Beer</td>
<td>14.7 (1.45)</td>
<td>16.5 (1.40)</td>
<td>21.3 (0.87)</td>
<td>25.9 (1.05)</td>
</tr>
<tr>
<td>Spirits</td>
<td>4.3 (0.48)</td>
<td>5.4 (0.46)</td>
<td>4.5 (0.29)</td>
<td>4.0 (0.35)</td>
</tr>
</tbody>
</table>

*P<0.01; **P<0.001 compared to NZSEI Class 1&2; †No longer significant after adjusting for income and education. \(^2^\)Excludes cottage cheese.
### Table 3. Geometric mean (95% tolerance factor) daily nutrient intakes by income adjusted for age, gender, and ethnicity

<table>
<thead>
<tr>
<th>Income</th>
<th>Number</th>
<th>Energy (MJ)</th>
<th>EI/RMR [mean(SE)]</th>
<th>Carbohydrate (g)</th>
<th>Starch (g)</th>
<th>Sucrose (g)</th>
<th>Fibre (g)</th>
<th>Fat (g)</th>
<th>SFA (g)</th>
<th>PUFA (g)</th>
<th>MUFA (g)</th>
<th>Cholesterol (mg)</th>
<th>Alcohol (g)</th>
<th>Calcium (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥$40,000</td>
<td>2670</td>
<td>9.1 (1.01)</td>
<td>1.34 (0.01)</td>
<td>121 (1.02)</td>
<td>51 (1.02)</td>
<td>26 (1.02)</td>
<td>93 (1.01)</td>
<td>38 (1.02)</td>
<td>11 (1.02)</td>
<td>11 (1.02)</td>
<td>126 (1.02)*</td>
<td>5.2 (1.05)</td>
<td>770 (1.02)</td>
<td></td>
</tr>
<tr>
<td>$30–≤$40,000</td>
<td>1273</td>
<td>9.2 (1.02)</td>
<td>1.36 (0.01)</td>
<td>123 (1.02)*</td>
<td>55 (1.04)**</td>
<td>25 (1.02)</td>
<td>93 (1.02)</td>
<td>38 (1.02)</td>
<td>11 (1.03)</td>
<td>28 (1.02)</td>
<td>125 (1.02)*</td>
<td>4.2 (1.06)**</td>
<td>749 (1.02)</td>
<td></td>
</tr>
<tr>
<td>$20–≤$30,000</td>
<td>1074</td>
<td>9.4 (1.02)</td>
<td>1.41 (0.02)**†</td>
<td>125 (1.02)*</td>
<td>55 (1.04)**</td>
<td>25 (1.03)**</td>
<td>96 (1.02)</td>
<td>39 (1.03)</td>
<td>29 (1.02)</td>
<td>29 (1.02)</td>
<td>12 (1.03)</td>
<td>3.6 (1.07)**</td>
<td>763 (1.03)</td>
<td></td>
</tr>
<tr>
<td>≤$20,000</td>
<td>273</td>
<td>9.7 (1.04)**</td>
<td>1.47 (0.03)**†</td>
<td>132 (1.05)*</td>
<td>59 (1.08)*</td>
<td>100 (1.05)**</td>
<td>92 (1.04)**</td>
<td>42 (1.05)**</td>
<td>30 (1.05)</td>
<td>30 (1.05)</td>
<td>117 (1.368)*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P<0.01; ** P<0.001 compared to income ≥$40,000; EI/RMR=total energy intake divided by resting metabolic rate; SFA=saturated fatty acids; PUFA=polyunsaturated fatty acids; MUFA=monounsaturated fatty acids; †No longer significant after adjusting for NZSEI and education.

### Table 4. Mean (SE) servings of food groups per month adjusted for age, gender, ethnicity, and total energy intake by level of income.

<table>
<thead>
<tr>
<th>Income</th>
<th>Number</th>
<th>Red meat</th>
<th>Chicken</th>
<th>Fish</th>
<th>Vegetables</th>
<th>Fruit</th>
<th>Eggs</th>
<th>Cheese‡</th>
<th>Milk</th>
<th>Bread</th>
<th>Cereal</th>
<th>Wine</th>
<th>Beer</th>
<th>Spirits</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥$40,000</td>
<td>2670</td>
<td>34.6 (0.36)</td>
<td>5.3 (0.12)</td>
<td>8.0 (0.15)</td>
<td>127.4 (1.16)</td>
<td>56.9 (0.89)</td>
<td>10.7 (0.21)</td>
<td>11.5 (0.24)</td>
<td>67.3 (1.30)</td>
<td>28.3 (0.31)</td>
<td>30.2 (1.45)</td>
<td>9.3 (0.36)</td>
<td>20.6 (0.82)</td>
<td>5.5 (0.28)</td>
</tr>
<tr>
<td>$30–≤$40,000</td>
<td>1273</td>
<td>34.6 (0.51)</td>
<td>5.3 (0.17)</td>
<td>7.8 (0.22)</td>
<td>123.9 (1.63)</td>
<td>51.6 (1.25)**</td>
<td>11.5 (0.30)</td>
<td>10.4 (0.34)</td>
<td>61.8 (1.83)</td>
<td>30.2 (0.44)**</td>
<td>28.6 (2.04)</td>
<td>6.9 (0.50)**</td>
<td>21.0 (1.16)</td>
<td>3.9 (0.39)</td>
</tr>
<tr>
<td>$20–≤$30,000</td>
<td>1074</td>
<td>35.7 (0.57)</td>
<td>5.7 (0.19)</td>
<td>8.5 (0.24)</td>
<td>122.2 (1.82)</td>
<td>49.8 (1.40)**</td>
<td>11.4 (0.33)</td>
<td>9.9 (0.39)**†</td>
<td>61.9 (2.07)</td>
<td>29.8 (0.50)</td>
<td>31.7 (2.28)</td>
<td>6.2 (0.57)**</td>
<td>22.8 (1.31)</td>
<td>3.4 (0.44)**</td>
</tr>
<tr>
<td>≤$20,000</td>
<td>273</td>
<td>36.1 (1.14)</td>
<td>6.6 (0.38)**†</td>
<td>8.9 (0.49)</td>
<td>117.1 (3.68)*</td>
<td>43.6 (2.83)**</td>
<td>12.3 (0.67)</td>
<td>9.5 (0.80)</td>
<td>61.0 (4.25)</td>
<td>28.4 (1.03)</td>
<td>25.3 (4.61)</td>
<td>3.4 (1.17)**</td>
<td>19.2 (2.70)</td>
<td>3.2 (0.90)</td>
</tr>
</tbody>
</table>

*P<0.01; ** P<0.001 compared to income ≥$40,000. †No longer significant after adjusting for NZSEI and education. ‡Excludes cottage cheese.
Table 5. Mean (SE) servings of food groups per month adjusted for age, gender, ethnicity and total energy intakes by level of education

<table>
<thead>
<tr>
<th>Education</th>
<th>University</th>
<th>Technical College</th>
<th>Trades</th>
<th>No tertiary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>466</td>
<td>596</td>
<td>1806</td>
<td>2741</td>
</tr>
<tr>
<td>Red meat</td>
<td>33.3 (0.86)</td>
<td>34.6 (0.76)</td>
<td>34.5 (0.44)</td>
<td>35.5 (0.36)</td>
</tr>
<tr>
<td>Chicken</td>
<td>5.1 (0.28)</td>
<td>5.3 (0.25)</td>
<td>5.3 (0.15)</td>
<td>5.7 (0.12)</td>
</tr>
<tr>
<td>Fish</td>
<td>8.1 (0.37)</td>
<td>8.2 (0.33)</td>
<td>8.2 (0.19)</td>
<td>8.3 (0.16)</td>
</tr>
<tr>
<td>Vegetables</td>
<td>132.8 (2.75)</td>
<td>128.5 (2.43)</td>
<td>126.0 (1.42)</td>
<td>122.2 (1.16)</td>
</tr>
<tr>
<td>Fruit</td>
<td>62.1 (2.11)</td>
<td>56.8 (1.86)</td>
<td>55.0 (1.09)*</td>
<td>50.5 (0.89)**</td>
</tr>
<tr>
<td>Eggs</td>
<td>9.3 (0.49)</td>
<td>10.5 (0.43)</td>
<td>10.8 (0.25)*†</td>
<td>11.8 (0.21)**†</td>
</tr>
<tr>
<td>Cheese‡</td>
<td>12.6 (0.57)</td>
<td>12.2 (0.51)</td>
<td>11.2 (0.29)</td>
<td>9.6 (0.24)**</td>
</tr>
<tr>
<td>Milk</td>
<td>72.6 (3.03)</td>
<td>68.6 (2.70)**</td>
<td>67.5 (1.56)*</td>
<td>58.9 (1.29)**</td>
</tr>
<tr>
<td>Bread</td>
<td>26.1 (0.73)</td>
<td>27.4 (0.65)</td>
<td>29.3 (0.38)**</td>
<td>29.7 (0.31)**</td>
</tr>
<tr>
<td>Cereal</td>
<td>32.4 (3.32)</td>
<td>31.6 (2.94)</td>
<td>29.1 (1.71)</td>
<td>28.8 (1.40)</td>
</tr>
<tr>
<td>Wine</td>
<td>12.1 (0.82)</td>
<td>9.5 (0.73)</td>
<td>7.7 (0.42)**</td>
<td>6.4 (0.35)**</td>
</tr>
<tr>
<td>Beer</td>
<td>11.9 (1.92)</td>
<td>15.8 (1.71)</td>
<td>20.3 (0.99)**</td>
<td>23.8 (0.82)**</td>
</tr>
<tr>
<td>Spirits</td>
<td>3.7 (0.64)</td>
<td>5.0 (0.57)</td>
<td>4.4 (0.33)</td>
<td>4.6 (0.27)</td>
</tr>
</tbody>
</table>

*P< 0.01; ** P<0.001 compared to University education group; †No longer significant after adjusting for NZSEI and education; ‡Excludes cottage cheese

After further adjusting for NZSEI and education, only starch, saturated fatty acids and alcohol intakes remained significant. With the exception of cholesterol intakes that were explained by level of education, the other initially significant differences were no longer significant after adjusting for NZSEI. Similar results for family income were obtained when nutrients were expressed as percentage contribution to total energy intakes and adjusted for age, gender, ethnicity, NZSEI, and education (individual data not shown).

Table 4 shows mean food group serving per month by family income, after adjusting for age, gender, ethnicity, and total energy intakes; there were trends towards higher chicken intakes and lower vegetable, fruit, wine, and spirit intakes in the lower income groups compared to the highest income group are also apparent.

After further adjusting for NZSEI, there were no longer significant differences for chicken intakes, and vegetable intakes were explained by level of education. Adjustment for NZSEI and education did not change the significance levels for fruit, wine, or spirits.

Mean nutrient intakes, adjusted for age, gender, and ethnicity, were calculated by level of tertiary education (individual data not shown). There were trends towards lower geometric mean (tolerance factor) fibre (No tertiary 25 (1.02) g/day versus University 27 (1.04) g/day, p < 0.001) and calcium intakes (No tertiary 728 (1.02) mg/day versus University 812 (1.04) mg/day, P < 0.001) in those with no tertiary education compared to those who had attended University.

Results were unchanged after further adjusting for family income and NZSEI. There were no significant differences in nutrients expressed as their contribution to total energy intake by level of education after adjusting for age, gender, ethnicity, NZSEI, and family income (individual data not shown).
Table 5 shows mean food group servings per month by education after adjusting for age, gender, ethnicity, and total energy intakes. There were trends towards lower vegetables, fruit, cheese, milk, and wine intakes; and higher egg, bread, and beer intakes; at lower levels of tertiary education compared to those with a University education.

After further adjusting for NZSEI and income, significant differences remained for fruit, cheese, milk, bread, wine, and beer intakes. Initially significant differences for vegetable and egg intakes were no longer significant after adjusting for NZSEI.

Discussion

Consistent associations across all three measures of socioeconomic status (adjusted for age and gender) showed that low socioeconomic status was associated with lower intakes of vegetables, fruit, cheese, and wine.

Some of the initially significant nutrient and food differences across socioeconomic status groups were explained by the inclusion of the other measures of socioeconomic status (e.g., total energy, carbohydrate, sucrose, total fat, protein, and chicken associations with income were no longer significant after adjusting for NZSEI). However, there were differences in the way that the different socioeconomic measures captured different aspects of diet.

Independent associations of dietary nutrients across occupation groups as measured by the NZSEI were higher dietary cholesterol, egg, and beer intakes; and lower cheese, milk, and wine intakes; in the lower occupational classes compared to the highest occupational classes after adjusting for the other socioeconomic measures.

Similarly, the following independent associations occurred between dietary nutrients and gross combined family income: higher starch and saturated fatty acids intakes—and lower alcohol, fruit, wine, and spirit intakes. A lower education level was associated with lower fibre, calcium, cheese, milk, and wine intakes; and higher bread and beer intakes; compared to those with a University education.

Table 6 compares the associations reported for the different measures of socioeconomic status. The 1989–90 LINZ survey reported median nutrient intakes by the Elley-Irving classification of occupations and by education (including primary education which reflected very old people). Results in Table 6 report a “Trend” if there were consistent associations across these socioeconomic variables, but excludes those with primary school education only.

In addition, the associations between nutrient intakes and NZDEP96, and NZSEI, from the 1997 National Nutrition Survey are shown. However, it was not possible to compare food intakes with these other two surveys because results were expressed as the proportion of people eating at least one serve per week. Some food groups were mentioned in the text as having a significant socioeconomic gradient and are noted.

Table 6 shows that there are some dietary differences between the different socioeconomic status measures. For example, higher dietary cholesterol was found in the lower socioeconomic classes for NZSEI, income, and the Elley-Irving classification of occupations, but no association was found for education or NZDEP96. Higher levels of starch were observed in the lower income groups.
Table 6. Significant (✓) and non-significant (X) associations between diet intake and measures of socioeconomic status from New Zealand surveys

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NZSEI</td>
<td>Income</td>
<td>Education</td>
</tr>
<tr>
<td>Energy</td>
<td>X</td>
<td>✓†</td>
<td>X</td>
</tr>
<tr>
<td>Fat</td>
<td>X</td>
<td>✓†</td>
<td>X</td>
</tr>
<tr>
<td>SFA</td>
<td>X</td>
<td>✓</td>
<td>X</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>✓</td>
<td>✓†</td>
<td>X</td>
</tr>
<tr>
<td>Starch</td>
<td>X</td>
<td>✓</td>
<td>X</td>
</tr>
<tr>
<td>Sucrose</td>
<td>✓†</td>
<td>✓†</td>
<td>X</td>
</tr>
<tr>
<td>Fibre</td>
<td>✓†</td>
<td>✓†</td>
<td>✓</td>
</tr>
<tr>
<td>Calcium</td>
<td>✓†</td>
<td>X</td>
<td>✓</td>
</tr>
<tr>
<td>Alcohol</td>
<td>X</td>
<td>✓</td>
<td>X</td>
</tr>
<tr>
<td>Milk</td>
<td>✓</td>
<td>✓</td>
<td>X</td>
</tr>
<tr>
<td>Cheese</td>
<td>✓</td>
<td>✓†</td>
<td>✓</td>
</tr>
<tr>
<td>Vegetables</td>
<td>✓†</td>
<td>✓†</td>
<td>✓†</td>
</tr>
<tr>
<td>Fruit</td>
<td>✓†</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Bread</td>
<td>X</td>
<td>X</td>
<td>✓</td>
</tr>
<tr>
<td>Eggs</td>
<td>✓</td>
<td>X</td>
<td>✓†</td>
</tr>
<tr>
<td>Wine</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Beer</td>
<td>✓</td>
<td>X</td>
<td>✓</td>
</tr>
<tr>
<td>Spirits</td>
<td>X</td>
<td>✓</td>
<td>X</td>
</tr>
</tbody>
</table>

✓ Indicates a significant association reported with socioeconomic variable. X Indicates no significant association reported.

† No longer significant after adjusting for other measures of socioeconomic status. SFA = saturated fatty acids.

- Difficult to compare as individual food items reported. Trend = a consistent trend was observed across medians (primary education excluded), but no comment was made in the text.

LINZ and NNS used 24-hour dietary recalls for nutrients and a food frequency questionnaire for frequency of food intakes. WDS used a food frequency questionnaire for both.
In contrast, no such patterns were observed across the NZSEI and Elley-Irving classification of occupations, education, or NZDEP96 quartiles. Overall, lower socioeconomic status was associated with a less healthy diet than the higher occupational classes. Furthermore, this pattern does not appear to have changed much between the 1988–1990s surveys and the 1997 survey.

Lower alcohol servings was observed in the lower income groups, with lower wine servings and higher beer servings in the lower NZSEI groups—and lower education groups, and lower wine servings only in the lower income groups.

The 1977 nutrition survey reported that the results “do not provide substantial evidence of major differences of social importance.” Similarly, the Elley-Irving occupational scale showed no association with alcohol; however, lower occupational status was associated with lower wine intakes in men and women, and lower spirits intake in men. The associations with NZDEP96 also showed lower alcohol consumption in the lower quartiles, with lower consumption of wine and spirits, but no clear association with beer intakes.

Both the NZSEI occupation class and education showed lower calcium, cheese, and milk intakes with lower levels of these measures. The LINZ survey also noted a lower cheese intake with lower levels of education, and lower cheese and calcium intakes were observed in lower NZDEP96 quartiles.

The NNS reported significant gradients in dietary fibre with NZDep96, as was observed with education in the current study. However, no such associations were observed using the Elley-Irving classification of occupations.

Independent associations were observed with lower fruit intakes in lower income (Table 4) and education (Table 5) groups. Similar associations were observed with the Elley-Irving classification of occupations in men only. The Elley-Irving classification of occupations (in men only) also reported higher egg servings in the lower socioeconomic groups, as was observed in the current study for the NZSEI classification of occupations (Table 2).

Although the socioeconomic measures were correlated, with Pearson’s coefficients ranging from 0.28 to 0.49, the correlations are not high enough to suggest that they are completely dependent on one another, and the results suggest that the different socioeconomic status measures provide different information. Horwath et al. noted that “education seemed to be a more sensitive discriminator of social class differences in dietary patterns” compared to the Elley-Irving classification of occupations.

The NZDEP96 uses nine markers of deprivation (e.g. income, housing, occupancy, and access to a telephone) but it is uncertain about the extent to which small-area measures reflect the individual effects. Davis et al have suggested that income may be a better predictor of health status than any other socioeconomic status measure. Education has been regarded as being interchangeable with income and occupation class.

It has been reported that an advantage of using education as a marker of socioeconomic status is that it remains fixed for the majority of people after 25 years of age. In contrast, income can fluctuate over time, so that in any given year, it may
not reflect usual income and is more liable to be affected by illness. Occupational
class is often more stable than annual income, but is also less likely to be less stable
than education. Furthermore, some people refuse to report income in such surveys,
and others do not know the household income.

Study limitations include the fact this was a working population and its results cannot
be easily generalised to the non-working population. A further limitation involves
using the Goldberg cut-off level for under-reporting when used with food frequency
questionnaires.

Non-differential misclassification is a problem of principal interest in nutrition
epidemiology, since it always works in the direction towards the null. In addition, the
cross-sectional design makes it difficult to draw inferences about causal pathways.
Another limitation is that the data reported in the current study were obtained prior to
the benefit cuts of 1990, so that any trends picked up here may have been exacerbated
after this time.

The current study showed that each of the different markers of socioeconomic status
appeared to capture some different dietary habits across the strata. Differences
between socioeconomic status markers and health outcomes (mortality and morbidity)
have been summarised previously.

This study appears to the first in New Zealand to simultaneously adjust for other
measures of socioeconomic status in order to determine independent nutrition patterns
by NZSEI, income, and education and to report food servings by socioeconomic
status. Knowledge of the socioeconomic behaviours could help target interventions.

Conclusions

Dietary intakes showed a generally more adverse pattern in the lower socioeconomic
strata. NZSEI and education were associated with food group selections, whereas
nutrient intakes were associated with income. More money available for food could
improve nutrition. Public health programmes to improve nutrition need to be targeted
at these groups and be coupled with personal support and structural changes that make
“healthy choices the easy choices.”

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References:


Metabolic characteristics of patients with apparently normal fasting plasma glucose

Geoff Braatvedt, Greg Gamble, Cam Kyle

Abstract

Aims To describe the prevalence of dysglycaemia in patients with fasting glucose <6.1 mmol/L.

Methods Consecutive patients referred for OGTT between July 2002 and December 2003 to eight Diagnostic Medical Laboratory depots in the Auckland region of New Zealand were invited to participate. In addition to a standard OGTT, patients’ BMI was calculated and HbA
1c, fructosamine, lipids, and insulin concentrations were measured. Patients were grouped according to fasting glucose of <5.5 mmol/L=normal, 5.5–6.0 mmol/L=“high fives”, 6.1–6.9 mmol/L=“old” impaired fasting glucose, and ≥7 mmol/L=diabetes.

Results 310 patients were studied. 111 patients had a fasting glucose of <5.5 mmol/L, and of these, 23 had IGT and 2 diabetes on OGTT; 85 patients had a fasting glucose 5.5–6.0 mmol/L, and 18 of these had IGT and 11 diabetes on OGTT; 75 patients had a fasting glucose of 6.1–6.9 mmol/L, and of these, 33 had IGT and 21 diabetes on OGTT; 39 patients had a fasting glucose ≥7 mmol/L and 38 were confirmed diabetic on OGTT.

Conclusion This study suggests that the upper limit of normal fasting glucose be lowered to <5.5 mmol/L in line with Australian and American Diabetes Society guidelines.

Type 2 diabetes is a growing epidemic in New Zealand with an estimated 115,000 patients having diagnosed diabetes in 2000. Population screening studies suggest that half of those with diabetes are undiagnosed and many more have impaired glucose tolerance (IGT) and are thus at risk of developing diabetes. The prevalence of IGT and impaired fasting glucose (IFG) in New Zealand is, however, unknown. Intervention studies have shown that patients with IGT can reduce their risk of developing diabetes by about 60% by combining exercise with lifestyle changes, and by about 30% with metformin use; thus diagnosing patients with IGT is worthwhile.

Furthermore, many patients with IGT have coexisting metabolic abnormalities that cluster as the metabolic syndrome, which increases the risk of macrovascular disease and again lends itself to intervention strategies to alter this risk.

In the late 1990s, the American Diabetes Association (ADA) and the WHO proposed a new category of glucose tolerance called impaired fasting glucose (IFG). Values of fasting glucose of ≥7 mmol/L were classified as consistent with diabetes and values 6.1–6.9 mmol/L as impaired fasting glucose, with values <6.1 mmol/L classified as normal.
A significant minority of subjects with IFG have either IGT or diabetes on subsequent oral glucose tolerance test (OGTT). Therefore, proceeding to OGTT was recommended at that time by both the New Zealand and Australian Diabetes’ Societies for patients with IFG \(^9,10\) (fasting glucose 6.1–6.9 mmol/L) even though the ADA did not take this approach, relying only on the fasting glucose for classification of patients.

A previous New Zealand study showed that a significant number of patients with diabetes on OGTT would be misclassified as non-diabetic using the ADA fasting criteria alone.\(^11\)

Recently the ADA\(^12\) has suggested a new upper limit of normal fasting glucose of 5.5 mmol/L thus expanding the IFG category from 6.1–6.9 to 5.6–6.9 mmol/L. This was based on the results of studies from Europe and the USA showing a significant minority of patients with fasting glucose of between 5.6–6.0 mmol/L (previously classified normal) as having IGT or even diabetes.

The Australian Diabetes Society has now recommended an OGTT in all subjects with IFG classified as 5.5–6.9 mmol/L.\(^13\) Recently, the New Zealand (NZ) Guideline Group (NZGG) has published guidelines for the management of type 2 diabetes\(^14\) in New Zealand. They recommend an OGTT for all patients with a fasting glucose of 6.1–6.9 mmol/L inclusive (previous IFG limits)—but for those with a fasting glucose between 5.5–6.0 mmol/L, only if the patient is not of European origin, has a family history of diabetes, or has “features of the metabolic syndrome.”

The NZ Guideline Group has stated that “a fasting glucose below 5.5 mmol/L is normal” (Management of Diabetes Guidelines Page 2), but has at the same time avoided extending the range of impaired fasting glucose below 6.1 mmol/L.

Therefore patients with fasting glucose in the 5.5–6.0 mmol/L range currently are officially classed as neither normal nor IFG in New Zealand. Furthermore, unlike Australia where an oral GTT is recommended in all such patients, many who do not meet the additional risk criteria stated above, live in a diagnostic “no man’s land” because an oral GTT is considered unwarranted.

The prevalence of IGT or diabetes in patients in New Zealand with a fasting glucose between 5.5–6.0 mmol/L is unknown. Furthermore, the metabolic characteristics of these patients are not described. This study thus aims to describe the metabolic features of patients with fasting glucose below 6.1 mmol/L.

**Methods**

Non-pregnant consecutive patients—referred by their GP (reasons for referral not recorded) between July 2002 and December 2003 for 75 g OGTT to 8 Diagnostic Medlab collection depots spread across the wider geographical area of Auckland (Orewa to Manurewa)—were invited to participate.

Height and weight were measured and BMI was calculated. Ethnicity was self-declared. A standard 75 g OGTT was then carried out. Fasting samples for lipids, HbA\(_1c\), fructosamine, and insulin in addition to glucose were taken at baseline. Glucose and insulin concentrations were measured at 60 and 120 minutes after glucose ingestion. Samples for glucose were collected in fluoride oxalate tubes and serum for insulin frozen at -20°C for later analysis in one batch.

Serum lipids, glucose, fructosamine and HbA\(_1c\) were analysed on the day of collection in one central laboratory. Insulin was measured on an Abbott Imx (normal range 5–13 mLU/L CV 5.3%), HbA\(_1c\) by cation exchange HPLC (Biorad Variant 2 normal range 4–6% CV 2%) and fructosamine on a Roche Hitachi Modular system (normal range 180–300 umol/L CV 4%).
Estimates of insulin resistance were made from clinical measurements (BMI) as well as serum cholesterol/HDL ratio and fasting triglyceride, fasting insulin (higher values imply insulin resistance), insulin/glucose ratio (higher values imply insulin resistance), and mathematical indices using homeostasis model assessment (HOMA-IR – higher values imply more insulin resistance\(^1\))\(^5\), the quantitative sensitivity check index (QUICKI\(^1\) – a lower score implies more insulin resistance) and McAuley score (lower values imply insulin resistance and scores<6.3 MmU\(^{-1}\)/L defines insulin resistance),\(^1\)\(^7\) which correlate with hyperinsulinemic euglycaemic clamp studies.\(^1\)\(^8\),\(^1\)\(^9\) HOMA was calculated as glucose × insulin/22.5 and QUICKI as 1/log (fasting insulin) + log (fasting glucose). Diabetes was defined as a fasting glucose ≥7.0 and/or 2 hr OGTT ≥11.1 mmol/L; IGT as fasting glucose <7.0 and 2 hour glucose of 7.8–11.0 mmol/L; IFG in 2 categories as fasting glucose 5.5–6.0 mmol/L (“high fives”), or 6.1–6.9 mmol/L (“old IFG”) and 2 hour glucose <7.8 mmol/L . Normal glucose tolerance was defined as a fasting glucose of <5.5 and 2 hour value <7.8 mmol/L.

The study was approved by the Auckland Regional Ethics Committee.

Comparisons between groups were made using analysis of variance (ANOVA, proc GLM, SAS Institute Inc, v9.1 software). Should the main effect reach statistical significance, the post hoc procedure of Dunnett was used to compare each group against normal. All tests were two-tailed and p<0.05 was considered significant.

**Results**

310 patients, not previously known to have diabetes, agreed to participate (90% of those asked). Table 1 displays the patients’ details. 244 patients (79%) were European, 41 (13%) Maori, 12 (4%) Pacific, and 13 (4.2%) Asian. 72 patients (23%) were classified as having diabetes and another 74 patients (24%) as IGT following the OGTT.

The 2-hour GTT result (normal, IGT, or diabetes) is compared with the fasting glucose result (normal, “high fives”, “old” IFG, diabetes) in Table 2. Whilst similar numbers of patients with a fasting glucose of <5.5 mmol/L (normal) and 5.5–6.0 mmol/L (“high fives”) actually had IGT on OGTT (21%), significantly more patients with “new” IFG had diabetes (13% vs 2%) \(p\leq0.005\). Seventy-two percent of patients with a fasting glucose of 6.1–6.9 mmol/L (“old” IFG) had IGT (44%) or diabetes (28%) on OGTT and, as expected, almost all (97%) with diabetic range fasting glucose (≥7 mmol/L) did indeed have diabetes based on 2-hour OGTT result.

Measures of insulin resistance are shown in Table 1. Patients with fasting glucose between 6.1–6.9 mmol/L (“old” IFG) had many features of insulin resistance when compared with patients with a glucose <5.5 mmol/L with patients with fasting glucose of 5.5–6.0 mmol/L having intermediate features.

The sensitivity, specificity, and positive and negative predictive value of diagnosing diabetes using a fasting glucose with the lower IFG category of 5.5–6.0 compared with current IFG category of 6.1–6.9 mmol/L (as compared to OGTT) is shown in Table 3.

**Discussion**

This study demonstrates that in patients referred for OGTT, a significant minority have either IGT or diabetes when the fasting glucose is below the current IFG threshold (5.5–6.1 mmol/L). Furthermore, many of these patients have features of the metabolic syndrome (lower McAuley score and higher HOMA – IR value then those with glucose <5.5 mmol/L) thus supporting the rationale to proceed to OGTT in all patients with a fasting glucose of 5.5–6.9 mmol/L inclusive, and not just for those with a fasting glucose of 6.1–6.9 mmol/L.
Table 1. Characteristics of 310 patients previously not known to be diabetic undergoing 75 g OGTT, classified according to fasting glucose

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal &lt;5.5 mmol/L</th>
<th>Impaired fasting glucose (“high fives”) 5.5–6.0 mmol/L</th>
<th>Impaired fasting glucose (“old IFG”) 6.1–6.9 mmol/L</th>
<th>Diabetes ≥7.0 mmol/L</th>
<th>ANOVA Main Effect p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>52.5±15.6</td>
<td>55.6±12.6</td>
<td>59.0±11.0*</td>
<td>55.0±16.0</td>
<td>0.016</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.2±6.0</td>
<td>30.5±5.7</td>
<td>30.3±5.6</td>
<td>31.7±6.0</td>
<td>0.11</td>
</tr>
<tr>
<td>% male</td>
<td>47.8</td>
<td>54.1</td>
<td>57.3</td>
<td>59</td>
<td>0.50</td>
</tr>
<tr>
<td>Plasma glucose (mmol/L)</td>
<td>5.0±0.3</td>
<td>5.7±0.2 *</td>
<td>6.4±0.2*</td>
<td>9.5±2.9*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HbA₁c %</td>
<td>5.7±0.4</td>
<td>5.8±0.4</td>
<td>6.2±0.6*</td>
<td>7.8±1.8*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Fructosamine (µmol/L)</td>
<td>224±19</td>
<td>229±32</td>
<td>239±26*</td>
<td>306±78*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>AUC insulin during OGTT</td>
<td>135±115</td>
<td>170±131</td>
<td>187±155*</td>
<td>118±99</td>
<td>0.01</td>
</tr>
<tr>
<td>Triglyceride mmol/L</td>
<td>1.7±0.9</td>
<td>2.0±1.5</td>
<td>2.2±1.7</td>
<td>2.1±1.2</td>
<td>0.07</td>
</tr>
<tr>
<td>Total cholesterol / HDL</td>
<td>4.0±1.2</td>
<td>4.3±1.1</td>
<td>4.3±1.2</td>
<td>4.7±1.3*</td>
<td>0.48</td>
</tr>
<tr>
<td>Fasting insulin mu/L</td>
<td>10.7±8.5</td>
<td>13.0±9.8</td>
<td>18.0±20.0*</td>
<td>18.0±16.0*</td>
<td>0.0004</td>
</tr>
<tr>
<td>HOMA</td>
<td>2.4±2.0</td>
<td>3.3± 2.5</td>
<td>5.4±6.1*</td>
<td>7.2±6.1*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>QUICKI</td>
<td>0.28±0.08</td>
<td>0.25±0.06</td>
<td>0.23±0.04*</td>
<td>0.21±0.03*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>McAuley score</td>
<td>7.05±2.13</td>
<td>6.39±1.95</td>
<td>5.86±1.81</td>
<td>5.79±1.91</td>
<td>&lt;0.0002</td>
</tr>
<tr>
<td>% McAuley score &lt;&lt;6.3</td>
<td>43.1</td>
<td>57.1</td>
<td>19.6</td>
<td>73</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>% BMI &gt;30 kg/m²</td>
<td>41.8</td>
<td>51.2</td>
<td>41.9</td>
<td>59</td>
<td>0.19</td>
</tr>
<tr>
<td>% triglyceride &gt;1.5 mmol/L</td>
<td>44.1</td>
<td>48.2</td>
<td>62.2</td>
<td>64.1</td>
<td>0.36</td>
</tr>
<tr>
<td>% HDL &lt;1.0 mmol/L</td>
<td>7.2</td>
<td>10.6</td>
<td>6.9</td>
<td>18</td>
<td>0.2</td>
</tr>
</tbody>
</table>

*Significant difference from normal (Dunnett’s test p<0.05). Data are mean±SD.
Table 2. Percentage of patients (n=310) with 2-hour OGTT result classified as normal (<7.8 mmol/L), impaired glucose tolerance (7.8–11.0 mmol/L) or diabetes (≥11.1 mmol/L)—compared to fasting glucose classification result (normal <5.5 mmol/L, “high fives” 5.5–6.0 mmol/L; “old IFG” 6.1–6.9 mmol/L and diabetes≥7.0 mmol/L)

<table>
<thead>
<tr>
<th>Variable</th>
<th>2-hour OGTT glucose result</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>IGT</td>
<td>Diabetes</td>
</tr>
<tr>
<td></td>
<td>n=164</td>
<td>n=74</td>
<td>n=72</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal (n=111)</td>
<td>77%</td>
<td>21%</td>
<td>2%</td>
</tr>
<tr>
<td>“New” IFG (n=85)</td>
<td>66%</td>
<td>21%</td>
<td>13%</td>
</tr>
<tr>
<td>“Old” IFG (n=75)</td>
<td>28%</td>
<td>44%</td>
<td>28%</td>
</tr>
<tr>
<td>Diabetes (n=39)</td>
<td>3%</td>
<td>0%</td>
<td>97%</td>
</tr>
</tbody>
</table>

Table 3. Sensitivity, specificity, and positive (PPV) and negative (NPV) predictive value (95% CI) for diagnosing diabetes on subsequent OGTT by fasting glucose alone (mmol/L)

<table>
<thead>
<tr>
<th>Fasting glucose (mmol/L)</th>
<th>Sensitivity (95% CI)</th>
<th>Specificity (95% CI)</th>
<th>PPV (95% CI)</th>
<th>NPV (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.5–6.1</td>
<td>75.8 (65.9–84.0)</td>
<td>87.1 (79.0–93.0)</td>
<td>84.7 (75.3–91.9)</td>
<td>79.3 (70.3–86.0)</td>
</tr>
<tr>
<td>6.1–6.9</td>
<td>44.1 (36.7–51.9)</td>
<td>99.0 (93.0–100)</td>
<td>98.7 (93.0–100)</td>
<td>51.5 (44.3–58.7)</td>
</tr>
</tbody>
</table>

In the UKPDS study, diabetes was diagnosed on the basis of symptoms in only 54% of patients. Of more concern, more than half of those patients had established microvascular complications of diabetes at diagnosis suggesting a period of 5 – 8 years of preceding undiagnosed diabetes. Those with the lowest fasting glucose at diagnosis had the lowest prevalence of microvascular complications at diagnosis and furthermore had a lower rate of progression of complications at follow up suggesting that early diagnosis of diabetes improves outcome.

Detecting patients early, during the IGT (or ‘pre-diabetes’) stage, is also worthwhile. Intervention studies confirm that the progression of patients with IGT to diabetes can be slowed significantly by combination of diet, exercise, and metformin use. Those patients with IGT who had the lowest fasting glucose result had the greatest success in preventing diabetes in these programmes. These data suggest that there is a continuum of fasting glucose results wherein patients progress from normal towards diabetes during which time intervention can delay or prevent the progression to diabetes or the development of microvascular complications.

Detecting these patients at an early stage relies on the development of simple screening algorithms that have high sensitivity and specificity primarily for the detection of diabetes, but which will (as a consequence) also detect those who are also clearly at risk with ‘pre-diabetes’.

The recent Australian AusDiab study used a protocol of performing a fasting glucose test in patients with one or more risk factors for diabetes (age >55 years, or >45 years if obese or hypertensive or a family history of diabetes, non European, established cardiovascular disease, women with previous gestational diabetes or
polycystic ovarian syndrome), then proceeding to OGTT if the fasting glucose result was between 5.5–6.9 mmol/L. This study had 80% sensitivity and specificity for the detection of previously undiagnosed type 2 diabetes but also detected many patients with pre-diabetes as well who may potentially benefit from lifestyle change.

Sensitivity for diabetes detection improved from 64% to 80% by lowering the cutoff from 6.1 to 5.5 mmol/L as the threshold for proceeding to OGTT, although there was a reduction in specificity from 94% to 80%. The pickup rate for IGT/IFG also increased from 35% to 52%.

The current reported study demonstrates that patients with apparently normal fasting glucose (5.5–6.0 mmol/L) have significantly higher rates of dysglycaemia compared with those patients with a fasting glucose of <5.5 mmol/L and that these patients tended to have more features of insulin resistance (Table 1).

It is acknowledged that the subjects of the current study were not randomly chosen for OGTT and the pre-test probability of diabetes in this group was presumably reasonably high for their doctor to have requested the test in the first place. Nevertheless, this study does provide evidence that relying on a fasting glucose alone will misclassify a significant number of patients with dysglycaemia on OGTT as ‘normal’, based on current fasting thresholds.

Taken together, these data suggest that the upper limit of normal fasting glucose in New Zealand should be considered to be <5.5 mmol/L in line with recent Australian recommendations. Screening for diabetes should continue to use the fasting glucose as the first step but all patients with a fasting glucose of 5.5–6.9 mmol/L should proceed to OGTT testing, also in line with Australian Diabetes Society guidelines.

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**Acknowledgement:** This study was supported by the ELI LILLY GRANT 2003 administered through New Zealand Society for the Study of Diabetes. Additional support from Roche diagnostics and Diagnostic Medlab is gratefully acknowledged.

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**References:**


Abstract

Aims Rheumatoid arthritis is associated with an excess mortality from cardiovascular disease, and this may be related to an atherogenic lipid profile. We set out to identify whether there was a correlation between disease activity and levels of different lipid fractions in a stable population of patients with rheumatoid arthritis on disease-modifying therapy.

Methods Patients with rheumatoid arthritis were selected from our database and requested to have inflammatory markers and a fasting lipid profile taken at their next visit for monitoring of their disease modifying therapy.

Results 204 patients were recruited for the study. A statistically significant relationship exists between reduced HDL and elevated CRP (p=0.01) and ESR (p=0.041). Elevated ESR, but not elevated CRP, was associated with raised LDL cholesterol (p=0.014). Fourteen patients (6.8%) were receiving statin therapy and 71 (34.8%) were taking prednisone. Use of prednisone, independent of dose, was associated with elevated triglyceride levels (p=0.041).

Conclusions This study supports previous work showing that rheumatoid arthritis is associated with an adverse lipid profile. While good disease control is clearly important, we should not neglect management of traditional cardiovascular risk factors.

Patients with rheumatoid arthritis have an increased mortality from cardiovascular disease, and untreated patients have an atherogenic lipid profile which can be positively influenced by the use of disease-modifying antirheumatic drugs (DMARD) therapy. There is, however, some doubt as to the significance of these changes—since it has been shown previously that the increased cardiovascular risk is not completely explained by traditional risk factors.

Interestingly, use of glucocorticoids which are known to cause hypercholesterolaemia in this population, has also been shown to increase high-density lipoprotein (HDL) and reduce the total cholesterol/HDL ratio, thus suggesting a causative role for the inflammatory response in generating this lipid abnormality. Previous studies have been hampered by analysis of stored specimens for lipid analysis which can falsely reduce HDL levels.

This study was designed to investigate whether—in our population of patients with stable rheumatoid arthritis on disease-modifying therapy—there was an association between inflammatory markers, glucocorticoid-use, and lipid levels using specimens analysed on the same day as collection.

Methods

Our database of patients currently taking disease-modifying therapy was searched; patients were invited to participate in the study if they had rheumatoid arthritis and had been on disease-modifying therapy for at least 6 months.
therapy for at least 3 months. Fasting lipid profile, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) were performed with the next routine blood monitoring for their DMARD therapy. DMARD-use was recorded along with any concurrent use of prednisone and lipid-lowering therapy.

The data was analysed using Microsoft Excel software to assess the presence and strength of any association between disease activity and different lipid fractions.

**Results**

300 patients were invited to participate initially, and positive responses were received from 204 patients within the 3.5 month period allotted for data collection, thus giving a response rate of 68%. Forty-six (22%) were male with an average age of 58.63 years (range 34 to 87 years). 159 (78%) of the patients were female with an average age of 59.61 years (range 22 to 90 years).

Seventy-one (34.8%) of patients were taking prednisone in doses ranging from 2 mg to 40 mg, and 14 (6.8%) were receiving lipid-lowering therapy with an HMG-CoA reductase inhibitor and another two (0.98%) with a fibrate.

Mono-therapy with methotrexate was used in 92 (45%) of patients, with a further 43 (21%) using leflunomide. Combination therapy (methotrexate and leflunomide) was used in 23 patients (11%), whilst single therapy with sulphasalazine was found in 15 (7%) of patients. The remaining 31 patients were on various regimes (either alone or in combination) using hydroxychloroquine, gold, azathioprine, and penicillamine. Small sample sizes for many of these groups prevented statistical analysis.

Suggested optimal lipid levels from the latest New Zealand Guidelines Group publication on assessment of cardiovascular risk were used for this analysis; these levels are presented in Table 1 along with the numbers of patients who did not meet these suggested criteria.

**Table 1. Optimal lipid levels suggested by the New Zealand Guideline Group, and numbers of patients not meeting this recommendation**

<table>
<thead>
<tr>
<th>Lipid fraction</th>
<th>Optimal value</th>
<th>Number of patients not meeting this recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol (TC)</td>
<td>&lt;4.0 mmol/L</td>
<td>191 (93.6%)</td>
</tr>
<tr>
<td>LDL Cholesterol</td>
<td>&lt;2.5 mmol/L</td>
<td>152 (74.5%)</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>&gt;1.0 mmol/L</td>
<td>21 (10.3%)</td>
</tr>
<tr>
<td>TC:HDL ratio</td>
<td>&lt;4.5</td>
<td>59 (28.9%)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>&lt;1.7 mmol/L</td>
<td>78 (38.2%)</td>
</tr>
</tbody>
</table>

LDL=low-density lipoprotein; HDL=high-density lipoprotein.

Associations between inflammatory markers and different lipid fractions were assessed using the Chi-squared test, and the p values obtained are shown in Table 2. Insufficient patients were on lipid-lowering therapy to permit statistical analysis of statin therapy with regard to inflammatory markers.

A statistically significant relationship is demonstrated between elevated ESR and raised LDL (p=0.041). Reduced levels of HDL was associated with raised ESR (p=0.041) and CRP (p=0.01). In addition, being on prednisone (independent of dose)
was associated with a statistically significant increase in triglyceride concentrations (p=0.041).

Table 2. P values for Chi-squared tests between lipid fractions & inflammatory markers and prednisone

<table>
<thead>
<tr>
<th>Lipid fractions &amp; inflammatory markers</th>
<th>ESR&gt;20</th>
<th>CRP&gt;5</th>
<th>On prednisone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol &gt;4.0</td>
<td>0.264</td>
<td>0.306</td>
<td>0.375</td>
</tr>
<tr>
<td>LDL &gt; 2.5</td>
<td><strong>0.014</strong></td>
<td>0.469</td>
<td>0.838</td>
</tr>
<tr>
<td>TC:HDL &gt;4.5</td>
<td>0.076</td>
<td>0.155</td>
<td>0.075</td>
</tr>
<tr>
<td>HDL &lt;1.0</td>
<td><strong>0.041</strong></td>
<td><strong>0.010</strong></td>
<td>0.283</td>
</tr>
<tr>
<td>Triglycerides &gt;1.7</td>
<td>0.512</td>
<td>0.630</td>
<td><strong>0.041</strong></td>
</tr>
<tr>
<td>ESR&gt;20</td>
<td>–</td>
<td>&lt;<strong>0.00001</strong></td>
<td>0.728</td>
</tr>
<tr>
<td>CRP &gt;5</td>
<td>–</td>
<td>–</td>
<td>0.762</td>
</tr>
</tbody>
</table>

Significant associations are shown in **bold**: CRP=C-reactive protein; ESR=erythrocyte sedimentation rate; LDL=low-density lipoprotein; HDL=high-density lipoprotein; TC=total cholesterol.

Discussion

We have demonstrated a statistically significant association between raised ESR & CRP and reduced HDL cholesterol in this population as well as confirming the results of a previous study, which showed a relationship between use of prednisone and increased triglyceride levels.

Elevated ESR is associated with statistically significant alterations in HDL and LDL cholesterol levels, but this is not the case for elevated CRP where a relationship was only seen with reduced HDL levels. This most likely reflects the fact that ESR and CRP are independent variables. CRP has been shown to reflect the acute phase response more closely than ESR in patients with rheumatoid arthritis because elevations in the ESR can be created by high titres of rheumatoid factor and immunoglobulins which may not rise acutely.6

This study is limited by the fact that we did not control for patients’ weight, menopausal status, and other comorbidities which are known to affect lipid profiles.7 In addition, no allowance was made for disease duration apart from the fact that patients had stable disease for at least 3 months. Unfortunately, small patient numbers prevented the analysis of any effect of lipid-lowering therapy on inflammatory markers as well as analysis of different DMARD sub-groups with regard to lipid levels and inflammatory markers.

Recent data has shown that better disease control (leading to improvement in quality of life and reduced radiographic progression of disease) can be achieved using intensive outpatient treatment at no additional cost.8 In addition, use of methotrexate has shown a reduced mortality mainly as a result of a reduction in cardiovascular deaths.9

Therefore, good control of disease should be the priority given that both quality of life and longer-term outcomes can be improved. Nevertheless, rheumatoid arthritis patients appear to have a high prevalence of abnormal blood lipid profiles—and given
that so few of our patients were receiving lipid-lowering therapy, it is clear that we are not managing traditional risk factors for cardiovascular disease as part of our routine care.

Data from the trial of atorvastatin in rheumatoid arthritis\textsuperscript{10} demonstrated that patients who received atorvastatin 40 mg had improvement in disease severity scores, swollen joint counts, and inflammatory markers with no increase in adverse events compared to placebo.

Whilst the routine use of statins as disease-modifying therapy for patients with rheumatoid arthritis is not yet routine practice, their use in selected patients with abnormal lipid profiles could also benefit their arthritis.

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**References:**

Botulinum toxin type-A (Botox-A) injections for treatment of sialorrhoea in adults: a New Zealand study

Subhaschandra Shetty, Patrick Dawes, Dean Ruske, Mohannad Al-qudah, Brett Lyons

Abstract

Aim We report our initial experience using Botox-A injection to the salivary glands to control sialorrhoea.

Methods Eight adult patients with significant sialorrhoea were referred from an inpatient rehabilitation unit, GP referral, and internal medicine department. All subjects underwent bilateral submandibular gland injections and, in addition, one patient (the first) also had intraparotid injections. Injections were performed with ultrasound guidance at Dunedin Hospital, New Zealand. Six patients received a total of 30 Units and two patients received 60 Units in the submandibular glands without any complications. Outcome was assessed using a drooling scale and VAS self report of sialorrhoea.

Results Of the eight patients treated, six reported a marked reduction in salivation following treatment, and one patient improved partially. One patient did not find the Botox injection helpful in controlling sialorrhoea and was offered escalation of the Botox dose with a subsequent partial response. No serious adverse events occurred, and no procedure-related complications were noted.

Conclusions Our initial experience suggests that injection of Botox-A injected at a relatively low dose to the submandibular glands can be used to achieve desired results for the treatment of sialorrhoea. This is an easily performed procedure with low morbidity and can be recommended as a first-line intervention in the treatment of adult sialorrhoea.

Sialorrhoea is an important clinical, social, and emotional issue, which contributes to poor quality of life and carer burden. It is associated with a wide range of disorders, as shown in Table 1.

Salivary gland secretion is controlled by the autonomic nervous system, mediated by adrenergic and cholinergic nerve endings, but primarily under parasympathetic cholinergic control. The major salivary glands (the paired parotid, submandibular, and sublingual) are responsible 95% of the 1.5 L of saliva secreted daily. In the unstimulated (basal) state, 70% of saliva is secreted by the submandibular salivary glands.

Sialorrhoea is defined as saliva emanating beyond the lip margins. The physical and psychosocial complications (associated with sialorrhoea) range from mild and inconvenient symptoms to severe problems that can have a significant negative impact on quality of life.

Various treatments are described for sialorrhoea, each having their own side effects. Systemic anticholinergics are often ineffective, produce blurred vision, urinary retention, and in 40% of cases, cardiac arrhythmia.
Surgical intervention may involve either rerouting of the submandibular ducts, excision of salivary tissue, or in uncommon situations (when voice function is lost) laryngectomy.5

Each surgery carries complications both specific to the procedure as well as to the general condition of the patient. Radiotherapy has been used to reduce salivary gland function. This is time consuming, and is associated with mucositis during treatment as well as injury to minor salivary glands caught in the treatment field, so that the end result may be less predictable—resulting in xerostomia.6

Table 1. Aetiology of sialorrhoea

<table>
<thead>
<tr>
<th>Neuromuscular/sensory dysfunction</th>
<th>Hypersecretion†</th>
<th>Anatomic‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental retardation</td>
<td>Inflammation (teething, dental caries, oral-cavity infection, rabies)</td>
<td>Macroglossia</td>
</tr>
<tr>
<td>Cerebral palsy</td>
<td>Drug side effects (tranquillizers, anticonvulsants)</td>
<td>Oral incompetence</td>
</tr>
<tr>
<td>Parkinson's disease</td>
<td>Gastro-oesophageal reflux</td>
<td>Malocclusions</td>
</tr>
<tr>
<td>Pseudobulbar*</td>
<td>Toxins (e.g. mercury)</td>
<td>Orthodontic condition</td>
</tr>
<tr>
<td>Bulbar palsy*</td>
<td></td>
<td>Head and neck (H&amp;N) surgical defect</td>
</tr>
<tr>
<td>Stroke*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Less common; † Usually controlled by increased swallowing; ‡ Frequently exacerbate existing problems.

Recently, *Botulinum* toxin-A (Botox-A) intraglandular application has been shown to significantly decrease saliva production and is considered a safe treatment.7,8 Botox-A acts to inhibit salivary production by binding to SNAP-25 (25 kDa synaptosome-associated protein), a cytoplasmic protein involved in the fusion of synaptic vesicles with the presynaptic membrane. This ultimately disrupts the secretory pathway for acetylcholine and produces a chemodenervation.10 Gradual re-innervation occurs as the neurones regenerate SNAP-25.

The Botox-A injection procedure has its own complications—they range from minor (bleeding, pain at site, flu-like symptoms, parotitis, and dry mouth) to severe (dysphagia, aspiration, facial nerve branch palsy, temporomandibular joint [TMJ] dislocation, and vascular injuries)

We present our personal experience treating eight adults with disabling sialorrhoea using Botox-A injections. We used low dosages and favoured bilateral injection of the submandibular glands. Limiting injection to submandibular glands aimed to minimise morbidity associated with xerostomia.
Methods

Patients—Eight adult patients with drooling (attributable to head and neck carcinoma, neurodegenerative diseases, quadriplegia, or idiopathic salivation) were treated by injection of Botox-A into the submandibular glands (and parotid glands in one case) at Dunedin Hospital in New Zealand. Patient demographics and clinical data are shown in Table 2.

Table 2. Demographics and clinical details

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>Duration of sialorrhoea</th>
<th>Total Botox-A dose (IU)</th>
<th>Improvement following injection (Global VAS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>84/M</td>
<td>Idiopathic sialorrhoea</td>
<td>4 years</td>
<td>30</td>
<td>Marked</td>
</tr>
<tr>
<td>2</td>
<td>50/M</td>
<td>Quadriplegia</td>
<td>3 years</td>
<td>30</td>
<td>Marked</td>
</tr>
<tr>
<td>3</td>
<td>35/M</td>
<td>Neurodegenerative + Quadriplegia</td>
<td>1 year</td>
<td>30</td>
<td>Marked (Patient died 5 months following injection due to cardiac disease)</td>
</tr>
<tr>
<td>4</td>
<td>74/M</td>
<td>Pharyngocutaneous fistula + Idiopathic sialorrhoea</td>
<td>11 months</td>
<td>30</td>
<td>Mild</td>
</tr>
<tr>
<td>5</td>
<td>77/F</td>
<td>Idiopathic hypersalivation</td>
<td>2 years</td>
<td>30</td>
<td>Marked</td>
</tr>
<tr>
<td>6</td>
<td>64/M</td>
<td>Parkinson’s disease</td>
<td>3 years</td>
<td>45</td>
<td>No change</td>
</tr>
<tr>
<td>7</td>
<td>42/M</td>
<td>Quadriplegia</td>
<td>2 years</td>
<td>30</td>
<td>Marked change</td>
</tr>
<tr>
<td>8</td>
<td>69/M</td>
<td>Lateral medullary syndrome</td>
<td>1 year</td>
<td>30</td>
<td>Marked change</td>
</tr>
</tbody>
</table>

All patients were informed of the details of therapy and its possible side effects and gave their written consent to undergo treatment and present themselves regularly for follow-up examinations.

During the first outpatient visit, the symptom ‘drooling’ was scored by means of the ‘questionnaire-based semi-quantitative assessment of drooling severity and frequency’ as shown in Table 3 and a VAS estimate of drooling severity.

Table 3. System for assessment of frequency and severity of drooling*

<table>
<thead>
<tr>
<th>Drooling Severity</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry (never drools)</td>
<td>1</td>
</tr>
<tr>
<td>Mild (wet lips only)</td>
<td>2</td>
</tr>
<tr>
<td>Moderate (wet lips and chin)</td>
<td>3</td>
</tr>
<tr>
<td>Severe (clothing becomes damp)</td>
<td>4</td>
</tr>
<tr>
<td>Profuse (clothing, hands, tray, objects become wet)</td>
<td>5</td>
</tr>
</tbody>
</table>

**Adapted from Thomas Stone et al, Dysphagia, 1998:3:5.**

The VAS scale introduced in this series (before and after injection) was a 100 mm horizontal line marked from 0 (normal salivation) up to 100 (worst possible drooling). Patients marked the line at the point they felt represented their current salivation state or rate.

All patients had high severity scores reflecting that clothing, hands, tray, and objects become wet because of drooled saliva. The frequency scores showed that all patients had frequent to constant drooling scores.
Injection technique—Using high-resolution ultrasound, the vascular anatomy, gland location, and size of the submandibular glands were carefully assessed immediately prior to injection. No anaesthesia was used, and the treatment was usually well tolerated. Botox-A® (ALLERGAN, New Zealand) 100 Mouse Units (U) was reconstituted with 0.9% sodium chloride solution to 100 U/4ml.

In the majority of the patients, 15 U (0.6 ml) of Botox-A was injected into each submandibular gland and, in one case, 7.5 U to both parotids. After defining the gland and its blood supply, the ultrasound transducer was positioned in such a way that injections with the needle were possible along the axis of the transducer, thus providing quick and easy visualisation of the needle entering the gland.

The injections were administered after clearly visualising the central part of the parenchyma of submandibular glands in real time mode with avoidance of blood vessels.

All patients were monitored for 1 hour post-injection. Side effects and procedure-related complications were carefully monitored; only pain at the injection site was noted in one patient. In the follow-up period (at 6 weeks and 6 months), all patients were examined and the scores were repeated; global VAS scores were also noted.

Efficacy and Results:

Major side effects were not seen even after repeated doses; in particular neither xerostomia nor dysphagia was seen. The risk for these potential adverse effects may be diminished but certainly cannot be excluded by our limiting the dose and number of treated glands.

Reduction in salivation was first noted 3 days (mean 5 days) after the injection. Seven patients reported substantial reduction of salivation and drooling at their first visit at 6 weeks after injection and the effect was maintained at 6 months in 5 patients—as shown in Figure 1 and Figure 2. One patient (#3) died from cardiac disease 5 months after the Botox-A injection; he had remained symptom-free till death. (His family confirmed his salivation had remained controlled until he died.)

Figure 1. Patients assessment of the level of sialorrhoea, as determined by VAS, at baseline and the point of maximum benefit after Botox-A injection

<table>
<thead>
<tr>
<th>Mean VAS Scores</th>
<th>Median VAS Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>80</td>
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<tr>
<td>80</td>
<td>60</td>
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<td>60</td>
<td>40</td>
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<tr>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>20</td>
<td>0</td>
</tr>
</tbody>
</table>

1=Baseline 2=At 6-Weeks 3= At 6-Months
Figure 2. Median drooling scores at follow-up periods following Botox injection compared to baseline (pre-injection)

Note: Patient #3 died at 5 months.

One patient, with Parkinson’s disease (patient #6) experienced no improvement after the first injection and proceeded to a second injection with higher dose [30 U], again with little perceived benefit. Patient #4, who had a pharyngocutaneous fistula, showed an initial response for 12 weeks after which his symptoms recurred. He has received further injections at 6 months [30 U/submandibular gland] and is still in follow-up showing gradual improvement in his salivation rate.

Unwanted side effects were assessed at visit 2 and 3 using a 0–4 scale (0=no adverse events; 1=mild or brief; 2=moderate; 3=severe and brief; 4=severe, disabling and long-standing). We asked about dysphagia, chewing and breathing difficulties, and generalised weakness. No serious side effects or procedure-related complications occurred during the treatment. One patient complained of injection site pain, which was considered mild and was controlled with paracetamol.

Discussion

This series represents the preliminary experience gained at Dunedin Hospital with intraglandular injection of Botulinum Toxin-A to reduce drooling in various disorders. Six out of eight patients reported excellent control of drooling.

Early reports describing the use of Botulinum toxin in the management of sialorrhoea described intraparotid injection, with the reduction in salivation lasting between 4 and 7 months. Overall, the duration of effect is longer than following intramuscular injection of Botulinum toxin, although there is both intra-individual and inter-individual variation.
More recently, Jongius et al\textsuperscript{12} have recommended injection into the submandibular glands only; their proposal being based on the consideration that the submandibular salivary glands are responsible for 60–70\% of basal salivary production. The theoretical benefit being that reducing basal secretion will counteract drooling while allowing secretion from the parotid glands during meals—thus maintaining lubrication for chewing and swallowing as well as reducing the risk of gingivitis, caries, and secondary ascending parotitis.

The reported total dose of Botox-A administered varies from 10 U to 100 U.\textsuperscript{2,4,11,12} A prospective, double blind, randomised trial has evaluated the efficacy of three different dosages (18.75, 37.5, and 75 U per parotid gland) of intraglandular injection of Botox-A for patients with sialorrhoea. Good control of salivation was achieved with 75 U of Botox-A per side, suggesting this is safe and effective treatment for patients with sialorrhoea.\textsuperscript{13}

A recent placebo-controlled study investigated the highest dosage of Botox-A ever used (450 U) for sialorrhoea; injection was made in to the parotid and submandibular glands and reduced salivation was described for approximately 1 month.\textsuperscript{14} Pal et al have suggested that accuracy when placing the toxin within the salivary glands plays an important role in dosages required and results.\textsuperscript{15}

We used relatively low dosages of Botox-A (15 U to each gland) and achieved significant effects using an ultrasound-guided technique. Effective low doses of Botox-A may help to prevent development of neutralising antibodies, which is a potential problem in 5–10\% of treated patients. An interval of less than 3 months between treatments and high doses of Botox-A are considered risk factors for the development of immunoresistance.\textsuperscript{16}

In late 2004, there was a news report of complications following the use of Botox A for managing sialorrhoea in children, the injections having been administered at Starship Hospital (Auckland, New Zealand).\textsuperscript{17} Specifically, extracapsular spread of the drug had caused paralysis of the pharyngeal muscles resulting in dysphagia for three children. Communication with the ORL-HNS service at Starship indicates that ultrasound was used to identify the submandibular glands; the factors thought most likely to have contributed to the dysphagia were underlying swallowing dysfunction related to cerebral palsy and extravasation of Botox beyond the submandibular gland capsule exacerbated by the use of a larger volume of diluted Botox (Botox-A 100MU diluted in 5 ml saline – 2 ml to each gland). Guidelines have since been developed—Starship now use 100 MU diluted in 1 ml saline, and they inject 30 MU (0.3 ml) to each gland.\textsuperscript{18,19}

Recent literature strongly recommends the use of ultrasound to guide the injection of Botox-A to salivary glands.\textsuperscript{1,4,9,11–13,15} In our experience, using ultrasound, with real-time needle positioning during infiltration, has made the treatment more precise and helped to avoid inadvertent migration of the toxin. Indeed, this single ultrasound-guided injection not only prevents the morbidity associated with multiple site injection, it is essential for targeting the correct region (intraparenchymal) of the salivary tissue.
Conclusion

The injection of Botox-A into the submandibular glands is an easily administered, effective treatment for adult drooling, regardless of the underlying cause of the condition; it has few side effects. The use of ultrasound guidance during the injection procedure may enhance efficacy and safety.

Although our results are encouraging, a full clinical trial would be needed to formally evaluate the most effective dosage, risks, and the benefits of Botox-A for palliative treatment of sialorrhoea. Indeed, Botox-A may prove to be a dependable therapeutic option for patients with sialorrhoea.

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References:


Caecal volvulus associated with intestinal malrotation immediately following caesarean section

Anthony Marren, Kenneth Wong

Intestinal obstruction complicates 1 in 2500 pregnancies.\(^1\) Colonic volvulus is the second most common cause, after adhesions.\(^1\) Most commonly, colonic volvulus occurs prior to the onset of labour.\(^1\) Less than 15 cases of colonic volvulus following caesarean section have been reported.

We present a case of caecal volvulus associated with previously unsuspected intestinal malrotation diagnosed shortly after caesarean section. Given the rising incidence of caesarean sections,\(^2\) it is important to be aware of this remediable condition—as delayed diagnosis is associated with a high maternal and fetal mortality rate, due to large bowel perforation.\(^1,2\)

Case report

A 29-year-old gravida 2 Caucasian woman at 37 weeks’ gestation presented with a 2-day history of abdominal pain, nausea, and vomiting to Gosford Hospital, New South Wales (NSW), Australia. She was afebrile and haemodynamically stable. The abdomen was soft, with a single fetus presenting cephalically.

The patient was admitted for observation with a provisional diagnosis of threatened preterm labour, and differential diagnoses of placental abruption and gastroenteritis. Her symptoms remained unchanged for 4 days. On day 5, she was noted to be oedematous, hyper-reflexic, and have 2–3 beats of clonus. Her serum biochemistry was consistent with pre-eclampsia (ALT 56; AST 47; Uric Acid 0.42; Spot Urine 95.58)

On suspicion of non-hypertensive pre-eclampsia, the patient received a lower-segment caesarean section on day 6 with epidural anaesthesia, whereupon a healthy baby was delivered.

Postoperatively, the patient complained of extreme abdominal discomfort. This was initially attributed to postoperative ileus of the bowel. Eight hours later, she was noted to be tachycardic (heart rate >100 beats per minute), hypotensive (systolic blood pressure <100 mmHg), and oliguric (urine output <20 ml per hour). Her abdomen was distended, with a large tympanic mass in the epigastrium.

Plain abdominal radiograph revealed a single dilated loop of large bowel in the upper abdomen, suggestive of a caecal volvulus (Figure 1). At urgent laparotomy, an extremely distended, gangrenous caecum with serosal splitting was found in the upper abdomen. This was manually detorted. She was also noted to have intestinal malrotation (transverse colon posterior to the small intestine and duodenum anterior to the origin of the superior mesenteric artery).

A right hemicolectomy was performed. She was discharged home 5 days later.
Discussion

The present case, only the second case reported in the literature, demonstrates the importance of considering intestinal obstruction and colonic volvulus when evaluating a pregnant patient with persistent abdominal pain, nausea, and vomiting. These are common symptoms associated with pregnancy and may be dismissed, or be attributed to obstetric disorders such as hyperemesis gravidarum and placental abruption.\(^1,2\)

Similarly, such symptoms following caesarean section may be mistaken as normal postoperative symptoms.\(^1,2\) Postoperative ileus is unusual following caesarean section as there is minimal handling of the bowel.\(^3\)

Pregnant patients are at high risk for caecal volvulus due to uterine displacement of the ascending colon.\(^1,2\) Caesarean section rapidly increases intra-abdominal space, thereby increasing the likelihood of volvulus.\(^3\) Our patient’s intestinal malrotation contributed to caecal hyper-mobility, predisposing her to volvulus. Generally, it is uncommon for intestinal malrotation to become symptomatic in adult life, thereby contributing to diagnostic delays.\(^3\)

Abdominal X-rays are diagnostic for caecal volvulus in 50\% of cases.\(^4\) However, there is a general reluctance to request radiography during pregnancy for fear of foetal radiation exposure. A plain abdominal radiograph generates 100 mrad.\(^5\) Carcinogenesis and teratogenic complications occur after exposure to 1 and 20 rads respectively.\(^5\) Therefore, a single diagnostic X-ray does not result in sufficient
radiation exposure to threaten the foetus. Magnetic resonance imaging is gaining acceptance as the imaging modality of choice for pregnant women with acute abdominal conditions.\(^6\)

If caecal volvulus is suspected, urgent surgical consultation should be arranged. Right hemicolecctiony is the definitive treatment.\(^3\) Caesarean section using a lower vertical midline incision should be considered to facilitate inspection of the bowel and subsequent resection.\(^1\)

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**References:**

Quality improvement in New Zealand healthcare. Part 4: achieving effective care through clinical indicators

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Abstract

Clinical indicators can be a powerful means of improving the effectiveness of patient care. In this fourth article in the Series on quality improvement we identify the key attributes of clinical indicators and provide a scheme for their critical appraisal.

Clinical indicators are objective measures of the process or outcome of patient care. They can be used to monitor care; to flag potential opportunities to improve care, and to provide evidence that a change in practice has resulted in improvement. Clinical involvement from the “bottom up” helps to ensure that indicators are used in a formative way with a focus on “quality improvement,” rather than as a summative mechanism for “top-down” external accountability which attempts to “assure” quality. In some cases, such external quality assurance can actually harm quality improvement efforts.

Performance indicators, key performance indicators (KPIs), and clinical indicators—what are they, how do they differ, and how are they used? In this article we will attempt to answer these questions and equip clinicians with the tools to spot useful clinical indicators.

Performance indicators were placed firmly on the healthcare agenda in 1986 when the Joint Commission on the Accreditation of Health Care Organisations (JCAHO) in the United States launched an “Agenda for Change” to modernise the accreditation process. The attempt to collect and report “performance” data was a centrepiece for the JCAHO’s new direction.¹

Performance data incorporated into accreditation was to be used to satisfy the demand by the payers of healthcare, for objective evidence on the quality of that care. At the same time, healthcare organisations were progressively embracing the concept of “continuous quality improvement” (CQI) and exploring the role of performance indicators in the quest to improve the effectiveness of care. Data generated through the use of reliable and valid performance measures were recognised as central to the CQI process.

Thus from the outset there have been two principal uses of performance indicators:

- **As summative** mechanisms for external accountability and seeking “assurance” of the quality of health care, and
- **As formative** mechanisms for internal quality improvement.²³

The distinction is very important because, as Freeman³ has pointed out, the use of performance indicators in assurance and performance management systems—
summative indicators—has the potential to undermine the conditions required for continuous quality improvement in the clinical setting. Summative performance indicators (e.g. accreditation, Pay for Performance) may increase compliance costs, meaning that there is less money available for CQI. If they are used to ‘punish’ behaviour they may also drive down innovation and trust, leading to gaming of data.

Clinical indicators are a subset of performance indicators. They have been variously defined but they are essentially “an objective measure of either the process or outcome of patient care in quantitative terms.” They are usually rate based with a numerator and denominator, both of which must be clearly defined. They do not measure quality directly, but flag potential problems and possible opportunity to improve care.

It is important to appreciate that “The benefits to be gained from the use of clinical indicators do not lie in the collection of the data, but in how those data are used; that is, in the data analysis and the actions taken to achieve sustained improvements in clinical practice. Clinical indicators do not ‘work’ unless used effectively by clinicians and managers to bring about improvements.”

There are different objectives for clinical indicators, depending on who is using the indicators and whether the assessment is intended to be summative or formative. They can be used by the “manager” to control clinical behaviour, usually with the aim of decreasing costs. They can also be used by the Ministry of Health as international benchmarks, and as a means to direct funding.

For clinicians, the prime objective is to use clinical indicators to improve patient care. They do this by measuring an aspect of care over time, using indicators as flags to possible problem areas and/or potential areas for improvement. Clinical indicators can also be used to provide evidence that any changes introduced have in fact resulted in improvements in care provided. Clinical involvement from the “bottom-up” helps to ensure that indicators are used as a formative mechanism for quality improvement in patient care, rather than as summative mechanisms for “top-down” external accountability with a focus on “assurance” rather than “improvement.”

Most of the clinical indicators in use in New Zealand hospitals are derived from the Australian Council on Healthcare Standards (ACHS) indicator sets that have been developed in conjunction with Australian and New Zealand Medical Colleges, Associations, and Societies since 1989. The aims of the ACHS indicator program are laudable (to increase the involvement of clinicians in evaluation and quality improvement activities, and to facilitate the collection of national data on the processes and outcomes of patient care)—but there are several problems with the current reliance on the ACHS indicator set.

Firstly, the ACHS clinical indicators are mostly not evidence-based, and they do not adequately represent the subspecialties within the many disciplines. Secondly, forced use of externally derived clinical indicators removes clinical ownership and makes their use for quality improvement less likely. And, thirdly, benchmarking against a standard can have the effect of encouraging complacency once the benchmark is reached, which is at variance with the continuing quality improvement ethos.
How to choose an indicator?

There are recognised criteria for selecting clinical indicators. A brief understanding of Donabedian’s model for quality improvement will help guide decisions (see Box 1).\(^7\)

**Box 1. Model for quality improvement**

- **Structure**: Available resources and policies (e.g. staffing ratios, availability of diagnostic equipment)
- **Process**: The interaction between clinicians and patients (e.g. diagnostic tests, management, and treatments)
- **Outcome**: The result for the patient. (e.g. survival rates, years of healthy life lost, disability, pain)

Outcomes are of prime interest, but there are problems with measuring these directly: it may take too long to observe outcomes (therefore need high volumes and/or early endpoints); they can be confounded by problems outside the healthcare sphere of influence (e.g. poor housing, poor incomes); and they are expensive to collect. There is a consensus that measuring process indicators is preferable if there is good evidence that the process being measured is related to outcomes of interest.\(^8\) For example, there is good evidence showing that giving aspirin and beta-blockers (process measures) to patients suffering an acute myocardial infarction improves their survival (the outcome of interest).\(^9,10\)

**Table 1. Key attributes of clinical indicators\(^11\)**

<table>
<thead>
<tr>
<th>Attribute</th>
<th>Description</th>
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<tbody>
<tr>
<td>Definable</td>
<td>Can the indicator be clearly defined? (includes definable numerator and denominator)</td>
</tr>
<tr>
<td>Clear Intent</td>
<td>Is the intent of the indicator easily understood and interpretable by all users?</td>
</tr>
<tr>
<td>Valid</td>
<td>Does the indicator measure what is intended and point to issues of quality? If an indicator does not do the job you have selected it to do, then discard it. Once you have decided that it is valid for the purposes required, ensure that it can be measured reliably.</td>
</tr>
<tr>
<td>Reliable</td>
<td>Is there demonstrated reliability (reproducibility) of data? Reliability will largely depend on standardised definitions and rigour of data collection mechanisms. It depends on the precision of the definition (numerator/denominator) and the accuracy of, or variation in, data reporting. It has been said that clinical indicators should probably be valid before being reliable, as reliability can be improved with effort over time.(^12)</td>
</tr>
<tr>
<td>Accessible</td>
<td>Are data easily accessible? Are data routinely collected or will the data have to be extracted from the patient record. Who will do this and how much will it cost?</td>
</tr>
<tr>
<td>Useful/utility</td>
<td>Does the indicator provide useful information to inform quality programs and stakeholders?</td>
</tr>
<tr>
<td>Practical benefit</td>
<td>Does the indicator have a strong cost/utility ratio?</td>
</tr>
<tr>
<td>Responsive</td>
<td>Is the indicator responsive with a potential for action and quality improvement?</td>
</tr>
<tr>
<td>Relevance</td>
<td>Does the indicator measure aspects of care which are relevant and significant?</td>
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For clinical indicators to be useful in improving patient care (and therefore outcomes) it is important for each clinical group in an organisation to identify which clinical indicators are likely to be useful for their improvement efforts. In this way, only those indicators that the clinical team identifies with are chosen, and there is likely to be better ownership and association with improvement efforts.
There are several key attributes to consider when choosing an indicator (see Table 1). There are nine key attributes in this Table—for example, an indicator should be clearly defined, have a clear intent, be practical to collect, and relevant. An indicator might not satisfy all the attributes—but if it does not, then the risks associated with this must be explicitly discussed and monitored.

Once an indicator is selected, it should be critically evaluated. Box 2 outlines a series of questions that may be applied to any proposed indicator in order to understand its usefulness and potential impact on clinical work. These questions attempt to extract information about the indicator in the managerial, clinical, and economic spheres.

**Box 2. Critical appraisal of clinical indicators**

<table>
<thead>
<tr>
<th>Level 1: Is this indicator technically correct?</th>
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<tbody>
<tr>
<td>• Are we measuring what we are setting out to measure?</td>
</tr>
<tr>
<td><em>Example: Cervical screening: The denominator should exclude all women who do not qualify for screening (e.g. those who have had hysterectomy with removal of cervix)</em></td>
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<table>
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<tr>
<th>Level 2: Is there evidence to support this indicator?</th>
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<tr>
<td>• How strong is the evidence that the process leads to the desired outcome?</td>
</tr>
<tr>
<td><em>Example: Reduced length of stay may indicate better performance but may also indicate that patients are discharged too quickly. This indicator needs to be backed up by monitoring unexpected readmissions to hospital.</em></td>
</tr>
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<tr>
<th>Level 3: Is this the correct indicator?</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Why pick this one?</td>
</tr>
<tr>
<td>• Will it help the patient?</td>
</tr>
<tr>
<td>• What is the purpose and for whom?</td>
</tr>
<tr>
<td><em>Example: Influenza vaccination rates are used at international level by governments as a comparator. This may lead to distortion in the expectation on clinicians and managers compared to another possibly more clinically important indicator that is not reported in the political sphere</em></td>
</tr>
</tbody>
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<tr>
<th>Level 4: Consequences and opportunity cost (“Opportunity Cost” is the cost in all aspects of giving up the next best choice when one decides on a certain course of action)</th>
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<tbody>
<tr>
<td>• What will stop happening when we focus on this indicator?</td>
</tr>
<tr>
<td>• Are we flexible to respond to unexpected outcomes of using this indicator, both positive or negative?</td>
</tr>
<tr>
<td>• Is the system prepared for the changes in workflow that might result from this indicator—e.g. increased procedures, tests, etc.</td>
</tr>
<tr>
<td><em>Example: Funding to increase retinal screening in New Zealand did not include funding for treatment of diabetic eye disease that was discovered as a result of that programme.</em></td>
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<th>Level 5: Funding</th>
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<tr>
<td>• Are payment mechanisms and incentives aligned across the system?</td>
</tr>
<tr>
<td>• Is this financially viable?—cost:benefit across the system</td>
</tr>
<tr>
<td>• Is implementation easy?</td>
</tr>
<tr>
<td>• Is information readily available and accessible</td>
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<tr>
<th>Level 6: Is this important?</th>
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<tr>
<td>• In the grand scheme of things, and is all the effort worthwhile?</td>
</tr>
<tr>
<td>• Are there some areas where the indicator system simply does not work?</td>
</tr>
<tr>
<td>• How does one measure care where the endpoint is not recovery?</td>
</tr>
<tr>
<td><em>Example: Although domestic violence has a high societal and health cost, it is not easy to determine indicators that quickly point to improved outcomes. This does not mean that domestic violence should be ignored (indeed, one should strongly guard against this in an environment that demands data) but it should be recognized that it can never compete with cardiovascular disease for a suite of indicators</em></td>
</tr>
</tbody>
</table>
Box 2 also provides examples of the questions one might ask to better understand the indicator and how good (or bad) it will be. It is unlikely that many indicators will satisfy all levels; however, the information gained from this exercise allows everyone to understand and explicitly state the limitations of the indicator. All can then understand why the indicator was picked, what it can and cannot achieve, and what else needs to be done to limit that indicator’s weaknesses. Box 2 also includes examples of issues that have arisen from previously used indicators, to show that these could have been foreseen with pertinent evaluation.

The analysis and interpretation of indicator data by clinicians (who are familiar with the clinical process) is important for quality improvement. Clinical indicators generate data—but data needs to be analysed and presented as useable information if it is going to be used to improve care. Furthermore, clinicians need to understand the basic principles and limitations of data analysis and presentation to be able to use the information appropriately. The usefulness of the data is primarily limited by the adequacy of data collection (“garbage in, garbage out”).

Clinical indicator data is collected over time, and the most effective way to present this as useful information is either through a run chart or a control chart. Both use a set of statistical rules to determine whether the pattern revealed by the data represents the normal fluctuations about a median that is observed in any process (common-cause variation) or whether there is something that needs further investigation (special cause variation). It is important to use these rules to avoid the common problem of seeing trends where none exist, or of over-reacting to common-cause variation (and thereby making the system of care more unstable). Several texts deal with this subject for those who want to know more about run charts or control charts.13–15

**Summary**

Clinical indicators can be a powerful means of effecting change if used correctly. It is important to understand who has defined the indicators and for what purpose. It is also vital that the indicators are adequately assessed in terms of the changes they will make on the whole system, before they are adopted. Even with this approach, clinicians and managers will still be surprised when something unexpected occurs, and should be in a position to promote or restrict this as it becomes apparent. This is made a lot easier with access to accurate and timely data visible to all.

**Conflict of interest:** No conflict.

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References:

Quack remedies

This excerpt is from an Editorial published in the New Zealand Medical Journal 1906, Volume 5 (20), p32.

The Bill introduced to the House this session with the object of checking the use of quack medicines and the publication of much of the literature by which the public are entrapped within the meshes of the quack doctor’s web, has unfortunately been killed. The subject, however, has been well ventilated, and the public have certainly been warned. It is not the fault of the Public Health Department if the sale of these much-advertised remedies is not very much curtailed. Dr. Mason has striven hard and courageously, by public lectures and by writing, to impress the public with the wickedness of the trade carried on and the necessity for Government interference.

The Minister of Public Health fully admits that something must be done, and we believe it is his firm intention to bring down a Bill next session which will meet with the approval of the House. What opposition Mr Hornsby’s Bill provoked was due, partly to defects inherent in the Bill, and partly to the ignorance of certain members whose intelligence was warped by the arguments of a few interested people.

Outside the interested ones, the opponents of a Bill of this kind are just the class who love to write letters to the editor of the daily papers. Very foolish letters they are for the most part. These men say “We have found these advertised remedies very useful, far better than the doctors’ prescriptions, and you want to prevent us getting them in order to heap up riches for a close profession.”

The facts to impress upon these deluded people are, that the Bill is not in any way promoted by or intended to benefit the doctors; that there is no wish to prevent the public getting any patent medicines which are not absolutely harmful or dangerous; that all that is desired is, to protect the public from the lying literature which accompanies these drugs and to keep them out of the clutches of those harpies who first drain their purses and then cast them off, bankrupt of hope and money.

If the public wish to pay about ten times its value for their carbonate of iron they can take it in the form of- Pink Pills; if they wish for the same drug at a reasonable price let them buy it as Blaud’s Pills; but why should they be misled by lying testimonials into thinking that this or any drug is a remedy for all the ills that flesh is heir to? Again, a craving for stimulants is easily enough acquired without allowing it to be fostered and encouraged by patent medicines, which are one-third alcohol.

The law provides heavy penalties, and rightly so, for anyone who issues a prospectus of a Company containing untrue statements, why should not the law insist that statements accompanying patent medicines be also approximately true? The aim and object of the practice of medicine and surgery is the prevention and cure of sickness and the alleviation of suffering, believing as we all do, that quack remedies on the whole enormously increase the sum of human sickness and suffering, it is our duty to do all in our power to suppress them.
Pulmonary grume

Syed Wamique Yusuf, Myrshia Woods, Joseph Swafford

A 53-year-old gentleman with glioblastoma multiforme presented to the emergency department with two syncopal episodes. On arrival he was found to be in atrial fibrillation. A CT pulmonary angiogram was performed (Figure 1).

Figure 1. CT pulmonary angiogram

Question—What is the diagnosis?
Answer and discussion

The patient suffered a pulmonary saddle embolus (grume) (Figure 2). In view of malignancy, he was managed conservatively with full-dose heparin, without any bleeding complications.

Syncope is present in up to 14–21% of cases, and atrial arrhythmia (mostly atrial fibrillation and flutter) is present in up to 15% of patients with pulmonary embolism. Presence of syncope and atrial arrhythmia in patients with pulmonary embolism is associated with a worse outcome.

Figure 2. CT pulmonary angiogram showing saddle embolus

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Uncomplicated community acquired pneumonia (CAP)—how much antibiotic?

The recommendation of 7–10 days’ antibiotic treatment for uncomplicated pneumonia is not based on scientific evidence. This matter has been studied in nine hospitals in Holland. Patients with mild to moderately severe CAP were treated with intravenous amoxicillin. Those who showed significant improvement after 72 hours were switched to either oral amoxicillin or placebo for five days. Clinical and radiological outcomes assessed at days 10 and 28 were not significantly different.

The authors emphasised that their conclusions do not apply to the population with severe community acquired pneumonia. They also excluded patients with severe immunodeficiency syndrome or a significant amount of pleural fluid.

Interesting, but only small numbers (119) were involved. One wonders whether some might have done as well with oral amoxicillin only? Also we suspect that similar patients in New Zealand would be treated with co-amoxyclov rather than amoxicillin.

Clopidogrel plus aspirin versus anticoagulation for prevention of strokes

Patients with atrial fibrillation have a five times higher stroke risk than those without this common arrhythmia. Oral anticoagulation with warfarin reduces the stroke risk by two-thirds. However, anticoagulants are patient-unfriendly, difficult to monitor, and are associated with a risk of severe bleeding of at least 1% a year.

Aspirin is safer and easier to use but reduces the stroke rate by only 20%. Alternative anticoagulant therapy with the oral direct-thrombin-inhibitor ximelagatran has proven to be equally effective, but showed unacceptable liver toxicity. So what about clopidogrel (75 mg per day) plus aspirin (75–100 mg per day) versus warfarin?

A recent randomised trial involving a direct comparison of intensive antiplatelet therapy with oral anticoagulation in 6706 high-risk patients with atrial fibrillation was terminated early because of clear evidence of superiority of oral anticoagulation therapy. We have also recently reported that clopidogrel plus aspirin is no better than aspirin alone in stroke prevention (NZMJ 2 June 2006).

Early inhaled nitric oxide therapy in premature newborns with respiratory failure

Your scribe was unaware of this beneficial therapy for premature infants. But is it beneficial? Two papers, in a recent NEJM, report on the safety and efficacy of early, low-dose, prolonged therapy with inhaled nitric oxide in premature newborns with respiratory failure.
One finds that inhaled nitric oxide therapy improves the pulmonary outcome for premature infants who are at risk for bronchopulmonary dysplasia when it is started between 7 and 21 days of age and has no apparent short-term adverse effects. Excellent, but—the other found that the treatment did not reduce the overall incidence of bronchopulmonary dysplasia, except among infants with a birth weight of at least 1000 g, but it did reduce the overall risk of brain injury. Both were randomised placebo-controlled trials with adequate numbers of patients.

An editorial addresses this dilemma and comes up with the conclusion that it is an expensive unproven treatment, and the use of inhaled nitric oxide in this setting should be limited to clinical trials.


Chronic insomnia in older adults

Most clinicians are aware that insomnia is a common condition in older adults and is associated with a number or adverse medical, social, and psychological consequences. It is also associated with frequent requests for sleeping pills—most of which requests are probably successful. What about alternatives? Apparently cognitive behavioural therapy (CBT) is the most widely used psychological intervention for insomnia. Methuselah pleads ignorance of CBT—so what is it?

In this paper it is said to include sleep hygiene, sleep restriction, stimulus control, cognitive therapy, and relaxation. In this trial, older adults (mean age 60.8 years) were randomised to CBT, zopiclone 7.5 mg nightly, or a placebo tablet each night. Their results suggest that interventions based on CBT are superior to zopiclone treatment both in the short- and long-term management of insomnia in older adults. And outcomes for zopiclone were no better than placebo.

What about a wee dram before retiring?

JAMA 2006;295:2851–8

Task transfer (or substitution)?

A new term that relates to the world wide shortage of doctors and nurses. The MJA devotes a whole issue (14 papers) to this topic—Australocentric, but relevant to New Zealand. We are told that task substitution can involve the creation of new autonomous roles (e.g. nurse practitioners) or roles in which non-medical practitioners work under the supervision of someone else (usually a medical practitioner) (i.e. delegated care). Supervision may be in person or remote (e.g. nurses or physiotherapists running minor illness and injury clinics using video links for medical supervision).

An American contributor points out that, as of 2006, there are 100,000 clinically active physician assistants and nurse practitioners (NPs)—comprising approximately one sixth of the workforce in the United States. And what’s more there are to be doctoral-level NPs. These practitioners will be trained to practise at the level of family physicians, with hospital-admitting privileges and full parity of reimbursement.
And, in the UK, the University of Birmingham is considering instituting a course to train medical care practitioners (MCPs)—as "MCPs may offer advantages over increasing the number of doctors or taking nurses out of nursing roles. The introduction of MCPs may also enhance service effectiveness and efficiency."

Radical, and scary.

Med J Aust 2006;185:2–38
Working with what we have before getting into bed with the tobacco industry

New Zealand tobacco control strategies have been successful in reducing overall smoking prevalence, but there is still a way to go. For a long time the approach of the tobacco control community has been one of ‘quit or die.’ Although most smokers want to quit, many find this difficult. For half of all smokers their dependence upon tobacco will be directly responsible for future morbidity and premature death.¹

We welcome the debate about smoking harm reduction opened by McCormick et al.² They propose that new strategies to reduce the harm associated with smoking be considered, and they suggest Swedish snuff (‘snus’, a form of oral ‘smokeless’ tobacco) as a substitute for cigarettes that provides the nicotine smokers require without the many harmful products contained in cigarette smoke.

We wholeheartedly agree that increasing the options for smokers who find quitting difficult or who do not want to quit is a matter of some urgency. However, there is still much more that could be done with existing and emerging therapies before introducing new tobacco products such as snus.

Our concerns with snus include:

- Insufficient evidence that this would be an effective intervention to reduce smoking in New Zealand. While there is ecological evidence from Sweden that introducing smokeless tobacco might lead to a reduction in the prevalence of people using smoked tobacco, no randomised controlled trials have been published showing that snus promotes quitting.

- Second, the implications and impacts for Māori of introducing another form of tobacco must be thoroughly considered.

- Third, introducing snus would involve an alliance with the tobacco industry, with its well-documented history of deception and manipulation.

- Fourth, Swedish snus may be safer than smoking but it is not completely without risk. It contains tobacco-specific carcinogenic nitrosamines (2.0 µg/g product wet weight), which, while at levels lower than cigarettes (e.g. Marlboro Full Flavour 6.3 µg/g) cannot compare with the undetectable levels in nicotine replacement therapy (NRT).³

NRT has been available for the past two decades and has been shown to be effective in aiding smoking cessation.⁴ NRT’s potential for helping smokers goes beyond smoking cessation, for example in reducing cigarette consumption in smokers not motivated to quit.⁵ Some of these smokers actually go on to stop smoking completely.

How can we improve NRT-based approaches to reducing tobacco-related harm? First, NRT product licenses generally recommend a 3–6 month treatment period only—but, given that smoking is a chronic disease of dependence, longer-term NRT could be considered for some smokers. Second, we support McCormick et al’s suggestion that evaluation of faster-acting NRT products be undertaken.
Most currently available NRT products deliver significantly lower quantities of nicotine less rapidly than cigarettes, and under-dosing is also common. We would like to see this avenue explored first before introducing another unregulated tobacco company product that is of unproven benefit and of potential harm.

New, faster-acting NRT products are currently in development and there is much more that can be achieved from products already available if only smokers and healthcare professionals could overcome their fear of NRT and be more liberal with its application.

Chris Bullen
Associate Director

Hayden McRobbie
Research Fellow

Simon Thornley
Public Health Registrar

Natalie Walker
Senior Research Fellow

Robyn Whittaker
Research Fellow

Clinical Trials Research Unit, School of Population Health, University of Auckland

References:
Tom Patrick Cannon

Tom was born in Christchurch in 1923 and went to St Bedes’ College. Tom was an extremely competent all-round sportsman. He was in the First XI cricket and First XV rugby teams at St Bedes’ and later an Otago and NZU Rugby Blue. He was also an excellent boxer.

Although he was only of medium height, he had a strong physique and possessed a deceptive swerve in his preferred rugby positions of first and second five-eighths.

He started his medical education at Otago comparatively late because he was too busy playing sport earlier; he was motivated to succeed and studied diligently. At that time “mature” students were comparatively rare.

After graduation, he was a House Surgeon at Christchurch Hospital in 1949 before continuing at Kew Hospital (Invercargill).

Next he demonstrated Anatomy at Otago before going to the UK for postgraduate training in Otolaryngology at the Royal National Throat, Nose, and Ear Hospital, London in the years 1952–54. His final UK appointment was at Northampton General Hospital in 1955 where he held a top position and gained valuable experience. He was next appointed Visiting ENT Specialist to North Canterbury Hospital Board eventually becoming Head of the ENT Department at Christchurch Hospital for several years.

After establishing his medical career, marrying, and raising children, he turned to sport again becoming a competent golfer. He often played at Shirley Golf Club. In addition, he represented the Residents in their annual fixtures against the Consultants in tennis and squash.

His professional appointments included Consultant to Van Asch School for the Deaf, Consultant to the West Coast Hospital Board, Accredited Civil Aviation Examiner, President of the Otolaryngological Society of New Zealand, and Patron and President of the Christchurch Branch of the Hearing Association.

Tom was known as a gregarious and friendly colleague who was liked and respected. He was always very obliging in helping other disciplines, and he had a talent and reputation for solving and managing problems, particularly in the field of chronic ear conditions.

The obituary was written by the Production Editor using information supplied by Thomas Milliken and Don Beaven.
GRANTS AWARDED JULY 2006

At the July meeting of the Scientific Advisory Group of the National Heart Foundation, a total of 34 grants were awarded. The awards included 9 Project Grants, 12 Fellowships/Scholarships, 3 Small Project Grants, 1 Grant-in-Aid, and 9 Travel Grants. A total of 7 Summer Studentships were also awarded to the Medical Schools at the University of Otago and the University of Auckland.

PROJECT GRANTS

Dr Cliona Ni Mhurchu  
Clinical Trials Research Unit, School of Population Health, University of Auckland  
*Nutrition labels: understanding and use by ethnicity and income in New Zealand*  
$137,951 for 1 year.

Ms Anna Pilbrow  
Christchurch School of Medicine and Health Sciences, University of Otago  
*Genotype and gene expression in heart failure*  
$99,781 for 2 years.

Dr Anthony Rodgers  
Clinical Trials Research Unit, School of Population Health, University of Auckland  
*Are cardiovascular medication treatment gaps reducing?*  
$52,374 for 6 months.

Professor Zoltan Endre  
Christchurch School of Medicine and Health Sciences, University of Otago  
*Monitoring progression of diabetic kidney and cardiac disease*  
$109,342 for 1 year.

Dr Simon Hales  
Wellington School of Medicine and Health Sciences, University of Otago  
*Seasonal patterns of cardiovascular disease in New Zealand*  
$125,000 for 2 years.

Professor Mark Richards  
Christchurch Cardioendocrine Research Group, Christchurch School of Medicine and Health Sciences, University of Otago  
*Renal impairment in decompensated heart failure*  
$150,791 for 3 years.

Dr Natalie Walker  
Clinical Trials Research Unit, School of Population Health, University of Auckland  
*Nicotine replacement therapy selection box trial*  
$46,364 for 3 years.

Dr Rob MacGinley  
Dunedin School of Medicine, University of Otago  
*Does a high salt diet lead to a rapid deterioration in vascular compliance?*  
$105,482 for 2 years.
Dr Ralph Maddison
Clinical Trials Research Unit, School of Population Health, University of Auckland

Environmental influences on nutrition and physical activity in New Zealand children
$70,000 for 1 year.

FELLOWSHIPS

Dr Nicolaas van Pelt
An Overseas Training Fellowship (for 1 year) was awarded to Dr Nicolaas (Niels) van Pelt who will work as a Cardiovascular Imaging and Research Fellow at the Thorax Centrum in Rotterdam, The Netherlands.

Dr Madhav Menon
An Overseas Training Fellowship (for 1 year) was awarded to Dr Madhav Menon who will work as an Interventional Cardiology and Research Fellow in Coronary Angiography at the Minneapolis Heart Institute, USA.

Dr Sandhir Prasad
An Overseas Training Fellowship (for 1 year) was awarded to Dr Sandhir Prasad who will work as an Interventional Cardiology Fellow at Vancouver Hospital, Canada.

Ms Hannah Badland
A Research Fellowship (for 3 years) was awarded to Ms Hannah Badland who is currently employed in the Centre for Physical Activity & Nutrition Research, Auckland University of Technology.

Dr Ralph Maddison
A Research Fellowship (for 3 years) was awarded to Dr Ralph Maddison who is currently employed in the Clinical Trials Research Unit, School of Population Health, University of Auckland.

Dr Jay Ritzema-Carter
A Research Fellowship (for 3 years) was awarded to Dr Jay Ritzema-Carter who is currently employed in the Department of Cardiology, Christchurch School of Medicine and Health Science, University of Otago.

Ms Katrina Poppe
A Postgraduate Scholarship (for 3 years) was awarded to Ms Katrina Poppe who is currently employed in the Departments of Medicine and Bioinformatics, University of Auckland.

Miss Cynthia Hsu
A Postgraduate Scholarship (for 3 years) was awarded to Ms Cynthia Hsu who is currently employed in the Department of Physiology, Faculty of Medical and Health Sciences, University of Auckland.
Mr Christopher Booker

A Postgraduate Scholarship (for 3 years) was awarded to Mr Christopher Booker who is currently employed in the Edgar National Centre for Diabetes Research, Department of Medical and Surgical Sciences, University of Otago.

Ms Katrina Ellis

A Postgraduate Scholarship (for 3 years) was awarded to Ms Katrina Ellis who is currently employed in the Christchurch Cardioendocrine Research Group, Christchurch School of Medicine and Health Sciences, University of Otago.

Ms Shirin Foroughian

A Postgraduate Scholarship (for 3 years) was awarded to Ms Shirin Foroughian who is currently employed in the Section of Epidemiology and Biostatistics & Social and Community Health, School of Population Health, University of Auckland.

Mr Hamish Prosser

A Postgraduate Scholarship (for 3 years) was awarded to Mr Hamish Prosser who is currently employed in the School of Biological Sciences, University of Canterbury.

GRANT-IN-AID

Dr Sally Rose

Department of Primary Health Care & General Practice, Wellington School of Medicine and Health Sciences, University of Otago

Women’s Lifestyle Study – Maori and Pacific Cohort

$14,108 for 16 months.

SMALL PROJECT GRANTS

Dr John Irvine

Christchurch School of Medicine and Health Sciences, University of Otago

Genes of the renin-angiotensin system, oxidative stress, diabetes and endothelial dysfunction as risk factors for cardiovascular disease

$14,969 for 1 year.

Associate Professor John Evans

Department of Obstetrics and Gynaecology, Christchurch School of Medicine and Health Sciences, University of Otago

Sex steroids and secretion of vasoactive peptides

$11,641 for 6 months.
Dr Patrick Gladding
Cardiology Department, Auckland City Hospital

*Herbal medication and aspirin study*
$15,000 for 1 year.

**TRAVEL GRANTS**

**Dr Cliona Ni Mhurchu**
Clinical Trials Research Unit, School of Population Health, University of Auckland

*1st World Congress of Public Health Nutrition, Barcelona, Spain*

**Dr Wendy Atkinson**
Department of Clinical Biochemistry, Canterbury Health Laboratories, CDHB

*Folic Acid, Vitamin B12 and One-Carbon Metabolism Research Conference, California, USA*

**Dr Vicky Cameron**
Christchurch Cardioendocrine Research Group, Christchurch School of Medicine and Health Sciences, University of Otago

*The American Heart Association Scientific Sessions, Chicago, USA*

**Ms Elizabeth Duncan**
Centre for Physical Activity & Nutrition Research, Auckland University of Technology

*10th International Congress on Obesity (ICO2006) and the Physical Activity & Obesity Conference (PAOC), Sydney, Australia*

**Dr Nicolaas van Pelt**
Erasmus MC, Thorax Centrum, Rotterdam

*The European Society of Cardiology/World Congress of Cardiology Conference, Barcelona, Spain*

**Mr Akbar Ashrafi**
Department of Medicine, University of Auckland

*CSANZ Annual Scientific Meeting, Canberra, Australia*

**Mr James Duncan**
Centre for Physical Activity & Nutrition Research, Auckland University of Auckland

*9th International Congress of Behavioural Medicine, Bangkok, Thailand*

**Ms Emma Newall**
Wellington School of Medicine and Health Sciences, University of Otago

*CSANZ Annual Scientific Meeting, Canberra, Australia*

**Ms Gillian Whalley**
Department of Medicine, University of Auckland

The European Society of Cardiology/ World Congress of Cardiology Conference, Barcelona, Spain
National Heart Foundation: 2006 Grant Applications

((Libraries: print out the PDF version and replace this page))
The Hawke’s Bay Medical Research Foundation Inc: Studentship Research (applications invited)

The Foundation is pleased to offer two Annual Studentships of up to $NZ5,000 each for Medical or Health related research.

Applications can obtain an Application Form together with the guidelines of assessment by phoning the Secretary:

- Tel: (06) 879 9199; Mobile: (0273) 135 135
- or by writing to: The Secretary, P O Box 596, Napier
- or email: jmbax@xtra.co.nz (website: www.hawkesbaymedicalresearch.org.nz)

Applications close 29 September 2006

(Priority will be given to applications from appropriate persons employed in Hawke’s Bay or having an association with the region.)

JM Baxter
Secretary