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In this Issue of the Journal

Have geographical inequalities in cause-specific mortality in New Zealand increased during the period 1980–2001?
Jamie Pearce, Catherine Tisch, Ross Barnett

This research examines whether various causes of mortality in New Zealand have become more geographically unequal during the period 1980 to 2001, a time of rapid social and economic change. We calculated mortality rates for all-cause mortality, as well as for nine of the leading causes of death among males and females, for District Health Boards (DHBs) for the periods 1980–1982, 1985–1987, 1990–1992, 1995–1997, and 1999–2001. We found that between 1980 and 2001, whilst all-cause mortality rates fell for both males and females, rates have risen for chronic obstructive pulmonary disease, diabetes mellitus, and cancer. The overall reductions in mortality rates have not been uniform across all regions with large decreases and increases in mortality attributable to specific causes in some DHBs. The findings of the research are important because they demonstrate that inequalities in health between regions of New Zealand increased rapidly over the past 20–25 years.

Ethnic differences in cardiovascular disease risk factors and diabetes status for Pacific ethnic groups and Europeans in the Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand
Gerhard Sundborn, Patricia A Metcalf, Dudley Gentles, Robert Scragg, David Schaaf, Lorna Dyall, Peter Black, Rod Jackson

Niueans had the lowest cardiovascular risk of the Pacific groups. The highest estimated cardiovascular risk was found in Samoans. Diabetes prevalence was highest in Tongan women (35.8%) and Samoan men (26.2%). Tongan women had a diabetes prevalence over double that of their men. More rigorous screening of diabetes in Cook Islanders is needed to meet detection rates of other Pacific Island communities.

The accuracy of ethnicity data in primary care and its impact on cardiovascular risk assessment and management—PREDICT CVD-8
Tania Riddell, Graeme Lindsay, Tim Kenealy, Rod Jackson, Sue Crengle, Dale Bramley, Susan Wells, Roger Marshall

This study found the accuracy of ethnicity data in primary care records was limited. However the clinical impact of ethnicity misclassification on cardiovascular risk assessment and management was modest because much of the misclassification did not alter cardiovascular risk classification. Nevertheless, efforts to improve the accuracy of ethnicity data collection in primary care need to continue in order to support the sector’s ability to monitor health service utilisation, outcomes, and performance-related indicators.
Comparison of three different methods of assessing cardiovascular disease risk in New Zealanders with Type 2 diabetes mellitus
Patricia A Metcalf, Susan Wells, Robert K R Scragg, Rod Jackson

We compared three equations to evaluate the risk of heart disease and stroke over the next 5 years in people with adult-onset diabetes. The New Zealand modified equation produced the highest risk scores, followed by the original Framingham equation risk scores. The risk scores calculated using the United Kingdom Prospective Diabetes Study (UKPDS) equation produced the lowest risk scores. This means that people with adult-onset diabetes living in New Zealand will be managed earlier and more intensively based on their risk using the New Zealand Study Guidelines than if the UKPDS or original Framingham equations were used.

Ethnic counts on mortality and census data (mostly) agree for 2001–2004: New Zealand Census-Mortality Study update
Tony Blakely, June Atkinson, Jackie Fawcett

We anonymously and probabilistically linked 2001 census and 2001–2004 mortality, allowing a determination of whether mortality data correctly ascertains ethnicity. Using the same methods, we have previously shown large undercounts for Māori and Pacific deaths in the 1980s and early 1990s. For 2001–04, Māori, Pacific, Asian, and European/Other counts on mortality data were accurate—which is good news.

Food frequency information—relationships to body composition and apparent growth in 4-year-old children in the Pacific Island Family Study
Elaine Rush, Janis Paterson, Vladimir Obolonkin

This report adds to important information about the growth and development of almost 1000 Pacific children born at Middlemore Hospital in 2000. When the child was 4 years of age parents provided information about how often over the last 4 weeks had the children eaten different foods. The 40 most frequently eaten foods are listed with bread, milk, apples or pears, breakfast cereal, bananas and oranges or mandarins, consumed around once a day by most children. Food patterns associated with increased body size included consuming more protein and dairy foods and less fruit and vegetables. The continued measurement of the diet and growth patterns of these children will add to evidence to target cost-effective improvements in nutrition provided by staple foods of Pacific and New Zealand children.

Oral healthcare for older people: ‘I can’t afford not to go to the dentist, but can I afford it?’
Lynne Giddings, Barbara McKenzie-Green, Linda Buttle, Keita Tahana

This qualitative study focused on how older people perceived oral health and what they did to care for their mouth and their teeth. The findings revealed that older people don’t ‘just go to the dentist’. They put a great deal of effort into doing the best
that they can, often with limited resources. As cost is a major challenge to good oral healthcare, subsidised care would make a difference.
“Social injustice is killing people on a grand scale”

Tony Blakely

The title of this editorial is a direct quote from the second paragraph of the World Health Organization (WHO) Commission on Social Determinants of Health (CSDH) report Closing the Gap in a Generation: Health Equity through Action on the Social Determinants of Health\(^1\) launched on 28 August 2008.

The CSDH report is the culmination of 3 years of work by 19 commissioners and staff, chaired by Sir Professor Michael Marmot, and including past Presidents and Ministers of Health, and Nobel laureates. Both inequalities in health within and between countries are brought into focus. For example, a child born in a suburb of Glasgow can expect a life 28 years shorter than another living only 13 kilometres away. And a girl in Lesotho is likely to live 42 years less than another in Japan.

The report is an authoritative, state-of-the-art review of the causes of (and likely policy remedies for) social determinants of health, internationally and nationally. The messages are clear, with statements such as “the unequal distribution of health-damaging experiences is not in any sense a ‘natural’ phenomenon but is the result of a toxic combination of poor social policies and programmes, unfair economic arrangements, and bad politics.”

Should, and do, health sector employees care? Yes, and yes. The majority of people working in health, in part at least, have altruism and fairness as a driver to their career choice. The unfairness of such international and intra-national distributions of health status is an affront to most value systems. And the waste of ‘human capital’ due to premature death or illness is a drain on economic and societal performance.

Is it an issue in New Zealand? Yes. We may not have the extreme variations in health status that are observed in some countries, but our ethnic inequalities in health are large and our socioeconomic and regional inequalities in health are present in similar magnitude to other developed countries.

Can we do anything about it? Yes. As outlined in the report, many actors have the power to bring about change: multilateral agencies (e.g. WHO itself); national and local governments; civil society (e.g. Cancer Society and National Heart Foundation); private sector (e.g. food industry); and research institutions.

In this editorial I will follow the CSDH structure (improving daily living conditions; tackling the inequitable distribution of power, money and resources; and measuring and understanding the problem) to address some selected New Zealand issues, weaving in examples of recent health sector activity and research findings (including three papers in this current issue of the NZMJ).

**Improve daily living conditions**

The CSDH focuses its recommendations on early childhood development, healthy places – healthy people (e.g. urban slums), fair employment and decent work, social
protection across the lifecourse, and universal healthcare. As a developed country with a social democrat tradition, New Zealand has done fairly well on many of these aspects. There are important gaps though. Examples include the quality of our housing stock especially for lower socioeconomic groups, something which has been actively researched and addressed in recent policy initiatives.

Urban design is a challenge to all countries, with intersecting agendas of health equity, obesity epidemics, and environmental sustainability. With brave political leadership there are, however, potential win-wins. Walkable cities should be good for both health (inequalities) and environmental sustainability.

New Zealand has a mostly universal healthcare delivery system, with free financial access to most services with the exception of co-payments in primary care, dental and other ‘allied’ services. The state has a key role in the efficient and equitable provision of universal healthcare.

Two issues warrant comment ahead of the upcoming New Zealand general election. First, private public partnership models in the provision of secondary healthcare services might be considered inconsistent with the tenets of a universal healthcare system. Second, the primary health care strategy and capitation formula is now fully implemented. While the funding formula for services to improve access (SIA) and health promotion (HP) components include ethnicity and deprivation, the main component primary care does not.

Thus, a PHO with a ‘high need’ population may have a 60% higher mortality rate than a ‘low need’ population, but only receive up to an additional 17% extra funding by successfully and fully accessing the SIA and HP components of funding (workings available from author on request). This seems insufficient to address differential health need and hence health inequalities. Any future government needs to consider including deprivation and ethnicity within the main funding formula for primary care.

Tackle the inequitable distribution of power, money, and resources

The CSDH focuses its recommendations on health equity in all policies, systems, and programmes, fair financing, market responsibility, gender equity, political empowerment, and governance. Whilst New Zealand has many strengths in these domains (e.g. universal suffrage since 1893, low perceived corruption compared to other OECD countries\(^2\)), there are notable problems too.

The proportion of the New Zealand population with net-of-housing incomes below the 50\(^{th}\) or 60\(^{th}\) percentile of the median (i.e. relative poverty) is high, especially for children and Māori and Pacific peoples.\(^2\)

In recent years, poverty rates and income inequality (ratio of income of 80\(^{th}\) to 20\(^{th}\) percentile) has fallen, largely as a result of the Working for Families package (http://www.workingforfamilies.govt.nz/). Incomes have improved notably in recent years for low to medium income earners—but not as much for the lowest decile of income earners.

Families with one or more adult social security benefit recipients are ineligible for the ‘in-work payment’ of NZ$60 per week for up to three children, even if one or more adults in the household are employed. This is the basis of the current Child Poverty
Action Group case against the Crown that is before the Office of Human Rights Proceedings (OHRP), with the concern being that children of beneficiaries are being unfairly discriminated against by a policy package that conflates work incentives and income security. The forthcoming OHRP report will require addressing by Government.

The Public Health Bill is on the legislative agenda, and includes a major shift of focus from communicable disease to both communicable and chronic disease prevention. It includes provisions for codes of conduct to be developed with industry, and the ability to enforce regulations within 2 years if these codes of conduct are not successfully implemented. An obvious candidate example here is food advertising—especially to children. At a more operational level, the Health Equity Assessment Tool (HEAT) is available to screen programmes and policies for their potential to reduce (or exacerbate) inequalities in health.³

**Measure and understand the problem and assess the impact of action**

It is imperative to know the size of health inequalities, and trends over time. New Zealand has done reasonably well in this regard. The Ministry of Social Development Social Report² provides essential information on trends in social factors over time.

**Figure 1. Māori and non-Māori life expectancy trends**

![Graph showing Māori and non-Māori life expectancy trends over time.](image)

**Source:** Blakely et al (2005)⁵, updated to include 2005-07 by Ministry of Health, (Health and Disability Intelligence), unpublished.
The health monitor series (e.g., health and nutrition surveys) promulgated by the Public Health Intelligence (now Health and Disability Intelligence) group with the Ministry of Health, and the New Zealand Census-Mortality Study (NZCMS), both provide monitoring data over time on inequalities in health.

Considering ethnic inequalities, the figure above presents nearly 60 years of life expectancy trends for Māori and non-Māori, utilising Ministry and NZCMS data. It is updated for 2005–07, premised on no remaining undercount of Māori deaths, an assumption supported by NZCMS data published in this issue of the NZMJ showing little if any such remaining undercount in 2001–04.

Thirty years of closing ethnic gaps post World War 2 were followed by widening ethnic inequalities during the 1980s and 1990s, explanations for which include the structural reforms and socioeconomic ramifications of these. Of note, though, is the recent improvements in both Māori and non-Māori life expectancy accompanied by a 2-year closing in the ethnic gap in life expectancy. This is good news, although the life expectancy gap is still large and ‘stark’.

In this issue of the NZMJ, Pearce and colleagues describe geographic (District Health Board – DHB) inequalities in mortality, arguing that relative inequalities between DHBs in mortality rates have widened from 1980–82 to 1999–01. Specifically, the relative index of inequality (a regression-based relative risk measure across all DHBs) increased from 1.11 to 1.24 for males, and from 1.13 to 1.17 for females, driven in turn by widening geographic inequalities in ischaemic heart disease, chronic obstructive pulmonary disease, and diabetes mortality rates. (The latter diabetes trends, however, must be treated with considerable caution due to coding problems on mortality data.) The all-cause mortality trends over time are small in magnitude, possibly non-statistically significant, and in the case of males largely due to a jump in 1999–01. Nevertheless, geographic inequalities in health, and the likely underlying mechanisms including health selective mobility, warrant consideration, and are of immediate concern to DHBs.

Also in this issue, Sundborn et al present cardiovascular disease risk factors and diabetes rates for four Pacific groups (Niuean, Samoan, Tongan, and Cook Islanders) in 2002–03. Niueans had the healthiest profile according to the indices in this paper. A range of other differences existed between these four Pacific groups. Interestingly, compared to earlier workforce survey data for Pacific people blood pressure had increased over time, whereas lipid profiles had improved. The ratio of known to undetected diabetes was notably higher in most Pacific groups compared to European, denoting higher screening rates and suggestive of effective primary care outreach by Pacific providers.

A limitation in New Zealand, and many other countries, is accurate data on the quality and timeliness of health care receipt, over time and by social group. Health services matter for inequalities in health, and increasingly so as manifest by dramatic treatment related improvements in cardiovascular case fatality rates due to improved treatments, and to a lesser (but growing extent) for cancer survival.

A challenge in New Zealand is to move beyond the current (and good by international standards) NZHIS datasets to use patient management systems, outpatient and
inpatient data to routinely monitor (and prevent) who is falling between the cracks in healthcare.

**Conclusion**

The CSDH report has the potential to become a landmark document, in much the same way that Alma Ata was 30 years ago. Whether it lives up to that potential will depend on the response from WHO and its member countries in the next year, and the uptake of the report’s kaupapa and direction by civil society, the private sector, and other stakeholders.

In New Zealand we have made progress on inequalities in health, underpinned by research and reasonably robust monitoring systems. Maintaining the momentum, and responding to new challenges such as climate change, urban environments, state-led action to reduce risk factors for chronic disease, equitable financing of health services, and improved information systems within the health sector—to name just a few—will be important.

**Declaration of interests:** Tony Blakely has led one WHO-funded research project (2001-02) on the association of risk factors with poverty world wide, and attended one knowledge network meeting in 2005 in the early stages of the WHO CSDH’s work. He receives research funding from the Health Research Council of New Zealand and Ministry of Health. He is a member of the Cancer Control Council.

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**References:**

How to spend a sixth of your entire economy on healthcare

Pauline Norris

Healthcare now eats up 16% of the entire United States (US) economy yet US health outcomes are not particularly good. Some of the factors leading to this are undoubtedly due to the specific history of the US, while others are general trends that provide lessons to those in other countries like New Zealand.

This editorial shares impressions from my recent visit to the US, about how a country of (in the large part) kind, intelligent, and reasonable people can end up in this predicament.

Create ill-health

Create and sustain large economic and social inequalities
American society is characterised by large inequalities in access to material resources, opportunity for advancement, and social inclusion. The US’s Gini index (a measure of income inequality) is 45, substantially higher than New Zealand’s 36.2, or the Nordic countries (24–28).

Widespread poverty, homelessness, and lack of basic medical care lead to significant physical and mental health problems. An estimated 3.5 million people in the US experience homelessness each year, and the fastest growing segment is families with children. At any one time, homeless adults suffer from an average of 8–9 illnesses.

Make food cheap, taste good, and super-size it

In Chicago airport, even I baulked at the size of the individual fresh-baked warm buns, dripping with icing, that were about 6 inches square. So I asked for a smaller one, but unfortunately they were only available in a row of 6!

Destroy opportunities for incidental or deliberate exercise (except in a gym)
A combination of urban design, lack of public transport, and the availability of cheap fuel can be used to minimise walking, cycling or running, and ensure that everyone drives everywhere, all of the time. If you locate shops and services out of town, such as by the side of highways, or in large malls, that can only be accessed by car, only the most determined person will visit on foot. Wide highways without footpaths or pedestrian crossings can make it impossible to cross from one side to another without getting in a car.

Turn real and imagined health problems into market opportunities
The American public are bombarded with advertisements for medicines, for medical procedures, for medical insurance, and for particular medical services. See Figure 1.
An increasing range of symptoms of everyday life are seen as medical problems. In one issue of the newspaper—USA Today (29/10/2007)—I found the following: advertisements for the Boston Medical Group (for erectile dysfunction); health insurance (as part of an advertisement for life insurance); electric scooters for mobility; ‘Resperate’ a system of structured breathing exercises to lower blood pressure; Viamedic—a company which delivers prescription medicines; overnight shipping of three ED medications; a full-page advertisement from Sanofi-Aventis recommending taking insulin earlier in diabetes treatment; and a full-page advertisement advising that children should have an hour’s play/exercise per day.

During 2 hours of television (Turner Network Television, 9–11pm, 29/10/2007) I was informed that Quaker Oats could reduce my cholesterol, Lasix Plus could allow me a good deal on laser eye surgery, Walgreens pharmacy could offer “special services” for caregivers, Asmanex would help my asthma (advertised twice), Ducolax could relieve my constipation, and that I should take Centrum Silver vitamin supplement, and Emergen-C vitamins (advertised twice). The actress Betty White even advertises discount medications for pets on US television.

Pharmaceutical solutions to these real and imagined health problems are not only widely advertised by also widely accessible. Tylenol (paracetamol) can be found in food vending machines, and all non-prescription medicines can be bought off the shelf in liquor stores, supermarkets, or elsewhere.

**Have no brake on prices**

In New Zealand the government’s monopsony position as the biggest payer for health services gives it considerable ability to put the brakes on price growth, on total expenditure, and to balance healthcare with other goals.
In US, the huge diversity of payers means that no one can do this. If they restrict what they pay for, customers can go elsewhere. The American healthcare system is not really a system at all; it is a whole range of competing providers and payers.

For example, Medicare, as the US government’s health insurer for the elderly and disabled, pays for a vast quantity of medication. But instead of using this position to gain price advantages (as PHARMAC does in New Zealand), Medicare offers patients a choice of about 2000 different prescription medicines plans. Medicare patients have to choose the plan they think will be most advantageous for them. This requires complex judgements about the medicines they currently take, and impossible judgements about what they are likely to need in the future.

What can be done if you find that all this money isn’t making people healthy?

In spite of the huge level of expenditure on health care, life expectancy for both males and females is lower in the USA than in New Zealand and the number of Americans without health insurance increased from 44.8 million in 2005 to 47.0 million in 2006.

Both candidates in the presidential election agree on the problems: the number of uninsured people and the cost of healthcare. (www.barackobama.com and www.johnmccain.com). Barack Obama is promising to extend healthcare coverage to every American, with lower premiums whereas John McCain is promising a tax credit to offset the cost of insurance. However the chances of either candidate being able to make significant changes should not be overestimated—as (in the past) the lobbying power of insurers and providers has stymied any attempts at significant reform.

Competing interests: None known.

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References:


Have geographical inequalities in cause-specific mortality in New Zealand increased during the period 1980–2001?

Jamie Pearce, Catherine Tisch, Ross Barnett

Abstract

Aims To monitor geographical inequalities in cause-specific mortality in New Zealand during the period 1980 to 2001, a time of rapid social and economic change.


Results Between 1980 and 2001, all-cause mortality rates fell for both males and females. However, age-standardised rates have risen for chronic obstructive pulmonary disease, diabetes mellitus, and cancer. The overall reductions in mortality rates have not been uniform across all regions as the all-cause mortality RR for each DHB ranged from 0.98 to 0.69 for males and 1.10 to 0.69 for females. The RRs for cause-specific mortality are more varied with large decreases and increases in mortality attributable to specific causes in some DHBs. There has also been a sharp rise in geographical inequalities in health measured using the RII, and this trend is consistent for most types of mortality.

Conclusions Although overall mortality rates decreased over the 1980s and 1990s, this trend has not been consistent for all causes of mortality or in all regions of the country resulting in higher geographical inequalities in all-cause and most types of mortality.

Recent research has demonstrated that among Organisation for Economic Cooperation and Development (OECD) countries, mortality rates continue to decrease each year. During the period 1960 to 2003 average life expectancy among OECD countries rose from 68.5 to 77.8 years. However, whilst these improvements in health are welcomed, earlier research has established that equal progress is not always made among all socioeconomic groups, or in all geographical areas of each country.

There is overwhelming evidence that rates of ill health are significantly higher among more socially and materially disadvantaged individuals, and these gaps (in relative terms at least) have widened, leading to the emergence of significant inequalities in health.

New Zealand is no exception to these international trends. Whilst life expectancy has risen during the period between 1980–82 and 2000–02 from 70.4 to 76.3 for males and 76.4 to 81.1 for females, there are significant variations in health between different socioeconomic and ethnic groups within the country; gaps that have widened
since the 1980s. For example, one study found that although there was a decrease in overall mortality rates among New Zealand men aged 15–64 between the periods 1975–77 and 1995–97, the relative inequalities in the premature mortality rates between social classes increased by approximately 25%. Similarly, using linked census-mortality data for the period 1980-84 and 1996-99, compared to non-Māori, non-Pacific the relative gap in life expectancy grew from 7.7 to 10.8 years for Māori and from 3.3 to 7.7 years for Pacific people.

In addition to variations in health between different socioeconomic groups in New Zealand, there are also gradients across geographical areas. Regional inequalities across the country have been noted for mortality, cancer incidence, and health-related behaviours such as smoking. Further, not only are there significant spatial variations in health, but geographical inequalities in health in New Zealand are also increasing.

Recent work has found that when ranking regions within New Zealand by deprivation, regional inequalities in mortality widened during the 1980s and 1990s by approximately 50%. However, with only a few exceptions, there is a paucity of New Zealand work that has considered geographical inequalities in cause-specific mortality. This is despite the growing international interest in geographical inequalities in health as well as the emerging recognition of the importance of geographical context in explaining health outcomes. Further, the reduction of health inequalities is a key priority of the New Zealand government and monitoring inequalities in health is an important first step towards achieving this target.

Given such concerns, the objective of this paper is to investigate the association between social inequalities and cause-specific mortality rates from an area perspective, building on earlier work examining geographical inequalities in all-cause mortality. We examine changes in cause-specific mortality rates for males and females between 1980 and 2001, by District Health Boards and consider whether geographical inequality in cause-specific mortality has risen during this period.

**Methods**

Mortality records were extracted for the period 1980 to 2001 from the New Zealand Health Information Service (NZHIS) Mortality Collection. For each year, the mortality data were configured to the 21 District Health Boards (DHBs) across the country using consistent geographical units (2001 boundaries). The DHBs were formed in 2001 and are responsible for the provision of health and disability services.

The boards have an average population of 194,000 and range from 31,000 to 489,000. The small number of unspecified and overseas deaths were excluded from the analyses. In addition, identical datasets were extracted for some of the leading causes of death in New Zealand (Table 1). The leading causes of death that were examined included ischaemic heart disease, cerebrovascular disease, chronic obstructive pulmonary disease (COPD), diabetes mellitus; prostate cancer (males only), breast cancer (females only), lung cancer, colorectal cancer, as well as total cancer (which included cases of prostate and breast cancer).
Table 1. Summary information for mortality cases in New Zealand 1980 to 2001.

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>ICD-10 Code(s)</th>
<th>Count of cases (1980–2001)</th>
</tr>
</thead>
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<tr>
<td></td>
<td></td>
<td>Males</td>
</tr>
<tr>
<td>All-cause mortality</td>
<td></td>
<td>310688</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>I20-I25</td>
<td>78879</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>I60-I69</td>
<td>21798</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>J44</td>
<td>13474</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>E10-E14</td>
<td>5024</td>
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<tr>
<td>Prostate cancer</td>
<td>C61</td>
<td>8797</td>
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<tr>
<td>Breast cancer</td>
<td>C50</td>
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<tr>
<td>Lung cancer</td>
<td>C33-C34</td>
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<td>Colorectal cancer</td>
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<tr>
<td>Total cancer</td>
<td>C00-C96</td>
<td>71951</td>
</tr>
</tbody>
</table>

Directly age/sex-standardised mortality rates (ASRs) were calculated for each DHB for the periods 1980–82, 1985–87, 1990–92, 1995–97, and 1999–2001 (mortality data for 2002 were not available at the time of study), using the total contemporary New Zealand population as the standard. Age-standardised rates were calculated for all-cause mortality, as well as for each of the nine causes of mortality. For each time period, the total population for each age-sex group (e.g. 1980, 1981, and 1982) was used as the denominator.

Age- and sex-specific population data for 36 groups (males and females 0–4, 5–9, 10–14 up to 85+) were supplied from the five Censuses that took place during this period. For inter-Census years, population estimates were calculated for each age-sex group through linear interpolation.

To examine whether changes in health status have been consistent across all regions of New Zealand, the (rate) ratio of the age-standardised rate in 1999–2001 compared to the rate in 1980–1982 was calculated for each health measure and each DHB. In order to identify whether inequalities in cause-specific mortality became geographically polarised over the study period, the Relative Index of Inequality (RII) was calculated for all-cause mortality as well as for each cause for the five time periods.

The RII provides a consistent measure of health inequalities across a population because it incorporates the mortality rates of all DHBs rather than comparing, say, just those areas with the highest and lowest mortality rates. Further, the metric provides an easily interpretable measure of the socioeconomic gap in mortality between different social groups or geographical areas. The RII was calculated by ranking DHBs by a measure of poverty in 2001 weighted by the total population in 2001. Poverty was measured using the 2001 New Zealand Deprivation Index (NZDep 2001), an index based on nine socioeconomic variables taken from the 2001 New Zealand census. The NZDep 2001 is available for Census Area Units (CAUs) which are the second smallest unit of dissemination of New Zealand census data and each area comprises of approximately 2300 people. DHB-level poverty was estimated using the mean NZDep 2001 score calculated from the constituent CAUs of each DHB. The RII is then obtained by regressing (using linear regression) each of the weighted scores on each of the health outcomes (e.g. age-standardised all-cause mortality). The regression coefficient from this model is the Slope Index of Inequality (SII). The RII can then be calculated as:

\[
\text{RII} = \frac{\text{intercept}}{\text{intercept-SII}}
\]

The index provides a measure of the extent of inequalities that can be best summarised as the averaged difference between the poorest and least poor in society. Furthermore, the RII is less sensitive to changing definitions of poverty over time, hence the measure allows comparisons between different time periods. It is also the most appropriate measure for the comparison of rates and ratio spreads. Further details on the RII are described elsewhere. All results are reported for all-cause mortality and each of the different causes of mortality, stratified by sex.
Results

Temporal trends in mortality—The 3-year averaged age-standardised rates of all-cause mortality reduced among both males and females during the period 1980 to 2001 (Tables 2 and 3). For males there was a slight increase in the all-cause age-standardised rates between 1980-82 and 1985-87 from 888.2 to 893.9 per 100,000 (Table 2). However, this small rise was followed by a reduction in each of the subsequent years, and by 1999–2001 the age-standardised rate was 778.0 per 100,000; an overall reduction of 14% over the study period. Similarly, between 1980 and 2001 the age-standardised rates of ischaemic heart disease, cerebrovascular disease, and lung cancer have all decreased by between 10% and 38%. However, for the remaining causes of death (chronic obstructive pulmonary disease, diabetes mellitus, prostate cancer, colorectal cancer, total cancer), the rates have increased by between 21% (colorectal cancer) and 88% (diabetes mellitus).


<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause mortality</td>
<td>888.2</td>
<td>893.9</td>
<td>844.7</td>
<td>808.6</td>
<td>778.0</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>250.9</td>
<td>242.1</td>
<td>211.5</td>
<td>185.4</td>
<td>182.4</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>70.1</td>
<td>62.8</td>
<td>56.5</td>
<td>50.9</td>
<td>56.9</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>33.1</td>
<td>36.1</td>
<td>33.2</td>
<td>39.6</td>
<td>42.9</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11.3</td>
<td>11.1</td>
<td>11.6</td>
<td>14.6</td>
<td>21.2</td>
</tr>
<tr>
<td>Prostate cancer</td>
<td>17.0</td>
<td>20.3</td>
<td>24.1</td>
<td>26.2</td>
<td>30.7</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>50.5</td>
<td>51.6</td>
<td>48.7</td>
<td>44.9</td>
<td>46.0</td>
</tr>
<tr>
<td>Colorectal cancer</td>
<td>25.6</td>
<td>27.3</td>
<td>29.4</td>
<td>29.1</td>
<td>31.1</td>
</tr>
<tr>
<td>Total cancer</td>
<td>175.6</td>
<td>187.3</td>
<td>195.4</td>
<td>195.0</td>
<td>218.7</td>
</tr>
</tbody>
</table>

For females, the all-cause age-standardised rates followed a similar trend to those for males with a slight increase between 1980–82 and 1985–97 (746.6 to 775.3 per 100,000) followed by a persistent decrease over the remainder of the study period (Table 3). By 1999–01, the all-cause age-standardised rate among females had fallen to 712.5 per 100,000, a reduction of 5% over the study period. Similar to males, there was a reduction in the mortality rates for ischaemic heart disease and cerebrovascular disease, but unlike males there was an increase in the rate of lung cancer.

Although the female rate was lower than that for males, it nevertheless increased by 80% (from 16.6 to 29.0 per 100,000) between 1980–82 and 1999–2001. There were also increases in the age-standardised rates of COPD, diabetes mellitus, breast cancer, colorectal cancer, and total cancer over the study period by between 4% (colorectal cancer) and 327% (chronic obstructive pulmonary disease).

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause mortality</td>
<td>746.6</td>
<td>775.3</td>
<td>735.9</td>
<td>732.4</td>
<td>712.5</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>170.6</td>
<td>174.4</td>
<td>156.6</td>
<td>139.3</td>
<td>146.6</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>101.2</td>
<td>92.9</td>
<td>84.3</td>
<td>78.8</td>
<td>85.6</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>10.5</td>
<td>15.1</td>
<td>20.0</td>
<td>27.3</td>
<td>34.3</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11.8</td>
<td>11.2</td>
<td>11.1</td>
<td>14.2</td>
<td>19.2</td>
</tr>
<tr>
<td>Breast cancer</td>
<td>28.4</td>
<td>31.3</td>
<td>31.3</td>
<td>31.4</td>
<td>31.8</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>16.6</td>
<td>19.5</td>
<td>22.8</td>
<td>25.1</td>
<td>29.0</td>
</tr>
<tr>
<td>Colorectal cancer</td>
<td>27.5</td>
<td>28.4</td>
<td>27.0</td>
<td>26.1</td>
<td>28.7</td>
</tr>
<tr>
<td>Total cancer</td>
<td>147.5</td>
<td>161.1</td>
<td>168.2</td>
<td>171.3</td>
<td>182.4</td>
</tr>
</tbody>
</table>

Geographical trends—Whilst national mortality rates have fallen over the study period, the reduction has not been consistent for all DHBs across the country (Tables 4 and 5). For males, the Rate Ratios (RRs) of the all-cause mortality rates in 1999–01 compared to the rates in 1980–82 were less than 1.0, which suggests that all areas experienced a reduction in all-cause mortality over the study period (Table 4). However, some regions (e.g. Whanganui, Tairawhiti, Lakes, and Northland) experienced only very small reductions while in others (such as the Capital and Coast, Otago, South Canterbury, Auckland, and Westland DHBs) the all-cause mortality rates declined by more than 15%.

With regards to the cause-specific analysis, similar trends to the all-cause analysis were noted for ischaemic heart disease and cerebrovascular disease with most DHBs being characterised by RRs of less than 1.0 although again the reduction was not equal throughout the country. However, for the other leading causes of death (COPD, diabetes mellitus; prostate cancer, lung cancer, colorectal cancer, and total cancer), mortality rates have tended to increase in most DHBs. Further, the increases in mortality from these causes were not consistent in all regions across New Zealand. For example, for diabetes mellitus the RRs ranged from 1.26 in Nelson-Marlborough to 4.71 in Whanganui, which demonstrates that age-standardised mortality rates had increased by between 26% and more than four-fold over the study period.

For females, there are some important differences to the male results (Table 5). First, the higher RR for females (0.95 compared to 0.88 for males) indicates that there has been a smaller relative decrease in all-cause mortality for females than for males over the study period. Second, although at the national level there was a reduction in female all-cause mortality, in some regions (most notably the Waitemata, Hawke's Bay, Hutt Valley, Tairawhiti, Whanganui, and Lakes DHBs) there was a slight increase in the age-standardised rates during the 1980s and 1990s. With the exception of the West Coast and Wairarapa DHBs, the remaining regions had a RR of between 0.9 and 1.0 thus suggesting that the reduction in all-cause mortality was less than 10% in most DHBs.
Table 4. Rate Ratio for age-standardised mortality rates in 1999–2001 compared to 1980–1982 (males)

<table>
<thead>
<tr>
<th>District Health Board</th>
<th>All cause</th>
<th>IHD</th>
<th>CVD</th>
<th>COPD</th>
<th>Diabetes mellitus</th>
<th>Prostate cancer</th>
<th>Lung cancer</th>
<th>Colorectal cancer</th>
<th>Total cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>0.96</td>
<td>0.94</td>
<td>0.64</td>
<td>1.55</td>
<td>2.34</td>
<td>2.03</td>
<td>1.40</td>
<td>1.89</td>
<td>1.51</td>
</tr>
<tr>
<td>Waitemata</td>
<td>0.85</td>
<td>0.67</td>
<td>0.84</td>
<td>1.40</td>
<td>1.84</td>
<td>1.40</td>
<td>0.83</td>
<td>1.18</td>
<td>1.14</td>
</tr>
<tr>
<td>Auckland</td>
<td>0.82</td>
<td>0.66</td>
<td>0.82</td>
<td>1.47</td>
<td>1.64</td>
<td>1.63</td>
<td>0.78</td>
<td>1.04</td>
<td>1.08</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>0.89</td>
<td>0.66</td>
<td>0.85</td>
<td>1.35</td>
<td>1.61</td>
<td>1.57</td>
<td>0.76</td>
<td>1.15</td>
<td>1.07</td>
</tr>
<tr>
<td>Waikato</td>
<td>0.93</td>
<td>0.78</td>
<td>0.88</td>
<td>1.51</td>
<td>1.77</td>
<td>2.06</td>
<td>0.92</td>
<td>1.37</td>
<td>1.27</td>
</tr>
<tr>
<td>Lakes</td>
<td>0.96</td>
<td>0.85</td>
<td>0.94</td>
<td>2.11</td>
<td>2.89</td>
<td>2.84</td>
<td>1.18</td>
<td>1.55</td>
<td>1.51</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>0.91</td>
<td>0.75</td>
<td>0.79</td>
<td>1.55</td>
<td>1.64</td>
<td>2.50</td>
<td>0.98</td>
<td>1.47</td>
<td>1.38</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>0.96</td>
<td>0.79</td>
<td>0.69</td>
<td>1.19</td>
<td>2.15</td>
<td>1.29</td>
<td>1.06</td>
<td>1.11</td>
<td>1.28</td>
</tr>
<tr>
<td>Taranaki</td>
<td>0.90</td>
<td>0.90</td>
<td>0.95</td>
<td>1.65</td>
<td>2.42</td>
<td>2.60</td>
<td>1.00</td>
<td>1.11</td>
<td>1.35</td>
</tr>
<tr>
<td>Hawke's Bay</td>
<td>0.89</td>
<td>0.84</td>
<td>0.87</td>
<td>1.18</td>
<td>2.08</td>
<td>2.62</td>
<td>1.31</td>
<td>1.03</td>
<td>1.41</td>
</tr>
<tr>
<td>Whanganui</td>
<td>0.98</td>
<td>0.98</td>
<td>0.77</td>
<td>1.30</td>
<td>4.71</td>
<td>2.28</td>
<td>1.06</td>
<td>1.43</td>
<td>1.51</td>
</tr>
<tr>
<td>Mid Central</td>
<td>0.90</td>
<td>0.81</td>
<td>0.92</td>
<td>1.43</td>
<td>2.23</td>
<td>1.87</td>
<td>0.87</td>
<td>1.06</td>
<td>1.22</td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>0.86</td>
<td>0.63</td>
<td>0.76</td>
<td>1.14</td>
<td>1.37</td>
<td>2.15</td>
<td>0.83</td>
<td>0.83</td>
<td>1.27</td>
</tr>
<tr>
<td>Capital and Coast</td>
<td>0.83</td>
<td>0.68</td>
<td>0.93</td>
<td>0.92</td>
<td>2.31</td>
<td>1.52</td>
<td>0.65</td>
<td>1.31</td>
<td>1.19</td>
</tr>
<tr>
<td>Wairarapa</td>
<td>0.88</td>
<td>0.74</td>
<td>0.48</td>
<td>1.10</td>
<td>1.72</td>
<td>1.46</td>
<td>0.66</td>
<td>0.96</td>
<td>1.00</td>
</tr>
<tr>
<td>Nelson-Marlborough</td>
<td>0.88</td>
<td>0.66</td>
<td>0.90</td>
<td>1.41</td>
<td>1.26</td>
<td>1.74</td>
<td>1.04</td>
<td>1.33</td>
<td>1.48</td>
</tr>
<tr>
<td>West Coast</td>
<td>0.69</td>
<td>0.63</td>
<td>0.65</td>
<td>1.25</td>
<td>2.70</td>
<td>4.29</td>
<td>0.70</td>
<td>1.08</td>
<td>1.05</td>
</tr>
<tr>
<td>Canterbury</td>
<td>0.85</td>
<td>0.70</td>
<td>0.77</td>
<td>1.23</td>
<td>1.44</td>
<td>1.31</td>
<td>0.97</td>
<td>1.46</td>
<td>1.25</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>0.82</td>
<td>0.62</td>
<td>0.69</td>
<td>1.28</td>
<td>1.69</td>
<td>1.60</td>
<td>0.67</td>
<td>0.73</td>
<td>1.15</td>
</tr>
<tr>
<td>Otago</td>
<td>0.83</td>
<td>0.67</td>
<td>0.69</td>
<td>0.95</td>
<td>1.71</td>
<td>2.27</td>
<td>1.06</td>
<td>1.23</td>
<td>1.31</td>
</tr>
<tr>
<td>Southland</td>
<td>0.87</td>
<td>0.89</td>
<td>1.19</td>
<td>1.35</td>
<td>2.31</td>
<td>2.37</td>
<td>0.97</td>
<td>1.50</td>
<td>1.50</td>
</tr>
<tr>
<td>New Zealand</td>
<td><strong>0.88</strong></td>
<td><strong>0.73</strong></td>
<td><strong>0.81</strong></td>
<td><strong>1.29</strong></td>
<td><strong>1.88</strong></td>
<td><strong>1.81</strong></td>
<td><strong>0.91</strong></td>
<td><strong>1.22</strong></td>
<td><strong>1.25</strong></td>
</tr>
</tbody>
</table>

(IHD=Ischaemic heart disease; CVD=Cerebrovascular disease; COPD=Chronic obstructive pulmonary disease)

Similar to males, in most DHBs there was a reduction in the mortality rates attributed to ischaemic heart disease and cerebrovascular disease. However, some DHBs saw an increase in mortality for these two causes of mortality and in Tairawhiti and Lakes DHBs mortality rates increased for both causes by as much as 35%. For all of the remaining leading causes of death (COPD, diabetes mellitus; breast cancer, lung cancer, colorectal cancer, and total cancer) the 3-year average age-standardised rates tended to increase in most DHBs during the 1980s and 1990s. For example, the RR for COPD ranged from 1.89 (Wairarapa) to 8.70 (Wairarapa), which suggests that the mortality rates for this cause rose by between 89% and more than eight-fold. Similarly, mortality rates due to diabetes mellitus rose in all DHBs by between 13% (Tairawhiti) and 246% (Northland). Interestingly, in the Whanganui and Lakes DHBs age-standardised mortality rates increased not only for all-cause mortality but also for all but one of the specific causes of mortality.

The RII for all-cause mortality was roughly equal over the first part of the study period but then rose sharply between 1995–97 and 1999–2001 (Table 6). Between 1980–82 and 1999–2001 the RII for all-cause mortality rose from 1.11 to 1.24 for males and from 1.13 to 1.17 for females. The results therefore show that the level of health inequalities in New Zealand equates to an increase in excess mortality, for the worst off areas, from 11% to 24% for males and 13% to 17% for females.
Table 5. Rate Ratio for age-standardised mortality rates in 1999–2001 compared to 1980–1982 (females)

<table>
<thead>
<tr>
<th>District Health Board</th>
<th>All cause</th>
<th>IHD</th>
<th>CVD</th>
<th>COPD</th>
<th>Diabetes mellitus</th>
<th>Breast cancer</th>
<th>Lung cancer</th>
<th>Colorectal cancer</th>
<th>Total cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td>0.99</td>
<td>0.87</td>
<td>0.82</td>
<td>2.81</td>
<td>2.46</td>
<td>1.50</td>
<td>1.90</td>
<td>0.85</td>
<td>1.44</td>
</tr>
<tr>
<td>Waitemata</td>
<td>1.03</td>
<td>0.83</td>
<td>0.81</td>
<td>3.06</td>
<td>1.21</td>
<td>1.22</td>
<td>1.56</td>
<td>1.06</td>
<td>1.22</td>
</tr>
<tr>
<td>Auckland</td>
<td>0.90</td>
<td>0.79</td>
<td>0.81</td>
<td>2.69</td>
<td>2.32</td>
<td>0.95</td>
<td>1.11</td>
<td>0.74</td>
<td>0.99</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>0.92</td>
<td>0.70</td>
<td>0.78</td>
<td>4.67</td>
<td>1.38</td>
<td>0.82</td>
<td>1.74</td>
<td>0.84</td>
<td>1.02</td>
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<tr>
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<td>0.82</td>
<td>0.84</td>
<td>3.45</td>
<td>1.33</td>
<td>1.24</td>
<td>1.68</td>
<td>0.83</td>
<td>1.17</td>
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<tr>
<td>Lakes</td>
<td>1.10</td>
<td>1.35</td>
<td>1.16</td>
<td>2.19</td>
<td>1.47</td>
<td>0.73</td>
<td>2.14</td>
<td>1.08</td>
<td>1.46</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>0.99</td>
<td>0.81</td>
<td>1.07</td>
<td>4.01</td>
<td>1.56</td>
<td>1.47</td>
<td>1.91</td>
<td>1.56</td>
<td>1.42</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>1.07</td>
<td>1.32</td>
<td>0.67</td>
<td>2.02</td>
<td>1.13</td>
<td>1.08</td>
<td>1.26</td>
<td>0.93</td>
<td>1.19</td>
</tr>
<tr>
<td>Wairarapa</td>
<td>0.96</td>
<td>1.02</td>
<td>0.98</td>
<td>8.70</td>
<td>2.26</td>
<td>1.18</td>
<td>1.42</td>
<td>0.96</td>
<td>1.35</td>
</tr>
<tr>
<td>Hawke’s Bay</td>
<td>1.04</td>
<td>1.08</td>
<td>0.97</td>
<td>3.65</td>
<td>1.77</td>
<td>1.68</td>
<td>1.89</td>
<td>1.28</td>
<td>1.48</td>
</tr>
<tr>
<td>Whanganui</td>
<td>1.10</td>
<td>1.17</td>
<td>1.21</td>
<td>4.72</td>
<td>1.75</td>
<td>0.98</td>
<td>2.91</td>
<td>1.15</td>
<td>1.48</td>
</tr>
<tr>
<td>MidCentral</td>
<td>0.95</td>
<td>0.93</td>
<td>0.96</td>
<td>5.09</td>
<td>1.63</td>
<td>1.04</td>
<td>1.78</td>
<td>1.07</td>
<td>1.21</td>
</tr>
<tr>
<td>Hutt Valley</td>
<td>1.04</td>
<td>0.77</td>
<td>0.78</td>
<td>2.23</td>
<td>1.59</td>
<td>1.55</td>
<td>1.46</td>
<td>0.88</td>
<td>1.41</td>
</tr>
<tr>
<td>Capital and Coast</td>
<td>0.94</td>
<td>0.86</td>
<td>0.92</td>
<td>3.13</td>
<td>2.38</td>
<td>0.98</td>
<td>1.43</td>
<td>1.04</td>
<td>1.18</td>
</tr>
<tr>
<td>Wairarapa</td>
<td>0.88</td>
<td>0.86</td>
<td>0.49</td>
<td>1.89</td>
<td>1.86</td>
<td>1.18</td>
<td>3.08</td>
<td>0.89</td>
<td>1.50</td>
</tr>
<tr>
<td>Nelson-Marlborough</td>
<td>0.98</td>
<td>0.99</td>
<td>1.01</td>
<td>3.54</td>
<td>1.29</td>
<td>1.62</td>
<td>1.60</td>
<td>1.10</td>
<td>1.41</td>
</tr>
<tr>
<td>West Coast</td>
<td>0.69</td>
<td>0.60</td>
<td>0.52</td>
<td>3.23</td>
<td>1.56</td>
<td>0.60</td>
<td>4.79</td>
<td>1.79</td>
<td>1.15</td>
</tr>
<tr>
<td>Canterbury</td>
<td>0.90</td>
<td>0.87</td>
<td>0.78</td>
<td>3.30</td>
<td>1.32</td>
<td>1.00</td>
<td>2.08</td>
<td>1.05</td>
<td>1.22</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>0.93</td>
<td>0.82</td>
<td>0.64</td>
<td>4.62</td>
<td>1.64</td>
<td>1.22</td>
<td>2.77</td>
<td>1.37</td>
<td>1.42</td>
</tr>
<tr>
<td>Otago</td>
<td>0.94</td>
<td>0.78</td>
<td>0.94</td>
<td>3.72</td>
<td>1.57</td>
<td>1.40</td>
<td>2.27</td>
<td>1.23</td>
<td>1.25</td>
</tr>
<tr>
<td>Southland</td>
<td>0.94</td>
<td>1.00</td>
<td>1.02</td>
<td>2.12</td>
<td>1.37</td>
<td>0.91</td>
<td>1.70</td>
<td>1.76</td>
<td>1.34</td>
</tr>
</tbody>
</table>

It is noteworthy that for some causes of death the RII values are considerably higher than those noted for all-cause mortality. For example, the RII for mortality attributed to diabetes mellitus was in excess of 2.0 for males and females, which suggests that there was an excess mortality of more than 100% in the poorest areas of New Zealand. However, for other causes of death, particularly colorectal cancer, the RII was consistently close to 1.0 which suggests that there were no significant inequalities attributable to this cause.

Nonetheless, the trend in the RII for the cause-specific mortality results are generally consistent with the all-cause analysis with an overall increase in inequality over the study period. The RII values are usually higher for males than females and the increase has been more consistent in the former.
Table 6. Relative Index of Inequality for all-cause and causes-specific mortality in New Zealand 1980–2001

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>All-cause mortality</td>
<td>1.11</td>
<td>1.12</td>
<td>1.12</td>
<td>1.14</td>
<td>1.24</td>
<td>1.13</td>
<td>1.16</td>
<td>1.12</td>
<td>1.14</td>
<td>1.17</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>0.96</td>
<td>1.03</td>
<td>1.04</td>
<td>0.99</td>
<td>1.15</td>
<td>1.03</td>
<td>0.99</td>
<td>0.99</td>
<td>1.04</td>
<td>1.07</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>1.10</td>
<td>1.06</td>
<td>0.99</td>
<td>1.09</td>
<td>1.07</td>
<td>0.94</td>
<td>1.07</td>
<td>0.96</td>
<td>0.91</td>
<td>1.02</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>0.99</td>
<td>1.06</td>
<td>1.08</td>
<td>1.04</td>
<td>1.27</td>
<td>1.00</td>
<td>1.56</td>
<td>1.40</td>
<td>1.10</td>
<td>1.18</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.79</td>
<td>1.43</td>
<td>1.75</td>
<td>2.11</td>
<td>2.49</td>
<td>1.80</td>
<td>1.31</td>
<td>2.32</td>
<td>2.85</td>
<td>2.13</td>
</tr>
<tr>
<td>Prostate (M) or breast (F) cancer</td>
<td>0.86</td>
<td>0.92</td>
<td>0.96</td>
<td>1.10</td>
<td>1.29</td>
<td>0.99</td>
<td>1.03</td>
<td>1.02</td>
<td>1.17</td>
<td>0.99</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>1.04</td>
<td>1.19</td>
<td>1.19</td>
<td>1.06</td>
<td>1.24</td>
<td>1.49</td>
<td>1.34</td>
<td>1.46</td>
<td>1.51</td>
<td>1.63</td>
</tr>
<tr>
<td>Colorectal cancer</td>
<td>0.90</td>
<td>1.01</td>
<td>0.90</td>
<td>1.07</td>
<td>0.93</td>
<td>1.08</td>
<td>1.00</td>
<td>1.02</td>
<td>0.98</td>
<td>0.93</td>
</tr>
<tr>
<td>Total cancer</td>
<td>1.03</td>
<td>1.05</td>
<td>1.02</td>
<td>1.06</td>
<td>1.11</td>
<td>1.08</td>
<td>1.07</td>
<td>1.00</td>
<td>1.13</td>
<td>1.10</td>
</tr>
</tbody>
</table>
Discussion

The main finding of this study is that during the 1980s and 1990s there were rising spatial inequalities in health for males and females in New Zealand as measured using all-cause mortality as well as for most of the leading causes of death. Whilst overall all-cause mortality declined by approximately 12% over the study period, this was not true of all regions with some DHBs witnessing considerable mortality reductions compared to only modest declines in others.

Further, relative inequality in mortality between areas of high and low social deprivation increased, which suggests that there have been larger reductions in mortality in less deprived regions of the country. The reduction in all-cause mortality was also greatest for males with females showing fewer relative gains and, in some areas, absolute increases in mortality occurred.

Not surprisingly, there has also been a widening in inequality for each of the leading causes of mortality. Whilst two of the major causes of death (ischaemic heart disease and cerebrovascular disease) have mirrored the overall reduction in all-cause mortality, for some causes of death, in particular chronic obstructive pulmonary disease, diabetes mellitus, all of the individual cancer types (except male lung cancer) and total cancers, there was an increase in the age-standardised rates. However, as with all-cause mortality, the national-level increases or decreases in the age-standardised rates of each cause have not been consistent in all regions.

Further, the rising spatial inequalities between rich and poor regions of the country noted for all-cause mortality are not consistent for all mortality types. Nonetheless, by the end of the study period for most of the leading causes of death (except colorectal cancer, and breast cancer among women) there was at least a small and increasing excess in mortality in more socially deprived regions of the country. Indeed for some causes of death (especially diabetes mellitus) there was a particularly strong socioeconomic gradient.

These trends are consistent with the international studies that have monitored geographical inequalities in health. Research in the UK, US, and Australia has noted that health has become more geographically polarised over the 1980s and 1990s. The current research is also consistent with earlier New Zealand research examining spatial inequalities in all-cause mortality, which found a spatial polarisation in life expectancy over the 1980s and 1990s. Further, our findings concur with other New Zealand studies that have examined ethnic and social inequalities in health.

There are several plausible explanations for rising geographical inequalities in health in New Zealand. First, the 1980s and 1990s saw the implementation of a neoliberal economic and social agenda in New Zealand which led to economic restructuring and substantial alterations to the welfare state, particularly in the areas of housing, health, and education. One important outcome of this transformation was a significant increase in levels of economic and social inequality between the rich and the poor. This changing social and political environment particularly disadvantaged lower socioeconomic groups and areas as well as Māori and Pacific people and is likely
to be an important explanation for the diverging health status between high and less deprived regions across the country.

Second, as has previously been suggested, selective migration patterns between New Zealand regions may help to explain why regional health status in New Zealand became more geographically polarised between 1980 and 2001. This interpretation is consistent with work in the UK which found that the differential migration patterns of ill people relative to healthy contributes to the widening geographical divide in health in that country.30

Compared to other OECD countries, New Zealand has high levels of immigration (19.5% of the New Zealand population were born overseas)31 and emigration, which is likely to result in the perpetual re-sorting of people by area. Most migrants into New Zealand are highly skilled and have high levels of educational attainment and tend to locate in the main urban centres, particularly Auckland.32 These selective trends in population turnover may partially explain the rising relative inequalities in health observed in this study.

Third, it is possible that there are characteristics of the DHBs that exert an independent influence on the health of the residents of those areas. This interpretation would be consistent with the substantial body of literature that has identified various ‘place effects’ that operate across the lifecourse, and influence the health outcomes and health inequalities of local residents.33

Researchers are continuing in their attempts to untangle the ‘compositional’ (individual-level) and ‘contextual’ (ecological) explanations for health inequalities. Potentially, the most pertinent place-based process that operates at the DHB level is the provision of healthcare.

The healthcare reforms of the 1980s and 1990s, which resulted in substantial co-payments, led to the under utilisation of healthcare services among the most at-risk groups.34 Poor access to primary health care services has been linked to worse health outcomes and increased hospitalisation among the more disadvantaged social groups.35,36 Moreover, the unequal rationing of primary health care services has been shown to have affected some regions more than others and is likely to contribute to the emerging inequalities in health between DHBs across New Zealand.

It should be noted that we have examined spatial inequalities in health across relatively broad geographical areas (the 21 DHBs in New Zealand). However, DHBs are likely to exhibit considerable internal heterogeneity particularly with respect to social deprivation. It is probable that operationalising smaller geographical units, which more precisely specify area-level socioeconomic status, would have revealed wider spatial inequalities in health.

Similarly, it is not possible using ecological data to ascertain the socioeconomic circumstances of individual mortality cases. Therefore, it cannot be assumed that what is identified at the area-level is necessary a reflection of what is occurring at the individual-level (the ecological fallacy). Also, in any mortality study there are always potential data quality issues, particularly in terms of misdiagnoses of the causes of death, or the effect of multiple causes.
Some minor problems may also have arisen as a result of using cross-sectional data based on the average mortality rate for each 3-year period. Depending upon the rate of population change, interpolation of rates based on census estimates may have resulted in some minor variations in estimates for some DHBs.

The findings of this research should be of significant interest to policy makers in New Zealand. Although reducing health inequalities has been identified in the New Zealand Health Strategy as a key government priority, our findings suggest that government policies have not been effective in reducing the spatial divide.

Given the increased importance of DHBs in promoting health and greater local accountability for monitoring and addressing adverse health outcomes we suggest that more attention needs to be paid to geographical differences in health and how the causes of ill health and mortality are likely to vary between different regions. This assertion is important because the causes of spatial variations in health are not simply a function of ethnic or social differences in the population composition of different DHBs.

Therefore, it is imperative that in the future, policy makers pay greater attention to local contextual and compositional factors affecting health and the extent to which DHB trends in health outcomes are similar or different to those in other regions. It is also important that future research monitors the inequalities between key at-risk groups and evaluates the government’s strategies to reduce the health divide.

**Competing interests:** None known.

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**Acknowledgements:** We thank Craig Wright from the Public Health Intelligence group at the Ministry of Health for providing the mortality and population data. CT’s postgraduate scholarship was funded through the GeoHealth Laboratory.

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**References:**

1. Organisation for Economic Co-operation and Development. Health at a Glance - OECD Indicators 2005: OECD; 2005. [http://www.oecd.org/document/11/0,3343,en_2649_34631_16502667_1_1_1_1,00.html](http://www.oecd.org/document/11/0,3343,en_2649_34631_16502667_1_1_1_1,00.html)
Ethnic differences in cardiovascular disease risk factors and diabetes status for Pacific ethnic groups and Europeans in the Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand

Gerhard Sundborn, Patricia A Metcalf, Dudley Gentles, Robert K R Scragg, David Schaaf, Lorna Dyall, Peter Black, Rod Jackson

Abstract

Aim The aim of this paper is to provide levels of cardiovascular disease (CVD) risk factors and diabetes status for Pacific ethnic groups and make comparisons amongst these groups (Samoan, Tongan, Niuean, Cook Islanders) with European New Zealanders by gender from the 2002–03 DHAH Survey.

Methods The DHAH was a cross-sectional population-based survey and was carried out in Auckland between 2002–03. A total of 1011 Pacific comprising of 484 Samoan, 252 Tongan, 109 Niuean, 116 Cook Islanders, and 47 Other Pacific (mainly Fijian) and 1745 European participants took part in the survey. Participants answered a self-administered questionnaire to assess whether they had previously diagnosed CVD risk factors (blood pressure, cholesterol, diabetes) and lifestyle risk factors (smoking, physical inactivity). All participants provided an early morning mid-stream urine sample, an initial blood test and full glucose tolerance test (GTT) for those not previously diagnosed with diabetes.

Results In both men and women, CVD risk among the Pacific groups were all significantly higher than Europeans. Niueans had the lowest Pacific CVD risk and Samoans had the highest estimated risk. Individual risk factors differed between the groups, however; the most observable differences were the more adverse lipid profile in Tongan men and the lower total cholesterol and micro-albumin in Niuean women when compared to their Samoan counterparts. Diabetes prevalence was highest in Samoan men (26.2%) and Tongan women (35.8%). Tongan women had a diabetes prevalence over double that of their men (17.8%), whereas in the other Pacific groups, male and female prevalence was very similar. Niueans had the lowest diabetes prevalence of both sexes (men 14.9%, women 10.8%). Undiagnosed diabetes as a proportion of total diabetes was similar in Samoan, Niuean and Cook Islands groups (1/4–5) suggesting efficient screening. Cook Islanders had a ratio of one undetected diabetes case for every two known cases.

Conclusion CVD risk factors, diabetes prevalence, and levels of undetected diabetes differed between the Pacific ethnic groups with Niueans having the healthiest profile. More rigorous screening of diabetes in Cook Islanders is needed if they are to experience similar detection rates as other Pacific Island communities in New Zealand. Greater attention is required to identify and manage CVD risk among all Pacific peoples to reduce the gap in CVD risk factors, morbidity and mortality when compared to European New Zealanders.
Cardiovascular diseases (CVD) are the leading cause of death in New Zealand (NZ), and accounted for 39% of all deaths in 2003.\(^1\) Diabetes is a leading cause of premature mortality and disability for NZers, and it has been estimated that diabetes contributes to one-quarter of the difference in life expectancy between Pacific and European NZers.\(^2\)

Pacific people in NZ (mostly of Samoan, Tongan, Niuean, or Cook Islands origin) had the highest mortality rate for cerebrovascular disease, the highest hospital discharge rate for stroke,\(^3\) and were found to be four times more likely to have diabetes than European NZers.\(^4\)

Most studies that have assessed CVD risk and diabetes prevalence in NZ do not differentiate between the various Pacific ethnic groups although this has been recommended.\(^5,6\) However, the few studies that have are either from workforce samples (and therefore not representative of the general population), are low in Pacific numbers, rely on self-reported measures of diabetes status, and are now over 10 years old.\(^5,7\) These studies found that CVD risk factors and the prevalence of known diabetes differed between the Pacific ethnic groups. This has meant that reliable information on CVD risk factors and diabetes prevalence of Pacific ethnic groups has been scarce.

The aim of this paper is to provide levels of CVD risk factors and diabetes status for Pacific ethnic groups and make comparisons amongst these groups (Samoan, Tongan, Niue, Cook Islands) with European NZers by gender from the 2002–03 DHAH Survey.

**Method**

The DHAH Survey was a cross-sectional study that surveyed people aged 35–74 years, between January 2002 and December 2003. All participants were selected from within the Auckland region. There were 1011 Pacific and 1745 European participants.

Adults were recruited using two sampling frames: one was a cluster sample where random starting point addresses were obtained from Statistics New Zealand and the probability of selection was proportional to the number of people living in that mesh block (response rate 61.3%); and the other was a random sample taken from the November 2000 Auckland electoral rolls stratified into 5-year age bands and included all people living in the Auckland area, with the exception of the Franklin and Rodney electorates (response rate 65%). Participants were interviewed in places close to where they lived and all completed a self-administered questionnaire and a series of health measurements were made.

Classification of ethnicity gave priority to Pacific over European ethnicity. This is similar to the method used by Statistics New Zealand.\(^8\) Participants who indicated belonging to more than one Pacific ethnic group were assigned to one ethnic group only. Those who were of Pacific and non-Pacific or non-Maori were assigned into their respective Pacific ethnic group. Those who belonged to more than one Pacific ethnic group were assigned to the smaller Pacific group as done by Census 2001.\(^9\) This gave priority firstly to Niuean, followed by Cook Islands, Tongan, and lastly Samoan ethnicity. Small numbers of Fijian (n=27) and ‘Other Pacific’ (n=27) participants meant that analysis of their results could not generate reliable findings. Ethical approval was obtained from the Health and Disability Ethics Committees.

All participants received information in the mail with instructions not to eat any food from 10pm onwards the night before their survey was scheduled and to drink water only. Included in the information pack was a sterile urine container that was used to collect an early morning urine sample (mid-stream). Fasting blood samples were taken from all participants. Participants were asked whether they had been diagnosed with diabetes, and if so how old they were when they were first told, and what their current treatment was. Those who did not have previously diagnosed diabetes mellitus were then
asked to complete a 2 hour Glucose Tolerance Test (GTT). This involved having a drink consisting of 75 g glucose after their initial blood test and final blood test was taken 2 hours later. Participants filled in survey questionnaires that obtained information on socioeconomic status (SES), smoking, and exercise. Two blood pressure readings were taken after the participant had been sitting for at least 10–15 minutes. Height, weight, and waist and hip circumferences were measured.

Fasting blood samples were assayed using enzymatic methods. Plasma glucose was measured using commercial reagents (Roche Products [NZ]), HbA1c was measured by high performance liquid chromatography, and micro-albumin was measured using an immunoturbidmetric method. Categorisation of glucose tolerance status was evaluated by 1998 World Health Organization (WHO) criteria using fasting glucose ≥ 7.0 mmol/L or 2-hour post-glucose load of ≥ 11.1 mmol/L for diabetes; fasting glucose < 7.0 mmol/L and 2-hour glucose between 7.8 and 11.0 mmol/L for Impaired Glucose Tolerance (IGT) and fasting glucose of 6.1–6.9 mmol/L for Impaired Fasting Glucose (IFG). All participants were then classified as ‘known’ (from their past history), ‘new’-ly diagnosed, having ‘IGT’ or ‘IFG’ or ‘normal’ glucose functioning.

Measurement of cholesterol and triglycerides was done using enzymatic methods. LDL cholesterol was estimated by the Friedewald formula.9 Exercise was assessed using a 3-month physical activity recall questionnaire.10 One question asked if participants had engaged in any vigorous activity at least once a week in the past 3 months long enough to make them breathe hard or sweat. The other question asked if they had engaged in any moderate activity (that did not cause them to breathe hard or sweat). Those that answered no to both questions were categorised as inactive.

Statistical analysis was undertaken using SAS (version 9.1) software. Participant data were weighted according to the sampling frame that they were obtained from means, standard errors, and prevalences calculated using dual-frame sampling methodology.11–13 SAS survey procedures (SURVEYMEANS, SURVEYREG, SURVEYFREQ AND SURVEYLOGISTIC) were used to calculate weighted means, adjusted means, percentages, and odds ratios, respectively.14 The Rao-Scott modified Pearson Chi-squared test was used where appropriate. Analyses have compared all Pacific ethnic groups to their European and Samoan counterparts. Samoans were used as the intra Pacific reference group as they comprised the largest Pacific sample.

Results

Table 1—Table 1 shows the proportions and mean levels of both lifestyle and CVD risk factors in men. As Samoan and Niuean men were slightly younger than European men and Tongan men were slightly older than Samoan men, subsequent analyses were adjusted for age. Compared to European men, all Pacific men were significantly shorter, had larger body mass index (BMI) measures, and higher NZDep2001 scores. Compared to Samoan men, Tongan men had a larger waist-to-hip ratio, lower systolic blood pressure (BP), lower high-density lipoprotein (HDL) cholesterol, higher ratio of total to HDL cholesterol and low-density lipoprotein (LDL) cholesterol, and lower urinary albumin.

Table 2—Table 2 show the proportions and mean levels of both lifestyle and CVD risk factors in women. All Pacific women were more than four years younger than European women. Therefore all subsequent analyses were adjusted for age. Compared to European women, all Pacific groups had significantly higher BMI, NZDep2001, waist to hip ratio, waist circumference, diastolic BP, 5-year CVD risk scores, and had lower HDL-cholesterol. There were no significant differences between Samoan and Cook Islands women.

In both men and women, 5-year CVD risk among the Pacific groups were all significantly higher than Europeans with the exception of Niuean men. However, Niuean men were the only group to be significantly different to their Samoan
counterparts. Five-year CVD risk was higher for men compared to women for all groups.

**Table 3**—Table 3 shows the fasting and 2-hour fasting glucose levels and proportions of those with newly diagnosed diabetes, known diabetes, impaired glucose tolerance (IGT), and impaired fasting glucose (IFG) by ethnic group for men. Compared to European men, all Pacific groups had higher HbA1c levels. Samoan and Tongan men had the highest proportions of total diabetes. Some differences were not statistically significant due to low numbers. For example: the proportion of total diabetes of 20.8% in Cook Islands men compared with 6.3% in European men.

**Table 4**—Table 4 shows the fasting and 2-hour fasting glucose levels and proportions of those with newly diagnosed diabetes, known diabetes, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) by ethnic group for women. Compared to European women, all Pacific groups had higher proportions of HbA1c and total diabetes mellitus.

These results show that Niuean women have a clearly distinct diabetes profile to other Pacific groups with the lowest plasma glucose levels, and had the smallest proportions of total diabetes, IGT, and IFG diabetes states.

Tongan women had the highest prevalence overall and Samoans had the highest prevalence for men. Most ethnic groups showed little inter-gender diversity in diabetes prevalence except for Tongans where Tongan women had a prevalence >2 times that of Tongan men.

**Figure 1**—Figure 1 shows the proportion of total diabetes that were ‘newly’ diagnosed and ‘previously’ diagnosed by gender and Pacific ethnicity. Ratios of ‘previously’ to ‘newly’ diagnosed diabetes were 5:1 for Niueans, 4:1 for Samoans and Tongans, and 2:1 for Cook Islanders and 2:1 for European men and women combined.
Table 1. Comparison of CVD risk factors among men aged 35–74 years (age adjusted). Values are mean (SE) or percent

<table>
<thead>
<tr>
<th>Variables</th>
<th>European (n=863)</th>
<th>Samoan (n=246)</th>
<th>Tongan (n=123)</th>
<th>Niuean (n=49)</th>
<th>Cook Islands (n=46)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>51.7 (0.18)</td>
<td>46.6 (0.77)***</td>
<td>53.8 (2.51)†</td>
<td>48.0 (1.52)*</td>
<td>50.0 (4.26)</td>
</tr>
<tr>
<td>Current smokers (%)</td>
<td>15.2 % (0.01)</td>
<td>34.2 % (0.05)***</td>
<td>36.3 % (0.08)**</td>
<td>18.4 % (0.07)</td>
<td>19.2 % (0.06)</td>
</tr>
<tr>
<td>Never smokers (%)</td>
<td>46.8 % (0.02)</td>
<td>30.4 % (0.05)***</td>
<td>25.5 % (0.08)*</td>
<td>41.6 % (0.08)</td>
<td>49.3 % (0.12)</td>
</tr>
<tr>
<td>Inactive leisure time (%)</td>
<td>24.3 % (0.02)</td>
<td>45.2 % (0.05)***</td>
<td>39.3 % (0.08)</td>
<td>29.1 % (0.07)</td>
<td>47.2 % (0.10)*</td>
</tr>
<tr>
<td>Exercise (min/week) (tolerance)</td>
<td>66 (1.18)</td>
<td>26 (1.91)***</td>
<td>29 (2.67)</td>
<td>56 (2.22)</td>
<td>33 (2.36)</td>
</tr>
<tr>
<td>NZDep2001</td>
<td>4.4 (0.10)</td>
<td>7.9 (0.24)***</td>
<td>8.7 (0.30)***</td>
<td>7.9 (0.34)***</td>
<td>7.6 (0.45)***</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.6 (0.16)</td>
<td>33.0 (0.47)***</td>
<td>34.4 (0.88)***</td>
<td>32.0 (0.75)***</td>
<td>30.8 (0.81)*** †</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.7 (0.25)</td>
<td>173.5 (0.69)***</td>
<td>174.7 (0.85)*</td>
<td>173.0 (1.04)***</td>
<td>172.3 (1.78)**</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>0.93 (0.002)</td>
<td>0.94 (0.01)</td>
<td>0.97 (0.01)†</td>
<td>0.93 (0.01)</td>
<td>0.93 (1.01)</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>97.8 (0.39)</td>
<td>105.9 (1.11)***</td>
<td>110.1 (2.05)*** †</td>
<td>102.1 (1.57)*** †</td>
<td>100.2 (2.93)†</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>126 (0.62)</td>
<td>133 (2.44)***</td>
<td>125 (2.57)†</td>
<td>129 (2.55)</td>
<td>35 (3.24)**</td>
</tr>
<tr>
<td>Diastolic</td>
<td>78 (0.35)</td>
<td>83 (1.26)***</td>
<td>81 (1.55)</td>
<td>79 (1.67)</td>
<td>81 (1.81)</td>
</tr>
<tr>
<td>Serum lipids (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>5.61 (0.04)</td>
<td>5.28 (0.08)***</td>
<td>5.62 (0.19)</td>
<td>5.36 (0.17)</td>
<td>5.72 (0.23)</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>1.31 (0.01)</td>
<td>1.24 (0.03)*</td>
<td>1.12 (0.04)*** †</td>
<td>1.31 (0.05)</td>
<td>1.27 (0.05)</td>
</tr>
<tr>
<td>Ratio Tot/HDL</td>
<td>4.51 (0.05)</td>
<td>4.52 (0.14)</td>
<td>5.18 (0.24)*** †</td>
<td>4.26 (0.17)</td>
<td>4.63 (0.17)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.65 (0.04)</td>
<td>1.68 (0.12)</td>
<td>1.67 (0.19)</td>
<td>1.51 (0.16)</td>
<td>1.56 (0.14)</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>3.57 (0.03)</td>
<td>3.31 (0.09)**</td>
<td>3.77 (0.17)†</td>
<td>3.38 (0.15)</td>
<td>3.74 (0.22)</td>
</tr>
<tr>
<td>Microalbuminuria (mg/L)♦</td>
<td>4.11 (1.06)</td>
<td>7.30 (1.22)***</td>
<td>4.45 (1.28)††</td>
<td>5.53 (1.46)</td>
<td>7.26 (1.78)</td>
</tr>
<tr>
<td>Five-year risk score of CVD</td>
<td>6.8 % (0.15)</td>
<td>9.4 % (0.70)***</td>
<td>10.8 % (1.13)***</td>
<td>7.1 % (0.37)††</td>
<td>9.1 % (0.54)***</td>
</tr>
</tbody>
</table>

*0.01<p<0.05, **0.001<p<0.01, ***p<0.001 compared to European; †0.01<p<0.05, ††0.001<p<0.01, †††p<0.001 compared to Samoan, ♦ = geometric mean.
Table 2. Comparison of CVD risk factors among women aged 35–74 years (age adjusted). Values are mean (SE) or percent

<table>
<thead>
<tr>
<th>Variable</th>
<th>European (n=882)</th>
<th>Samoan (n=238)</th>
<th>Tongan (n=132)</th>
<th>Niuean (n=60)</th>
<th>Cook Islands (n=70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>52.1 (0.18)</td>
<td>46.9 (0.80)***</td>
<td>48.0 (1.67)*</td>
<td>46.2 (1.20)***</td>
<td>46.1 (1.02)***</td>
</tr>
<tr>
<td>Current smokers (%)</td>
<td>13.3 % (0.01)</td>
<td>18.0 % (0.04)</td>
<td>7.5 % (0.03) †</td>
<td>12.7 % (0.06)</td>
<td>30.5 % (0.09)</td>
</tr>
<tr>
<td>Never smokers (%)</td>
<td>53.9 % (0.02)</td>
<td>54.8 % (0.06)</td>
<td>84.0 % (0.05)*** †††</td>
<td>49.6 % (0.11)</td>
<td>38.4 % (0.08)*</td>
</tr>
<tr>
<td>Inactive leisure time (%)</td>
<td>23.3 % (0.02)</td>
<td>37.7 % (0.05)**</td>
<td>47.0 % (0.07)**</td>
<td>36.6 % (0.09)</td>
<td>45.8 % (0.08)*</td>
</tr>
<tr>
<td>Exercise (min/week) (tolerance)</td>
<td>59 (1.16)</td>
<td>23 (1.57)***</td>
<td>15 (2.31)**</td>
<td>37 (2.33)</td>
<td>17 (2.41)*</td>
</tr>
<tr>
<td>NZDep2001</td>
<td>4.5 (0.11)</td>
<td>8.5 (0.27)***</td>
<td>9.0 (0.16)***</td>
<td>8.0 (0.38)***</td>
<td>7.9 (0.63)***</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.3 (0.19)</td>
<td>36.3 (0.57)***</td>
<td>36.3 (1.05)***</td>
<td>35.3 (1.27)***</td>
<td>35.4 (0.78)***</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>163.5 (0.23)</td>
<td>160.8 (0.51)***</td>
<td>165.1 (0.87)†††</td>
<td>162.8 (0.76)†</td>
<td>160.3 (1.25)*</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>0.81 (0.002)</td>
<td>0.87 (0.01)***</td>
<td>0.87 (0.01)***</td>
<td>0.85 (0.01)*** ††</td>
<td>0.87 (0.01)***</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>87.1 (0.45)</td>
<td>105.1 (1.06)***</td>
<td>106.4 (1.73)***</td>
<td>101.0 (1.80)***</td>
<td>102.7 (2.00)***</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>116 (0.60)</td>
<td>128 (1.55)***</td>
<td>121 (3.85)</td>
<td>126 (2.64)***</td>
<td>128 (2.53)***82</td>
</tr>
<tr>
<td>Diastolic</td>
<td>72 (0.34)</td>
<td>78 (1.00)***</td>
<td>78 (1.59)***</td>
<td>79 (1.57)***</td>
<td>(2.97)**</td>
</tr>
<tr>
<td>Serum lipids! (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>5.50 (0.03)</td>
<td>5.40 (0.12)</td>
<td>5.45 (0.13)</td>
<td>5.01 (0.09)*** †</td>
<td>5.88 (0.34)</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>1.62 (0.01)</td>
<td>1.36 (0.03)***</td>
<td>1.31 (0.05)***</td>
<td>1.39 (0.06)***</td>
<td>1.31 (0.04)***</td>
</tr>
<tr>
<td>Ratio Tot/HDL</td>
<td>3.57 (0.04)</td>
<td>4.07 (0.10)***</td>
<td>4.36 (0.21)***</td>
<td>3.77 (0.15)</td>
<td>4.72 (0.40)**</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.20 (0.02)</td>
<td>1.30 (0.06)</td>
<td>1.39 (0.08)*</td>
<td>1.34 (0.08)</td>
<td>1.78 (0.27)*</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>3.33 (0.03)</td>
<td>3.43 (0.10)</td>
<td>3.51 (0.11)</td>
<td>3.01 (0.08)*** ††</td>
<td>3.76 (0.25)</td>
</tr>
<tr>
<td>Microalbuminuria (mg/L)</td>
<td>3.46 (1.06)</td>
<td>7.67 (1.51)**</td>
<td>5.20 (1.45)</td>
<td>3.53 (1.39)†</td>
<td>7.95 (1.40)***</td>
</tr>
<tr>
<td>Five-year risk score of CVD</td>
<td>3.0 % (0.10)</td>
<td>5.2 % (0.24)***</td>
<td>5.6 % (0.35)**</td>
<td>4.3 % (0.57)*</td>
<td>6.2 % (0.63)***</td>
</tr>
</tbody>
</table>

*0.01<p<0.05, **0.001<p<0.01, ***p<0.001 compared to European; †0.01<p<0.05, ††0.001<p<0.01, †††p<0.001 compared to Samoan, ♦ = geometric mean.
Table 3. Comparison of diabetes factors among men aged 35–74 years (age adjusted). Values are mean (SE) or percent

<table>
<thead>
<tr>
<th>Variables</th>
<th>European (n=863)</th>
<th>Samoan (n=246)</th>
<th>Tongan (n=123)</th>
<th>Niuean (n=49)</th>
<th>Cook Islands (n=46)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting</td>
<td>5.35 (0.04)</td>
<td>6.19 (0.18)***</td>
<td>6.02 (0.30)*</td>
<td>5.81 (0.20)*</td>
<td>5.74 (0.23)</td>
</tr>
<tr>
<td>2 hour</td>
<td>5.64 (0.08)</td>
<td>6.43 (0.41)</td>
<td>6.00 (0.38)</td>
<td>6.20 (0.57)</td>
<td>6.46 (0.69)</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.6 % (0.02)</td>
<td>6.5 (0.12)***</td>
<td>6.3 (0.15)***</td>
<td>6.2 (0.14)***†</td>
<td>6.3 (0.23)**</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>6.3 % (0.01)</td>
<td>26.2 % (0.04)***</td>
<td>17.8 % (0.06)*</td>
<td>14.9 % (0.05)</td>
<td>20.8 % (0.08)</td>
</tr>
<tr>
<td>Impaired Glucose Tolerance</td>
<td>8.4 % (0.01)</td>
<td>4.8 % (0.01)*</td>
<td>7.6 % (0.05)</td>
<td>5.7 % (0.02)</td>
<td>6.2 % (0.04)</td>
</tr>
<tr>
<td>Impaired Fasting Glucose</td>
<td>2.9 % (0.01)</td>
<td>3.1 % (0.01)</td>
<td>4.2 % (0.02)</td>
<td>4.7 % (0.03)</td>
<td>2.7 % (0.02)</td>
</tr>
</tbody>
</table>

*0.01<p<0.05, **0.001<p<0.01, ***p<0.001 compared to European; †0.01<p<0.05, ††0.001<p<0.01, †††p<0.001 compared to Samoan.

Table 4. Comparison of diabetes factors among women aged 35–74 years (age adjusted). Values are mean (SE) or percent

<table>
<thead>
<tr>
<th>Variables</th>
<th>European (n=882)</th>
<th>Samoan (n=238)</th>
<th>Tongan (n=132)</th>
<th>Niuean (n=60)</th>
<th>Cook Islands (n=70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting</td>
<td>5.02 (0.03)</td>
<td>6.00 (0.19)***</td>
<td>6.70 (0.69)*</td>
<td>5.53 (0.21)*</td>
<td>5.85 (0.25)**</td>
</tr>
<tr>
<td>2 hour</td>
<td>5.48 (0.07)</td>
<td>7.28 (0.49)***</td>
<td>9.03 (1.12)**</td>
<td>6.01 (0.47)</td>
<td>7.54 (0.50)**</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.5 % (0.02)</td>
<td>6.3 (0.12)***</td>
<td>6.6 (0.27)***</td>
<td>6.0 (0.10)***</td>
<td>6.2 (0.12)***</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>5.3 % (0.01)</td>
<td>25.3 % (0.05)***</td>
<td>35.8 % (0.07)***</td>
<td>10.8 % (0.03)†</td>
<td>17.8 % (0.04)**</td>
</tr>
<tr>
<td>Impaired Glucose Tolerance</td>
<td>15.4 % (0.01)</td>
<td>12.1 % (0.02)**</td>
<td>6.4 % (0.03)</td>
<td>7.1 % (0.03)</td>
<td>13.9 % (0.04)</td>
</tr>
<tr>
<td>Impaired Fasting Glucose</td>
<td>1.6 % (0.01)</td>
<td>5.1 % (0.03)</td>
<td>1.4 % (0.01)</td>
<td>8.2 % (0.04)</td>
<td>2.3 % (0.02)</td>
</tr>
</tbody>
</table>

*0.01<p<0.05, **0.001<p<0.01, ***p<0.001 compared to European; †0.01<p<0.05, ††0.001<p<0.01, †††p<0.001 compared to Samoan.
Discussion

These results reinforce the huge health disparities that exist between European and Pacific New Zealanders. Furthermore, they clearly show that there are important differences in the cardiovascular disease (CVD) risk profile and diabetes prevalence between Pacific ethnic groups. Niuean men and women had the healthiest profile. They had the lowest inactivity levels, highest average exercise times, smallest waist to hip ratios, and the healthiest lipid profiles resulting in the lowest CVD risk scores of the Pacific groups.

For diabetes, Niuean men and women also had the lowest HbA1c, and prevalence of total and previously diagnosed diabetes. This finding reflects the more favourable health-related socioeconomic profile that Niueans have compared to other Pacific ethnic groups as reported earlier.6

Among women, Samoans had the next best CVD risk profiles, followed by Tongans and Cook Islanders, however for diabetes prevalence the order followed a trend more similar to the SES profile already highlighted elsewhere6 with Cook Islanders having the next lowest prevalence followed by Samoans and Tongans.

Among men, Cook Islanders had the next best CVD risk profile to Niueans, then Samoans, and finally Tongans; however for diabetes, the next lowest prevalence was observed in Tongan followed by Cook Islanders and Samoans.
Unexpectedly, Cook Islands women had the poorest CVD profile despite their high SES and lengthy residence in New Zealand. This may be attributed to their higher level of smoking. However, they had the lowest prevalence of diabetes aside from Niueans.

The only other studies that offer comparable data are the Workforce Diabetes Survey (WDS)15 from 1988–1990 and the South Auckland Diabetes Project (SADP)7 from 1991–1994. Workforce based surveys, however, are not directly comparable to the DHAH due to a possible ‘healthy worker’ bias. Age groupings also differ between these studies which would compromise comparability, although all statistics are age adjusted. With these caveats, overall BMI levels in men were higher in the present study compared to the WDS survey.

Compared to the WDS, all ethnic groups had higher proportions of diabetes. The prevalence for both Cook Islanders and Niueans doubled, Samoans tripled, whilst the Tongan prevalence was more than 6 times higher than those measured 13 years earlier.

Comparing measures of known diabetes (previously diagnosed) to the South Auckland Diabetes Project (SADP) carried out between 1991–94 demonstrated a similar pattern. Niueans reported the smallest difference (1.4 times higher in the current study), Cook Islanders were intermediate at about double the SADP prevalence, whilst the Tongan and Samoan groups reported prevalence was approximately 4 times higher.

The Samoan and Tongan communities have on average resided in New Zealand for a shorter period than the Niuean and Cook Islands communities and therefore may be more at risk of negative health consequences that come with migration, especially from countries more accustomed to traditional lifestyles.

Samoans, Tongans, and Niueans had similar ratios of known diabetes to undiagnosed diabetes (4:1; 4:1; 5:1 respectively). The Cook Islands group however had a smaller ratio (2:1) than the other Pacific groups as did Europeans. A larger ratio indicates better screening of diabetes as fewer cases are left undetected. The large ratios of the Samoan, Tongan, and Niuean groups could be due to aggressive screening for diabetes of Pacific providers and the higher use of Pacific providers by these groups.

The smaller ratio of the Cook Islands group, in spite of a relatively high prevalence of diabetes, may be due to lower utilisation of Pacific providers, and poorer screening of diabetes in Pacific people from mainstream health providers.

The Cook Islands community are more geographically spread throughout New Zealand than other Pacific ethnic groups.16 Of the four largest Pacific groups, the Cook Islands population had the smallest proportion (60%) living in the Auckland region. This pattern may also occur within the Auckland region as well, and if so, could decrease the use of Pacific providers of Cook Islanders as they are less likely to live in areas of high Pacific density.

Thus Cook Islanders are more likely to access mainstream health providers. Higher levels of integration/acculturation into NZ society as a result of their lengthier stay, could also contribute to their lower use of Pacific providers. The same argument could
be put forward for Niueans, however they are most likely to live in the Auckland region (80%), and have the lowest prevalence of diabetes of the Pacific ethnic groups.

Within the gender groupings, more marked differences in CVD risk factors and diabetes prevalence were observed in Pacific women. These differences do not appear to be moderated by differences in BMI, as BMI Pacific in women were within 1 BMI unit of each other as opposed to 3.6 BMI units in Pacific men. This finding may indicate that monitoring the health of Pacific women better determines ethnic disparities in health.

In men, all Pacific groups had lower smoking prevalence by more than 10% compared to the 1988-90 WDS study, and Tongans by 20% (however they still remained most likely to smoke). Among women a rise in smoking prevalence of 10% was reported by Cook Islanders. However, all other groups reported similar levels to the WDS. Sex-specific prevalence mirrored those found in the Pacific where ‘Tongan men are most likely to smoke and Niuean men least, and Cook Islands women are most likely to smoke and Tongan women least. All groups reported higher exercise levels compared to the WDS. This is unexpected considering that the WDS was workforce based. An altered social definition/perception of what constitutes ‘activity’ and the positive aspects that come with it, could in part be responsible for this difference considering the time-frames and settings.

Compared to the WDS, systolic blood pressure was higher in all groups with the exception of Tongan). Diastolic blood pressure was slightly higher in all groups except for Cook Island men. These measures contrast with trends of the general New Zealand population previously reported, that showed a decrease in blood pressure in New Zealand over time, however these trends are from a non-Polynesian sample.

Niueans had the healthiest lipid profile for both men and women. All ethnic groups had a better profile compared to levels measured in 1988–90. This trend is similar to those of greater NZ and maybe a result of national public health measures that have encouraged lower cholesterol consumption through food regulation and health promotion. As in the WDS survey, Pacific women’s HDL cholesterol levels were significantly lower than European women’s. However, for men this trend was only observed for the Samoan and Tongan groups.

Pacific people’s levels of microalbuminuria were generally higher than Europeans. Samoan men and Samoan and Cook Island women had significantly higher levels of microalbuminuria than their European counterparts. Compared to levels from the 1988–90 WDS all groups had lower levels of microalbuminuria in the current study.

Elevated microalbuminuria concentrations have been associated with obesity, high BP, and triglyceride concentrations. The lower levels of microalbuminuria measured in this study is reassuring and may indicate positive gains that are being made in the prevention of CVD in Pacific peoples.

A limitation of this study is that using electoral roll based and cluster sampling frames did not allow for ethnic specific response rates to be determined. Although the overall response rate was not as high as in previous Auckland risk factor studies, it has been shown in the Atherosclerosis Risk in Communities Study that response rates lower...
than those in our study produced relatively small errors in the estimates of prevalence of common cardiovascular disease risk factors.

Caution should be taken when interpreting findings related to the smaller Pacific ethnic groups (Cook Islands, and Niuean) due to the higher proportion of participants that identified with more than one ethnic group. For the larger groups (Samoan and Tongan) approximately 5% reported affiliation to more than one ethnic group, however for the Niuean and Cook Islands groups this was approximately 11%.

Summary

These results reinforce the huge health disparities that exist between European and Pacific New Zealanders and clearly show that there are important differences in the cardiovascular disease (CVD) risk profile and diabetes prevalence between Pacific ethnic groups.

Niuean men and women had the healthiest risk profiles. There were marked differences in both CVD risk factor and diabetes prevalence by gender and Pacific Island groups. These differences are likely to be explained in part by SES and migrant histories.

The high ratio of known to undiagnosed diabetes suggests that screening programmes in Samoan, Tongan, and Niuean communities have been successful. This could in part be due to ‘by Pacific for Pacific’ services provided by Pacific Primary Health Care Organisations (PHO) as well as more robust screening of Pacific people by mainstream healthcare services. However for Cook Islanders there is evidence that more aggressive screening for diabetes is needed.

For all Pacific communities, the high levels of CVD risk factors highlights the need for greater focus on CVD risk assessment and management. Addressing smoking and physical activity levels are also a priority for specific groups of the Pacific community.

Competing interests: None known.

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References:

The accuracy of ethnicity data in primary care and its impact on cardiovascular risk assessment and management—PREDICT CVD-8

Tania Riddell, Graeme Lindsay, Tim Kenealy, Rod Jackson, Sue Crengle, Dale Bramley, Susan Wells, Roger Marshall

Abstract

**Background** Accurate ethnicity data are a prerequisite for evidence-based cardiovascular risk assessment and management according to national guidelines.

**Aims** (i) To investigate the accuracy of ethnicity data in primary care medical records by comparing them with self-identified ethnicity. (ii) To determine the clinical impact of ethnicity misclassification on cardiovascular risk assessment and management.

**Methods** A random sample of 870 patients from 18 general practices (who had ethnicity collected from their medical record as part of cardiovascular risk assessment using PREDICT, a web-based decision support tool) were sent a postal questionnaire asking their self-identified ethnicity using the 2001 Census ethnicity question.

**Results** Data were available for 665 people (77% response rate) who completed the postal questionnaire. Ethnicity in the primary care record and self-identified ethnicity from the questionnaire were identical for 68% of respondents at Statistics New Zealand Level 2 coding. Data concordance varied from 9.8% for the non-New Zealand European ethnic group to 90.9% for New Zealand European. The primary care record agreed with self-identified ethnicity for 64.9% of Māori respondents. Fortunately, when the same ethnicity data were categorised using the Statistics New Zealand ethnic group prioritisation rules and applied within PREDICT, which adds a risk weighting for Māori, Pacific, and Indian subcontinent peoples, the impact of misclassification was small. The main reason was that about half of misclassifications occurred between ethnic groups classified in the same high cardiovascular risk category. For about 6% of Māori, Pacific, and Indian subcontinent people in our study this misclassification could potentially have delayed risk assessment and resulted in under-treatment. In contrast, about 1.5% of those with other ethnicities may have undergone a premature risk assessment and been over-treated.

**Conclusion** The clinical impact of ethnicity misclassification on cardiovascular risk assessment and management in primary care is modest because much of the misclassification does not alter cardiovascular risk classification. Nevertheless, efforts to improve the accuracy of ethnicity classification in primary care need to continue in order to support the sector’s ability to monitor health service utilisation, outcomes, and performance related indicators.

The capture of ethnicity data in New Zealand has historically posed a significant challenge to the quality of our health statistics. Primary care is usually the first point of contact with health services and as the healthcare provider patients see most is
ideally placed to collect comprehensive accurate patient data, including ethnicity data. However, prior to the introduction of the Ethnicity Data Protocols for the Health and Disability Sector in 2004 there were no standards for the collection, coding and recording of ethnicity data in primary care.

Ethnicity data, if collected at all, have been recorded in an inconsistent and often idiosyncratic way. This has been cause for concern for health researchers who monitor Māori health inequalities in New Zealand.

National guidelines recommend that people who identify with the ethnic groups Māori, Pacific, and Indian subcontinent peoples:

- Undergo a cardiovascular risk assessment 10 years earlier than other ethnicities (for example, Māori men at age 35 years; Māori women at age 45 years); and,
- Have their calculated 5-year CVD risk adjusted upward by 5% (for example a 5-year CVD risk of 15% for a Māori man would be adjusted to 20%).

(*Where Indian subcontinent peoples include Indian, Fijian Indian, Afghani, Bangladeshi, Nepalese, Pakistani, Sri Lankan, and Tibetan.)

PREDICT, a web-based clinical decision support programme for cardiovascular disease (CVD) based on New Zealand guidelines was implemented in ProCare, a large Auckland PHO, from 2002. Implementation was undertaken progressively over several years.

The ProCare Network (Auckland, Manukau, and Waitemata) comprise approximately 178 general practices and 480 general practitioners; it has the largest enrolled Primary Health Organisation (PHO) population and the largest enrolled Māori population in New Zealand.

In this study we undertook a survey to compare self-identified ethnicity with primary care ethnicity to determine the impact of misclassification on cardiovascular risk assessment and management recommendations.

Methods

General practices—Practices were eligible if they met the following study criteria: belonged to or were affiliated with ProCare; used MedTech patient management system; had performed 20 or more cardiovascular risk assessments using PREDICT in the 12-month period to 30 June 2005; and, had at least one permanent general practitioner or long-term locum. From this group, practices were randomly sampled using MS Excel’s random numbers table function.

Patients—Patients were eligible if they had undergone a PREDICT-CVD risk assessment in the 12-month period 1 July 2004–30 June 2005; and if they had sufficient patient details to enable a mail-out of the questionnaire. Where a patient had more than one PREDICT assessment undertaken the most recent record was analysed.

Patient ethnicity data—At the practices, patient ethnicity data were extracted from the PREDICT record retained in the patient management system (PMS). Although ethnicity data, where available, were automatically drawn into PREDICT from the PMS, the practitioner also has the ability to manually enter or change a patient’s ethnicity within the PREDICT template.

Patient self-identified ethnicity data were collected via a postal questionnaire. The questionnaire used the ethnicity question recommended by the 2004 Ethnicity Data Protocols for the Health and Disability Sector—i.e. the 2001 Census ethnicity question that has been rigorously tested by Statistics New Zealand (Appendix 1).
Ethnicity grouping for analysis—Patients were assigned to four ethnic group categories that, according to the national guidelines are at higher risk (Māori, Pacific, and Indian subcontinent ethnic groups) or lower risk (‘All other’ ethnic groups) of developing CVD.

PREDICT uses the rule set of Statistics New Zealand Level 2 codes (Appendix 2: code 21 for Māori, 30–37 for Pacific, 43 and 44 for Indian subcontinent ethnicities but excluding Japanese and Korean, and codes 10–12, 40–42 and 51–54 for All other ethnic groups).

Sampling and questionnaire process—Sample size calculations suggested that, if 10% of patients were misclassified for ethnicity, a sample of 600 people would have given classification estimates within 2.5% of the true value (with 95% confidence). To allow for non-response we aimed to sample 800 people.

A 20% random sample of eligible patients in each participating practice were sent a combined letter/questionnaire, a participant information sheet and freepost return address envelope. The ethnicity question was the only information requested. A second mail-out was conducted and subsequent telephone follow-up of non-responders undertaken one month following the second mail-out.

Ethical approval—The PREDICT project was approved by the Auckland Ethics Committee (AKY/03/12/314).

Results

Eighteen of the 29 ProCare general practices that met the study eligibility criteria were randomly selected and invited to participate. Two practices declined and two other practices from the remaining 11 eligible practices were randomly selected and consented. The selected practices came from the wider Auckland region representing the ProCare Network. All were group general practices except for one Accident and Medical clinic.

There were a total of 4373 people from the 18 practices with a recorded PREDICT CVD risk assessment in the 12-month period to 30 June 2005. Of these patients, 22 were ineligible (16 deceased, 6 mock or test patients) for the questionnaire mail-out.

A random sample of 20% from each practice (n=870) were sent the postal questionnaire. Of these, 669 returned completed questionnaires (77% response rate); 665 had ethnicity data in their primary care record; and 4 did not have sufficient data for comparative purposes. There were statistically significant differences between questionnaire responders (n=669) and non-responders (n=201) with regard to ethnicity and age.

The European ethnic group had a significantly higher response rate (82.6%) than any other ethnic group. The response rates for the non-European ethnic groups were not significantly different from each other—Indian subcontinent (72.4%), Māori (68.7%), Pacific (66.7%), and Other ethnic groups (64.3%). Responders were older (median age group for responders was 55-64 years compared to median age group of 45-54 years for non-responders).

Table 1 shows a classification of self-identified ethnicity codes against codes recorded in the PREDICT database. For about two-thirds of the sample the primary care ethnicity record agreed with self-identified ethnicity at the Statistics New Zealand Level 2 coding hierarchy. Agreement varied from a low of 0% notably for the ethnic descriptions for which ‘Not further defined’ applied, to a high of about 90% for New Zealand European. Agreement for Māori was below that of the aggregated sample.
Table 1. Agreement between primary care and self-identified ethnicity, in 665 patients, at Statistics New Zealand Level 2 codes and descriptions

<table>
<thead>
<tr>
<th>Ethnicity codes</th>
<th>Ethnicity descriptions</th>
<th>N</th>
<th>Agreement* with self-identification n (%)</th>
<th>Disagreement with self-identification n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>European NFD#</td>
<td>82</td>
<td>0 (0)</td>
<td>82 (100.0)</td>
</tr>
<tr>
<td>11</td>
<td>New Zealand European</td>
<td>320</td>
<td>291 (90.9)</td>
<td>29 (9.1)</td>
</tr>
<tr>
<td>12</td>
<td>Other European</td>
<td>50</td>
<td>13 (26.0)</td>
<td>37 (74.0)</td>
</tr>
<tr>
<td>21</td>
<td>Māori</td>
<td>57</td>
<td>37 (64.9)</td>
<td>20 (35.1)</td>
</tr>
<tr>
<td>30</td>
<td>Pacific people NFD#</td>
<td>8</td>
<td>0 (0)</td>
<td>8 (100.0)</td>
</tr>
<tr>
<td>31</td>
<td>Samoan</td>
<td>40</td>
<td>34 (85.0)</td>
<td>6 (15.0)</td>
</tr>
<tr>
<td>32</td>
<td>Cook Island Māori</td>
<td>16</td>
<td>15 (93.8)</td>
<td>1 (6.2)</td>
</tr>
<tr>
<td>33</td>
<td>Tongan</td>
<td>23</td>
<td>20 (87.0)</td>
<td>3 (13.0)</td>
</tr>
<tr>
<td>34</td>
<td>Niuean</td>
<td>7</td>
<td>7 (100.0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>35</td>
<td>Tokelauan</td>
<td>0</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>36</td>
<td>Fijian</td>
<td>8</td>
<td>3 (37.5)</td>
<td>5 (62.5)</td>
</tr>
<tr>
<td>37</td>
<td>Other Pacific peoples</td>
<td>0</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>40</td>
<td>Asian NFD#</td>
<td>6</td>
<td>0 (0)</td>
<td>6 (100.0)</td>
</tr>
<tr>
<td>41</td>
<td>Southeast Asian</td>
<td>3</td>
<td>3 (100.0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>42</td>
<td>Chinese</td>
<td>14</td>
<td>8 (57.1)</td>
<td>6 (42.9)</td>
</tr>
<tr>
<td>43</td>
<td>Indian</td>
<td>17</td>
<td>17 (100.0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>44</td>
<td>Other Asian</td>
<td>6</td>
<td>0 (0)</td>
<td>6 (100.0)</td>
</tr>
<tr>
<td>51</td>
<td>Middle Eastern</td>
<td>1</td>
<td>0 (0)</td>
<td>1 (100.0)</td>
</tr>
<tr>
<td>52</td>
<td>Latin American/Hispanic</td>
<td>1</td>
<td>1 (100.0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>53</td>
<td>African</td>
<td>3</td>
<td>3 (100.0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>54</td>
<td>Other</td>
<td>3</td>
<td>0 (0)</td>
<td>3 (100.0)</td>
</tr>
<tr>
<td>All ethnic groups combined</td>
<td>665</td>
<td>452 (68.0)</td>
<td>213 (32.0)</td>
<td></td>
</tr>
</tbody>
</table>

*Agreement = complete agreement between ethnicity codes and descriptions in MedTech and the self-identified ethnicity/ethnicities at Statistics New Zealand Level 2; #NFD = Not further defined.

Table 2 shows the same data reclassified into four main ethnic categories with respect to the degree of cardiovascular risk for different ethnic groups in the national guidelines. The process of collapsing down to broad higher risk or lower risk ethnicity groups increased the overall agreement level to 95.8% (Kappa statistic 0.91, good agreement).
Table 2. Primary care ethnicity compared to self-identified ethnicity, in 665 patients, with a PREDICT cardiovascular risk assessment

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Patient questionnaire</th>
<th></th>
<th>Indian subcontinent</th>
<th>All others</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary care record</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori#</td>
<td>48</td>
<td>4</td>
<td>0</td>
<td>5*</td>
<td>57</td>
</tr>
<tr>
<td>Pacific#</td>
<td>3</td>
<td>95</td>
<td>3</td>
<td>1*</td>
<td>102</td>
</tr>
<tr>
<td>Indian subcontinent#</td>
<td>0</td>
<td>0</td>
<td>21</td>
<td>1*</td>
<td>22</td>
</tr>
<tr>
<td>All others#</td>
<td>4+</td>
<td>5+</td>
<td>2+</td>
<td>473</td>
<td>484</td>
</tr>
<tr>
<td>Total</td>
<td>55</td>
<td>104</td>
<td>26</td>
<td>480</td>
<td>665</td>
</tr>
</tbody>
</table>

* These patients would be risk assessed 10 years earlier than, and given an absolute 5-year risk 5% more than, recommended by the guidelines; + These patients would be risk assessed 10 years later than, and given an absolute 5-year risk 5% less than, recommended by the national guidelines for cardiovascular risk assessment and management; # Patients reporting Māori ethnicity in addition to any other ethnicity are classified as Māori; patients reporting Pacific ethnicity, except Māori, are classified as Pacific.

In the same table, 87.3% (48/55) of self-identifying Māori were correctly assigned as Māori in their primary care record. For Pacific and Indian subcontinent groups, 91.3% and 80.8% were respectively classified correctly according to their self-identifying ethnicity.

Discussion

This study found the accuracy of ethnicity data in the primary care record was limited when directly compared to self-identified ethnicity at the Statistics New Zealand Level 2 code.

About one-third of Māori in our sample had ethnicity data that did not fully agree with their self-identified ethnicity suggesting ongoing systematic misclassification of Māori in primary care records. In some cases, misclassification was due to the failure of primary care data to capture multiple ethnicities.

Therefore analyses of primary care data using Statistics New Zealand Level 2 ethnicity codes and descriptions may be misleading. This makes the monitoring of ethnic differentials in health status, access to health services, and evaluation of health outcomes (including performance-related indicators and health service development) questionable.

When these data were reclassified, using ethnic group prioritisation rules, into high CVD risk ethnic groupings (i.e. Māori, Pacific, and Indian subcontinent peoples) or ‘All others’ (lower CVD risk ethnic groups), according to national guidelines for cardiovascular risk assessment and management, the potential clinical impact was less than would be expected given the degree of misclassification.

The reason for this lesser impact was that about half of the misclassifications were between ethnicities within the high CVD risk ethnic groupings. Nevertheless, in this sample a total of 11 people who self-identified as Māori, Pacific, or Indian subcontinent ethnicity would have been misclassified into the lower risk ethnic grouping (‘All others’); potentially had their risk assessment undertaken 10 years later.
than recommended; their 5-year cardiovascular risk under-estimated by 5%; and, been under-treated as a consequence.

A much smaller proportion—1.5% of ‘All others’—may have had their risk assessment undertaken 10 years early and their risk overestimated because of misclassification into the high-risk ethnicity groups. Although the potential impact of misclassification may appear small it must be considered in the context of stark ethnic inequalities in CVD morbidity and mortality in New Zealand.

Reducing these inequalities requires a broad range of approaches. High quality ethnicity data, and associated improvements in clinical management, will contribute to reducing these inequalities.

This study is one of the first in New Zealand to directly compare the accuracy of primary care records with self-identified ethnicity.

Another study recently compared the ethnicity data of children on the National Immunisation Register (where data were collected from parents via a protocol-based informed consent process in the Meningococcal B campaign) with ethnicity data on PHO registers. There was significant ethnic group misclassification in the primary care record for about one-third of Māori, a quarter of Pacific and a fifth of Asian children. There are also discrepancies in ethnicity recording among non-European ethnic groups in New Zealand hospital records compared to self-identified ethnicity. However agreement between two independent databases (PREDICT and the National Health Index) is moderately good.

Other researchers have reported that the coverage of ethnicity data recording in primary care in New Zealand has improved since the establishment of PHO registers.

Our study has some limitations. Its generalisability may be limited due to the sample coming from one primary care network, albeit the largest in New Zealand, and the lack of precision due to small numbers in some ethnic groups. There were significant differences between questionnaire responders and non-responders, and even with a response rate of 77%, non-response may have contributed to selection bias. For example, we may still have under-estimated the level of misclassification of Māori in primary care records due to the higher proportion of Māori compared to European non-responders.

Ethnicity data reflect important dimensions of difference and power in New Zealand society. It is not acceptable that Māori are differentially affected by ethnicity data misclassification. Doctors, nurses, healthcare managers, policymakers, and researchers must be cognisant of this and the limitations of ethnicity data in routinely collected health datasets.

Protocols recommended by the Ministry of Health for the standardised collected of ethnicity data throughout the health sector should be nationally implemented and evaluated. In addition, with the increasing use of electronic registers to identify patients eligible for interventions and the expanding use of electronic decision-support tools, that incorporate ethnicity data from medical records, the accuracy of these data will become more and more clinically important.
PHOs and general practices need to be supported and resourced to overcome the barriers to quality ethnicity data collection and systematic assessment of their at-risk patient populations.

Competing interests: None known.

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References:

Appendix 1. New Zealand 2001 Census ethnicity question and standard ethnicity question for the health sector

Appendix 2. Statistics New Zealand ethnic group priority order

<table>
<thead>
<tr>
<th>Priority order</th>
<th>Level 2 ethnic group code</th>
<th>Ethnic group description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>21</td>
<td>Māori</td>
</tr>
<tr>
<td>2</td>
<td>35</td>
<td>Tokelauan</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>Fijian</td>
</tr>
<tr>
<td>4</td>
<td>36</td>
<td>Niuean</td>
</tr>
<tr>
<td>5</td>
<td>33</td>
<td>Tongan</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>Cook Island Māori</td>
</tr>
<tr>
<td>7</td>
<td>31</td>
<td>Samoan</td>
</tr>
<tr>
<td>8</td>
<td>37</td>
<td>Other Pacific Island</td>
</tr>
<tr>
<td>9</td>
<td>30</td>
<td>Pacific Island NFD#</td>
</tr>
<tr>
<td>10</td>
<td>41</td>
<td>South East Asian</td>
</tr>
<tr>
<td>11</td>
<td>43</td>
<td>Indian</td>
</tr>
<tr>
<td>12</td>
<td>42</td>
<td>Chinese</td>
</tr>
<tr>
<td>13</td>
<td>44</td>
<td>Other Asian</td>
</tr>
<tr>
<td>14</td>
<td>40</td>
<td>Asian NFD#</td>
</tr>
<tr>
<td>15</td>
<td>52</td>
<td>Latin American / Hispanic</td>
</tr>
<tr>
<td>16</td>
<td>53</td>
<td>African</td>
</tr>
<tr>
<td>17</td>
<td>51</td>
<td>Middle Eastern</td>
</tr>
<tr>
<td>18</td>
<td>54</td>
<td>Other</td>
</tr>
<tr>
<td>19</td>
<td>12</td>
<td>Other European#</td>
</tr>
<tr>
<td>20</td>
<td>10</td>
<td>European NFD#</td>
</tr>
<tr>
<td>21</td>
<td>11</td>
<td>NZ European</td>
</tr>
</tbody>
</table>

Source: Table adapted from Ethnicity Data Protocols for the Health and Disability Sector; #NFD = Not further defined
Comparison of three different methods of assessing cardiovascular disease risk in New Zealanders with Type 2 diabetes mellitus

Patricia A Metcalf, Susan Wells, Robert K R Scragg, Rod Jackson

Abstract

Aim To compare three methods of assessing 5-year absolute risk of cardiovascular disease (CVD) in adults with type 2 diabetes; the Framingham CVD equation, the UK Prospective Diabetes Study (UKPDS) coronary heart disease plus stroke equations and the New Zealand Guidelines Group (NZGG)-modified Framingham CVD equation.

Methods Participants were 423 people with newly (n=118) or previously diagnosed (n=305) Type 2 diabetes mellitus aged 35 to 74 years with no past history of cardiovascular disease or nephropathy from an interviewed study population of 4049 adults. Absolute 5-year CVD risks were calculated in 5-year age bands by gender; Māori, Pacific, and European ethnicity; and newly and previously diagnosed diabetes.

Results The mean 5-year CVD risk score was 2.9% (95%CI: 2.40–3.42; p<0.0001) lower for the UKPDS risk engine compared to the original Framingham equation in absolute terms, and 7.6% (95%CI: 7.05–8.08; p<0.0001) lower than the NZGG-modified Framingham equation. In general, 5-year CVD risks were highest using the NZGG-modified equation, intermediate using the original Framingham equation and lowest using the combined UKPDS coronary heart disease plus stroke equations, in all age groups by gender, ethnicity, and time of diagnosis of Type 2 diabetes. However, the 5-year CVD risks are themselves potentially low as they include treated blood pressure and lipid values. Compared to the UKPDS 15% level of risk, the NZ Guidelines modified 15% level of risk results in people with diabetes being recommended for CVD drug management 10 to 17 years earlier.

Conclusions In general, among people with Type 2 diabetes, the Framingham equations showed higher 5-year CVD risk estimates compared to combined UKPDS coronary heart disease plus stroke equations and the NZGG-modified Framingham equation showed the highest 5-year CVD risks. In practice, people with type 2 diabetes will be managed earlier and more intensively based on their risk estimated by the current NZGG guidelines than if the UKPDS or original Framingham equations were used.

In 1991, Anderson et al published an updated equation for predicting CVD risk from the Framingham Heart Study of people aged 30 to 74 years originally free of CVD that included a coefficient for presence of diabetes. However, various groups recommend intensive drug treatment at lower levels of CVD risk in people with diabetes due to concerns that the Framingham risk equation underestimates CVD risk in these patients.
To address the lack of a risk equation specifically designed for people with Type 2 diabetes, the United Kingdom Prospective Diabetes Study (UKPDS) published an equation for predicting absolute risk of new coronary heart disease (CHD) events, and a separate prediction equation for stroke events. In contrast to the Framingham equations, the UKPDS CHD equation replaces age with age at diagnosis, adds time since diagnosis of diabetes, and includes HbA1c. They reported that the UKPDS CHD and Framingham CHD equations produced similar 10-year coronary heart disease risk estimates for women, but that the Framingham equation substantially underestimated coronary heart disease risk in men.

In 2003, the New Zealand Guidelines Group (NZGG) published guidelines for assessing cardiovascular risk for people living in New Zealand. They used a Framingham equation for cardiovascular rather than coronary risk and because it was thought that the equation underestimated risk in some population groups, added a once only 5% upward risk adjustment for people meeting certain criteria. For those with diabetes, this upward risk adjustment was made if they had diabetes over 10 years duration, or an HbA1c consistently over 8% or had microalbuminuria. Other criteria for the upward adjustment included being of Māori, Pacific, or Indian subcontinent (or South Asian that includes Indian, Fijian Indian, Sri Lankan, Pakistani, Bangladeshi, Nepali, Afghan, and Tibetan) ethnicity, having a family history of premature cardiovascular disease or having the metabolic syndrome.

We calculated and compared the 5-year CVD risks in people with Type 2 diabetes using the Framingham CVD, UKPDS CHD plus stroke and the NZGG-modified Framingham CVD equations.

Participants and Methods
The Auckland Diabetes, Heart and Health Survey was carried out between December 2001 and November 2003. Adults aged 35 to 74 years were recruited from two sampling frames: one was a cluster sample where random starting point addresses were obtained from Statistics New Zealand and the probability of selection was proportional to the number of people living in that census mesh block (response rate 61.3%); and the other was a random sample taken from the November 2000 Auckland electoral rolls stratified into 5-year age bands and included all people living in the Auckland area (response rate 60%).

Participants fasted from 10pm the evening before the interview and collected a first morning urine sample which they brought with them to the study centre that morning. A 75-gram oral glucose tolerance test was carried out in participants who had not been previously diagnosed with diabetes, and a fasting and 2-hour postvenous blood samples were collected for glucose measurement. Plasma glucose was measured using an enzymatic method (Roche Products [NZ]). Participants were classified as having newly diagnosed diabetes mellitus using 1998 World Health Organization (WHO) criteria using fasting glucose ≥7.0 mmol/L or 2-hour post glucose load of ≥11.1 mmol/L for diabetes. Serum cholesterol was measured using the enzymatic methods of Allain et al, and HDL-cholesterol was measured using a combination of a polyion and a divalent cation (Roche). Urinary albumin was measured using an immunoturbidimetric method. Haemoglobin A1c was measured by High Performance Liquid Chromatography on a Biorad Variant II instrument.

The inter-batch percentage coefficients of variation for low control material were glucose 2.1, cholesterol 1.4, HDL 2.0, and HbA1c 1.7; those of abnormal control were glucose 1.3, cholesterol 1.2, HDL 2.7, and HbA1c 2.1. An Omron-Hem-706 oscillometric blood pressure pulse monitor was used to
measure blood pressure twice in the sitting position after a rest of more than 5 minutes and the average calculated.

Out of the 4049 participants interviewed, 584 participants had newly or previously diagnosed diabetes mellitus and were aged greater than 20 at diagnosis (those diagnosed prior to age 20 were excluded). We excluded 98 people who had a past history of cardiovascular disease events, a further 25 with urinary albumin >300 mg/L (NZGG threshold for overt nephropathy) and 38 Asian people due to their small numbers, leaving 423.

Five-year coronary heart disease risks were calculated using the Framingham CVD\(^1\) equation, combining the UKPDS56\(^3\) CHD equation and the UKPDS60\(^4\) stroke equation, and the NZGG\(^5\)-modified Framingham CVD equation. The following equations were used to calculate the Framingham CVD risk\(^1\) estimates:

\[
\begin{align*}
\mu &= 18.8144 - 1.2146 \times \text{female} - 1.8443 \times \log(\text{age}) + 0.3668 \times \text{female} \times \log(\text{age}) - 1.4032 \times 
\log(\text{systolic blood pressure}) - 0.3899 \times \text{smoker} - 0.5390 \times 
\log(\text{cholesterol/HDL}) - 0.3036 \times \text{diabetes} - 0.1697 \times 
\text{diabetes} \times \text{female}.
\end{align*}
\]

\[
\sigma = \exp(0.6536 - 0.2402 \times \mu)
\]

5-year risk (%)=\((1 - \exp((-\log(5) - \mu)/\sigma))\times100.
\]

Where log is the natural logarithm, diabetes = 1 for people with previously diagnosed diabetes, and 0 for people with newly diagnosed diabetes, female is 1 for a female and 0 for a male, smoker is 1 for a current cigarette smoker and 0 for a non-smoker and chol/HDL is total cholesterol ÷ HDL-cholesterol. If a person with a past history of CHD or stroke has a 5-year risk <20% then the risk is set at 20%.

The NZGG-modified guidelines adds 5% (once only) if the person has any of the following factors: Māori, Pacific, or Indian ethnicity, triglycerides >8 mmol/L, cholesterol/HDL ratio >8, systolic blood pressure >170 mmHg and diastolic blood pressure >100 mmHg, urinary albumin \(\geq\) 20 mg/L, HbA\(_1c\) \(\geq\) 8%, the presence of the metabolic syndrome or diabetes duration \(\geq\) 10 years.

The UKPDS CHD\(^3\) risk was calculated using the following equations:

\[
q = 0.0112 \times 1.059^{(\text{age at diagnosis} - 55)} \times 0.525^{(1.350^{\text{smoker}} \times 1.144^{(\text{HbA}\_1c - 6.72)} \times 1.073^{(\text{bpsys} - 135.7)^{10}} \times 3.110^{\log(\text{cholesterol/HDL}) - 1.59})} \times 1.078^{(\text{duration})}.
\]

UKPDS 5-year CHD risk (%) = (1 – \exp((-q \times (1 – 1.078^5)/(1 – 1.078)))) \times 100.

Where sex = 1 for a female and 0 for a male, bpsys is systolic blood pressure in mmHg, duration is the duration of the diagnosed diabetes and chol/HDL is total cholesterol ÷ HDL-cholesterol.

The UKPDS stroke\(^4\) risk was calculated as follows:

\[
q1 = 0.00186 \times 1.092^{(\text{age at diagnosis} - 55)} \times 1.547^{(\text{smoker})} \times 0.700^{(\text{female})} \times 1.122^{(\text{bpsys} - 135.5)^{10}} \times 
1.111^{(\text{cholesterol/HDL} - 5.11)}.
\]

UKPDS 5-year stroke risk (%) = (1 – \exp(-q1 \times 1.145^{(\text{duration})} \times (1 - 1.145^5)/(1 - 1.145))) \times 100.

With abbreviations as for the UKPDS CHD equation.

Statistical analyses were carried out using SAS version 9.3.\(^8\) Participant data were weighted according to the sampling frame that they were obtained from and the clusters and strata taken into account using dual-frame sampling methodology.\(^9\)–\(^12\) A SAS survey procedure (SURVEYREG) was used to calculate mean 5-year cardiovascular disease risks by age groups, by gender, ethnicity, and time of diagnosis of diabetes. PROC SURVEYMEANS and PROC SURVEYFREQ were used to estimate weighted means and percentages, respectively.

Ethical Committee approval was obtained from the Auckland Ethics Committees.

**Results**

The clinical and biochemical characteristics by gender and time of diagnosis of diabetes are shown in Table 1. Overall the NZGG-modified Framingham equation gave the highest mean 5-year CVD risk of 18.6%, followed by the original Framingham equation mean CVD risk of 14.0%, and the mean UKPDS CVD risk of 11.0%.
The mean risk score was 2.9% (95%CI: 2.40–3.42; p<0.0001) lower in absolute terms for the UKPDS risk engine compared to the Framingham equation and 7.6% (7.05–8.08; p<0.0001) lower for the UKPDS risk engine compared to the modified Framingham equation. The mean 5-year CVD risk was 4.6% (4.24–4.77; p<0.0001) higher for the modified Framingham risk compared with the original Framingham risk.

Figure 1 shows mean 5-year absolute risks of CVD calculated by the three equations by age group and ethnicity.

Table 1. Mean (SE) or percentages for clinical and biochemical characteristics of participants with Type 2 diabetes by gender and time of diagnosis

<table>
<thead>
<tr>
<th>Variables</th>
<th>Variables</th>
<th>Males</th>
<th>Females</th>
<th>Newly diagnosed</th>
<th>Previously diagnosed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>Number</td>
<td>202</td>
<td>221</td>
<td>118</td>
<td>305</td>
</tr>
<tr>
<td>Age (years)</td>
<td>Age (years)</td>
<td>58.9 (0.70)</td>
<td>57.3 (0.67)</td>
<td>55.9 (0.91)</td>
<td>58.9 (0.57)</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>Systolic blood pressure (mmHg)</td>
<td>139.5 (1.57)</td>
<td>136.5 (1.50)</td>
<td>139.3 (2.05)</td>
<td>137.4 (1.28)</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>Cholesterol (mmol/L)</td>
<td>5.14 (0.07)</td>
<td>5.41 (0.07)</td>
<td>5.60 (0.09)</td>
<td>5.16 (0.06)</td>
</tr>
<tr>
<td>HDL-cholesterol (mmol/L)</td>
<td>HDL-cholesterol (mmol/L)</td>
<td>1.23 (0.02)</td>
<td>1.34 (0.02)</td>
<td>1.29 (0.03)</td>
<td>1.28 (0.02)</td>
</tr>
<tr>
<td>Cholesterol: HDL ratio</td>
<td>Cholesterol: HDL ratio</td>
<td>4.43 (0.09)</td>
<td>4.28 (0.08)</td>
<td>4.62 (0.11)</td>
<td>4.24 (0.07)</td>
</tr>
<tr>
<td>Current cigarette smoker</td>
<td>Current cigarette smoker</td>
<td>19.4%</td>
<td>15.9%</td>
<td>17.4%</td>
<td>17.6%</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>HbA1c (%)</td>
<td>7.6 (0.11)</td>
<td>7.4 (0.11)</td>
<td>6.9 (0.15)</td>
<td>7.7 (0.09)</td>
</tr>
<tr>
<td>Duration (years)</td>
<td>Duration (years)</td>
<td>4.0 (0.55)</td>
<td>7.0 (0.52)</td>
<td>–</td>
<td>7.7 (0.48)</td>
</tr>
<tr>
<td>UKPDS 5-year CVD risk (%)</td>
<td>UKPDS 5-year CVD risk (%)</td>
<td>14.6 (0.61)</td>
<td>7.8 (0.58)</td>
<td>8.8 (0.84)</td>
<td>11.9 (0.52)</td>
</tr>
<tr>
<td>Framingham 5-year CVD risk (%)</td>
<td>Framingham 5-year CVD risk (%)</td>
<td>16.3 (0.57)</td>
<td>11.8 (0.55)</td>
<td>13.9 (0.77)</td>
<td>14.0 (0.48)</td>
</tr>
<tr>
<td>NZ-modified Framingham risk (%)</td>
<td>NZ-modified Framingham risk (%)</td>
<td>20.9 (0.58)</td>
<td>16.5 (0.56)</td>
<td>18.3 (0.79)</td>
<td>18.7 (0.49)</td>
</tr>
<tr>
<td>Framingham – UKPDS risk (%)</td>
<td>Framingham – UKPDS risk (%)</td>
<td>1.73 (0.38)</td>
<td>4.00 (0.37)</td>
<td>5.12 (0.51)</td>
<td>2.08 (0.31)</td>
</tr>
<tr>
<td>NZ modified – Framingham risk (%)</td>
<td>NZ modified – Framingham risk (%)</td>
<td>4.59 (0.09)</td>
<td>4.67 (0.09)</td>
<td>4.41 (0.12)</td>
<td>4.73 (0.07)</td>
</tr>
</tbody>
</table>

In general, the original Framingham CVD risks were between the UKPDS CVD risks and the modified Framingham risks (Figures 1A–D and Figures 2 A–D).

The mean modified Framingham CVD risk estimates were significantly higher than the UKPDS CVD risks in people with Type 2 diabetes aged 35 to 39 years and 45 years and over, and significantly higher than the original Framingham mean CVD risks in people aged 45 years and over (Figure 1A). In Europeans, the mean modified Framingham CVD risks were significantly higher than the UKPDS CVD mean risks in people aged 55 to 65 years (Figure 1B).

The modified Framingham mean CVD risks were significantly higher than the UKPDS mean risks in Māori aged 40 years and over (Figure 1C), and significantly higher than the original Framingham CVD mean risks in Māori aged 65 to 69 years. The NZGG-modified Framingham CVD mean risks were significantly higher than the UKPDS mean CVD risks in Pacific people aged 45 years and over (Figure 1D), and significantly higher than the original Framingham CVD mean risks in Pacific people aged 50 to 64 years.
Figure 1. Five-year risks of cardiovascular disease calculated using the UKPDS56 + UKPDS60 and Framingham equations, and the NZ Guidelines Group recommendations by age group (A: All; B: 91 Europeans; C: 119 Māori; D: 213 Pacific)

* indicates p<0.05 between NZ guidelines and UKPDS risk in that age group.
× indicates p<0.05 between NZ guidelines and Framingham risk in that age group.
+ indicates p<0.01 between Framingham and UKPDS risk in that age group.
Note: Horizontal lines have been drawn at the 15% level of absolute risk.
Figure 2. Five-year risks of cardiovascular disease calculated using the UKPDS56 + UKPDS60 and Framingham equations, and the NZ Guidelines Group recommendations by age group (A: 202 males; B: 221 females; C: 118 people with newly diagnosed diabetes; and D: 305 people with previously diagnosed diabetes)

* indicates p<0.05 between NZ guidelines and UKPDS risk in that age group.
× indicates p<0.05 between NZ guidelines and Framingham risk in that age group.
+ indicates p<0.01 between Framingham and UKPDS risk in that age group.

Note: Horizontal lines have been drawn at the 15% level of absolute risk.
Figure 2 shows mean 5-year absolute risks of CVD calculated by the three equations by age group, gender, and time of diagnosis of diabetes. The modified Framingham mean CVD risks were significantly higher for men aged 45 to 69 years compared to the mean UKPDS risks (Figure 2A), and significantly higher than the original Framingham CVD mean risks in men aged 50 to 54 years.

Apart from women aged 40 to 44 years, the modified Framingham CVD mean risks were significantly higher in all age groups than the mean UKPDS CVD risks (Figure 2B), and significantly higher than the original Framingham CVD mean risks in women aged 45 years and over.

The modified Framingham CVD risks gave significantly higher mean risks in people aged 45 and over with newly diagnosed diabetes (Figure 2C) and people with previously diagnosed diabetes (Figure 2D) compared to the mean UKPDS CVD risks. The modified Framingham CVD risks were also significantly higher in the 45 years and over age groups in people with previously diagnosed diabetes compared to the original Framingham CVD risks (Figure 1H).

**Discussion**

Compared to the UKPDS 5-year CVD risk estimates, both the original Framingham equation and the modified Framingham equation risk estimates recommended by the NZ Guidelines Group were higher in people with Type 2 diabetes mellitus. The modified Framingham equation consistently produced the highest risk estimates. A possible reason for the lower UKPDS CVD risks is that the authors discarded the first 4 years of events because all-cause mortality was not significantly different from that in the general population. Thus, all patients in the UKPDS were assumed to have survived the first 4 years event-free.

A major strength of our study was that participants were from a random sample of the Auckland population, and that both newly and previously diagnosed cases of diabetes were included. A further advantage was that all relevant CVD risk factors required for CVD risk estimation were measured. For example, HDL-cholesterol measurements were included—whereas in some other studies comparing Framingham and UKPDS, HDL cholesterol measurements were not available and had to be imputed. On the other hand, the equations do not include coefficients for treatment of blood pressure or of dyldiapaemia, but assume that risk is due to current levels of risk factors.

A potential limitation of the current study was that the type of diabetes was not recorded. The UKPDS risk engine was only designed for use in people with Type 2 diabetes while the original Framingham equation estimates of risk for people with diabetes included both Type 1 and Type 2 diabetes. However, we excluded people reporting a diagnosis of diabetes prior to 20 years of age, who would be more likely to have Type 1 diabetes.

A further limitation of our study was the lack of information on LVH; a variable in the Framingham equations which is more common among diabetic patients, thus leading to the possibility of underestimating CVD risk using the Framingham equation.

The NZ guidelines recommended a modified Framingham CVD risk assessment, with an extra 5% 5-year risk being added for Māori, Pacific, and South Asian people, and
those with poorly controlled diabetes, with diabetes for more than 10 years or with diabetes and microalbuminuria.5

Therefore, higher CVD risk estimates would be expected among Māori, Pacific, and South Asian people using the NZGG-modified Framingham equation compared to the original Framingham and UKPDS equation estimates. Of note, 5-year CVD risks using the modified equation were also systematically higher for Europeans (Figure 1B) and on average the NZGG-modified CVD risks would lead to people with diabetes being recommended for drug management (i.e. CVD risk ≥15%) 10 to 17 years earlier than if risk was calculated using the UKPDS risk engine (Figures 1A–D and Figures 2A–D).

While the population-level predictive validity of the original Framingham risk equation has been validated in New Zealand16, the NZGG-modified Framingham equation does not appear to have been validated.

With respect to combining the two UKPDS risk engines (CHD plus stroke), there is possible undercounting of peripheral vascular disease and congestive heart failure in the CHD equations, but possible overcounting of transient ischaemic attack on the stroke equations due to the overlap of some diseases resulting in approximately correct numbers.

People with Type 2 diabetes will be managed earlier and more intensively based on their risk estimated by the current NZGG guidelines than if the UKPDS or original Framingham equations were used.

Competing interests: None known.

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References:

Ethnic counts on mortality and census data (mostly) agree for 2001–2004: New Zealand Census-Mortality Study update

Tony Blakely, June Atkinson, Jackie Fawcett

Abstract

Background The New Zealand Census-Mortality Study (NZCMS) previously demonstrated substantial undercounting of Māori and Pacific deaths on mortality data relative to census data for the 1980s and 1990s. The recent linkage of 2001–04 mortality data to 2001 census data allows us to determine whether any such ‘numerator-denominator’ bias persists.

Methods 2001 census anonymously and probabilistically linked to 3 years of subsequent mortality data (82,404 eligible mortality records), allowing a comparison of ethnicity recording.

Results Using a ‘total’ definition of ethnicity, there was a close agreement of census and mortality counts: 7419 Māori on the 2001 census compared to 7536 Māori according to mortality data—a census to mortality ratio of 0.98; Pacific—2451 and 2493, ratio 0.98; Asian—1236 and 1215, ratio 1.02; non-Māori non-Pacific non-Asian—73,089 and 72,051, ratio 1.01.

Using a ‘sole’ definition of Māori ethnicity, census counts were only 86% of mortality counts, indicating that mortality data is not recording as many people with two or more ethnic groups as would be expected based on census data. This ‘sole’ bias was more pronounced in the South Island.

Conclusion There is now little bias in ethnic group counts between census and mortality data for a ‘total’ definition of ethnic group. Calculations of mortality rates by ethnicity using unlinked census and mortality data and a total definition of ethnicity should be unbiased. These results strongly support using the census definition of ethnicity on all health datasets.

The New Zealand Census-Mortality Study (NZCMS) previously quantified the gross undercount of Māori and Pacific deaths (and overcount of non-Māori non-Pacific (nMnP) on mortality data prior to 1995.1,2 This meant that historic mortality trends in New Zealand by ethnicity were incorrect, and needed recalculation.3–5

With the advent in 1995 of an ethnicity question on the death registration form that approximated the self-defined question on the 1996 census, undercounting on 1996–99 mortality data was much less for Māori deaths (only 7% using the prioritised or total definitions of Māori ethnicity) and there was no apparent difference for Pacific deaths.2 This highlights the importance of collecting ethnicity information on all administrative datasets in a manner as close as possible to that in the census.

In this paper, we use updated NZCMS data for the 2001 census linked to all deaths in the following three years to determine any discrepancy between mortality and census...
We present ratios of census to mortality counts for total, prioritised and sole definitions of ethnicity.

**Methods**

**Study design**—The methodology has been described in detail in technical reports. Briefly, 81.5% of eligible mortality records (all ages) for the 3 years following the 2001 census were anonymously and probabilistically linked to census records. For the purposes of determining numerator-denominator bias, the linked records were further restricted to highly probably links (HPL) where ethnicity had no effect on linkage probability. Ethnic groupings according to census and mortality data were then compared, using cross-classifications of the weighted HPL dataset. The weights were calculated to make the HPL dataset representative of all eligible mortality records, and are described in detail elsewhere.

**Ethnicity definitions**—Since September 1995, the death register collected data on ‘ethnic group’ using an approximation to the 1996 census self-identified ethnicity question, with up to three ethnic groups coded per person. We use three definitions of ethnicity to examine discrepancies in counts between census and mortality data:

- **Total** ethnicity was assigned as Māori if any ethnic group the decedent was classified as on their census or mortality record was Māori. Likewise for Pacific, Asian, and nonMāori nonPacific nonAsian (nMnPnA). Note that sums of counts across ethnic groups in any total series will be greater than the actual number of decedents due to some decedents being assigned to two (or more) ethnic groups.

- **Prioritised** ethnicity. The prioritised Māori group was the same as the total Māori group. The prioritised Pacific group was the total Pacific group, minus any decedents who were also classified as Māori. The prioritised Asian group was the total Asian group, minus any decedents who were also classified as Māori or Pacific. The remainder were assigned as nMnPnA. Note that conceptually this nMnPnA group is like a ‘sole nMnPnA’ group—i.e. those with only a nMnPnA ethnic group.

- **Sole** ethnicity was assigned as Māori if only one category was identified, and that was Māori; likewise for Pacific and Asian. Those remaining were labelled as ‘Remainder’, and equate to the total nMnPnA group plus some extra decedents that, say, had both Māori and Pacific ethnic groups.

**Results**

Table 1 shows the key findings. There was close agreement between the ethnicity counts on the mortality and census data when either the ‘total’ or ‘prioritised’ definitions of ethnicity were used (i.e. census to mortality ratios all close to 1.0). However when a ‘sole’ definition is used the census count for Māori was less than the count in the mortality data (i.e. ratio of 0.86). This discrepancy arises because fewer mortality records were assigned two or more ethnic groups than expected compared to census data, and thus the sole Māori counts on the mortality data exceed those on the census.

Table 1 also shows the census to mortality ratios for 1996–99 as a comparison over time. The modest undercounting of Māori deaths in 1996–99 (‘total’ and ‘prioritised’ definitions of ethnicity, and as indicated by ratios greater than 1.0) is no longer present in 2001–04. However, the overcounting of Māori ‘sole’ deaths by mortality data relative to census data persists over time.

Ethnicity counts of census and mortality data, by sex, age, and deprivation, show no systematic variation in the census to mortality ratios by these demographic factors (see tables elsewhere). The one exception was region, where census to mortality ratios in the South Island were 1.05 for ‘total’ Māori compared to 0.79 for sole Māori,
indicating that the under-reporting of multiple ethnic groups on mortality data (relative to that expected on census data) is most prominent in the south.

Table 2 shows the full cross-classification of census and mortality data for the prioritised definition of ethnicity. The marginal totals are as in Table 1. The full cross-classification shows the specific mismatches between files. Thus, the majority of mismatches were between Māori and nMnPnA groups.

Table 1. Census ethnicity and death registration form ethnicity totals and ratios for all ages in 2001–04 (n=82,404 deaths; for total, prioritised, and sole ethnicity definitions), and census to mortality ratios only for 1996–99

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>7,419</td>
<td>7,539</td>
<td>0.98</td>
<td>1.07</td>
</tr>
<tr>
<td>Pacific People*</td>
<td>2,448</td>
<td>2,493</td>
<td>0.98</td>
<td>–</td>
</tr>
<tr>
<td>Asian People</td>
<td>1,236</td>
<td>1,215</td>
<td>1.02</td>
<td>–</td>
</tr>
<tr>
<td>nMnPnA ‡</td>
<td>73,089</td>
<td>72,051</td>
<td>1.01</td>
<td>1.03</td>
</tr>
<tr>
<td>Prioritized ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>7,419</td>
<td>7,539</td>
<td>0.98</td>
<td>1.07</td>
</tr>
<tr>
<td>Pacific People</td>
<td>2,373</td>
<td>2,439</td>
<td>0.97</td>
<td>0.99</td>
</tr>
<tr>
<td>Asian People</td>
<td>1,170</td>
<td>1,155</td>
<td>1.01</td>
<td>1.02</td>
</tr>
<tr>
<td>nMnPnA ‡</td>
<td>71,442</td>
<td>71,274</td>
<td>1.00</td>
<td>0.99</td>
</tr>
<tr>
<td>Sole ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>5,931</td>
<td>6,891</td>
<td>0.86</td>
<td>0.86</td>
</tr>
<tr>
<td>Pacific People</td>
<td>2,196</td>
<td>2,274</td>
<td>0.97</td>
<td>0.90</td>
</tr>
<tr>
<td>Asian People</td>
<td>1,098</td>
<td>1,086</td>
<td>1.01</td>
<td>1.01</td>
</tr>
<tr>
<td>Remainder #</td>
<td>73,179</td>
<td>72,153</td>
<td>1.01</td>
<td>0.99</td>
</tr>
</tbody>
</table>

All the numbers are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. The census to mortality ratio is the census count divided by the death registration form count. Note that the sum of observations for Prioritized and Sole series both add to 82,404, but the total ethnicity definition counts sums to more.

*Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.
† The 1996–99 ratios are sourced from Table 12 of Ajwani et al (2002), except for the total ethnicity ratios for Pacific and Asian which were not previously calculated.
‡ The ‘total nMnPnA’ group was defined those people with one or more self-(undertaker-) defined ethnic groups, of which one was nMnPnA. The ‘prioritised nMnPnA’ is best thought of those remaining after all census respondents or decedents with any one of Māori, Pacific or Asian ethnicity have been ‘prioritised out’. Put another way, one might think of the ‘prioritised nMnPnA’ group as actually being the ‘sole nMnPnA’ group.
# The ‘Remainder’ group in the sole series includes those any people who reported nMnPnA ethnic group (i.e. the ‘total nMnPnA’ group) plus some extra decedents or census respondents who were recorded as, say, both Māori and Pacific and therefore not eligible to be either ‘sole Māori’ or ‘sole Pacific’.
Table 2. Cross-classification of census by death registration form ethnicity using the prioritised definition of ethnicity, 2001-04

<table>
<thead>
<tr>
<th>Death registration form ethnicity</th>
<th>Māori</th>
<th>Pacific</th>
<th>Asian</th>
<th>nMnPnA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Census</td>
<td>6,621</td>
<td>21</td>
<td>6</td>
<td>774</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>36</td>
<td>2,250</td>
<td>6</td>
<td>81</td>
</tr>
<tr>
<td>Asian</td>
<td>6</td>
<td>42</td>
<td>1,059</td>
<td>69</td>
</tr>
<tr>
<td>nMnPnA</td>
<td>879</td>
<td>126</td>
<td>87</td>
<td>70,353</td>
</tr>
</tbody>
</table>

All the numbers are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. Minimum cell size is 6.

Discussion

Disagreements in ethnic group coding between mortality and census data still occurred in 2001–04. However, it was much improved compared to the early 1990s and even the late 1990s. Our results suggest that if analysts and researchers use the ‘total’ definition of ethnicity on both census and mortality data in the early 2000s, they should be able to generate ethnic mortality rates with little—if any—numerator-denominator bias. However, we do not recommend calculating ‘sole’ ethnic mortality rates with unlinked census and mortality data.

These results are reassuring. It appears that ensuring the death registration form ethnicity question is as close as possible to that on the census (albeit the 1996 census question) results in close agreement of ‘total’ ethnic group counts, at least. A key recommendation, therefore, is that all datasets in the health sector (and other sectors too) must use an ethnicity question that approximates the census question. Not doing so is likely to result in low quality ethnic group data, hampering analysis, planning and funding.

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Note: Access to the data used in this study was provided by Statistics New Zealand under conditions designed to give effect to the security and confidentiality provisions of the Statistics Act 1975. The results presented in this study are the work of the authors, not Statistics New Zealand.

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References:


Food frequency information—relationships to body composition and apparent growth in 4-year-old children in the Pacific Island Family Study

Elaine Rush, Janis Paterson, Vladimir Obolonkin

Abstract

Aims To record at the 4-year measurement point for the Pacific Island Family Study the most frequently eaten foods and to identify associations with growth and body composition.

Methods A food frequency questionnaire relating to consumption of 111 foods over a 4 week period was administered to the 739 children, to be completed by a parent. Body composition of the children was measured by anthropometry and body fatness by bioimpedance analysis. Patterns of association between body composition and food frequency were examined using correlation analyses.

Results The foods most frequently consumed were bread (1.32 times/day) and total milk (1.63), followed by breakfast cereal (0.83), and fruits (0.78-0.83). 77% of respondents consumed white bread only while 85% consumed standard milk and 7% consumed milk less than once a month or never. Recommended frequency of consumption for fruit was attained by 60%, while only 35% achieved the recommended level for vegetables. Traditional Pacific food consumption made up 5% of the dietary pattern. Protein consumption was positively associated with weight and BMI at 4 years, along with weight gain (0 to 4 years), while frequency of fat consumption was negatively correlated with these variables, in addition to body fat %. Dairy consumption showed a positive correlation with body fat % and BMI.

Conclusions This diet and body size analysis as part of a longitudinal study provides practical evidence to inform practical dietary advice and food policies. Further research is needed to explore association of growth with food patterns and quality.

Worldwide patterns of diet and activity affect body composition and future health throughout the lifecycle. Obesity rates in New Zealand have risen sharply over the past decade. In 2002/2003 in a national survey of health using ethnic-specific body mass index (BMI) cut-off points 56% of New Zealand adults aged ≥20 years were classified as overweight (35%) or obese (21%) while more than 80% of Pacific adults were classified as overweight or obese.

In the 2002 National Children’s Nutrition Survey, more than 60% of Pacific children were classified as overweight or obese using the Cole cut-offs to define overweight and obesity; furthermore, the Pacific population in New Zealand are over-represented in adverse social and health statistics.

These statistics have immediate implications for child health and wellbeing and longer term developmental consequences. Obese children are more likely to be obese
in adulthood and obesity in early life is associated with cardiometabolic syndrome in adulthood.

Energy intake exceeding energy expenditure (over-nutrition) will result in an increase in body fatness but children have an added requirement for specific nutrients to enable optimal growth. Growth, particularly at critical periods, is determined by nutrient intake and energy expenditure matching.

Dietary recommendations throughout the life course include foods from each of the protein, cereal, fruit and vegetable, and dairy groups. Food patterns, related to the variety, frequency, and portion sizes of foods from each group determine nutrient quality and balance. Food frequency questionnaires may be used to determine key foods, dietary balance and patterns from a whole food perspective.

The food frequency questionnaire developed for New Zealand children in 2000 shows good short term repeatability and identified dietary patterns in the 2002 child nutrition survey. Pacific children were identified as eating more breakfast cereal, drinking less milk but more sugary drinks than European children.

The Pacific Island Family (PIF) Study was designed to increase knowledge of the health, psychosocial, and behavioural characteristics of Pacific peoples with recruitment of mothers (n=1376) of children born at Middlemore Hospital in South Auckland in 2000.

Initial data showed that 6 weeks after the birth of the baby 40% of the Pacific Island families reported that sometimes food ran out due to lack of money. We have tracked the growth of these children to 4 years and show that compared with the World Health Organization (WHO) growth standard Pacific babies were born heavy, and over 4 years increased weight faster and between 2 and 4 years increased height faster than the reference breastfed child independent of pre and post natal factors. Maternal smoking decreased the rate of weight gain and children who were not breast fed gained weight faster.

The aim of this investigation was to record the most frequently eaten foods, analyse the dietary pattern and to identify associations of food choices with body composition and growth characteristics at the 4-year measurement point of the PIF Study.

**Methods**

**Survey**—From the 1376 children recruited at birth, 1048 (76.2%) were retained at age 4 years. The qualitative food frequency questionnaire used in the New Zealand Child Nutrition Survey (2002) was administered to the parent of each of 739 children as part of the 4-year assessment in the Pacific Island Family Study.

“How often over the last 4 weeks” was asked for 111 foods with options for other foods to be included in each section. Six foods were considered specifically Pacific: cooked green banana, cassava, taro, coconut cream, boiled corned beef, and canned corn beef. Frequency of consumption was then multiplied by a fraction per day (Table 1) to weight the responses so that they could be summated as frequency/day.
Table 1. Frequency of consumption of food and the weighting factor applied to standardise to a daily rate

<table>
<thead>
<tr>
<th>Frequency of consumption</th>
<th>Weighting factor /day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never or less than once a month</td>
<td>1/200</td>
</tr>
<tr>
<td>1-3 times a month</td>
<td>2/30</td>
</tr>
<tr>
<td>1-2 times a week</td>
<td>1.5/7</td>
</tr>
<tr>
<td>3-4 times a week</td>
<td>3.5/7</td>
</tr>
<tr>
<td>5-6 times a week</td>
<td>5.5/7</td>
</tr>
<tr>
<td>Once a day</td>
<td>1</td>
</tr>
<tr>
<td>“More than once a day”</td>
<td>2</td>
</tr>
</tbody>
</table>

Each food was classed as either a source of carbohydrate, fat, protein, dairy or fruit and vegetables (Table 2). For example pies, burgers, and sausage meats were categorised as protein, legumes/baked beans were also protein, caloric drinks were carbohydrate and spreads and sauces, convenience meals and snacks were classified by their major ingredient. Foods were then subclassified as nutrient high or low. The British nutrient profiling score was used to discriminate foods according to their nutrient (and energy) density.

Table 2. Classification of the 111 foods in the children’s nutrition survey food frequency questionnaire by major nutrient or as dairy or fruit and vegetable and by nutrient and energy density

<table>
<thead>
<tr>
<th>Class</th>
<th>Total number</th>
<th>Higher nutrient-lower energy</th>
<th>Lower nutrient-higher energy</th>
<th>Selected example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruit and vegetable</td>
<td>27</td>
<td>26</td>
<td>1</td>
<td>Tomato sauce</td>
</tr>
<tr>
<td>Protein</td>
<td>27</td>
<td>18</td>
<td>9</td>
<td>Sausage roll</td>
</tr>
<tr>
<td>Fat</td>
<td>8</td>
<td>*1</td>
<td>7</td>
<td>*Avocado (higher nutrient)</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>40</td>
<td>11</td>
<td>29</td>
<td>Biscuits, cakes</td>
</tr>
<tr>
<td>Dairy</td>
<td>9</td>
<td>6</td>
<td>3</td>
<td>Cheese</td>
</tr>
</tbody>
</table>

Birth weight was obtained from the birth records. Body weight and height were measured at 2 and 4 years by using standardised equipment and procedures. BMI was calculated as weight in kilograms divided by squared height in meters.

At 4 years, direct measurements of resistance (R), reactance (X), impedance (Z), and phase (θ) to a 50 kHz signal using a bioimpedance analyser (Model Imp 4, Impedimed, Queensland, Australia) with a tetrapolar arrangement of self-adhesive electrodes (Red Dot 2330, 3M Healthcare, St Paul, MN, USA) were made.

These measurements were carried out with the child lying supine, the arms near but not touching the body and the legs abducted. The skin of the right hand and foot was swabbed with alcohol before the electrodes were placed. Source electrodes were placed on the dorsal surface of the foot over the distal portion of the second metatarsal, and on the hand on the distal portion of the second metacarpal.

Sensing electrodes were placed at the anterior ankle between the tibial and fibular malleoli and at the posterior wrist between the styloid processes of the radius and ulna. The child was lying still for at least 5 min before the measurements were made. The average of repeated measurements of R and X agreeing to within 1 ohm of each other was used in subsequent analyses. Resistance measurements were used to derive body fat percentage using the prediction equation of Rush et al. Statistics—Results are presented as means ± SD. Patterns of association between body composition and frequency per day of food groups and subcategories of nutrient density were investigated using
bivariate Pearson correlation. Data were analysed using SPSS (version 13) software (SPSS Inc, Chicago, IL). P values <0.05 were considered significant.

Results

All measurements were made and a food frequency questionnaire for the child completed by the parent for 355 girls and 384 boys. Table 3 shows the physical characteristics of the study population. On average, the z-scores for BMI, weight, and height are significantly above the ideal child.\(^1\) Male and female were similar and for purposes of this descriptive analysis have not been separated.

Table 3. Characteristics of study cohort

<table>
<thead>
<tr>
<th></th>
<th>N=739</th>
<th>Mean (SD)</th>
<th>Mean Z-score (WHO)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight, kg</td>
<td>3.61(0.597)</td>
<td></td>
<td>0.455(1.24)</td>
</tr>
<tr>
<td>Weight 2 y, kg</td>
<td>14.6(1.96)</td>
<td></td>
<td>1.03(1.11)</td>
</tr>
<tr>
<td>Height 2 y, cm</td>
<td>89.2(4.13)</td>
<td></td>
<td>-0.264(1.27)</td>
</tr>
<tr>
<td>BMI 2 y, kg.m(^{-2})</td>
<td>18.4(1.98)</td>
<td></td>
<td>1.68(1.27)</td>
</tr>
<tr>
<td>Weight 4 y, kg</td>
<td>20.8(3.85)</td>
<td></td>
<td>1.64(1.28)</td>
</tr>
<tr>
<td>Height 4 y, cm</td>
<td>106(4.44)</td>
<td></td>
<td>0.582(1.05)</td>
</tr>
<tr>
<td>BMI 4 y, kg.m(^{-2})</td>
<td>18.3(2.40)</td>
<td></td>
<td>1.94(1.38)</td>
</tr>
<tr>
<td>% body fat</td>
<td>21.0(5.32)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight gain 0–2 y g.day(^{-1})</td>
<td>11.1(1.86)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight gain 0–4 y g.day(^{-1})</td>
<td>17.2(3.73)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

From the food frequency questionnaire bread and milk were the reported foods most frequently eaten (Table 4). Three choices were given for the “main type of bread you usually eat? tick one box”. White bread only was ticked by 77% and a further 11% had white and grain (wholemeal or mixed grain) breads ticked as well as white bread. Only 11% ticked that only grain breads were consumed. Milk was consumed by 7% never or less than once a month. For the “kind of milk” there also were also multiple responses possible—85% of those who drank milk ticked standard milk, 11.5% light blue, and 3% each the trim and extra calcium, 1% consumed soy.

The mean total number of times that specific foods were reported as consumed by a child each day was 30. The first most frequently consumed 20 foods accounted for 37% of the frequency of eating and the next 20 for 21%—i.e. 58% of the total frequency was included in 40 items in the questionnaire.

Nutrient poor food including powdered drinks, noodles, tomato sauce, and potato crisps and corn snacks were in the top 20. Total milk including flavoured and food drinks was consumed at a similar rate to bread 1.63 vs 1.32 times in a day. More traditional Pacific food consumption was also investigated and on average was 5% of the dietary pattern. Taro was eaten on average 0.29 times a day, cassava 0.12, canned corned beef 0.22, boiled corned beef 0.13, fish 0.30, and cooked green banana 0.26. Foods high in white flour or sugar made up more than 20% of the diet.
Table 4. Forty most frequently eaten foods ranked by mean frequency of consumption each day

<table>
<thead>
<tr>
<th>Food item and class</th>
<th>Frequency/day</th>
<th>Food item</th>
<th>Frequency/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bread, including toast and bread rolls C+</td>
<td>1.32</td>
<td>Other item of the Dairy group D+</td>
<td>0.42</td>
</tr>
<tr>
<td>Milk (not flavoured) D+</td>
<td>0.86</td>
<td>Biscuits C–</td>
<td>0.39</td>
</tr>
<tr>
<td>Apples or pears FV+</td>
<td>0.83</td>
<td>Nutella C–</td>
<td>0.39</td>
</tr>
<tr>
<td>Breakfast cereal C+</td>
<td>0.83</td>
<td>Ice cream D-</td>
<td>0.38</td>
</tr>
<tr>
<td>Banana, raw C+</td>
<td>0.79</td>
<td>Canned spaghetti with tomato sauce C+</td>
<td>0.37</td>
</tr>
<tr>
<td>Oranges or mandarins FV+</td>
<td>0.78</td>
<td>Jam or honey C–</td>
<td>0.37</td>
</tr>
<tr>
<td>Food drink D+</td>
<td>0.58</td>
<td>Other vegetables FV+</td>
<td>0.36</td>
</tr>
<tr>
<td>Chicken P+</td>
<td>0.57</td>
<td>Other fruit FV+</td>
<td>0.36</td>
</tr>
<tr>
<td>Rice C+</td>
<td>0.56</td>
<td>Soup FV+</td>
<td>0.35</td>
</tr>
<tr>
<td>Powdered fruit drink C–</td>
<td>0.51</td>
<td>Canned or cooked fruit FV+</td>
<td>0.35</td>
</tr>
<tr>
<td>Noodles C-</td>
<td>0.50</td>
<td>Tomatoes FV+</td>
<td>0.35</td>
</tr>
<tr>
<td>Tomato sauce or ketchup FV–</td>
<td>0.50</td>
<td>Crackers or crispbreads C–</td>
<td>0.35</td>
</tr>
<tr>
<td>Eggs, boiled, poached, fried or scrambled, etc P+</td>
<td>0.49</td>
<td>Peanut butter F–</td>
<td>0.33</td>
</tr>
<tr>
<td>Yoghurt or dairy food (all types) D+</td>
<td>0.49</td>
<td>Fried potatoes C–</td>
<td>0.32</td>
</tr>
<tr>
<td>Mixed vegetables FV+</td>
<td>0.46</td>
<td>Chocolate coated or cream filled biscuits C–</td>
<td>0.32</td>
</tr>
<tr>
<td>Other potatoes C+</td>
<td>0.46</td>
<td>Other Convenience meals/snacks item C–</td>
<td>0.32</td>
</tr>
<tr>
<td>Juice C–</td>
<td>0.46</td>
<td>Other items of the Spreads, sauces group F–</td>
<td>0.30</td>
</tr>
<tr>
<td>Carrots FV+</td>
<td>0.45</td>
<td>Sausages P–</td>
<td>0.30</td>
</tr>
<tr>
<td>Other items of Eggs, Meat, Poultry and Fish P+</td>
<td>0.44</td>
<td>Fish P+</td>
<td>0.30</td>
</tr>
<tr>
<td>Potato crisps, corn snacks or chips C–</td>
<td>0.43</td>
<td>Lettuce or green salad FV+</td>
<td>0.29</td>
</tr>
</tbody>
</table>

FV=Fruit and vegetable; P=Protein; F=Fat; C=Carbohydrate; D=Dairy; +Higher nutrient, lower energy; – Lower nutrient, higher energy.

Associations (Pearson r, p<0.05) between growth and body size characteristics with frequency/day of food categories were systematically examined and key aspects of the pattern of association detected are summarised in Table 5.

Birth weight was positively associated with the proportion of nutrient dense food (p=0.003), fruit and vegetable intake (p=0.0005) and negatively with the nutrient poor protein foods (p=0.02). The more times carbohydrate foods were consumed in a day the lower the birth weight (p=0.04).

Four-year weight, BMI, body fatness, and weight gain over the 4 years showed a consistent pattern not seen for the 2-year old body composition variables. Higher frequency of consumption of fruit and vegetables and of fat foods was associated with less weight gain (p=0.05, 0.009) and a smaller BMI (p=0.04, 0.008). Total protein and dairy consumption were also positively associated with a larger body size (p<0.05).

The most influential foods on 4-year BMI were the relative frequency of consumption of all fat foods (-ve, p=0.008) and good protein (+ve, p=0.01) foods.
Table 5. Pattern of association between growth and body size characteristics and frequency of consumption of different food groupings (relative to total food frequency)

<table>
<thead>
<tr>
<th>FFQ items</th>
<th>Nutrient Dense</th>
<th>Fruit/veg</th>
<th>Dairy</th>
<th>Fat</th>
<th>CHO</th>
<th>Total Protein</th>
<th>Good Protein</th>
<th>Bad Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight</td>
<td>+ve</td>
<td>+ve</td>
<td></td>
<td>-ve</td>
<td></td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
<tr>
<td>Weight, 4y</td>
<td></td>
<td></td>
<td></td>
<td>-ve</td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
<tr>
<td>Height, 4y</td>
<td>-ve</td>
<td>+ve</td>
<td></td>
<td>-ve</td>
<td></td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
<tr>
<td>BMI, 4y</td>
<td>-ve</td>
<td>+ve</td>
<td></td>
<td>-ve</td>
<td></td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
<tr>
<td>Body fat%, 4y</td>
<td>-ve</td>
<td>+ve</td>
<td></td>
<td>-ve</td>
<td></td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
<tr>
<td>Weight gain, 4y</td>
<td>-ve</td>
<td></td>
<td></td>
<td>-ve</td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
</tbody>
</table>

+ve means that there was a significant positive association and –ve a significant negative association. Blank cells indicate no association seen.

The recommended frequency of consumption fruit (2+ a day) and vegetables (3+ a day) for 2–12 y old children in NZ was compared to the frequency reported. For fruit 442 (60%) consumed fruit at least twice a day and the four top fruit were apples or pears, oranges or mandarins at ~0.8 serve day; banana consumption was similar.

Vegetables were eaten at least three times a day by 255 (35%) the most popular being tomato sauce, mixed vegetables, and carrots. Noodles were eaten at least once a week by 659 (89%) and 375 (50%) consumed breakfast cereal at least once a day. Rice was eaten at least once a day by 150 (20%).

Discussion

Food patterns, growth and their associations have been examined in this cohort of Pacific Island children in South Auckland. Foods frequently eaten by these Pacific Island children have been identified and bread and milk are the foods most frequently consumed. Associations of the quality of the food pattern with body size and growth trajectory have also been demonstrated.

A higher proportion of fruit and vegetables in the diet is associated with a higher birth weight but lower BMI and weight gain over 4 years. Conversely protein and dairy foods, both good sources of protein, are associated with increased weight gain and BMI. A higher proportion of fat foods are associated with less weight gain. Each finding will be discussed in turn.

Bread and milk are staples of the NZ diet for children and adults alike. White bread and standard milk were consumed by the majority of children frequently—10% of the diet. These common food sources point to areas where nutrient density could be improved very easily—addition of whole grain flours to the bread and removal of saturated fat from the milk—small changes in composition with very little potential for change in taste and palatability would make a big difference to nutrient density and intake.
Price and availability would also need to be addressed to make this a viable intervention. Consumption of whole grains and less saturated fat, affordable price and access are all known to be associated with improved health.\textsuperscript{20}

We have reported previously that at birth this cohort were not food secure\textsuperscript{13}. We have also reported that at birth babies of non smokers were larger than those of smokers\textsuperscript{14,21} and that breast feeding was associated with a smaller body size at age 4.\textsuperscript{14}

Higher fruit and vegetable intake is a marker of a more healthy (and maybe wealthy) lifestyle.\textsuperscript{22} In this study a higher birth weight was associated with a higher fruit and vegetable intake reported at age four. A lower BMI at age 4 was also associated with a higher fruit and vegetable intake.

This study is limited in a number of aspects including many confounders and unknowns. The food frequency questionnaire is not quantitative as portion size is not asked but it does quantify the number of times in a given period that foods are consumed.

At age 4 the reported foods have been ranked and a 4-week pattern analysed. The data was collected over a year—there is no correction for seasonal availability. Similarly there is no correction for socioeconomic status, body size for amount of food consumed or physical activity to name a few key confounders. But it is unlikely that there were large changes in the family food environment over the last four years\textsuperscript{23} and the associations with birth weight support this.

The foods most frequently eaten agree with findings in the national children’s nutrition survey and endorse the validity of the questionnaire. Specific ethnic foods such as taro were included in the analysis but were only 5% of the foods chosen. Conversely high sugar and white flour foods comprise 20% of the foods.

Food classification and consequent analysis or scoring food patterns is difficult given the complexity of identifying “good” and “bad” foods. Consumption of foods high in whole grain breads and cereals, fruits and fruit juices, and raw vegetables, and low in processed meat, butter, cheese, margarine, and meat in an adult European population is predictive of low prospective weight change.\textsuperscript{24}

Responses to food frequency questionnaires rank a population well\textsuperscript{25} but are fraught with inconsistency. Although the median and the mean of the reported number of food items per day look reasonable (26 and 30 respectively) the maximum and minimum were 7 to 150 food items per day which reflects the difficulty of getting accurate information from questionnaires related to food consumption.

We did not exclude any data to avoid adding more bias. But broad food group and nutrient density classification methods such as that used here\textsuperscript{15} are useful to identify areas where dietary patterns and frequency of consumption are associated with increased body fatness or other measures of health, growth, and development.

We have provided a list of foods most commonly consumed by Pacific children at 4 years and identified some potential for change in the food supply. We demonstrate that measures of nutrient quality of foods and within food groups of diet over the previous 4 weeks are associated with growth over the past 4 years and present body size. The issue of protein and dairy foods being associated with larger body size merits further investigation.
Competing interests: None known.

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References:


Oral healthcare for older people: ‘I can’t afford not to go to the dentist, but can I afford it?’

Lynne Giddings, Barbara McKenzie-Green, Linda Buttle, Keita Tahana

**Aim** The purpose of this study was to examine challenges older people encounter in maintaining satisfactory oral health status.

**Method** This interpretive qualitative study involved 19 in-depth interviews with participants aged 65 to 87 years. Data were examined using a three-level analytic process. NVivo Qualitative Software assisted data management.

**Results** Participants in this study didn’t ‘just go’ to the dentist. Much effort went into solving the dilemmas and tensions of maintaining their oral health through a process of option balancing. Balancing personal and financial costs, they continually assessed their ‘bottom line’. They negotiated issues of: dental cost versus service quality; basic treatment versus functionality and social appearance; future living costs versus current dental costs; and ‘how long will it need to last’ versus ‘how long will I live’?

**Conclusion** Problems exist in the provision of affordable oral health care for the older population in Aotearoa New Zealand. They struggle to afford dental care. They receive little financial support to access oral healthcare services and are dependent on developing their own strategies to enable such care. Health professionals and policy decisionmakers’ challenge is to bridge this gap.

As a result of global population ageing and technological advances in dental services, edentulism (no natural teeth) has decreased and oral healthcare needs have substantially increased. The ageing process itself, the increasing numbers of older people, and the dynamic relationship between oral health and general health, produces this care need. The correlation between poor oral health and conditions such as respiratory infections, glycaemic control in diabetic patients, and cardiovascular disease has been established. As Ship asserts “oral health and function decline, in some cases rapidly, in medically, behaviourally, and physically compromised older persons”. Whether edentulous or dentate the older person can experience changes in chewing ability, and are at higher risk of nutritional deficiency as a result of poor oral health. Recent studies have reported that older people experience embarrassment if they have diseases of their mouth, teeth and/or gums and suggest that such situations can disrupt social life. Older people’s quality of life can thus be substantially affected by poor oral health. International research has highlighted the relationship between good oral health and preventive activities, such as daily hygiene practices and fluoridisation, together with regular dental care services. Key to the effectiveness of these practices is peoples’ ability to afford and access these services.
Affordability is a critical issue for many older people as their incomes decrease and oral healthcare needs increase. One Australian study reported a decrease in the purchase of dental insurance in the 70 and over aged group together with reports of difficulty in meeting dental costs.

The majority of the ageing population in Aotearoa New Zealand is caught by their increased need for oral healthcare services and their diminishing income. The situation is compounded by changes in their ability to physically access dental services. With the current government funding focus on subsidising general healthcare, oral healthcare is missing out. This gap in healthcare provision is evidenced by the paucity of specific social and public policies on the subsidised provision of oral healthcare services for the elderly.

The current focus of oral health policy and funding is on preventative care for children and adolescents. While such policy is essential for the general health of future generations, this does not negate the need to invest in the maintenance of oral health for older people. Yet, the majority of older people in Aotearoa New Zealand still continue to be “responsible for the full costs of their oral healthcare”.

Carter et al in reference to dependent elderly state that “oral health and oral disability is not seen as part of overall health by central government funders, and this (in part) negatively influences the delivery of care”. This argument could be extended to the community dwelling older population.

If good general health and a satisfactory quality of life are dependent on maintaining oral health, then policy needs to be implemented so that such services become affordable and accessible to our ageing population. The purpose of this study was to examine the challenges that older people face to maintain their current oral health status and to inform policymakers of this critical health situation.

Method

Semi-structured interviews (45 to 90 minutes duration) were conducted with 19 older adults (5 male and 14 female), aged 65 to 87 years. Participants were predominantly white, middle-class and retired. The philosophic assumption underpinning this qualitative interpretive study was that the challenges to maintaining oral healthcare could be examined by focusing on older adult’s experiences of this phenomenon. A qualitative approach can add depth and texture that complements the available quantitative data. Ethical approval for the study was obtained from the AUT University Ethics Committee.

Purposive sampling using the snowball technique was employed for recruitment. Initial participants responded to flyers distributed to retirement villages and health clinics. Once enrolled, participants were encouraged to invite others to contact the researchers. Inclusion criteria were English speaking men and women over 65 years, who could comprehend the study details.

Interviewers (x3) were trained in qualitative data collection techniques. Training sessions (2x2 hours) included interviewing techniques and the application of open ended reflective questioning. Removing all identifying information from the audio taped and hard copy transcripts ensured confidentiality and public anonymity.

A three-level iterative analytic approach as described by Grbich was applied. At the first level of analysis prominent concepts were systematically coded. The relationships between concepts were then examined, followed by a movement of concepts into themes. Each theme was dimensionalised according to the meaning participants attached to the concepts, the strategies that arose out of that meaning and the conditions which shifted a participant’s strategy with regard to their oral health. Analysis for all themes followed this iterative approach to coding, conceptualising and examining relationships between concepts and themes. Two investigators collaborated on all phases of data
analysis. QRS NVivo software was used for organisation of data and concept modelling. Analytic decisions were by mutual agreement and were documented.

**Results**

A full description of the themes conceptualised during data analysis has been reported in a previous article. In this paper we focus on how the participants navigated the complex process of option balancing. This process was used by the participants to arrive at decisions regarding their oral healthcare given their resources. The major condition which shifted decision-making was affordability.

Option balancing included all or some of the following dimensions: the need to weigh future and current health service requirements; price against dental service access and perceived professional competence; deciding between tooth extraction and tooth preservation; functionality versus appearance; health maintenance with ongoing dental checkups against problem-oriented dental visits, and value against years left. Those in a couple relationship also talked of prioritising the person whose needs were most urgent. Not all option balancing resulted in decisions that led to optimal dental health.

For these participants option balancing was mediated by ‘bottom lines’ which were figured according to individual perspectives and personal circumstances. For some, the bottom line was met when they required a root canal. For others, when treatments became too costly.

I mean if I had toothache or things like that all the time I might have them out but I wouldn’t waste the money on having caps or root canals (9/250).

As this example illustrates, the bottom line could be future-oriented and decided before they entered the process of option balancing.

While many participants decided that, regardless of personal and financial costs they would continue to access dental services, all participants indicated affordability of dental services was the major consideration.

…As you get older of course, the money pot that you have is steadily eroded…The bank balance withers away and up goes the price of most commodities, but dentistry, for some reason, you don’t look forward to having to [go]. You shudder when you think how much he is going to [cost] (18/472).

The following findings and examples illustrate the complexity of the option-balancing process.

Decisions led to strategies for preventive or problem-oriented dental visits. Some participants found their negative childhood experiences played a role in their decision not to seek preventive dental care. Others decided that preventive visits had become too expensive (a bottom line) and commenced a pattern of problem oriented care. While in contrast, in spite of negative experiences and cost, others continued to maintain a schedule of preventive dental visits.
One participant reported her response to the dentist.

He [the dentist] said to me…well, why do you want to keep your teeth? I said, “Well I eat with them” (1/165).

Regardless of the care pattern, participant strategies involved balancing cost containment with access and quality of outcome.

I went to a dentist and they said I would have to have all my teeth removed but my husband…he said ‘no’. He had to lose his teeth at 19 like a lot of young people did in those days…He hated his false teeth so he said ‘I don’t care what it costs go to someone else’ (2/85).

Dental services were carefully considered, particularly when there was a change in participants’ ability to access the clinic. To decide about service quality, cost, and accessibility they asked friends, neighbours, and those with local knowledge, with the aim of finding the best accessible service at the lowest cost.

I didn’t know the area and I asked him about it and he said there is that one…in [suburb that] was very good and…the cheapest one around (33/313).

Some prioritised maintaining their current dentist and would ‘co-opt’ transport from friends or family or use taxis when they could no longer drive.

She gets a taxi to go and she goes to the hygienist quite often. I don’t think she’s got that much money to waste…I think she is trying to preserve the teeth she’s got rather than…at this age having to get them out and having dentures (33/214).

Relationships with the dental staff were included in discussions about dental clinic choice with some participants prioritising the quality of that relationship. They reported during interview their discussions with dentists, their comfort with questioning the dental staff, learning new dental care techniques and assistance with decisions about treatments as well as their preferences in these matters.

I like the younger ones because I feel they are up with all the new technology (23/199).

Cost containment extended to the type of services accessed. Those with dentures would access technicians rather than a dental clinic and attend to their teeth but not always their gum conditions.

I’ve usually gone to a technician because it was always cheaper …at one time you had to go to the dentist to get your dentures, but I’ve only been to a technician (22/213).

Others chose between regular dental and hygienist treatment.

I would go to a dentist by choice [who has] a degree because I feel that I would have more confidence in a fellow that’s got further in his dental expertise. I don’t care whether it’s a man or a woman (19/319).

A number of participants negotiated directly with the dentist.

I got a quote from him, $300 or something and I had an argument…whether if I paid cash would he take the GST off…which he did. He put some money in his pocket and I went away with a set of dentures much cheaper than I intended on paying. But if he mentioned my lower teeth I wouldn’t have given him a look (18/128).

Other participants negotiated within the family.

She had dentistry done initially. Well, her teeth are falling apart and she is a regular attender and that’s what keeps her going. This sort of thing, her going and me not, saves us money (18/426).

A constant backdrop that accompanied this deliberating, deciding and action process was ‘making ends meet’ or planning for the unexpected. One participant had
experiences of Accident Compensation Corporation (ACC) payments that did not meet her accident related costs and found that her spare money had almost disappeared.

"...it [dental work] took all the spare cash I had...a bit frightening to think that okay I might have something else and I won’t be able… and then of course I did my glasses in…So I was down financially over $5000 (22/114).

Other participants had made earlier decisions about their dental care based on perceived time left to live.

Now I’m 85 I wonder what better things I might have done when I was 74 and I had taken the advice that I was offered, which I didn’t (1/9).

Most participants’ reports revealed the uncertainty that ageing brings. Decisions to have particular treatments or not involved a balancing between cost and expected length of life.

I remember saying to him “Well hang it all I am 74 it can’t really matter can it” (1/290).

For all participants, uncertainty extended to the continued health of their body, teeth, and mouth as well as their acceptability to others.

I like to know that they are clean, that when I smile they are not all grubby and dirty although they are getting older looking. But…I certainly would hate to be without them…so in that way they’re precious (5/343).

Participants reported in their ageing they experienced discoloured teeth, fillings falling out, teeth cracking and decay. Infection and inflammation of the gums were more frequent. Dentures required replacement or adjustment as gums receded. Food choices were limited and foods became trapped between teeth, making social occasions difficult. Others felt watched and sometimes judged for the state of their teeth.

I think it’s the cost with most people when they are older. They are frightened of being without something at the back of them and a lot of them have only got their pensions…I go to [suburb] where there is a lot of older people and to me their mouths don’t look as though they are very healthy and it’s probably because they can’t afford to go. I think dental health is part of ordinary health. They shouldn’t have to pay so much to have to go to the dentist like they don’t have to pay so much to go to the doctor (22/374).

People balanced cost against the real need of comfort and nutrition versus their personal appearance to have an acceptable public self or a self that was authentically them.

If you’ve got a nice healthy mouth and teeth then the rest of your body is going to be a lot healthier isn’t it? (1/22)

Participants talked about their teeth and mouth looking aged and old like them, but were ‘them’ and that was important.

I’m still quite conscious that some of the gaps in my mouth show when I laugh and I talk a lot and so people I’m with must be very familiar with what my mouth looks like (4/362).

Some participants covered their mouth with their hand when talking, while others maximised their attractiveness by the use of particular toothpastes, mouthwashes, flossing, and whitening products—and made regular dental clinic visits. Many participants had learned earlier in life that chewing gum (particularly in public) was unacceptable and either would not use these oral health products or would do so privately where no-one could see.
While a number of dimensions were taken into account during option balancing processes, and while participants had differing bottom lines, the final consideration which shifted their decision was cost. The first to go were regular dental visits. Whether the participant went to the dentist from a problem oriented or preventive perspective, for most they went ‘because they could not afford not to’ while wondering ‘whether they could afford it’.

Discussion

Increasing healthcare needs of older people—With the ageing of our Aotearoa New Zealand population, the oral healthcare needs of older people is on the increase. Yet our healthcare system appears poorly prepared for this situation. Although the inter-relationship between oral health and general health is well known by health professionals, there is evidence that they place oral healthcare as a low priority when caring for the elderly. Oral health assessments are poorly conducted and few professionals give advice concerning oral health maintenance.

This study has shown that for an older person, the maintenance of a healthy mouth and teeth is not a simple matter. The intersection of increasing general healthcare needs and decreasing resources and income can cause a shift from preventive to problem oriented dental care. While many receive subsidies for general healthcare, dental care is minimally supported.

In Aotearoa New Zealand, people on lower incomes have access to emergency dental care only. It was not surprising to find therefore, that affordability was at the centre of these participants’ deliberations concerning their oral healthcare. Only one participant reported the use of a refund programme, namely ACC. Even though an appeal was lodged, this participant was refunded a small proportion of the actual costs incurred from an accident.

While an increase in the availability of dental care insurance could assist those who are still working or in their early retirement years, research has shown that those who have been retired for many years can no longer afford insurance costs.

Government funders, maybe rightly so, are concerned with cost effectiveness and there is an increasing focus on the provision of accessible and affordable primary healthcare. Yet older peoples’ oral healthcare needs do not feature prominently in future planning. For example, a 1997 report to the National Health Committee on preventive dental strategies for older populations recommended “a systematic review of the current formula by which publicly-funded dental care is allocated” (p2). However, Carter et al reported that the results of their “Christchurch study show little evidence of implementation of the recommendations of that report” (p8). Additionally, there is little indication that the current government plans any focused response to these recommendations.

It would seem from this study and from international research that this hands off approach will not meet the current and future needs for oral healthcare for older people.

This study has not provided epidemiological data. Critically, it gives voice to those who otherwise may not be heard, the older people in an urban New Zealand community. It provides insight into the lengths older people go to maintain their oral
health and quality of life. These findings stand alongside national and international research that report affordability and access as central to the maintenance of oral health for older people. They could also complement the findings from the current national Oral Health survey commissioned by the Ministry of Health to be published in 2009.

The participants’ stories reflect determination and steadfastness; they challenge health practitioners, policymakers and public funding agencies to become equally determined to support policies that direct funding towards accessible oral healthcare for older people. To not do so would be more costly. Research has clearly linked the relationship between poor oral health and exacerbations of chronic health conditions.

**Future research and study limitations**—This study raises questions for further investigation. From the stories of these participants it is evident that dental practitioners are also involved in a balancing act when advising older people about their dental healthcare needs. We propose to examine the situation related to older people from the perspective of these practitioners.

A limitation of this study is the homogeneity of the participants. Most participants were from working or middle class backgrounds and owned their own homes and cars. With only five men, the majority of participants were women. Additionally, we recruited few Māori. The findings therefore, are more representative of mainstream European oral healthcare experiences. A more ethnically heterogeneous sample could have added complexity to this study.

**Conclusion**

Health professionals’ lack of awareness of oral healthcare needs combined with the limited public funding in this area, does not auger well for the general health standards of Aotearoa New Zealand’s ageing population. The message given by the participants in this study is clear, ‘access to oral healthcare matters’. Their stories of ‘balancing’ affordability with their quality of life need to be listened to. Publicly funded oral healthcare for the older population is an urgent healthcare need. A decision by health professionals and policymakers to take joint action and change current policy to include publicly funded oral healthcare for the older population will make a difference.

**Competing interests:** None known.

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References:

   http://www2.stats.govt.nz/domino/external/web/prod_serv.nsf/fb471f7feb8c88d5fcc256af1000fa1c1/c0170b59e8f7b3b3b2cc256e699071f0b4e7OpenDocument
Percutaneous transcatheter obliteration of aortic paravalvular leak

Ali Khan, Peter N Ruygrok, Paisan Nakpathomkun, Ivor L Gerber

Abstract

A 76-year-old man who underwent aortic valve replacement for severe calcific aortic stenosis developed a significant paravalvular leak. Because the risk of re-operation was felt too great, a percutaneous transcatheter obliteration of the defect using an Amplatzer vascular plug was undertaken, with an excellent clinical outcome.

A 76-year-old man with symptomatic severe aortic stenosis and coronary disease underwent elective aortic valve replacement (AVR) and two-vessel coronary bypass surgery. During surgery his aortic valve was noted to be severely calcified, particularly in the right coronary and non-coronary sinuses, where it was difficult to achieve complete decalcification.

A 29 mm bovine pericardial prosthetic valve was implanted. The post-bypass transoesophageal echocardiogram (TOE) identified a small paravalvular leak (PVL) in the region of non-coronary sinus where most calcification had been observed. At the time it was felt that further surgical intervention was unlikely to be beneficial.

The patient reported no noticeable improvement of his pre-surgical dyspnoea (NYHA class III) at the 2-month follow-up visit and a prominent aortic early diastolic murmur was audible. A transthoracic echocardiogram showed at least moderate eccentric aortic regurgitation. A subsequent TOE revealed a significant PVL but no valvular regurgitation and the left ventricle appeared to have enlarged.

The patient was re-discussed at the combined cardiology-cardiac surgery meeting and it was felt that the chance of significant morbidity and mortality, associated with re-operation was high, and the potential for benefit doubtful. After discussion with the patient, an attempt to partially obliterate the PVL by a percutaneous transcatheter approach was planned.

The procedure was undertaken under general anaesthesia with TOE and fluoroscopic guidance, preceded by DC cardioversion of atrial fibrillation (AF) to sinus rhythm. Via a retrograde approach from the right femoral artery, a 6 French delivery sheath was advanced across the defect, through which a 10 mm Amplatzer vascular plug was carefully positioned and delivered, with definite angiographic and echocardiographic reduction in the degree of aortic regurgitation (Figure 1). The procedure time was 55 minutes and fluoroscopy time 8 minutes.

The patient tolerated the procedure well with no complications and was discharged the next day on warfarin anticoagulation for recurrent AF. At 6-week follow-up he reported definite symptomatic improvement (NYHA Class II) and a TOE showed stable position of the plug with only a mild-moderate residual PVL (Figure 2).
Discussion

Early para-prosthetic leakage after AVR is relatively common and may be related to particular valve types, but the majority of defects are small and generally have a benign course. Some, however, may cause progressive left ventricular dilatation and heart failure from a high regurgitant load, and may also cause haemolysis. This usually occurs due to incomplete apposition of the sewing ring of the prosthetic valve to the native aortic tissue and suture dehiscence, and develops more commonly in patients with significant annular calcification or localised infection.1,2

The rate of detection of PVL has increased due to improved diagnostic techniques. Doppler echocardiography is particularly useful in differentiating transvalvular from paravalvular regurgitation.3 Until recently, management has been limited to medical therapy or re-operation which has been associated with a high mortality and substantial risk of recurrent paravalvular leak.4

Percutaneous transcatheter closure of aortic prosthetic PVLs has recently been described in selected patients, with a reasonable chance of success. This can however be technically challenging, with only a few case reports in the literature.5,6 Careful assessment of the defect by pre-procedural TOE and peri-procedural selective contrast injection is important in the estimation of defect size and geometry, and in selection of an appropriate obliteration device.

The presence of vegetations or thrombus, active infection or an unstable rocking valve, are clear contra-indications. Obliteration of PVL is feasible in patients with a ‘tilting-disc’ mechanical valve provided care is taken to ensure the device does not interfere with leaflet movement before released. No specifically designed transcatheter device has been developed or approved for percutaneous PVL closure but there are reports of use of the Amplatzer atrial septal occluder, Rushkind umbrella, Cardioseal Clamshell septal occluder, and Gianturco detachable coils.7–9 Larger defects may require more than one device.

In our case we used the Amplatzer device since it is compressible and relatively conformable. Potential procedural complications include interference with valve

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Figure 1. Amplatzer plug positioned in the paravalvular defect

Figure 2. TOE at 6-week follow-up showing stable device position
leaflet movement, embolisation of thrombus, air, atheroma or the device itself, haemolysis, bleeding, and infection.\textsuperscript{10}

**Conclusion**

With increasing numbers of aortic valve operations being undertaken (particularly in the elderly), the burden of significant PVL requiring repeat intervention will increase. In those patients in whom the risk of re-operation is high, percutaneous obliteration appears to be a technically feasible and safe alternative.

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**References:**

A case of pneumococcal aortitis presenting with back pain

Daniel Garofalo, Matthew Dawes

A 61-year-old man presented to hospital with a 2-day history of sudden onset lower back pain and chills. His medical history included hypertension, obesity, and chronic renal impairment. On examination he was sweaty, tachycardic, and dehydrated but afebrile. He had tenderness over the lumbar spine. Initial investigations showed a white cell count of 11.21×10^9/L with a left shift and a C-reactive protein (CRP) of 329 mg/L. The chest X-ray (CXR) revealed cardiomegaly but no evidence of consolidation.

Initial differential diagnoses included osteomyelitis/spinal abscess but a magnetic resonance imaging (MRI) scan could not be performed due to the patient’s habitus. Twenty-four hours post admission two blood cultures grew a fully-sensitive Streptococcus pneumoniae. Intravenous benzylpenicillin was commenced at 1.2 grams 6 hourly.

A computed tomography (CT) thorax/abdomen scan showed no evidence of pneumonia and the abdominal aorta was ectatic but there were no aneurysms. There were streaky soft tissue changes posterior to the proximal abdominal aorta, suggestive of aortitis, with retroperitoneal fluid tracking into the pelvis, but no signs of vertebral osteomyelitis or paravertebral collection. A transoesophageal echo excluded vegetations or dissection. A bone scan was normal.

A working diagnosis of pneumococcal abdominal aortitis was made. The patient remained on intravenous antibiotics with low grade temperatures and was reviewed by vascular surgery. There were ongoing complaints of intermittent pains in the chest, neck, interscapular region, and knee. The CRP remained elevated.

Two weeks after admission the patient developed left calf pain, and a Doppler ultrasound confirmed a deep vein thrombosis. There was an incidental finding of a left popliteal aneurysm measuring 3.9 cm. Anticoagulation was started and a CT angiogram requested. This showed, almost 3 weeks after the original CT, aneurysms all along the aorta, from the left carotid origin down to the iliac arteries (Figure 1). At this point surgical intervention was considered impracticable.

On day 24 after admission the patient suffered a cardiac arrest (pulseless electrical activity) and died.
Figure 1. CT aortogram showing multiple aneurysms on a tortuous abdominal aorta and right iliac artery
Discussion

Bacterial aortitis is rare in the antibiotic era and the mechanisms of arterial infection are varied. Infection may complicate prosthetic vascular grafts or can be spontaneous in native vessels. If spontaneous this may be due to:

1) Seeding of an abnormal vessel during a bacteraemia (e.g. pre-existing aneurysm or atherosclerotic plaque becoming secondarily infected);

2) Septic embolisation of the vasa vasorum (e.g. from endocarditis) with subsequent development of a mycotic aneurysm;

3) Direct infection secondary to trauma; or

4) Direct spread of infection from a local extra-vascular source. 1,2

It is likely that mechanism 1) is responsible in our arteriopathic patient.

Pneumococcus is an unusual cause of aortitis. The most common pathogens include Salmonella and Staphylococcus, occurring in 38% and 19% of a series of 21 patients, respectively. 3 In a review of 37 patients with pneumococcal aortitis (reported since 1908) 4 approximately 50% of blood cultures were positive and the region of involvement was: 54% abdominal aorta; 30% descending thoracic aorta; and 15% ascending aorta.

Only 5 of 19 patients, with pneumococcal aortitis, treated with surgery and antibiotics, died. 4 Our patient demonstrated the rapid progression (2–3 weeks) of a pneumococcal aortitis within an initially atherosclerotic but non-aneurysmal abdominal aorta despite treatment with intravenous antibiotics. This is in keeping with previous reports of a dynamic progressive phase between initial infection and subsequent infected aneurysm formation. 5

The classical description of aortitis is non-specific including fever, abdominal and back pain, pulsatile abdominal mass (if there is an aneurysm) and positive blood cultures. Typically it presents in older males with atherosclerosis or vascular risk factors (as exemplified by our case). 4 The non-specific presentation and rarity of bacterial aortitis means clinical diagnosis is difficult and delayed but it should remain, with other endovascular infections, as a differential diagnosis in febrile patients. 6

The classical description of aortitis is non-specific including fever, abdominal and back pain, pulsatile abdominal mass (if there is an aneurysm) and positive blood cultures. Typically it presents in older males with atherosclerosis or vascular risk factors (as exemplified by our case). 4 The non-specific presentation and rarity of bacterial aortitis means clinical diagnosis is difficult and delayed but it should remain, with other endovascular infections, as a differential diagnosis in febrile patients. 6

CT scanning is the initial imaging technique and can show features such as periaortic inflammation or aneurysms. MRI may further define the extent of disease and help with planning surgery. 2

Bacterial aortitis has a mortality that approaches 100%, often from arterial rupture, if treated by medical therapy alone. 6 This compares with a mortality rate of 20% in cases of pneumococcal bacteraemia not associated with aortitis. 7 Optimal management requires clinical suspicion, early diagnosis, and prompt surgery in addition to intravenous antibiotics preoperatively and for an extended period postoperatively (4 to 6 weeks). 4

Controversy exists with respect to the best surgical approach with the overriding concern being infection of graft material. 8 Wide resection of infected tissue is recommended with prosthetic bypass through clean tissue planes. 1 This approach results in mortality rates reported between 20 to 60%. 6
This case describes a rare cause of back pain and sepsis. However, bacterial aortitis does require prompt recognition to allow for consideration of early surgical management. Our patient demonstrates the rapid clinical and radiological deterioration of pneumococcal aortitis when managed with antibiotics alone.

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**References:**

Leri’s hand

Sujit Nair, Olliver Byass

Clinical

A 29-year-old woman presented to her general practitioner with a painful swollen hand.

What is the diagnosis?
Answer

*Melorheostosis* (Leri’s disease)

**Findings**

Antero-posterior (AP) and oblique view plain radiograph shows hyperostosis and cortical bone expansion of middle finger metacarpal and phalanges. These appearances are described as ‘dripping candle wax’ (see arrows in Figure 1).

**Discussion**

Melorheostosis is a rare benign hyperostotic bone dysplasia that affects both sexes. It predominantly affects the appendicular skeleton. The usual age of presentation is late adolescence or early adulthood. Patients are often asymptomatic, with the condition diagnosed as an incidental finding.

Melorheostosis affects both soft tissue and bone and can result in pain, swelling, contracture, restriction of joint movements, and bone deformity. Hyperostosis and dripping candle wax sign on plain radiograph indicates melorheostosis and seldom need any further imaging.

Treatment options include medications (to relieve pain), physiotherapy, and surgery (to correct deformity).

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Anton’s syndrome secondary to cerebral vasculitis

Rayid Abdulqawi, Khaled Ashawesh, Saqib Ahmad

A 35-year-old woman presented with a 2-day history of progressive visual loss, noted by her husband. On examination, she was alert and she had no light perception in either eye; pupils were equal in size with normal light reflex. Fundoscopy revealed a normal optic disc and retina.

A computed tomography (CT) scan of the brain, shown in Figure 1, revealed large wedge shaped areas of low attenuation of both occipital lobes (arrows) in keeping with acute infarcts.

Subsequent magnetic resonance (MR) angiography (Figure 2) showed paucity of flow in both posterior cerebral arteries with areas of irregularity (arrows) suggestive of vasculitis affecting both posterior cerebral arteries. There was no systemic evidence of vasculitis in investigations.

High-dose steroid therapy led to a marked improvement in visual fields and moderate improvement in the visual acuity (6/18 both eyes).

Figure 1. CT head shows large wedge shaped areas of low attenuation of both occipital lobes in keeping with acute infarcts

Figure 2. MR angiography showing paucity of flow in both posterior cerebral arteries with areas of irregularity
Discussion

Anton's syndrome is a form of cortical blindness in which the patient denies the visual impairment. The patient may attempt to walk, bumping into objects and injuring themselves. Anton's syndrome is caused by damage to the occipital lobe which extends from the primary visual cortex into the visual association cortex.

Major causes are cerebrovascular disease, cardiac surgery, cerebral and coronary angiography,\(^1\) and head injury. Cerebral vasculitis is a rare cause.\(^2\)

Our case highlights recognition of this rare form of cortical blindness so that immediate treatment can be initiated.

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**References:**

Dr WB Tripe


It is with much regret we have to record the death of Dr. William Borrowdale Tripe, which took place at his residence, Upper Willis Street, Wellington, on February 16th. Dr. Tripe has been in practice in Wellington since 1878. He was born July 25th, 1822, and was therefore 85 years of age. He was the son of a surgeon in the Royal Navy, and had many friends in the Army and Navy.

He was educated in London, and qualified from the London Hospital. In New Zealand he practised his profession first in Rangiora, then in Picton, moving finally to Wellington. For many years he was Honorary Consulting Physician to the Wellington Hospital.

Always a courteous gentleman, exact in the performance of his duties, careful to do all in his power to maintain the honour of his profession, he was sincerely liked by everyone, and though of late years increasing weakness prevented him engaging in active practice, his white hair and well knit frame were often seen about near his home, and will be much missed in Wellington.

Dr. Tripe is survived by his wife and a family of nine sons and two daughters.

NZMJ Note: Additional details at http://www.ccdhb.org.nz/hhist/staff/TripeWB_N.html (navigate to other staff via the grey arrows on the webpage)
Melatonin and sleep disorders

Being both interested and ignorant I read this paper. First the facts—melatonin is a chronobiotic, a hormone that adjusts the timing of the central biological clock, including the timing of the sleep-wake cycle. Then the hypotheses about when it should be used—children with neurodisabilities, those with delayed sleep-phase syndrome, the jet-lagged, the blind and maybe the elderly demented. And even perhaps in the healthy who do not sleep well?

The dosage range, 0.5–12 mg, is wide and the timing disputed. The authors of this paper observe “that there no doubt that the prescribing of melatonin is continuing to increase despite a poor scientific base, and with no consistent guidance on the dose and when it should be given.”

Fortunately we seem to be spared from this debate in New Zealand?

Clinical Medicine 2008:8:381–3

Outcomes of patients with transient ischaemic attack (TIA) after hospital admission or discharge from the emergency department

This paper is based on the premise that TIA are a warning of stroke and that half of such strokes are within a month.

All TIA presentations to EDs in a large metropolitan and rural region of Sydney and its surroundings, New South Wales, between 2001 and 2005 were extracted from state health department databases and followed up over 1 year.

Of 2535 presentations to an ED with TIA during the 5-year period, 1816 patients were admitted to hospital (71.6%) and 719 were discharged from the ED (28.4%).

At 28 days the discharged group had a significantly higher rate of further TIA or strokes. Hence the authors recommend admission. Presumably the benefit accrues from investigation and appropriate treatment. An accompanying editorial agrees but raises the issues of workload and resources. It also includes a useful tool, ABCD, for assessment of such patients.

ABCD tool for assessment of patients with transient ischaemic attack

A. Age ≥60 years = 1 point.
B. Blood pressure ≥140/90 mmHg = 1 point.
C. Clinical features: unilateral weakness = 2 points, speech impairment alone = 1 point.
D. Duration >60 minutes = 2 points, 10–59 minutes = 1 point.
E. Diabetes = 1 point.
Total. 0–3 = low risk of stroke, 4–7 = high risk of stroke.

Conservation care versus early surgery in patients with sciatica caused by lumbar disc herniation

Opinion is divided on this issue. Two papers from the Netherlands report on a randomised trial involving 283 patients with sciatica for 6–12 weeks caused by lumbar disc herniation.

Early surgery achieved more rapid relief of sciatica than conservative care, but outcomes were similar by one year and these did not change during the second year. So that settles that—or does it? It transpires that 44% of those in the conservative arm subsequently had surgery. And to further complicate the issues, the authors performed an economic analysis—they claim that faster recovery from sciatica makes early surgery likely to be cost effective compared with prolonged conservative care.

So—opinion is still divided on this issue.


Atrial fibrillation and heart failure—rhythm control versus rate control

It is common practice to restore and maintain sinus rhythm in patients with atrial fibrillation and heart failure. This multicentre trial attempts to elucidate whether restoration of sinus rhythm is better than rate control. 1376 patients recruited from 123 centres in 6 countries were randomised to each arm of treatment.

Baseline characteristics of the groups were well matched. The primary outcome, death from cardiovascular cause, was similar (p=0.59). Secondary outcomes such as death from any cause, stroke or worse heart failure were also not significantly different in either treatment arm. So rhythm control is not better than rate control.


Accurate blood pressure management

Most physicians and their staffs do a poor job of measuring patient blood pressure, said hypertension experts at the scientific meeting of the American Society of Hypertension (ASH) held in New Orleans recently.

Clarence E. Grim, MD, a clinical professor of medicine at the Medical College of Wisconsin in Milwaukee pointed out that an error of –5 mmHg for diastolic pressure in the 90 to 95 mmHg range would miss 21 million US patients with hypertension.

Adding an erroneous 5 mmHg to diastolic readings in the 85 to 90 mmHg range would cause 27 million people to be misdiagnosed with hypertension, driving up medical costs and exposing patients to adverse effects of unnecessary medication.

How to avoid these “grim” errors—use a properly fitted cuff, have the patient seated with the cuff at heart level and have the patient relaxed and rested for 5 minutes.

(Article can be obtained from http://jama.ama-assn.org/cgi/content/full/299/24/2842)

JAMA 2008;299:2842–4
HIV transmission: the ongoing importance of antenatal screening

We write to report three cases of mother-to-child HIV transmission in New Zealand which occurred in the years 2002–2006. In all three cases, HIV antibody testing was not performed as part of antenatal screening, and children were subsequently diagnosed with HIV infection. The women and children have since immigrated to Australia where their ongoing care is being undertaken.

Prenatal HIV transmission can be prevented, however interventions depend on the woman and her health care provider being aware of her HIV status. These cases support the recent decision by New Zealand’s Ministry of Health to recommend universal antenatal HIV screening, and enable strategies to prevent perinatal HIV transmission.

Case 1: A 35-year-old woman, born in Vietnam, migrated to New Zealand in 1998. Her daughter was born via Caesarean section in 2002. Her husband was diagnosed with HIV infection in 2003. Subsequently she and her daughter were both found to be infected with HIV.

Case 2: A 32-year-old woman, originally from Zimbabwe, migrated to New Zealand in 2002. Her son, who was born in 2004, was diagnosed with HIV infection at the age of 3, and subsequently she was also confirmed to be HIV infected.

Case 3: A 31-year-old woman from East Timor migrated to New Zealand in 2002. Her son, who was born in 2006, was diagnosed with HIV infection after developing a respiratory illness in 2007, and subsequently the mother was also diagnosed with HIV infection.

These three cases of newly diagnosed HIV infection in children exposed perinatally via mothers unaware of their HIV status, highlight the missed opportunity for antenatal HIV testing which may have limited further perinatal HIV transmission. In 2004, a New Zealand Ministerial Review of antenatal HIV screening was undertaken. A pilot programme was initiated following the review, involving an education program for midwives and general practitioners which aimed to increase uptake of HIV testing amongst pregnant women. It was undertaken, first of all, in the Waikato District Health Board from March 2006.

This programme was successful, with a 99.7% uptake of antenatal HIV testing in the first year. Subsequently, the Ministry of Health published guidelines in February 2008 which recommend offering all pregnant women an HIV test.

All three women had their antenatal care in New Zealand, two prior to the Ministerial review of antenatal HIV testing. The third case occurred during the time of implementation of the pilot project. In addition, even prior to the recommendation for universal HIV screening in pregnancy, two of the women came from regions with high HIV seroprevalence; 0.5%–1% in Vietnam and 18% in Zimbabwe. There is minimal data on HIV seroprevalence in East Timor.
These three cases highlight the limitations of a selective screening approach. In light of this, many developed countries with both low and high HIV seroprevalence now recommend universal antenatal HIV screening including the United States, the United Kingdom, and Australia. Knowledge of a woman’s HIV status is of benefit for her own health, to limit further transmission to partners and is essential to enable uptake of interventions during pregnancy to reduce HIV transmission to her child. With a combination of antiretroviral therapy to the mother and child post partum, elective Caesarean section, and avoidance of breastfeeding, mother-to-child transmission of HIV has been reported as less than 2%.

These cases reinforce the importance of routine offer of an antenatal HIV test in both Australia and New Zealand. It is not a guarantee that perinatal transmission will not occur, but is the first step in minimising the chance of mother-to-child HIV transmission.

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References:
The ethics of chiropractic

The recent article by Gilbey inspired me to monitor the ethical behaviour of UK chiropractors.

I googled “Chiropractic Clinics UK” (31/07/2008) and evaluated the contents of the first 10 websites of individual chiropractic clinics listed. My aim was to find out whether chiropractors adhered to their own ethical code: it states, amongst other things, that “chiropractors must not use any title or qualification in such a way that the public may be misled as to its meaning or significance. In particular, chiropractors who use the title ‘doctor’ and who are not registered medical practitioners must ensure that they make it clear that they are registered chiropractors and not medical practitioners.” It furthermore states that “If chiropractors, or others on their behalf, do publicise the information used must be factual and verifiable. The information must not be misleading or inaccurate in any way.”

Thus I extracted the chiropractors’ use of the title ‘doctor’ and the therapeutic claims made on the 10 websites. The claims were subsequently checked against the published evidence.

The results are summarised in Table 1. Six of the 10 clinic directors used the title ‘doctor’ without making it clear whether or not they are registered medical practitioners. All but one website advertised chiropractic for conditions for which there is no good evidence of the effectiveness of chiropractic manipulations. Many of these conditions are unrelated to back and neck or other musculoskeletal problems.

Table 1. Data extracted from 10 websites

<table>
<thead>
<tr>
<th>Clinic</th>
<th>Listed title of Director</th>
<th>Indications not supported by good evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Dr</td>
<td>Whiplash, PMS, a variety of organic and systemic problems</td>
</tr>
<tr>
<td>B</td>
<td>no title</td>
<td>Jaw joint pain, infantile colic, childhood asthma, bed wetting</td>
</tr>
<tr>
<td>C</td>
<td>Dr</td>
<td>Sleeping problems, lethargy/lack of energy, digestive/bowel disorders, heartburn, pregnancy and infancy, infantile colic</td>
</tr>
<tr>
<td>D</td>
<td>Dr</td>
<td>None</td>
</tr>
<tr>
<td>E</td>
<td>Dr</td>
<td>Poor concentration, grinding teeth, whiplash, asthma, colic, sleeping and feeding problems, RSI, breathing difficulties, hyperactivity, bed wetting, frequent infections especially in the ears</td>
</tr>
<tr>
<td>F</td>
<td>Dr</td>
<td>Whiplash, RSI</td>
</tr>
<tr>
<td>G</td>
<td>no title</td>
<td>Infantile colic, growing pains of babies, correcting the effects of the rough and tumble of life</td>
</tr>
<tr>
<td>H</td>
<td>Dr</td>
<td>Bed wetting, colic, ear infections, PMS, pregnancy, whiplash</td>
</tr>
<tr>
<td>I</td>
<td>no title</td>
<td>Whiplash, RSI</td>
</tr>
<tr>
<td>J</td>
<td>no title</td>
<td>Bursitis, rotator cuff tears, tendonitis, whiplash</td>
</tr>
</tbody>
</table>

RSI = repetitive strain injury.
These findings add to the findings by Gilbey\textsuperscript{1} and confirm previous findings from North America.\textsuperscript{5} They suggest that many chiropractors violate their own ethical code. Langworthy et al recently showed that only 23\% of UK chiropractors discuss serious risks of spinal manipulation with their patients before treating them.\textsuperscript{6} This lack of obtaining informed consent would constitute a further infringement on ethical guidelines.\textsuperscript{2}

In the UK, chiropractors are independent primary healthcare professionals regulated by statute since the Chiropractors Act of 1994. The General Chiropractic Council has the duties of protecting the public, setting standards and developing the profession.\textsuperscript{2}

My analysis is, of course, based on very small numbers and therefore not conclusive. Its results nevertheless suggest that these duties are performed less than optimally.

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Reference:


Colquhoun reply to chiropractic-defending letters from Kelly, Bale, and Roughan

I’m grateful for the opportunity to reply to the defences of chiropractic from Kelly, Roughan, and Bale in your last issue.

I’d like first to deal with the minor matter of titles, before getting onto the more important question of evidence. I notice that Brian Kelly signs his letter “Dr Brian Kelly B App Sci (Chiro)” in his letter to NZMJ. He seems to be a bit less careful in his use of titles on his own school’s website where his President’s welcome is signed simply “Dr Brian Kelly”, a title he adopts in at least three other places. Karl Bale (CEO/Registrar, Chiropractic Board New Zealand) points out that “Failure to qualify the use of the title ‘Doctor’ may contravene the provisions of the Medical Practitioners Act 1995”. Karl Bale also points out that some ruthless sales methods characteristic of chiropractic are contrary to the Chiropractic Board’s code of ethics.

It seems to me quite remarkable that none of the letters mentions the ‘subluxation’ that lies at the heart of their subject. Could that be because they are reluctant to admit openly that it is a mere metaphysical concept, that no one can see or define? It is sad that so many patients are subjected to X-rays in search of this phantom idea. It is this metaphysical nature of chiropractic that separates it quite clearly from science.

Brian Kelly says “How can any reader take seriously, anything suggested by a writer who opines that a 19th Century journalist possessed superior “intellectual standards” to “the UK’s Department of Health” and “several university vice chancellors”. The views of the Davenport Leader on chiropractic were mild compared with those of the great H.L. Mencken (1924) who wrote “This preposterous quackery flourishes lushly in the back reaches of the Republic, and begins to conquer the less civilized folk of the big cities.”...The problem is that the Department of Health is full of arts graduates who may be very good at classics but can’t understand the nature of evidence. And the UK has one vice-chancellor, a geomorphologist, who defends a course in his university that teaches that “amethysts emit high yin energy” I’ll admit, though, that perhaps ‘intellect’ is not what’s deficient in this case, but rather honesty.

Your correspondents seem to confuse the duration of a course with its intellectual content. You can study homeopathy for years too, but after all that they are still treating sick people with medicines that contain no medicine. Anyone who works in a university knows that you can easily get accreditation for anything whatsoever if you choose the right people to sit on the committee. I have seen only too many of these worthless pieces of paper. “Amethysts emit high yin energy” was part of an accredited course (at the University of Westminster) too, Need I say more?

Now to the real heart of the problem, namely the question of evidence. Brian Kelly says that the book by Singh and Ernst shows “extreme bias”, but what that book actually shows is an extremely scrupulous regard for evidence, Ernst is in a better position to do this than just about anyone else. He has qualified and practised both regular and alternative medicine, and he was appointed to his present position, as
professor of complementary and alternative medicine to assess the evidence, Perhaps most importantly of all, his position allows him to do that assessment with complete lack of bias because, unlike Kelly, his livelihood does not depend on any particular outcome of the assessment. I’m afraid that what Kelly describes as “extreme bias” is simply a display of pique because it has turned out that when all the evidence is examined dispassionately, the outcome is not what chiropractors hoped.

The fact of the matter is that when you look at all of the evidence, as Singh & Ernst do, it is perfectly clear that chiropractic is at best no better than conventional treatments even for back pain. For all other conditions its benefits fail to outweigh its risks – contrary to the many claims by chiropractors. Both the New Zealand and the UK governments have got themselves into an impossible position by giving official recognition to chiropractic before the evidence was in.

Since the conventional manipulative treatments are cheaper, and may be well be safer, and because they involve no quasi-religious ideas like “subluxation” or “innate intelligence”, the only reasonable conclusion is that there is no need for chiropractic to exist at all. They do nothing they do that could not be done as well by medical practitioners and physiotherapists. What will governments do about that, I wonder?

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References:

Colquhoun's opinion versus science—a response from the New Zealand Chiropractors' Association

In response to Colquhoun's letter regarding the concept of subluxation in chiropractic we offer the following:

Chiropractic is concerned with the relationship of body structure (primarily the spine) to function (as co-ordinated by the nervous system). It is the only profession dedicated to the analysis, correction and prevention of the vertebral subluxation complex, otherwise known simply as “subluxation”.

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Yes, the concept of subluxation is a central premise in chiropractic. Colquhoun's dismissal of it as a “mere metaphysical construct” or “phantom idea” serves only to demonstrate his lack of knowledge and understanding in this arena.

The term 'subluxation' has a colourful history and has been the subject of debate both within and outside the chiropractic profession.\(^1,2\) Contrary to Colquhoun's personal opinion and unsubstantiated references, the concept is sound and scientific research from various fields is helping to confirm its existence and effects.

Unfortunately, to date most funds for complementary medicine research have been diverted into simplistic or poorly designed clinical trials which are of limited or no value for studying such phenomena. However, progress is being made using neuro-physiological measurements and functional imaging. Such research will allow for various phenomena detected by trained CAM practitioners to be objectively measured (e.g. subluxations in chiropractic, cranial rhythms in osteopathy, and Qi in Chinese medicine).

The term subluxation is derived from \(sub\) = less than, \(luxation\) = dislocation. The original simplistic concept of ‘a bone out of place pressing on a nerve’ has been significantly developed and refined with increasing knowledge of spinal function and nerve physiology (no longer limited to just the action potential).\(^2-4\)

A comprehensive, but by no means complete, description of the subluxation is the five component model known as the “Vertebral Subluxation Complex” which details key features of this important clinical entity under the categories of spinal kinesiopathology (abnormal motion or position of spinal bones), neuropathophysiology (abnormal nervous system function), myopathology (abnormal muscle function), histopathology (abnormal soft-tissue function), and pathophysiology (abnormal function of the spine and body as a whole). Each of these components can and has been observed, measured and documented.\(^2-6\)

New research and contemporary thinking is focusing on the afferent aspect of nerve communication; specifically on how altered input from spinal joints affects central nervous system processing (a phenomenon often termed dysafferentation or more simply ‘garbage in – garbage out’).\(^7,8\)

There are two NZ chiropractors (who incidentally hold PhDs) currently researching various neuro-physiological aspects of chiropractic at the University of Auckland's Medical School, a prestigious institution that Colquhoun would be hard pressed to cast aspersions on.

Studies continue to elucidate and characterise the numerous and varied devastating effects that subluxations can have on overall health and function. The World Health Organization accepts it as a listing in the latest international classification of disease and related health problems, referred to as M99.1 Subluxation complex (vertebral). Doctors of chiropractic are the only professionals specifically trained and dedicated to analysing and correcting vertebral subluxations.

The Council on Chiropractic Practice has published comprehensive evidence-based guidelines on chiropractic care (www.ccp-guidelines.org) including evidence ratings for various aspects of clinical practice.
Investigation of neuro-musculo-skeletal dysfunction is a growing area of interest involving chiropractic researchers, clinicians, and other independent researchers. Much of the evidence for the effects of subluxation increasingly comes from outside the chiropractic profession and is found in such varied fields as the basic sciences, medicine, psychology, biophysics and engineering, neurophysiology, and the relatively new field of psycho-neuro-immunology.\textsuperscript{2,3,9}

It is only through inter-disciplinary co-operation that we can hope to further understand the complexities and interactions of the human organism as an integrated whole. Much progress has been made and more is yet to be documented. Traditionalists in medicine may find it hard to accept the move away from pure reductionist models.

As with any subject, one can choose to form closed opinions based on biased discourse, or can hold an open mind with a healthy dose of scepticism. As Colquhoun should be aware, “expert opinion” like Ernst’s rates as the least robust form of evidence but he relies heavily upon it when making his rather tiresome and repetitive assertions.

Modern chiropractors are not claiming that all disease or dysfunction is a result of subluxation. Disease and dysfunction can of course occur with, because of, or despite subluxations. Chiropractors recognise that lifestyle, environment, nutrition, toxins, genetics, and emotional state all influence health and well-being.

Finally to address Colquhoun’s statement that chiropractors “do nothing that could not be done as well by medical practitioners and physiotherapists” and “what will governments do about that, I wonder?”, we recommend he read the New Zealand Government’s thorough Report on the Commission of Inquiry into Chiropractic in 1979 to find out what has already been done and recommended.\textsuperscript{10}

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References:


A rejoinder to Professor Kevin Dew’s letter “Who is confused by courtesy titles?”—and response

Thank you for the opportunity to reply to some of the points raised in the letter by Professor Kevin Dew in your last issue.¹

When referring to my previous rejoinder,² Professor Dew is quite right to point out my oversight when I suggested that complementary and alternative medicine (CAM) “has little or no theoretical rational”; however, it is somewhat of an overstatement to then suggest it is indicative of my limited knowledge of the area. In hindsight, I should have added the word ‘sound’ before ‘theoretical rationale’, or used the phrase I used in my original article ([the] “scientific rationale…[is] not as strong as for mainstream medicine”).³

As such, I suggest that if I am correct once, but commit an oversight the second time, then Professor Dew’s interpretation is entirely unwarranted. What my slip does show, however, is the value of peer-review and editing to detect instances when authors use inappropriate words, fail to explain things clearly, or misspell the occasional name (as the original paper was peer-reviewed, but my rejoinder was not), in addition to more serious issues, such as overgeneralisation, inappropriate design, and biased interpretation of findings.

Traditionally, social psychologists draw upon the expertise of others in their area when conducting research—that is what I did when referring to scientific rationale. This makes sense, as clearly not all researchers can be acknowledged experts in the area of their research.

Professor Edzard Ernst, holder of the Laing Chair in Complementary Medicine at the University of Exeter (UK), who is widely reputed to be an expert in his area, has this to say about the scientific rationale of some popular CAM practices: “The primary premise that subluxation is the cause of all illness has no scientific rationale” (chiropractic); “Their scientific rationale is not fully convincing. In particular, the theory of the overriding importance of alignment lacks a scientific rationale (osteopathy); “No evidence has been found to confirm the existence of Qi or meridians” (acupuncture); “There is no known neurophysiological basis for connections between organs or other body parts and specific areas of the feet” (reflexology); and “Presently there is no scientific rationale for understanding how remedies devoid of pharmacologically active molecules produce clinical effects” (homeopathy).⁴

When Professor Dew refers to “Gibley’s [sic] rigorous methodology of looking at the yellow pages”, he could inadvertently undermine the rigour of my research if his readers were to infer that there was some justifiable sense of irony in his words. I did indeed review the Yellow Pages—that is hardly surprising, given it was stated as the source of my secondary data. To be precise, I reviewed the listings for each CAM practice, for each directory area, at least twice whilst conducting my analysis.
When I did not get two identical consecutive counts, I reanalysed the data until I did. My analysis paid particular attention to the fact that many CAM practitioners appear twice under the same heading, in both box and line listings. I clearly stated in my methods section that when this was the case I would use the entry most likely to mislead, as the potential to mislead was the primary focus of my paper. Furthermore, using the Yellow Pages as a source of secondary data is not at all uncommon, as a quick search of the article database http://scholar.google.co.nz/ will quickly reveal. For example, in New Zealand health research, Jopson and Reeder used the New Zealand Yellow Pages and were funded by the Cancer Society of New Zealand Inc.

It is not surprising at all that Professor Dew managed to find instances where he perceived that practitioners stated clearly the area in which they were a ‘doctor’ (although I am not convinced that calling oneself Doctor X in a box advert for a named chiropractic clinic does indeed make clear a practitioner’s title may be one of courtesy).

A common pitfall for researchers is to overlook the fact it is often very easy to quickly find some evidence in support of one’s hypothesis; unfortunately, due to the phenomenon of ‘researcher bias’, whatever is found may tend to be biased in favour of one’s hypothesis and thus of little value.

My original article sought to ascertain the ratio of practitioners who use the title doctor, in a way that could lead people to believe they were consulting mainstream medical practitioners, in relation to those who do not. This is quite different from seeking evidence to support an a priori hypothesis that some practitioners make clear they are doctors of a particular type of CAM. So, in my original exploratory study, in which I had no a priori hypothesis—and would thus be less prey to researcher bias—I sought simply to count instances where the title doctor was used without clear qualification (no pun intended), relative to instances where the title doctor was not used. I was unsure of what I would find and, from the outset, intended merely to let the data speak for itself, as most social scientists would do.

Professor Dew suggests the argument should be moved on to answering a number of research questions. I wholeheartedly agree and should point out that I did suggest a further research idea in my original paper. To get more value from the proposal that Professor Dew suggested in his most recent letter, perhaps a quantitative component could also be included (e.g. on a scale of 1 to 10, how likely do you think this person is a qualified medical practitioner like your family doctor, is based upon proper research trials for its efficacy, etc) along with a meaningful comparison group.

So, rather than an uncontrolled one-shot design exploring the (qualitative) beliefs of people visiting a CAM practitioner who use the title of doctor, perhaps an experimental design could be implemented, whereby the perceptions of people consulting CAM practitioners who call themselves doctors could be compared to the perceptions of people consulting practitioners who do not call themselves doctors. After a predetermined number of consultations had occurred, it would then be possible to answer three interesting and pertinent questions: i) do CAM practitioners using the title ‘doctor’ receive more consultations than those not using the title; ii) does the mean client perception of the two groups differ; and iii) does the use of a prestigious title, such as doctor, affect clinical outcome.
A similar proposal could also be conducted using other occupations that use the honorific title doctor, such as vets and dentists.

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References:

A plea for sophistication

Andrew Gilbey makes an effort at distancing himself from a priori theorising when it comes to undertaking research in relation to the title of ‘doctor’ but his suggestions on future research demonstrate how difficult this is. I refer to his suggestion for quantitative research where participants would respond to a question “on a scale of 1 to 10, how likely do you think this person is a qualified medical practitioner like your family doctor, is based upon proper research trials for its efficacy, etc”. This could be interpreted, by respondents at least, as equating the practice of the family doctor with the findings of ‘proper research trials’. (As an aside—what sense anyone would make of the word ‘proper’ here is anyone’s guess—and this would require even more research. Here we encounter the problem of ‘auxiliary’ hypotheses when testing a theory.’ Is Andrew Gilbey’s idea of proper the same as everyone else’s?).

I have been involved, alongside clinical practitioners, in research closely analysing interactions between patients and health professionals. There is a great deal of work that is undertaken in clinical consultations that is not based on “research trials”. This is not something to be concerned about—but is an inevitable consequence of the very
complex nature of the clinical consultation, where prescribing a drug that has made it through the trial process can be just one component.

Clinicians are weighing up complex issues of drug interactions, co-morbidities, social factors (such as their impression of whether patients are likely to comply with advice given), physiological resistance to medications and so on. This is a very heady mix. Research trials may provide some help for clinicians in some situations, but it is very clear that clinicians are drawing on their experience and their own understanding and values. In addition, clinicians cope with the uncertainties inherent in clinical practice.  

To provide a misleading question in a quantitative survey (suggesting that what family doctors do is based on research trials) perpetuates a myth—but a myth that clinicians themselves have long since discarded. A research tool of the nature proposed would be at best useless, and at worst misleading.

I think we desperately need to move the debate beyond crude dichotomies between ‘bad’ and irrational alternative medicine and ‘good’ and rational orthodox medicine. It is clear that most General Practitioners in New Zealand have moved well beyond this simplistic view, seen in the very high numbers of GPs who refer to CAM therapists.

Clinical practice is far more complex than this crude dichotomy implies, and requires a more sophisticated understanding from researchers.

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References:
Properties of “light” cigarettes sold in New Zealand

“Light” or “mild” cigarettes have historically been marketed to appeal to health concerned smokers, and positioned as an alternative to quitting. But despite this marketing strategy, there is evidence that “light” cigarettes often deliver as much tar as regular cigarettes. Furthermore, there is epidemiological evidence that suggests no significant health benefit in terms of lung cancer, heart disease or chronic lung disease for smoking “light” versus other cigarettes.

Even more concerning is that the marketing appears to work in that there is evidence that smokers do indeed use “lights” as an alternative to quitting. In New Zealand, there is also some local survey evidence that smokers believe that “light” cigarettes do less harm than regular cigarettes. In response to misleading marketing around “lights”, the Framework Convention on Tobacco Control (FCTC) requires ratifying countries to enact laws that prohibit misleading descriptors and specifically mentions the terms: “light”, “mild”, “ultra-light”, and “low tar”.

As part of the New Zealand arm of the International Tobacco Control Policy Evaluation Survey (ITC Project), we purchased factory-made cigarettes of the 11 leading cigarette varieties in New Zealand (based on sales figures').

Purchases were made in August 2007 at two urban supermarkets (suburban and central Wellington) and a store in a rural town (in the Wairarapa) with six packs of each variety per location. One pack of each variety was used for physical property testing (at Roswell Park Cancer Institute) and four packs of each variety were used in the emissions testing using both the ISO and Canadian Intense conditions testing regimens at an independent contract laboratory (Labstat International, Kitchener, Ontario, Canada).

The Canadian regime more closely mimics real-life smoking, as it involves blocking cigarette vent holes to enhance nicotine delivery. Five cigarettes per pack were tested and the product testing followed methods previously reported. A full description of the methods and results are available in an online report. We undertook comparisons between brands marked “light” or “mild” (n=4) and other cigarettes (n=7), and these are reported herein.

For most characteristics, the “lights” were fairly similar to regular cigarettes (e.g. for: cigarette length, filter length, wet weight, dry weight, tipping paper length, vacuum porosity, pressure porosity etc). All variants tested had vent holes except for one regular brand variant. However, “lights” were significantly more ventilated (34% vs 8% on average) and this was associated with a lower pressure drop or “draw resistance” (see Table 1).

At first glance at the table, “lights” might appear less health damaging than regular cigarettes due to lower levels of tar and carbon monoxide (CO) inhaled per cigarette (Table 1). However, the differences between regular brands and “lights” for tar and nicotine yields were less (and some differences were not statistically significant e.g. for CO) for the Canadian test that is thought to better approximate actual smoker
behaviour than with the ISO test. Importantly though, studies of smokers’ behaviour show that they generally smoke to achieve specific levels of nicotine delivery, and will inhale deeper and take more puffs when smoking lower nicotine content cigarettes (as reviewed by Hammond et al11). In doing so they will also inhale more tar, CO and other toxic constituents. Therefore, other relevant measures are the ratios of tar to nicotine and CO to nicotine (last four rows in Table 1), as this gives an indication of the amount of tar and CO that will be inhaled to receive a given dose of nicotine.

Table 1. Test results for brand variants of “lights” and regular factory-made cigarettes which are popular in the New Zealand market

<table>
<thead>
<tr>
<th>Property*</th>
<th>Regular brand variants (average results – 7 variants)**</th>
<th>“Light” brand variants (average results – 4 variants)**</th>
<th>Test for difference (ANOVA unless indicated otherwise)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physical properties (showing those with significant differences)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure drop (mm water)</td>
<td>110.21</td>
<td>101.63</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Ventilation (%)</td>
<td>7.58</td>
<td>33.91</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td>Filter weight (g)</td>
<td>0.11</td>
<td>0.13</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td><strong>Tar levels</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ISO tar average inhaled per cigarette (mg)</td>
<td>12.64</td>
<td>8.47</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td>Canadian tar average inhaled per cigarette (mg)</td>
<td>31.87</td>
<td>28.38</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>ISO tar per puff (mg)</td>
<td>1.61</td>
<td>1.09</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td>Canadian tar per puff (mg)</td>
<td>3.10</td>
<td>2.96</td>
<td>p = 0.020</td>
</tr>
<tr>
<td><strong>Nicotine levels</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ISO nicotine average inhaled per cigarette (mg)</td>
<td>1.10</td>
<td>0.76</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td>Canadian nicotine average inhaled per cigarette (mg)</td>
<td>2.46</td>
<td>2.09</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td>ISO nicotine per puff (mg)</td>
<td>0.14</td>
<td>0.10</td>
<td>p &lt; 0.001 (NP)</td>
</tr>
<tr>
<td>Canadian nicotine per puff (mg)</td>
<td>0.24</td>
<td>0.22</td>
<td>p &lt; 0.002 (NP)</td>
</tr>
<tr>
<td><strong>Carbon monoxide levels</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ISO carbon monoxide average inhaled per cigarette (mg)</td>
<td>10.99</td>
<td>7.60</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Canadian carbon monoxide average inhaled per cigarette (mg)</td>
<td>24.52</td>
<td>23.81</td>
<td>p = 0.11</td>
</tr>
<tr>
<td>Property*</td>
<td>Regular brand variants (average results – 7 variants)**</td>
<td>“Light” brand variants (average results – 4 variants)**</td>
<td>Test for difference (ANOVA unless indicated otherwise)</td>
</tr>
<tr>
<td>-----------</td>
<td>----------------------------------------------------------</td>
<td>--------------------------------------------------------</td>
<td>-----------------------------------------------------</td>
</tr>
<tr>
<td>ISO carbon monoxide per puff (mg)</td>
<td>1.40</td>
<td>0.98</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Canadian carbon monoxide per puff (mg)</td>
<td>2.39</td>
<td>2.50</td>
<td>p = 0.97 (NP)</td>
</tr>
</tbody>
</table>

Ratios of tar/CO to nicotine

| ISO tar ratio (mg tar per mg nicotine) | 11.51 | 11.34 | p = 0.011 (NP) |
| Canadian tar ratio (mg tar per mg nicotine) | 12.95 | 13.79 | p = 0.20 (NP) |
| ISO carbon monoxide ratio (mg CO per mg nicotine) | 10.03 | 10.48 | p = 0.64 (NP) |
| Canadian carbon monoxide ratio (mg CO per mg nicotine) | 9.98 | 11.68 | p = 0.004 (NP) |

* For full definitions and details of the brands, see the full online report.  
** In each of these categories there were two of the same brands which had both “regular” and “light” variants that were tested.  
NP – Non-parametric test (Kruskal Wallis test for two groups) comparing medians was used where variances were not homogeneous and analysis of variance (ANOVA) was not appropriate.

These results indicate that the “lights” smokers who block vent holes with their fingers (as to some extent mimicked by the Canadian test method), would potentially obtain higher doses of tar and CO per dose of nicotine inhaled (with the latter being statistically significant). Higher tar to nicotine ratios for a “light” versus a regular brand have been described previously for New Zealand cigarettes, but only two brands were tested.  

In conclusion the results show that the amount of tar, CO and nicotine per cigarette were fairly similar (especially when considering the Canadian test results). Also the yield of CO per dose of nicotine was significantly higher for the “lights” compared to regular brands when using the test method where vent holes were blocked. Therefore for smokers who block vent holes with their fingers (as they can do unknowingly, or as a compensatory behaviour) to achieve delivery of a specified level of nicotine (as most do), “lights” may result in as great or greater exposure to toxic constituents. Hence smoking “lights” will probably be at least as hazardous as smoking regular cigarettes, despite the impression given by the marketing of “lights” being a less toxic alternative. This provides a plausible mechanism for the epidemiological observations of no difference in risk of adverse health effects between smokers of “light” and regular cigarettes.

These findings should strengthen the hands of authorities in New Zealand such as the Commerce Commission which is currently investigating misleading tobacco product marketing. Other jurisdictions (e.g. the European Union, Australia, and Canada) have all banned misleading descriptors from cigarette packs, and there are calls for plain
packaging for tobacco products so as to eliminate the potential for all misleading marketing (eg, via colours, wording and imagery).13

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- The Health Research Council of New Zealand which has provided the core funding for the ITC Project.

Competing interests: The authors declare no competing interests.

References:

Ciproxin HC eardrops application for funding


The only ototopical antibiotic medication available in New Zealand (NZ) which to date has not been shown to be ototoxic is ciprofloxacin with hydrocortisone (Ciproxin HC). Unfortunately it is not funded in NZ and therefore significantly more expensive than commonly available and potentially ototoxic eardrops.

Last year the NZSOHNS made an application to PHARMAC for funding of ciprofloxacin with hydrocortisone eardrops (Ciproxin HC). Regrettably, PHARMAC has now declined this application.

In contrast to the NZSOHNS opinion, in their response PHARMAC maintains that there was no evidence that ciprofloxacin with hydrocortisone (Ciproxin HC) was a better or safer alternative to commonly funded antibiotic eardrops. Safety in the presence of a tympanic membrane perforation was not proven and not guaranteed by the manufacturer.

The New Zealand Society of Otolaryngology Head and Neck Surgery stands by their position statement (below). Further efforts to achieve funding for ciprofloxacin with hydrocortisone eardrops will be made.

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**STATEMENT FROM THE NEW ZEALAND SOCIETY OF OTOLARYNGOLOGY HEAD AND NECK SURGERY ON THE USE OF EAR DROPS WITH OTOTOXIC POTENTIAL IN THE PRESENCE OF TYMPANIC MEMBRANE PERFORATION, VENTILATION TUBES AND MASTOID CAVITIES WITH OPEN MIDDLE EAR**

1. It is preferable to use non ototoxic drops in the presence of tympanic membrane perforation, ventilation tubes and mastoid cavities with open middle ear.
2. If potentially ototoxic eardrops are used then they should be used only in the presence of infection and discontinued immediately after infection has resolved. The treatment should preferably be limited to a maximum of two weeks.
3. If potentially ototoxic eardrops are prescribed for the treatment of ear infection, with either a tympanic membrane perforation, ventilation tube or open middle ear/mastoid cavity, then the reason for use and the potential ototoxicity should be discussed with the patient/parent and documented (risk 1:1,000 to 1:10,000).
4. If potentially ototoxic eardrops are prescribed, then the patient should be advised to return to the doctor if vertigo, hearing loss or tinnitus develop during or soon after treatment.
5. Use of potentially ototoxic eardrops is acceptable in the presence of an intact tympanic membrane.
Response from PHARMAC

Thank you for the opportunity to respond to Drs Baber and Neef about ciprofloxacin/hydrocortisone (Ciproxin HC) eardrops.

Clinical aspects of this issue are explained by the relevant minutes of the Pharmacology and Therapeutics Advisory Committee (PTAC), available online at [http://www.pharmac.govt.nz/2008/02/01/PTAC%20minute%20-%20February%202008.pdf](http://www.pharmac.govt.nz/2008/02/01/PTAC%20minute%20-%20February%202008.pdf). The application, for the treatment of otitis media with tympanic membrane perforation and associated conditions such as chronic suppurative otitis media, was considered by PTAC at its February 2008 meeting.

In essence, PTAC has recommended that the application for funding be declined because of insufficient evidence to suggest greater efficacy than currently funded ear drops or better safety in the presence of tympanic membrane perforation. The Committee also suggested the applicants approach the manufacturer for evidence on safety of use and, if applicable, request a change in the manufacturer’s data sheet to reflect this. (PTAC had noted that the data sheet [http://www.medsafe.govt.nz/profs/datasheet/c/Ciproxinhcotic.htm](http://www.medsafe.govt.nz/profs/datasheet/c/Ciproxinhcotic.htm) indicates that safety and efficacy have not been studied in the presence of a perforated tympanic membrane, and the eardrops are therefore contraindicated in patients with known or suspected perforation, or where there is a risk of perforation).

Note: The relevant record from that meeting is as follows; sections of the minute have been withheld under Section 9 (2)(a) of the Official Information Act 1982, as indicated by “[ ]”.

Peter Moodie
Medical Director
PHARMAC

Relevant minutes of the Pharmacology and Therapeutics Advisory Committee Meeting held on 21 & 22 February 2008:

**Ciprofloxacin/Hydrocortisone (Ciproxin HC) for treatment of otitis media with a perforated tympanic membrane (TM) and associated conditions such as chronic suppurative otitis media (CSOM)**

The Committee reviewed applications from Dr J Gathercole and the New Zealand Society of Otolaryngology Head and Neck Surgery for the listing of ciprofloxacin 0.2% with hydrocortisone 1.0% ear drops (Ciproxin HC) on the Pharmaceutical Schedule for the treatment of otitis media with a perforated tympanic membrane (TM) and associated conditions such as chronic suppurative otitis media (CSOM).

The Committee noted that an earlier application for the funding of ciprofloxacin with hydrocortisone ear drops was made in 2003 and at that time the Antibiotic Subcommittee recommended declining the application in light of concern about quinolone resistance and insufficient information supplied to validate the statements made in the application.
The Committee noted that the New Zealand Society of Otolaryngology Head and Neck Surgery and Dr Gathercole were concerned about the ototoxic potential of currently funded aminoglycoside ototopical agents when used to treat middle ear infections in the presence of a non-intact tympanic membrane. Members noted that both applicants suggest ciprofloxacin with hydrocortisone ear drops are non-ototoxic and should be funded.

The Committee reviewed the evidence provided by both applicants and further evidence regarding efficacy of ototopical quinolones, ototoxicity of aminoglycoside ear drops and antibiotic resistance from the use of ototopical agents.

Members considered that evidence for efficacy of ciprofloxacin with hydrocortisone ear drops in otitis media with perforated TM was limited. The Committee reviewed one randomised, double blind, controlled trial (Couzos et. al. MJA, 2003) comparing topical 0.3% ciprofloxacin with framycetin (0.5%), gramicidin and dexamethasone (Sofradex) for CSOM in 147 children aged 1-14 years. A highly significant absolute difference of 24.6% in clinical cure (resolution of otorrhoea) was reported in favour of ciprofloxacin compared with Sofradex (76.4% vs 51.8%; P=0.009). However, only those children who had a post-treatment assessment (n=111) were included in the statistical analysis; an intention-to-treat analysis was not undertaken. There was no difference in TM perforation size or hearing.

The Committee noted further evidence regarding the efficacy of topical quinolones (without steroid) from two Cochrane reviews by Macfadyen et al., 2005 and Macfadyen et al., 2006 (Macfadyen et al., The Cochrane Library, 2007). The reviews indicated that topical quinolones were superior to systemic antibiotics and topical antiseptics but the difference between topical quinolones and non-quinolones was unclear. The reviewers considered that the studies evaluated in the reviews were of varying methodological quality and poorly reported, and while the evidence presented related to short-term clearance of aural discharge, long-term outcomes and safety were unclear.

The Committee noted the position statement from the New Zealand Society of Otolaryngology Head and Neck Surgery on the use of potentially ototoxic ear drops. Members considered that the statement is consistent with Australian and American guidelines and that there is a small risk of ototoxicity (in the order of 1:1000 to 1:10,000) from the use of ototopical aminoglycosides in situations where there is a direct pathway to the middle ear. Members also noted that the Society recommends, where possible, avoiding the use of potentially ototoxic ear drops in the presence of a non-intact TM.

The Committee reviewed further evidence regarding ototoxicity of aminoglycoside ear drops from a review by Roland et al (Otolaryngology – Head and Neck Surgery, 2004) and Matz et al (Otolaryngology – Head and Neck Surgery, 2004) and noted that the evidence was largely from animal studies with some case reports in humans. Members noted that there are anatomical differences between the human ear and experimental animal ear and, as such, data needs to be extrapolated with caution. Members also considered that ototoxicity in humans may be underappreciated because the earliest and most severe auditory manifestations may occur at higher frequencies which are usually not tested in humans; the vestibular manifestations, if unilateral, may be subtle; and some damage may be misattributed to the condition.

The Committee also noted an opinion from [ ], consultant otolaryngologist regarding ototoxicity of ototopical ear drops. The Committee noted [ ] views on the use of ototoxic ears drops and [ ] concern around the safety of quinolone ear drops and antibiotic resistance. The Committee noted that there was no evidence provided in support of [ ] view. The Committee agreed that there was insufficient evidence regarding the safety of quinolone ear drops when used in the presence of a non-intact TM.

The Committee noted that quinolone resistance has been raised as a concern because of increasing quinolone use. The Committee reviewed evidence from a review by Weber et al (Otolaryngology – Head and Neck Surgery, 2004) on development of resistance with the use of ototopical antibiotics. The Committee noted that there was grade B evidence to indicate that no significant antibiotic resistance develops from use of ototopical antibiotics. Members noted further support for this from a review article by J Kline (Amer J Managed Care, 2002), which recommends using ototopical antibiotics rather than systemic antibiotics for treating middle ear infections to reduce the risk of developing bacterial resistance. The Committee also noted the opinion of Dr Mark Thomas, a member of the Anti-infective Subcommittee, who considered that the use of ciprofloxacin ear drops would provide relatively minor selection pressure for emergence of resistant organisms.
The Committee acknowledged the New Zealand Society of Otolaryngology Head and Neck Surgery’s concern around the medico-legal risk from the use of potentially ototoxic ear drops in treatment of otitis media in the presence of a non-intact TM. However the Committee noted that potentially ototoxic ear drops had been used, off-label, to treat middle ear infections in the presence of a non-intact TM for many years and considered that the risk of ototoxicity from aminoglycoside ear drops was low.

The Committee considered that there may be an unmet need for a safer alternative in certain populations such as low socio-economic, Maori, and Pacific Island people in whom chronic middle ear conditions are more prevalent.

However, the Committee considered that there was insufficient evidence to suggest ciprofloxacin with hydrocortisone ear drop were a safer alternative to use in the presence of a non-intact TM. The Committee noted that the manufacturer of ciprofloxacin with hydrocortisone ear drops states that the safety and efficacy of ciprofloxacin with hydrocortisone ear drops have not been studied in the presence of a perforated tympanic membrane and ciprofloxacin with hydrocortisone ear drops are, therefore, contraindicated in patients with known or suspected perforation, or where there is a risk of perforation of the tympanic membrane.

The Committee **recommended** that the application for funding of ciprofloxacin 0.2% with hydrocortisone 1.0% ear drops (Ciproxin HC) be declined because of insufficient evidence to suggest that they were more efficacious than currently funded ear drops or were safer to use in the presence of a non-intact TM.

The Committee also suggested that the applicants approach the manufacturer for evidence on safety of using ciprofloxacin with hydrocortisone ear drops to treat otitis media in the presence of a TM perforation and if applicable, a change in the manufacturer’s data sheet recommendation to reflect this. **The Decision Criteria relevant to this recommendation are:** (i) The health needs of all eligible people within New Zealand; (ii) The particular health needs of Maori and Pacific peoples; (iii) The availability and suitability of existing medicines, therapeutic medical devices and related products and related things; (iv) The clinical benefits and risks of pharmaceuticals; (vii) The direct cost to health service users, and (viii) The Government’s priorities for health funding, as set out in any objectives notified by the Crown to PHARMAC, or in PHARMAC’s Funding Agreement, or elsewhere.
High Court Appeal – Health Practitioners Disciplinary Tribunal (Med05/15D)

Previous Journal Notice
A notice about this Health Practitioners Disciplinary Tribunal (the Tribunal) hearing was published in NZMJ Vol.120 No.1265.

Tribunal Finding & Penalty
The Tribunal found Dr Harriett Rosalind Elles Martin, medical practitioner, guilty of professional misconduct.

The Tribunal ordered the practitioner:

- be censured
- pay a total fine of $15,000
- pay costs of $20,000

Appeal
The practitioner appealed to the High Court as to part of the substantive finding and as to the penalty ordered by the Tribunal.

The High Court dismissed the appeal on the substantive finding, upholding the Tribunal’s finding of professional misconduct. The court modified the penalty order and reduced the fine.

The fine of $5,000 in relation to part 1 and part 2 of the charge was reduced to $3,000. The fine of $10,000 in relation to part 3 of the charge was reduced to $7,000 (Martin v Director of Proceedings (High Court, Auckland, CIV-2006-404-005706, 2 July 2008, Courtney J)).

The full Tribunal decisions relating to the case can be found on the Tribunal web site at www.hpdt.org.nz Reference No: Med05/15
Introducing mental health nursing: a consumer-oriented approach

Brenda Happell, Leanne Cowin, Cath Roper, Kim Foster, Rose McMaster.

This is a 397-page soft-covered text which aims to introduce undergraduate nursing students to mental health nursing practice. It takes an introductory approach which would be helpful for nursing students in both general and mental health settings.

This is an innovative text which takes a consumer-oriented approach to nursing practice rather than a biomedical approach. The authors are experienced mental health nurses and a consumer all currently in education. The text is structured around concepts rather than the traditional approach of structuring around psychiatric diagnosis in similar texts for beginning practice. While a biomedical approach is included, a social model of health (with an emphasis on consumers’ individual experience of mental health problems) is the conceptual model for the text.

The chapters all include case studies and critical thinking exercises which make use of both printed and online resources. These exercises are thought-provoking and well integrated into the overall focus of each chapter. A comprehensive range of subjects are addressed including the environment within which nurses practice, nursing roles, defining, understanding and treating mental health problems, and nursing research.

The main strength of the text is its consumer focus and very good critical thinking activities. The main weakness of the text is that it is specifically pitched at the Australian mental health context with mostly Australian resources identified and included. As a result of this it probably is not as relevant to New Zealand undergraduate nursing students.

In conclusion it is an innovative easy-to-read text that includes many useful experiential activities integrated into the content. It has a strong clinical and consumer orientation but is very Australian in its focus.

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Occupational and physical therapy for children with rheumatic diseases: a clinical handbook


This book provides a quick and easy to use reference for Doctors and allied health professionals caring for children with rheumatic diseases. The book is divided into six sections. Sections 1–3 provide a brief clinical overview of paediatric rheumatic diseases, their assessment and management. Section 4 provides detailed information on occupational and physiotherapy assessments and interventions while section 5 provides an overview of the medical, nursing and social work management strategies. Finally, in Section 6, information sheets and resources for patients, their families, and schools are provided.

There are detailed practical guidelines for assessing and managing pain, sleep, fatigue, activities of daily living, and the school environment as well as for assessing function and managing problems within each specific joint region. The handouts, which can be reproduced as well as being available online, provide useful guidance on such practical matters as choosing a school backpack and shoes and participating in sporting activities. Such information is invaluable to patients and their families and frequently poorly addressed by health practitioners. This book is clearly laid out and easy to use. Most information is presented in easy to follow tables with excellent supporting clinical photographs.

Management of paediatric rheumatological conditions has undergone major transformation in the last decade including early more intensive use of intra-articular steroids, methotrexate and biological agents such as anti-tumour necrosis factor therapies. A multidisciplinary team approach is critical in managing children with rheumatic diseases.

In New Zealand there is limited access to paediatric rheumatologists as well as nurses, occupational therapists, and physiotherapists with specific training for dealing with children with rheumatic diseases. As such, this book provides an excellent resource for general practitioners, paediatricians, and allied health professionals.

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