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This Issue in the Journal

Gender inequality in New Zealand life expectancy: decomposition by age and cause
Peter Sandiford

That men have shorter lives than women is well known. What is less well known is that the difference in life expectancy between men and women peaked in the mid-1970s and has since been declining, but only in non-Maori. In this study the specific causes of death that account for this difference in life expectancy are determined. It was found that men die more frequently than women of causes that account for over two-thirds of all deaths. However, official health policy does not appear to consider sex differences in life expectancy to be inequitable and only recently has men’s health begun to attract the sort of attention that it is receiving in other developed countries.

Does mortality vary between Pacific groups in New Zealand? Estimating Samoan, Cook Island Māori, Tongan, and Niuean mortality rates using hierarchical Bayesian modelling
Tony Blakely, Ken Richardson, Jim Young, Paul Callister, Robert Didham

Pacific mortality rates are traditionally presented for all Pacific people combined, yet there is likely differences between separate Pacific ethnic groups. We used New Zealand Census-Mortality Study (NZCMS) data for 2001–2004, and compared all causes of death in other Pacific groups compared to a baseline rate (Samoan): 1.21 times higher for Cook Island Maori; 0.93 for Tongan; and 1.07 for Niuean. Cardiovascular disease (CVD) death rates showed even greater differences: 1.66 times higher for Cook Island Māori; 1.11 for Niuean; and 0.86 for Tongan. Therefore Cook Island Māori have relatively high rates of heart disease when compared to other Pacific peoples. Future health research and policy should take these differences into consideration when studying the health status of Pacific peoples.

Mum’s the word: factors that influenced young adults’ participation in the New Zealand Meningococcal B immunisation programme
Marian Bland, Geraldine M Clear, Adrianna Grogan, Kath Hoare, Jan Waldock

This research report relates to the recent MeNZB vaccination campaign in New Zealand. This vaccine was developed in response to the Meningococcal B epidemic. The research explores how 11 young adults (aged 16–19 years) made the decision about whether or not to have the vaccination, and the factors that influenced their decision. Convincing the participants of the need for, and safety of, the vaccine were significant factors in their decision.
Sero-prevalence of leptospirosis in workers at a New Zealand slaughterhouse
Jackie Benschop, Cord Heuer, Patricia Jaros, Julie Collins-Emerson, Anne Midwinter, Peter Wilson

Leptospirosis is an endemic bacterial disease of livestock in New Zealand and our most important occupationally-acquired infectious disease in humans. Meatworkers and farmers are at most risk and major underreporting is suspected. In 2008, 121 cases were notified with 45% of these so ill they required hospitalisation. The same serovars occur in livestock and humans, indicating transmission from animals to humans. Since vaccination of all livestock is not practiced at the moment, better knowledge of risk factors for transmission is crucial to prevent further infection in meat workers in the New Zealand meat industry. Our 2008 pilot cross-sectional study found a 9.5% seroprevalence amongst 242 healthy meat workers at a North Island abattoir. This seroprevalence was higher than that reported from previous studies in the 1980s, and despite the increased use of personal protective equipment, and that workplace exposure was predominantly to sheep. This suggests significant workplace exposure to leptospirosis in this population of workers. Further studies are planned to determine whether the prevalence of leptospirosis is similar in other slaughterhouses, and to determine the link between sero-prevalence, which indicates past exposure, and incidence, that indicates recent exposure. These will assist in developing a better understanding of risk factors important for the reduction of exposure of this occupationally acquired disease.

Acute gastrointestinal illness in New Zealand: information from a survey of community and hospital laboratories
Rob Lake, Nicola King, Kerry Sexton, Philippa Bridgewater, Donald Campbell

Acute gastrointestinal illness causes diarrhoea, vomiting and other symptoms, and is often caused by harmful organisms in food and water. This survey was part of a larger study to estimate the number of people who have this illness in New Zealand each year. The survey asked community and hospital laboratories how many faecal samples they received in 2005, and what testing was done. Overall, it was estimated that approximately 250,000 samples were received, and these came from the proportion of approximately 800,000 ill people who visited their GP about the illness and were asked to provide a sample.
Sudden infant death and co-sleeping: stronger warning needed

Edwin A Mitchell

Dr McIntosh and colleagues, in this issue of the NZMJ, remind us that more than 50% of sudden infant deaths occur while an infant is sleeping with parents in the same bed—What is the mechanism of sudden infant deaths associated with co-sleeping?; http://www.nzma.org.nz/journal/122-1307/3914)—and they speculate on the possible mechanisms for this association.

The marked decline in SIDS in the early 1990s was due to the recommendation not to place infants to sleep on their fronts. The gradual decline in SIDS mortality in the late 1990s and early 2000s has been attributed to the change from side to supine sleeping position. However, there are still 50–60 cases of SIDS a year in New Zealand. Each death is a tragedy and they are potentially avoidable.

As most infants now sleep supine, there will be little further gain from focussing on infant sleeping position. Maternal smoking, especially in pregnancy, is the next major risk factor for SIDS. The recommendation to stop smoking is an important public health message but is very resistant to change.

What is the risk from bed sharing?—After maternal smoking, bed sharing or co-sleeping is probably the next major risk for SIDS. Our group convincingly showed that it was a risk factor for SIDS in 1992. The epidemiological evidence is summarised in Table 1.

Table 1. Summary of the risk of SIDS with bed sharing

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>The risk of SIDS with bed sharing is high when the mother smokes or smoked in pregnancy</td>
<td>4,7</td>
</tr>
<tr>
<td>The risk of SIDS with bed sharing is higher in younger infants</td>
<td>5,8</td>
</tr>
<tr>
<td>There is a small increased risk of SIDS from bed sharing in infants of non-smoking mothers less than 3 months of age</td>
<td>6</td>
</tr>
<tr>
<td>Bed sharing infants who are placed back in their cot are not at increased risk of SIDS</td>
<td>6</td>
</tr>
<tr>
<td>The longer the infant bed shares during the night the greater the risk of SIDS</td>
<td>4</td>
</tr>
<tr>
<td>Infants who bed share with mothers who have drunk 2 or more units of alcohol are at risk of death</td>
<td>9</td>
</tr>
<tr>
<td>Co-sleeping on a couch or sofa is associated with a high risk of SIDS in some studies, but not others</td>
<td>6,9</td>
</tr>
<tr>
<td>Infants bed sharing with older siblings are at increased risk, although there are no data either way for twins sharing (sometimes referred to as co-bedding)</td>
<td></td>
</tr>
</tbody>
</table>
What is the down side of advising that infants should not bed share?—The major concern is that breastfeeding will be reduced.\textsuperscript{10} Bed sharing is associated with a longer duration of breastfeeding, but the effect is small.\textsuperscript{11} It is also argued that it improves mother-infant bonding. One might speculate that improved bonding from bed sharing should result in less abuse of infants, but ecological evidence does not support this contention. Communities with high bed sharing rates do not have lower child abuse rates.

What is the mechanism of sudden infant death with co-sleeping?—Several mechanisms have been proposed; these include airway obstruction, thermal stress, and head covering. Dr McIntosh and colleagues postulate that the mechanism is subtle airway obstruction produced by the jaw being displaced backwards and occluding the upper airways.

This is certainly plausible and is more likely than overt “overlaying”; that is unlikely except in the rare situation that the parent is so intoxicated that they are unaware that the infant is beneath them. However, thermal stress and head covering are alternative mechanisms. Indeed head covering is associated with a very high risk of SIDS even in infants sleeping alone.\textsuperscript{12} Covering of the infants head occurs more frequently when bed sharing with a parent than sleeping separately.\textsuperscript{13} Airway obstruction, thermal stress or head covering might interact with arousal defects, such as can be produced by \textit{in utero} smoke exposure, or by gene polymorphisms.

What should we recommend?—The Ministry of Health states\textsuperscript{14}:

“Co-sleeping (a parent who sleeps with their baby in bed) is dangerous when the:

- Baby’s mother has smoked during pregnancy
- Adult in bed with the baby has been drinking, or taking drugs or medicines that might reduce their awareness of the baby
- Co-sleeping adult is excessively tired.

There is also a small increase in the risk of SUDI from co-sleeping for babies less than 3 months old, whether or not the mother smoked during pregnancy.”

It is important to realise that no group either of infants or parents has been identified where bed sharing is associated with a reduced risk of SIDS. The current advice has been given so quietly it has not been heard. Indeed a recent survey found that only 46\% identified bed sharing as a risk for SIDS, compared with 84\% for sleep position and 73\% for smoking.\textsuperscript{15}
The Wellington coroner, Judge Gary Evans, has recommended to the Director General of Health:

1. That the public health advice in relation to safe infant care practices and safe sleeping environment be strengthened and broadened so as to make it clear that:
   i. Bed sharing by adults and siblings with infants under 6 months exposes the infant to the risk of death and should be avoided.
   ii. The safest place for babies to sleep for the first six months of life is in a cot beside the parental bed.

2. That steps be taken by the Ministry (of Health) to ensure that the same advice is given by public health educators and health professionals in those public health sectors over which the Ministry has influence.

Parents have the right to know what risks they are exposing their infants to, and health professionals have the responsibility to provide evidence based advice. The issue now is how best to give this message. The ‘softly-softly’ approach has not worked.

At the risk of alienating lactation consultants, Māori, and others, I believe a different, stronger and no-nonsense approach should be taken.

Competing interests: None known.

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Gender inequality in New Zealand life expectancy: decomposition by age and cause

Peter Sandiford

Abstract

**Aim** To quantify gender inequality in life expectancy at birth (LEB) in New Zealand and the contribution to it made by different age groups and causes of death. To examine the response of the health sector.

**Method** Determination of the trend in sex differences in LEB. Multiple decrement decomposition of LEB differences into components ages and causes. Review of the gender equity policies and priorities of New Zealand’s main health sector stakeholders.

**Results** A difference between the sexes in LEB of 4.7 years for Māori and 4.0 years for non-Māori, reverses the historically lower gender disparity among the Māori. Over half of the sex difference in LEB is accounted for by heart disease and all types of cancer and almost a quarter by accidents and suicide but male survival disadvantage is evident in many other causes of death. The health sector is beginning to acknowledge the survival disadvantage of men as inequitable, and reducing disparity as a legitimate goal for health policy.

**Conclusion** Although gender inequality in LEB is declining among the non-Māori it remains high among the Māori. Smoking habits may explain some of the difference in LEB but policies must also address the causes of sex differences in accidental death and suicide.

There is rightly considerable concern in New Zealand with ethnic and socioeconomic inequalities in health, but oddly little has been said about gender inequalities in health, despite the well-known fact that men live significantly shorter lives than women.

This paper presents the long-term trend in these inequalities and quantifies the contribution made by different causes for men’s shorter life expectancy. It looks at how health system stakeholders have responded to these inequalities in terms of their policies and health programmes. Whilst noting recent interest and initiatives in the field of men’s health, it questions whether there was a valid reason for past failure to act on these, in the face of longstanding inferior male health outcomes.

**Methods**

Life tables for 1934 through 2007 were obtained from Statistics New Zealand. Age-cause-specific death rates were obtained from the WHO mortality database, which receives its data from the Ministry of Health death registration system. The most recent data available from this source was for the years 2000–2002 and so the corresponding life table was used for this analysis. Mortality rates were standardised to the 2001 total population as published in WHO Mortality Database. Differences in life expectancy between men and women for 2000–2002 were decomposed into age and cause of death components using Arriaga’s method as described by Preston et al. This consists of first
calculating the contribution to differences in life expectancy between males and females of all-cause differences in the various age groups and then further decomposing these into the various causes of death of interest. The contribution of age group $x$ to $x + n$ to the overall gender difference in life expectancy, $n \Delta_x$, is given by:

$$n \Delta_x = \frac{l_{x \text{ male}}}{l_{0 \text{ male}}} \cdot \left( \frac{n L_{x \text{ female}}}{l_{x \text{ female}}} - \frac{n L_{x \text{ male}}}{l_{x \text{ male}}} \right) + \frac{T_{x+n \text{ female}}}{l_{x \text{ female}}} \cdot \left( \frac{l_{x \text{ male}}}{l_{x \text{ male}}} - \frac{l_{x+n \text{ male}}}{l_{x+n \text{ female}}} \right)$$

where $l_{x}$, $L_{x}$, and $T_{x}$ are conventional life table functions. For calculating the contribution of the open-ended age-group $\Delta_{\infty}$, the following formula is used:

$$\Delta_{\infty} = \frac{l_{x \text{ male}}}{l_{0 \text{ male}}} \cdot \left( \frac{T_{x \text{ female}}}{l_{x \text{ female}}} - \frac{T_{x \text{ male}}}{l_{x \text{ male}}} \right)$$

Now the specific contribution to the difference in life expectancy between males and females of cause $i$ in age group $x$ to $x + n$ under the assumption of a constant distribution of deaths within each age-group of the population is estimated by:

$$n \Delta_{x}^i = \frac{n R_{x i \text{ female}}}{n m_{x \text{ female}}} - \frac{n R_{x i \text{ male}}}{n m_{x \text{ male}}}$$

where:

- $R_{x i}$ is the proportion of all deaths in age group $x$ to $x + n$ caused by $i$; and
- $m_{x}$ is the all-cause mortality rate in age group $x$ to $x + n$.

The response by health sector stakeholders to gender inequalities in health was assessed by referring to their websites for policies and priorities, downloading where necessary the relevant documents, and by conducting word searches within the sites for references to equity, gender and inequality.

Results

Sex differences in survival—Male life expectancy in New Zealand in 2000–2002 was 4.8 years less than female life expectancy. This disparity represents a decline from the 1970s when it was over 6 years but it has really only just returned to the degree of inequality that was last seen in about 1950 (Figure 1). Moreover, gender inequality in Māori survival remains close to historical highs, and for the first time has surpassed that in the non-Māori population.

Decomposition of the sex difference in 2000–2002 life expectancy by age revealed that although women have lower mortality at all ages, approximately half of the female survival advantage occurs at ages 65–84. Among the Māori, though, it is more evenly spread with this age group responsible for just 31%. Interestingly, only 2.3% of the difference can be attributed to higher male infant mortality and only 3.3% to all ages under 15 years. The overall low infant and child mortality in both sexes is mainly responsible for this small contribution, because male babies still have a 20–30% higher risk of dying than female babies.
Figure 1. Trend in the gender difference in life expectancy for Māori and non-Māori

Table 1. Decomposition of the gender differences in 2000–2002 life expectancy at birth into component causes of death

<table>
<thead>
<tr>
<th>Cause of death (ICD10 codes)</th>
<th>Standardised mortality rate (deaths/10,000/year)</th>
<th>Effect on the difference in life expectancy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>Ischaemic heart disease, diseases of pulmonary circulation &amp; other forms of heart disease (I20-I24, I25-51)</td>
<td>23.8</td>
<td>15.7</td>
</tr>
<tr>
<td>All malignant neoplasms except prostate, lung, breast and uterine (including cervix) (C00-C32, C67-C96)</td>
<td>15.7</td>
<td>10.4</td>
</tr>
<tr>
<td>Cancer of the prostate (C61)</td>
<td>3.7</td>
<td>0</td>
</tr>
<tr>
<td>All accidents (V01-X59, Y40-Y86, Y88)</td>
<td>3.8</td>
<td>1.8</td>
</tr>
<tr>
<td>Suicide, self-inflicted injury and other external causes (X60-84, Y10-36, Y87, Y89)</td>
<td>2.1</td>
<td>0.6</td>
</tr>
<tr>
<td>Cancer of the trachea, bronchus and lung (C33,C34)</td>
<td>5.0</td>
<td>2.7</td>
</tr>
<tr>
<td>Chronic bronchitis, emphysema and asthma (J40-J46)</td>
<td>5.7</td>
<td>3.4</td>
</tr>
<tr>
<td>Arterial embolism and thrombosis (I71-I78)</td>
<td>1.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Diabetes (E10-E14)</td>
<td>2.5</td>
<td>1.7</td>
</tr>
<tr>
<td>Cervical cancer (C53)</td>
<td>0</td>
<td>0.3</td>
</tr>
<tr>
<td>Uterine cancer (not cervical) (C45-C55)</td>
<td>0</td>
<td>0.4</td>
</tr>
<tr>
<td>Cerebrovascular disease (I60-I69)</td>
<td>7.4</td>
<td>6.6</td>
</tr>
<tr>
<td>Breast cancer (C50)</td>
<td>&lt;0.1</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Analysis of the contribution of specific causes of death to the gender difference in life expectancy is summarised in Table 1. The causes listed, including female cancers,
when taken in combination explain 95% of the sex difference in life expectancy (4.52 of 4.77 years).

Figure 2. Breakdown of the cancer contribution of the sex difference in life expectancy by site of neoplasm

The conditions listed in the table are responsible for 81% of all deaths in New Zealand from 2000–2002. Those that adversely affect male survival comprise 69% of all deaths in New Zealand. It should be noted that male mortality rates are significantly higher than female mortality rates for many other causes of death not listed in this table because their contribution to the total life expectancy differential was minimal.

It could be argued that if sex-specific diseases such as genital organ neoplasms, obstetric causes and even breast cancer (although this also occurs in men) had been excluded from the analysis one would have had a fairer or more accurate indication of the effects of gender as opposed to sex on inequality in life expectancy. However, this results in an even greater proportion of the net difference in life expectancy between men and women being accounted for than when the sex-specific causes are included (4.64 out of 4.77 years or 97%).

Figure 2 shows the breakdown by type of cancer of the 1.49-year difference in life expectancy attributable to cancers other than prostate, lung, breast, uterus and cervix. It is evident from this figure that male mortality disadvantage is spread across a wide range of malignancies. Motor vehicle accidents made up almost half of the difference in LEB due to accidents. Drowning comprised 10% of this grouping.
The health sector response to gender inequalities in survival—The survival disadvantage of males over females is well known. It is a legitimate question, therefore, to ask what actions, if any, the Ministry of Health and other key health sector actors in New Zealand have taken to reduce these inequalities. Ministry of Health policy on reducing health inequalities is articulated in the New Zealand Health Strategy.

Reducing social inequalities in health is clearly an important goal for the government. There is a strong emphasis on reducing inequalities between ethnic groups, and to a lesser extent on income inequalities in health outcomes, but the New Zealand Health Strategy makes no specific mention of sex/gender as an element of these inequalities. The Ministry of Health published a monograph on inequalities in health in 2002. While this acknowledged the lower life expectancy of men it went little further than to declare that “our knowledge in this area is relatively underdeveloped”.

Perhaps for that reason, in November 2004 the Public Health Advisory Committee commissioned a literature review on men’s health. While noting that health outcomes are poorer for men than for women, it suggested that more in-depth research was needed to provide a foundation for effective strategies to improve men’s health.

In June 2008 the then Associate Minister of Health announced $3 million of funding for a new men’s health programme. Its focus was on raising their awareness of health and encouraging them to make more use of health care. A Men’s Health Innovations Fund was established, social marketing campaigns were developed, and efforts were made to support workplace-based clinics. However, if the Ministry of Health website is any indication of current government concern for men’s health, then the absence of an updates or news since 2008 would suggest that it is no longer of significant policy interest and there is apparently no intention to continue or revive the Innovation Fund.

The aforementioned call for more in-depth research on men’s health is not yet reflected in the Health Research Council’s priorities. Its recent consultation document does suggest a “Research for Health Equity” Health Impact Target but the male population is not one of its priority groups (these are Māori, Pacific peoples, children and youth, older adults, and people with disability). On the other hand, the HRC does identify as priorities some of the specific conditions with significant sex disparities in mortality rates, such as cardiovascular disease, cancer and COPD, without making reference to the gender inequality dimension.

The Accident Compensation Commission, though operating in a field with substantial gender inequality, does not acknowledge this in its Strategic Plan nor does it have as a goal the reduction of gender inequality in injury incidence, mortality or disability resulting from accidents. In contrast, it does acknowledge ethnic inequalities in claim rates, and seeks to “remove disparities in rehabilitation outcomes” for Māori, Pacific and Asian peoples.

Some awareness of the significance of gender inequalities in health is emerging from charities and non-government organisations. Suicide Prevention Information New Zealand has recently highlighted the need for a greater focus on male suicide prevention, the Cancer Society of New Zealand made men’s health a major focus for their work in 2008, and Age Concern has organised Men’s Health Week activities,
emphasising the “unnecessarily low” life expectancy of men. The Public Health Association of New Zealand, however, does not mention gender inequalities in health in its policy on reducing health inequalities and a recent issue of its publication PHA News that looked specifically at health inequalities barely mentioned gender.

Discussion

Gender inequality in LEB may be on the decline for the population as a whole, but the rising trend among the Māori is of some concern. This may simply be a delayed trend of the same pattern that increased gender inequality among the non-Māori until the mid-1970s and which is now producing what appears to be a sustained decline. Until we know more about the causes of that rise and fall, and likewise for the trend in Māori gender inequality, one cannot be confident that either the Māori level will start to fall, or that non-Māori men will continue to catch up to non-Māori women in their life expectancy.

It would also be of interest to examine how gender inequality is changing among different socioeconomic groups. Work by Tobias and Cheung on 1995–1997 data showed that gender inequality increased with socioeconomic deprivation from 4.0 years in decile 1 to 6.5 years in decile 10 in the population as a whole, but not for the Māori. As Māori gender inequality in LEB has increased, a corresponding socioeconomic gradient may have emerged.

It is informative to explore ‘what if’ scenarios of different strategies to reduce gender inequalities. For example, age-specific reductions in all-cause mortality rates have a different effect on life expectancy for women than for men. Similarly, equal reductions in cause-specific mortality rates for both sexes will extend life expectancy for one more than the other if there is a difference in their respective mortality rates at the outset (e.g. for ischaemic heart disease). Alternatively, one might consider the potential impact on gender equity of targeted interventions that reduce mortality for males at a greater rate than for the females.

Apart from prostate and lung cancer (and in the opposite direction breast cancer), the contribution of other cancers to the difference in life expectancy was large and relatively evenly spread, including both gastrointestinal and non-gastrointestinal cancers. This has been noted previously and a large part, but not all of the difference, was attributed to differences between the sexes in smoking and possibly alcohol consumption. Indeed, changes in smoking prevalence have probably made a major contribution to the decline in sex differences in LEB, as it has in the United States.

Given that sex differences in life expectancy are well documented and that their causes have been the subject of academic enquiry for many years (see for example Verbrugge), despite a relative paucity of studies specifically on men’s health, the belated and so far rather feeble health sector response to these inequalities seems bizarre, especially given the high level of concern over ethnic and income differences in health outcomes.

It is possible that policymakers did not consider gender differences in life expectancy to be inequitable or they thought there was nothing that can be done about them. This is quite plausible, given the commonly held belief that the shorter lifespan of men is “an unremarkable consequence of the difference between the sexes.” However, the
changes in magnitude, and variation between countries of the ‘gender gap’ in survival, not to mention the importance of ‘avoidable’ causes of death as components of the difference, should firmly dispel any notion that the male disadvantage in life expectancy is attributable solely or even mainly to purely biological differences between the sexes. Even if it were, though, why would that disqualify sex differences in life expectancy from being the focus of efforts to reduce inequalities in health?

Another possibility, advanced by Goodyear-Smith, is that policymakers view women as socially and economically disadvantaged relative to men, and feel a need to ‘redress the balance’ by prioritising services for women. According to this view, to meet women’s health needs services need to be made more appropriate to their specific requirements, while the corresponding view for responding to men’s health needs tends to be that men themselves must change to become less ‘masculinist’, on the grounds that this is what makes them engage in behaviours and lifestyles that place them at increased risk.9

A third explanation that has been given for the low priority awarded to men’s health is that men have lower overall morbidity, despite their shorter life expectancy, making it unclear which sex has worse health, despite the obvious gender gap in mortality. It is beyond the scope of this paper to examine this question in detail but it should be noted that although women undeniably use primary care services more frequently than men, data from the 2006 Disability Survey show that disability rates for men were about 10% higher than in women. Given that the male disadvantage predominates in younger ages, the overall difference in Disability Adjusted Life Years lost is probably significantly greater than in prevalence rates.

With other diseases the picture is indeed mixed. Data from the 2002/03 New Zealand Health Survey show higher self-reported prevalence rates of heart disease, stroke, and diabetes in men than in women, while women had a higher self-reported prevalence of cancer, COPD, osteoporosis and arthritis (although many of these differences were not statistically significant).11 These findings are intriguing since men undoubtedly have higher age-standardised mortality rates for all-cause cancer, COPD and asthma.

Case and Paxton12 tackled this same paradox of higher male mortality in conjunction with higher female self-reported morbidity in the United States. They found that higher rates of self-reported ill-health among women were due to differences between men and women in the mix of chronic conditions that they were suffering from while men were more likely to die and be hospitalised from certain smoking-related illnesses than women suffering from the same conditions.

Attitudes to male disadvantage in life expectancy and health generally may be changing, not just in New Zealand, but also worldwide.13,14 The Australian Medical Association issued a position statement on Men’s Health in 2005 and the Royal Australian College of General Practice established a policy on men’s health in 2006. The Australian Government is developing a National Men’s Health Policy and has begun by publishing a paper outlining its justification for this decision. In Europe, a growing men’s health movement, including organisations such as the European Men’s Health Forum is lobbying for recognition in policy and practice of the special health needs of men. In the UK, men’s health has received a boost from the introduction of new gender equality legislation and the Irish Government made a commitment to
produce a policy for Men’s Health and Health Promotion in its 2001 Health Strategy (although it seems that this has yet to be fulfilled).

The decomposition presented in this paper may help guide effective policies to reduce the gender gap in survival, and the techniques used can be repeated to monitor their success with specific causes of death.

Competing interests: None known.

Disclaimer: The views expressed in this publication are those of the author and do not in any way necessarily reflect the views of the Waitemata District Health Board.

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References and endnotes:

1. A distinction is conventionally made between ‘gender’ and ‘sex’ with the former relating to the roles played by men and women and the latter referring more to their biological differences. In this paper I maintain that distinction, but use the term ‘gender inequality’ to refer also to the difference in health outcomes between men and women, and in particular, the difference in life expectancy.


Does mortality vary between Pacific groups in New Zealand? Estimating Samoan, Cook Island Māori, Tongan, and Niuean mortality rates using hierarchical Bayesian modelling

Tony Blakely, Ken Richardson, Jim Young, Paul Callister, Robert Didham

Abstract

Background Pacific mortality rates are traditionally presented for all Pacific people combined, yet there is likely heterogeneity between separate Pacific ethnic groups. We aimed to determine mortality rates for Samoan, Cook Island Māori, Tongan, and Niuean ethnic groups (living in New Zealand).

Methods We used New Zealand Census-Mortality Study (NZCMS) data for 2001–04, for 380,000 person years of follow-up of 0–74 year olds in the 2001-04 cohort for which there was complete data on sex, age, ethnicity (total counts), natality, and household income. Given sparse data, we used hierarchical Bayesian (HB) regression modelling, with: a prior covariate structure specified for sex, age, natality (New Zealand/Overseas born), and household income; and smoothing of rates using shrinkage. The posterior mortality rate estimates were then directly standardised.

Results Standardising for sex, age, income, and natality, all-cause mortality rate ratios compared to Samoan were: 1.21 (95% credibility interval 1.05 to 1.42) for Cook Island Māori; 0.93 (0.77 to 1.10) for Tongan; and 1.07 (0.88 to 1.29) for Niuean. Cardiovascular disease (CVD) mortality rate ratios showed greater heterogeneity: 1.66 (1.26 to 2.13) for Cook Island Māori; 1.11 (0.72 to 1.58) for Niuean; and 0.86 (0.58 to 1.20) for Tongan. Results were little different standardising for just sex and age. We conducted a range of sensitivity analyses about a plausible range of (differential) return migration by Pacific people when terminally ill, and a plausible range of census undercounting of Pacific people. Our findings, in particular the elevated CVD mortality among Cook Island Māori, appeared robust.

Conclusions To our knowledge, this project is the first time in New Zealand that clear (and marked in the case of CVD) differences in mortality have been demonstrated between different Pacific ethnic groups. Future health research and policy should, wherever possible and practicable, evaluate and incorporate heterogeneity of health status among Pacific people.

Pacific people have mortality rates intermediary between Europeans and Māori when considered as a single group. But the Pacific population is heterogeneous, and health status (including mortality rates) is likely to vary between specific Pacific groups (e.g. Samoan, Tongan, Cook Island Māori, and Niuean).

First, there are demographic and migration variations between the Pacific ethnic groups that might generate variations in health status. For example, migration histories to New Zealand vary. Cook Islanders and Niueans have been New Zealand
citizens since 1901 and Tokelauans since 1916. Migration of these ethnic groups to New Zealand thus has a longer history and may have been less health-selective than other Pacific ethnic groups. With the increasing need for unskilled labour in the 1960s and 1970s, immigration from the Pacific grew. A Treaty of Friendship was signed with the Samoan Government in 1962, and the Western Samoan Quota scheme was established to facilitate migration from Samoa.

In 1945, the Pacific population in New Zealand was just over 2000 people, Samoans being the largest group. In recent times, the Pacific population was 202,233 in 1996, rising to 231,801 in 2001, and increasing further to 265,974 in March 2006.\(^2\) The Cook Island Māori and Niuean population in New Zealand are now substantially higher than their respective ‘home’ population (4 and 14 times greater, respectively, at the 2006 Census) and more than 70% are New Zealand-born. In contrast, the ‘home’ Samoan population is larger than the New Zealand population, and 60% of those residing in New Zealand are born New Zealand. The highest overseas-born proportion is seen amongst Tongans.

Table 1 shows a range of sociodemographic characteristics for the four largest Pacific ethnic groups, Samoan, Cook Island Māori, Tongan, and Niuean. Cook Island Māori and Niuean are least likely to self-identify with a single ethnic group. Cook Island Māori have the lowest rate of formal qualifications, and Tongan the lowest median income.

Second, health-related risk factors vary between Pacific ethnic groups. At the 2006 Census, 38% of the Cook Island Māori population smoked, compared to 28% and 29% for Samoan and Tongan (Table 1). Sundborn et al (2008) have recently published estimates of a range of risk factors for Samoan, Tongan, Niuean, and Cook Island Māori participating in the Diabetes Heart and Health Study.\(^3\)

Being a workforce survey, the population was considerably older than the census population. The smoking prevalence also differed from census data, raising concerns about representativeness of the Pacific population. Nevertheless, the study found substantial variation in health risk factors between different Pacific groups.

The aim of this paper, therefore, is to present mortality rates for the four largest Pacific ethnic groups living in New Zealand, using linked census-mortality data for 2001–04, thereby bypassing the problems of non-comparable collection of data for Pacific ethnic groups between census and mortality data (i.e. numerator-denominator bias). However, mortality is an uncommon outcome, and Pacific populations are small and young. Thus, given the small number of deaths in some Pacific strata, we have used hierarchical Bayesian (HB) regression modelling that allows smoothing of posterior mortality rates by shrinkage towards a prior covariate structure.\(^4\)
### Table 1. Demographic data for Samoan, Cook Island Māori, Tongan, and Niuean living in New Zealand at the 2006 census (and 2001 census for selected variables)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Census year</th>
<th>Samoan</th>
<th>Cook Island Māori</th>
<th>Tongan</th>
<th>Niuean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>2006</td>
<td>131,103</td>
<td>58,011</td>
<td>50,478</td>
<td>22,476</td>
</tr>
<tr>
<td></td>
<td>2001</td>
<td>115,000</td>
<td>52,600</td>
<td>40,700</td>
<td>20,100</td>
</tr>
<tr>
<td>% of total Pacific population</td>
<td>2006</td>
<td>49%</td>
<td>22%</td>
<td>19%</td>
<td>8%</td>
</tr>
<tr>
<td></td>
<td>2001</td>
<td>50%</td>
<td>23%</td>
<td>18%</td>
<td>9%</td>
</tr>
<tr>
<td>Sole any-Pacific † #</td>
<td>2006</td>
<td>77%</td>
<td>66%</td>
<td>82%</td>
<td>68%</td>
</tr>
<tr>
<td>Sole group-specific Pacific ‡ #</td>
<td>2006</td>
<td>66%</td>
<td>53%</td>
<td>71%</td>
<td>41%</td>
</tr>
<tr>
<td>Median age (years) #</td>
<td>2006</td>
<td>20.9</td>
<td>18.9</td>
<td>18.6</td>
<td>19.6</td>
</tr>
<tr>
<td>Current smokers #</td>
<td>2006</td>
<td>28%</td>
<td>38%</td>
<td>29%</td>
<td>33%</td>
</tr>
<tr>
<td>% adults with formal educational qualification #</td>
<td>2006</td>
<td>69%</td>
<td>55%</td>
<td>64%</td>
<td>60%</td>
</tr>
<tr>
<td>% living in Auckland #</td>
<td>2006</td>
<td>67%</td>
<td>60%</td>
<td>80%</td>
<td>79%</td>
</tr>
<tr>
<td>Born in NZ</td>
<td>2006</td>
<td>60%</td>
<td>73%</td>
<td>56%</td>
<td>74%</td>
</tr>
<tr>
<td></td>
<td>2001</td>
<td>58%</td>
<td>70%</td>
<td>53%</td>
<td>70%</td>
</tr>
<tr>
<td>% speaking own language</td>
<td>2006</td>
<td>63%</td>
<td>16%</td>
<td>61%</td>
<td>25%</td>
</tr>
<tr>
<td>% reporting religion</td>
<td>2006</td>
<td>86%</td>
<td>70%</td>
<td>90%</td>
<td>70%</td>
</tr>
<tr>
<td>% living in extended family</td>
<td>2006</td>
<td>35%</td>
<td>32%</td>
<td>39%</td>
<td>35%</td>
</tr>
<tr>
<td>Median personal income for adults</td>
<td>2006</td>
<td>$21,400</td>
<td>$19,800</td>
<td>$17,500</td>
<td>$21,500</td>
</tr>
<tr>
<td>Own home (a)</td>
<td>2006</td>
<td>23%</td>
<td>21%</td>
<td>19%</td>
<td>21%</td>
</tr>
<tr>
<td>Access to telephone</td>
<td>2006</td>
<td>83%</td>
<td>76%</td>
<td>81%</td>
<td>79%</td>
</tr>
</tbody>
</table>

(a) % of adults in fully or partly owned home

‡ ‘Sole any-Pacific’ mean the person did not self-identify as any non-Pacific groups, but might have self identified as two or more specific Pacific groups (e.g. Samoan and Niuean).

‡ ‘Sole specific-Pacific’ means the person only identified one Pacific ethnic group—and nothing else (e.g. Samoan only)

# Only 2006 reported due to either missing data for 2001 or little change from 2001.


### Methods

**Data**—Linked census-mortality data from the 2001–04 cohort of the New Zealand Census-Mortality Study (NZCMS) were used; details of the linkage, weighting for incomplete linkage of mortality data to census data, and variables specifications can be found elsewhere.\(^5\) Data was restricted to 380,000 person-years for 0–74 year olds with non-missing data on ethnicity, sex, age, and equivalised household income (62% of all eligible person years). Analyses were conducted on aggregated data for the 240 strata formed by cross-classifying ethnicity (4 groups) by sex (dichotomous) by age (5 groups) by income (tertiles) by natality (New Zealand-born versus overseas-born).

The ethnicity variable was classified using a “total count” definition.\(^6,p13\) For example, all of the following people would be categorised as Niuean: self-identified Niuean only; self-identified Niuean and Samoan; self-identified Niuean and New Zealand European; and self identified Niuean and Māori. Based on these examples, using the total count method, the Niuean and Samoan person would be
counted in both Niuean and Samoan groups in this paper. (The contribution of Pacific self-
identification to health status of European and Māori responses are not directly addressed in the paper.)
For the HB modelling, five age-groups were used: 0–14 years, 15–34 years, 35–44 years, 45–64 years,
and 65–74 years, centred at the 35–44 age group, and scaled so that each unit increase in scaled age
corresponded to an actual increase of 10 years. Thus, the above age ranges are represented by their end-
points which, after centring and scaling become elements from the set {-3, -1, 0, 2, 3}. To allow for the
non-linear increase in mortality with age, a linear spline for age with knots at the 35–44 and 45–64 age
groups was included in the prior mean [equation (3), below].
We used equivalised household income, categorised into tertiles within strata of sex and age-group,
and based on income for the New Zealand Pacific population. Income ranks were median centred and
scaled (divided by 10). Thus, income ranks (coded as 1, 2, 3) are transformed to (-0.1, 0, 0.1) and the
income effect was assumed to be log linear.
Hierarchical Bayesian Poisson regression—We largely follow the HB methods used previously in
the NZCMS by Young et al (2006). In brief, the method was as follows. Assuming death is a Poisson
process such that for Pacific ethnicity \(j = 1, \ldots, 4\) and stratum \(i = 1, \ldots, 60\) with deaths \(d_{ij}\),
mortality rate \(\lambda_{ij}\), and person-years at risk \(P_{ij}\), and using the notation \(x \sim D(a, b)\) to represent a
random variable \(x\) distributed as \(D\) with mean \(a\) and variance \(b\), a three-level Poisson model was
defined by:
\[
d_{ij} | \lambda_{ij}, P_{ij} \sim \text{Poisson} [\lambda_{ij} P_{ij}], \quad (1)
\]
\[
\lambda_{ij} | X_i, \beta_{ij}, \zeta \sim \text{gamma}[\mu_{ij}, \mu_{ij} / \zeta^2], \quad (2)
\]
\[
\log(\mu_{ij}) = X_i \beta_{ij}, \quad (3)
\]
\[
\beta_{ij}, \zeta \sim \pi. \quad (4)
\]
The mortality rate \(\lambda_{ij}\) had a gamma distribution with mean \(\mu_{ij}\) and variance \(\mu_{ij}/\zeta^2\), and the prior mean \(\mu_{ij}\)
had a structure that depended on covariates \(X_i\) and parameters \(\beta_{ij}\) through a log-link function. Second-
level parameters, \(\beta_{ij}\) (the regression “hyper-parameters”) and \(\zeta\) (the mortality rate variance or “shape”
hyper-parameter) were assigned independent prior distributions (“hyper-priors”) at the third level of the
hierarchy.
Extending the Young et al (2006) model to allow for variation by Pacific ethnicity, the regression
hyper-parameter vector was partitioned as \(\beta_j = (\beta_{0j}, \beta_{sex}, \beta_{age}, \cdots)\) to allow the intercepts (\(\beta_{0j}\)) to
vary by Pacific ethnicity. A standard approach was adopted for \(\beta_{ij}\), with uniform prior distributions for
each component.
The prior covariate structure influences the mean of the posterior rate, but the degree of influence
depends on the overall support for the prior covariate structure in the data, as well as on how much
local information is available. Given the structure of the model defined by equations (1) and (2), the
conditional posterior distribution for the mortality rate is also gamma with mean
\[
E[\lambda_{ij} | y, \beta_{ij}, \zeta] = B_{ij} \mu_{ij} + (1 - B_{ij}) y_{ij}, \quad (5)
\]
where \(y_{ij} = d_{ij} / P_{ij}\) is the observed mortality rate in the \(i\)th stratum of the \(j\)th ethnicity,
\(y = (y_{1}, y_{2}, \ldots)\) and
\[
B_{ij} = \zeta / (\zeta + \mu_{ij} P_{ij}). \quad (6)
\]

Thus, the conditional mean for $\lambda_{ij}$ is a weighted average of the prior mean $\mu_{ij}$ and the observed mortality rate ($y_{ij}$). The $B_{ij}$, which lie between zero and one, are known as shrinkages because larger values shrink the conditional posterior mean mortality rates towards the prior mean. The gamma shape parameter $\zeta$ provides a measure of the influence of the prior mean. The relatively uninformative uniform shrinkage prior of Christiansen and Morris (1997) was adopted for $\zeta$.7,8

A priori, following previous NZCMS work14, we expected interaction of age and income, and sex and income as predictors of the mortality rate. Thus the components of the regression hyper-parameters in equation (3) for the most complex prior model were

$$\beta_j = (\beta_{0j}, \beta_{sex}, \beta_{age}, \beta_{CoB}, \beta_{inc}, \beta_{age+inc}, \beta_{sex+inc}).$$

(7)

To allow comparison across the four ethnic groups, posterior mortality rates were directly standardised to the total Pacific population distribution using three combinations of sex, age, income and natality (country of birth; CoB). The first model (model 1) computed posterior mortality rates for each age, sex, ethnicity stratum using underlying data stratified by the same variables. Standardisation was by sex and age. Model 2 computed posterior rates for each age, sex, natality, and ethnicity stratum, using underlying data stratified by the same variables, and standardised by sex, age, and natality. Model 3 (the “full” model) added income as well.

All analyses and plots were done using the R environment (http://www.r-project.org) for statistical computation v2.2.0 available from the Comprehensive R archive Network (CRAN) website (http://cran.r-project.org) or SAS v8.2 (SAS Institute Inc., Cary, North Carolina). All HB analyses used WinBugs 1.4, available from (http://www.mrc-bsu.cam.ac.uk/bugs/winbugs/contents.shtml), and the R2WinBUGS package version 2.0-4.

Sensitivity analyses—We examined the likely impact of two possible biases: return migration and census under-enumeration. Methods were straightforward and are best described along with the results are presented below.

Results

Posterior mortality rates standardised for sex and age (model 1), plus natality (model 2) plus income (model 3), are shown in Table 2. The relative positions of the four ethnic groups did not alter greatly with this sequential posterior standardisation process. For the final fully standardised model, all-cause mortality rate ratios compared to Samoan were: 1.21 (95% credible interval 1.05 to 1.42) for Cook Island Māori; 0.93 (0.77 to 1.10) for Tongan; and 1.07 (0.88 to 1.29) for Niuean.

Table 2. Posterior all-cause mortality rates (per 100,000) and rate ratios (95% credibility intervals) by Pacific groups from models extended to include natality and household income

<table>
<thead>
<tr>
<th>Group</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Rate ratios c.f. Samoan</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rates</td>
<td></td>
<td></td>
<td>Model 1</td>
</tr>
<tr>
<td>Cook Island</td>
<td>288 (255–325)</td>
<td>295 (260–333)</td>
<td>294 (260–332)</td>
<td>1.18 (1.02,1.37)</td>
</tr>
<tr>
<td>Niuean</td>
<td>252 (208–297)</td>
<td>257 (211–306)</td>
<td>260 (215–308)</td>
<td>1.03 (0.84–1.24)</td>
</tr>
<tr>
<td>Samoan</td>
<td>244 (224–266)</td>
<td>243 (222–264)</td>
<td>243 (222–265)</td>
<td>1.00</td>
</tr>
<tr>
<td>Tongan</td>
<td>234 (197–269)</td>
<td>228 (193–264)</td>
<td>226 (190–262)</td>
<td>0.96 (0.80–1.12)</td>
</tr>
</tbody>
</table>

Model 1 = data stratified by ethnic group, sex and age only; sex and age included as independent variables in prior model; posterior rates directly standardised to the NZ Pacific population using a total definition of ethnicity; Model 2 = model 1, but extended to be additionally stratified by natality; additionally including natality as independent variable; posterior rates standardised as for model 1; Model 3 = model 2, but extended similarly for household income.
Table 3. Posterior all-cause and cause-specific mortality rates (per 100 000) and rate ratios (95% credibility intervals) by Pacific groups for the fully adjusted model 3

<table>
<thead>
<tr>
<th>Group</th>
<th>All-cause</th>
<th>CVD</th>
<th>Cancer</th>
<th>Injury/Suicide</th>
<th>All-cause</th>
<th>CVD</th>
<th>Cancer</th>
<th>Injury/Suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cook Island</td>
<td>294 (260–332)</td>
<td>111 (90, 135)</td>
<td>64 (50–80)</td>
<td>31 (21–42)</td>
<td>1.21 (1.05–1.42)</td>
<td>1.66 (1.26–2.13)</td>
<td>0.85 (0.56–1.09)</td>
<td>0.93 (0.60–1.34)</td>
</tr>
<tr>
<td>Niuean</td>
<td>260 (215–308)</td>
<td>75 (51–104)</td>
<td>66 (44–88)</td>
<td>18 (6.0–32)</td>
<td>1.07 (0.88–1.29)</td>
<td>1.11 (0.72–1.58)</td>
<td>0.87 (0.54–1.19)</td>
<td>0.53 (0.17–0.99)</td>
</tr>
<tr>
<td>Samoan</td>
<td>243 (222–265)</td>
<td>68 (57–79)</td>
<td>76 (65–89)</td>
<td>33 (26–42)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Tongan</td>
<td>226 (190–262)</td>
<td>58 (40–78)</td>
<td>73 (56–94)</td>
<td>27 (16–41)</td>
<td>0.93 (0.77–1.10)</td>
<td>0.86 (0.58–1.20)</td>
<td>0.96 (0.71–1.26)</td>
<td>0.82 (0.46–1.28)</td>
</tr>
</tbody>
</table>

HB models used data stratified by sex, age, country-of-birth, and income. Standardisation was to the total Pacific population using a total definition of ethnicity.
Table 3 and Figure 1 show the all-cause and cause-specific mortality rates and rate ratios for the fully adjusted model. Cardiovascular disease (CVD) mortality rate ratios, compared to Samoan, were: 1.66 (1.26 to 2.13) for Cook Island Māori; 1.11 (0.72 to 1.58) for Niuean; and 0.86 (0.58 to 1.20) for Tongan. That is, CVD mortality was nearly twice as high among Cook Island Māori compared to Tongan.

Moderating these strong CVD differences were weaker (and perhaps opposing) non-statistically significant differences in cancer. For injury/suicide mortality combined, Niuean people had a rate ratio of 0.53 (0.17 to 0.99) compared to Samoan people. Injury/suicide mortality rates for Samoan, Cook Island Māori, and Tongan groups were similar.

Checks of posterior predictive distributions of mortality rates against empirical estimates produced no evidence of model lack-of-fit.

**Sensitivity analyses: return migration and census under-enumeration**—The results of basic sensitivity analyses about the sex and age-adjusted (only) HB mortality rates for 0–74 year olds are shown in Table 4. The sensitivity analyses are crude. For example, input parameters (e.g. percentage returning to Pacific country) are applied to overall rates, not by strata of sex, age and so on. We consider return
migration and census under-enumeration in this paper, but other possible systematic biases are considered elsewhere.\footnote{9}

Return migration might occur when people are unwell, and they decide to return to their home country (country of birth, one would assume) to die. We set 8\% as the best estimate of underestimation of Pacific deaths due to return migration, and 4\% and 12\% as low and high scenarios (see elsewhere for justification\footnote{9}). When such bias is the same for all four Pacific ethnic groups, the rates vary but rate ratios do not (Table 5), meaning no bias in the rate ratios estimated in this project. However, would return migration when terminally ill be the same for the four Pacific groups in this project? We view return migration as more likely among Tongan and Samoan people, because: there are still substantial Samoan and Tongan populations (and health care facilities) in the home Island to act as ‘pull factors’ for return migration; and lower proportions of Samoan and Tongan people are born in New Zealand, probably predicting greater return migration for these groups when terminally ill.

Scenario A (10\% of terminally ill Samoan and Tongan people returning home to die, and 5\% of Niuean and Cook Island Māori returning home to die when terminally ill) is our best estimate of such differential bias. For all cause-mortality, this would reduce the Cook Island: Samoan excess rate ratio by about a quarter from 1.21 to 1.15, but would only reduce the CVD rate ratio by about 13\% from 1.63 to 1.55. The two remaining differential return migration scenarios (B = 12\% Samoan and 3\% Cook Island Māori; C = 25\% Samoan; 0\% Cook Island Māori) are both in our view unlikely, if not implausible. Thus we conclude differential return migrant bias is an unlikely cause of differences between Pacific-specific ethnic groups in CVD mortality.

Census under-enumeration of Pacific people is likely, and we selected a 4\% undercount as an overall estimate, and 2\% and 6\% as low and high scenarios about this estimated census undercount (see elsewhere for justification\footnote{8}). With respect to the calculation of mortality rates, these percentages cause an overestimate of the mortality rate.

Pacific mortality rates adjusted for such overestimation, when nondifferential across the four Pacific groups, have no impact on the rate ratios compared to Samoan (Table 5)—just the rates themselves. Is it plausible that census undercounting might vary between the four Pacific groups, and hence overestimation of mortality rates might vary by Pacific group? As Niuean and Cook Island Māori have automatic New Zealand citizenship, there might be less willingness among Samoan and Tongan population to be enumerated due to the history of action against ‘over-stayers’. We posited a 2\% census undercount for Niuean and Cook Island Māori, and 6\% for Samoan and Tongan people. However, this only serves to widen the mortality gap between Cook Island Māori and Samoan people (Table 4). Therefore, we conclude that differential census under enumeration across the four Pacific groups in our study is very unlikely (if not impossible) to cause the observed differences between mortality rates for Pacific-specific ethnic groups.
Table 4. Sensitivity analyses about hierarchical Bayesian (HB) all-cause and cardiovascular disease (CVD) mortality rates for scenarios of; return migration when terminally ill; census undercounting

<table>
<thead>
<tr>
<th></th>
<th>Rates</th>
<th>Rate ratios</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cook</td>
<td>Niuean</td>
</tr>
<tr>
<td>HB all-cause rate and</td>
<td></td>
<td></td>
</tr>
<tr>
<td>rate ratios</td>
<td>294</td>
<td>260</td>
</tr>
<tr>
<td>Amount of return</td>
<td></td>
<td></td>
</tr>
<tr>
<td>migration for terminally</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ill</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-differential</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Best estimate = 8%</td>
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<td>283</td>
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<td>271</td>
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<tr>
<td>High scenario 12%</td>
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<td>295</td>
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<tr>
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<td>Tongan; 5% Niuean and</td>
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<td>Cook</td>
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<tr>
<td>B. 12% Samoan; 3% Cook</td>
<td>303</td>
<td>276</td>
</tr>
<tr>
<td>C. 25% Samoan; 0% Cook</td>
<td>294</td>
<td>324</td>
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<tr>
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<tr>
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<td>4% as average</td>
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<tr>
<td>A. 6% Samoan and Tongan</td>
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</tr>
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<td>2% Niuean and Cook</td>
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<td>126</td>
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</tr>
<tr>
<td>A. 10% Samoan and Tongan; 5% Niuean and Cook</td>
<td>117</td>
<td>79</td>
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<td>B. 12% Samoan; 3% Cook</td>
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<tr>
<td>C. 25% Samoan; 0% Cook</td>
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<th>1.63</th>
<th>1.10</th>
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<tbody>
<tr>
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<td>57</td>
<td>1.63</td>
<td>1.10</td>
<td>1</td>
<td>0.85</td>
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<tr>
<td>High scenario = 6%</td>
<td>104</td>
<td>71</td>
<td>60</td>
<td>55</td>
<td>1.63</td>
<td>1.10</td>
<td>1</td>
<td>0.85</td>
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</tr>
</tbody>
</table>

| Differential, about best estimate of 4% as average | 6% Samoan and Tongan; 2% Niuean and Cook | 109 | 74 | 64 | 55 | 1.70 | 1.15 | 1 | 0.85 |

† Note that the CVD rate ratios are not identical to those in Table 4 due to use of integer values for the rates as the starting point for sensitivity analyses. E.g. for Cook Island Maori the rate ratio in Table 4 is 1.66; nevertheless 111/68 = 1.63 for purposes of Table 5.
Discussion

To our knowledge, this project is the first in New Zealand to show clear (and marked in the case of CVD) differences in mortality have been demonstrated between Pacific ethnic groups. This finding is important for further health research and policy. At present, policy approaches to Pacific health disparities often assume that they are the same across ethnic subgroups.

Our results demonstrate that this is not the case. There is parallel emerging evidence from the New Zealand Ministry of Health and University of Auckland on differences between Pacific groups in youth health status (work in progress) and some indicators relating to adult health status (Health and Disability Intelligence, Ministry of Health, work in progress), as well as published evidence of diversity among Pacific ethnic groups for mental health. 10

We have also demonstrated the added value of using hierarchical Bayesian methods for sparse data problems. We anticipate that the use of such methods will accelerate in the future both within New Zealand and internationally, for a range of research questions including the monitoring and understanding of the health of ethnic minorities.

We present quantitative sensitivity analyses in this paper about return migration to the Pacific when terminally ill, as well as census under enumeration of Pacific people, and conclude that neither bias could plausibly give rise to the observed variations in mortality—especially the elevated CVD mortality among Cook Island Māori.

Migration to New Zealand due to ill health should also not alter the findings in this paper, as we had data on usual residency on the 2001 census cohort and years in New Zealand on the mortality data that (assuming reasonable data accuracy) allowed us to exclude non-residents and ineligible deaths. We have also conducted basic comparative analyses on the 1996-99 NZCMS cohort9; Cook Island Māori also had an elevated CVD mortality rate in this period.

Explanations for the elevated CVD mortality risk of Cook Island Māori relative to the other Pacific ethnic groups include: (1) lesser degrees or greater waning of health-selective migration, and (2) greater degree of uptake of adverse risk factors (as evidenced, for example, by their higher smoking prevalence). Against these hypotheses is that inclusion of natality in our models had little effect.

In summary, Cook Island Māori have notably elevated CVD mortality compared to Samoan, Tongan and Niuean ethnic groups, and Niueans have lower injury and suicide mortality. No obvious explanation for these differing mortality risks is evident from our analysis. Researchers should, if at all possible, try to analyse and interpret results separately for specific ethnic groups within the Pacific grouping. This additional analysis should not only aim to explain the causes and contexts of these differences but also should assist in the development of policies that overcome these within-Pacific group disparities.

Policymakers and their advisors need to be aware of differences in health status and risks between Pacific ethnic groups, and consider Pacific ethnic group-specific policies and programmes where relevant, alongside pan-Pacific approaches.
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References:


http://www.moh.govt.nz/moh.nsf/0/3195F8D3155E1C2ACC2571FC00131A6D
Mum’s the word: factors that influenced young adults’ participation in the New Zealand Meningococcal B immunisation programme

Marian Bland, Geraldine M Clear, Adrianna Grogan, Kath Hoare, Jan Waldock

Abstract

Aims To identify the factors that influenced young adults (aged 16–19 years) when deciding whether or not to be immunised during the MeNZB campaign.

Method Semi-structured interviews were held with 11 young adults (7 who consented to the Meningococcal B immunisation and 4 who did not) to explore their immunisation decision. A qualitative descriptive data analysis was then undertaken to reveal the latent and manifest themes arising from the interviews.

Results Similar influences on the decision-making process were described by all the young adults, regardless of the outcome. These included weighing up the risks involved, and making collaborative decisions with their families, especially their mothers. Situating the decision in a personal context required the young adults to consider factors such as needle-phobia, herd immunity, and the accessibility of the immunisation.

Conclusion The information used in immunisation campaigns involving young adults must be carefully balanced so that the situation becomes real to the target group, while also helping inform others, especially parents, who will be involved in the decision-making process.

Between 2004 and 2006, young New Zealanders (aged under 20 years) were the focus of a mass immunisation programme against the New Zealand serogroup B Meningococcal disease (Meningococcal B). A vaccine, MeNZB, was developed in response to what was described as a “devastating 14-year epidemic” (p.1) of the disease in New Zealand.¹

The Meningococcal Vaccine Strategy (The Strategy) aimed for a 90% uptake of the vaccine in the target group, with a projected 70% reduction in disease rates.¹ By the official end of the mass MeNZB campaign in 2006, 86% of 5–17 year olds had been immunised, but the uptake in those aged 18-19 years was only 54%.²

The Strategy was unusual in that it offered young adults what was probably their first opportunity to make their own decisions in relation to immunisation in a mass vaccination campaign, yet little is known of young adults’ attitudes towards vaccinations.³ A review of research on the sociology of immunisation concluded further qualitative research was required into the thinking and decision-making that informs choices about immunisation.⁴ This pilot study therefore sought to investigate the factors that had influenced young adults when they made their decision about whether or not to have the MeNZB vaccination.
Method

Approval for this qualitative descriptive study was obtained from the Central Regional Ethics Committee, and the UCOL Research Committee. Inclusion criteria for the study required the participants to have been aged between 16-19 years at the commencement of the MeNZB roll-out, speak English, and live in the Palmerston North area.

It was intended to recruit a total of 20 young adults, 10 who had consented to immunisation, and 10 who had not. Data collection began in September 2006. Despite advertising in free community newspapers, distributing flyers in places where young people were likely to gather, and presentations to youth representatives, recruitment was extremely slow.

With the consent of Central Regional Ethics, recruitment was then extended to include high schools and other youth-focused groups. An incentive payment of a $10 phone card was also offered to each participant, but these changes did not significantly improve recruitment rates. In May 2007, with 11 participants enrolled in the study (7 who had completed the full MeNZB immunisation, and 4 who declined to be vaccinated) and the major phase of the immunisation campaign officially ended, further attempts at recruitment were abandoned.

Demographic characteristics of the 11 participants are included in Table 1.

Table 1. Demographic characteristics of the study participants

<table>
<thead>
<tr>
<th>Age range</th>
<th>17–21 years at last birthday</th>
</tr>
</thead>
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<td>17 years</td>
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<tr>
<td>18 years</td>
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<td>19 years</td>
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<tr>
<td>20 years</td>
<td>3</td>
</tr>
<tr>
<td>21 years</td>
<td>1</td>
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<table>
<thead>
<tr>
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</tr>
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</tr>
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<td>10</td>
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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>New Zealand European/European</td>
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<table>
<thead>
<tr>
<th>Highest school qualification</th>
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</thead>
<tbody>
<tr>
<td>NCEA level 2</td>
<td>27%</td>
</tr>
<tr>
<td>NCEA Level 3 or equivalent</td>
<td>73%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Previous immunisation history</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 participants were confident they were ‘fully’ immunised, but the remainder were unsure of their previous immunisation history.</td>
</tr>
</tbody>
</table>

Once written consent had been obtained, participants were interviewed individually to explore their MeNZB decision. All of the authors engaged in this process, although only one author was present for each interview. A brief schedule was used to guide the semi-structured interviews, which were conducted in a mutually convenient private setting.

Participants were interviewed once, with interviews lasting up to 45 minutes. These were audio-taped and transcribed verbatim. Identifiers were removed, and pseudonyms chosen by the participants. A descriptive thematic analysis was undertaken by two researchers, initially by analysing each interview as a single entity, and then by cross-referencing the emerging themes with a focus on the key research aims. Differences and similarities in the latent and manifest themes arising from the data were explored, and overarching themes finalised. These findings were then confirmed by all the authors.
Results

Four of the 11 participants decided not to participate in the MeNZB immunisation programme. The processes they engaged in to reach this decision were similar to those who did participate – *weighing up the risks, making collaborative decisions, and considering the personal context.*

**Weighing up the risks**—Making the decision about vaccination involved weighing up the risks involved. Fears about the safety of the vaccine had to be balanced against the likelihood of contracting Meningococcal B. Regardless of their vaccination decision, all the young adults referred to the serious outcomes associated with Meningococcal B. Knowing someone who had contracted the disease helped ’make it real’:

> Two people in the same town actually contracted the disease. One person was in a few classes of mine and things like that. …. Everything about it was really just shocking.  
> (Sarah, immunised)

Knowledge of the risks associated with the disease did not necessarily make it any more real to individuals:

> I know what it does, and I know how it spreads but I mean, I know it’s bad, but I still share drinks and lip gloss and things like that, and it’s just, it’s not real to me, that it can happen to me or someone I know.  
> (Sara, not immunised)

Graphic images in the media, such as the photos of Baby Charlotte, helped some of the young adults appreciate the serious nature of the disease, and encouraged them to consent to the vaccine. Others were “*grossed out*” by visual representations of the disease, or found it difficult to relate the information to their age-group.

Responses to the personalities who featured in the MeNZB promotional material (such as Nessian Mystik, Jason Gunn) were also mixed, with most considering them better suited to younger age groups. The campaign emphasis on young adults aged less than 20 had also given several participants the impression that the mere fact of attaining the age of 20 would magically confer increased immunity.

Even when the likelihood of contracting Meningococcal B was considered to be a significant risk, the decision to vaccinate was complicated by concerns about the perceived safety of the vaccine being offered.

> Yeah, no long-term information. Not knowing what was going to happen in the long-term sort of thing. I mean obviously it’s gonna protect you against Meningitis but you don’t know what could happen or anything.  
> (Charlotte, not immunised)

> I didn’t want to get sick from it [the vaccine]. I just felt it wasn’t a very well-researched vaccine.  
> (Maree, not immunised)

In addition, concern was expressed that the information provided about the vaccine was biased. Participants (or their parents) found it difficult to get what they considered to be balanced information on the risks and benefits.
Making collaborative decisions—Searching for evidence about the need for, and potential risks associated with, the vaccine was an activity more likely to be undertaken by parents than the young adults:

Mum found some more stuff about the effectiveness of it and things like that, that we never got to see and it wasn’t really publicised to people at school

(Sara, not immunised)

Mum did a bit of research into it and it was just like “nah”

(Maree, not immunised)

Having a health professional in the family meant that several of the young adults were happy to accept what they considered to be an informed opinion on the relative merits of the vaccine:

My dad’s a GP, so he strongly advised me, he said “Just get it done, it’s free, so you might as well get it done” and that was the underlying factor why I had it done

(Sally, immunised)

Parents, especially mothers, were hugely influential in helping make the decision about immunisation, although the majority of young adults felt the final decision was theirs:

Mum had said earlier when the ads started coming on TV that me and my brother should get them done when they came around the schools. So she thought I should get it but I thought so too.

(Hillary, immunised)

Mum basically said “no.” I mean, if I had wanted it I could have got it but she basically, I agree with her, that it hadn’t really been around for that long, well researched or anything.

(Charlotte, not immunised)

Several of the young adults were also influenced by teachers they respected at their school, especially the science teachers, and appreciated having someone outside of the family who offered an independent opinion. However Maree felt “hounded” at school over her decision not to have the vaccination, being repeatedly asked to explain her position. Confidence in her ability to fight off the disease meant she questioned whether she needed the vaccine:

It’s the vaccine itself. I just don’t agree with putting foreign things into your body when I’m already healthy and I know I’ve got a perfectly healthy immune system as it is. I just don’t get the point.

(Maree, not immunised)

While there was little discussion with peers about the merits of the vaccine, accounts of the short-term side effects others experienced was of more significance:

I talked to some girls [in the hostel] who had got it done and they were saying, they’ve got these flu symptoms, and that it [the injection] really hurt

(Polly, not immunised)

Observing first hand the experiences of others was also disconcerting for those who went ahead with the immunisation:

But it’s not easy to watch someone be dragged out on the stretcher for the same thing you’ve just been injected with.

(Sallie, immunised)
Putting it in a personal context—The unique personal context of each of the study participants was instrumental in the decision-making processes they engaged in, and their subsequent comfort with that decision. Themes related to the personal context included easy access to the vaccine, needle phobia, and previous experiences with vaccinations.

The majority of participants had easy and free access to the vaccine, as it was rolled out in high schools and tertiary institutions. Jac was working, and rapidly nearing the age when he would need to pay for the vaccine. He made four visits (the first to get information and a consent form, and then three for the vaccine administration) to his general practitioner to complete his vaccination while it was still free.

Despite the vaccine being free, the fact that it necessitated three injections at regular intervals was problematic. Five participants described how their needle-phobia had to be factored into their decision about whether to have the MeNZB vaccination:

Well, I made the decision, in consultation with my mum, but basically it’s, the biggest factor is I don’t like needles and things so having three injections. ..//.. Pretty much the deciding factor for me was if I can avoid any sort of injection or blood test or anything, I will. I hate them. ..//.. If it had come in pills then I would possibly have had it.

(Sara, not immunised)

Sallie, also needle phobic, decided to go ahead with the vaccine, but asked for additional support, with unexpected consequences:

I don’t like injections or needles at all. I actually said to the health nurse “don’t let me watch, I don’t want to see”, and she said “look over there” and I turned around and saw my friend get it, which was even worse (laughs).

(Sallie, immunised)

Mia reported to the author interviewing her that she was crying when it was her turn for the vaccination, and was asked why:

Oh the injection! Yeah, I hate injections. They were quite good about it, like they were a bit mean, but they got a doctor, they got someone to hold me down, so, ah, like pull my face away so they could put it [the needle] in. ..//.. She kind of pushed my face into her boobs

(Mia, immunised)

The timing of the vaccinations was also a consideration. Sara was reluctant to expose herself to potential vaccination side-effects that might disrupt her sporting and travel plans. Polly, moving into a university hostel, would have had to make arrangements for two of the vaccinations in a new part of the country, and just never got round to it.

Regardless of whether they were vaccinated or not, all the young adults reported they were comfortable with their decision. Stories of people contracting Meningococcal B despite having completed the immunisation programme though did alarm them.

It’s a bit disheartening to hear now that people who have had the vaccine are contracting Meningococcal B. That’s a bit disturbing. ..//.. You can’t really be sure if the vaccine is faulty. ..//.. I suppose it’s more a worry that you’ve been immunised, you think you’re safe and then you can still potentially get it.

(Jac, immunised)

None of the four young adults who chose not to have the vaccine would change their decision, although one acknowledged that if someone she knew personally got the disease she would probably reconsider. Two took comfort from the possibility of herd as most of their friends were vaccinated, with Sara commenting:
I thought if my friends are immunised, well then hopefully that decreases my chances of getting it because I can’t catch it off them.

(Sara, not immunised)

Discussion

The significance of family input into the decision-making processes of these young adults was ultimately the major influencing factor in whether they decided to be immunised against Meningococcal B. Parents, especially mothers, were largely responsible for evaluating whether the vaccine was both necessary and safe, and advising the young adult accordingly. Despite the acknowledged importance of this advice, all but one of the young adults felt that ultimately the decision was their own. There was little evidence of peer discussion as part of the decision-making process, other than to share stories of vaccination side-effects.

Three participants made a deliberate decision to not have the MeNZB vaccination. Concern about the long-term safety of the vaccine, and its short-term side effects, were cited as the key factors in their decision. Another, in the process of moving into a university hostel, felt bullet-proof once she turned 20 because she was now one year older than those targeted by the immunisation campaign, despite the risk factors associated with living in a hostel.

Weighing up the risk was fundamental to the immunisation decision. The complexity of risk perception is such that its relationship with risk behaviour is still not fully explained. Several factors contributed to the perception of risk, such as the graphic nature of some of the promotional material. Responses to that material were mixed. Images of Baby Charlotte, for instance, motivated several of the participants to have the vaccine, while other young adults failed to relate the experiences of an infant to their own situation. The potential threat of the disease was just “not real” to most of these young adults, except for those who personally knew of someone who had contracted it.

Perceptions of the risk of contracting the disease were then balanced against confidence in the short and long-term efficacy and safety of the vaccine. Internationally, there has been a long history of vaccine controversies. Immunisation is one of the few times when “governments attempt to force a medical solution on citizens, regardless of their personal desires or beliefs, and this may be part of the problem” (pp.38-39). Suspicion about the integrity of the information supplied by the Ministry of Health relating to the MeNZB vaccine meant that other sources of information including the internet, and the opinion of trusted health professionals, or teachers at school, were also sought. These findings are similar to those from research into the MeNZB decision-making processes of parents of children aged up to 15 years.

Although schools-based immunisation programmes are an efficient mechanism for reaching large numbers of children quickly, research has indicated parental preference for immunisation at general practices. Schools must balance what they consider to be the best interests of their students with the need to respect their autonomy. In a democracy people would expect to have a choice, and their decision-making processes respected. Research into the experiences of public health nurses with high school students during the MeNZB roll-out highlighted the dilemma those nurses
faced when parents had consented to the immunisation, but the young person then refused.\textsuperscript{9}

The young adults in this study generally demonstrated confidence in their decision, although those who had chosen not to be immunised took comfort in the perceived safety net afforded to them from herd immunity, with so many of their peers being immunised. This comfort may have been misguided, as nationally only half of those aged 17-19 were immunised\textsuperscript{7} and there was no expectation in The Strategy of herd immunity.\textsuperscript{10}

Making the risk of a potentially serious disease real to young adults is an ongoing challenge.\textsuperscript{5} While the majority of those targeted in this campaign (children aged from 6 months onwards) were not old enough to be able to make the immunisation decision themselves, a significant number of high school students were, and campaign material needs to reflect that reality.

The findings of this study support one of the recommendations arising from the evaluation of the MeNZB campaign\textsuperscript{2} that “advertising resources should be devoted to documenting and advertising situations that describe the disease and its impact on young people aged 16-19 years” (p.73). For instance, ‘survivor stories’ featuring young adults are likely to be more relevant for this age group than the story of a small infant. The use of peers, celebrities and the media may not necessarily be a major influence\textsuperscript{11} and participants in this research responded to the same information in very diverse ways.

It has been suggested\textsuperscript{12} that immunisation campaigns for young adults need strategies that can accommodate the diversity of those within the adolescent and young adult age groups and developmental stages.\textsuperscript{13} The MeNZB data for those aged 17-19 years was reported separately in the evaluation report\textsuperscript{2}, but data for younger adults (15-16 year olds for instance) was essentially grouped with all other school-aged children. Expanding reporting categories to include all school-aged young adults considered competent to make autonomous decisions about vaccination may help inform further understanding of the health decisions of this group.

The limitations of this small study are acknowledged. The data collection process commenced towards the end of the campaign roll-out, and participants had difficulty recalling the sources of the information they used to inform their immunisation decision. They were hazy about their immunisation status prior to the MeNZB campaign, meaning no conclusion can be drawn about how parental attitudes to immunisations generally may have impacted on this specific campaign.

Finally, the sample size for this qualitative study was small, and only one ethnic group in New Zealand was represented. Nevertheless, the data that was collected revealed the complexity of the decision-making processes for these young adults associated with the uptake of the MeNZB vaccine. The current Gardisal vaccination campaign reinforces the importance of understanding more about how young people make decisions about immunisation.

### Conclusion

Young adults are unique from other populations in relation to immunisation.\textsuperscript{13} The MeNZB immunisation campaign has been credited with playing an important role in
the decline of the Meningococcal B epidemic in New Zealand. Yet only 56% of young adults aged 17-19 years nationally chose to have the MeNZB vaccination, which is an indication of the complexity of engaging of this age group in such decisions. Difficulty in making the risks associated with Meningococcal B more meaningful to the participants in this research was compounded by concerns about the safety and efficacy of the vaccine, and perceptions that information was biased.

In addition, media activities also impacted on public perceptions of the situation. Education is essential to ensure that young adults are fully aware of the risks about specific conditions and the associated vaccine so they can make well-informed decisions. The influential role that parents played in the decision-making process is such that they must also be targeted in any promotional material related to young adults.

**Competing interests:** None known.

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**References:**


Sero-prevalence of leptospirosis in workers at a New Zealand slaughterhouse

Jackie Benschop, Cord Heuer, Patricia Jaros, Julie Collins-Emerson, Anne Midwinter, Peter Wilson

Abstract

Aims To undertake a pilot study to measure the sero-prevalence to Leptospira serovars Hardjo and Pomona in an occupationally exposed group. To evaluate worker age, sex and previous clinical episodes of leptospirosis as risk factors for sero-positivity.

Methods A cross-sectional sero-prevalence study was conducted in February and March 2008 at a predominantly sheep slaughterhouse in Hawke’s Bay, New Zealand. A single blood sample was collected from 242 meatworkers, comprising 145 men and 97 women. Sera were tested by the Microscopic Agglutination Test (MAT) with a titre cut point of 1:24, using serovars Pomona and Hardjo as antigens. Age, sex, and details of previous clinical episodes of leptospirosis were recorded.

Results Overall sero-prevalence was 9.5%. Ten (4.1%) workers were positive to serovar Hardjo (titres 1:24–1:192), 13 (5.4%) were positive to serovar Pomona (titres 1:24–1:768), and one worker was positive for both serovars. Sero-prevalence was 13.1% and 4.1% in men and women, respectively. The median age for sero-positive workers was 54 years while that for sero-negative workers was 48 years. Twenty-three workers (9.5%) reported a leptospirosis disease episode 1–35 years previously, and 14 of those were sero-positive in the current study.

Conclusion The sero-prevalence observed suggests significant exposure to leptospirosis from sheep in meatworkers in the slaughterhouse studied. This sero-prevalence was similar to that reported in a survey in 1982. Further study is needed to determine the link between sero-prevalence and incidence, whether the prevalence of leptospirosis is similar in other slaughterhouses, and to develop a better understanding of risk factors important for the reduction of exposure of this occupationally acquired disease.

Leptospirosis in New Zealand is predominantly an occupationally acquired zoonosis occurring at a higher frequency here than in other countries where the disease is notifiable.1 Farmers, meat and forestry workers are most at risk. Where serovar was determined, Leptospira borgpetersenii serovar Hardjo was the most frequently diagnosed serovar from notified human cases in 2007 (44%). In the same year, 27% of cases were L. interrogans serovar Pomona positive and 19% were L. borgpetersenii serovar Ballum positive.2 Leptospirosis can result in severe illness and in some cases death.

During 2003-2005, leptospirosis resulted in 207 hospitalisations in New Zealand.3 However, the majority of cases are relatively mild and may be misdiagnosed as
influenza. Thus, while 121 cases were notified in 2008, the true incidence of leptospirosis is probably many times that reported.4,5

*Leptospira* species in New Zealand have many animal hosts, mainly livestock species, but also rodents and other wildlife.6 Earlier research and disease control has focused largely on dairy cattle and domestic pigs. It is estimated that 90% of dairy and 10% of beef herds in New Zealand are vaccinated.4 Recent evidence suggests that 9% of farmed deer herds are vaccinated.7 In beef cattle, herd and animal sero-prevalence to serovar Hardjo is estimated to be over 50%,8 while the sero-prevalence of serovar Pomona is lower.

A nationwide survey of 110 deer farms found Hardjo alone was present on 61% of farms, Pomona alone on 3.6%, and both together on 16.4% of farms.9 A slaughterhouse survey in lambs found 49% of lines had one or more animals with titres to Hardjo, Pomona or both.10 There is evidence that more clinical disease in sheep and deer is emerging, particularly with morbidity and mortality in lambs and young deer.9,11

Leptospirosis in New Zealand meatworkers and meat inspectors was investigated between 1979 and 1984, with sero-prevalence reported between 4% and 10%.12-14 In 1990 to 1992 the occupational distribution of cases included a higher proportion of livestock farm workers, (57%), compared with meatworkers, (43%).4 However, during the period 2001 to 2003 the proportion of cases reported in meatworkers was 38% and in farmers, 37%.15 Results from 2007 showed that 47% of notified cases were in meatworkers compared with 36% in farmers.2 Further, 13 of 15 patients admitted to the intensive care unit of Hawke’s Bay Hospital with a diagnosis of leptospirosis from 1999 to 2005, were employed as meatworkers or meat inspectors.3

Thus, in terms of occupational risk, the epidemiology of infections in humans appears to be changing, with transmission in slaughterhouses becoming more important. Recently, simulation studies based on culture results from apparently healthy lines of sheep at a Manawatu slaughterhouse, estimated the exposure risk for workers ranged from 5, (eviscerator), to 26, (offal handler) carcasses shedding *Leptospira* organisms per working day.16

The highest regional incidence of notified cases of leptospirosis in NZ in 2004 (13.1 per 100,000) and 2005 (8.4 per 100,000) was in Hawke’s Bay, compared with national figures of 2.3 and 2.1 per 100,000, respectively.3 Hawke’s Bay is one of the regions identified in an August 2007 report from the Department of Labour as being a suitable region for further leptospirosis research.4 This pilot cross-sectional serological study, involving blood collection and interview, was therefore conducted amongst meatworkers at a slaughterhouse in this region.

The aims were to measure the sero-prevalence to *Leptospira* serovars Hardjo and Pomona, and to evaluate worker age, sex, and previous clinical episodes of leptospirosis as risk factors for sero-positivity.

**Materials and Methods**

This study was approved by the Massey University Human Ethics Committee: Southern A, application 05/123.
Data collection and handling—The slaughterhouse was located at Takapau in southern Hawke’s Bay, New Zealand. This plant processes predominantly lamb and mutton and employs 960 staff in peak season, from November until approximately June each year. Since 2006, bobby-calves have also been slaughtered for a six week period in July and August.

Two investigators visited the plant on five occasions in November 2007 during the period of worker induction to inform staff of the proposed study. Initially, 325 workers agreed to participate. Absenteeism, resigning from the job, being afraid of needles, or inability to be released from work because of a shortage of staff resulted in 242, (145 men and 97 women), being blood sampled and interviewed in February/March 2008. Workers fell into two groups: those not on the slaughter-board, and so considered to be at low risk of occupational exposure to leptospirosis, \((n=125)\); and, those on the slaughter-board, and considered to be at high occupational exposure to leptospirosis, \((n=117)\). The latter group included eight meat inspectors.

To ensure minimal interference to production for the required 30 minutes on each day for sampling and interview, a “rover” was used to relieve workers who were drawn strategically from different parts of the slaughterhouse.

To investigate age, sex and previous clinical episodes of leptospirosis, a questionnaire was administered at the time the blood sample was taken. The case definition of a previous diagnosis of leptospirosis was that the worker had clinical signs consistent with leptospirosis, had time off work for the disease, and had sought assistance from a health professional. Fever, severe headache, back pain, sore eyes, sweating, general debility and feeling “very unwell” were considered to be signs consistent with leptospirosis.

To ensure consistency of delivery and to limit interviewer bias, questionnaire administration was limited to four trained individuals. Data were treated in confidence. Participants were notified of their serum blood test results by mail within two to three weeks of blood sampling. Ninety-two percent of participants consented to having an independent occupational physician receive a copy of their results, ensuring medical advice and follow up of sero-positive workers.

Data were entered into a database, and quality control measures applied, to assure accuracy. The continuous variable age was categorised into terciles \((20–40; 41–47; \text{ and } 48+ \text{ years of age})\). Summary statistics and unadjusted odds ratios were calculated to investigate the association between each worker’s serology result and age, sex and previous history of leptospirosis.

Serological testing—Blood samples were collected by one of two certified phlebotomists using plain vacutainer tubes for serum collection. After sampling, blood tubes were kept cool \((5^\circ \text{C})\), transported to Massey University and stored in a cooler over night to retrieve the maximal volume of serum. Tubes were then centrifuged at approximately 1500 \(g\) for 10 minutes and the serum was drawn off. Sera were tested by the microscopic agglutination test (MAT) in the Leptospirosis Research Unit laboratory, Massey University, using a modification of the technique described by Faine et al (2000). Doubling dilutions from 1:24 to 1:3072 were made and serovars Pomona and Hardjo used as antigens. A cut-off point of \(\geq1:24\) dilution was used to designate a positive titre.

Results

Of the 242 sampled workers, 23 (9.5%; 95% CI: 6.4% - 13.9%) had a positive titre to either \(L. \text{interrogans}\) serovar Pomona \((n=13)\) or \(L. \text{borgpetersenii}\) serovar Hardjo \((n=9)\), or both \((n=1)\) (Figure 1). Titres were generally higher to Pomona (range: 1:24 to 1:768) than to Hardjo (range: 1:24 to 1:192).

The sero-prevalence in men was 13.1%, \((19/145)\) (95% CI: 8.6% - 19.6%), while in women it was 4.1%, \((4/97)\) (95% CI: 1.6% - 10.1%). Compared with women, the unadjusted odds ratio for having a positive titre for leptospirosis for men was 3.51 (95% CI: 1.27 – 12.40).

Sero-positivity was also associated with age. The median age of those positive being 54 years, (IQR: 47 - 59 years), and those negative being 48 years (IQR: 35 – 56 years). There was a dose-response relationship between the unadjusted odds ratios for having a positive titre and the age terciles. Compared with workers in the lowest age
tercile, (18 – 40 years), the unconditional odds ratio for having a positive titre for leptospirosis for workers aged 41 to 53 years was 4.79 (95% CI: 1.15 – 32.48); and for workers aged 54 to 69 years was 6.94 (95% CI: 1.84 – 45.37).

Twenty-three workers met the case definition for a previous diagnosis of leptospirosis. A further five workers reported suspicion of having previously had leptospirosis, but they did not meet the case definition for this study. In the 23 that met the case definition, the previous disease episodes occurred from one to 35 years ago, (median: 8 years; IQR: 5 -14 years). Six of these 23 reported two previous episodes of disease. Fourteen of the 23 workers (58%), reporting at least one previous leptospirosis disease episode, had positive titres in the current study. For these 14 the most recent leptospirosis disease episode occurred from 1 to 15 years prior to the current study. For the nine workers that meet the case definition and were sero-negative, the most recently reported clinical disease episode occurred from 3 to 35 years ago.

Compared with workers having no previous clinical episode, the unadjusted odds ratio for having a positive titre for leptospirosis for workers having had at least one previous clinical episode was 36.29 (95% CI: 12.86 – 111.40), suggesting that the previous diagnosis of leptospirosis was accurate.

Sixteen of the 23 workers that met the case definition for previous leptospirosis provided information on test results for that disease episode. Eleven of the 16 reported a positive blood test, three reported a negative blood test, and two recalled a blood test being taken but were unsure of the result.

A scatterplot of reciprocal titres against years since last clinical episode for all 32 workers that met the case definition for previous leptospirosis disease, and/or were sero-positive in the current study, is presented in Figure 2.

Discussion

This paper presents a sero-survey of leptospirosis of an occupationally exposed slaughterhouse work-force, reporting a 9.5% seroprevalence. To the authors’ knowledge, exposure to leptospirosis in workers in New Zealand has not been reported since 1982. The sero-prevalence in the population of workers in the current study (9.5%) was higher than that measured in a population of 1248 meatworkers (6.2%) from the 1979–1982 study.

Workers from the earlier study were either processing pigs only (n = 26); sheep and cattle only (n = 578); or sheep, cattle and pigs (n = 644). The higher seroprevalence in the current study was in the face of the work-place exposure being predominantly to sheep, and that the workers had a high level of compliance with the use of protective clothing. This survey was a pilot, involving one slaughterhouse only, so data may not be representative of slaughterhouse workers elsewhere.

The MAT is currently the gold standard test for the sero-diagnosis of leptospirosis. Detectable serum antibodies are present 7-10 days after infection resulting in poor sensitivity in newly acquired infections. This may have resulted in a minor under-estimation of the sero-prevalence in this study. However, this is unlikely to be significant, since the persistence of seropositivity in previously clinical cases
presented here (Figure 2) suggests that the annual incidence rate is less than the 9.5% prevalence rate.

No inference of incidence can be made from this study since MAT titres following previous disease appear to persist for variable times with Indian\textsuperscript{20} and New Zealand\textsuperscript{13, 14} studies reporting that this can be for up to ten years. Data from 14 of the 23 sero-positive workers in this study reporting a previous clinical episode of leptospirosis showed titres were generally higher for the more recent cases (Figure 2). However, further research is necessary to resolve the complex issue of persistence of a titre after a clinical episode.

A higher seroprevalence and generally higher titres were observed to Pomona than Hardjo (Figure 1). The meatworker with the highest titre (Pomona 1:768) reported having recently been unwell. On receipt of his results the occupational physician arranged follow up testing which confirmed infection.

Our finding of a higher sero-prevalence in males than females is in agreement with notified cases in New Zealand both recently\textsuperscript{2} and previously in a review of notifications from 1990 to 1998.\textsuperscript{1} This difference is possibly due to the differing work patterns of men and women in the slaughterhouse. Women are more likely to work in processing areas (e.g. packing), rather than on the slaughter-board where there is a higher risk of exposure to the organism, mainly through urine. In this study population 48\% (47/97) of women worked in processing areas, compared with 36\% (52/145) of men.

**Figure 1. Frequency histogram of positive reciprocal titres to serovars Pomona and Hardjo in study participants**
In 2006, when this study was first proposed, it was designed to investigate seroconversion rates in a longitudinal study. Access to meat plants that logistically could handle the enrolling and subsequent repeated sampling of initially sero-negative workers was not possible at that time, even for a cross-sectional study. However, there
was a change in industry awareness and concern about leptospirosis in New Zealand in 2007. This was associated with the death of a meatworker from the disease and was widely reported in the public media, facilitating this cross-sectional study. In addition, the Department of Labour released the report “Leptospirosis: Reducing the impact on New Zealand workplaces” in August 2007 identifying current information gaps, one of which this study begins to address.

The result of this study should prompt further investigation into the incidence rate for leptospiral infections in meatworkers in this plant and elsewhere, along with other risk occupations, on a longitudinal basis, encompassing a wider geographical distribution.

While this study has provided useful information in a pilot study context, it contains potential limitations. Selection bias may have resulted in either an under-estimation of the sero-prevalence. This could result from the “healthy worker effect”, whereby workers ill or convalescent with leptospirosis would not be available for sampling.

Furthermore, this study analysed only the two serovars most common in domestic livestock in New Zealand thus possibly under-estimating the true sero-prevalence of leptospiral infection per se. *Leptospira borgpetersenii* serovar Ballum has been associated with rodents in New Zealand but has also been associated with disease in New Zealand livestock species (Fraser Hill, personal communication), albeit rarely. This serovar was reported in 19% of the notified human leptospirosis cases in New Zealand in 2007. Further, serological evidence of serovar Copenhageni is occasionally observed in domestic livestock including deer. Thus transmission of this serovar could occur from livestock, or directly from its rodent reservoir host.

The study sample of 242 participants was representative of the sex distribution in the eligible population of 960 employees. The eligible population was 63% male (351 women and 609 men), the study population was 60% male (91 women and 145 men). However, the 242 participants sampled were slightly older than those in the eligible population. Age terciles in the eligible population were 17 – 31, 32 – 47 and 48 + years; while those in the study population were 20 – 40, 41 – 47, and 48+ years.

As we wish to make inference for the entire workforce, it is important to consider the effect of voluntary participation, that those with a high level of interest in leptospirosis (including those with a clinical history), are more likely to present for sampling.

This study used the dilution of 1:24 as the titre cut point to indicate sero-conversion and sero-prevalence should be interpreted in that context. There is debate about the appropriate cut-point for a diagnostic titre indicating current leptospiral infection, or previous exposure. The optimal cut-point depends on the endemicity of disease in the population of interest, which in the present case, was apparently healthy meatworkers with a potentially high risk of exposure to the organism. However, earlier published surveys in meatworkers, meat inspectors and dairy farmers in New Zealand also used a cut-point of 1:24, thus the choice of this titre in the present study allows direct sero-prevalence comparison with earlier studies.

This study used only one serum sample because our interest was in sero-prevalence, not acute infection. For a definitive diagnosis of current leptospiral infection the MAT should be performed on paired serum samples, for which the recognised criterion is a four-fold rise in titre or sero-conversion. In NZ this is adopted for accident
compensation for workplace infection. Thus while sero-prevalence is described, no conclusion on the current infection status of the individual meatworker can be drawn from these data.

We intend to use these pilot study results as a baseline for studies in other occupationally exposed groups involving other livestock species, and including farmers, meatworkers in other plants, technicians and veterinarians. During 2001-2003 most reported human leptospirosis infections in New Zealand were associated with contact with cattle, either on their own or with other animals. Additionally, establishing a baseline sero-prevalence in a non-occupationally exposed population is important to enable appropriate interpretation of the relevance of the present result.

In this communication we have reported the sero-prevalence of leptospiral titres in workers at a Hawke’s Bay, predominantly sheep, slaughterhouse, and the association with worker age, sex and previous history of clinical disease. Multivariable analysis of additional data collected in the questionnaire will be applied to further evaluate risk factors for sero-conversion. This will include adjusting for the confounding effects of age and lifestyle exposure. In addition, strain-typing of organisms isolated from both animal and human isolates will be undertaken in future studies. Those studies should provide a greater understanding of disease prevalence, incidence, and transmission pathways and assist in reducing in the risk of infection in occupational risk groups.

Competing interests: None known.

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References:
Acute gastrointestinal illness in New Zealand: information from a survey of community and hospital laboratories

Rob Lake, Nicola King, Kerry Sexton, Philippa Bridgewater, Donald Campbell

Abstract

Aim To describe and quantify laboratory testing of faecal samples for enteric pathogens as a component of the reporting pyramid of acute gastrointestinal illness (AGI) in New Zealand.

Method Postal survey of community and hospital laboratories throughout New Zealand conducted in mid-2006, requesting data from the 2005 calendar year.

Results Of the 47 laboratories eligible for the survey, responses were received from 35 (74%, 16 hospital laboratories, 12 community laboratories, five hospital and community laboratories, and two Public Health Laboratories). Based on survey data and extrapolation it was estimated that approximately 250,000 faecal samples were received by New Zealand laboratories in 2005. The majority of these (77%) were requested by primary healthcare providers on people in the community. Routine testing of these samples would include bacteria (Salmonella, Shigella, Campylobacter, Yersinia) and parasites (Cryptosporidium, Giardia) and (depending principally on the age of the patient) rotavirus. Testing for other pathogens was comparatively infrequent. The frequency of detection of a pathogen in community samples was estimated as approximately 20%.

Conclusion The positivity rate of 20% for faecal samples from people in the community is consistent with overseas results. Although there was considerable variation in the testing methods employed by the laboratories the methods were considered appropriate based on consultation with ESR Public Health and Reference Laboratory staff. These data on the number and type of samples, and positivity rate, will assist in the determination of a reporting pyramid for AGI in New Zealand.

This survey of community and hospital laboratories was one of the elements of a study of acute gastrointestinal illness (AGI) in New Zealand initiated by the New Zealand Food Safety Authority (NZFSA). The other components were a survey to determine the prevalence of AGI in the community, and an investigation of the incidence of AGI-related visits to General Practitioners (GPs).

The overall objectives of the study were to determine the magnitude and distribution of AGI in the New Zealand population, and describe and estimate the magnitude of under-ascertainment at each stage of the national communicable disease surveillance process. The laboratory survey facet was intended to describe and quantify laboratory testing of faecal samples for enteric pathogens as a component of the reporting pyramid of AGI in New Zealand.
This paper represents a distillation of key data from the full study report, which can be accessed at the NZFSA website: http://www.nzfsa.govt.nz/science/research-projects/index.htm

Method

Through examination of several sources, 45 community and hospital laboratories that performed relevant analyses of faecal samples were identified. Laboratories were contacted by telephone (principally via the laboratory manager) and all except one agreed to participate in the survey. With the addition of the two Institute of Environmental Science and Research Limited (ESR) Public Health Laboratories, the postal survey was sent to 46 laboratories.

The survey instrument was adapted from a questionnaire developed originally by the United States’ Centers for Disease Control and Prevention for Campylobacter. The survey asked questions about: samples numbers and sources; decision making and testing conducted; test methods; pathogen identification rates and further testing; samples and isolate referral; and reporting of results. The source questionnaire was expanded to cover the broader range of pathogens to be covered by the AGI study, as well as additional information such as testing criteria. The information requested on testing methods was reduced to a brief description.

The instrument was pre-tested on ESR and private diagnostic laboratory staff and piloted on five laboratories in May 2006. As a result some questions were slightly amended. The revised survey was sent to the remaining 41 laboratories in June 2006. A reminder letter offering a non-monetary reward (the reward was also sent retrospectively to responding laboratories) was sent to non-responding laboratories on 17 August 2006, which generated one additional response. The survey was declared closed at the end of September 2006. Numerical data in the questionnaire were sought for the calendar year 2005.

Returned surveys were analysed by entry of data into an Excel spreadsheet for calculation of statistics and extrapolated estimates. Additional data on norovirus testing were obtained from the ESR Norovirus Reference Laboratory (NRL), but these data have been excluded from the general analysis and are presented separately where appropriate.

Results

Response—Of the 47 laboratories eligible for the survey, responses were received from 35 (74%). Of these, 16 (46%) were hospital laboratories, 12 (34%) were community laboratories, five (14%) were hospital and community laboratories (i.e. samples were received from both hospital patients and people in the community), and two (6%) were Public Health Laboratories. Geographical coverage was representative with 17/20 laboratories in upper North Island District Health Boards (DHBs) responding, 8/13 from lower North Island DHBs, and 8/11 from South Island DHBs.

Stool specimens—Of the 35 responding laboratories, 34 were able to report the numbers of stool samples received in 2005. The details are shown in Table 1. An additional 356 stool samples were tested by the NRL for norovirus. Many of these were referrals from primary laboratories and so are likely to be included in their total.

The sources of stool samples for the reporting laboratories were calculated by combining reported source percentages from the laboratories, with the sample numbers. This indicated that stool samples were requested by hospital health care professionals (18.6%), primary healthcare providers (77.1%), and other (4.2%, including occupational healthcare providers, public health services, and direct requests from the public).
Table 2. Number of stools received by laboratory type in 2005 as reported by responding laboratories

<table>
<thead>
<tr>
<th>Lab type:</th>
<th>Hospital only</th>
<th>Community only</th>
<th>Hospital &amp; Community</th>
<th>Public Health</th>
<th>All Laboratories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stools</td>
<td>Responses</td>
<td>Total (%)</td>
<td>Mean</td>
<td>Median</td>
<td>Max</td>
</tr>
<tr>
<td></td>
<td>15/16</td>
<td>32,255 (17.5%)</td>
<td>2150</td>
<td>858</td>
<td>11,500</td>
</tr>
<tr>
<td></td>
<td>12/12</td>
<td>134,379 (72.9%)</td>
<td>11,198</td>
<td>5717</td>
<td>56,997</td>
</tr>
<tr>
<td></td>
<td>5/5</td>
<td>16,644 (9.0%)</td>
<td>3329</td>
<td>985</td>
<td>13,162</td>
</tr>
<tr>
<td></td>
<td>2/2</td>
<td>974 (0.5%)</td>
<td>487</td>
<td>487</td>
<td>867</td>
</tr>
<tr>
<td></td>
<td>34/35 (97%)</td>
<td>184,252</td>
<td>5419</td>
<td>2076</td>
<td>56,997</td>
</tr>
</tbody>
</table>

**Testing performed**—Laboratories were asked which pathogen tests would be conducted if the request was for “faecal (or enteric) pathogens”. Thirteen laboratories replied with specifics. For bacteria, all would perform tests for *Salmonella*, *Shigella*, *Campylobacter* and *Yersinia*. Additional routine tests reported were *E. coli* O157 (7/13 laboratories), *Aeromonas* (6/13 laboratories), *Vibrio* (2/13 laboratories) and *Plesiomonas* (2/13 laboratories). For parasites 12/13 laboratories reported testing routinely for *Cryptosporidium* and *Giardia*. Depending on the patient’s age, specimen characteristics, or clinical details, testing for rotavirus would also be performed routinely by all 13 laboratories.

Laboratories were asked about discarding of specimens without testing. The primary reported reasons for discarding a specimen were its age, leakage, problems with labelling, and multiple specimens from the same person within the same period (usually reported as one day). The number of discarded specimens was reported as low. The mean percentage of samples discarded was approximately 2%, although only a few laboratories were able to estimate a percentage.

**Numbers of samples for specific testing**—The number of faecal samples tested by laboratories in 2005 for bacteria, viruses, parasites, and toxins is summarised in Table 2.

Testing for other types of toxins (*S. aureus, B. cereus, C. perfringens*) was reported by only two laboratories, and one of these reported that only 30 samples were tested.

**Testing methods**—The brief descriptions of testing methods for bacterial pathogens revealed considerable variation across laboratories in terms of enrichment, agar and incubation temperatures. None of the methods were considered to be ineffective, but the experience of laboratory workers could be a factor in the recognition of some bacteria. The situation was similar for parasite testing, with a wide variety of methods reported. For ova detection microscopy was the most common method, and this was also reported as the method of choice for some laboratories testing for *Giardia* and *Cryptosporidium*. The experience of laboratory workers would again be a factor in consistent and successful testing.
### Table 3. Number of faecal specimens tested for specific pathogens in 2005

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Responses (of 35 laboratories returning the survey)</th>
<th>No. of faecal samples tested</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salmonella spp.</td>
<td>26</td>
<td>109,332</td>
</tr>
<tr>
<td>Shigella spp.</td>
<td>26</td>
<td>107,981</td>
</tr>
<tr>
<td>Campylobacter spp.</td>
<td>26</td>
<td>108,016</td>
</tr>
<tr>
<td>Yersinia spp.</td>
<td>26</td>
<td>107,858</td>
</tr>
<tr>
<td>Listeria spp.</td>
<td>3</td>
<td>53</td>
</tr>
<tr>
<td>STEC incl. E. coli O157:H7</td>
<td>18</td>
<td>9422</td>
</tr>
<tr>
<td>Aeromonas spp.</td>
<td>14</td>
<td>15,670</td>
</tr>
<tr>
<td>Vibrio spp.</td>
<td>8</td>
<td>7776</td>
</tr>
<tr>
<td>Plesiomonas spp.</td>
<td>13</td>
<td>20,416</td>
</tr>
<tr>
<td>Enterobacter sakazakii</td>
<td>2</td>
<td>764</td>
</tr>
<tr>
<td>Bacillus spp.</td>
<td>2</td>
<td>275</td>
</tr>
<tr>
<td>Clostridium spp.</td>
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<tr>
<td>Staphylococcus aureus</td>
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<tr>
<td><strong>Viruses</strong></td>
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<tr>
<td>Rotavirus</td>
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</tr>
<tr>
<td>Norovirus&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>673</td>
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<tr>
<td><strong>Protozoa/parasite</strong></td>
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<tr>
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<tr>
<td>Cryptosporidium</td>
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<td><strong>Toxins</strong></td>
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<td></td>
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<tr>
<td>C. difficile toxin</td>
<td>17</td>
<td>14,752</td>
</tr>
</tbody>
</table>

<sup>a</sup> Data on norovirus testing provided by the NRL have been incorporated with data from two laboratories that provided responses to the norovirus questions.

The majority of laboratories stored specimens pending further testing. Refrigerated storage (27/31 reporting laboratories) was most common, with only four laboratories reporting storage at ambient temperature. The most common retention time period was one week (19/31 laboratories). Further testing of samples in which no pathogen had been detected by routine screening appeared to be uncommon. Of the responding laboratories, 21/33 reported no further testing, while the 12 remaining laboratories would perform such testing, most frequently at the request of a doctor/clinician or after discussion with Public Health Units. Nevertheless, these laboratories indicated that such further testing was infrequent.

**Non-detection of pathogens**—Laboratories were asked how many samples they received, for which no pathogen was detected. Seventeen laboratories were able to provide percentage estimates, and it appeared that in over two thirds of samples (mean
reported estimate 76.5%, 95%CI 71.2-81.8%; mean based on sample numbers for each responding laboratory: 67.7%) no pathogen was detected. The rate using only data from the eight community laboratories able to provide an estimate (i.e. excluding hospital, and hospital and community laboratories) indicated that in 77.4% (95%CI 73.0-82.8%) of samples from people in the community no pathogen was detected.

Reporting—Healthlink (a computer network) or print-based reporting formats were the most common means of sending results to GPs and Public Health Units. Direct contact (by telephone) between the laboratory and test requestor would occur to clarify testing requirements, or report results of public health significance (e.g. the isolation of pathogens with serious adverse outcomes).

Referral of bacterial isolates for further characterisation—All but one of the responding laboratories (n = 34) reported whether they sent bacterial isolates to the ESR Enteric Reference Laboratory (ERL) for further characterisation (such as speciation and typing). The consistency of referrals varied markedly by bacterial genus: Salmonella (34 laboratories); Shigella (32); Yersinia (17); STEC (30); Vibrio (18); Enterobacter (9); Listeria (2).

Estimated total samples and GP consultations—The total number of faecal samples per year, extrapolated from the mean values in Table 1 to add the missing six hospital, five community, and one hospital and community laboratories suggests that the total number of samples for 2005 may be approximately 250,000.

A previous report\(^1\) indicates that patients (over 5 years of age) with AGI symptoms presenting to GPs in New Zealand are requested to provide stool samples in:

- Less than 25% of cases by 42% of GPs;
- 25–50% of cases by 31% of GPs; and,
- Over 50% of cases by 23% of GPs.

Using the projected 250,000 stool samples submitted in 2005, of which an estimated 77% are derived from primary healthcare providers, who request a sample from an average of 25% of patients, it can be estimated that there were approximately 790,000 GP consultations by people in New Zealand with AGI symptoms in 2005.

Discussion

This survey provides a useful but incomplete picture of faecal sample testing for enteric pathogens in New Zealand. The response rate was lower than anticipated, given that all but one laboratory had agreed to participate when contacted prior to sending the survey. Work pressures and commercial sensitivity about confidential information were cited by some laboratories as reasons for not responding. Devising procedures to protect commercial information may assist in improving response rates in future.

The actual number of samples may be higher than the 250,000 estimate, given the number of community laboratories that did not complete the survey. This estimated total number of samples represents a rate of 0.06 samples per person per year for a New Zealand population of 4,098,900 (based on Statistics New Zealand population estimates for 2005). The majority (estimated 77%) of these samples derive from
primary health care providers, and using the estimate that pathogens were detected in just over 20% of community based samples, this suggests that approximately 45,000 samples have pathogens detected.

Anecdotal comments by laboratory staff suggest that “clearance samples” from ill workers are few. For comparison there were approximately 18,000 notified cases of infection with bacterial and parasitic pathogens in 2005. This excludes cases caused by enteric viruses or C. difficile, which are major causes of acute gastroenteritis, but are not notifiable unless in an outbreak situation or in a person with a “high risk” occupation.

The positivity rate of 20% for faecal samples from people in the community is consistent with overseas results. Community based studies in the Netherlands and the United Kingdom (UK) found bacterial positivity rates of 16% and 19.5% respectively, while the UK overall positivity rate was 24% (includes samples positive for viruses and parasites). A Canadian study in 2001 found that a pathogen (bacteria, parasite, C. difficile, or virus) was identified in 29.4% of stool samples.

Although there was considerable variation in the testing methods employed by the laboratories the methods were considered appropriate, based on consultation with ESR Public Health and Reference Laboratory staff. It is likely that if pathogens are present, then they will occur in high numbers and sensitivity (such as enrichment, for bacterial pathogens) should be less important. Therefore variation in methods may not be a significant factor in detection. However, for several bacterial genera, few laboratories submit isolates to the ERL for further characterisation. This will limit the value that might be obtained by such analyses, such as the ability to identify outbreaks.

The estimate that 25% of patients with AGI are asked to provide a stool sample is similar to other countries, where a range of 14 – 27% of those seeking medical care with an acute diarrhoeal illness are asked to submit a stool sample. The estimate of 790,000 GP consultations by people with AGI should be treated with caution as this survey did not ask laboratories to differentiate between stool samples submitted by patients with AGI and those submitted for other reasons.

These data will assist in the determination of a reporting pyramid for AGI in New Zealand from which a ratio for the number of non-notified cases for every notified AGI case can be estimated.

Competing interests: Philippa Bridgewater was involved in survey and questionnaire design. For reasons of confidentiality, returned surveys were analysed by ESR staff only.

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References:


‘Party pill’ drugs—BZP and TFMPP

Ushtana Antia

Abstract

BZP and TFMPP are amphetamine-like recreational drugs and the major active components of ‘party pills’. The pharmacodynamic effects of these neurally active drugs are thought to be dependent on their activity at DA and 5-HT receptors and several studies report drug-drug interactions at a pharmacodynamic level. Their metabolism involves the hepatic P450 enzymes CYP2D6, CYP1A2 and CYP3A4 resulting in inhibited metabolism of other drugs and medicines, as well as compromised metabolism in poor metabolisers for CYP2D6. Basic pharmacokinetic properties are described for both BZP and TFMPP when taken alone and in combination. Several studies have shown that these drugs cause several drug-drug interactions.

History and use

While the majority of recreational drugs have a history of medicinal use, a group of compounds consisting of piperazine derivatives has been developed specifically for distribution as a recreational drug with psychoactive properties. Capsules or tablets containing these substances were marketed as ‘party pills’ or ‘herbal highs’ (despite the purely synthetic origins) and have been widely used in several countries prompting scrutiny into their safety, and leading to public debate, legislation and scientific enquiry.

BZP and TFMPP have proven central effects that mimic those of other illicit drugs, resulting in their recreational use despite recent legislation in most countries including New Zealand. Information about their action, metabolism, concentrations in the body and interactions (with each other and with other drugs) is vital to clinicians dealing with complications arising from their use and concomitant use of other illicit drugs or medicines, toxicologists and law enforcement agencies wishing to detect them, and pharmacologists seeking to understand the effects of this new class of recreational drug.

BZP and TFMPP are piperazine derivatives which contain a six-membered heterocycle comprised of two nitrogen atoms linked by two ethyl chains (seen in Figure 1). Recreationally used derivatives of this simple ring usually contain a benzyl or phenyl substituent linked to a nitrogen atom on the piperazine group. The benzyl or phenyl group may be further substituted. BZP and TFMPP, illustrated in Figure 1, are the most commonly used compounds of this group of drugs.
BZP was first reported to have amphetamine-like effects in 1972 while TFMPP is said to reproduce the psychedelic effects of MDMA and other empathogenic drugs. The desired subjective effects of ‘party pills’ range from the amphetamine-like effects in BZP-containing ‘energy pills’ (with names like ‘Bolts’ and ‘Charge’) to ‘ecstasy’-type pills (‘Bent’ and ‘d-Lite’) containing both BZP and TFMPP.

Other piperazines have also been used in ‘party pill’ preparations including methoxyphenyl piperazine (MeOPP), fluorophenyl piperazine (pFPP) and chlorophenylpiperazine (mCPP) (Figure 2), among others.

In the USA, BZP was temporarily classified as a Schedule I Controlled Substance in 2002 followed by permanent classification on March 18, 2004. TFMPP was also temporarily classified under Schedule I in 2002 but due to the lack of information about its effect on people the scheduling was not renewed, leaving TFMPP as an uncontrolled substance after March of 2004. The sale of TFMPP is also controlled in Denmark, Sweden, Belgium, Greece and Australia.

Due to a lack of evidence demonstrating the potential for abuse and harm the sale of these compounds was initially restricted to persons aged 18 years and over in New Zealand, however on April 1 2008 they were reclassified as Class C1 drugs by the Misuse of Drugs (Classification of BZP) Amendment Act 2008 (New Zealand), which bans their sale and purchase.

‘Party pills’ are often used in place of stimulants such as methamphetamine and methylenedioxyamphetamine (MDMA or ‘ecstasy’) and are perceived as a safer option. According to an industry estimate in 2005, approximately 150,000 doses of these pills were sold each month in New Zealand. In some cases ‘party pills’ are used in addition to other illicit drugs to enhance the stimulant or euphoric effects of drugs such as methamphetamine and MDMA.
The use of these compounds in New Zealand was significant, with an estimated one in five of the 13 - 45 year old New Zealanders surveyed (20.3 %; n = 2010) having tried ‘party pills’ at least once. The use of ‘party pills’ was found to be higher for males across a number of age groups (13 - 14 year olds, 20 - 24 year olds, 30 - 34 year olds, 35 - 39 year olds and 40 - 45 year olds). In New Zealand, BZP and TFMPP were initially classified under Schedule 4 of the Misuse of Drugs Amendment Act 2005 (an amendment to the Misuse of Drugs Act 1975) as restricted compounds, available for legal sale to any person aged over 18 years.

Pharmacodynamics

Subjective effects—Studies reporting self-administration of BZP suggest that BZP has a high potential for abuse or addiction, which is a feature of many stimulant drugs. For example, in primate studies, rhesus monkeys trained to discriminate between amphetamine and saline selected BZP in preference to saline. Monkeys trained to self-administer cocaine were also found to self-administer BZP. In animal studies, BZP has also been reported as having stimulant effects. Studies using rats trained to discriminate between bupropion (a norepinephrine and dopamine reuptake inhibitor) and saline show self administration of BZP and other CNS stimulants, including d-amphetamine, cocaine, methylphenidate and caffeine.

The stimulant effects of BZP are also supported by human studies. When BZP (20 mg or 50 mg) was administered to human subjects in performance tasks, auditory vigilance improved in comparison to the placebo group, a result shared with subjects administered with d-amphetamine. BZP also rated significantly different from placebo on ‘amphetamine scores’ (amphetamine was used as a positive control in this study) and BZP scored higher than amphetamine on subjective drug liking in former stimulant addicts. However, as individuals with a history of stimulant abuse can sometimes demonstrate withdrawal, which elevates subjective responses to stimulants, the results of studies in former addicts may not be directly comparable to the effects in users without a history of stimulant abuse.

TFMPP’s effects have been rated as between MDMA and other psychedelics such as lysergic acid diethylamide (LSD) or psilocybin. Like many psychedelic drugs, TFMPP does not lead to self-administration in primates. However, rats were found to generalise to TFMPP when trained to discriminate between MDMA and saline, and generalized to MDMA when trained to discriminate between TFMPP and saline. These results explain the inclination to regard ‘party pills’ with high TFMPP content as ‘MDMA-like’.

Mode of action—The pharmacodynamic effects of BZP revolve around its effects on monoaminergic neurotransmitters in particular dopamine (DA), serotonin (5-HT) and noradrenaline (NA).

BZP’s stimulant effects are largely due to its profound effect on dopaminergic neuronal transmission demonstrated by cocaine-like inhibition of dopamine uptake, amphetamine-like dopamine release and agonist activity on postsynaptic dopamine receptors.

BZP also causes the release of 5-HT into the rat nucleus accumbens, and stimulation of the 5-HT1 receptor has been noted while 5-HT2 receptors were not affected.
In addition to its dopaminergic and serotonergic effects, BZP also has noradrenergic effects which is a possible cause of tachycardia and related physiological effects\textsuperscript{24}. TFMPP has been reported to have LSD-like effects, and like LSD has been shown to act on a number of 5-HT receptor subtypes, for example, 5HT\textsubscript{1B}, 5-HT\textsubscript{1C} and 5HT\textsubscript{2} \textsuperscript{25, 26}. In rats, TFMPP acts as an agonist and has behavioural, neuroendocrine and serotonin turnover effects (lowered anxiety, hyperthermia), but displays antagonist activity in the peripheral nervous system\textsuperscript{27}. However this study did not distinguish between the different subtypes of serotonin receptor.

When taken together these drugs are responsible for elevated levels of both DA and 5-HT, an effect similar to that of MDMA. The levels of DA and 5-HT released from a combined dose of BZP and TFMPP are far greater than the individual effects of BZP and TFMPP\textsuperscript{5} suggesting a pharmacodynamic interaction in rats.

**Adverse events**—BZP, like many stimulants, has been anecdotally reported as having a host of physiological effects, for example, tachycardia, elevated systolic and (to some extent) diastolic blood pressure and mydriasis\textsuperscript{1, 2}. More serious effects reported include insomnia, headaches, sweating, hot or cold flushes, nausea, vomiting, anxiety, agitation, confusion, dystonia, palpitations and collapse\textsuperscript{11}, severe adverse effects such as toxic seizures, respiratory acidosis, metabolic acidosis, hyponatraemia\textsuperscript{28}, acute renal failure\textsuperscript{29}, psychosis (in one event of co-administration with cannabis and nitrous oxide)\textsuperscript{30}, and even death (though the circumstances of such cases are often clouded by poly-drug use).

It is important to note that most cases of severe adverse events have only been reported following poly-drug use. Indeed BZP is frequently taken along with other drugs such as alcohol, tobacco, cannabis or amphetamines\textsuperscript{11} therefore the majority of cases reporting serious effects report poly-drug use\textsuperscript{28, 30, 31} or do not test for other drugs and in most cases do not even test for BZP, instead relying on information from self-reported drug use. Two deaths following accidents involving BZP use and one involving TFMPP use were also reported to involve alcohol and / or other drugs\textsuperscript{32}. It is unreasonable to attribute a set of adverse events to BZP use in cases where the use of other drugs are not ruled out.

Full toxicological screening is required, as self reporting for the use of illicit drugs is generally unreliable\textsuperscript{33} and easily biased by the mode of questioning\textsuperscript{34}. A more recent study has found that BZP concentrations correlate to seizure frequency, while co-ingestion of alcohol reduces seizure frequency but increase confusion and agitation\textsuperscript{35}.

In summary, the physical effects that can reliably be attributed to BZP (in the absence of interfering or interacting substances) are limited to stimulant-like effects such as tachycardia and elevated blood pressure\textsuperscript{16} and higher plasma levels of BZP are associated with seizures\textsuperscript{35}. More severe symptoms only arise (as is expected) after concomitant use of other illicit drugs.

**Metabolism**

Knowledge about the metabolism of BZP and TFMPP can provide useful information about the pharmacology and potential toxicity of these substances. Research about the metabolism of BZP in rats has indicated that while a number of metabolites may be possible, little metabolism was observed. It has been proposed that the hydroxylation
and dealkylation pathways catalysed by enzymes of the cytochrome P450 families may produce the majority of BZP metabolites \(^{36,37}\). TFMP metabolism occurs to a small extent, and has been found to occur primarily through the action of enzymes belonging to the cytochrome P450 (CYP) family. Of these enzymes, CYP2D6, CYP1A2 and CYP3A4 are thought to be involved in TFMP metabolism \(^{38}\).

The metabolism of BZP and TFMP in rats \(^{39}\) provided insight into probable routes for their metabolism in humans. By using rats as a model for human CYP2D6 metaboliser status (female Dark Agouti, male Dark Agouti and male Wistar rats as poor, intermediate and extensive metaboliser models, respectively), CYP2D1 (the rat orthologue of human CYP2D6) was claimed to be the principal enzyme responsible for the metabolism of TFMP, accounting for 80.9% of TFMP metabolism in rats. CYP1A2 and CYP3A4 also contributed to the metabolism, but to a lesser extent, 11.5% and 7.6% respectively \(^{40}\). It was proposed that TFMP metabolism occurs via hydroxylation of the phenyl group and dealkylation of the piperazine ring. Subsequent degradation, acetylation and conjugation (glucuronation and sulfonation) can result in a number of metabolites \(^{39}\).

BZP metabolism in rats is reported to be limited and thought to occur primarily through the action of the same three isoenzymes of the cytochrome P450 family. The specific enzymes responsible have not been confirmed but it has been proposed that BZP metabolism in humans also occurs via aryl-hydroxylation and N-dealkylation \(^{36,41}\) again by the P450 enzymes implicated in TFMP metabolism (namely CYP2D6, CYP1A2 and CYP3A4). Since BZP and TFMP are structurally and chemically similar, parallels have been drawn between the metabolic routes of the two compounds. As with TFMP, further metabolism of hydroxylated or dealkylated BZP can result in a range of metabolites \(^{36}\).

BZP and TFMP are metabolised by CYP2D6, CYP1A2 and CYP3A4 in the human liver \(^{38}\). The metabolism of BZP and TFMP has been shown to be adversely affected by the presence of the inhibitors of these enzymes and other substrates. It has also been shown unsurprisingly that these enzymes have different affinities for TFMP and BZP. An important concern (also raised by Staack \(^{40}\) for TFMP metabolism) is that of compromised metabolism of TFMP and BZP in ‘poor metabolisers’ demonstrating low levels of CYP2D6.

In a clinical context, the interaction between BZP and/or TFMP and therapeutic drugs is a justified concern that should be further investigated. There is a potential for BZP and TFMP to interfere with a number of commonly used medications such as paroxetine—a selective serotonin reuptake inhibitor and CYP2D6 substrate, olanzapine—an antipsychotic and CYP1A2 substrate, and carbamazepine—an anticonvulsant and CYP3A4 substrate.

**Pharmacokinetics**

An early attempt to study the pharmacokinetic properties of ‘party pill’ drugs in humans \(^{42}\) was terminated prematurely due to adverse events. The design of this study was responsible for its failure to provide meaningful results and for its early termination. \(^{43,44}\) Specifically, the concomitant use of BZP and TFMP with alcohol is explicitly discouraged on the packaging of most ‘party pills’ but in this research participants were given large doses of BZP (300 mg) and TFMP (74 mg) in addition
to alcohol (57.6 g / 6 units), which is further evidence of the role of poly-drug use in the high incidence of adverse events. Furthermore, the co-administration of all three drugs meant that the investigators could not attribute the adverse effects to any one of these drugs. In addition to this flaw in study design the purity of the drugs was not ascertained and commercially available ‘party pills’ were used instead of pure drugs.

As a study into the contents of commercially available ‘party pills’ has shown, the content of these preparations can vary significantly from the dosage and even the ingredients listed on the packaging. Ten of the twelve preparations studied in this study demonstrated differences from 62% to 133% of the stated amount of drug. Successful pharmacokinetic studies in humans, with well-defined criteria to protect the participants and prevent adverse events have been conducted since.

**BZP**—BZP reached its maximum plasma concentration ($C_{\text{max}}$) of 262 ng/mL 75 minutes after a single 200 mg oral dose of BZP alone. The elimination half-life for BZP was 5.5 hours and clearance (Cl/F) was 99 L/h. Additionally, plasma concentrations of 4-OH BZP and 3-OH BZP respectively were found to peak at 7 ng/mL ($T_{\text{max}} = 60$ min) and 13 ng/mL ($T_{\text{max}} = 75$ min) in this study. Urinary metabolites were also reported, specifically an N-sulphate conjugate of BZP and an O-sulfate conjugate of its hydroxylated metabolites, in addition to 4-OH BZP and 3-OH BZP.

**TFMPP**—Plasma concentrations of TFMPP following a single 60 mg oral dose peaked at 24 ng/mL ($T_{\text{max}} = 90$ minutes). TFMPP had two disposition phases with calculated half lives of 2 hours and 6 hours, with Cl/F of 384 L/hour. A single plasma metabolite, 4-OH TFMPP ($C_{\text{max}} = 20$ ng/mL; $T_{\text{max}} = 90$ min), was detected in this study. Urinary metabolites included 4-OH TFMPP and an N-glucuronide of TFMPP, with some evidence of conjugates of 4-OH TFMPP.

**Tissue distribution of BZP and TFMPP**—The fact that these drugs have observable effects on mood and behaviour suggests that they cross the blood-brain barrier. Therefore a disparity should be exhibited between plasma and brain tissue concentrations. For example, concentrations of methamphetamine in plasma do not reflect those found in the brain. A study of the tissue distribution of BZP and TFMPP has also noted a significant difference in the amounts of distribution of these drugs in the rat.

The organ with the highest concentration of BZP was the kidneys with a concentration ratio between the plasma and kidneys of approximately 1:20, while the TFMPP concentration ratio between the plasma and the lungs (organ with the highest TFMPP concentration) has a ten-fold difference at approximately 1:200, thirty minutes after the dose. This study reported that the ratios of BZP and TFMPP between plasma and all other analysed tissue (brain, liver, kidneys, lungs, heart) were 1:40 and 1:385 respectively, thirty minutes after the dose. Therefore the presence of a more obvious distribution phase in the human plasma profile of TFMPP when compared to BZP is in agreement with tissue distribution data from the rat.

**Concentration-dependent subjective effects**—A positive relationship has been reported between plasma drug concentrations and subjective ratings indicating that both BZP and TFMPP have concentration-dependent subjective effects suggesting
that elevated concentrations of these drugs (due to compromised clearance or larger doses) may result in elevated effects on mood \(^ {51}\).

As TFMPP and BZP do not persist in plasma for longer than 24 hours, these results also suggest that subjective effects of these drugs should last no longer than 24 hours at the given dose. However it is important to note that the drug effects are not the same for every individual, with a minority demonstrating the opposite relationship between concentration and effect.

Conversely, reports from animal studies\(^5\) have indicated that the subjective effects of these drugs are synergised when they are co-administered. This suggests that the interaction resulting in synergism between these drugs occurs at a pharmacodynamic level. This further suggests that by combining BZP and TFMPP, the doses of each can be reduced without compromising the effect of the drugs which may explain why, when these drugs are sold in combined drug preparations, the doses of each drug are routinely far lower than in the single drug preparations.

**Drug-drug interactions**

**Inhibition of P450 enzymes by BZP and TFMPP**—Studies have implicated hepatic P450 enzymes, namely CYP2D6, CYP1A2 and CYP3A4, in the metabolism of BZP and TFMPP \(^ {38,40,41}\). With many prescription medicines being metabolised by P450 enzymes, adverse events resulting in the compromised clearance of a prescription drug can manifest as either toxic overdose or therapeutic failure (in the case of pro-drugs). It has been shown that BZP and TFMPP inhibit the metabolism of several P450 isoenzymes.\(^ {38,52}\)

Both BZP and TFMPP were found to inhibit the metabolism of dextromethorphan, caffeine, ethinylestradiol and tolbutamide, probe substrates for CYP2D6, CYP1A2, CYP3A4 and CYP2C9 respectively, thus potential drug-drug interactions may be predicted. The inhibitory effect of BZP and TFMPP on these enzymes is of clinical importance because the enzymes studied are involved in the metabolism of many commonly used prescription and over-the-counter drugs. While clinicians attempt to prevent drug-drug interactions, these interactions might still occur in patients using but failing to disclose the use of ‘party pill’ drugs.

**Metabolic interactions between BZP and TFMPP**—It has been shown that BZP and TFMPP are metabolised by the same enzymes, namely CYP2D6, CYP1A2 and CYP3A4.\(^ {38}\) Therefore, their co-administration is likely to result in compromised metabolism of one or both of these compounds. Evidence of an interaction is both relevant and concerning because nearly 30% of ‘party pill’ preparations contain BZP and TFMPP in combination. Indeed, this study also reported a significant inhibition of the metabolism of BZP in the presence of TFMPP and vice versa.

**Pharmacokinetic interactions between TFMPP and BZP**—Pharmacokinetic interactions have been demonstrated between related piperazines and other recreational drugs. Mixtures containing cocaine and the piperazine analogue, mCPP, have been reported in the Netherlands. In a single case report the urinary concentrations of the major metabolite of mCPP were drastically reduced, allegedly due to drug-drug interactions with cocaine, a known CYP2D6 inhibitor.\(^ {53}\) In the ‘party
scene’, it is known that piperazines are taken in combination with caffeine (a CYP1A2 substrate) in the form of energy drinks and sodas.

It is known that elevated caffeine levels in the body can lead to caffeine intoxication; a range of symptoms from restlessness to gastrointestinal disturbance to rapid heartbeat and psychomotor agitation. Deaths resulting from extreme overdose of caffeine have been reported.\textsuperscript{54-56} PK interactions leading to elevated caffeine concentrations are a potentially lethal consequence of combining BZP and/or TFMPP with caffeine.

Other recreational drugs have been shown to cause clinically important interactions with prescription medicines. For example, an episode of severe and prolonged effects following a small dose of MDMA by an individual under ritonavir and saquinavir treatment for HIV-1 infection has been reported.\textsuperscript{57} This effect was linked to P450 inhibition (specifically CYP2D6 and CYP3A4 inhibition) by the antiretroviral drugs, leading to reduced clearance of MDMA. Due to the similarities in the metabolism of MDMA and the piperazine-based drugs, similar adverse effects might be observed in cases where antiretroviral drugs or other potent P450 inhibitors are co-administered with ‘party pill’ drugs.

As previously mentioned, the co-administration of BZP and TFMPP has been reported to lead to elevated pharmacodynamic effects in the rat as mentioned earlier. An interaction study has compared the pharmacokinetic properties of BZP and TFMPP after a combined oral dose (100 mg of BZP plus 30 mg of TFMPP) with their individual pharmacokinetics.\textsuperscript{58} Some differences in the pharmacokinetic properties of TFMPP were noted, specifically, the $C_{\text{max}}$ of TFMPP was 28 ng/mL, reached 75 minutes after the dose, with an elimination half life of 2.3 hours and no detectable plasma metabolites. This result showed some significant differences to the pharmacokinetic properties of TFMPP alone as demonstrated by Antia et al.\textsuperscript{47} Meanwhile, BZP reached peak plasma concentrations of 295 ng/mL at 60 minutes, with an elimination half-life of 4.3 hours and only one plasma metabolite, 4-OH BZP. Apart from the loss of the metabolite 3-OH BZP, there were no significant changes to the pharmacokinetics of BZP when compared to those of BZP alone.\textsuperscript{46}

Interactions between BZP and TFMPP were not found to affect the $T_{\text{max}}$ and $t_{1/2}$ of either drug but the difference in lag time of TFMPP between the single and combined doses is significant. In the single dose of TFMPP a lag time of 30 minutes is observed, however for the combined dose, this lag time is not evident. It has been suggested that BZP (due to localised effects) stimulates stomach emptying may explain an absence of a lag time for TFMPP in the combined dose.\textsuperscript{58}

A limitation on the interpretation of the results of this study is the difference in dose between the single and combined treatments. Ideally, if the same doses of TFMPP were given for the single and combined treatments, the effect of inhibiting the elimination phase would be that the plasma concentrations of TFMPP would increase (i.e. $C_{\text{max}}$ would be higher in the combined treatment). This effect is suggested by the equivalence of $C_{\text{max}}$ between the two groups, despite the combined treatment group taking only half the dose of TFMPP. While this was not as robust as a direct comparison at the same doses, it gave ethical justification for halving the dose used in
combination. Using the same dose would almost certainly have resulted in an elevated $C_{\text{max}}$ and potentially led to adverse events. Interestingly, one subject has reported experiencing different subjective effects at different doses of BZP (50 mg, 100 mg and 200 mg).\textsuperscript{51}

A further finding from the human pharmacokinetic interaction study is the loss of one hydroxylated metabolite of each drug, 3-OH BZP and 4-OH TFMPP. This study has implicated elevated levels of Phase II metabolites of BZP and TFMPP in the combined dose compared to the single dose, suggesting that while metabolism by CYP enzymes may be reduced, the clearance of the drug is not affected due to the increase in alternative metabolites, namely the N-sulphate of BZP and the N-glucuronide of TFMPP.\textsuperscript{59} The results from the interaction study suggest that the majority of the interactions between BZP and TFMPP are metabolic, lending support to claims that these drugs are metabolised by the same P450 isoenzymes.

**Poor metaboliser status**—Several P450 enzymes are highly polymorphic, resulting in individuals with either higher or lower levels of an enzyme, leading to significant differences in the clearance of drugs metabolised by those enzymes.

One of enzymes implicated in the metabolism of BZP and TFMPP, CYP2D6, is known to be polymorphic with up to 14 \% of Caucasians demonstrating compromised metabolism of substrates of this enzyme.\textsuperscript{60} Studies have implied that the CYP2D6 status of an individual could have a significant impact on their metabolism of TFMPP.\textsuperscript{38} It has been reported that while there was no significant differences in the pharmacokinetics of BZP in poor and extensive metabolisers, a significant increase in the $C_{\text{max}}$ of TFMPP has been reported in a CYP2D6 PM compared to a population of extensive metabolisers\textsuperscript{59}.

Furthermore, 4-OH TFMPP (a metabolite detected in all other participants) was absent in all plasma samples of the PM. This is in agreement with in vitro data that implicates CYP2D6 in the metabolism of TFMPP and shows compromised metabolism in CYP2D6 PMs. The limitations of this work include that only phenotypic poor metaboliser for CYP2D6 was used, and the phenotype of the population of extensive metabolisers was not confirmed.

**Discussions**

In New Zealand, the use of ‘party pill’ drugs is likely to continue despite regulations banning their sale because there were reports of stockpiling of BZP-containing ‘party pills’ immediately prior to the change in the regulation of these drugs in April 2008.\textsuperscript{61} Non-disclosure of illicit drug use is a well-documented phenomenon\textsuperscript{33, 34} and could be a potential concern for clinicians prescribing medicines that might interact with ‘party pill’ drugs or those dealing with emergency room admissions caused by ‘party pills’. Furthermore, with the criminalisation of BZP and TFMPP there is also a potential need for simple and rapid techniques that allow the routine testing for these drugs similar to those used for amphetamines in urine, saliva and sweat.\textsuperscript{62}

The metabolism of BZP and TFMPP has been reported to be catalysed by P450 enzymes CYP2D6, CYP1A2 and CYP3A4. In turn, BZP and TFMPP inhibit substrates of these enzymes. In addition to drug-drug interactions, the metabolism and pharmacokinetic properties of these drugs can be affected by enzyme polymorphisms.
Pharmacokinetic studies on BZP and TFMPP demonstrate that both drugs reach their maximum concentrations more than one hour after an oral dose. If the maximum drug effects only occur one hour after the dose is taken it is possible that users will re-administer the dose within the first hour in an effort to achieve the desired effect. This practice could significantly increase the incidence of adverse events because users could underestimate the resulting drug effects.

Furthermore, the presence of a distinct distribution phase for TFMPP, and clear proof of distribution of both BZP and TFMPP in animal models raises the issue of concentrations of these drugs in the brain. While the plasma concentrations of these drugs provide important information pertaining to how long they persist in the body, the concentration of these drugs (and their metabolites) in the brain is potentially more useful for establishing a relationship between the pharmacokinetic and pharmacodynamic properties.

Drug-drug interactions affect the success of therapy when prescription drugs are involved and increase the likelihood of adverse events. The metabolism of BZP and TFMPP in humans has been shown to occur via three enzymes which are known to metabolise a range of medicines. This suggests that BZP and TFMPP are likely to interact with the majority of prescription and many recreational drugs, potentially leading to adverse effects due to compromised drug clearance. Furthermore, the concentrations of BZP and TFMPP, and subsequently the onset, duration and size of their effects could be altered by their combined use as well as the metaboliser status of CYP2D6, CYP1A2 and CYP3A4 phenotypes of the user. This phenomenon is clearly seen in the elevated TFMPP concentrations in a CYP2D6 PM individual and has a high potential for clinically relevant adverse events.

Pharmacokinetic interactions with drugs that have a small therapeutic window can lead to severe adverse events. Furthermore, pharmacodynamic interactions with psychoactive drugs such as amphetamines or methylphenidate (Ritalin) could potentially cause dramatic effects. As many recreational drugs are combined in order to enhance their subjective effects, these interactions could potentially be responsible for a number of the reported adverse events reported following ‘party pill’ use.

Caffeine and alcohol are also co-administered, often at large doses, with BZP and/or TFMPP and can have effects on the absorption, metabolism and elimination of these drugs. Smoking status may also have a role in interaction studies due to the effects of smoking on induction of CYP1A2. Furthermore, interactions may exist between different ‘party pill’ constituents. While pharmacokinetic interactions between BZP and TFMPP have been investigated, there are several other piperazine-based drugs with similar metabolic pathways and inhibitory effects on P450 enzymes. In vitro human liver microsomal assays have reported potential metabolic interactions with analogues of BZP and TFMPP and other drugs and medicines.

At this stage no information is available in order to assist with better clinical outcomes following adverse reactions to BZP and TFMPP. As the self-reporting of illicit drug use is unreliable, users may underestimate both the number of drugs used and the dosage in order to avoid persecution, resulting in clinicians in emergency departments having to work with incomplete or false information, posing problems in dealing with hospitalisations due to adverse drug events.

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What is the mechanism of sudden infant deaths associated with co-sleeping?

Christine G McIntosh, Shirley L Tonkin, Alistair J Gunn

Abstract

The risk of Sudden Infant Death Syndrome (SIDS) has fallen dramatically in the “Back to Sleep” era; however, half the cases now occur when the infant has been sleeping in bed with another person. Despite the association of SIDS with co-sleeping, parents are receiving mixed messages. It is often presumed that co-sleeping deaths are due to ‘overlaying’, when the adult rolls on top of the baby, stopping baby from breathing. We examine research that shows that it is not necessary to cover the face, or squash the body of a baby to restrict or prevent breathing and cause oxygen deprivation. At birth, the temporo-mandibular joint is not yet fully formed, and thus the jaw can be easily displaced upwards and backwards pushing the tongue into the upper airway to form a partial or complete block of the airway. Indeed, this can happen with firm flexion of the infant’s head so that the chin pushes against its own chest. Further research is needed, but on present evidence, all parents should be advised to sleep their baby in a cot or similar next to their parent’s bed, until baby is at least 6 months of age.

Unexplained infant death and co-sleeping

Recent coroners’ reports and related media publicity in New Zealand have drawn attention to the significant number of young infants who still die while sharing a bed with their parents or others. For example, a recent Wellington coroner’s inquest examined the cases of seven babies who died suddenly and unexpectedly and found that four died while sharing a bed. The remaining cases involved other known unsafe sleeping environments, including prone sleeping, v-shaped pillows, and loose covers over the face. The associated case summaries make an apparent presumption that the adults probably had rolled upon the infants during the night—i.e. ‘overlaying’. However, although this may be the cause of some deaths, there is little direct evidence in most cases. Other potential contributing factors include covering the infant’s face with the adult’s blanket or duvet, rebreathing, thermal stress, or splinting of the infants chest by the adult’s arm.

Based on close examination of the circumstances of similar deaths with co-sleeping, and of the anatomy and physiology of infants in the very narrow age range of these deaths, we would like to propose that there may be another, more subtle explanation for some cases of SIDS occurring while co-sleeping.

In the present discussion paper, the terms co-sleeping and bed-sharing are used interchangeably to describe a baby sharing the same sleep surface with another person at times when the adult is asleep, however, technically bed-sharing may used to refer
to when a baby is brought into an adult bed for feeding or settling without the intention of sleeping.5

The epidemiology of sudden infant death syndrome (SIDS) has changed dramatically in some respects since the 1991 *Back to Sleep* campaign. In the UK, along with a remarkable fall in the risk of SIDS, the proportion of children dying while co-sleeping with an adult has risen from 12% to 50%.6 Other changes included an increase in the proportion of SIDS associated with financially deprived families (from 47% to 74%), maternal smoking in pregnancy (57% to 86%), and pre-term infants (from 12% to 34%).6 This is consistent with data in other countries.4,7

The same is true in New Zealand. For example, as highlighted by the 2005 Auckland Coroners Report, 19 sudden infant deaths were classified as “not due to sepsis or birth defects” (obscure natural causes).8 Of these, 12 were sleeping with another person, 10 of these were with their parents, and 2 were with siblings; 9 of these 12 were in double beds.

It is noteworthy that even amongst the other deaths attributed to “infection” 6 were co-sleeping at the time they died. Thus of a total of 39 dead babies, 18 were sleeping with a person who did not wake to their distress although with them in bed at the time. Similarly, in the recent Wellington Coroners report on 7 unexplained infant deaths: “All either slept with their babies or had slept them on their tummies or on pillows where they had been found face down or partly covered in a blanket”.1,2

**Why is co-sleeping dangerous for sleeping babies?**

Sudden Infant Death has several features that are unique. The age range is surprisingly limited; it peaks at 2 months of age and is rare after 6 months.9 This remains true for co-sleeping infants; indeed the large UK case-control Avon study suggested that the age of infants dying of SIDS while bed-sharing has fallen (from a median of 88 days before 1992, to 54 days subsequently).6 Further, autopsies suggest an asphyxial death, frequently showing pulmonary oedema and petechial haemorrhages in internal organs.3,10

Although the mechanisms contributing to SIDS in general are incompletely understood, there is evidence that in part it may be related to a brainstem abnormality in the neuroregulation of cardiorespiratory control, with independent reports of nonspecific gliosis in the brainstem of infants dying of SIDS and of abnormalities of serotonin activity in the medulla oblongata, as comprehensively reviewed by Kinney and colleagues.11 Presumptively, such an inherent susceptibility must interact with other intrinsic and external risk factors.

In particular, the narrow age of vulnerability suggests that whatever the risk factors are the infant is able to ‘grow out of them’—i.e. there must be an anatomical or physiological predisposition which resolves with age. Given the magnitude of the difference in size between an adult and even say a 6-month infant, it seems unlikely that mere growth of the baby would be enough to explain this very narrow range.

There is increasing evidence for anatomical factors.12 At birth, the infant head is very large (third to quarter of total length). The neck is short with the small chin almost touching the chest. The toothless mandible is flat, without any vertical ramus, and is attached to the skull by a loose capsule (as shown in Figure 1).
Within the mandible the relatively large infant tongue fills the oral space between the soft palate and the gums. Stark and Thach have shown in newborn infants that pressure on the jaw from neck flexion, submental pressure, or mandibular pressure from face masks caused backwards displacement of the jaw, leading to obstruction of the airway.\textsuperscript{13,14}

**Figure 1. The mandible and its articulation at different ages (reproduced with permission from Tonkin et al\textsuperscript{12})**

(a) Infant skull showing the surface tympanic membrane and the horizontal mandible. The mandible is mobile and can be pushed up and backwards by any pressure, towards the tympanic membrane.
(b) Adult skull and mandible. The mature head of the mandible and the mandibular fossa, which restrict its backward movement, are displayed.

The increased incidence of oxygen desaturation in premature infants placed in semi-upright infant car seats compared with lying in cots\textsuperscript{15,16} has been shown to be associated, at least in part, with flexion of the head on the body and marked narrowing of the upper airway on respiration-timed upper airway X-rays.\textsuperscript{16} Although most of these observations were made in preterm infants, there is evidence that the similar albeit lesser frequency of oxygen desaturations in term infants restrained in car seats\textsuperscript{17–19} may also be related to forward slumping of the head on to the chest and consequent pressure on the chin.\textsuperscript{20} Indeed, even at term, displacement of the jaw with oxygen desaturation can be obtained by any pressure on the chin, including forward flexion of the infant’s head onto its own chest in some cases.\textsuperscript{21}
An example of a baby with a very narrow upper airway when the mother pressed on her baby’s chin is shown in Figure 2. We have seen a number of infants up to 5 months old who were referred to the Auckland Cot Monitor Service by the Auckland Starship Children’s Hospital after major, unexplained apnoeic episodes in circumstances where detailed reconstruction of the scene and events suggested that the lower jaw was being pressed upon whilst they were asleep. For example, we have reported eight cases of apparent life-threatening events in young full term infants restrained in car safety seats who were admitted to hospital, and found to be otherwise normal.19

Figure 2. Inspiratory-timed lateral neck X-rays

![Left, an example of a normal upper airway in a young infant. Right, an infant whose mother has pressed gently on the chin with a finger. Note the marked narrowing of the upper airway due to upwards and backwards displacement of the jaw.]

Scene reconstruction showed that while asleep in their car safety seats these infants' heads flexed forward, so that their chins pressed down onto their chests. This position was associated with intercostal recession on inspiration in all cases, consistent with the hypothesis that this position was associated with upper airway restriction.19 Further, we have shown that a simple foam insert that allows a baby’s head to rest upright, in a neutral position, while restrained in a car seat was associated with improved oxygen saturations in preterm and young term infants,16,20 and with improved upper airway size as shown by lateral timed radiographs.16

Upper airway compromise and SIDS during co-sleeping

These findings suggest the hypothesis that co-sleeping could lead to SIDS through inadvertent pressure on the infant’s jaw, for example if the mother, or other person, unknowingly pressed their body or limb against the baby’s chin. Alternatively, if baby was sleeping with the adult’s arm under its head or if baby was placed higher up on
the bed with its head on a pillow, there could be pressure from the pillow that would tend to flex baby’s head forward, again leading to pressure on the jaw. In New Zealand as in much of the developed world adult beds are often markedly softer than infant cots—the weight of the adult will cause depressions in a bed where a baby may be forced into a flexed position.

This hypothesis is consistent with the known epidemiology of SIDS in general, and particularly of that described above with SIDS during co-sleeping. First, the youngest infants, with the least mature temporo-mandibular joints, are at highest risk. Second, not all cases are associated with apparent drug use or alcohol intake; even in those cases, the danger may be not from overt overlaying but from the adult being less responsive to direct contact with baby or to movement by the baby. Next, it is noteworthy that backwards displacement of the jaw leads to obstruction of the pharynx, and thus to reduced air flow, and so no sounds can be made. This is consistent with the lack of reported outcry by infants who die of SIDS.12

What other factors could be involved?

Smoking is highly prevalent in cases of SIDS and occurred in the majority of cases in the UK Avon case-control study.6 Nicotine exposure reduces hypoxic arousal,22 and so speculatively might augment susceptibility due to underlying brainstem abnormalities.11 Critically, however, it may be argued that without blocking of the airway the babies would not have needed to respond in the first place. Consistent with this, the association between co-sleeping and SIDS remains after controlling for maternal smoking.23

Unusual sleeping circumstances such as shared sleeping on a couch are associated with a high rate of SIDS.23 However, in absolute terms this is still rather uncommon and the great majority of co-sleeping SIDS cases both internationally and in New Zealand occur in the family bed.23 We also cannot rule out a possible role for other factors such as covering of the head by bedding, which is also highly associated with SIDS.3

In one study, covering appeared to precede death, as shown by sweat on the face of the infant, and so may well be causally related to the death,3 presumably due to obstruction of the external airway leading to hypoxia and rebreathing (carbon dioxide build up). However, the infants in that study were older than average, which is not consistent with the known younger age in infants who die of SIDS while co-sleeping.6

Why weren’t these cases sleeping separately?

The most likely reasons for co-sleeping in the SIDS cases reported in New Zealand include: a lack of appreciation of the importance of sleeping baby on a separate sleep surface, a cultural preference for co-sleeping, and an association with financial deprivation of families that makes purchase of a cot for only a few months’ use challenging for parents.

The message that young babies should sleep in the same room as their parents but on a separate sleep surface has been weakened by advocacy for bed-sharing to promote bonding and breastfeeding.24 Since breastfeeding is associated with many positive benefits, this is an important issue. Nevertheless, before giving advice we must be
mindful that there is no compelling evidence that it is ever possible for a young infant to share its bed with a sleeping adult in absolute safety.

Thus, in New Zealand it is recommended that baby should sleep in its own space, beside the parents’ bed.5 There are now programmes that lend cradles to parents, from SIDS New Zealand, Pregnancy Help, Moe Ora, and some maternity hospitals. Although further research is clearly needed, anecdotally, the authors believe that with the assistance of these initiatives effective breastfeeding can be achieved without baby being left to sleep with the adult, in the adult bed.

Alternatively, one proposed option to help baby sleep safely next to mother is the wahakura (flax cradle) developed by Dr David Tipene-Leach that can be placed on the parents’ bed. The wahakura may help provide a safe sleeping place with easy access for breastfeeding, while at the same time baby would be protected from any pressure on the face. The wahakura have not yet been the subject of research to confirm their safety, and the relative effect of these different strategies on breastfeeding success is not known.

Summary

The safest way for an infant to sleep is on its back, on its own sleep surface, but in the same room as its parent. There must be no head flexion, covering of the face, or pressure on the lower jaw.

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Will the financial crisis get under the skin and affect our health? Learning from the past to predict the future

Tony Blakely, Melissa McLeod

What will be the impact of the 2008–09 global financial crisis and consequent recession on the health of New Zealanders? In this viewpoint article we attempt to predict this health impact by reflecting on the lessons from the economic and social changes of the 1980s and 1990s in New Zealand, and using an epidemiological framework to discuss: what exposures or determinants of health (e.g. unemployment) are changing as a result of the financial crisis, and which social groups exposures are having the largest changes?; what health outcomes are these exposures likely to affect?; and what contextual factors (e.g. background disease rates) might influence these effects?

How might the current economic recession get under the skin?

The media and editorial columns of journals have been awash with coverage and anticipation of the impacts of the global financial crisis.1,2 No country will go untouched by the economic recession. However the relative impact on employment, housing, education, nutrition, mortality, child health, mental health, and violence will vary greatly according to an individual countries economic capacity, initial level of inequality, and the availability of social, medical and public health services.3

A major concern for middle and low-income countries is the impact on development aid from rich countries, and rich individuals, reducing donations.1,2 In this paper, we focus on New Zealand and three key knock-on effects of the financial crisis that in turn may have health impacts: unemployment; reduced incomes for some individuals and families (be that due to business income reducing, unemployment, or reduced investment income); and a reduced rate of increase in government funding of the health system in part due to reduced tax revenue.

There are no simple answers to what drives improvements in mortality. All of macroeconomic factors (e.g. GDP4), social factors (e.g. education and income5,7), behaviour (e.g. smoking, diet7,8), and health services9 make some contribution, but the exact contribution of each factor depends on context (e.g. background disease burden, history of migration) and many interactions.10–17 Economic recessions present one example of a ‘shock’ to the economic and societal system that may impact on health status, mortality, and life expectancy.

Internationally, downward trends in mortality have continued in the face of economic recession,18,19 and sometimes mortality declines are actually accelerated in economically difficult times.20 The recent failure of the ex-Soviet states has been the notable exception, with dramatic worsening of life expectancy accompanying the social upheaval and economic collapse.21–23

In New Zealand, there has been a general trend towards improved life expectancy for Māori and non-Māori since the 1950s (Figure 1). However, improvements in life
expectancy have not occurred in every decade. For example, non-Māori male life expectancy was stagnant in the 1960s and 1970s coinciding with the peak in the ischaemic heart disease mortality epidemic.

Importantly for the current paper, Māori life expectancy stagnated in the 1980s and early 1990s whilst non-Māori life expectancy showed strong increases. The structural reforms of the 1980s and 1990s, and in particular the high unemployment rates that peaked in 1991–92 at 25% for Māori compared to 8% for European, almost certainly contributed to the divergence of Māori and non-Māori life expectancy trends in the 1980s and 1990s as shown in Figure 1.

Figure 1. Life expectancy trends since 1950 for Māori and non-Māori

Unemployment—The unemployment rate at the end of the March quarter for 2009 had inched up to 5.6% (Household Labour Force Survey), with unemployment more concentrated among youth (e.g. 19.1% among 15–19 year olds), Pacific and Māori (13.1% and 11.2%), and those living in Northland (8.5%) and Auckland region (6.5%). (People who reported more than one ethnic group are counted once in each group reported.) This means that the total number of responses for all ethnic groups

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can be greater than the total number of people who stated their ethnicities. The unemployment rate is projected to increase to 7.2% in the March 2010 quarter (New Zealand Institute of Economic Research June 2009 forecast, http://www.nzier.org.nz/Site/Publications/Consensus_forecasts.aspx), less than the current OECD average of 7.8% (which is expected to increase further by 2010). It is not clear which industries will be hit hardest by the projected increases in unemployment rates. However, in times of high unemployment, ethnic minority groups and low socioeconomic groups are inevitably most at risk of losing employment. These groups (as well as those unemployed prior to the recession) are also less likely to gain employment when the job market is flooded with relatively more educated potential employees.

To summarise thus far, the unemployment rate is projected to reach levels two thirds of those in the early 1990s, and the burden is again likely to be unevenly distributed across ethnic groups.

But does being or becoming unemployed damage your health? Yes. In the published literature, unemployment has been associated with increased self harm and suicide and decreased mental health status. From New Zealand we have cohort study evidence of elevated suicide rates during 1991–94 among those unemployed at the 1991 Census, and increased rates of self-harm among those made unemployed during freezing work factory closures.

A well-designed Swedish record linkage study adjusted thoroughly for early life events, and found a 50% increase in mortality rates for the four years following unemployment (for a period of 3 or more months) during the 1992–94 recession. A recent meta-analysis of 87 longitudinal studies found a moderately strong association of being made unemployed with deteriorating mental health, and vice versa re-employment being associated with improvements in mental health.

An econometric analysis for 26 European countries has just been published. This study was designed to predict the possible health impacts of the current financial crisis by assessing the mortality impact of rapid rises in unemployment from 1970 to 2007. They found that a 1% rise in unemployment was associated with a 0.79% (95% confidence interval 0.16 to 1.42) increase in suicide rates in those aged less than 65 years, a 0.79% (0.06 to 1.52) rise in homicide deaths, a 1.39% (0.62% to 2.14%) decrease in road traffic crash deaths, and no discernible impact on overall mortality.

Of particular importance, this study also found evidence that the association of changes in unemployment rates with suicide was less in countries that invested more in active labour market programmes (e.g. labour market training, special youth programmes, transition from school to work programmes).

**Income**—Individual or household income is widely thought to be a major determinant of health. In the current environment, there is uncertainty and stress for individuals around retaining employment, the continued viability of businesses and the safety of investments. Employment loss and to a lesser degree the reduction in income from those who remain in employment will force households to cut expenditure.

Of particular importance to public health is the reduced ability of households to afford nutritious foods and in some cases to afford food at all (food insecurity). In New Zealand, the proportion of people living in households who do not have a regular source of sufficient food has increased from 10% in 1977 to 30% in 2007.
Zealand, a lack of food security is higher in those of Pacific or Maori ethnicity, for larger households and those of lower socioeconomic status. Another manifestation of reduced household income will be an increase in household crowding, or a shift to lower quality but more affordable accommodation. This is of particular concern for Maori, Pacific and low socioeconomic groups who are already more likely to live in overcrowded accommodation. Household crowding is associated with an increased risk of a number of infectious diseases, and possibly with mental health, and violence.

Reduced household income also has implications for the affordability of healthcare, including the ability to pay part charges for primary care visits and prescriptions, but also the costs of over the counter medicines, transport and time off work to attend appointments. In parallel, if co-payments for healthcare increase (e.g. due to reduced Government funding), this may further reduce income among marginalised groups.

However, there are inter-related reasons why, among those who do not become unemployed, the health impacts via income in the forthcoming economic recession may not be too severe. First, people losing income from investments may have generally higher incomes, and the association of change in income with change in health is much weaker at high incomes (i.e. diminishing marginal returns).

Second, people reliant on investment income are often retired, and have a superannuation entitlement (generous by international standards at two thirds of the average wage) to fall back on. Third, and perhaps most importantly of all, the global and New Zealand sharemarkets started to bounce back in 2009. For example, the NZX50 since March this year has clawed back nearly 50% of its loss in value since September 2008.

Reduced funding for Vote:Health—Government new spending on Vote:Health in the last 10 years or more has increased annually by an amount well in excess of either inflation or the average annual increase in Government spending. With the current financial crisis, reduced tax revenue, Government priorities on restraining expenditure growth in Health and improving productivity, these rates of annual increase in funding will diminish—if not even be zero or decreasing after allowing for inflation and additional health care inflation adjustment. That is, health services may need to contract or be delivered in new ways. The impact on peoples’ health from such stasis or even contraction will depend on which services are affected, and how access is affected.

The current National-led Government recently announced a number of changes to the New Zealand health system including the development of a National Health Board and Shared Services Establishment Board (21 October 2009) following the recommendations of the Ministerial Review Group (i.e. Horn Report). These changes signal an increased and explicit focus on prioritisation of new investment, and disinvestment. It is important that any prioritisation considers equity, not just efficiency.

What about context?

We have already noted how in recent econometric analyses the association of unemployment rates with suicide rates seems to vary with the level of each country’s
social spending. What contextual factors that dampen or exaggerate the health impacts of the financial crisis might be important this time around?

First, there have been significant changes to the health system since the 1980–1990s, not least of which included a significant restructure of primary care services. The Primary Health Care Strategy 2001 set a new direction for primary care with the establishment of Primary Health Organisations. These organisations receive capitation funding to work with local communities, reduce health inequalities, provide co-ordinated care and improve access to quality primary care services for their enrolled populations.

Such changes to primary care should place us in a better position to manage the impacts of the economic recession. However, despite major increases in funding to primary care, there remain disparities in access to primary care by both ethnicity and socioeconomic status, which risk being exacerbated in the current financial crisis.

Second, the health impacts depend on what diseases are prevalent in society. Of particular importance, 1–74 year old cardiovascular disease mortality has fallen by a staggering two thirds for European/Other in the last 25 years, and 40–45% for Māori. Cardiovascular disease deaths were probably an important mechanism whereby ethnic inequalities in mortality widened in the 1980s and 1990s.

The decreased prevalence of cardiovascular disease may naturally constrain the total mortality effects of unemployment in the current economic crisis. However, other diseases such as mental illness and suicide remain prevalent in New Zealand society, and detectable impacts on cause specific morbidity and mortality through these diseases are likely.

Finally, as demonstrated in the 1980–1990s, policies both within and outside of the health sector have important modifying impacts on health. Cut-backs in policy around social services, education and housing seem extremely unwise.

**Our best estimate … and policy recommendations**

The current global economic crisis will have health consequences for the New Zealand population. As in the 1980s and 1990s the distribution of health impacts are likely to be concentrated in those who are already socioeconomically deprived, and in ethnic minority groups. Increases in suicide rates are likely; these will be amplified if primary and mental health services were weakened due to parallel funding restraints. Short-term morbidity from mental illness, infectious diseases, and acute incidents of cardiovascular disease seem likely to increase.

However, it seems unlikely that any detectable aberration to the long-term decline in total mortality will occur that can be attributed to the financial crisis and consequent unemployment. And road traffic crash mortality may even decrease faster with the ‘assistance’ of recession and other manifestations of the crisis. The long-term impacts on other causes of morbidity and mortality are very difficult to anticipate, due to uncertainty about changes (if any) in the prevalence of mediating risk factors such as smoking, nutrition (food insecurity aside) and alcohol that may arise from competing influences of stress, reduced income, unemployment, job insecurity, increased active transport, and such like.
It may be that the just announced restructuring of the New Zealand health system, and reduced future Vote:Health funding, precipitated in large part by the global financial crisis, will be the mechanism by which health status is most impacted. It will take time to determine if this is so.

In the prioritisation of publicly funded services, and to monitor and reduce the impacts of the economic recession on health, a prudent list of policy recommendations includes:

- Protecting and maintaining the quality of, and improving access to publicly funded health services in the face of funding constraints, particularly for Māori, Pacific, and low income people and communities
- Valuing the contribution of prevention activities, many of which are highly cost-effective and designed to improve equity (e.g. tobacco control, in particular tobacco taxation)
- Monitoring of unemployment and health indicators by ethnicity and deprivation (e.g. using cross-sectional and longitudinal survey data to monitor (changing) mental health of those made unemployed, and their (changing) access to health services)
- Vigilance of primary and mental health services to people at risk of mental health and cardiovascular consequences of unemployment—including possible explicit inclusion of ‘unemployment’ as a predictive factor in risk assessment.
- Investment by the state in social spending that reduces the amount of unemployment, and in social spending that assists those made unemployed (e.g. bridge back to work programmes).

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References:
Climate science, denial and the Declaration of Delhi

George Laking, Alistair Woodward, Scott Metcalfe, Alexandra Macmillan, Graeme Lindsay, Joanna Santa Barbara, Anne MacLennan, Imogen Thompson, Susan Wells; for OraTaiao: New Zealand Climate and Health

Abstract

Human-induced climate change is now the central health issue facing humanity. The World Medical Association recently adopted the Declaration of Delhi, committing the medical profession to mitigate and adapt to the effects of climate change. This is new professional territory for many doctors. Even so, the profession has often engaged with issues outside ‘the health sector’ when the stakes are high, for example leaded petrol, road safety, tobacco, and nuclear weapons.

The scientific basis to the declaration merits scrutiny in light of commonly used contrary arguments. Decisions in medicine, as elsewhere, must be taken on the evidence to hand, weighing up the risks, given that complete knowledge is seldom available and time is precious. There are strong analogies between clinical experience and our approach to planetary climate.

The relevant context for scientific observations on climate is the world’s multi-gigatonne annual CO₂-equivalent greenhouse gas emissions. Emissions drive changes in concentrations of greenhouse gases, which matters when they are rapid or prolonged. The current variation in global temperature is alarming, even when within ‘normal range’. Climate models inform and guide present-day decision-making, and perform well in explaining observed warming. They corroborate other evidence that tells us that CO₂ and other greenhouse gases are harmful at current atmospheric concentrations.

As a profession and as global citizens, we need to move beyond dissent and denial about anthropogenic climate change. The WMA correctly says that circumstances now require us all to take action.

Ten years into the third millennium, climate change looms as the central issue facing humanity’s collective future. It should be no surprise that climate change now appears in our professional domain.¹ ²

Most of us will have attested at the start of our career to something like the World Medical Association (WMA) Declaration of Geneva, ³ which begins

“ISOLENLY PLEDGE to consecrate my life to the service of humanity”.

Although our day-to-day focus as clinicians is more on individual humanity, the pledge requires us to also consider the collective wellbeing of populations over time. That wellbeing strongly depends on climate and the environment, and so in October 2009 the World Medical Association adopted the Declaration of Delhi, “to provide a response ... to the challenges imposed on health and healthcare systems by climate change”⁴.
The Delhi Declaration begins by noting the most likely effects predicted by the Intergovernmental Panel on Climate Change (IPCC, AR4). The IPCC has “very high confidence” (its code for “at least a 9 out of 10 chance of being correct”) that climate change currently contributes to the global burden of disease and premature death. These and related effects are “projected to progressively increase in all countries and regions”.

With this in mind, the WMA has committed the medical profession to actions to mitigate and adapt to the effects of climate change. These are summarised as advocacy, leadership, education and capacity building, surveillance and research, and collaboration. The WMA Declaration of Delhi is a global call to action for the entire medical community, and our colleagues are responding internationally.

Most readers of this Journal devoted their education to what was viewed as ‘the Health Sector’. We did not anticipate our interest in human welfare would require a good understanding of geophysics and politics. So it is understandable if many of us feel outside our professional comfort zone when we consider how the medical profession should respond to climate change.

Many times in the past the profession has engaged with issues outside the health sector when the stakes for health were high. Road safety, tobacco and nuclear weapons are three recent examples.

But climate is new territory, and many doctors will seek to increase their understanding of the background, particularly when dissenting voices are heard in the public media. Here we explore the scientific basis to the Declaration of Delhi, with reference to commonly used contrary arguments.

**Due personal verification**

The first challenge to the Declaration of Delhi is whether we should accept it. After all, the earlier Geneva Declaration on the Duties of Physicians enjoins that:

“A PHYSICIAN SHALL certify only that which he/she has personally verified”, and consequently we have had scepticism and resistance to *ex-cathedra* statements drummed into us from Day One of medical school. Scepticism means asking questions, not taking matters on face value, and not being swayed by authority unless we decide independently there is good reason to act.

But true scepticism does not mean refusing to act in the absence of certainty. Doctors are well aware that decisions must be taken on the evidence to hand, weighing up the risks, on the basis that complete knowledge is seldom available and time is precious. Our patients’ well-being often demands we act on the basis of incomplete and emerging information.

What applies at the bedside is true also in the public domain. Our experience of medical controversy reminds us that science is frequently disputed, particularly when commercial interests are at stake. Think back, for example, to the arguments over the effects of lead on child health. For the better part of a century a few prominent scientists, supported by industry, advanced doubt as reason to delay removal of lead from paint and petrol. In recent history, we as a profession have taken a stand on similar controversial issues, on the grounds that the evidence may not be perfect, but
still sufficient for action. Examples include passive smoking, immunisations, and cardiovascular risk. The Declaration of Delhi puts the profession in such a position now with climate change.

It is fair to ask how we as doctors might personally verify anthropogenic global warming, especially in the absence of our preferred evidentiary tool, the randomised controlled trial. The evidence mainly resides in a single incomplete case report, the geophysical history of planet Earth. A very short supporting case series is suggested by our neighbours Venus and Mars. Clearly verification must take a different form to how we would approach a controversy over drug therapy or food supplements.

**Some contrary arguments**

Arguments commonly cited against climate change were mooted in an open letter written in 2007 by “100 Prominent Scientists” to the Secretary-General of the United Nations. This group claimed that recent observations of phenomena such as glacial retreats, sea-level rise and the migration of temperature-sensitive species are not evidence for abnormal climate change, “for none of these changes has been shown to lie outside the bounds of known natural variability”.

The claim did not specify a time-scale, which means that the “bounds of natural variability” were potentially very wide. For example, the characteristic sea level for the planet, based on evidence from the last 500 million years, is about 100 metres higher than the present day. Just because something falls within the bounds of known natural variability, does not mean it is desirable.

In clinical practice, variation of physiological parameters within a reference range may often be cause for concern. For example, in the case of human temperature, 700 recordings from 148 healthy subjects varied between 35.6°C and 38.2°C, being a 2.6°C (7%) variation with a mean of 36.8°C and 37.7°C an upper limit of normal. Doctors commonly see rising temperatures within the normal range, and must judge whether this is “natural variability” or the first flicker of something more serious.

Another example is weight change. A 5 kg fall in a patient’s weight might be within the bounds of normal, if it occurred over the course of a year. If the weight were lost over a month, we would tend to consider a potentially serious explanation. It is often the rate as much as the magnitude of change that is important.

The 100 Scientists also noted a recent apparent lull in global warming, saying that despite computer projections of temperature rises, there has been no net global warming since 1998. “That the current temperature plateau follows a late 20th-century period of warming is consistent with the continuation today of natural multi-decadal or millennial climate cycling.”

Such arguments fail to see the wood for the trees, in this case by picking an arbitrary short series of just the last 10 years and an abnormally warm single year (1998) as the starting reference point. Clinically this is akin to taking false reassurance from an isolated set of ‘good’ laboratory values (a false-negative), when looking at all results over time would show a more serious evolving pattern. The climate change deniers have used data selectively, where the IPCC has assiduously used all available data to properly compare pre- and post-industrial trends. The most recent global data,
released by the World Meteorological Organisation, show that 2000-2009 has been the warmest decade ever since direct measurements began.\textsuperscript{17}

The 100 Scientists concluded that “it is not possible to stop climate change, a natural phenomenon that has affected humanity through the ages.” This insistence on an explanation in terms of natural phenomena is akin to ‘diagnostic anchoring’, in which data not compatible with a starting diagnostic assumption are systematically excluded from consideration.\textsuperscript{18} We know in medicine that signs of pathology may often be modified or masked, so that ‘natural appearances’ become misleading. For example, factors such as age or treatment with corticosteroids or partially effective antibiotics may attenuate the fever response.

The same applies to the short-term climate trend, which may be damped down or reinforced by factors such as the El Nino Southern Oscillation (the periodic change in atmospheric conditions around the Pacific affecting rainfall patterns and temperatures world-wide).\textsuperscript{19} Longer-term temperature increases are also being artificially masked by aerosols in the atmosphere.

In medicine, we have to recognise the underlying true febrile process even when it may be modified by other factors. Similarly, we have to recognise the climate's complexity when interpreting apparent lulls in global warming and likely future warming trends.

In the end, the most decisive tool for interpreting change tends not to be rate or magnitude but rather context. If the person whose temperature has just been recorded as 37.7°C is a possibly neutropenic oncology patient with malaise, the temperature should at the very least be rechecked within an hour. It would be a brave doctor who failed to act on a rising temperature in this context, simply on the grounds that it was still within the normal range.

If a person lost 5kg of weight over a month in the setting of an intensive diet and exercise programme, this might be explicable. But if that weight were lost in the context of fevers and night sweats, there would be a strong suspicion of something more sinister.

Although the analogy between clinical practice and planetary climate is necessarily imperfect, it reminds us to look for context. The relevant context for scientific observations on climate is the world’s approximate 50 gigatonne annual CO\textsubscript{2}-equivalent greenhouse gas emissions.\textsuperscript{16}

\textbf{Airborne aetiology}

At the heart of the 100 Scientists’ position lies a very simple idea, that “… carbon dioxide (CO\textsubscript{2}), [is] a non-polluting gas that is essential to plant photosynthesis.” This blanket claim (that CO\textsubscript{2} is non-polluting) must raise a red flag to doctors, conditioned as we are by Paracelsus’ dictum that

“\textit{All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy}”.\textsuperscript{20}

CO\textsubscript{2} and other greenhouse gases become increasingly polluting at elevated concentrations. By analogy, in the clinical setting, electrolytes such as potassium are
vital at normal concentrations, but quickly become life-threatening when concentrations rise above normal.

With this in mind we review in detail the global ‘greenhouse effect’, and the part played by CO$_2$. (We are indebted in the following account to the work of Held and Soden, who have documented progress in understanding of greenhouse gases over the last century.)

In 1827, Fourier described the heat-trapping ability of the atmosphere, which functions in essence as a one-way filter. Visible frequencies of sunlight are transmitted freely and warm the Earth. The return journey as radiated heat is blocked because the atmosphere at these frequencies is highly absorbent.

In 1861, Tyndall discovered that atmospheric heat is absorbed mainly by water vapour and CO$_2$, trace gases making up less than 1% by weight. From his laboratory observations, he concluded that water vapour acted as “a blanket, more necessary to the vegetable life of England than clothing is to man”. At the levels described by Tyndall, CO$_2$ is indeed a non-polluting gas essential to photosynthesis (albeit that when plants are stressed, including by heat, they photosynthesise less and move more to respiratory metabolism; such a potential further positive feedback mechanism bodes poorly for the future climate).

Observational data suggesting that the climate could warm in response to atmospheric CO$_2$ were first published by Arrhenius at the end of the 19th century. The effect does not need to be strong; like compound interest on a loan it simply needs to persist over time.

In 1905, Chamberlin made a key interpretative advance in writing that water vapour,

“... confessedly the greatest thermal absorbent in the atmosphere, is dependent on temperature for its amount, and if another agent, as CO$_2$, not so dependent, raises the temperature of the surface, it calls into function a certain amount of water vapour which further absorbs heat, raises the temperature and calls forth more vapour ...”.

Simply put, warm air holds more water as vapour before it starts to rain. In the early 1960s, data from the first Venus probes suggested that a water-mediated greenhouse effect had overtaken that planet’s atmosphere. Since that time, positive feedback between water and greenhouse gases such as CO$_2$ has been a central element in models of global warming. More recent experiments from NASA’s Aqua satellite have confirmed this link. The observed increase in atmospheric heat absorption per degree rise in temperature is approximately 2.04 W/m$^2$/K.

**Predictive models work**

It is true that greenhouse gases such as CO$_2$ and water are but one part of a much larger and complex global system. Climate modellers face a significant technical challenge when they attempt to estimate greenhouse gas-mediated temperature gains in the future. But it is important to note the modellers do not claim to foretell what will happen in the future. Instead, they offer models as a guide to what may happen, based on best efforts to understand causal relations and complexity.
The climate models used in the IPCC AR4 were tested against the conditions of the last 40 years and produced backcasts that fitted closely with observations at global and regional levels.\textsuperscript{16} As Held and Soden wrote in 2000,

“… it is useful to watch an animation of the output of such a model, starting from an isothermal state of rest with no water vapour in the atmosphere and then ‘turning on the sun,’ seeing the jet stream develop and spin off cyclones and anticyclones with statistics that closely resemble those observed, watching the Southeast Asian monsoon form in the summer, and in more recent models, seeing El Niño events develop spontaneously in the Pacific Ocean.”\textsuperscript{21}

Doctors are familiar with this kind of uncertainty. We are constantly required to make forecasts, judging risk and prognosis on a daily basis. We regularly use algorithms,\textsuperscript{27} imperfect as they may be, to assess risk and individualise clinical decision-making.

Cardiovascular risk is one such example. Quantitative risk models cannot of course predict the date on which an individual patient will suffer a heart attack or stroke. But an algorithm based on age, gender, current blood pressure, lipid profiles, diabetes, and smoking history, expressed as a simple colour chart or computer programme, provides an invaluable guide to decisions about treatment. This has been validated in New Zealand through a back casting exercise, demonstrating how well the model could ‘predict’\textsuperscript{28} results from previous epidemiological studies.\textsuperscript{28–30}

But unsurprisingly, if your starting position is to categorically deny that CO\textsubscript{2} or other greenhouse gases can trap heat in the atmosphere, it is unlikely your models will predict human-induced global warming.

**The science demands action**

Due scepticism has an important role to play, particularly in fields such as evidence based medicine that are complex and contested. It is essential though to distinguish between appropriate scepticism and counterproductive ‘denialism’. Denialism includes the use of rhetorical arguments, at times selective and influenced by economic interests beyond the science, \textit{inter alia},\textsuperscript{15} to give the impression of legitimate argument where there is none. There are common patterns in the tactics employed by the tobacco industry in its beat-up of ‘the smoking controversy’, those who deny that HIV causes AIDS, and the climate change ‘sceptics’ (see Diethelm & McKee 2009\textsuperscript{15} for more detail). This kind of denial is dangerous, and must be questioned diligently.

Diligent questioning has also been leading scientists to update the latest IPCC assessment, which is now more than two years old. The science in this area is fast-moving. There is mounting evidence the IPCC predictions may have been too conservative, where more recent comprehensive reviews\textsuperscript{31–33} indicate that climate change is proceeding at or beyond the upper projections of the 2007 IPCC assessment.

This is not a criticism of IPCC models, so much as an ongoing refinement process, similar to updating a medical diagnosis and prognosis as evidence accumulates. The purpose of climate models is not to foretell the future, but to inform and guide present-day decision-making in light of future possibilities.
The climate observations, IPCC models, and physical mechanisms are consistent; the mechanisms mooted are plausible and can be demonstrated experimentally at the correct scale; and the process can be observed in real life and reproduced by models. There is no plausible alternative explanation that passes these basic tests.

In summary, there are more than sufficient grounds to reject the arguments of the 100 Scientists\textsuperscript{12} and kindred Climate Change Deniers.\textsuperscript{15} Change is not necessarily normal: small changes can matter especially when they are rapid and cumulative, and variation within normal ranges (e.g. variation in either absolute temperature or rates of change of temperature) is not always benign; the context of greenhouse gases makes such variation alarming. There is ample evidence the climate models perform well. CO\textsubscript{2} and other greenhouse gases are causing substantial climate change at current industrial atmospheric concentrations, and will continue to harm unless these concentrations are reduced.

Good medicine recognises risk and urgency and is willing to act on presumptive diagnoses and emerging yet incomplete information. The warnings of the IPCC are stark, and have serious implications for health workers throughout the world.\textsuperscript{2}

As Sir Austin Bradford-Hill pointed out,\textsuperscript{2} scientific work is inherently incomplete and uncertain, and yet we are required to use such knowledge as we have and to act now in the face of uncertainty. Uncertainty is not in itself a reason to postpone or avoid action.

New Zealand adopted this precautionary principle when we signed up to the Rio Convention after the Earth Summit of 1992. In the health arena, New Zealand has acted on this principle in its response to epidemic Group B meningococcal disease. In the absence of trials of vaccine efficacy, we undertook safety trials and implemented a vaccination programme, as the most ethical option.\textsuperscript{34} In contrast, we have watched helplessly while HIV-AIDS deniers seriously retarded work on that illness in Africa, causing much increased mortality and morbidity.\textsuperscript{15,35}

The same principles hold true whether the scale of action be clinical or global. As Peter Doherty (the Nobel Prize-winning Australian immunologist) says, paralleling climate science with biomedical research:

“This experiment, which involves 6.8 billion human beings, as well as every other complex life form on our small planet, can only be done once. Can we afford to explore the extent of its possibilities? I fail to comprehend how any competent scientist could argue that our current strategies are sustainable. Comparable intimations of disaster during for instance the testing of a new drug would lead to the immediate termination of the trial.”

As a profession and as global citizens, we need to move beyond dissent and denial. We were able to do this for lead, tobacco and immunisations. As reasonable as it is to verify the evidence on climate change, circumstances now require us all to take action.\textsuperscript{1,2,4,6–9,37–47}

\textbf{Competing interests:} This paper is authored by individual health professionals belonging to, and on behalf of, OraTaiao: New Zealand Climate and Health (\texttt{www.nzchg.webs.com}). Professor Alistair Woodward was a member of the writing teams that prepared the fourth (AR4)\textsuperscript{48} and earlier assessment reports for the IPCC (2004–07 and before).
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Rectus sheath haematoma: an anticoagulation dilemma

Giovanni Losco, Samba Siva Reddy Pulusu, Murray Pfeifer

Abstract
Rectus sheath haematoma (RSH) is an uncommon condition with a propensity for difficult and mistaken diagnosis. We describe a case where management was complicated by the patient’s requirement for anticoagulation.

Case report
An 85-year-old man was admitted to hospital with an 18-hour history of abdominal pain and lower abdominal distension. He had been found at home in a state of collapse. Paramedics had treated him at home for hypotension, by the administration of intravenous therapy, before bringing him to hospital.

On admission, he gave a history of having noticed a small swelling in the right iliac fossa a day before presentation, which had increased in size on the day of admission. A recent pneumonia had resulted in persistent coughing. His relevant past history included atrial fibrillation for which he was on warfarin, pulmonary embolism, and deep vein thrombosis, several years earlier.

On examination in the Emergency Department, he was noted to be haemodynamically stable. Abdominal examination revealed right lower quadrant swelling, tenderness and guarding. The admission haemoglobin was 145 and international normalised ratio (INR) was 4.1. Computed tomography (CT) of the abdomen showed an abdominal wall haematoma, lying within the rectus sheath (Figure 1).
In consultation with the local haematology service, warfarin was stopped and INR reversed with vitamin K and Prothrombinex-VF (CSL Limited, Broadmeadows, Victoria, Australia). The INR normalised to 1.0 and there was clinical improvement with stable abdominal girth and haemoglobin. Four days after admission the patient became hypoxic. CT pulmonary angiography showed multiple pulmonary emboli. His abdominal symptoms had stabilised, so he was commenced on treatment dose low molecular weight heparin.

Four days later he had a recurrence of abdominal pain and an increase in abdominal girth, indicating further bleeding. Initially the decision was made to continue heparin due to the significance of his pulmonary emboli. Two days later, his pain and girth worsened and, in consultation with medical colleagues, his heparin was stopped. Unfortunately he rapidly deteriorated and died as a result of pulmonary embolism.

**Discussion**

RSH results from damage to the superior or inferior epigastric arteries or their branches or from direct tear of the rectus abdominis muscle. The immediate cause of rupture may be external trauma to the abdominal wall, iatrogenic trauma from surgery, excessively vigorous contractions of the rectus abdominis muscle often seen in strenuous exercise or repeated valsala with severe coughing, vomiting or straining at stool. Anticoagulation, especially heparin injection, has been described as an important aetiological factor.\(^1\)

RSH can mimic any acute abdominal condition and consequently has a high rate of misdiagnosis.\(^2\) One study found an incidence of RSH of 1.8% among 1257 patients admitted to hospital with abdominal pain.\(^3\) Usually self-limiting, it can cause hypovolaemic shock and death. Historical data shows overall mortality is around 4% but increases to 25% in patients who are anticoagulated.\(^4\) A number of recent case reports suggest that the frequency and severity of cases is increasing, and that anticoagulation is playing a more prevalent role (6 out of 7 in one case series).\(^5\)

Abdominal pain is the most common symptom but anorexia, nausea, vomiting, diarrhoea, constipation, tenesmus, and bladder irritability can also manifest. Abdominal examination usually reveals a palpable, painful, firm, non-pulsatile mass corresponding to the rectus sheath. The mass does not move with respiration. Ultrasound and CT scanning are commonly used to help confirm the diagnosis.\(^3,6\)

Conservative management with rest, analgesia, compression, ice packs, reversal of anticoagulation, and transfusion is successful in the majority of cases.\(^7\) Definitive management with angiography and embolisation of the bleeding vessel\(^8\) or open surgery with clot evacuation, ligation of the bleeding vessel and closed-suction drainage should be considered in selected patients who fail or are unsuitable for conservative management. This includes those with enlarging haematomata, haemodynamic instability despite resuscitation, signs of peritonitis, inadequate pain control or persistent gastrointestinal or urinary symptoms.

Our case presented some challenging issues. Despite a past history of venous thromboembolism (VTE) several years earlier, the patient’s indication for anticoagulation on admission to hospital was stroke prophylaxis in atrial fibrillation.
In the presence of clinically significant bleeding where a warfarin-induced coagulopathy is considered a contributing factor, current Australasian guidelines recommend aggressive warfarin reversal using vitamin K and Prothrombinex, with or without fresh frozen plasma.

In our patient this regimen was highly effective until the unexpected finding of VTE created a new dilemma. Clinically this was the most life-threatening pathology and as the patient’s abdomen had stabilised, we decided anticoagulation was appropriate with expectant management of his RSH. Unfortunately, he succumbed to his VTE despite treatment and although there was concurrent deterioration in the RSH, intervention was not considered for this given his worsening respiratory condition.

This case highlights the difficulty that can arise in managing patients with RSH and a requirement for anticoagulation. Our patient profile is known to have a high mortality. Involvement of the local haematology service as well as surgeons and interventional radiologists where appropriate is recommended.

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References:
Incarceration of an umbilical hernia following colonoscopy

Maxine M Beetham, M Imran Khan

Abstract

We describe the unique case of a patient who developed incarceration of a loop of small bowel in an umbilical hernia following an uneventful diagnostic colonoscopy. It was treated with laparotomy, with release of the incarcerated bowel and closure of the defect with nylon sutures.

Case report

A 72-year-old lady underwent a diagnostic colonoscopy for iron deficiency anaemia. Her background history included a small para-umbilical hernia which had been present for many years. The colonoscopy was a routine procedure carried out under intravenous sedation. The colonoscope was passed round to the caecum with some sigmoid looping but once through there, it passed easily into the caecum. A circumferential tumour was identified in the proximal transverse colon which was able to be negotiated and biopsied. She was discharged home post procedure but was readmitted the next morning with a history of abdominal pain and vomiting after her discharge from hospital. On examination she was afebrile with stable vital signs. Her abdomen was tender with guarding. The umbilical hernia was noted to be swollen, painful and irreducible. Bowel sounds were present. She had neutrophilic leucocytosis.

Pain abdominal X-rays showed distended loops of small bowels compatible with small bowel obstruction. Computerised tomography of the abdomen (Figure 1) showed a strangulated and obstructed loop of small bowel in the para-umbilical hernia.

Figure 1
The patient underwent laparotomy with release of the incarcerated, but viable loop of small bowel. The umbilical defect was closed with interrupted nylon sutures. Biopsies taken at the time of colonoscopy confirmed adenocarcinoma of the colon. She later went forward for elective right hemicolectomy which was an uncomplicated procedure with an uneventful recovery.

**Discussion**

Colonoscopy is now accepted as the gold standard procedure for investigations of the colon.\(^1\) Its safety profile has improved considerably over time with advances in technology.\(^2\) However, as it is an invasive procedure, it carries a risk of complications. Bleeding and perforations rates vary widely in literature but are on average less than 0.3% to 0.1% respectively for diagnostic colonoscopy.\(^3\) The complication rates of therapeutic colonoscopy are about twice that of the diagnostic procedure.\(^3\)

Rare complications include splenic injury,\(^4\) the development of small bowel obstruction from internal hernia following a colonoscopy,\(^5\) and incarceration of the scope in an inguinal hernia.\(^6\)

A literature search including MEDLINE and MESH showed no prior report of incarceration of small bowel in an umbilical hernia after colonoscopy, making this the only documented case of an incarcerated abdominal wall hernia reported post colonoscopy. It is unlikely to be a coincidence as the patient had the umbilical hernia for many years without any complications and her symptoms started soon after discharge from the hospital following the procedure.

The likely mechanism is perhaps an increase in intra-abdominal pressure following air distension of the colon during colonoscopy forcing the small bowel into the hernia and compromising its blood supply.

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Hypereosinophilic syndrome with lung involvement

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Hypereosinophilic syndrome (HES) refers to a group of leucoproliferative disorders characterised by an overproduction of eosinophils that leads to organ damage. HES occurs at any age, though most cases occur between 20–50 years of age. Most cases of HES frequently affect the lungs, heart, and gastrointestinal tract, although a few cases of peripheral vascular involvement have been reported. The recognised causes of eosinophilia are allergy and asthma, infections like parasitic, fungal, interstitial lung disease-like histiocytosis X, sarcoidosis, collagen vascular diseases, malignancy (including non-Hodgkin’s lymphoma, myeloblastic leukaemia, and non-small cell carcinoma of the lung), pulmonary eosinophilic syndromes like tropical pulmonary eosinophilia and Churg-Strauss syndrome, and chronic eosinophilic pneumonia.

A 10-year-old boy admitted to our hospital with dyspnoea for 10 days. The medical history was unrevealing. Laboratory values disclosed a white cell count 56,000mm$^{-3}$, and in the differential white cell count 47% eosinophils. Bronchoalveolar lavage data revealed marked eosinophilia (34%). Chest X-ray examination showed symmetric bilateral alveolo-interstitial infiltrates in the central parts of the lungs (Figure 1).

Figure 1. Chest X-ray showing symmetric bilateral alveolo-interstitial infiltrates in the central parts of the lungs
Computed tomography confirmed dense patchy alveolar shadows and interstitial infiltrates (Figures 2a,b).

Based on the clinical picture, radiological findings, and laboratory results, the probable diagnosis was HES with lung involvement.

There was complete resolution of all signs and symptoms followed corticosteroid therapy.

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References:
The quality of life of New Zealand doctors and medical students: what can be done to avoid burnout?

Marcus A Henning, Susan J Hawken, Andrew G Hill

Abstract
Life as a doctor or medical student poses particular challenges and stressors which can impact on quality of life. This paper sets out to review what is known about the quality of life of doctors and medical students and the ramifications of a poor quality of life. This paper summarises the national and international literature on what is known about quality of life and burnout with regards to both medical students and doctors in terms of the origin of these issues and various risk factors. This paper further recommends ways of addressing these issues from an undergraduate level, for doctors in practice, and then in the workplace. It is critical that the New Zealand medical workforce addresses these issues in a timely manner. In addition, the paper proposes that if doctors, particularly those involved as clinical teachers, have a poor quality of life, the learning environment for medical students may be adversely affected. Exploration of the evidence around these important issues and their relevance to the New Zealand context are considered with suggested solutions.

Background
Quality of life can be defined in many ways. Dimenäs et al\(^1\) suggested that there are three areas related to the notion of quality of life, namely subjective well-being, health and welfare. Subjective well-being refers to a person’s perception of their life situation. Health can be defined in objective and subjective terms and imply an evaluation of physical and mental status. Welfare is a measure of environmental factors.

The World Health Organization (WHO) has developed a definition of quality of life that includes aspects of physical, mental, and social well being.\(^2\) Quality of life is measured in terms of an individual’s perception and level of satisfaction about their life. Other factors include culture, values, goals, expectations, standards and concerns. As such, the WHO Quality of Life (WHOQOL) survey incorporates areas of physical health, psychological state, level of independence, social relations, personal beliefs, and environmental characteristics.\(^2\) Table 1 below illustrates the domains and facets used in the measurement of quality of life according to the Australian version of the WHOQOL-100, the most detailed version of the WHOQOL series.

Quality of life and burnout are integrally linked.\(^3\)–\(^5\) The increasing rates of burnout and poor quality of life for doctors has potentially serious ramifications for doctors’ lives and patient care.\(^6\) One study has suggested that physicians “have long considered burnout (i.e. emotional exhaustion, depersonalisation, and decreased feelings of personal accomplishment) to be an occupational hazard” and that this sense of disempowerment is linked with the notion of quality of life, such that physicians in the US have a higher risk of depression, substance abuse and anxiety compared with
the general population. Eckleberry-Hunt et al have argued that several factors can influence lowered quality of life and lead to burnout symptoms. These include lack of social support, depression, disempowerment and sleep deprivation. It is thus likely that burnout and quality of life indicators may measure very similar entities related to the notion of ‘wellness’. In this model, burnout is viewed as being at the extreme end of quality of life issues going awry.

### Table 1. Domains and facets of the Australian version of the WHOQOL-100

<table>
<thead>
<tr>
<th>Domain</th>
<th>Facet</th>
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<tbody>
<tr>
<td>Physical health</td>
<td>Pain and discomfort</td>
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<td>Energy and fatigue</td>
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<td>Sleep and rest</td>
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<td>Psychological health</td>
<td>Positive affect</td>
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<td></td>
<td>Thinking, learning, memory and concentration</td>
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<td></td>
<td>Self-esteem</td>
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<td>Body image and appearance</td>
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<td></td>
<td>Negative affect</td>
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<td>Level of independence</td>
<td>Mobility</td>
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<td></td>
<td>Activities of daily living</td>
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<td></td>
<td>Dependence on medication or treatments</td>
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<td></td>
<td>Work capacity</td>
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<td>Social relationships</td>
<td>Personal relationships</td>
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<td></td>
<td>Social support</td>
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<td></td>
<td>Sexual activity</td>
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<td>Environment</td>
<td>Physical safety and security</td>
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<td>Home environment</td>
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<td>Financial resources</td>
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<td></td>
<td>Health and social care: accessibility and quality</td>
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<td></td>
<td>Opportunities for acquiring new information and skills</td>
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<td></td>
<td>Participation in and opportunities for recreation/leisure activities</td>
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<tr>
<td></td>
<td>Physical environment</td>
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<tr>
<td></td>
<td>Transportation</td>
</tr>
<tr>
<td>Spiritual domain</td>
<td>Spirituality/religion/personal beliefs</td>
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<tr>
<td></td>
<td>Overall quality of life and general health</td>
</tr>
</tbody>
</table>

### Quality of life issues for doctors

Shanafelt et al investigated the association between career satisfaction, burnout, and quality of life. These researchers found that nearly 40% of American surgeons in their sample met the criteria for burnout. In addition, burnout was associated with psychological aspects of quality of life, and nearly 30% of all surgeons surveyed displayed significantly low scores for this domain. They further found that younger surgeons were more at risk of burnout and lower measures of quality of life than more established or older surgeons. Paradoxically, 70% of the sample enjoyed being a surgeon and would not have it any other way. Of note, quality of life issues relating to crime and finance are strong motivating factors for doctors to seek relocation to New Zealand from South Africa.

There has been a significant interest in New Zealand and internationally in the areas of work related stress, mental health issues and burnout across various medical
disciplines. In New Zealand there have been reports of up to 10% of general practitioners (GPs) showing psychological symptoms of concern and a related study showed similar rates for New Zealand physicians and surgeons. Forty-six percent of GPs felt that work had affected their physical health and of greater concern is that 57% indicated they often thought about leaving general practice. A study of emergency physicians across the USA, UK, and Australasia indicated that levels of work-related stress and depression were similar across the sites. A recent study of young surgeons in New Zealand and Australia revealed that almost 27% of the participants reported burnout. Risk factors included being female, working in small hospitals, and working more than 60 hours per week.

Similarly, a recent study of New Zealand physicians preparing for written examinations, found that two thirds reported their preparation for the examinations had adversely affected their relationship with their partner or spouse. Of those with children, 90% of participants felt it adversely affected their relationships. Other reported morbidities included insomnia (37%), headaches (35%), anxiety/panic (17%), and depression (15%). A survey of practicing physicians in the Waikato and Bay of Plenty reported that 28% of participants experienced high levels of two or three aspects of burnout. Similar results were shown in a survey of Christchurch public hospital clinicians.

Quality of life issues for medical students

Issues linked with the notion of quality of life amongst medical students have also been well documented. Rosenthal et al stated that medical students are more likely to manifest depressed symptoms than their nonmedical peers. Goebert et al disclosed that 21% of their US respondents indicated depressed states of mind, which was significantly higher than those found in the general population (8%-15%). Furthermore, reports for suicidal ideation were higher in student years with a peak at fourth year of 9.5% (as opposed to 6.6% at year 1) and lower rates in later postgraduate years. In a similar study in Australia, it was found that between 16% to 25% of students reported some degree of suicidal ideation before examinations.

A further study in Turkey revealed that medical students self-reported significantly worse scores on the General Health Questionnaire, Beck Depression Inventory and State-Trait Anxiety Inventory as the students transitioned from years 1 to 2 suggesting marked problems in these areas. These scores were significantly higher than comparable students studying in physical education and economics. These findings are similar to other studies noting that medical students were particularly vulnerable to conditions such as depression, anxiety, suicidal ideation, inability to cope with problems and distress, and dissatisfaction with social support networks.

Problems with quality of life: How do they originate?

Quality-of-life issues are relevant to everyone and in this article quality of life issues pertaining to doctors and medical students have been discussed. There are some interesting causal factors postulated, that need to be acknowledged before solutions can be proposed and implemented.

Psychiatrists experience higher rates of burnout than other groups of doctors and, therefore, this group may be predisposed to burnout due to personality characteristics
and work related factors. A recent study indicated that two thirds of the psychiatrists surveyed reported moderate to high levels of emotional exhaustion and a similar proportion reported low levels of personal accomplishment. Kumar et al have proposed an aetiopathological model that encompasses predisposing, precipitating and perpetuating factors associated with this phenomenon.

Predisposing factors, with respect to psychiatrists, include characteristics such as neuroticism, openness and agreeableness. Moreover, Kumar et al found that psychiatrists scored higher than physicians and surgeons on items related to neuroticism, openness, and agreeableness, but lower in conscientiousness, implying that when combined with the personalised nature of their occupation this medical sector are more prone to burnout than other medical professionals. A further predisposing factor discussed in this article proposed that the training experience of psychiatrists plays a significant role in causing stress and burnout, due to the often adversarial encounters when working with patients with mental illness.

Precipitating factors for burnout in psychiatrists include patient violence, difficult or hostile relatives or patient suicide. Perpetuating factors depend on how a doctor perceives and responds to stressful situations. This is influenced by numerous factors including gender. For example, women may respond to stress by career dilution and working part time. They may also respond by limiting the demands of intimacy, and thus they are less likely to marry, and have fewer children.

Other studies in reference to other medical disciplines also tend to somewhat resonate with the quality of life problems faced by psychiatrists. In a longitudinal study of UK medical graduates, it has been reported that doctors who are most stressed had higher neuroticism scores, and those reporting the most emotional exhaustion had both higher neuroticism scores and were more likely to be introverted. In addition, it was found that lower conscientiousness on a personality measure predicted greater stress. Extroverts reported greater satisfaction with medicine as a career.

The causal link between quality of life, stress and burnout has been addressed in a longitudinal study of UK doctors. McManus et al noted that an increasing emphasis on higher professional standards increases stress, lowers perceived levels of quality of life, and increases likelihood of burnout in doctors. Depersonalisation (cynicism) was found to be a protective factor against stress but was considered a maladaptive coping mechanism. Academic work demands have also been reported to be associated with higher risk of burnout and stress. Lastly, GPs reported the main causes of stress were excessive paperwork, health reforms, bureaucracy, excessive hours and on call work.

A qualitative study in the UK found that medical students reported the pressure of academic and professional demands provided significant sources of stress. It is difficult to discern whether there is an increase in problems with obtaining a good quality of life or if there is greater awareness about issues such as depression which have highlighted recognition of the problem.

Nonetheless, a commonly cited link with depression is sleep deprivation, such that students who experienced less or disturbed sleep are more prone to experiencing varying degrees of depression. Furthermore, increased pressure of assessments and applying for residency placements adversely affect quality of life perceptions.
addition, increases in psychological distress have also been associated with similar increases in perfectionism, and more critically, increased levels of perfectionism have been linked to the more severe reports of suicidal ideation. Kaptein et al revealed that New Zealand medical students are more anxious about the health-related aspects of living in the postmodern world than European medical students and that female students are more anxious than their male counterparts. One suggested explanation for the difference was the greater prominence given to issues of health and environment in New Zealand compared to Europe, suggesting that students with more awareness about the issues of quality of life are more likely to report and discuss concerns related to cause and effect.

The periods of transition from school to university, from pre-clinical to clinical and from clinical to qualification, are considered to be taxing. Students find that they are expected to develop a professional persona and find the expectations of a sense of clinical confidence and competence to be a major stressor. “Many students perceive(d) lack of guidance from the medical school and individual tutors on academic requirements and individual welfare issues as a significant source of stress”.

Ross et al found that students felt that lack of money and high levels of coursework were significant causes of stress. Environmental issues related to financial stress heavily impact on the wellbeing of medical students in New Zealand. This is perhaps unsurprising in New Zealand medical students given their high tuition fees and subsequent debt; it is suggested that “first-year house officers have an average debt of $65,000; with 10% owing more than $100,000”.

**Solutions and strategies to improve quality of life**

Overseas research suggests that poor coping styles of physicians in response to high work demands are magnified by a reluctance to seek help. This is an area of concern and suggests a need to target the emotional well-being and health concerns of those within the profession. A key factor in the ability to care for others is the ability to care for oneself. Developing strong work networks that promote personal and professional reflection can assist in the exploration of practice and reduce anxiety-provoking behaviour. Positive protective factors for burnout include having interests outside professional life.

It is suggested that the way forward is to address issues of quality of life for doctors at many levels, starting at medical school selection and training through to developing a culture of support in the work place. We propose the following solutions to some identified questions:

**What can be done at an undergraduate level?**

1. The selection criteria for medical students needs to be considered prudently in terms of personality characteristics that may predispose to burnout and other quality of life issues, such as neuroticism.
2. Educators need to be explicit in the teaching and modelling of self care in the medical school curriculum including:
   a. Resilience, self-care skills training
b Facilitation of good study habits and consider strategies for coping with examination stress

c Modelling of self care

d Curriculum attention to burnout (awareness raising)

3. Medical schools should support students in engaging in positive social relationships. One of the key WHO factors for quality of life is the development of interpersonal skills.\textsuperscript{35} This cultivation of mind and empathy is important when establishing relationships with patients and developing clinical practice.\textsuperscript{35} The promotion of well-being in oneself heightens the capability to care for others especially in uncertain health contexts.

Mentoring needs to be evaluated with respect to its potential benefit for medical students. A review of all studies on mentoring in medical education reveal that although the programmes appear positive, there are challenges with their delivery, and the long term outcomes are not known. Longer term effects of a mentoring programme and a cost benefit analysis need to be investigated.\textsuperscript{39}

**What can be done for doctors in practice?**

1. Where practical, such as in a hospital, non-clinical managerial and support staff should be trained to identify early warning signs of burnout in junior and senior medical staff and monitoring well being.\textsuperscript{40}

2. Promoting quality of life programmes that include engaging in healthy exercise, developing healthy sleep patterns, and facilitating time out activities such as retreats and regular meditation.\textsuperscript{41, 42}

3. Establishment of peer groups and one-on-one support systems should be explored more widely. Most physicians prefer a one-on-one support system.\textsuperscript{14} Peer groups and supervision have been successfully introduced to help in self care for GPs by the RNZCGP,\textsuperscript{43} and similarly for surgeons by the RACS.

4. Close supervision and support of junior doctors. Increased supervision by senior doctors is important as the relationship between the supervising consultant is central to the learning experience of the intern and can impact on career decisions.\textsuperscript{44} It is also important to note that any proposals to reduce workload for junior doctors (see below) or increase the supervision of junior doctors by senior doctors must be monitored for their effect on senior doctors.

**What can be done about the workplace?**

1. Scrutiny and change with respect to medical culture should be investigated. In an exploration of compassion fatigue, Huggard urges that in order to care for the carers, health care organisations need to “develop respect and care for their employees in the same way that they require their employees to care for their patients”.\textsuperscript{36} Similarly Kumar et al propose that “organisational leaders must work with their staff to develop a workplace environment where the acknowledgement and resolution of such
workplace distress is normalised and not contained within a culture of secrecy and shame”.\textsuperscript{27}

2. Changes in working conditions should be considered. In a local study,\textsuperscript{45} surveying a small group of Resident Medical Officers (RMOs) and Senior Medical Officers suggested some strategic changes in work conditions. The RMOs sought the following work provisions: prohibition of PGY1s working nights in the first six months of employment; restrictions around provision of 24-hour on-call duty; the requirement for alternate weekends off; and unlimited training reimbursements for costs incurred on the pathway to vocational registration.\textsuperscript{45}

Conclusion

High rates of burnout in medical students and doctors are areas of great concern. There are several contributing factors including predisposing personality traits, training experience, workplace culture and workload. Potential strategies involve addressing the problem at all levels from initial selection processes, medical undergraduate education and postgraduate training, improved support systems, and changes in working conditions.

Further research is required into the effectiveness of elements of mentoring and supervision, and identifying protective and preventative factors in promoting quality life and reducing the risk of burnout. All medical students and doctors need to challenge ongoing learning and workplace practices that perpetuate less than optimal self-care practices; thereby making positive changes to workplace culture and learning environments, improving the quality of life of medical students and doctors, ultimately leading to better patient care.

Competing interests: None known.

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References:


President’s Address (part 4)


With regard to marriages much good might be done by the Health Department taking over this work. It has been suggested that no one be allowed to issue a license to marry until the prospective bride and bridegroom present certificates of good health signed by a legally qualified medical practitioner who has known the parties for at least six months.

Now I do not believe in this or any method of dragooning people. It has also been suggested to raise the minimum marriage age of a man to 25 and a woman to 21, which would (according to Dr. Rentoul) decrease degeneracy, pauperism and disease. Also that no man over 65 should be given in marriage unless the woman be over 45. Again, although recognising that such regulations would be good for the race in one sense, yet one must admit that one can carry regulations too far when one interferes with the sacred rites of the people. As to ages in New Zealand, a man of 23 is equal as a rule physically to a man of 25 in England; and a woman of 21 to an English woman of 23. We all recognize this. Yet it was only recently that some hospital boards allowed girls to start nursing before they were 23.

Such a rule is explained of course on the same grounds as that the Railway Department persists in putting the names of stations so high up that the passengers cannot see them from the carriage windows. The custom of the Old Country was followed, and although at Home it is now the custom on most of the new lines to have the names of stations painted in huge letters opposite the windows at different places, in New Zealand the railway station signs are still put in such a position as to cause trouble and mixed astigmatism.

But to return to our subject. If marriage indirectly ever brought under the jurisdiction of Health Department, at least an attempt might be by the issue of pamphlets to people marrying telling them some useful physiological facts. Better still if there were classes in hygiene in all schools of the nation, say evening continuation classes, where girls could be taught most useful information. Schools for mothers, similar to those established in St. Pancras, London, might also be established in all the large centres, where instruction could be given in the duties of motherhood and infant feeding.

In recognising that the health of a nation both physical and moral is its greatest asset, we must recognise the all powerful influence of women. We must have healthy mothers who are not familiar only with factory labour, but who can also sew, cook, wash, and attend intelligently to children. The best immigrants to New Zealand are strong and healthy children born in the dominion. Once they arrive do not cram their undeveloped brains but train them up gradually by means of kindergartens, schools, continuation classes, etc., to develop into healthy mothers and strong fathers.
Homeopathic products—well at least they can cause no harm

Those who see little to recommend in homeopathic remedies have been comforted by the fact that they were harmless as they contain nothing. But recently the US Food and Drug Administration (FDA) has issued warnings that a popular over-the-counter cold remedy could cause loss of sense of smell in users and this has drawn attention to potential risks associated with so-called homeopathic remedies. Apparently the medications in question contained zinc in less than homeopathic concentration. Not so harmless after all.


On the other hand—homeopathy can be very harmful

The World Health Organization (WHO) has said that homeopathy should not be used to treat several serious diseases such as HIV, tuberculosis, malaria, influenza, or diarrhoea in infants. Their interest in the topic was aroused by doctors in the UK and Africa who drew attention to the continuing promotion of such complementary therapies in many developing countries. Clearly medications containing nothing have no place in the treatment of potentially lethal diseases.


Chronic obstructive pulmonary disease (COPD), inhaled corticosteroids, and the risk of pneumonia

Inhaled corticosteroids, given with and without long-acting $\beta_2$-agonists, reduce the occurrence of disease exacerbation and improve quality of life for patients with COPD, but they have also been associated with increased risk of pneumonia. Two meta-analyses have pointed this way. However, this one produces a contrary view. The authors pooled data from seven large clinical trials of inhaled budesonide (320–1280 $\mu$g/day), with or without formoterol, versus control regimen (placebo or formoterol alone) in patients with stable COPD and at least 6 months of follow-up. There were 7042 patients, of whom 3801 were on inhaled budesonide and 3241 were on control treatment, with 5212 patient-years of exposure to treatment. There was no significant difference between the cohorts in terms of occurrence of pneumonia or other serious adverse events. In musing on this an editorial commentator notes that this review was on hospitalised patients which would increase the likelihood of an authentic diagnosis of community-acquired pneumonia, but some uncertainty remains. He also notes that there was no increase in mortality rates in any of the three meta-analyses.

Renal-artery stenosis management

Stenosis of the renal artery is associated with both hypertension and chronic kidney disease, although it is not clear whether these associations are causal. Treatment has traditionally focused on correcting renal-artery stenosis, with endovascular revascularisation having gradually replaced open surgical techniques. The authors of this report question the benefits of revascularisation. They randomised 806 patients with renal-artery stenosis to medical treatment with or without revascularisation. Patients in each arm of the trial were well matched in terms of their exposure to antihypertensive, antiplatelet and cholesterol-lowering drugs. And the conclusion of the study was that revascularisation carried substantial risk but was not associated with any benefit with respect to renal function, blood pressure, renal or cardiovascular events, or mortality.


Should children at risk of developing urinary tract infection (UTI) have antibiotic prophylaxis?

Antibiotic prophylaxis in children who have had urinary tract infection (UTI) to prevent further infection is a common practice, the rationale being that such treatment may reduce the development of further renal scarring by the prevention of recurrent acute pyelonephritis, particularly in those children who have vesico-ureteric reflux (VUR). Whether such treatment achieves these aims is the subject of this meta-analysis. Eight trials involving 677 children were evaluated and they conclude that although prophylaxis is effective in reducing the number of positive urine cultures found on subsequent screening, there is no reduction in the number of symptomatic infections nor of new renal scarring, which is the primary aim of the intervention. So they do not recommend prophylaxis as a routine, but they speculate that there may be a role for prophylaxis in children with more complex urological conditions, who are at the highest risk such as infants with high-grade VUR and recurrent pyelonephritis. Enter clinical judgement.


Rotator cuff tears—which ones require surgical repair?

The management of rotator cuff tears is a matter of controversy, especially for tears with limited symptoms. While surgery is certainly a recognised treatment option, not all rotator cuff tears actually warrant surgical management. 59 shoulders with such lesions were evaluated with MRI at baseline and serially. The baseline scans demonstrated 33 full-thickness tears, 26 partial-thickness tears, and 4 combined full-thickness and partial-thickness tears. 58 of the 59 tears involved the supraspinatus tendon and 10 involved multiple tendons. The study revealed that progression of rotator cuff tears is more likely in subjects who are older than 60 years and have full-thickness tear, and fatty infiltration of the rotator cuff muscle(s). Hence they recommend MRI study of such shoulders as this will define which will benefit from surgical repair.

Diabetes and tobacco

Thirty years ago, cigarette smoking was seen by some doctors as giving sustenance to young people with Type 1 diabetes and older women with Type 2 diabetes.

- Today 1 in 9 European New Zealanders now have diabetes or pre-diabetes.
- 1 in 3 Polynesian New Zealanders now have diabetes or pre-diabetes.
- 1 in 5 Māori now have diabetes or pre-diabetes.

Last year tobacco tax provided $963 million excluding GST to the government. In spite of the measures for tobacco control including legislation, social marketing campaigns, quit campaigns, and education, 45% of Māori aged 15–64 smoke and their rates of tobacco smoking remain the highest in the world.

It is well established that taxation plays a key role in lowering rates of smoking. We applaud the efforts of the Māori Party and Hone Harawira, MP in announcing that the Māori Affairs Select Committee will hold a Hearing on Māori Tobacco Use in 2010.

Diabetes New Zealand supports the following measures to ensure New Zealand is smokefree no later than 2020:

1. An increase in the tax on cigarettes and loose tobacco.
2. A ban on all retail displays of cigarettes and tobacco.
3. An immediate end to individual retailers being allowed to discount cigarettes or tobacco.

Professor Sir Don Beaven               Sarah Thomson
Patron                                Chief Executive

Diabetes New Zealand

(Sir Don Beaven tragically died following the writing of this letter. An obituary appears at http://www.nzma.org.nz/journal/122-1307/3919/content.pdf)
Majority support by Māori and non-Māori smokers for many aspects of increased tobacco control regulation: national survey data

The Māori Affairs Select Committee is undertaking an Inquiry into “the tobacco industry in Aotearoa and the consequences of tobacco use for Māori”\(^1\). The very high levels of smoking among Māori,\(^2\) the important contribution of smoking to poor health and disparities in health,\(^3,4\) and the substantial impact of tobacco use on Māori social and economic development, support the timeliness and importance of this Inquiry.

A possible outcome of the Inquiry is to recommend substantial strengthening of the measures in place to reduce smoking prevalence by promoting and supporting smoking cessation and reducing smoking uptake. Such measures might include introducing a range of proposed new tobacco control policies, strengthening and intensification of existing interventions, or implementing more radical ‘endgame’ solutions. The latter is probably more efficient at ending the tobacco epidemic and could aim to reduce the use of smoked tobacco products such that the large-scale commercial distribution and sale of smoked tobacco product effectively ceases (e.g. in 10 years time).

The aim of this study is to describe the level of support for additional tobacco control policy measures among Māori and non-Māori participants from a nationally representative sample of New Zealand smokers.

These data comes from the New Zealand arm of the International Tobacco Control Policy Evaluation Survey (ITC Project).\(^5\) The data were collected in a national computer-assisted telephone survey (Wave 1) between March 2007 and February 2008, and in a follow-up survey between March 2008 and February 2009 (Wave 2). Subjects were selected from adult smokers in the New Zealand Health Survey (NZHS) who were willing to participate in further research.

Out of 2438 potential respondents, 1376 (including 607 Māori smokers) completed the Wave 1 questionnaire giving a response rate of 56.4%, and 923 (37.9%) completed a Wave 2 interview. Taking into account the response rate of the NZHS and willingness to participate in further research reduces the overall response rate in Wave 1 was 32.6%.

The particular questions relating to the tobacco industry and regulation were largely derived from Wave 4 of the four-country ITC survey. However, the New Zealand arm of the study added some additional questions in both Wave 1 and Wave 2. Further detail on the survey methods are available in an online Methods Report\(^6\) and in other journal article publications from this project.\(^7,8\)

In the analysis, where data are available from both waves, results are presented from Wave 1 due to the higher number of participants. Results are presented as a comparison of views among Māori and European/Asian/other smokers. All of the
presented results were weighted and adjusted for the complex sample design of the NZHS to make the sample representative of all Māori and European/Asian/other New Zealand smokers.

Table 1 shows that among Māori and European/Asian/other smokers there was strong majority support for greater regulation of the tobacco industry and additional government action on tobacco. There was overwhelming support for introducing regulations to reduce the toxicity and addictiveness of cigarettes. There was also strong majority support for a range of other specific tobacco control policy options, including, point of sale display bans, restricting availability of tobacco for sale to premises where children are excluded, and tax increases on tobacco products (provided the revenue was used for health promotion and to help smokers to quit).

A substantial minority of smokers also supported the introduction of plain packaging of cigarettes. Except for the measures to reduce the addictiveness and toxicity of cigarettes, support was stronger among Māori smokers, and was statistically significantly stronger for several issues. These findings add to previously reported data on support for extensions to smokefree legislation in many outdoor settings and also in private cars where children are present. There was also support among almost half of all smokers for the endgame solution of banning cigarette sales in ten years time, provided effective non-smoked nicotine products were available.

Some of these findings have been replicated in other settings. For example, the proportion of smokers agreeing that tobacco products should be more tightly regulated was 61.7% in the USA, 63.8% in Canada, 68.9% in Australia, and 71.1% in the UK in the ITC 4-country study. These data refer to support among smokers. Support is likely to be greater still among non-smokers. For instance, 69% of a 2008 national survey of New Zealand smokers and non-smokers agreed that there should be a complete ban on retail tobacco displays. Half agreed ‘cigarettes and tobacco should not be sold in New Zealand in 10 years time’ (30% disagreed), and 53% agreed that ‘tobacco companies should not be allowed to promote tobacco by having different brand names’.

The findings suggest there is strong support among smokers for intensification of current tobacco control efforts as well as for more radical ‘endgame’ approaches to tobacco control. Support was at least as strong and usually greater among Māori smokers. Levels of support among non-smokers will almost certainly be higher still. This high level of support suggests that the public are well ahead of policy-makers. The findings bring into focus successive governments’ failure to fully implement proven tobacco control measures. There has been a failure to introduce or even consider potential endgame solutions to the greatest single cause of avoidable illness and premature death among the New Zealand population. The findings suggest that the Māori Affairs Select Committee should feel empowered to recommend a clear endgame strategy or at least a rigorous programme of measures aiming to end the tobacco epidemic among Māori and all other New Zealanders.
Table 1. Support for increased tobacco regulation among Māori and European/Asian/other smokers in New Zealand
(with all results weighted and adjusted for the complex sample design to represent the national population of smokers in New Zealand)

<table>
<thead>
<tr>
<th>Question asked</th>
<th>Māori (95%CI)</th>
<th>European/Other/Asian (95%CI)</th>
<th>Crude odds ratios—Māori vs European/Other (95%CI)</th>
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</thead>
<tbody>
<tr>
<td>(all Wave 1 data unless otherwise indicated)</td>
<td>n=607 in W1</td>
<td>n=679 in W1</td>
<td>n=369 in W2</td>
</tr>
<tr>
<td><strong>General questions about tobacco industry and regulation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Agree or strongly agree that tobacco companies should be more tightly regulated</td>
<td>70.5% (65.6–75.4)</td>
<td>62.3% (57.9–66.8)</td>
<td>1.44 (p=0.017) (1.07–1.95)</td>
</tr>
<tr>
<td>Agree or strongly agree that the government should do more to tackle the harm done by smoking</td>
<td>65.9% (60.8–70.9)</td>
<td>54.6% (50.0–59.2)</td>
<td>1.60 (p=0.001) (1.20–2.15)</td>
</tr>
<tr>
<td>Agree or strongly agree that if effective nicotine substitutes that are not smoked became available, the government should then set a date to ban cigarette sales in ten years time (W2)</td>
<td>46.2% (39.0–53.4)</td>
<td>44.4% (38.9–49.9)</td>
<td>1.07 (p=0.704) (0.74–1.55)</td>
</tr>
<tr>
<td><strong>Specific questions about tobacco regulation policies</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Support a little or a lot laws that would reduce the toxins in cigarette smoke</td>
<td>84.8% (81.2–88.3)</td>
<td>87.8% (84.8–90.9)</td>
<td>0.77 (p=0.199) (0.52–1.15)</td>
</tr>
<tr>
<td>Support a little or a lot laws that would reduce the addictiveness of cigarettes</td>
<td>84.4% (80.4–88.4)</td>
<td>86.1% (83.0–89.2)</td>
<td>0.87 (p=0.511) (0.59–1.31)</td>
</tr>
<tr>
<td>Support a little or a lot complete bans on displays of cigarettes inside shops and stores</td>
<td>62.5% (57.3–67.6)</td>
<td>58.2% (53.6–62.7)</td>
<td>1.20 (p=0.223) (0.90–1.60)</td>
</tr>
<tr>
<td>Agree or strongly agree that tobacco products should only be sold in special places where children are not allowed to go (W2)</td>
<td>66.6% (60.1–73.1)</td>
<td>58.5% (53.0–63.9)</td>
<td>1.42 (p=0.065) (0.98–2.05)</td>
</tr>
<tr>
<td>Agree or strongly agree that tobacco companies should be required to sell cigarettes in plain packages (W2)</td>
<td>42.3% (35.0–49.5)</td>
<td>36.8% (31.5–42.1)</td>
<td>1.26 (p=0.230) (0.86–1.83)</td>
</tr>
<tr>
<td>Support a little or a lot an increase in the tax on tobacco if all the extra money was used to promote healthy lifestyles including helping smokers wanting to quit</td>
<td>64.6% (59.4–69.9)</td>
<td>56.4% (51.8–60.9)</td>
<td>1.42 (p=0.021) (1.05–1.90)</td>
</tr>
</tbody>
</table>
Richard Edwards*, Nick Wilson, George Thomson, Deepa Weerasekera, Tony Blakely

Department of Public Health, University of Otago, Wellington
*richard.edwards@otago.ac.nz

Competing interests: Although we do not consider it a competing interest, for the sake of full transparency we note that some of the authors have undertaken work for health sector agencies working in tobacco control.

Acknowledgements: The ITC Project New Zealand team thank: the interviewees who kindly contributed their time; the Health Research Council of New Zealand which has provided the core funding for this Project; and our other project partners (see: http://www.wnmeds.ac.nz/itcproject.html).

References:


Protecting victims of domestic violence

There are currently two Bills aimed at protecting victims of domestic violence which are now before the New Zealand Parliament. With the Domestic Violence (Enhancing Safety) Bill it would enable police to issue on the spot protection orders to protect victims of domestic violence. It would also enable sentencing judges in criminal courts to issue protection orders on behalf of victims. The Child and Family Protection Bill focuses on keeping children safe in circumstances where there has been family violence. Its objectives and advantages include, for example, clarification that when protected person dies their children remain protected, addressing issues of psychological violence of children.

How would you identify a victim? It is commonly agreed that there are four elements which describe instances where there has then victimisation. Firstly there it is an imbalance of power between individuals. Secondly there is an abuse of that power by the superior or stronger individual. Third, the inferior/weaker individual (the victim) suffers in some manner whether that be, for example, physically, psychologically or otherwise. Fourth, the victim is often in a situation of helplessness either where they are unable to receive help and/or unable to seek it for themselves. What's interesting is that these criteria apply just as much to animals as they do to people.

Research has shown that a significant number of women will stay in circumstances where there is domestic violence thereby putting themselves, and their children, at significant risk rather than leave the family pet with an abusive partner/spouse to fear that harm will come to the animal. In recognition of this some shelters have enabled families fleeing domestic violence to also bring their pets. In further recognition of this research finding, certain legislatures overseas have allowed family pets to be included in protection orders. "A victim is a victim no matter what the species" is a byline quoted by many who recognize the link between domestic violence and animal abuse. Protections would preferably be provided for animals on their own merits but the reality of course is that not all people view or value animals as sentient, animate creature's capable of experiencing pain. However recent political attention to the issue of domestic violence and recognition of its huge cost to society lends weight to the argument that if the cost is going to be exacerbated by not looking after the pets then perhaps it would be a relatively simple cost efficient initiative to enable pets to be included in protection orders.

This is not a groundbreaking revelation in view of the research and the leadership in this area already provided by legislators and politicians overseas—but perhaps it is an initiative that New Zealand might consider learning from and duplicating.
Ian Robertson is a veterinarian and lawyer who has combined his training and expertise to become an internationally recognised animal welfare law specialist. He is an external adviser to the World Organisation for Animal Health (OIE), a member of the International Advisory Board of Compassion in World Farming (CIWF), the Director of International Animal-Law and a Prosecutor for the Ministry of Agriculture and Forestry.
Response to Narasimhan and Clausen's letter on heterophile antibodies and troponin results

Narasimhan and Clausen highlight the importance of recognising possible assay interference due to heterophilic antibodies (HAb)¹ and having strategies in place for addressing this, although they raise several issues which need to be critically qualified. Firstly, although it is plausible to conjecture that increased exposure to animals in a rural population may increase the prevalence of HAb, and in particular human anti-animal antibodies (HAAA), the authors supply no data to support this.

Secondly, manufacturers of immunoassays are aware of the potential for false positive and false negative results due to HAb routinely include “blocking agents”, usually pooled globulin from several species, to minimise the problem. No assay, however is entirely protected from this type of interference and manufacturers include a warning to this effect on package inserts supplied with reagents. The authors indicate that the Beckman Coulter Access AccuTnI assay was selected because it gave the “best” results, but are not clear on the criteria used to determine this.

In those cases where there is a high suspicion of HAb interference, they use Scantibodies Heterophilic Blocking Reagent (HBR)² in order to combat the problem, although have not indicated how they would come to have such a suspicion in the first place and in what proportion of samples they would employ this manoeuvre. Such interference usually only comes to light when there is discordance between results and the clinical context³ and is dependent upon a good clinician-laboratory interface to recognise in the first instance. Furthermore, they have not indicated that there are several strategies that are well recognised and documented in the clinical laboratory community for addressing potential interference apart from HBR³, including analysis by an alternative analytical platform and non-linearity of results on serial dilution.

Most critically, the results obtained following the application of HBR cannot be reported, although a clear difference may be interpreted as due to the presence of HAb interference. In this instance, the best strategy is to arrange for assay on an alternative analytical platform, as indicated above, with a view to providing results that can be reported.

With improved assay formulations to overcome the effects of HAb interference, the proportion of clinically significant interference may be in the order of 0.2%⁴. Notwithstanding that, prevalence figures may be flawed given that assay results may still be inaccurate due to interference, though do not come to light through being obviously clinically discordant. HBR, furthermore may not be a foolproof way of detecting assay interference. We recently reported a case with assay interferences affecting cortisol, free T4 and free T3 in the same patient, though through different mechanisms⁵. HBR pre-treatment provided a clue to the presence of spuriously low cortisol, though had no effect on the free T4 or Free T3 assays. Other strategies were employed to fully characterise the interference in these assays⁵.
In the case of troponins, it should also be noted that elevations may be observed in several clinical contexts\(^6\) and are not exclusively attributable to acute myocardial infarction (MI), thus one should not necessarily suspect interference if an otherwise unexplained elevation is observed. The recent universal definition of MI\(^7\) also stipulates a pattern of rising or falling pattern of troponins, which if present, is evidence against interference, where constant levels are usually observed over time.

We would therefore like to assure the clinical community that laboratories are well aware of the potential for assay interference through HAb and several other mechanisms\(^3\), although rely upon good clinician feedback in order to recognise and investigate further. HBR is only one of many strategies for addressing this, and not foolproof. Arrangement for assay by an alternative analytical platform is usually the most expedient and practical approach for most laboratories.

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Clinical Director and Consultant in Chemical Pathology  
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References:

Rewriting the inverse care law

In 1971, a general practitioner in Wales, Dr Tudor Hart, proposed the Inverse Care Law in a paper published in *The Lancet*. He wrote,

“The availability of good medical care tends to vary inversely with the need for the population served. This inverse care law operates more completely where medical care is most exposed to market forces, and less so where such exposure is reduced. The market distribution of medical care is a primitive and historically outdated social form, and any return to it would further exaggerate the maldistribution of medical resources.”

New Zealand now has an elegant variation of this law, which can be stated in this way; “The need to pay for treatment of an acute illness varies inversely with the severity of the medical condition.”

Experience shows that a patient threatened with a coronary occlusion merely has to lift the phone and dial a number. The ambulance and the hospital will, between them, do the rest, and do it for nothing. That was the experience 3 weeks ago of a friend of mine. He got three stents inserted and he was back home again in 2 days.

By contrast, here is the case of a shop worker who recently consulted her doctor for a troublesome acute illness. Fee $47, plus unspecified charges at the chemist’ shop. A few days later, she was seen for review. Another $47. The doctor wanted the case to be further reviewed urgently by a specialist, who supplied reassurance at short notice for $160 and told her to finish the course of antibiotics. The GP suggested a third consultation, but the patient, having almost fully recovered, declined this.

Had she agreed to the third consultation, the total bill, excluding the pharmacy charges, would have been $300, plus unspecified costs to the taxpayer. She is paid a casual rate of $12.50 an hour, so she would have had to put in three 8-hour days to meet the costs of one minor illness.

If you want to know what she thinks about these charges, you would have to ask her, but I do know that she is satisfied with the level of care and attention.

Roger Ridley-Smith
Retired GP
Wellington
Resource constraint is all the more reason to not let up on inequalities

Nationwide publicly funded health and health care organisations are in the process of major service reviews in light of the knowledge that the fiscal envelope for Vote Health next year and in subsequent years will be constrained. This is a process in which the commitment to reducing health inequalities, which has been a feature of health systems planning in this country for the last decade, can become subsumed in the need to curb costs in the short term.

Earlier this year we undertook an exercise in our DHB to estimate our health care expenditure by ethnicity, this being the most important marker of inequalities for our population. We took the crude expenditure per head (before adjustment for age) for Maaori and Pacific peoples and compared that to non-Maaori/non-Pacific groups. We estimated that if our Maaori and Pacific peoples were able to experience the same levels of health as our more advantaged populations, as a DHB we would save $62.2 M in direct healthcare costs.

If our figures are indicative of the costs of health inequalities for Maaori and Pacific peoples in other DHBs across the country, then this could translate into approximately $338 M of savings. Population inequalities related to socioeconomic deprivation, over and above those already captured using ethnicity, engender further costs—at least as much again. Other inequalities such as those related to gender and disability also result in increased costs. There are of course many other sectors that bear excess costs in relation to health inequalities (ACC, Social Welfare, Housing etc), and there are very significant tangible and less tangible social costs of these health inequalities for our communities.

Those with greatest need stand to gain most, so addressing inequalities makes sense both in terms of clinical and cost effectiveness. Continuing to ensure that health and health care organisations maintain their focus on health inequalities in these cost-constrained times is not only the right thing to do, it is likely to be the most economically prudent.

Maintaining quality in the face of resource scarcity is also an important health systems goal, and it is timely to remember that equity and access sit alongside safety, effectiveness and efficiency in the domains of quality for New Zealand health care organisations.

Investment in prevention is important to reduce health inequalities, and has been shown to be cost effective. Service areas which health needs analysis work in our DHB highlight as particularly important in relation to health inequalities, where there is evidence of effective interventions, and which may be vulnerable to spending cuts include:

- Smokefree lives
- Cardiovascular disease prevention
• Diabetes prevention
• Mental health early intervention
• Child and youth health.

While official statistics are yet to be reported, there is general acceptance that the economic recession, which has increased pressures on health budgets, has negatively impacted the social determinants of health. Given these determinants are a major driver of health inequalities, this is even more reason for health services to prioritise services which can help to mitigate these inequalities.

Health inequalities are not only unjust and unfair; they are costly to this country, and in times of tightening fiscal pressure, it is important that we do not let up on pursuing health equity.

Note: Double vowels [e.g. ‘aa’ in Māori] are used rather than macrons [e.g. ā] where appropriate in Te Reo words in this article in keeping with the Tainui convention, as Manawhenua of the Counties Manukau district.

Gary Jackson
Clinical Director Public Health
Planning and Performance Team
Counties Manukau District Health Board

Doone Winnard
Public Health and Clinical Advisor
Te Kaahui Ora Māori Health Team
Counties Manukau District Health Board

References:

A survey of New Zealand patients’ preferred characteristics of their general practitioners

Although there has been a great deal of research regarding the doctor/patient consultation, little if any research has addressed the basic questions of what sort of doctor patients prefer. Anecdotal reports suggest that patients place more emphasis on how “nice” a doctor is than may be expected. This perhaps partially explains the increasing popularity of many alternative health care practitioners. Although their treatments may in some cases have no scientific rationale or evidence, alternative health care practitioners often have much more time for their clients and are usually highly skilled in terms of their bedside manner.

In order to answer this question, 109 patients attending 5 general practice surgeries in the Bay of Plenty, New Zealand completed a short survey on which characteristics were preferred in their general practitioners (GPs). Respondents’ median age was 44 yrs (4 responses missing), and there were 41 males and 67 females (1 response missing).

First, participants were asked (Table 1), “When considering your family doctor (GP), how important are the following characteristics?” Overall, patients thought that it was very important that their doctor was knowledgeable, a good listener, had good bedside manner and was generally nice. They were not too concerned whether their doctor was the same age, gender or ethnicity.

Table 1

<table>
<thead>
<tr>
<th>GP characteristic</th>
<th>Not important at all</th>
<th>Not very important</th>
<th>No firm opinion</th>
<th>Quite important</th>
<th>Very important</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowledgeable</td>
<td>4</td>
<td>3</td>
<td>-</td>
<td>10</td>
<td>96</td>
</tr>
<tr>
<td>Good listener</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>15</td>
<td>88</td>
</tr>
<tr>
<td>Good bedside manner</td>
<td>1</td>
<td>5</td>
<td>9</td>
<td>36</td>
<td>51</td>
</tr>
<tr>
<td>Nice</td>
<td>1</td>
<td>4</td>
<td>3</td>
<td>46</td>
<td>54</td>
</tr>
<tr>
<td>Same age as me</td>
<td>59</td>
<td>24</td>
<td>14</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Same gender as me</td>
<td>47</td>
<td>24</td>
<td>16</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Same ethnicity as me</td>
<td>58</td>
<td>24</td>
<td>12</td>
<td>6</td>
<td>6</td>
</tr>
</tbody>
</table>

Second, participants were asked (Table 2) how they would feel about a number of factors that are largely irrelevant to the skills for which a medical practitioner would be expected to be consulted. The main findings from these questions were that strong religious beliefs and very old doctors were not preferred.
**Table 2**

<table>
<thead>
<tr>
<th>GP characteristic</th>
<th>Would not like this at all</th>
<th>Would not prefer this, but not too bothered</th>
<th>No opinion either way</th>
<th>This would be quite good</th>
<th>This would be very good</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very religious</td>
<td>10</td>
<td>23</td>
<td>68</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Very old</td>
<td>17</td>
<td>26</td>
<td>60</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Has movie star looks</td>
<td>5</td>
<td>11</td>
<td>70</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>Very much into alternative medicine</td>
<td>4</td>
<td>17</td>
<td>47</td>
<td>27</td>
<td>12</td>
</tr>
<tr>
<td>Very young—look like they’ve just qualified</td>
<td>8</td>
<td>23</td>
<td>70</td>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

Finally, when asked if they had to *choose between* a very clever doctor and a very nice doctor, 31% chose the latter.

This small, quirky survey therefore found that patients place a lot of value on their doctor being “nice” and for one-third of people this was more important than their clinical skills.

Shaun Holt
Tauranga

Andrew Gilbey
Palmerston North
Sir Donald Ward Beaven

31 August 1924 – 4 November 2009

Sir Don Beaven died in a house fire at Little Akaloa, Banks Peninsula. He was one of Canterbury's most admired health leaders for achievements and humanity.

When the plaudits fly for Sir Don Beaven, few will mention he was a self-confessed stirrer, an avowed republican, a socialist sympathiser. But then, so many and mighty were his achievements, and so much good did he do for others, the effects of early hardship and family dislocation can easily blur into a little-known background.

The boy whose mother scrimped and saved to send him to Christ's College after her husband abandoned the family in the Depression, who hated school because he was bullied, who could get into university only by winning a scholarship, became the face and voice of medicine in Canterbury. Possibly no other man was as much loved and admired in the community.

Sir Don, foundation professor of the University of Otago's Christchurch School of Medicine and a former chairman of the Canterbury Area Health Board, was a world authority on diabetes and an innovator in treatment and prevention of the disease. He was a leader in the promotion of healthy eating and medical research, who backed his words with the deeds of a pioneer wine producer and olive grower. He fearlessly confronted politicians, bureaucracies and agricultural interests. Yet he was a man of grace and modesty, who dismissed his host of honours and awards as due simply to luck.

He spoke frequently of his good fortune. "I was so lucky to live at a time of change in the health system," he said of the post-World War II years, when advances such as penicillin and new surgical procedures were allowing doctors to make a difference. In later years, perhaps bruised from brushes with authorities, he yearned for those times when "Doctors felt lucky to look after people" and felt "a strong feeling of privilege" to work with patients. "It was a unique time to be in medicine," Sir Don said.

Descended from a partner in well-known Christchurch engineering firm Andrews and Beaven, he was born in 1924 and attended Fendalton Primary School. He described his father, traumatised by 5 years of active service in World War I, as "emotionally impoverished and unable to form close bonds." Sir Don was 12 when his father walked away, leaving the family on an economic knife edge.

Sir Don then knew the discomfort of appearing in threadbare clothing at Christ's College. Small and vulnerable, he was bullied. He spent most of his time sequestered in the library for safety. As he grew wiser, he bunked classes, making a start to his sailing career with a small yacht he and his brother bought from doing odd jobs.
A doctor shortage caused by the war prompted the Government to establish a scholarship system for low-income students to attend medical school at Dunedin. Encouraged by an uncle, Sir Don applied. He joined a class of 40 who formed a closely knit group among colleagues from well-to-do homes. He said the feeling of having to prove himself drove him, and his group, to succeed.

He enjoyed the years 1944 to 1948 at Dunedin, not least as a member of an alpine climbing group. Making rapid advance in mountaineering skills, he climbed with Edmund Hillary and George Lowe at Mt Cook. He would later tackle high peaks of the European Alps.

Graduating in 1949, Sir Don worked as a medical intern at Christchurch Hospital. He benefited from the enlightened mentoring of progressive senior staff there, after the "old guard" approach of his Dunedin teachers. He then took up the post of resident doctor at Karama, 100 kilometres north of Westport. He described the practice in this remote place, where electricity for his surgery was generated by the diesel engine of a war-surplus torpedo boat, as consisting mainly of childbirth and sawmill accidents.

Colleague Sir David Hay says the Karama experience taught Sir Don self reliance and initiative. He developed the art of listening to patients, which served him well thereafter. Still feeling the inequities of poverty, he responded positively to the tutelage of a well-read local shopkeeper who gave him hot dinners and dissertations on socialism.

Reading some of the man's stock of socialist books reinforced Sir Don's humanitarian beliefs and sparked his interests in the link between poor housing and ill-health. He determined to seek a future in public health and, 15 months after arriving at Karama, left to work his passage on a cargo ship to Britain for post-graduate study. He worked as a medical registrar in London and Middlesex. While studying, he developed the interest in diabetes that would dominate his medical career.

Sir Don spent his leisure time climbing and sailing. The latter took him to French ports, from which he explored the coastal districts. There he discovered wine and first speculated on its health benefits. In Britain he attended socialist meetings, getting to know members of the Labour Party. He would later expound on his beliefs in co-authoring a book of social commentary with such Leftist thinkers as Brian Easton and Erich Geiringer.

Sir Don returned to Christchurch Hospital in 1955, as senior resident physician. He also took a new position as clinical tutor to medical students and extended his study of diabetes. His research and writing attracted the attention of the United States' Harvard Medical School, which invited him to do further work there. A Fulbright Scholarship made this possible and he took up the invitation in 1958. However, the costs were still high. Sir Don had married former Dunedin violinist Teresa Fahey in 1956. The couple sold wedding presents to help finance their trip.

Sir Don and his wife had two daughters. The couple later separated. Sir Don married Gillian Hobbs in 1993. Gillian says she was privileged to share with him 18 of the 20 years since he retired. In this time, he was a "a larger than life figure, whose great warmth touched everyone he met", yet he preferred, and managed to keep his family life private.
After 2 years in the US, Sir Don returned to Christchurch. He became a specialist consultant to Christchurch Hospital and senior lecturer at Otago University's medical unit there. When Otago opened its Christchurch School of Medicine, in 1971, Sir Don was appointed professor of medicine and head of the academic department. He attracted top overseas staff and led them in groundbreaking research. Under him, the school developed international standing.

Lack of funding for medical research was a bugbear for Sir Don. As joint founder of the Canterbury Medical Research Foundation, he worked to establish consistent funding. Some senior doctors and administrators opposed the development of research in hospitals, so the founders publicised their cause.

Getting community backing ensured the foundation's success. The exercise also demonstrated Sir Don's ability to win people's minds to a cause, an attribute that would serve him well in medical politics. He was instrumental in setting up diabetes societies. He became an effective member and chairman of the Canterbury Area Health Board.

His work in health over the past 40 years drew many accolades. Queen's honours, including knighthood, might have surprised some who remembered his republican and socialist leanings but he always claimed his awards honoured the work of his wife and colleagues. He became a popular public figure. He gave many addresses, published eight books and hundreds of articles, sat on boards and committees and held medical appointments and fellowships around the world. Sir David says Sir Don "was a man of high standing and of high standards, in everything he did".

His interest in wine led Sir Don, with six medical and mountaineering friends, to experiment with grape growing in Canterbury in the mid-1970s. Driven partly by a belief that New Zealanders should adapt to a more Mediterranean lifestyle, they tried 20 plant varieties near Halswell. Sceptics scoffed at their attempts and Sir Don admitted they never made any great wine. But their efforts laid a foundation for Lincoln University researchers and winemakers such as Danny Schuster to build on. Canterbury's wine industry owes much to the determination of Sir Don and others to overcome what Sir Don called "ridicule" and "negativism". Sir Don became an authority on wine and wrote columns on it for newspapers and magazines.

He was similarly enthused about the efficacy of olives, after studying the link between diet and longevity in Italy. He established one of Canterbury's early olive groves in the 1990s, on hills around the Banks Peninsula holiday house where he and Gillian loved to go. He constantly urged reductions in the intake of fatty foods, promoting olive oil to replace butter. His advocacy provoked battles with the dairy and meat industries over what he called "aggressive" lobbying and advertising.

The holiday house among the olive trees, on the brittle slope above the sparkling waters of Little Akaloa, had a Mediterranean flavour. Though their Christchurch home was an oasis of fine art and music, Sir Don and his wife got away to Little Akaloa whenever they could. It was there that Sir Don tapped a vein of mellowed optimism. Fondling an olive branch in 2004, he lamented that New Zealanders were still eating five or six times more fat than their livers could handle. But then he paused to add: "The situation is not all doom and gloom." And the twinkle returned to his eye
as he observed community awareness of diabetes was growing and health policy-makers were taking note.

Sadly, it was in this place, where Sir Don found peace, that he met his death.

Sir Don is survived by wife Gillian and daughters Sarah and Lisa.

This obituary originally appeared in The Press newspaper (Christchurch) and was written by Mike Crean.

A public memorial celebration for the late Emeritus Professor Sir Don Beaven will be held in the Auditorium, Christchurch Town Hall, on Friday 18 December 2009. Access to the Auditorium will be available from 1.30pm, and the Memorial Celebration will commence at 2.00pm.

The NZMJ welcomes any additional obituaries on Sir Don Beaven.
Dorothy Field Usher Potter  


Dr Dorothy Potter was the elder daughter of Auckland surgeon Victor Usher and his wife Doris. She was educated at Hilltop School for Girls in Auckland and at Woodford House, Havelock North, before completing her medical intermediate at the University of Auckland and her medical training at Otago, from where she graduated in 1948.

She obtained a house surgeon post in Napier where she was influenced towards ophthalmology by the late Dr Jim Gray and in 1950 she went to England and worked at the Royal Westminster and Central Eye Hospital in London, obtaining her Diploma in Ophthalmology in 1952.

While in England she was influenced by many big names in ophthalmology but in particular she developed a strong admiration for Professor Ida Mann who was the first woman Professor of Ophthalmology in Britain and the first woman professor (in any subject) at the University of Oxford.

Ida Mann did pioneering work on the embryology of the eye and its developmental abnormalities, the long-term ocular effects of gas warfare, and extensive public health ophthalmology amongst Australian aborigines.

Tragically, while Dorothy was in London, her father died in a boating accident on Lake Rotoiti and she returned home from England before having time to obtain the Fellowship. However, she was later admitted to Fellowship of the Royal Australian and New Zealand College of Ophthalmologists and the Royal College of Ophthalmologists of the United Kingdom.

Back in Auckland in 1953, Dorothy was declined a position at Auckland Hospital—the senior surgeons reportedly would not employ a female even though they were short staffed—but she was able to secure a locum position at Hamilton Hospital. While there she met and later married Charles Potter, a Wairarapa sheep farmer, and they settled in Masterton where she commenced the first ophthalmic practice in the region. From then until her retirement, Dorothy divided her professional life between Masterton and Wellington.

From the outset she limited her practice to non-surgical ophthalmology but this decision in no way hindered her career. She developed an early interest in glaucoma which resulted in a joint research project with Ida Mann into the low incidence of glaucoma amongst Māori (King Country, Bay of Plenty, East Cape, Wairarapa) compared with NZ Europeans and Afro-Americans, and she subsequently presented
the findings at various international ophthalmology meetings. She founded the NZ Glaucoma Society and later the Glaucoma Trust Fund to promote research into the disease—the work of these organisations is now embodied in Glaucoma New Zealand.

She was among the first NZ ophthalmologists to prescribe and fit contact lenses, at a time when their fitting was a much more difficult process than it is now, and was a long-time member of the NZ Contact Lens Society. During the 1970s she became interested in ocular allergy and with her colleague Dr Bruce Duncan published a paper on the subject. After developing an interest in aviation ophthalmology she helped establish the NZ branch of the Australia and New Zealand Medical Aviation Society and became a licensed examiner for the Civil Aviation Department. She also revived the defunct Wellington branch of the Medical Women’s Association in 1971 and was made an honorary life member in 1991.

Throughout her career Dorothy was a very proactive member of the Ophthalmological Society of New Zealand and became its first woman president in 1984. Her presidential address was the first of several papers she devoted to publicising the extensive work of Ida Mann (who had similarly struggled to achieve recognition in a male-dominated profession). For 5 years she toiled to obtain an official coat of arms for the Society and understandably was not an enthusiastic supporter of its amalgamation with the Royal Australian College of Ophthalmologists in 1997.

For all these endeavours Dorothy was deservedly awarded the CBE in the New Year Honours List of 1993.

Dorothy was a generous compassionate person who was tireless in seeking the best for her patients, and her energy and perseverance enabled her to be both an influential ophthalmologist and a champion for women in medicine. Despite significant resistance she achieved much in what was largely a man’s world.

Dorothy was predeceased by Charles, and is survived by her son Victor, daughter-in-law Mary, and grandchildren Melanie and Hugh.

Obituary composed by Victor Potter, Colin Fenton, and Thiers Halliwell.
Elsie Craig Gibbons

Dr Elsie Gibbons died on 17 September 2009 at the age of 94 years having devoted her professional life to the care of children.

She came from a respected Wellington Family and was educated at Samuel Marsden Collegiate School where she was a talented athlete and hockey player and was Dux of the School in 1933.

She graduated in 1941 from the Otago Medical School in a class that only included seven women. Elsie was awarded a University Blue in hockey.

She was on the Resident Staff of Wellington Hospital and later in a Wellington general practice.

In 1946 she set forth on her postgraduate training working in the London Children's Hospital, Queen Elizabeth Hackney and in Scotland. She obtained the Diploma in Child Health and in 1947 was admitted as a Member of the Royal College of Physicians of Edinburgh and then honoured with the Fellowship in 1994.

Elsie commenced her general Paediatric practice for 40 years, from her attractive home in Abel Smith Street, Wellington. She was a visiting paediatrician to the Karitane Hospital in Melrose and also to the Home of Compassion Hospital at Island Bay which in the early days provided a significant contribution to child care in the Wellington region. Mother Aubert would be proud of her.

Elsie was attached to the Kimi Ora School in Thorndon and encouraged integration of care for the physically handicapped. She was formerly the Director of the Child and Family Clinic, concentrating more on behaviour problems, working with Doctors Richard Bush, Susan Perry and the late Patricia Buckfield.

Elsie was a dynamic person with widespread interests outside the medical field. She encouraged the younger women medical graduates and was a strong leader in the Medical Women's Association and made significant contribution to the International Executive. She was on the Board of Governors of Marsden Collegiate School from 1966–71 and then on the Trust Board from 1972–99—a remarkable contribution.

In her later years developed her wonderful garden demonstrating her botanical knowledge—a bird watcher too. She was a discerning traveller with few places in the world that she missed from the Arctic to the Antarctic and from Spain to China.

Elsie retired to Eastbourne and at the Service in St Alban's Anglican Church a group of Marsden School Bellringers joined the large congregation of friends and colleagues in paying warm tributes to her life work in child care.

H J Weston wrote this obituary.
Supplementary contribution from Glenys Arthur

Dr Elsie Gibbons (30 July 1917–17 September 2009) was one of five children. She is survived by Peter and Patricia. Elsie remained single but her private life was filled by her large extended loving family. (There are 44 great great nephews and nieces.)

Elsie left college skilled in the domestic crafts of a lady. She liked embroidery which she often did to her own designs. Elsie loved children, travel, nature, outdoors and reading. However, she was also independent of mind, unconventional and did not always go with the flow. She believed in education especially for women. It is therefore not surprising that Elsie decided to study medicine. Elsie's family suspected that she faced a number of hurdles in her early professional years but they said that they know little of how she felt. (Elsie later told female colleagues of some frustrations during those years.)

Elsie's third year of medicine was interrupted by travel to the United Kingdom to watch the Coronation of King George VI and to be presented at Court.

Elsie's family said that she has always been inquisitive hence her broad interests. She disliked waste and was considered frugal. Her sense of adventure challenged the outdoors. She was not afraid to rough it. In shorts, wearing a backpack and often carrying a Youth Hostel pass she was the original backpacker, Elsie's annual overseas trips were accompanied by a minimum of luggage. Her houses contained an eclectic mix of spoils from her travels—china, animat figurines, indigenous crafty things and prints. Elsie was also a successful business woman in her private paediatric practice. She invested in real estate and was admired for her shrewdness in selecting good tenants. Elsie's family thought that she was remarkable.

Elsie was a great believer in the place of women practitioners in medicine. She joined the New Zealand Medical Women's Association and became a country member when the Wellington Branch went into recession in 1939. The Wellington Branch resumed in 1971 with Elsie an enthusiastic member. She joined the executive 1975 and was President from 1977–79.

In 1972, Elsie was successfully nominated for the position of Vice President of the West Pacific Region of the Medical Women's International Association. This involved extensive travel to member countries of the West Pacific region, frequent attendance at executive meetings of MWIA in Switzerland and attendance at International Congresses in various countries. Elsie did all this at her own expense.

Colleagues and friends found Elsie a generous hostess. They loved visiting her elegant interesting homes with seeing her beautiful gardens. Many patients and staff that she worked with have expressed their appreciation of Elsie.

The kindly, interesting, fun loving person that she was Elsie will be missed.

Glenys Arthur wrote this obituary. (The assistance of Peter Gibbons and various member of the greater Gibbons family is acknowledged in writing this obituary.)
Dorothy Barnard Kelly (née Smith)  
(1907–2009)

Dorothy Kelly died in Wimbledon, UK on Sunday 11 January 2009, aged 101.

She was the daughter of Thomas Waldock Smith (1874–1953) accountant, and his wife Deborah McAdam (1875–1935), and was born at Petone, in Wellington, New Zealand, on 28 August 1907.

After attending Wellington Girls School, where she was awarded first a 3-year Junior National Scholarship in 1921 and then a senior one in 1922, she attended the University of Otago Medical School from March 1925 to December 1929. Dorothy was awarded the degrees of Bachelor of Surgery, University of New Zealand and Bachelor of Medicine on 17 July 1930.

She later worked at the Waikato Hospital, Hamilton (1931), the Auckland Hospital and the District Hospital, Whangarei (1932).

Moving to England, she finished her training at the Elizabeth Garrett Anderson Hospital in London, and worked in London's Queen's Hospital for Children in 1933. She also studied physiology and anatomy at London's University College Hospital where she met her old tutors from Otago University. They were assisting the pioneering plastic surgeons Sir Archibald McIndoe (1900–1960) and his cousin Sir Harold Gillies (1882–1960).

In April 1934, she married Daniel Kelly, then a Captain in the Indian Medical Service who came from co. Roscommon in Ireland. They had two children, a boy and a girl. Dorothy followed her husband to India where she joined the Royal Army Medical Corps, eventually gaining the honorary rank of Major and being made MBE.

However, the couple separated and she returned to England, eventually going into partnership in 1958 as a GP.

One of her partners was Dr Ilse Friedheim (d. October 1990 aged 89 years) a German Jewish refugee she had originally met in India. The senior partner was the redoubtable and well-connected Dr Norah Trouton (1893–1988). She had qualified as early as 1918 at London University as an MRCS and had also worked some years in India.

Dr Kelly continued the practice, in Battersea's Albert Bridge Road until her retirement in 1988. Living for the most part alone in Battersea she later had to move to an old people's home in Wimbledon when she had a stroke in early 2004.

Dr Kelly leaves her son and daughter, five grandchildren, and three great-grandchildren. She was buried in Hendon Cemetery, London on Wednesday 29 January 2009.

Mark Thomas (a grandson) wrote this obituary, which appears on the Otago Medical School Alumnus Association website (http://medicalalumni.otago.ac.nz/). We thank them for the reprint permission.
Mervyn Wilfrid Archdall

1913–2009

Mervyn was born in Cambridge UK. His early years were spent in Armidale (NSW, Australia) where his father, Canon Archdall, headed a theological college. His later formative years were spent in Auckland where he attended Kings College (his father being its headmaster).

He also enjoyed 3 years at the Otago Medical School and then finished his medical training in England; then he spent 2 years at the Royal Infirmary in Edinburgh to obtain the FRCP. As a registrar at Southend-on-Sea he enlisted in the eighth army and saw service in the desert, then back to England for the D-Day invasion and eventually Germany.

As ship’s surgeon he travelled back to Auckland, where he was appointed to the consultant staff of Green Lane Hospital as a physician specialising in gerontology and in private practice with a major interest in medical insurance.

He married Peg Brown in 1950 and they had two daughters. Mervyn was a good physician and a gentleman always immaculately attired. He enjoyed gardening, trout fishing, architecture and design, and painting landscapes.

David Rogers (retired surgeon and brother in law of Mervyn) wrote this obituary.
John Stuart Boyd-Wilson


John was born in Wellington, the youngest of 2 boys and 2 girls. Educated at Kelburn Normal Primary School and Nelson College, he did his Medical Intermediate at Victoria, completing his degree at Otago University. He was on the OUSA, and as Capping Controller in 1947 was instrumental in breaking the barriers that prevented women from participating in the Capping Concert.

In 1947 he married Cog Low, a recently qualified Dunedin nurse.

In 1950 he began his house duties at Wellington Hospital, with the aim of training as a physician.

Pulmonary tuberculosis shortened his second year and he spent 6 months in Cashmere Sanitorium. Bored by the inactivity he walked out of Cashmere and in 1952 started his radiological career as a registrar. With Cog and his two daughters, Jan and Lindy, he travelled to Edinburgh and at the end of 1955 obtained his London DMRD.

At the request of Wellington neurologist, Dr Jack Bergin, he spent a year at the National Hospital for Nervous Diseases in London, studying neuroradiology. From 1956-59 he was a full time specialist radiologist in Wellington, joining the part time staff in 1960 with responsibility for neuroradiology. In the same year he started his private practice in Brandon St.

These are the bare bones of John's early life and do little justice to the person he was to his family, his colleagues and to the profession. His father, Edwin Boyd-Wilson, was Professor of Modern Languages at Victoria, and instilled in John a love of language. There are family stories of a dictionary being a regular companion at the dinner table, and any reply to a poorly written letter from a colleague or family member would include the necessary correction of spelling or syntax.

This precision was evident in his skill as a builder. He single-handedly renovated Brandon St. at night and weekends, and, as Cog explains, he moved every room in their Khandallah home and put it somewhere else. Aged 60 he reroofed the house.

His father was a founder member of both the Victoria University and the Tararua Tramping Clubs, and love of the New Zealand outdoors stayed with John all his life. The family spent their holidays at Waikaremoana, in the early years camping at the far end of the lake, and more recently at their cottage at Onepoto.

He made several significant contributions to his profession. From 1972–76 he was on the committee of the RANZCR and chairman from 1976–78.
In 1971 he challenged the 30 year old schedule of fees which capped the amount a private patient could be charged above the Government approved subsidy. It would refund 2 guineas, subsequently reduced to pounds, for a barium meal and the radiologist could charge no more than 4 pounds. At his own expense he sought legal advice whether the Government could do this. It was found to be unconstitutional. The restriction was withdrawn, so making private radiology economic again.

In 1978 he prepared the Medical Association submission to the Commission of Inquiry into Chiropractic. The Association subsequently commented: "Members should be in no doubt as to the debt they owe him for the dedicated manner in which he carried out his task. Together with briefings from the Association's counsel and attendance at all 60 of the hearings, it is clear that it could only have been achieved by major inroads into busy professional time." For his work he was made a Fellow of the Association.

In 1971 he was the instigator of the move to revitalise the Medical Assurance Society. Upset at the way he was treated while seeking finance for radiology equipment for Bowen Hospital, he decided, with his good friends Gavin Glasgow and Tangi Martin, to replace the old board by seeking proxies from members. The successful six month campaign demonstrated his ability to organise and his command of the English language. His letters to members won overwhelming proxy support. He was a natural leader and lead his troops against the odds with calm courage.

Anything John undertook he did with meticulous attention to detail. He had a penetrating and original mind.

In 2001 he retired to Waikanae and typically set about changing the new house to his liking. But this was constrained by ill health and progressive disability. Until the last he was cared for by the nurse he had married sixty three years earlier, Cog, together with the support of their children, Jan, Lindy, Tim and Pete.

Dr Humphrey Rainey (General Practitioner Upper Hutt) wrote this obituary.
Murray James Loughlin

After a long and debilitating illness, Murray James Loughlin passed away peacefully at home on 17 November 2009.

Born in Wellington on 8 April 1931, Murray’s early schooling was in Auckland. In 1944 he obtained a place at Auckland Grammar School.

In 1948 he won a Medical Bursary, passing Medical Intermediate after just 1 year. Murray transferred to Otago University in 1950, and graduated MBChB in 1954 with distinction, obtaining the Casement Aitken Memorial Prize and prizes in the final surgical and O&G examinations.

House surgeon and Registrar posts followed at Dunedin and Kew Hospitals, where he gained experience in trauma, orthopaedic surgery, and hospital administration. His work ethic and surgical skills were noted favourably by senior staff.

In 1959 Murray was appointed as Medical Officer in Charge of Tapanui Hospital and also bought a general practice. Soon after he left for London for postgraduate study. He obtained his London surgical fellowship (FRCS) at the first attempt in December 1961.

After 2 years as surgical and orthopaedic registrar in England, Murray moved back to Tapanui to take charge of the hospital there, and to the practice he had previously purchased. His first appointment as Surgeon Superintendent was at Gore Hospital under the Southland Hospital Board. When he left, his departure was felt as “a grievous” loss for Southland. He moved to Wairoa Hospital from 1968–70.

Murray was appointed to the post of Medical Superintendent of Tauranga Hospital in December 1969, where he remained for 18 years. In 1973 he obtained special leave to go to Manila with the World Health Organization (WHO), in a consulting capacity to examine hospitals, their roles, architecture, and manpower.

In 1974–1975 he was appointed by the Minister of Health to be a member of the Legal and Administrative Committee, working on the White Paper—‘A Health Service for New Zealand’.

Many changes at Tauranga took place during Murray's tenure of office. Among these were: the commissioning of new outpatient and X-ray departments, and the completion of the Board’s Offices. Drafting the country’s first Service Development Plan and the development of the chaplaincy and associated facilities. The establishment of the roof garden was a unique event, as was the carpeting of the hospital, distinguished features of Tauranga Hospital today.
Among his many other activities and achievements were:

Honorary Life Member, NZ Medical Superintendents’ Association; Member of the NZ College of Community Medicine; Member, Board of Governors, Tauranga Boys College (1975-1981); Member, Board of Governors, Bay of Plenty Polytechnic (1980-1981); Invested as Serving Officer of the Order of St John (1980). Member of the Rotary Club of Tauranga (President 1979–1980). Member of the Bay of Plenty Hunt Club.

In November 1988 Murray retired as Superintendent of Tauranga Hospital.

In 1990–1994 through Worley Gillman Ltd, Murray had the opportunity to design projects: The first for the Asian Bank, undertaking a 15-year National Development Plan for the Government in the Philippines, and the second to review the rapidly changing role of District Hospitals in the provinces in Vietnam.

Following these projects, Murray accepted a role of Branch Medical Advisor to the local Accident and Compensation Commission (ACC). He retired due to ill health in 2005.

Murray married his first wife, Joan Cook in 1956. They had four children. Murray also has eight grandchildren and two great grandchildren.

He married his present wife, Heather in 1983. His personal interests were car restoration, photography, landscaping, and touring New Zealand in his motor-home which he designed himself.

His fortitude and strength was an inspiration to the many people who cared for him during his long illness. Heather nursed him at home with remarkable devotion and good humour. Murray died peacefully at home on 17 November 2009.

This obituary was compiled by Heather Loughlin (Murray’s wife) with assistance from Dr AW Reid.
Medical Benevolent Fund

NZMA Members, and families of deceased Members, may apply for aid when in situations of financial hardship or distress.

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
Tel: 0800 656161
Clinical Management of Thyroid Disease


This book is written for clinicians with an interest in thyroid disease, and is aimed primarily at physicians rather than surgeons.

It is comprehensive and divided into 5 parts. The first deals with the anatomy, embryology, and physiology of the normal thyroid gland.

The anatomy section is basic but as this book is not aimed at surgeons is probably adequate.

The physiology section however is very detailed and includes the genetics of thyroid hormone synthesis and common mutations.

The second part deals with paediatric thyroid disease, including screening for congenital disease. Paediatric thyroid disease is relatively uncommon and the main areas are reasonably well covered. The next 3 parts deal with adult thyroid disease, and is divided into hyper- and hypofunction, other related syndromes, and thyroid cancer.

The treatment of these conditions is heavily biased towards medical management and the sections on surgery for these conditions is very brief and doesn’t really deal very well with the surgical issues and decision making involved for these conditions.

The only thyroid cancers that are included are the well-differentiated papillary and follicular cancers and also medullary cancer. These sections are relatively brief, and MEN syndrome associated with medullary cancer is not well covered.

Overall the book covers most aspects of thyroid disease quite well. Particularly the physiology and genetics of thyroid disease. As it is aimed at physicians it is biased towards medical management of thyroid conditions, and the surgical aspects are less well covered.

The book is well written and easy to read. It will be a good reference for thyroid physicians and for those clinicians wanting to increase their knowledge on thyroid disease. It is, however, lacking in the surgical aspects of thyroid disease.

Birgit Dijkstra
Consultant Surgeon
Department of Surgery
Christchurch Hospital