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C Shaw, T Blakely, P Crampton, J Atkinson

This study examined the relationship between parental socioeconomic position and specific causes of death in New Zealand children aged 1–14 between 1981 and 1999. It showed that children from lower socioeconomic households are more likely to die from all causes of death (except for cancer). For example, children in lower socioeconomic groups are 87% more likely to die from non road traffic injury, 36% more likely to die of road traffic injury, and 81% more likely to die of ‘other’ causes of death (e.g. communicable diseases). Children in lower socioeconomic households are consistently, and unfairly, exposed to the risk factors for diverse causes of mortality.

B McNoe, J Langley, A-M Feyer

The aim of this study was to identify and describe all work-related traffic fatalities in New Zealand between 1985 and 1998 inclusive. At an average of 31 deaths per year (28% of work-related fatalities), work-related traffic crashes represent the single largest category of work-related death in New Zealand. If commuters are excluded from the estimate, the average is approximately 17 deaths per year (16% of work-related fatalities).

Toxic effects of BZP-based herbal party pills in humans: a prospective study in Christchurch, New Zealand
P Gee, S Richardson, W Woltersdorf, G Moore

Herbal party pills are now widely used by young New Zealanders. The principal ingredient of these pills is 1-benzylpiperazine (BZP); a synthetic stimulant. BZP is illegal in many countries (but not in New Zealand) and very little is known about the effects of BZP on humans. This study describes the observed patterns of human toxicity related to the use of BZP-based ‘herbal party pills’. The results of this study indicate that BZP can cause unpredictable and serious toxicity in some individuals.

Ethnic differences in nicotine metabolic rate among New Zealanders
R Lea, N Benowitz, M Green, J Fowles, A Vishvanath, S Dickson, M Lea, A Woodward, G Chambers, D Phillips

Maori have one of the highest rates of smoking in the World and smoking is responsible for about 31% of Maori deaths. Ancestral or genetic factors, which lead to differences in the way Maori metabolise nicotine, might play a role in this problem.
ESR conducted a novel study to show that Maori smokers tend to metabolise nicotine at a slower rate compared to Europeans. This knowledge may help explain why Maori are more susceptible to becoming addicted to smoking and might mean that Maori require more tailored cessation therapies based on their unique genetic and metabolic makeup.

**Secondhand smoke in New Zealand homes and cars: exposure, attitudes, and behaviours in 2004**  
J Gillespie, K Milne, N Wilson

Studies show that exposure to secondhand smoke increases the risk of serious health effects among non-smokers. This paper assesses exposure to secondhand smoke in New Zealand homes and cars, and describes people’s attitudes and behaviours to establishing smokefree settings. Smoking bans were more likely to be imposed in homes than in cars. Although most people (73.6%) surveyed said they lived in homes with self-imposed smoking bans, secondhand smoke remains a significant public health problem, especially for Maori and low-income New Zealanders. Further public health campaigns are needed to increase the number of smokefree homes and cars.

**Hong Kong case-control study of sudden unexpected infant death**  
T Nelson, K-F To, Y-Y Wong, J Dickinson, K Choi, L-M Yu, Y Ou, C-B Chow, E Wong, N Tang, M Hjelm, L Chen

A 4-year study shows that sudden infant death syndrome (SIDS) is very uncommon (0.16 per 1000 live births) in Hong Kong. As elsewhere, prone sleep position and smoking by the mother were risk factors. Bedsharing was only risky when the baby slept with someone other than the parents. Parents whose baby unexpectedly dies have a right to a diagnosis. Cause of death was often recorded as ‘unknown’ or ‘unascertained’ instead of SIDS, thus emphasising the need for a child mortality review process in Hong Kong.

**Perceptions of New Zealand adults about complementary and alternative therapies for cancer treatment**  
J Trevena, A Reeder

We report the results from the first survey of adult New Zealanders’ attitudes towards complementary and alternative (CAM) therapies for cancer treatment. There was a high level of acceptance of CAM therapies: two-thirds of respondents thought that complementary therapies could be beneficial when used alongside conventional treatments, whereas one-third thought that alternative therapies could be effective when used instead of conventional treatments. We recommend that evidence regarding the efficacy of CAM therapies be made more widely accessible.
Attitudes of hospital medical practitioners to the mandatory reporting of professional misconduct
S Raniga, P Hider, D Spriggs, M Ardagh

339 New Zealand doctors answered questions about three hypothetical scenarios to examine their attitudes towards the mandatory reporting of colleagues. The scenarios were; an alcohol impaired practitioner, a senior colleague with recent behavioural change, and a surgeon expressing racist views. Most respondents indicated they would act if a colleague was falling below professional standards, although there was only limited support for mandatory reporting. Instead they preferred to consult senior colleagues or sometimes counsel the practitioner themselves.
Widening the lens on child health

Robin Kearns, Shanthi Ameratunga, Pat Neuwelt

In this issue of the Journal, Shaw, Blakely, Crampton, and Atkinson\(^1\) provide stark evidence of inequalities in child mortality across a range of causes. Their findings provide another sobering reminder that the cliché of New Zealand being ‘a great place to bring up kids’ holds true for some, but it cannot be presumed to be the case for all.

The authors admit that theirs is an analysis focussing on ‘proximal risk factors’ such as acquisition of diseases and the experience of severe injuries. The results bear further witness to the gradients of life chances experienced across Western countries. The steepest gradients in the Shaw paper belong to ‘non road traffic injury’ and ‘other’. These categories carry the potential to apportion blame through the so-called ‘accident’ of a child acting, or being acted upon, in a certain way.\(^2\)

Wisely, however, the authors point to ‘distal mechanisms’ such as policies on transport, income, and food as having ultimate influence over the grim occurrences that precipitate child deaths. Elsewhere, researchers have grappled with untangling these complex distal relations between human development and what Hertzman\(^3\) terms ‘the social/economic/psychosocial conjunction’. Clearly, a study reliant on census and mortality records cannot examine the relative importance of ‘distal’ determinants. What, then, can such work suggest to members of the health professions and research communities?

First, given their rigorous methodologies and convincing conclusions, perhaps it is now time to hold back from searching for more evidence of (and mechanisms for) inequalities, and instead divert energy towards influencing policy. The ‘distal’ determinants discussed in this study, range well beyond conventional domains of health and healthcare. It therefore behoves all involved in healthcare and research to convey health-promoting options to those crafting policy in cognate fields.

Transport planners, for instance, invariably design roads with cars and drivers pre-eminently in mind.\(^4\) The needs of other legitimate road users such as child pedestrians are seldom at the forefront of design briefs. How often have communities had to lobby for safe pedestrian crossings? While it should not necessarily be the case, it is arguably health professionals and public health researchers who are most appraised of the vulnerabilities of especially the young and old. It is incumbent on us to make a case for those whose voices are least heard.

Second, there is a need to complement quantitative analyses of indicators (such as that by Shaw et al) with indepth explorations of experience. Experience can be accessed through encountering others and observing their environments in situ. With their retreat from home visiting, members of the medical profession increasingly encounter people only within clinical and institutional settings. Healthcare interventions will always be a necessary (but not sufficient) ingredient in addressing inequalities in child health. While processes and places of everyday life most accurately reveal exposure to the distal determinants of health, the synergies between diverse domains of human experience have only recently been considered in policy.\(^5\)
A critical awareness of processes such as mobility, and places such as housing and streets, can be gained by health professionals through more sustained dialogue with social scientists as well as developing a heightened awareness of what is happening in their own neighbourhoods. Both sources of critical awareness warrant comment.

Encounters in both general practice and the so-called ‘qualitative turn’ in social science are founded on narrative. In a clinical encounter as well as in an indepth interview, the opening query invariably begins with the there words “Tell me about...”. Away from a clinical setting, this can be the invitation to articulate richly described commentaries that may not be so easily appropriated as enumerated variables. Through analysis of the ensuing narratives, we can begin to discern relations between the domains of disadvantage that converge within everyday life.

To take one example, housing need and food poverty are invariably treated as separate welfare issues. Food banks and social housing are even operated by different sectors of society. Yet, as Cheer et al demonstrated through the stories of Otara families, levels of rent were strongly influencing the ‘discounting’ of health through compromised food purchasing. Perhaps the relative lack of interest in such matters provides evidence of what Eyles and Woods termed the ‘inverse interest law’—that the more commonplace the problem and the more people affected, the less will be the medical interest.

The study by Shaw et al demonstrates that children are differentially at risk of premature death according to social class in New Zealand. However, there is a danger of under-estimating the ways in which children at large are disadvantaged in terms of health-promoting opportunities vis a vis adults. Granted, children of the affluent are generally better fed, clothed, and housed than many poorer adults. But, as a demographic cohort, we rarely acknowledge the ways that children’s freedoms are curtailed with ‘downstream’ health implications.

For instance, in our risk averse society, many children’s recreation is corralled within playgrounds, limiting their sense of adventure. Few are asked what sort of city they would prefer. Such observations reflect a society that sees children as inherently vulnerable, rather than regarding adult-installed infrastructure such as roads as inherently dangerous. The gradients of child mortality will only decrease in slope when children are seen as already being citizens rather than mere youngsters on the way to becoming adults.

One reflection of their presumed vulnerability is that our cities are increasingly populated with children who are driven to and from school. This chauffeuring is frequently claimed to be in children’s interests, yet children who are driven often express a clear preference for walking. More recent work has confirmed that children have well-formed understandings of the broad benefits of walking. The questions informing this type of qualitative research can be applied in everyday life. How often do we ask children what makes them feel safe, healthy, and hopeful? How often do we walk with our children to school, observing what interests them, as well as the behaviour of drivers?

A recent study gave children in primary schools cameras and asked them to photograph whatever they perceived as dangerous in their neighbourhood. The results were revealing. Many impediments to children’s security might pass unnoticed by adults but not by children: broken glass on the pavement, cars parked near pedestrian
crossings, dogs that bark aggressively behind fences.\textsuperscript{10} The message is we need to find new ways to listen to children. This requires us to slow down—not just on the roads, but in general so as to create space for children to be included in a broader spectrum of social life.

We are not alone in having researchers reveal dire trends in the lives and deaths of children.\textsuperscript{11} Indeed, the title of a recent book describes Australia as having ‘turned its back on children’. Significantly, however, the same volume complements grim statistical findings with chapters such as \textit{How do children flourish?} and \textit{Creating a civil society}.\textsuperscript{12} To this extent, we can learn from these Australian colleagues. For while Shaw et al serve us well in presenting a rigorous analysis of the evidence, we cannot stop at recognising gradients.

We must also ask ourselves (as well as our politicians, policymakers, and children themselves) ‘what would make all New Zealand children thrive and flourish’ and then, with courage and conviction, set about promoting and implementing policies that would create a truly civil society.

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\textbf{References:}


The hazards of driving to work

Jennie Connor, Alexandra Macmillan

McNoe and colleagues\(^1\) are to be commended for making the effort that was required to identify and enumerate work-related traffic deaths. That it should take such an effort and yield data that is still incomplete is a clear signal that we need to modify the way that such information is routinely recorded. As the authors point out, surveillance that would identify important trends in these events, such as changes due to the casualisation of labour, requires data to be accessible in a timely fashion.

By including traffic fatalities occurring while commuting, as well as in the course of work, the authors make a significant statement recognising the joint responsibility of employers and employees for the trip to work. This is an important step towards reducing such deaths. The location of workplaces relative to where employees live and the times of day employees are required to travel are seldom controlled by the employees themselves, and this is particularly so for the least well off. These can be important determinants of the risk of traffic injury.

McNoe has shown that commuting deaths are a significant fraction of all work-related injury deaths. Traffic-related deaths make up the largest group of work-related deaths (about 30%), and of these 44% occur while commuting.\(^1\) Deaths of commuters are the events most likely to have been underestimated, since the reason for the trip is poorly reported in available records. The Auckland Car Crash Injury Study\(^2\) recorded this information for all drivers in serious crashes in 1998–9 in the Auckland region. We found that 15% of drivers in crashes that resulted in hospitalisation or death were commuting, and another 5% were working at the time (unpublished data). Work-related injury prevention needs to extend its reach to the issue of commuting if this level of injury is going to be appreciably reduced.

Of course there are other hazards of driving to work, especially in situations where commuters spend long periods of time in their cars every day and where congestion is a problem. In comparison with more active modes of transport (public transport, cycling and walking), commuting by car can result in a chronic reduction in physical activity, deterioration in air quality for the whole community, and significant stress for some. The relationships between low levels of exercise and obesity, diabetes, and coronary heart disease are well known and are generating considerable concern. The health effects of traffic-related air pollution have also been estimated in New Zealand and account for more deaths than traffic crashes in Aucklanders over 30 years of age.\(^3\) While the current urban environment requires car ownership to access many vital social services, the severance effects of heavy traffic on neighbourhoods, and the isolative nature of car commuting result in a reduction in social connectedness for many.

However, any significant change to the mode of travel to work will require organisational support and changes in environmental infrastructure as well as a greater awareness of the benefits. Apart from improving the quality and quantity of public transport and better provision for pedestrians and cyclists on the roads, a
promising approach is the development of organisational travel plans. These plans aim to reduce single occupant car commuting through a variety of workplace interventions, including promotion of walking and cycling, improved organisational infrastructure for cyclists, financial assistance for public transport, and disincentives for car users.

Two complementary approaches to reducing the hazards of driving to work are needed. One is to address the issues relevant to commuting by car to and from each specific workplace, as part of work-related injury prevention. An example of this would be eliminating the need for staff to drive home in the early hours of the morning by changing shift times or providing alternative transport. The longer term, more sustainable, public health approach is to change the way people travel to and from work. This would both reduce the exposure to health risks such as the commuter traffic injury described by McNoe, and bring other health benefits to the individuals and their communities.

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References:


Caroline Shaw, Tony Blakely, Peter Crampton, June Atkinson

Abstract

Background Socioeconomic inequalities in all-cause child mortality exist in New Zealand; however the inequalities in cause-specific mortality have not been examined. This study examines child mortality inequality by household income between 1981 and 1999, by cause of death.

Methods Data was used from a record linkage study of census and mortality records of all New Zealand children aged 0–14 years on census night 1981, 1986, 1991, 1996 followed up for 3 years for specific causes of mortality between ages 1–14 years. All cohorts were combined to calculate mortality rates, rate ratios, and rates differences for each cause of death.

Results Socioeconomic differences in child mortality (low income compared to high income) were observed for injury (non road traffic) (RR 1.87, 1.35 to 2.58), road traffic injury (RR 1.36, 1.01 to 1.82), and ‘other’ causes of death (RR 1.81, 1.32 to 2.47). ‘Other’ and non-road traffic injury deaths together contributed 70% of the total gap in child mortality between the rich and the poor.

Conclusions Socioeconomic differences existed across most broad causes of child death. The major contributors to mortality inequality are diverse, suggesting that the similar distal causes of inequality (e.g. poverty) play out through a myriad of proximal causes. Fortunately there appears to be some scope for policymakers to modify some of the proximal and distal causes of these inequalities.

Summary Statistics New Zealand Security Statement—The New Zealand Census Mortality Study (NZCMS) is a study of the relationship between socioeconomic factors and mortality in New Zealand, based on the integration of anonymised population census data from Statistics New Zealand and mortality data from the New Zealand Health Information Service. The project was approved by Statistics New Zealand as a Data Laboratory project under the Microdata Access Protocols in 1997. The data-sets created by the integration process are covered by the Statistics Act and can be used for statistical purposes only. Only approved researchers who have signed Statistics New Zealand’s declaration of secrecy can access the integrated data in the Data Laboratory. (A full security statement is in a technical report at http://www.wnmeds.ac.New Zealand/nzcms-info.html.) For further information about confidentiality matters in regard to this study please contact Statistics New Zealand.

The existence of socioeconomic inequalities in all-cause child mortality in developed countries, including New Zealand, is well described.1–7 All-cause mortality includes a diverse range of causes of child mortality with dissimilar aetiologies. Despite this apparent diversity, socioeconomic gradients have been reported internationally in almost all of the common causes of child mortality. For example, socioeconomic gradients have been demonstrated in motor vehicle deaths,5,8,9 child pedestrian injury deaths,10 fire deaths,5,6,10 drowning,5,6 mortality from congenital conditions,5–7 sudden infant death syndrome,8,11 and cancer.5,7,9
This paper describes socioeconomic inequalities by cause of death groupings in New Zealand children between 1981 and 1999, and assesses which causes of death contribute most to inequality in all-cause child mortality. We then examine the pathways through which parental socioeconomic position (SEP) may be embodied in health, and expressed as child mortality. While much of the latter discussion is theoretical (and possibly reductionist due to the complexity of pathways) it attempts to move beyond simple description, and aims to consider reasons.

Methods

The data in this study came from the New Zealand Census-Mortality Study. Four population cohorts were constructed by anonymously and probabilistically linking individual census and mortality records over four time periods from 1981 to 1996, inclusive. New Zealand Health Information Service provided mortality data for 0–14 year olds for the periods 1981–84, 1986–89, 1991–94, and 1996–99. Four cohorts were created, following children aged 0–14 years on census night for 3 years, with analysis being conducted on those deaths that occurred in children aged 1–14 years. (Note that this study is not well suited to the study of infant mortality due to being a closed cohort.)

The percentage of eligible mortality records linked ranged from 66%–71% in each of the four census cohorts, and the percentage of those links estimated to be correct links was in excess of 96%. Linkage varied by age, rurality, ethnicity, and small area deprivation—so linkage weights were applied to overcome any potential misclassification bias of mortality outcome caused by differential success of linkage. For example, if 20 out of 30 deaths in one strata of sex, age, ethnicity and cause of death were linked, then a weight of $\frac{30}{20} = 1.5$ was applied to those 20 linked pairs. Non-linked census respondents were then weighted down slightly to ensure that the total weighted number of children in the cohorts equalled the census night population. Sensitivity analyses published elsewhere suggest these weights work well to adjust for (any) linkage bias.

To be included in the analysis, children must have been at their usual residence on census night, which had to be a private dwelling. All family types were included in the analysis. However an adult over the age of 16, who was also in their usual residence, had to be present on census night. These restrictions resulted in the exclusion of 7–9% of children in each cohort.

The ‘exposure’ (socioeconomic position) was measured using household income. When income was available on all adults in the house, it was collated and equivalised for household size using the New Zealand-specific Jensen equivalisation index. The equivalisation process adjusts for the number of adults and children in each household, recognising that larger families require more income to have the same standard of living as smaller families. Incomes were consumer price index adjusted to 1996 and then attached to each child in the household. Children were divided into three equal-sized income groups, with cut points of low (<NZ$20,600), medium (NZ$20,600 to NZ$33,000), and high (NZ$33,000).

The ‘outcome’ (cause of death) was divided into 6 groups: road traffic crash (RTC) (ICD9 E810-825), other injury (ICD E800-809, 826-929), cancer (ICD-140-209), congenital (ICD-740-759), suicide/homicide (ICD E950-999), and ‘other’ (all remaining ICD codes). The three most common causes of death in ‘other’ were communicable diseases, asthma, and respiratory infection. Pooled results are presented for all cohorts combined, as the purpose of this paper was to assess the socioeconomic gradient by cause of death, not assess any change over time.

Standardised rates, rate ratios, rate differences, and 95% confidence intervals were calculated across levels of income, using the age and ethnic group composition of the 1991 NZ census population as the external standard. Results were standardised by ethnicity, as: ethnicity is a strong determinant of socioeconomic position; ethnicity is also a strong determinant of health independent of socioeconomic position; and the ethnic composition of New Zealand children changed over this period.

The number of children identified as Maori or Pacific increased by 20.7% and 45% respectively, compared to a 13% decline in non-Maori/non-Pacific children between 1981 and 1999. Results are presented for both sexes together to maximise statistical power and because it is not possible for sex to confound the relationship between SEP and child mortality (i.e. whilst the child’s sex predicts child mortality, it is not associated with household measures of SEP).

The programme of work of the New Zealand Census Mortality Study has approval from the Wellington Ethics Committee (Reference number 98/7).
Results

Over the cohorts under study there were 2466 (weighted) deaths in children aged 1–14 years. The person years in each income group over the period was 2,329,754 in the low income group; 2,296,849 in the medium income group; and 2,210,060 years in the high-income group. Seven to 9% of all children were excluded from each cohort because they or their parent/caregiver were not at home on census night and an additional 1,406,383 person years (approximately 20% of children) were not available for this analysis due to missing income information.

The numbers of deaths; age and ethnicity-standardised mortality rates; rate ratios; and rate differences by cause of death are presented in Table 1 and Figure 2. Both show that socioeconomic gradients in child mortality were seen in all causes of death except cancer. Gradients were most strongly seen in injury (non road traffic), followed by ‘other’ causes of death and road traffic injuries. Point estimates suggested socioeconomic gradients for both suicide/homicide and mortality from congenital causes. However the confidence intervals included 1 for both of these causes of death. Cancer mortality was the notable exception with a pattern of increasing mortality with higher income is seen, although this was non-significant.

Table 1. Number of deaths in children aged 1-14 years; and standardised (Std) rates, rate ratios (SRR), and rate differences (SRD) by equivalised household income (1981–1999)

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Income</th>
<th>Deaths</th>
<th>Std Rate</th>
<th>95%CI</th>
<th>SRR</th>
<th>95%CI</th>
<th>SRD</th>
<th>95%CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Cause</td>
<td>Low</td>
<td>834</td>
<td>36.5</td>
<td>(33.4–39.5)</td>
<td>1.44</td>
<td>(1.25–1.66)</td>
<td>11.1</td>
<td>(6.9–15.4)</td>
</tr>
<tr>
<td></td>
<td>Medium</td>
<td>609</td>
<td>28.2</td>
<td>(25.4–30.9)</td>
<td>1.11</td>
<td>(0.95–1.29)</td>
<td>2.8</td>
<td>(-1.2–6.9)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>519</td>
<td>25.3</td>
<td>(22.4–28.3)</td>
<td>1.00</td>
<td></td>
<td>0.0</td>
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<tr>
<td>Road Traffic Crash (RTC)</td>
<td>Low</td>
<td>207</td>
<td>8.7</td>
<td>(7.2–10.2)</td>
<td>1.36</td>
<td>(1.01–1.82)</td>
<td>2.3</td>
<td>(0.1–4.4)</td>
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<td>Medium</td>
<td>132</td>
<td>6.1</td>
<td>(4.8–7.4)</td>
<td>0.96</td>
<td>(0.70–1.32)</td>
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<td>(4.9–7.9)</td>
<td>1.00</td>
<td></td>
<td>0.0</td>
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<tr>
<td>Injury (non-RTC)</td>
<td>Low</td>
<td>198</td>
<td>8.9</td>
<td>(7.3–10.4)</td>
<td>1.87</td>
<td>(1.35–2.58)</td>
<td>4.1</td>
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<td>111</td>
<td>5.3</td>
<td>(4.0–6.7)</td>
<td>1.13</td>
<td>(0.78–1.62)</td>
<td>0.6</td>
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<tr>
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<td>102</td>
<td>4.7</td>
<td>(3.5–6.0)</td>
<td>1.00</td>
<td></td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Cancer</td>
<td>Low</td>
<td>105</td>
<td>4.6</td>
<td>(3.5–5.6)</td>
<td>0.84</td>
<td>(0.60–1.17)</td>
<td>-0.9</td>
<td>(-2.6–0.8)</td>
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<tr>
<td></td>
<td>Medium</td>
<td>105</td>
<td>4.7</td>
<td>(3.6–5.8)</td>
<td>0.87</td>
<td>(0.62–1.22)</td>
<td>-0.7</td>
<td>(-2.4–1.0)</td>
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<td>High</td>
<td>108</td>
<td>5.4</td>
<td>(4.1–6.8)</td>
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<td></td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Congenital</td>
<td>Low</td>
<td>87</td>
<td>3.7</td>
<td>(2.8–4.7)</td>
<td>1.52</td>
<td>(0.95–2.43)</td>
<td>1.3</td>
<td>(-0.1–2.6)</td>
</tr>
<tr>
<td></td>
<td>Medium</td>
<td>60</td>
<td>2.9</td>
<td>(2.0–3.7)</td>
<td>1.17</td>
<td>(0.72–1.91)</td>
<td>0.4</td>
<td>(-0.9–1.7)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>45</td>
<td>2.5</td>
<td>(1.5–3.4)</td>
<td>1.00</td>
<td></td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Suicide/Homicide</td>
<td>Low</td>
<td>33</td>
<td>1.4</td>
<td>(0.8–2.0)</td>
<td>1.17</td>
<td>(0.62–2.22)</td>
<td>0.2</td>
<td>(-0.6–1.0)</td>
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<tr>
<td></td>
<td>Medium</td>
<td>39</td>
<td>1.8</td>
<td>(1.1–2.5)</td>
<td>1.53</td>
<td>(0.82–2.85)</td>
<td>0.6</td>
<td>(-0.3–1.5)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>27</td>
<td>1.2</td>
<td>(0.6–1.7)</td>
<td>1.00</td>
<td></td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Low</td>
<td>207</td>
<td>9.2</td>
<td>(7.7–10.8)</td>
<td>1.81</td>
<td>(1.32–2.47)</td>
<td>4.1</td>
<td>(2.1–6.2)</td>
</tr>
<tr>
<td></td>
<td>Medium</td>
<td>159</td>
<td>7.3</td>
<td>(5.9–8.7)</td>
<td>1.42</td>
<td>(1.03–1.97)</td>
<td>2.2</td>
<td>(0.2–4.1)</td>
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<tr>
<td></td>
<td>High</td>
<td>105</td>
<td>5.1</td>
<td>(3.8–6.5)</td>
<td>1.00</td>
<td></td>
<td>0.0</td>
<td></td>
</tr>
</tbody>
</table>

Deaths are weighted deaths, rates are age and sex standardised and per 100,000, SRD are per 100,000.
Figure 1. Mortality rates for various causes of death among children aged 1–14 by equivalised household income (1981–1999)

The chart illustrates the contribution of different causes of death to overall mortality and to absolute inequality. It demonstrates that the causes of death that contribute proportionately most to child mortality (road traffic injuries and ‘other’ causes of death) are not the same as the causes of death that contribute most to inequality. Indeed ‘other’ causes of death and non-road traffic injury contribute approximately 70% of the absolute inequality in child mortality. (These percentages were calculated by dividing the standard rate difference for the low-income group, for each cause of death, by the standard rate difference for the low-income group in all-cause mortality.)
**Discussion**

This study illustrates the inequitable distribution of child mortality in New Zealand, with socioeconomic gradients seen in most common causes of child mortality in New Zealand, despite often diverse aetiological pathways. The contribution of two very dissimilar causes of death to 70% of absolute inequality suggests some commonality of process, which leads to the outcome of child mortality.

The large study size of the combined New Zealand Census-Mortality Study cohorts allows reasonably precise estimates of cause-specific socioeconomic gradients in child mortality. However, despite the use of an entire population sample, some causes of death were simply too uncommon in New Zealand children to allow exact determination of gradients, meaning within each group there are a number of causes of death, with differing aetiology. There will also be some imprecision around the percentage contribution of differing causes of death to absolute inequality, but we are confident that this will not alter the broad findings.

Similar socioeconomic gradients were seen by maternal education (data not presented). This suggests that selection bias is not the explanation for the observed gradients (data were available for nearly all children on the educational level of their mothers). Moreover, the findings in this study are unlikely to be due to health selection (whereby parents with children who die are likely to become socioeconomically deprived in the period before death, thus ‘appearing’ to be of low socioeconomic position) as the most common cause of child mortality is injury, an unanticipated acute event.

The socioeconomic gradients by household income shown here will be confounded by other socioeconomic factors, but the intention of this paper is not to look at the
independent effect of income. Rather we use income to represent the process of social stratification of child mortality that occurs within a society.

This study also confirms that excess mortality risk is not simply a poverty-related phenomenon in New Zealand; children in the middle-income group had mortality rates that were, in the most part, higher than those in the high-income group for a number of causes of death. This has important implications for the type of public health interventions that will be required to reduce inequalities; programmes targeting children in low-income households will miss the opportunity to prevent excess mortality in middle-income children.

Figure 3 illustrates one explanatory model that can be used to consider how these socioeconomic gradients are generated and how diverse causes of death can show such similar patterns. This is a modified version of the framework proposed by Diderichson and Hallqvist (cited in Laflamme 2000). This model suggests that family SEP exposes children differentially to either specific risk(s) or health promoting assets, which avert the risk. Children may also have a different experience of this particular risk or disease depending on family socioeconomic position. This model highlights that the wider context of policy and socio-cultural environment may influence all of these layers, family socioeconomic position, and the exposure and experience of risk. The remainder of the discussion focuses on applying this model to causes of child mortality.

**Figure 3. Model of the socioeconomic determinants of fatal injury**

![Figure 3. Model of the socioeconomic determinants of fatal injury](image)

Source: Laflamme 2000

**Road traffic mortality**—Taking road traffic injuries as an example, within this group there are two main types of deaths, child pedestrian deaths, and motor vehicle passenger deaths. The aetiology of child pedestrian deaths is well studied; Table 2
illustrates the differing exposures that children of different socioeconomic groups have to both health injurious, as well as health-promoting resources.

Table 2. Mechanisms by which differential exposure to child pedestrian risk could occur

<table>
<thead>
<tr>
<th>Poor children have increased exposure to:</th>
<th>Rich children have increased exposure to</th>
</tr>
</thead>
<tbody>
<tr>
<td>· High traffic flow in neighbourhood(^{22})</td>
<td>· Parental or adult supervision while walking to or from school(^{25,27})</td>
</tr>
<tr>
<td>· Fast traffic speeds in neighbourhood(^{22,23})</td>
<td>· Safe places to play(^{28})</td>
</tr>
<tr>
<td>· High density parking in neighbourhood(^{22,23})</td>
<td>· Car ownership(^{25})</td>
</tr>
<tr>
<td>· Walking to school(^{24})</td>
<td>· Fenced driveways(^{29})</td>
</tr>
<tr>
<td>· Higher number of roads crossed while walking to school(^{25,26})</td>
<td></td>
</tr>
</tbody>
</table>

Note: References 22, 24, 25, 27, and 29 are New Zealand research.

The other main cause of mortality in this group is vehicle crashes in which children are passengers. International evidence mostly finds a socioeconomic gradient,\(^{6,10,30}\) but the precise pathways from SEP to injury are less well studied. It seems likely that children in lower socioeconomic groups are more likely to be in cars that are less crash-worthy (in both design and lack of maintenance). Children of lower socioeconomic groups may also be more likely to be in vehicle crashes through being in cars with drivers more predisposed to crash.\(^{31}\)

Factors subsequent to the event could also be important in generating socioeconomic gradients in mortality (i.e. differential experience of the risk described in the model in Figure 3). For example, children in lower socioeconomic groups (as measured by type of car driven) are less likely to be restrained by either car seats or belts,\(^{32}\) thereby being more susceptible to severe injury or mortality in the event of a crash. There is some research suggesting that, in adults, obesity is associated with increased mortality after a car crash.\(^{33}\) This has not been researched in children, but is worth consideration given the strong association between lower SEP and obesity in New Zealand children.\(^{34}\) Car factors may also play a role, as children in higher socioeconomic groups may be less likely to sustain severe or life threatening injuries thanks to features such as airbags, and intrusion bars.

**Non-road traffic injury mortality**—The most common causes of mortality in the non-road traffic injury category in 1-14 year olds in New Zealand are drowning and fire deaths.\(^{35}\) While this study was not able to look at these specific causes, international evidence supports the existence of socioeconomic differences in both drowning and fire mortality.\(^{5,6,10}\)

Risk factors associated with fires, such as poor housing conditions and parental smoking, cluster in poorer households.\(^{36}\) Moreover, children in households of higher SEP are more likely to have access to smoke alarms and telephones, both of which reduce risk of fire deaths.\(^{36,37}\)

Possible reasons for socioeconomic gradients in drowning include differing exposure to pools, differences in safety aspects around water such as parental supervision and pool fencing, and differing experience of water (studies in both New Zealand and
Australia suggest that children in lower socioeconomic groups have poorer swimming skills compared to their peers).\(^{38,39}\)

**‘Other’ mortality**—The group of ‘other’ is a heterogeneous group of mortality causes, with the largest causes of death being communicable diseases, asthma, and respiratory infections. There is evidence that risk factors for infectious diseases such as meningococcal disease and pneumonia cluster in more deprived households.\(^{40,41}\)

Given the strength of the gradients observed in this study, the specific causes of death need to be studied more closely, in order to determine where differential exposure to (and experience of) risk by SEP occurs.

**Cancer mortality**—Similar to most other international studies,\(^{42–46}\) this study found no evidence of socioeconomic gradients in child cancer mortality. (An association between increasing cancer mortality and decreasing SEP was previously reported for just the 1991-94 cohort,\(^9\) however it would appear that this was a chance finding.) This is in contrast to adults in New Zealand in whom cancer is increasingly patterned by SEP.\(^{47}\)

**Congenital mortality**—The findings in this study provide some support for the association described by others between SEP and mortality from congenital conditions,\(^5–7,48\) although understanding this relationship is complicated by the heterogeneous nature of congenital abnormalities and the multiple factors that influence mortality outcome (incidence, prevalence and case fatality). The differential distribution of risk factors for congenital anomalies such as low folic acid intake,\(^{49,50}\) obesity,\(^{50}\) and proximity to landfills,\(^{51}\) could contribute to the observed socioeconomic gradients from congenital causes of mortality.

**Suicide/homicide mortality**—The lack of any association between suicide and homicide deaths with SEP was somewhat surprising, as there is a strong relationship between suicide and lower SEP for adults in New Zealand.\(^{52}\) Evidence from the USA shows a relationship between poverty and child homicide,\(^5,6\) and there is some evidence to suggest that risk factors for these types of death are disproportionately placed in lower socioeconomic households.\(^{53}\) However, our New Zealand findings were based on a small number of deaths, reflected by the wide confidence intervals.

To understand the process of how socioeconomic inequalities lead to health outcomes, this discussion has been framed around exposure to risk and risk-experience. These are relatively proximal risk factors; the experience of risk factors for mortality are determined by more distal mechanisms, for example transport policy, determinants of income, availability of food and national policy on folic acid fortification.

In conclusion, intervening to reduce the child mortality inequalities in New Zealand shown in this paper should be a key priority for government. This study has shown that disease-related mortality and non-road traffic injury mortality are the largest contributors to child mortality inequalities in New Zealand. Addressing these issues needs to be given priority; this includes further research into inequalities in child injury morbidity and also into interventions that reduce inequalities. In addition, the influence of existing and future policy needs to be evaluated carefully for potential effects on the determinants of child health and mortality.

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References:


# Work-related fatal traffic crashes in New Zealand: 1985–1998

Bronwen McNoe, John Langley, Anne-Marie Feyer

## Abstract

**Aim** To identify and describe all work-related traffic fatalities in New Zealand between 1985 and 1998 inclusive.

**Methods** Potential cases were identified from databases held by three national agencies. The circumstances of the deaths in each fatal incident were reviewed directly from coronial files to determine work-relatedness.

**Results** The rate of work-related fatal injury involving vehicles on a public road was 2.01 per 100,000 workers per year. The rate for worker deaths was 1.11 and that for commuting deaths was 0.89 per 100,000 workers per year.

**Conclusions** There is a substantial number of work-related crash fatalities each year and these represent a sizeable portion of the total burden of work-related fatalities.

The annual rate of acute unintentional fatal injury in New Zealand is 26.0 per 100,000 person per year.\(^1\) About half of these fatalities occur as the result of motor vehicle traffic injury.\(^1\) Work-related injury also contributes substantially to the overall injury burden. In New Zealand, two studies have attempted to describe the extent of work-related fatalities, neither have included traffic fatalities.\(^2,^3\) The more recent, the Work-Related Fatal Injury Study, determined the rate of fatalities due to work-related activity was 5.03 per 100,000 workers.\(^3\)

We have identified two population-based studies that report on work-related traffic fatalities. The first was in Australia and identified that 37% of work-related fatal injury was due to motor vehicle traffic injury.\(^4\) The second study was in the United States and found that roadway crashes accounted for more than 23% of workplace fatalities.\(^5\) When bystanders to work (a person who is killed as the direct result of someone else’s work activity) are included, these figures are likely to increase further.

Current priorities and strategies for injury prevention in New Zealand are established without a good understanding of work-related traffic injury. Currently agencies responsible for routine injury data collection in New Zealand cannot provide accurate estimates of the number of work-related traffic crashes or fatalities in New Zealand. New Zealand Police do not identify in their Traffic Crash Report whether crashes were work-related or not. Crashes involving commercial vehicles are identified, but some of these commercial vehicles may have been used for non-work activity at the time of the crash.

The notifications database maintained by Occupational Safety and Health (OSH) and the national compensated claims database maintained by the Accident Compensation Corporation (ACC) do not provide an adequate base from which to estimate the extent of the problem because of reporting and coverage issues.\(^6\)
If a similar situation exists in New Zealand to the United States and Australia, where the largest single category of work-related fatalities is traffic injury, then the absence of accurate information on the number and circumstances is a major barrier to developing priorities for injury prevention.

The aim of this study was to identify and describe all work-related traffic vehicle fatalities that occurred on a public road in New Zealand between 1985 and 1998 inclusive. This paper describes the methods used in the study and gives a synopsis of the overall findings obtained. Other papers provide an analysis of bystander deaths as well as an international comparison of work-related traffic fatalities between New Zealand, Australia, and the United States.

Methods

The methodology adopted in this study closely followed that in a similar Australian study4 so as to allow meaningful comparisons. A work-related traffic case was defined as a person who suffered a traumatic death that occurred in New Zealand and involved a traffic vehicle, to which workplace exposures contributed as a necessary factor to the death, and which can be attributed to those exposures. A traffic vehicle was defined operationally as a conveyance in which, any person or property may be transported on a public road. In other words, this study was not confined to incidents involving motor vehicles but also includes transportation such as push bikes. Work-related activity encompassed most aspects of work, including working, commuting, activity during a recess period (e.g. lunch break), at an employer sponsored social function, and during training or a non-work period if the incident arose because of work.

Three sources of data were used to identify potential deaths due to a “traffic crash” that occurred in New Zealand between 1985 and 1998. The New Zealand Health Information Service (NZHIS) maintains a record of all injury deaths within New Zealand. Those records classified with an external cause code (E-code) of traffic incident or similar (E810-E829, E846-E848, E919) were identified as potential cases.

The Land Transport Safety Authority (LTSA) maintains a database that contains the official police records of reported motor vehicle traffic crashes in New Zealand. All motor vehicle traffic fatalities in that database were initially identified as potential cases. The Accident Compensation Corporation administers New Zealand’s Accident Compensation Scheme, which provides no-fault accident insurance for all New Zealand citizens, residents, and temporary visitors to New Zealand. All work-related fatal traffic crashes resulting in Accident Compensation Corporation entitlement claims were identified from the Accident Compensation Corporation motor vehicle account as potential cases. The death must have occurred between 1 January 1985 and 31 December 1998. Exclusions from the study included those aged over 85, suicide deaths, domestic violence, persons performing home duties, criminal activity as work, and delay of greater than 1 year between the incident and death.

The absence of a work-related identifier in any of the three data sources referred to above necessitated the use of an alternate source of information to identify work-related fatalities. For this study, the primary source of data to determine work-relatedness for this study was from coronial inquest files. Certain deaths, including unnatural deaths are notifiable to the police and the coroner. The role of the coroner in these cases is to establish the cause and manner of death by way of a coronial inquest. Although work-relatedness of an incident is not required, in many instances information recorded within the coronial file can be used to make a determination of work-relatedness.

From the three source databases, 12,519 potential cases were identified. These were electronically matched by the victims’ name to the coronial register, which contains a list of decedents name, date of birth, and coronial inquest number. The commercially available data linkage software Automatch7 was used for the matching process. Cases that remained unlinked were hand matched, where possible, with a coronial number. The matching process produced 10,993 coronial numbers (88%). It is likely that some duplication existed within the remaining 1,526 files given that three data sources were used. Of the 10,993 names, coronial files were identified for 10,809 (98%) of potential cases.

Each identified file was requested in a random sequence from Coronial Services at the Department of Justice where coronial files are stored. Demographic details (name, age, sex, date of death) from the source databases were checked against the coronial file to ensure a correct match had been made.
Due to variation and inaccuracies in the format and spelling of names, both in the databases and on the coronial register, the matching process was, at times, imprecise. Up to three attempts were made to match the correct coronial file. Of the potential cases identified, 98% were correctly matched to a coronial file number. A further 15 cases that had not been identified by the source databases were identified from within coronial files reviewed. Each potential case identified was reviewed for work-relatedness. Deaths were classified as definitely work-related, possibly work-related, definitely not work-related, and indeterminate. The files for all cases identified as definitely or possibly work-related were photocopied and transferred to the study headquarters for further assessment.

Victims were classified as one of the following: worker, commuter, and bystander to work.

**Definition of a worker:**

“Persons who work for pay, profit or payment in kind, in a job, business or on a farm, and persons who worked without pay in a family business or on a farm”. This includes employees, employers and self-employed persons, working full time, part-time and ad-hoc hours.

“Persons who worked in an official volunteer capacity for an organisation”.

“Students, who comprised any person who was studying and whose death was the result of an incident, that occurred during school time, while they were performing a task directly connected with their course”.

**Definition of a commuter:**

“People who satisfied the workers definition, but died as a result of an incident that occurred while travelling directly from home to work, work to home, or between two jobs. If the incident occurred while the person was travelling in the course of their work duties, the person was not classified as a commuter but as a worker”.

**Definition of a bystander:**

“All persons who were killed directly as a result of someone else’s work activity, even though the deceased was not working at the time”.

Cases were coded in random order by three coders over a 5-month period. Demographic details of the deceased, circumstances surrounding the injury, and work details (such as industry in which fatality occurred) were some of the details recorded on worker or commuter fatalities. Because of the large number of bystanders and the lack of detail in the coronial files on the working component of the crash, bystanders were simply counted, the type of vehicle involved in the crash recorded, and (when available) the active contribution of working activity to the crash noted. Variables pertinent to the analysis presented here include: gender, age, occupation (coded according to the New Zealand Standard Classification of Occupations 1996) and industry (coded according to the Australian and New Zealand Standard Industrial Classification 1996).

Inter- and intra-rater reliability was assessed for both case determination and the coding of coronial files using a sample of cases. For case review, there was 97% agreement, both between coders and by coders over time. High levels of agreement also existed for the coding of coronial files, for most variables being higher than 90%. The two exceptions to this were for industry and occupational coding. The lower level of agreement between coders for industry and occupation (80–86%) was of concern. To ensure a higher level of reliability, every work-related coronial file was thus re-reviewed for occupation and industry, and corrections made where necessary.

Data were analysed using Statistical Procedures for the Social Sciences (SPSSx) software. Population rates per 100,000 person years and 95% confidence intervals were calculated (assuming a Poisson distribution). For the purposes of deriving rates, census data were used to approximate the number of persons of working age who were exposed to the road traffic environment, while at work, or when going to or from work. It was assumed that everyone in the working population could be exposed. Estimates of the exposed population were available by gender, age group, ethnicity, occupation, and industry. Census data were available for 1986, 1991, 1996, and 2001, with the population in the intervening years being determined by interpolation using a linear function for the inter-census years to span the time frame 1985 to 1998 inclusive. The number of workers in each level were summed over time to give an estimate of person-years at risk.
Results

The total number of coronial files reviewed was 10,809 for which 4034 files (37%) contained insufficient detail to make a work-related determination.

This study identified 234 incidents resulting in 241 deaths where the deceased was engaged in work-related activity on a public road, and a further 183 incidents resulting in 192 deaths where the deceased was commuting to or from work on a public road. In addition, although not engaged in work themselves, 1447 people died in the process of another person’s work activity on a public road (bystanders). This paper describes worker and commuter fatalities only.

The overall rate of work-related traffic fatalities of workers and commuters on a public road, between 1985 and 1998, was 2.01 per 100,000 workers per year. This was comprised of decedents working 1.11 and commuting 0.89 per 100,000 workers per year. No obvious trend in either category was apparent over the period studied.

The majority of the work-related traffic fatalities were male, for both decedents working (93%) and commuting (80%). The highest number of worker deaths occurred in the 25–34 year age group (n=64, 27%). The highest number of commuter deaths was in the 15–24 year age group (n=68, 35%). Nearly three-quarters of worker deaths (n=172, 71%) occurred between age 20 and 49. Nearly three-quarters of the commuter deaths occurred under age 40 (n=138, 72%). The mean age was substantially lower for commuter fatalities (33 years) than for worker fatalities (39 years).

The age-specific rates differed for worker and commuter fatalities (Figure 1). For workers, the rate of fatal injuries increased with increasing age group, with the highest rate observed in the 65–84 year age group. For commuters, the rate of fatalities declined with increasing age group, with the highest rate observed in the 15–24 year age group.

The only information relevant to the nature of the work, which was consistently available in the coronial files was that on the industry and occupational group of the decedents.

For decedents working, the highest number and rate of fatal injuries occurred in the transport and storage industry (10.1 per 100,000 workers per year) which accounted for about 40% of the fatalities (Table 1). The sub grouping road freight transportation had a relatively high number of workers killed (n=83), with a correspondingly high rate (33.7).

Although the number of fatalities was small, high rates were observed in milk vending (19.4), road and bridge construction (8.8), postal and courier services (6.5), plumbing services (6.3), and taxi and other road passenger transport (excluding bus transportation) (6.2).

Commuting fatalities were more evenly spread across a number of occupations. The highest numbers of fatalities occurred in manufacturing (n=36) as well as agriculture, farming, and fishing (n=33) (Table 1). The highest rates were observed in livestock farming (7.6), log saw milling (7.6), automotive repair and service (4.5), services to agriculture (4.2), plumbing services (4.2), horticulture and fruit growing (2.6), and meat processing (2.4).
The pattern observed in occupational groupings was similar to that of industry (Table 2). For the worker group, the highest number (with rates approximately 17 times the population overall) occurred in the plant and machine operators and assemblers, most notably to drivers and mobile machinery operators (111 deaths) with a rate of 21.8 per 100,000 workers (Table 1). Within this group high rates were observed in heavy truck or tanker drivers (28.6), light truck and van drivers (15.1), and taxi drivers (12.4). Agricultural/earthmoving and equipment operators also had high rates (9.1) particularly ‘earthmoving machinery operators’ (14.1) and roading/paving machine operators (38.6). Other occupations that had significantly higher rates of fatal injuries than the total working population were couriers and deliverers (10.7), and commercial travellers and sales representatives (4.9).

For commuters, the highest numbers of fatalities occurred in the plant and machine operators and assemblers and the agricultural forestry and fishing occupations. In the plant and machine operators, the deaths were predominantly to stationary machine operators and assemblers (20 deaths) and in the agricultural industry to market orientated agricultural and fishery workers (34 deaths) and market orientated animal producers (17 deaths).
<table>
<thead>
<tr>
<th>Industry Grouping</th>
<th>Workers</th>
<th></th>
<th>Rate per 100,000</th>
<th>95% C.I.</th>
<th>Commuters</th>
<th></th>
<th>Rate per 100,000</th>
<th>95% C.I.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transport, Storage</td>
<td>96</td>
<td>39.8</td>
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<td>8.2-12.3</td>
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<tr>
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<td>1.1</td>
<td>0.7-1.6</td>
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<td>0.2-1.3</td>
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<td>0.6</td>
<td>0.2-1.3</td>
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<td>0.4</td>
<td>0.1-1.0</td>
<td>1</td>
<td>0.5</td>
<td>0.1</td>
<td>0.0-0.5</td>
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<td>Cultural, Recreational Services</td>
<td>5</td>
<td>2.1</td>
<td>1.2</td>
<td>0.4-2.7</td>
<td>4</td>
<td>2.1</td>
<td>0.9</td>
<td>0.3-2.4</td>
</tr>
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<td>Property, Business Services</td>
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<td>1.7</td>
<td>0.2</td>
<td>0.1-0.6</td>
<td>9</td>
<td>4.7</td>
<td>0.5</td>
<td>0.2-0.9</td>
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<tr>
<td>Health, Community Services</td>
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<td>0.3</td>
<td>0.1-0.7</td>
<td>9</td>
<td>4.7</td>
<td>0.6</td>
<td>0.3-1.2</td>
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<td>Education</td>
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<td>0.2</td>
<td>0.0-0.6</td>
<td>6</td>
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<tr>
<td>Accommodation, Cafes, Restaurants</td>
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<td>-</td>
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<td>27</td>
<td>11.2</td>
<td>-</td>
<td>-</td>
<td>39</td>
<td>20.3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>241</strong></td>
<td></td>
<td></td>
<td></td>
<td><strong>192</strong></td>
<td></td>
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Table 2. Occupational grouping of workers and commuters of work-related fatal traffic crashes: 1985–1998

<table>
<thead>
<tr>
<th></th>
<th>Workers</th>
<th></th>
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<th></th>
<th>Commuters</th>
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<tbody>
<tr>
<td></td>
<td>Number</td>
<td>%</td>
<td>100,000</td>
<td>95% C.I.</td>
<td>Number</td>
<td>%</td>
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<tr>
<td>Plant and Machine Operators/Assemblers</td>
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<td>50.2</td>
<td>5.5</td>
<td>4.5-6.5</td>
<td>39</td>
<td>20.3</td>
<td>1.8</td>
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<td>Elementary Occupations</td>
<td>27</td>
<td>11.2</td>
<td>1.9</td>
<td>1.3-2.8</td>
<td>16</td>
<td>8.3</td>
<td>1.1</td>
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<tr>
<td>Technicians and Associate Professionals</td>
<td>23</td>
<td>9.5</td>
<td>1.0</td>
<td>0.6-1.5</td>
<td>14</td>
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<tr>
<td>Agriculture Forestry and Fishery Workers</td>
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<td>1.0</td>
<td>0.6-1.6</td>
<td>34</td>
<td>17.7</td>
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<tr>
<td>Service and Sales Workers</td>
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<td>0.4</td>
<td>0.2-0.8</td>
<td>15</td>
<td>7.8</td>
<td>0.5</td>
</tr>
<tr>
<td>Legislators, Administrators, Managers</td>
<td>9</td>
<td>3.7</td>
<td>0.4</td>
<td>0.2-0.7</td>
<td>8</td>
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<tr>
<td>Trades Workers</td>
<td>8</td>
<td>3.3</td>
<td>0.4</td>
<td>0.2-0.7</td>
<td>20</td>
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<td>Professionals</td>
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<td>0.1-0.5</td>
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<td>Clerks</td>
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<tr>
<td>Total</td>
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<td>100.0</td>
<td>-</td>
<td>-</td>
<td>192</td>
<td>100.0</td>
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</table>
The relative importance of work-related traffic crashes to other work-related injury is important in terms of prioritising areas for injury prevention interventions. Previous comparable work estimated was that approximately 75 deaths (excluding traffic crashes) occurred due to work-related activity each year. In the present series we estimated an average of 31 (worker and commuter deaths) per year. In total then we estimate there were an average of 106 work-related deaths per year. Therefore, an estimate of the contribution of work-related traffic crash (worker and commuter deaths) to the annual overall burden of work-related fatal injury was 29%. This estimate ranged between 24% and 40% for particular years in the time period studied.

Discussion

At an average of 31 deaths per year, work-related traffic crashes represent the single largest category of work-related death in New Zealand. Even if commuters are excluded from the estimate, the average is approximately 17 deaths per year and this still represents a major contributor to the annual burden of work-related death of approximately 100 per year. Because of the difficulties associated with case ascertainment, the estimates produced are likely to be a conservative, particularly for commuting incidents that are more difficult to identify from coronial files than worker fatalities. This in part is due to the lack of uniformity of recording systems for coronial findings throughout New Zealand. Direct comparisons of these results with findings elsewhere need to be treated with caution because of differences between national collections including different case definitions and case ascertainment procedures. One of the major differences is the inclusion in this study of commuter fatalities. In broad terms, however, similar patterns are observed in this study to those found elsewhere in the developed world namely that traffic injury generally forms the largest category of work-related death and the most common industry in which traffic injuries occur is road transportation.

Work-related traffic fatalities were found to be predominantly male for decedents working and commuting. This large portion of male deaths may be due to a number of factors. Firstly, it may reflect exposure. Males are undertaking nearly twice the number of commuting journeys as females. Males also typically dominate many of the occupations involving professional driving. Secondly, a bias may exist in the data, where the work component is more explicitly stated in the coronial file for males than for females. Finally, particularly in occupations such as forestry and fishery (where workers are predominantly male), it seems likely that commuting is undertaken at riskier times of the day or for longer periods. The highest number of commuting fatalities occurred in the agricultural forestry and fishing industry and in the manufacturing industry. This may reflect that these workers are commuting longer distances at riskier times of the day. In these industries, many workers start work early in the morning and will therefore travel to work at their circadian low points and may well be at risk for fatigue-related crashes.

Rates of work-related traffic injury in this study were relatively constant between ages 15 and 64 but rose markedly over the age of 65. However, the confidence intervals around the later estimates suggest it should be viewed with caution. In terms of raw numbers, approximately two-thirds of working decedents were under the age of 45.

The lower numbers of occupational fatalities observed amongst older workers in this study may reflect the age distribution of driving occupations. As professional drivers...
Age, they may be likely to move from the road to static workplace-based positions that are less physically demanding, thus decreasing their exposure to road incidents. For example, a cohort study conducted on professional drivers in the Scandinavian countries demonstrated that over a 10-year period, about one-quarter of the cohort had changed tasks within their trade (for example, from bus driver to supervisor). A further quarter had left their trade altogether.\textsuperscript{17}

Age may have a detrimental effect on risk of occupational injury in that some particular tasks, such as some physically demanding activities and continuous rapid information processing, decline in older age groups. Conversely, age may afford the opportunity for greater experience on the road, which will benefit tasks that require experience and refined skills.\textsuperscript{5,18}

The number and rate of commuting incidents was highest in the 15–24 year age group and declined with increasing age. Nearly two-thirds (64.1\%) of decedents commuting were under the age of 44. This may reflect driver behaviour in those age groups. Young people always feature prominently in crash statistics whether they are commuting or not. It may be that young people travel longer distances because they enjoy driving, because they need to travel further to obtain work, or because of where they live, all of which would increase their exposure. There is likely to be an overestimate of risk based on populations. Data on the number of driver hours exposed are not available but it is likely that a risk estimate based on these denominator would be different to that based on a population. For example, truck drivers will spend more hours on the road than plumbers. In New Zealand, currently there is no routine surveillance of work-related traffic injury nor even any means of systematically and simply identifying events. New Zealand’s changing employment climate—including the increasing casualisation of the labour force, variable working hours, and multiple job handling—may effect the rate of work-related injury on the road, but current data collection processes will be unable to detect this. Therefore, it is important that relevant agencies have a good understanding of this problem so that they can accurately and effectively identify and prioritise areas for work-related injury prevention.

This study has enabled an estimate of the burden of work-related traffic injury to New Zealand but because of delays in the availability of coronial files the information provided is not particularly timely and was very resource intensive to produce given the number of coronial files that needed to be manually reviewed. An added problem was that the information contained in coronial files often does not provide much (if any) information on the work-relatedness of the fatal incident.

Since all injury deaths are investigated by the coroner, the coronial system provides an obvious means of determining whether a death was work-related. A Bill (Coroners Bill) is currently before the New Zealand Parliament that proposes a major reform to the coronial system. This provides an excellent opportunity to put in place uniform systems for identifying important classes of death such as work-related deaths and capturing key features of these deaths in a uniform manner.

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References:

1. Injury Prevention Research Unit. URL: http://www.otago.ac.nz/ipru


Toxic effects of BZP-based herbal party pills in humans: a prospective study in Christchurch, New Zealand

Paul Gee, Sandra Richardson, Wolfram Woltersdorf, Grant Moore

Abstract

Aim This study describes patterns of human toxicity related to the use of 1-benzylpiperazine (BZP)-based ‘herbal party pills’.

Methods From 1 April 2005 to 1 September 2005 all presentations associated with party pill use were captured on a prospective data collection form.

Results There were 61 patients who presented on 80 occasions to the Emergency Department of Christchurch Hospital, New Zealand. Patients with adverse effects took an average of 4.5 tablets/capsules. Patients with mild to moderate toxicity experienced symptoms such as insomnia, anxiety, nausea, vomiting, palpitations, dystonia, and urinary retention. Some adverse reactions persisted up to 24 hours after ingestion. Fifteen toxic seizures were recorded. Two patients suffered life-threatening toxicity with status epilepticus and severe respiratory and metabolic acidosis.

Conclusions Herbal party pills have been sold without regulation since 2000, and are now widely used by young New Zealanders. The principal ingredient of these pills is 1-benzylpiperazine (BZP). They appear to have a narrow safety margin when used recreationally by some humans, possibly because of intrinsic pharmacodynamic properties, self-dosing variability, or genetic polymorphism. Those with seizure disorders or coronary disease should avoid BZP as should those taking prescription sympathomimetics or anticholinergics. Coingestion with MDMA oramphetamine should also be cautioned against. The results of this study indicate that BZP can cause unpredictable and serious toxicity in some individuals. Furthermore, the results of this study should be carefully considered in any discussion on the legal status of piperazine-based party pills.

(Herbal) party pills have become widely available and are very commonly used amongst young New Zealanders during the past 18 months (since mid-2004). These pills have been marketed as ‘herbal’ and ‘safe’. The accumulating evidence of toxicity challenges these claims, however.

Party pills are taken for their ability to increase alertness as well as elevate mood and energy. The main ingredient in most party pills in New Zealand (NZ) is 1-benzylpiperazine (BZP) which is predominantly a synthetic sympathomimetic of approximately one-tenth the potency of dexamphetamine (see Figure 1 for structural comparison). BZP is one of a family of piperazine-based psychoactive compounds. It is sometimes mixed with a similar compound trifluormethylphenylpiperazine (TFMPP) in an attempt to mimic the psychoactive effects of methylenedioxymethamphetamine (MDMA or ‘ecstasy’).
BZP is chemically synthesised and is not a naturally occurring substance. It is most commonly classified under the class of ‘designer drugs’. Most BZP on the NZ market seems to be manufactured and imported from East Asia. The chemical process to manufacture BZP is straightforward and there are reports that it is being locally manufactured in kitchens.

BZP was originally synthesised by Wellcome Research Laboratories UK as a potential anthelmintic for livestock.\textsuperscript{3} It was not used because it was relatively ineffective and caused adverse effects such as seizures in mammals. Decades later, it was found that BZP caused hyperactivity, involuntary head movements, and a reduction in reaction times in humans—reactions also associated with amphetamines.\textsuperscript{1}

A cluster of human studies was done in the 1970s to investigate BZP as a potential antidepressant drug.\textsuperscript{1,4,5} Research was halted after it was found to have subjective and physiological effects very similar to dexamphetamine. One study showed that chronic amphetamine users could not distinguish between equipotent doses of BZP and dexamphetamine.\textsuperscript{6} The researchers recommended that BZP be placed under the same statutory control as amphetamines. A BZP prodrug was investigated as an antidepressant in Hungary in the 1980s but abandoned in phase 2 trials because of adverse side effects.\textsuperscript{6} BZP is a schedule 1 illegal stimulant in the USA\textsuperscript{7} and is controlled in all states of Australia.

BZP has a complex action working directly and indirectly on central monoamine receptors. It can cause the stimulation independent release of noradrenaline as well as blocking synaptic reuptake.\textsuperscript{8}
BZP also shows amphetamine like stimulation and reuptake inhibition of dopamine (DA) and serotonin. These neurotransmitters are responsible for the psychoactive properties of BZP.\textsuperscript{9,10} The peripheral actions of BZP on alpha-2 adrenoceptors mediate reflex tachycardia and hypertension.

The pharmacokinetics and human metabolism of BZP are incompletely understood, although BZP is known to be poorly metabolised and is largely excreted unchanged by the kidneys. Staack et al have recently carried out studies on metabolic pathways and postulated several enzymatic steps.\textsuperscript{11} The cytochrome P450 enzyme system CYP2D6 appears to be a central component in the degradation of BZP. This enzyme is known for its genetic polymorphism, which may explain the erratic distribution of adverse toxic effects, especially when coadministered with other drugs such as MDMA.\textsuperscript{12} Another enzyme involved in the breakdown of BZP is catechol-O-methyltransferase (COMT), which is also known to express genetically determined variations of activity. No information is available on interactions with other prescribed or recreational drugs, effects on carrier protein binding, or toxicity of metabolites. Additive effects are likely but more research is required in the area.

BZP is occasionally misrepresented to users as the illicit drug known as ‘ecstasy’. For several years BZP has been sold free of any legal constraint. As of July 2005, BZP is legally available for sale only to adults over 18 years of age in New Zealand. It is available under at least 120 brand names/synonyms (including Frenzy, Bliss, Charge, Herbal ecstasy, A2, and Legal X). It is sold in capsules, pill, or powder form from an increasing number of retailers.

Patients presented to Christchurch Hospital’s Emergency Department (ED) with BZP toxicity as early as 4 years ago. Presentations were very infrequent up till 2004, however, when a sudden escalation began. In 2005, four to five patients per weekend have been seen with adverse and toxic effects from these pills. This increase in presentations is consistent with the increasing number of outlets seen in Christchurch. There is almost no human toxicity research available that can help us manage these cases. Experimental research was based on much smaller “therapeutic” doses. There have been recent case reports of deaths associated with BZP in combination with other sympathomimetics though no deaths attributed to BZP alone.\textsuperscript{13,14} There are no series describing BZP toxicity in humans.

**Methods**

Christchurch Hospital’s Emergency Department has an annual census of 65,000 patients, and services a city population of 340,000. All ambulance and emergency self-referrals are seen in this facility.

An increase in presentations was detected in late 2004 and a pilot retrospective audit of BZP presentations was undertaken to detect general patterns of toxicity. From this study, a standardised reporting form was developed. From 1 April 2005 to 1 September 2005 all presentations associated with party pill use were prospectively captured. Several representative cases had their hospital visits cost-analysed to estimate the financial impact of BZP patients on our institution. Selected cases with severe toxicity had urine or blood samples sent to confirm the presence of BZP or other illicit substances.

**Results**

During the 5 months of data collection (1 April 2005 to 1 September 2005), 61 patients attended a total of 80 occasions with adverse effects after ingestion of party pills. The
male to female ratio was 1:1.3. The age range was 15 to 36 years with a mean of 20.4 and a mode of 18 years (see Figure 2). Patients reported the number of pills they had taken in 61 instances (not known or unrecorded in 19 instances); the average was 4.5 tablets (range was 1 to 25). Alcohol was coingested on 39/80, marijuana in 12/80, and nitrous oxide used in 10/80 presentations respectively. Four patients used multiple illicit coningestants which included MDMA, LSD, and ritalin.

Figure 2. Age distribution of BZP users admitted to Christchurch Hospital’s Emergency Department from April to September 2005 for adverse reactions

Patients experienced symptoms such as anxiety, vomiting, headache, palpitations, confusion, collapse, and seizures. Some symptoms had persisted for up to 24 hours after ingestion. Symptoms and their frequency are listed in Figure 3.

Vital signs showed expected sympathomimetic effects in patients with tachycardia and hypertension. Electrocardiograph recordings showed all patients were in sinus rhythm and most had a sinus tachycardia. A prolonged QTc was noted in 32% of patients. All other intervals were within normal limits and no supraventricular or ventricular arrhythmias were detected. Vital sign recordings and QTc are recorded in Figure 4.

One patient presented with minor symptoms of BZP toxicity and a plasma sodium of 118 mmol/L. Serum osmolality measured 242 mosmol/kg and other biochemical and haematological indices were normal. The measurement was repeated to rule out sampling error. The sodium returned to normal 5 hours later.

Seizures after BZP-use occurred in 14 patients, with one patient having had seizures on two occasions. Seizures when witnessed or described were of the grand mal type. Seizures occurred on average 3.9 hours after reported ingestion of party pills with a range between 30 minutes and 8 hours.
Figure 3. Symptoms of BZP ingestion noted in 80 admissions of 61 patients attending Christchurch Hospital’s Emergency Department in mid-2005

Figure 4. Vital signs of BZP-toxic patients, N=80

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature mean (°C)</td>
<td>37.8 (range 34.3 to 38.6)</td>
</tr>
<tr>
<td>Heart rate mean (bpm)</td>
<td>119 (range 72–170)</td>
</tr>
<tr>
<td>Systolic blood pressure mean (mmHg)</td>
<td>140 (range 70–180)</td>
</tr>
<tr>
<td>Diastolic blood pressure mean (mmHg)</td>
<td>77 (range 70–109)</td>
</tr>
<tr>
<td>ECG QTc mean 424mS (normal &lt;430)</td>
<td>32% had a QTC between 430–490</td>
</tr>
</tbody>
</table>

Patients who had seizures appeared not to have taken more tablets than non-seizing-patients (average taken 4.3 pills vs 4.55 in non-seizing patients p=0.75).
Following are details of three cases from the severe toxicity group.

**Patient 1**—A 16-year-old female was out at a sporting event with friends. She had taken three party pills at 1900 hours (7pm) and took one more pill at 2030 hours (8:30pm). No alcohol had been used. She had no suicidal or self-harm intent. At 2300 hrs (11pm) she collapsed in a crowd and had a witnessed tonic clonic seizure. The ambulance arrived when the patient was postictal. Seizure activity started again and two doses of diazepam were required to stop the seizures. The patient was totally unresponsive, with a Glasgow Coma Score (GCS) of 3/15 and she was intubated for airway control then transferred to Christchurch Hospital. On arrival she had a heart rate of 149, a blood pressure of 70/55 mmHg, blood sugar level of 5.6 mmol/L, and a temperature of 36°C.

She had three further seizures in the Emergency Department and her first blood gas showed a severe combined metabolic and respiratory acidosis with a pH of 6.87, pCO₂ of 60 mmHg, pO₂ of 115 mmHg on supplemental oxygen, HCO₃⁻ of 10.7 mmol/L (23.0–29.0), and base excess of −23 mmol/L (-3 to +3). The patient was transferred to Intensive Care Unit (ICU). She was extubated and 12 hours later she had a GCS of 15/15. This patient had no history of seizure disorders or drug abuse. A week later she reported that she “felt unwell but better” and appeared to have suffered no apparent long-term adverse effects. Subsequent toxicological analysis of urine revealed the presence of BZP and metabolites and no other identifiable illicit drugs or alcohol.

**Patient 2**—An 18-year-old female patient had a total of five seizures and had a recorded plasma pH of 6.64 (again a mixed metabolic and respiratory acidosis). This patient was intubated and transferred to the ICU. Urinalysis from this patient also confirmed the presence of BZP with no other toxic agents. Patient 2 was subsequently extubated and recovered with no apparent long-term effects.

**Patient 3**—A 25-year-old male patient took two party pills with alcohol in the evening, then he took two more the following morning. He then had a tonic seizure 3 hours later while driving a car. The front passenger took control to avert a head-on collision and was able to bring the vehicle to a halt. The seizure lasted approximately 3 minutes followed by a postictal phase. The patient had a pulse of 170 bpm, blood pressure of 148/75 mmHg, and blood sugar level of 5.4 mmol/L. On arrival to the ED he was drowsy but conversant with no focal neurological signs. He had no known seizure disorder or alcohol dependence. Plasma biochemistry was normal; and urine showed metabolites of BZP, ethanol, and no other drugs.

During the study period benzodiazepines were administered in 14/80 cases for general agitation, in 11/80 cases for panic attacks or palpitations, and for seizures in 3/80 cases (the remaining 12 seizure cases stopped fitting spontaneously). Antiemtics and intravenous fluids were required in 11/80 cases. Two patients required urinary catheterisation for retention.

Forty-nine patients were seen and treated in the ED with an average length of stay (LOS) of 4.2 hours. The average cost of these consultations was $NZ350 per visit which includes investigations, doctor time, staff nurse time, and fixed overhead expenses. Twenty-nine patients were admitted for a period of observation (average LOS 11 hours at an average cost of $NZ500 per visit). Two patients were admitted to
the ICU then stayed a further day on an inpatient ward (average cost of $NZ3500 per visit).

**Discussion**

This study group is the largest cohort with BZP toxicity recorded internationally. This study was possible because of the unrestricted availability and use of BZP in New Zealand. Females presented with adverse effects more frequently than males. This may be because the BZP-containing party pills are not dosed per weight; therefore females being generally smaller may be taking a relatively higher dose than BZP is available in dose packages ranging from 70 mg to 1000 mg in Christchurch so analysis of pill numbers taken bears no relation to actual dose taken. This is reflected in the average number of pills taken in seizure versus non-seizure patients of 4.3 and 4.5, respectively.

Many patients take multiple doses of BZP because the first dose does not produce the desired effects immediately. Previous research confirmed that the physiological effects of BZP are not felt for up to 2 hours after oral ingestion. Slow onset of action and slow abatement of symptoms are characteristic for this drug when taken orally. Exceeding recommended package doses may result in increased toxicity with some patients experiencing palpitations and/or vomiting for up to 24 hours after ingestion. Furthermore, some users now inject BZP intravenously to experience a faster onset of action, although this is reported as being painful due to is alkalinity (raw BZP in solution has a pH≥12).

Most patients with minor toxicity present with palpitations, agitation, nausea, and vomiting. Other effects observed were intractable vomiting, confusion, and collapse. Some presented with insomnia or inability to pass urine. Most of these patients responded to reassurance, a period of observation, and very selective use of benzodiazepines. The number of patients who present to hospital for treatment probably represent a very small fraction of users in any particular weekend. Indeed, it has been estimated by the Social Tonics Association New Zealand (STANZ) that more than 8 million doses of BZP have been sold in NZ to date. Also observed (but not tallied) were patients with facial dystonia and trismus. One patient presented with minor symptoms and a plasma sodium measured at 118 mmol/L. Acute hyponatraemia has been well-described with MDMA and is possibly caused by the stimulated release of antidiuretic hormone. A similar mechanism may be responsible with BZP. There have also been reports of BZP causing either a toxic paranoid psychosis or exacerbations of existing mental illness. Such events were not observed during the study period, however.

Of greatest concern are 14 patients who had seizures after the ingestion of party drugs. BZP appears to induce toxic seizures in neurologically normal subjects. Two displayed airway compromise and metabolic derangements that were potentially fatal. It is not clear whether this is a dose-related effect as yet—one patient reported taking 12 tablets before a seizure and one reported having only taken two tablets prior to having a seizure. In animal studies, 10 mg/kg of BZP is enough to induce seizures in most laboratory rats. Genetic polymorphism in the cytochrome P450 or COMT system may possibly account for severe toxicity in some patients. One of the 14
patients had known epilepsy but the remainder had no past history of neurological disorders.

Based on this study’s results, the authors make the following recommendations for the management of BZP toxicity. Patients with seizure disorders, psychiatric illness or coronary disease should avoid BZP as should those taking prescription sympathomimetics or anticholinergics (prescription antidepressants). Coingestion with MDMA or amphetamine should also be cautioned against, as this combination could lead to fatal toxicity.\(^{12,13}\) And users should not drive for at least 8 hours after ingesting BZP.

When patients present to healthcare-facilities with BZP toxicity they should receive an electrocardiograph and an estimation of plasma sodium. Those with moderate to severe toxicity may require treatment with benzodiazepines, intravenous fluids, and antiemetics. These patients should be observed for 6–8 hours post-BZP ingestion in case of delayed seizure. Toxic seizures should be treated with benzodiazepines and airway management. Barbiturates may be required in status epilepticus.\(^{17}\)

The World Anti-Doping Agency and the New Zealand Sports Drug Agency have banned BZP in competitive sport from 2005. The Misuse of Drugs (Amendment) No. 3 Bill has now been enacted creating a new category of controlled but not banned substances (Schedule D). BZP has been placed on this schedule and it is now illegal to sell BZP to minors. The Ministry of Health (MOH) in NZ has determined that there is inadequate information about BZP to put stronger controls on its distribution at present.\(^{18}\) They have commissioned research into BZP toxicity and studies are underway at the National Poisons Centre and other centres.

More research is needed into the pharmacokinetics and dose response of BZP in humans, as is research to monitor the social impact of having designer drugs legally available in NZ. There are at least three other piperazine-based substances and other psychoactives that could potentially be marketed in NZ under Schedule D. These substances are not classified as foods, dietary supplements, or medicines so no evidence of safety in human consumption is required before they can be sold to the public.

Many users are currently taking BZP-based pills without significant adverse effects. However, the results of this study indicate that BZP can cause unpredictable and serious toxicity in some individuals. BZP is currently a legal stimulant in NZ and this status makes it available and attractive to a far wider market of users than if it were illicit. Moreover, it has propagated a culture of accepting pill use as a normal behaviour at parties. These factors should be carefully weighed in any consideration of the legal status of piperazine-based party pills.

In 2006, the Drug Policy Unit at the MOH plan to review the available evidence on the safety of BZP.

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Ethnic differences in nicotine metabolic rate among New Zealanders

Rod Lea, Neal Benowitz, Michael Green, Jeff Fowles, Anasuya Vishvanath, Stuart Dickson, Marino Lea, Alistair Woodward, Geoffrey Chambers, David Phillips

Abstract

Aims To estimate (a) the prevalence of gene variants associated with slow nicotine metabolism in the general Maori population and (b) nicotine intake and metabolic rate in Maori and European smokers.

Methods The procedure involved (a) genotyping 85 Maori participants for cytochrome P-450 2A6 (CYP2A6) gene variants, which are associated with reduced nicotine metabolic rate (ie CYP2A6*9 and *4); and (b) measuring salivary cotinine (COT) and \textit{trans}-3’-hydroxycotinine (3-HC) as biomarkers of nicotine intake and metabolic rate in 12 female smokers from the Hawke’s Bay Region (6 Maori and 6 European).

Results (a) The frequencies of the slow nicotine metabolising variants, CYP2A6*9 and *4, were significantly higher in Maori compared to European (p<0.01). Indeed, the prevalence of the CYP2A6*9 variant in these Maori was among the highest in the world (~20%). (b) In smokers, the Maori group had ~35% lower 3-HC:COT ratios indicating a reduced metabolic rate, as well as 2-fold lower cotinine levels per cigarette smoked, indicating reduced nicotine intake (p<0.05). The CYP2A6*9 allele was significantly more frequent in Maori smokers (70%) compared to Europeans (30%), p=0.03.

Conclusions The findings of this study provide evidence that Maori are genetically slower nicotine metabolisers compared to Europeans. Although more research is required, this study may help explain ethnic differences in smoking initiation and may also have important implications for smoking cessation programs—since metabolic differences between groups with varying ancestry implies that different optimal dosages of nicotine replacement therapy may be required for successful quitting.

In New Zealand (NZ), the prevalence of cigarette smoking is around 22% for the general population. However the smoking rates are markedly higher for Maori (~46%) compared to Europeans (20%) and, for reasons that are unclear, the rates for female Maori are among the highest in the world—52% nationally and up to 60% in some regions.\textsuperscript{1} Extensive targeted campaigns for smoking cessation in the 1990s has led to a reduction in tobacco consumption in NZ, yet the high prevalence of smoking for Maori has not decreased.\textsuperscript{1}

High rates of smoking are associated with elevated rates of smoking-related diseases, and it has been estimated that smoking is responsible for around 30% of Maori deaths compared to about 17% nationally.\textsuperscript{2} As these statistics emphasise, identifying the determinants of the high smoking prevalence in Maori and using this information to develop new targeted cessation strategies is of major public health importance in NZ.
Whilst cultural and economic factors contribute to ethnic differences, these do not explain all of the prevalence disparity between Maori and Europeans. Data from the 2001 NZ Census show that ethnic differences for smoking prevalence exist across all socioeconomic strata. Identifying the underlying metabolic and/or genetic differences between groups with different ancestral backgrounds may be important, since this knowledge could provide new insights into the most effective ways of reducing tobacco-related disease in Maori.

Nicotine is the primary, although not the sole, compound for initiation and maintenance of sustained smoking behaviour. Smokers tend to consume a regular number of cigarettes per day—presumably to maintain the desired pharmacological effects of nicotine. The sustained daily levels of nicotine ingested by smokers are partly determined by the rate at which nicotine is metabolised in the liver. Variation in the rate of nicotine metabolism is also thought to influence an individual’s initial risk of becoming a smoker as well as their degree of dependence on tobacco.

Benowitz and colleagues have shown that nicotine metabolic rate varies widely among individuals, and among the major ethnic/racial groups in the United States (i.e. Asian, African, and Caucasians/Europeans). In particular, these researchers showed that Chinese-American smokers exhibit (on average) a 35% reduction in nicotine metabolic rate and take in less nicotine per cigarette compared to European-American smokers.

Information on ethnic differences in nicotine metabolism may have important implications for smoking cessation programs—as a slower metabolic rate implies that lower optimal dosages of nicotine replacement therapy (NRT) may be required for certain populations of Asian origin.

After nicotine is absorbed through the lungs by cigarette smoking it is primarily (~80%) metabolised to cotinine (COT) by the liver enzyme—Cytochrome P-450 2A6 (CYP2A6). COT is subsequently metabolised by CYP2A6 to trans-3’-hydroxycotinine (3-HC). The ratio of the 3-HC and COT concentration (3-HC:COT ratio) in saliva is highly correlated with oral clearance of COT in smokers (r=0.9), which in turn reflects intrinsic metabolic clearance of nicotine by the liver via the CYP2A6 enzyme.

Therefore, a single 3-HC:COT ratio derived from a saliva sample taken first thing in the morning can be considered a reliable index of CYP2A6 activity and hence the rate of hepatic metabolism of nicotine. COT concentration on its own is highly correlated with plasma cotinine (r=0.99) and is a widely used biomarker for the dose of inhaled or ingested nicotine (i.e. nicotine intake).

Variation in CYP2A6 enzyme activity (i.e. nicotine metabolic rate) is strongly influenced by genetics with a heritability of ~60% in Caucasians. Several DNA sequence polymorphisms in the CYP2A6 gene have been associated with nicotine metabolic rate, degree of tobacco dependence, and susceptibility to smoking-related disease. Large variation in CYP2A6 allele frequencies has been observed between ethnic groups worldwide. Thus, CYP2A6 gene variants are potentially useful biomarkers for ethnic differences in nicotine metabolism and tobacco dependence.

Two variants of the CYP2A6 gene (CYP2A6*9 and *4 alleles), which have been associated with slow nicotine metabolism, are far more prevalent in Asian populations.
Specifically, individuals possessing 1 or 2 copies of CYP2A6 *9 or *4 exhibit significantly reduced, or complete absence of, nicotine metabolism via the CYP2A6 enzymatic pathway. Given the putative ancestral (genetic) links between the NZ Maori population and South East Asia we suspected similar frequencies might exist for these slow nicotine metabolising gene variants in Maori.

The present study investigated:

(a) The prevalence of the CYP2A6*9 and *4 alleles in the general Maori population, and

(b) Nicotine intake and metabolic rate in a sample of Maori and European smokers using salivary metabolites as markers of CYP2A6 enzyme activity.

**Materials and Methods**

**Participants**—(a) We estimated the population prevalence of two functionally important CYP2A6 gene variants known to be common in Asians. This was achieved by screening a pre-existing bank of Maori DNA samples (n=44), which was considered to be fairly representative of the general Maori population in terms of age, sex. For this sample, the term “Maori” was defined by (i) self-report using the 2001 census definition for ethnicity and (2) an ancestral definition—i.e. having four Maori grandparents. Smoking status of these participants was not determined. Renewed ethics approval for this aspect of the research was granted by the Wellington Ethics Committee in 2004.

(b) We also recruited 12 female smokers from the Hawke’s Bay region. Six participants were classified as “Maori” defined as described above. Because of the heritable (genetic) nature of nicotine metabolic rate, it was important to control for genetic admixture as much as possible. The Iwi (tribes) represented in the Maori group included Ngati Rakaipaaka, Ngati Kahungunu, Nga Puhi, Tainui, and Tuhoe.

A comparison group of six European female smokers with no reported Maori ancestry were also recruited from the Hawke’s Bay region and were matched to the Maori group for age. All participants were fully informed about the nature of the research and were required to sign a consent form before commencement. Ethics approval for the study was obtained from the Hawke’s Bay Ethics Committee. All participants were above 18 years of age.

**Questionnaire**—A questionnaire, designed to fit the NZ context, was used to obtain the relevant smoking information from the participants. The measures included in the questionnaire were cigarette consumption (i.e. number of cigarettes smoked per day), brand, strength and type of cigarettes/tobacco smoked as well as the time to first cigarette and Fagerstrom Test for Nicotine Dependence (FTND). (The FTND scale is commonly used to assess levels of nicotine addiction whereby a value of 0 represents low dependence and 10 is very highly dependent).

**Genotyping of CYP2A6 variants**—Participants provided buccal cell swabs for DNA analysis. The DNA was extracted and purified using commercially available BuccalAmp DNA Extraction Kits (Epicentre). We obtained good DNA yield and quality using this non-invasive sampling method.

The CYP2A6*9 single nucleotide polymorphism (SNP) was genotyped using an allele-specific PCR technique. The primers for this assay, 2A6*9S and 2A6*9AS, have been previously described by Yoshida et al. and correspond to nucleotide positions -395 to -376 and -48 to -28 on the CYP2A6 gene (Accession number AC008537) respectively.

The PCR mixture consisted of approximately 50ng of genomic DNA, 1 x PCR Buffer [67-mmol/L Tris–hydrochloric acid (pH 8.8), 16.6-mmol/L ammonium sulfate, 0.45% Triton X-100, 0.2 mg/mL gelatine and 1.5mmol/L MgCl2], 0.25-mmol/L deoxyribonucleoside triphosphate (dNTP), 0.4 μmol/L of each primer and 1U of Taq DNA polymerase in a final reaction volume of 25 μL.

Thermal cycling and agarose gel electrophoresis were performed as stated by Yoshida et al. Genotyping of the CYP2A6*4 variant was conducted using the exact primers and protocols as published in the paper by Nakajima et al.

**Measurement of nicotine intake and metabolism**—We utilised a non-invasive method to assess nicotine intake and metabolic rate in New Zealand smokers. Participants were asked to provide
approximately 1–2 ml of oral fluid (saliva) in a sterile, airtight plastic collection tube upon waking in the morning and before consuming a cigarette, coffee, or food. Samples were kept at 4°C in the participant’s home until collection. We have found that the concentration of COT and 3-HC in saliva samples does not change significantly even when stored at room temperature for 7 days (coefficient of variation < 5%) (unpublished data). The 3-HC:COT ratio determined from saliva was used as an index for nicotine metabolic rate as described by Dempsey et al.9 For smokers with fairly constant smoking habits, cotinine levels vary only by about 15% over the course of the day. COT has an in vivo half-life of approximately 24 hours. Thus, measurements of salivary COT taken upon waking in the morning were considered an indication of the previous days total ingested nicotine (intake).

Metabolite analyses of the saliva samples were performed at ESR’s accredited analytical chemistry laboratory using LC-MSMS instrumentation.

Statistical analyses—The primary test variables included in the statistical analysis were CYP2A6 genotypes, salivary COT concentration, 3-HC:COT ratio, number of cigarettes smoked per day, COT/cigarette, and FTND score. Gene frequencies between general population groups were compared statistically using chi-squared analysis.

To compare means between smoking groups, independent samples T tests were performed. Where appropriate the significance of the T Test was confirmed using an analogous non-parametric test (i.e. Mann-Whitney U). This overcomes problems associated with asymmetrically distributed data. Fisher’s Exact Test was used to compare gene frequencies between smoking groups. A p value of ≤0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 12 software.

Results

(a) Estimating prevalence of slow nicotine metabolising alleles in the general Maori population

To estimate the general population frequencies for CYP2A6*9 and *4 alleles, we generated genotype data for a group of Maori with no European grandparents. Figure 1 shows the allele frequencies for these Maori as well as other ethnic groups from around the World (data previously published13,14). The CYP2A6*9 allele ranged in frequency from around 8% in Caucasians to 15–22% in Asian groups. The variant was found to be prevalent in the Maori sample (about 20%) and was >2 times more common compared to Caucasians (p<0.001).

The CYP2A6*4 allele on average is less prevalent than CYP2A6*9 but also tended to be more frequent in Asian subgroups compared to non-Asian groups. In our Maori sample we observed a frequency of around 9% for the CYP2A6*4, which is >4 times higher than in Caucasians (p<0.001).
Figure 1. Frequency of slow nicotine metabolising alleles CYP2A6*9 (A) and CYP2A6*4 (B) among different worldwide ethnic groups (x-axis). Data for non-Maori groups are from Schoedel et al, 2002 (Caucasians, AA CNI, Chinese), and Yoshida et al, 2003 (Japanese and Korean)\textsuperscript{13,14}

A. Slow Nicotine Metabolising Allele (CYP2A6*9)

\[ n = \text{number of alleles tested in each group.} \]
B. Slow Nicotine Metabolising Allele (CYP2A6*4)
(b) Estimating nicotine intake and metabolic rate in smokers

To examine the hypothesis that differences exist for nicotine intake and metabolic rate between Maori and European smokers, we employed a non-invasive method to test smokers for both metabolic and genetic variants of CYP2A6 activity.

Descriptive analyses of the questionnaire data showed that there was variation in the strength, brand, and type of cigarettes/tobacco smoked among the participants. Of the 6 Maori smokers, 5 reported that they smoked *Port Royal* brand cigarettes whilst 1 Maori participant smoked *Horizon* brand. Of the European group, 2 participants smoked *Park Drive* brand, 2 smoked *Benson and Hedges* brand, 1 smoked *Holiday* brand, and 1 smoked *Rothmans* brand. Of the Maori smokers, 5/6 typically rolled their own cigarettes compared to only 1/6 of the European smokers.

Comparative analyses of the primary test variables are shown in Table 1. The Maori group had ~50% lower salivary COT compared to the European group (p=0.03). According to cotinine levels normalised for number of cigarettes smoked, the Maori smokers took in less nicotine per cigarette on average compared to the European smokers (p=0.002).

The 3-HC:COT ratio (nicotine metabolic rate) was significantly lower (~35%) in the Maori smokers compared to European smokers (p=0.04). Both the Maori and European group smoked an equal number of cigarettes per day on average (n=16). The FTND score was slightly higher on average in Maori smokers but not significantly different from the European group. The CYP2A6*9 allele was significantly over-represented in the female Maori smokers compared to the European smokers (70% vs 30%, respectively). The CYP2A6*4 allele was not observed in this group of smokers.

<table>
<thead>
<tr>
<th>Smoking measure</th>
<th>Maori (n=6)</th>
<th>European (n=6)</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-HC/COT ratio†</td>
<td>0.36 ± 0.06</td>
<td>0.54 ± 0.06</td>
<td>0.04</td>
</tr>
<tr>
<td>Salivary COT (ng/ml)†</td>
<td>123.06 ± 21.9</td>
<td>261.9 ± 48.3</td>
<td>0.03</td>
</tr>
<tr>
<td>No. of cigarettes per day</td>
<td>16</td>
<td>16</td>
<td>ns</td>
</tr>
<tr>
<td>COT/cigarette†</td>
<td>8.5±1.7</td>
<td>18.8±1.9</td>
<td>0.002</td>
</tr>
<tr>
<td>FTND score*</td>
<td>4</td>
<td>3</td>
<td>ns</td>
</tr>
<tr>
<td>CYP2A6*9 allele²</td>
<td>70%</td>
<td>30%</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Values are means ± SE; P values are 2-sided and were determined by independent T test. P values ≤0.05 are statistically significant. Where appropriate the significance was confirmed using non-parametric Mann-Whitney U test. *FTND is Fagerstrom Test for Nicotine Dependence; *Slow metabolising allele of the Cytochrome P450-2A6 gene.

**Discussion**

The high smoking prevalence in Maori is one of the greatest health concerns facing NZ. Characterising the nicotine metabolic profiles that are unique to Maori may help (a) explain why young Maori people seem to be more susceptible to establishing
tobacco dependence and becoming long-term smokers; and (b) inform targeted smoking cessation programs perhaps allowing more tailored NRT to be prescribed for people with Maori ancestry.

We have conducted a study to assess nicotine metabolism in New Zealand. Specifically, we determined frequencies of the CYP2A6 gene slow-metabolising variants, *9 and *4, and showed both to be significantly more prevalent in the general Maori population compared to Caucasians (see Figure 1). To estimate levels of nicotine intake and metabolic rate, we also measured nicotine metabolites in saliva from smokers belonging to the most at-risk societal group in NZ—female Maori.

Comparison of ethnic (ancestral) groups indicated that the Maori smokers had approximately 35% slower nicotine metabolic rate through the CYP2A6 liver pathway compared to Europeans. The amount of nicotine ingested per cigarette was also lower in the Maori group.

These findings suggesting that Maori metabolise nicotine more slowly are consistent with cigarette consumption data showing that Maori tend to smoke fewer cigarettes per day. Interestingly, these trends are similar to those reported in Asian smokers, who have on average a 35% slower nicotine metabolic rate and ingest less nicotine per cigarette and smoke fewer cigarettes per day compared to Caucasians. We also demonstrated a marked difference in CYP2A6*9 allele frequencies between the Maori and European smokers, which is consistent with the significant differences in general population prevalence of this variant shown in Figure 1. The genetic differences we have found support the argument that DNA variants, which translate into functional metabolic changes, should be considered when attempting to explain differences in smoking-related traits among groups with different ancestral backgrounds.

It is important to address the limitations and future directions of this work. Firstly, further analysis of a much larger sample of smokers (including males and being representative of the general population) is required to confirm the findings of the present study and to accurately estimate the difference in metabolic rate among Maori and European.

Other NZ subgroups such as Pacific Islanders should also be investigated. Larger studies will also allow statistical assessment of the ethnic variation in brand and type of cigarettes smoked. In addition, future research should include data from non-genetic modifiers of nicotine metabolic rate (e.g. caffeine and alcohol consumption) to adjust for potential confounding effects.

Nevertheless, our findings raise some interesting questions about the role of nicotine metabolism in tobacco dependence in relation to Maori. A recent prospective study of adolescent smokers has provided compelling evidence that genetically slow metabolisers have an increased risk of tobacco dependence. It was hypothesised in this report that slower nicotine inactivation may lead to prolonged and/or higher brain exposure which might enhance the initial neurophysiological processes that lead to dependence.

Therefore, our data suggesting that female Maori smokers are more likely to be genetically slower nicotine metabolisers might partially explain why young Maori females are the most likely ethnic subgroup in NZ to establish and maintain an
addiction to tobacco smoking.\textsuperscript{19} If more rigorous genetic research of Maori smokers supports this notion, then there may be a case for designing future-targeted prevention campaigns to include information about genetic/metabolic predispositions.

The results of our study might also help explain why Maori find it more difficult to quit smoking using NRT. NRT is currently the main pharmacological smoking cessation treatment in NZ and is largely subsidised by the Government.

A recent evaluation of the NZ Quitline, a telephone smoking-cessation counselling service, revealed that significantly fewer Maori (10\%) were able to quit smoking using NRT after 12 months compared to European (14\%) \textit{(P = 0.026)}.\textsuperscript{20}

It is plausible that genetically reduced nicotine metabolic rate may influence a group’s response to NRT and likelihood of quitting. Specifically, slower metabolism may mean that nicotine replacement levels, attained through patches and/or gum, are far greater than the personalised levels attained through cigarette smoking causing people to relapse to smoking due to the onset of adverse events (e.g. insomnia, nausea, and/or headache).

It is important to note, that being a slow metaboliser is not unique to Maori people and a significant proportion of individuals with no Maori ancestry are also slow nicotine metabolisers. Thus, the ultimate aim of this line of research is to utilise genetic and metabolic information for \textit{individualization} of NRT. Whilst there have been several studies aimed at customising NRT through questionnaire-based methods, none so far have investigated the utility of measuring nicotine metabolic rate as a predictor of smoking cessation.

Being able to accurately predict the rate of nicotine metabolism based on CYP2A6 enzyme activity and/or genotype could facilitate personalised dosing of NRT to ensure optimal nicotine levels are met and side effects are avoided or reduced. In turn, this should improve effectiveness of NRT for the individual and help increase the overall smoking quit rates. The implementation of new targeted cessation strategies integrating knowledge from genetics, clinical medicine, population health programmes, and tobacco legislation looks to be our best strategy for driving down the smoking prevalence in NZ and subsequently reducing the burden of smoking-related disease.

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Secondhand smoke in New Zealand homes and cars: exposure, attitudes, and behaviours in 2004

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Abstract

Aims To assess exposure to secondhand smoke (SHS) in New Zealand homes and cars and to describe attitudes and behaviours that relate to establishing smokefree settings.

Methods In 2004, a nationwide telephone survey randomly selected and interviewed 2731 respondents. This sample was weighted to represent the national population and was comprised of a general adult population sample (n=1507), a sample selected from the Maori electoral roll (n=924), and a sample of current smokers and people who had recently quit smoking (n=300).

Results 19.6% of the general population and 42.7% of the Maori sample reported current smoking. Of all current smokers, 47.2% smoked indoors at home and 70.8% smoked in their cars. Maori participants were significantly more likely to be exposed to SHS in their homes than non-Maori participants. There was also some evidence for lower socioeconomic status being related to higher SHS exposure. Extrapolating from the adult exposure data in households with children, it was estimated that 18.9% of children were potentially exposed to SHS indoors at home. However, most respondents (73.6%) lived in homes with total smoking bans.

Conclusions SHS exposure remains a significant problem especially for Maori and low-income New Zealanders. There is a need for further public health campaigns to increase the prevalence of smokefree homes and cars.

Evidence of a link between secondhand smoke (SHS) exposure and serious health effects among non-smokers was officially recognised in the mid-1980s when several scientific committees and national organisations concluded that exposure to SHS is a cause of lung cancer. Since then, numerous studies have shown that SHS exposure increases the risk of developing a range of other smoking-related illnesses, including heart disease, stroke, cancer, and respiratory illnesses, as well as many childhood illnesses.

Although several New Zealand studies have contributed to the body of evidence on the effects of (and risk factors for) SHS exposure, there are limited data at the national level on SHS-related attitudes and behaviours. One Wellington study reported low levels of awareness of the implications of passive smoking for health and a high prevalence of smoking in the presence of children. Furthermore, an investigation of hair nicotine levels in children showed there to be no significant difference in children’s hair nicotine levels whether household members reported smoking inside or outside.
One possible explanation for this may be that residents do not always enforce their rules or they may need to remove the smoking further from the house (e.g. expand the smokefree area to the whole property).

One New Zealand study, which explored socioeconomic status and exposure to SHS in a population of 7725 New Zealanders, concluded that exposure to SHS was higher among those with lower socioeconomic status. Findings from a major New Zealand cohort study concluded that adults who had never smoked and who had lived with smokers had about 15% higher premature mortality than adults who had never smoked and lived in smokefree households. This finding is supported by Woodward and colleagues who estimated that exposure to SHS causes around 350 deaths annually in New Zealand.

As well as directly harming the health of children, parental smoking behaviour has been found to be associated with smoking by New Zealand adolescents. One study reported a clear dose-response association between exposure to SHS in the home and adolescent smoking status.

The New Zealand Government has taken several legislative steps to reduce SHS-related harms experienced by non-smoking citizens. These include the passage of the Smoke-free Environments Act 1990 and subsequent amendments to this Act in 1993, 1997, and 2003. The 2003 amendments meant that, from 10 December 2004, smoking has been completely banned in enclosed areas of all workplaces, including licensed premises. This leaves private settings such as homes and cars as the main indoor environments in which non-smokers are potentially exposed to SHS.

To address the issue of SHS exposure in private settings, the New Zealand Ministry of Health contracted the Health Sponsorship Council (HSC) to develop and deliver a social marketing campaign that aimed to reduce exposure to SHS in private settings, particularly in homes. As part of the campaign’s evaluation, baseline data on SHS exposure, attitudes, and behaviours were collected prior to the April 2004 campaign launch. The aims of this study were to measure the level of exposure to SHS in New Zealand homes and cars, and to describe modifiable risk factors such as participants’ smoking behaviours in domestic settings and their attitudes towards smoking restrictions, SHS, and smoking around others.

**Methods**

**Study samples**—Three samples were drawn for the 2004 telephone survey. These included a general population sample of 1507, a current smoker and recent quitter sample of 300, and a Maori sample of 924. Fieldwork was carried out by TNS New Zealand (a market research company) using Computer Assisted Telephone Interviewing (CATI) during March/April 2004. The general population and current smoker/recent quitter samples were obtained using a random digit dialling process to access private households containing land-line numbers. This method ensured that all landline numbers, including unlisted phone-numbers and all service providers, could be accessed. Both samples included all ethnicities and were stratified to ensure that similar proportions of males and females were recruited.

To be eligible to participate in the survey, the respondent had to be at least 15 years of age, was to have the next birthday in the household, and had to have sufficient comprehension of the English language. In addition to these criteria, the current smoker/recent quitter sample required respondents to be currently smoking at least once a month or to have quit smoking in the past year.

The Maori sample was derived from electoral roll data. People who identified as Maori on the general or Maori electoral rolls were randomly selected and their names and addresses tele-matched to all landline numbers listed with the Telecom white pages. This process gave a list of numbers where there
was a higher than average probability of contacting a Maori person. Numbers were then randomly
selected from the list and contacted by interviewers. The eligibility criteria for the Maori sample were
the same as for the general population sample, with the additional criteria that the participant self-
identified as Maori.

Response rates were calculated using the following formula:

\[
\text{Response rate} = \frac{\text{number interviewed (complete and partial)}}{\text{[number interviewed (complete and partial) + number of refusals + non-contacts + other unsuccessful interviews (e.g. language difficulties)]}}
\]

The response rate for the general population sample was 26%; for the Maori sample the response rate
was 62%; and for the smoker/recent quitter sample the response rate was 61%; thus giving an overall
response rate of 42%. Relatively short survey periods (approximately 1 month) may have negatively
impacted on the response rates, as interviewers had limited time to make call-backs to non-contacts.

Assessing potential bias—Prospective participants who refused to participate in the survey were a
potential source of bias (if key characteristics among this group differed from those of survey
participants). To help assess potential selection bias, people who refused to participate in the whole
survey were asked if they would participate in a short survey to gather data on their demographic
background and reasons for not participating. The ethnicities of the non-respondents were found to be
very similar to those of the general population sample (i.e. for non-respondents compared with the
general population respectively: Maori 7.9% vs 7.5%, New Zealand European 73.6% vs 76.8%, and
Other 16.4% vs 15.4%).

There were no statistically significant differences between survey non-respondents and survey
respondents with respect to presence of children in the household and number of smokers in the
household. The main reason non-respondents gave for choosing not to participate in the survey was
that they did not have time (41.6%). Thirteen percent were not interested in taking part and 11.0%
reported that they were “in the middle of doing something”.

Data weightings—Data were analysed in SPSS version 11 software. To calculate population
probability weightings, distributions of age by ethnicity for the eligible population were obtained from
the 2001 census. These distributions were divided into smokers and non-smokers using estimates from
national smoking survey data purchased by the Ministry of Health.21

To calculate probability weightings for the combined sample (n=2731), the estimated population
frequencies by age, smoking status, and ethnicity were divided by the number of responders in each
group. Thus, the weights are proportional to the number of people in the general population that each
survey respondent represents. Probability weights applied to the Maori sample included age and
smoking status, while probability weights for the current smoker/recent quitter sample included age
and ethnicity. The magnitude of association was measured by using rate ratios.

This paper presents weighted results for all participants, representing the three samples above (n=2731)
unless stated otherwise. Results are also reported for the total Maori sample (which is made up of 1087
participants who self-identified as Maori from all three samples) and the total non-Maori sample
(which contains 1640 non-Maori participants). The non-Maori sample was made up of all respondents
who answered the ethnicity question and did not identify as Maori. Four participants did not report
their ethnicity and therefore could not be categorised into the Maori or non-Maori samples.

Results

Smoking in homes and cars—Nearly one-fifth (19.6%) of the general population
sample (n=1507) reported current smoking, that is they smoked at least once a month.

Just under half of this group (46.0%) reported that they smoked indoors at home.

Nearly one-half (42.7%) of the Maori sample (n=924) reported current smoking, and

of this group 45.4% reported that they smoked indoors at home (these samples do not
include the additional smoker/recent quitter group, n=300).

Respondents in older age groups were more likely to report that they smoked indoors
at home than younger age groups (p value for trend <0.001). For example, around
three-quarters (76.3%) of all current smokers aged 66 years and over smoked indoors
at home compared with around two-fifths (36.8%) of current smokers in the 15–18 year age group.

Respondents who were less educated were the most likely group of people to report smoking indoors at home (i.e. 18.1% of all respondents who smoked indoors at home had less than a School Certificate qualification, compared with 16.5% of respondents who had School Certificate or the equivalent and 14.5% of respondents who had the University Entrance qualification [p value for trend=0.04]).

Respondents who were on personal incomes of less than NZ$30,000 per year were slightly more likely to report smoking indoors at home than respondents who were on an income greater than $30,000 (rate ratio [RR]=1.19; 95% CI=1.01–1.41).

The main reason (40.0%) given by current smokers for not smoking inside their home was to protect other people from SHS exposure (Table 1). Of those respondents who reported current smoking and travelling in private cars (n=272), 70.8% reported that they smoked while in private cars. The most common reason given for current smokers not smoking in their cars was that they were protecting other people (in particular children) from SHS exposure (39.7%).

Table 1. Current smokers’ reasons for not smoking or not smoking more often in the home (n=788)

<table>
<thead>
<tr>
<th>Response</th>
<th>Percentage responding</th>
<th>95% confidence interval (CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protect others from secondhand smoke</td>
<td>40.0</td>
<td>32.9–47.1</td>
</tr>
<tr>
<td>Protect home (smell, burns, discolouring, etc)</td>
<td>22.5</td>
<td>16.5–28.5</td>
</tr>
<tr>
<td>Other*</td>
<td>17.4</td>
<td>14.8–20.1</td>
</tr>
<tr>
<td>Don’t know</td>
<td>8.7</td>
<td>4.6–12.8</td>
</tr>
<tr>
<td>Not a regular smoker</td>
<td>6.0</td>
<td>2.6–9.4</td>
</tr>
<tr>
<td>Not allowed to</td>
<td>4.8</td>
<td>1.7–7.9</td>
</tr>
<tr>
<td>Not my home</td>
<td>0.6</td>
<td>0.0–1.7</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

*E.g. “Like to sit in the fresh air to smoke”, “trying to cut back”, and “only smoke socially”.

Smoking around children—Those respondents who reported current smoking and had children living in their homes (n=401) were less likely to report smoking indoors at home than current smokers who did not have children at home (n=387) (RR=0.62, 95% CI=0.53–0.71). Among respondents who reported current smoking and had children living with them, almost half (45.7%), reported that they did not smoke at all when they were around children (both indoors and outdoors), while two-fifths (39.9%) reported smoking less when they were around children. There were no statistically significant differences between Maori and non-Maori respondents with respect to reported smoking behaviour around children.

Of the respondents who reported that they did not smoke at all when they were around children, nearly half (46.5%) said that this was because they did not want to expose
children to SHS. Setting a good example for children was also reported as an important reason for not smoking in the presence of children (25.6%).

**Reported SHS exposure in the home**—Maori respondents were significantly more likely than non-Maori respondents to report SHS exposure in their home during the previous 7 days (RR=1.09, 95% CI=1.02-1.17). Respondents who were exposed to SHS at home were more likely to be exposed 7 days a week rather than a few days per week (Table 2). Sixteen percent of Maori respondents and 7.9% of non-Maori respondents reported being exposed to SHS every day for the 7 days prior to the survey.

### Table 2. Reported exposure to SHS during the previous 7 days in private homes by ethnicity

<table>
<thead>
<tr>
<th>Days exposed per week</th>
<th>Maori (n=1087)</th>
<th>Non-Maori (n=1640)</th>
<th>All respondents (n=2731)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>70.4 (67.7–73.1)</td>
<td>82.3 (80.5–84.2)</td>
<td>78.2 (76.7–79.8)</td>
</tr>
<tr>
<td>1–2</td>
<td>8.2 (6.6–9.8)</td>
<td>5.5 (4.4–6.6)</td>
<td>6.5 (5.6–7.4)</td>
</tr>
<tr>
<td>3–4</td>
<td>3.3 (2.2–4.4)</td>
<td>2.5 (1.7–3.3)</td>
<td>2.7 (2.1–3.3)</td>
</tr>
<tr>
<td>5–6</td>
<td>1.6 (0.9–2.4)</td>
<td>1.3 (0.8–1.9)</td>
<td>1.4 (1.0–1.8)</td>
</tr>
<tr>
<td>7</td>
<td>16.2 (14.0–18.4)</td>
<td>7.9 (6.6–9.2)</td>
<td>10.7 (9.5–11.9)</td>
</tr>
</tbody>
</table>

**Potential exposure of children to SHS**—Table 3 shows the reported levels of adult exposure to SHS by number of children in the household. If it is assumed that children were exposed to SHS at similar frequencies to the adult survey respondent, the results suggest that most (80.7%) children in the general population live in households where there is no potential to be exposed to SHS (Table 3). However, one-fifth of children were potentially exposed (18.9%) and among these the most common frequency of exposure was likely to be every day over the 7 days prior to the survey (9.5%).

**Restrictions on smoking in the home**—Three-quarters (73.6%) of all respondents reported living in homes with total smoking bans. There were no significant differences between the proportion of Maori and non-Maori respondents who reported that they lived in homes with total smoking bans. Almost 1 in 10 respondents (9.9%) reported that smoking was allowed anywhere in their home and a further 16.2% of respondents reported that smoking was only allowed in set areas in their home.

Smoking was allowed anywhere outside the home in three-quarters (75.9%) of all respondents’ properties, while 15.3% lived in homes where smoking was allowed only in set areas outside and 8.3% had total smoking bans in outside areas. Figures were similar for both Maori and non-Maori respondents.

Over half (53.9%) of respondents who lived in households in which smoking was allowed had (at some point in the previous year) asked people to go outside if they wanted to smoke, and around one-third (34.4%) had removed items such as ashtrays, which reminded people of smoking. Nearly a quarter (22.8%) of participants who reported smoking had tried to quit smoking during the previous year.
Table 3. Adult respondents’ reported exposure to SHS in the previous 7 days (n=2731 respondents) by number of children in the household

<table>
<thead>
<tr>
<th>No. of children living in household</th>
<th>Adult respondents’ reported exposure to SHS in the home % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Not exposed</td>
</tr>
<tr>
<td>None (n=1412)</td>
<td>81.0 (79.0–83.1)</td>
</tr>
<tr>
<td>One (n=448)</td>
<td>78.4 (74.6–82.2)</td>
</tr>
<tr>
<td>Two (n=468)</td>
<td>82.1 (78.6–85.6)</td>
</tr>
<tr>
<td>Three (n=273)</td>
<td>82.8 (78.3–87.3)</td>
</tr>
<tr>
<td>Four (n=89)</td>
<td>80.6 (72.4–88.8)</td>
</tr>
<tr>
<td>≥ Five (n=41)</td>
<td>87.5 (77.4–97.6)</td>
</tr>
<tr>
<td>Any children (n=1319)</td>
<td>80.7 (78.6–82.8)</td>
</tr>
</tbody>
</table>

Attitudes towards smoking restrictions and smoking around others—Most respondents (65.0%) favoured some form of restriction on smoking inside the home (i.e. either only in set areas (34.0%) or not anywhere in the home (31.0%)) (Table 4).

Table 4. Attitudes towards smoking restrictions inside the home

<table>
<thead>
<tr>
<th>Respondent groups</th>
<th>No restrictions</th>
<th>Smoking only in set areas</th>
<th>No smoking anywhere</th>
<th>Don’t know</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maori* (n=1087)</td>
<td>26.4 (23.8–29.0)</td>
<td>45.3 (42.3–48.3)</td>
<td>24.4 (21.9–27.0)</td>
<td>3.9 (2.8–5.1)</td>
</tr>
<tr>
<td>Non-Maori* (n=1640)</td>
<td>29.8 (27.6–32.0)</td>
<td>32.5 (30.2–34.8)</td>
<td>31.9 (29.6–34.2)</td>
<td>5.7 (4.6–6.8)</td>
</tr>
<tr>
<td>Smoker (n=788)</td>
<td>39.6 (36.2–43.0)</td>
<td>44.5 (41.0–48.0)</td>
<td>12.5 (10.2–14.8)</td>
<td>3.4 (2.1–4.7)</td>
</tr>
<tr>
<td>Non-smoker (n=1943)</td>
<td>25.9 (24.0–27.9)</td>
<td>30.5 (28.5–32.6)</td>
<td>37.4 (35.3–39.6)</td>
<td>6.2 (5.1–7.3)</td>
</tr>
<tr>
<td>All respondents (n=2731)</td>
<td>29.4 (27.7–31.1)</td>
<td>34.0 (32.2–35.8)</td>
<td>31.0 (29.3–32.7)</td>
<td>5.5 (4.6–6.4)</td>
</tr>
</tbody>
</table>

*Includes smokers and non-smokers.

Respondents who reported that no one smoked inside their home (in the week prior to the survey) were more than three times more likely to agree that smoking should not be allowed anywhere inside the home, as opposed to being in set areas only or anywhere inside the home (RR=3.56, 95% CI=3.16-3.94).

Two-fifths (40.2%) of all respondents thought smoking should not be allowed in private cars. Respondents who did not smoke were the most likely group to hold this view (46.0%), while respondents who smoked at least once a month were the least likely group (23.2%).

Of all respondents who were thinking about making their home smokefree, or definitely planning to make their home smokefree in the next 30 days (n=197), 84.5% felt confident that they would be able to make their home smokefree.
The majority of respondents (92.8%) disagreed or strongly disagreed with the statement that smoking around children is acceptable (Table 5). Similarly, around three-quarters of respondents (73.0%) disagreed or strongly disagreed that smoking around people who do not smoke is acceptable. Non-smokers were significantly more likely than smokers to disagree or strongly disagree that smoking around non-smokers is acceptable (RR=1.26, 95% CI=1.08–1.47).

Around three-quarters of respondents disagreed or strongly disagreed that it is “okay” to smoke around non-smokers inside homes (72.8%) or cars (75.8%) when there are windows open. Most respondents (59.0%) disagreed with the statement that the dangers of SHS have been exaggerated. However, nearly one in three respondents agreed (28.4%) with this statement. Finally, the majority of respondents (85.3%) agreed that people have the right to live in an environment free of tobacco smoke.

Table 5. Respondent agreement/disagreement with statements about SHS and smoking around others (n=2731)

<table>
<thead>
<tr>
<th>Statement provided by interviewer</th>
<th>Level of agreement % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>“It’s OK to smoke around children”</td>
<td>Strongly agree</td>
</tr>
<tr>
<td></td>
<td>2.6 (2.0–3.2)</td>
</tr>
<tr>
<td>“It’s OK to smoke around non-smokers”</td>
<td>4.8 (4.0–5.6)</td>
</tr>
<tr>
<td>“It’s OK to smoke around non-smokers inside homes if windows are open”</td>
<td>6.1 (5.2–7.0)</td>
</tr>
<tr>
<td>“It’s OK to smoke around non-smokers inside cars if windows are open”</td>
<td>4.7 (3.9–5.5)</td>
</tr>
<tr>
<td>“Dangers of SHS have been exaggerated”</td>
<td>16.0 (14.6–17.4)</td>
</tr>
<tr>
<td>“People have the right to live in an environment free of tobacco smoke”</td>
<td>69.9 (68.2–71.6)</td>
</tr>
</tbody>
</table>

Discussion

Main findings—In this survey, 19.6% of the general population sample and 42.7% of the Maori sample reported smoking at least once a month. Smoking estimates in our survey are slightly lower than those reported in other national smoking prevalence surveys in New Zealand. For example, in 2003, the Ministry of Health reported that 25.8% of the general population and 46.4% of Maori reported any smoking. Differences in reported smoking prevalence may be due to differences in survey sampling methods, since the Ministry collects data in face-to-face interviews, as opposed to a telephone survey.
Nearly one in two respondents who smoked (47.2%) reported smoking inside their home. Those respondents who reported smoking indoors at home tended to be in the older age groups, be less educated, have lower incomes, and were less likely to have children living in their household. This pattern is consistent with a study by Whitlock and colleagues that found that respondents with lower incomes were more likely to be exposed to SHS than those respondents on higher incomes.\textsuperscript{13}

Around one-fifth of respondents (21.3%) reported being exposed to SHS at least 1 day per week, with Maori respondents significantly more likely to be exposed to SHS in their homes than non-Maori respondents. This SHS exposure is likely to be contributing to the substantial health inequalities between Maori and non-Maori in this country. Nevertheless, the differences are not as great as expected by the ethnic differences in smoking prevalence, which could be due to the relative success of smokefree home implementation by Maori whanau (families) even when smokers are present.

Nineteen percent of children were potentially exposed to SHS at least 1 day per week; the most common frequency of exposure was likely to be every day (9.5%) over the 7 days prior to the survey. Children’s potential exposure levels reported in this paper are lower than exposure levels indicated in a recent New Zealand Action on Smoking and Health (ASH) survey, in which 27.1% of Year 10 students (14–15 year-olds) reported that their parents smoked inside their home during 2004.\textsuperscript{23}

The estimates are also lower than those reported for other developed countries; e.g. in 1997 about 43% of Australian children,\textsuperscript{24} 33% of Canadian children,\textsuperscript{25} and 41% of British children\textsuperscript{26} lived with one or more parents that smoked, and so may be exposed to SHS in the home.

Almost half (45.7%) of respondents who smoked reported that they did not smoke around children, and two-fifths smoked less when they were around children. These findings were similar to a Wellington study (conducted in 1999) which reported that 51% of the respondents did not smoke around children and a further 17% smoked less when they were around children.\textsuperscript{11}

Three-quarters of respondents reported living in homes with total indoor smoking bans. However, smoking was allowed anywhere outside the home in three-quarters of all respondents’ properties. We have no information on behaviours around how rigorously such indoor smoking bans are enforced.

Most respondents (65.0%) favoured some form of restriction on smoking inside the home. Respondents who reported that no one smoked inside their home were over three times more likely to agree that smoking should not be allowed anywhere inside the home. Non-smokers were much more likely than smokers to disagree or strongly disagree that smoking around non-smokers is not acceptable.

In this survey, 40.2% of respondents thought that there should be total bans on smoking in private cars. This compares with another study conducted in Wellington that reported that 54% of respondents thought that smoking should be banned in cars when there were passengers present (note that a slightly different question was asked in this study).\textsuperscript{11}

\textbf{Limitations of this study}—Although the sub-optimal response rate is fairly typical of a CATI-type survey, it may have contributed to selection bias (as detailed in the
methodology section). Another potential source of selection bias was that some of the survey-eligible population were not able to be contacted, for reasons such as having an unlisted number (Maori sample only), or not having a land-line. Individuals who lived in large households also had less chance of being contacted (i.e. one land-line shared between more people), and so this may have lowered representation by respondents in lower SES groups, who tend to live in more crowded households and tend to be more likely to smoke. The lower proportion of smokers (relative to a national Ministry of Health survey using face-to-face interviews) may reflect such selection bias. Nevertheless, data from the non-respondent survey suggests that non-respondents were still reasonably similar to respondents, e.g. in terms of distribution by ethnicity, number of children in the household, and the proportion living with a smoker. Furthermore, a recent study concluded that there is no evidence that declining response rates in national tobacco surveys have resulted in less accurate or biased estimates of smoking behaviour. This study found that under and over-representation of population subgroups has not changed as response rates have declined.

The survey was not accompanied by any validation of self-reported smoking status and smoking-related behaviours (e.g. by testing cotinine levels or nicotine in children’s hair). Therefore it is possible that some smokers were misclassified as non-smokers and that behaviours to reduce SHS exposure of others were over-reported. Such ‘social desirability bias’ is plausible given that smoking is becoming increasingly denormalised in New Zealand society, especially in indoor settings and around children. Nevertheless, several studies have explored the reliability of self-reported cigarette consumption, with several concluding that cross-sectional surveys of self-reported smoking status are a reliable surveillance tool for monitoring changes in population smoking behaviour.

Research and policy implications—To improve the validity of future studies, more effort to achieve higher response rates may be desirable (e.g. by offering modest rewards to survey respondents or using face-to-face survey designs, albeit at greater cost). Validation studies to actually measure tobacco smoke residues in homes and cars, as well as salivary cotinine in respondents, could also be considered.

In terms of tobacco control policy, it is clear that SHS exposure in private settings remains a health hazard for a substantial proportion of Maori and low-income New Zealanders—which highlights the need for current and future public health campaigns to promote smokefree homes and cars.

Primary health workers, particularly those who carry out home visits, can also play an important role in promoting smokefree homes and cars. Improving the capacity of smoking cessation services and achieving higher quit rates is also likely to increase the prevalence of smokefree homes and cars.

The association between smoking restrictions in a range of environments and the smoking behaviour of teenagers suggests that restrictions in the home and public places can help prevent teenage smoking. New Zealand data also indicate that parental smoking behaviour and smoking restrictions in the home are associated with smoking uptake by adolescents. In the future, this information could be communicated to parents, using social marketing approaches to encourage the establishment and maintenance of smokefree homes.
One Australian study tested community support for the banning of smoking in cars while children were travelling as passengers. It reported that 72% of respondents agreed that smoking should be banned in cars in which children were present. Although there are moves in some US states to institute smokefree cars (e.g. when children are present), the practicalities of enforcing such a ban might mean that social marketing campaigns to promote smokefree cars are more appropriate. Nevertheless, many jurisdictions have successfully enforced laws around other in-car behaviours (e.g. seat belt wearing, use of child car seats, and restrictions on mobile phone use when driving).

In summary, SHS exposure in homes and cars remains a significant public health problem in New Zealand, and further efforts by the health sector and other agencies to reduce exposure to SHS (particularly for Maori and low-income New Zealanders) are needed.

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References:


Hong Kong case-control study of sudden unexpected infant death

Tony Nelson, Ka-Fai To, Yuk-Ying Wong, Jim Dickinson, Kai-chow Choi, Ly-Mee Yu, Yvonne Ou, Chun-Bong Chow, Eric Wong, Nelson Tang, Magnus Hjelm, Lawrence Chen

Abstract

Aim To document causes of all unexpected child deaths under 2 years of age during a 4-year period (1999–2003), and to identify factors associated with sudden infant death syndrome (SIDS) in Hong Kong.

Methods The case-control component of the study compared information from SIDS deaths (n=16) with healthy controls (n=223) identified randomly from all births in Hong Kong. Coroner records of all deaths under 2 years of age were later reviewed.

Results SIDS risk factors included prone sleep position, smoking by mother, bedsharing with someone other than the parents, and baby found with head covered. Eighteen deaths were officially classified as SIDS but, on review of the coroner records, there were 33 potential SIDS deaths (many labelled as unascertained/unknown).

Conclusion Hong Kong SIDS incidence has fallen from 0.3/1000 (95% CI: 0.18–0.46 in 1987) to 0.16/1000 (95% CI: 0.11–0.22 in 1999–2002). Despite the small number of cases, key SIDS risk factors are shown to be important in this population. Hong Kong needs to take steps to standardise the investigation and management of these deaths and to establish a child mortality review mechanism to provide feedback to the public, to the health authorities, and to health professionals.

Sudden infant death syndrome (SIDS) was originally defined as the sudden death of an infant or young child, which is unexpected by history, and in which a thorough postmortem examination fails to demonstrate an adequate cause of death.\(^1\) Other definitions have been suggested,\(^2\) and recently a new definitional and diagnostic approach has been proposed.\(^3\)

At a time when New Zealand was reporting very high rates of SIDS (3.6-7.4/1000 live births in 1986), Hong Kong had very low rates (0.3/1000 lives births in 1987).\(^4,5\) In the United States and Europe during the same period, reported rates were in the region of 1 to 2/1000 live births.\(^6\)

Subsequent research studies in a range of countries have shown that certain childcare practices are associated with SIDS. Placing the baby to sleep on the front (prone) has been identified to be the most important risk factor; but other factors include smoking by the mother or father, the baby sleeping in the same bed as someone else (bedsharing), baby sleeping in a room separate from the parents, and baby not using a pacifier (dummy).

A striking reduction of SIDS incidence of more than 50% has been observed in many countries after parents were advised not to place their babies on their front to sleep.\(^7\)
However the prone sleep position alone does not explain widely disparate SIDS rates, and interaction with other childcare practices appears to be important. This study aimed to identify factors associated with an increased risk of SIDS in Hong Kong and to document the causes of all unexpected child deaths under 2 years of age during a 4-year period.

Methods
This study covered the period 1 February 1999 to 31 January 2003 and was in two main parts: a case-control study and a descriptive study.

Case-control study—This compared information of those children who died (cases) with a group of children who did not die (controls). Due to data privacy concerns, cases could only be identified and contacted with the help of the mortuary staff. All families of unexpected child deaths were required to attend the mortuary for identification purposes, and at this stage, the mortuary staff sought agreement from families of suspected SIDS deaths to contact them. At the same time, arrangements were made (when possible) for a paediatric pathologist to perform the postmortem examination using a modified international standardised protocol. Potential cases were missed when the mortuary staff failed to contact the research nurse. The Beckwith definition was used to decide whether deaths were due to SIDS.

To ensure that the controls represented all children born in Hong Kong, they were selected from both government and private hospitals in proportion to the anticipated births at these hospitals. The majority of hospitals participated in this process (representing 90% of anticipated births). To obtain controls that were matched to the expected ages of the deaths, dates of interview (“nominated date”) were randomly selected using computer-generated numbers for all 1460 days of the study. The age at interview and the “nominated time” were randomly selected according to anticipated age distribution and estimated time of death distribution of the deaths (based on previous New Zealand and Hong Kong data). A date of birth was then calculated from this data. The randomly allocated “nominated time” was during one of four time periods: morning routine (6am–12 midday); afternoon routine (12 midday–6pm); evening routine (6pm–12 midnight); night-time routine (12 midnight–6am). The reference sleep was the longest sleep during this nominated time period.

Both cases and controls were interviewed at home (or at another mutually agreed venue) using a questionnaire similar to the one used by European Concerted Action on SIDS study. The Hong Kong Observatory provided meteorological data related to the estimated time of death or reference sleep.

The study was approved by the Clinical Research Ethics Committee and Survey Ethics Committee of the Chinese University of Hong Kong.

Descriptive study—This involved obtaining the details of all deaths of children under the age of 2 years that were reported to the coroner during the study period. The new Hong Kong Coroners Ordinance was published in April 1997 and implemented in 1998. The coroner decides whether or not a Death Report is required. If a Death Report is requested, a detailed investigation is undertaken by the Police. If the coroner considers that there is no likelihood of any criminal or negligent acts, then a Death Report will not usually be requested and an autopsy may not be required.

Based on the details available, deaths were provisionally classified into four groups: A = not SIDS; B = probably not SIDS; C = possibly SIDS; D = SIDS. Death reports and/or related information were then viewed in detail by the correspondence author (T Nelson) for the B, C, and D groups.

Death certificates of all deaths not reported to the coroner for the period February 1999 to December 1999 were also viewed to assess the possibility of any SIDS deaths bypassing the coroner reporting system.

Statistical analysis—For the case-control study, EpilInfo data entry and data-checking programmes based on those used in the ECAS study were used. Univariate analyses were performed using Chi-squared tests, Fisher’s exact tests or Mann-Whitney tests as appropriate. Logistic regressions (using exact method as appropriate) were employed to control for sex and socioeconomic status. The confidence intervals for the incidence of SIDS were calculated using Clopper-Pearson method. All statistical analyses, odds ratios, and their associated 95% confidence intervals calculation were done
using SPSS 11.5, StatXact-4 (version 4.0.1 Cytel Software Corporation) and LogXact-4 for Windows (version 4.1, Cytel Software Corporation). All tests were two-sided with significance level at 0.05.

**Results**

**Case-control study**—Families of 18 children who had unexpected deaths were interviewed, but two of the deaths were subsequently shown not be due to SIDS (these 2 deaths were due to ornithine carboxylase deficiency and acute bronchiolitis respectively). One paediatric pathologist (KFT) performed the postmortem examination of 16 of these 18 deaths; 11 of these 16 deaths were diagnosed as being consistent with SIDS, 2 as consistent with SIDS but with marked fatty change of the liver (suggesting potential underlying inborn error of metabolism), 1 as consistent with SIDS with an underlying obstructive uropathy, 1 as interstitial pneumonia, and 1 as a urea cycle defect (ornithine carboxylase deficiency). This latter case was excluded from the case-control analysis. Detailed discussion of this case diagnosed as interstitial pneumonia concluded that SIDS could not be excluded and hence this case was retained in the case-control analysis.

Two of the 16 autopsies were undertaken by a forensic pathologist of the Department of Health and neither death was diagnosed as SIDS. However one case, diagnosed as pneumonia, was considered to be compatible with SIDS after careful review of the history and autopsy report. The other case, a 19-month-old child diagnosed with acute bronchiolitis, was excluded. The diagnoses for the 16 children included in the case-control analysis were thus SIDS (14), interstitial pneumonia (1), and pneumonia (1).

Including the latter two cases, 7 of the 16 SIDS deaths were considered to have some atypical features on history (e.g. age over 1 year) or presence of other significant findings on autopsy (e.g. interstitial pneumonitis) that may not be considered as SIDS using stricter definitions;\(^3\) thus they were classified as group C.\(^9\)

The majority of case interviews were undertaken at the case-family’s home; some interviews took place in hospital or at another neutral venue. Although we attempted to interview case-families within 3 weeks of the death, this was not always possible and the longest delay was 3 months. Of 268 planned controls, 223 were interviewed (147 were the first control selected and 76 were one of the 3 potential backup controls approached during the postpartum period).

The majority of controls (197/223) were initially approached during the postnatal period and 26 were identified retrospectively from the labour ward delivery records. Most of the interviews were carried out within 3 days of the nominated date with one or both parents, or with the caregiver. The majority 90% (201/223) of control interviews took place in the mother’s home and the remainder at another venue such as the mother’s office, park, or restaurant. There was no significant difference between the mean age of infants who died (20.7 weeks at death) and controls (19.4 weeks at nominated date), thus showing that the method of matching for age when selecting controls, worked well.

Seventy-three percent of the controls were selected from government hospitals and 27% from private hospitals, which was very similar to the anticipated distribution of total births at these hospitals. Significant differences were noted for child’s gender, parents’ marital and socioeconomic status, mother’s age, and parents’ occupation (Table 1). Although not statistically significant, there was a trend to lower mean birth
weight for infants who died (2980 gms) than controls (3219 gms), and for lower gestational age of infants who died (37.4) than controls (39.3).

Table 1. Demographic variables for cases and controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Infants who died (n=16)</th>
<th>Controls (n=223)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baby’s sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>4 (25%)</td>
<td>124 (55.6%)</td>
<td>3.8 (1.2–12.0)*</td>
</tr>
<tr>
<td>Male</td>
<td>12 (75%)</td>
<td>99 (44.4%)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Mother’s marital status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>12 (75.0%)</td>
<td>218 (97.8%)</td>
<td>1</td>
</tr>
<tr>
<td>Cohabiting</td>
<td>4 (25.0%)</td>
<td>5 (2.2%)</td>
<td>14.5 (3.5–61.2)**</td>
</tr>
<tr>
<td><strong>Parents’ socioeconomic status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average or above</td>
<td>8 (50.0%)</td>
<td>196 (87.9%)</td>
<td>1</td>
</tr>
<tr>
<td>Below average</td>
<td>8 (50.0%)</td>
<td>27 (12.1%)</td>
<td>7.3 (2.5–20.9)**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>mean (SD)</th>
<th>mean (SD)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baby’s age at death/nominated (weeks)</td>
<td>20.7 (18.0)</td>
<td>19.4 (18.8)</td>
<td>0.7</td>
</tr>
<tr>
<td>Baby’s birth weight (gms)</td>
<td>2981 (702)</td>
<td>3219 (450)</td>
<td>0.2</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>37.4 (3.3)</td>
<td>39.3 (4.3)</td>
<td>0.07</td>
</tr>
<tr>
<td>Mother’s age (years)</td>
<td>27.6 (6.4)</td>
<td>31.6 (5.1)</td>
<td>0.02</td>
</tr>
<tr>
<td>Father’s age (years)</td>
<td>33.8 (8.8)</td>
<td>35.2 (5.9)</td>
<td>0.2</td>
</tr>
</tbody>
</table>

*p<0.05; **p<0.01.

Univariate and logistic regression analyses controlling for sex and socioeconomic status showed that recognised SIDS risk factors such as sleep position, smoking by mother and father, and bedsharing were significant in this study (Table 2). However bedsharing was only important when the infants shared a bed with someone other than the parents. Smoking was only a consistent risk factor for those mothers who were smoking during the 2nd trimester.

Amount of the last feed and illness in the last week were also significant risk factors. The latter variable combined responses from a number of variables related to the presence of symptoms, medical attention and medications being taken between 7 and 1 day before death/reference sleep or in the last 24 hours.

These included diagnosis with gastroenteritis; bronchiolitis or pneumonia; visited doctor because of illness; admitted to hospital because of illness; symptoms of fever; cough, cold, earache or vomiting; change in bowel motion or change in colour or smell of urine; or given antipyretic, cough mixture, or antibiotic. Smoking by the partner, use of an adult pillow, and outside temperature were only significant risk factors on univariate analysis.

The analyses were repeated on the subgroup of “typical SIDS” (under 1 year of age with no atypical features n=9, and controls under 1 year of age n=204), and (despite the small numbers) mother smokes, bedsharing, head and body covered, and amount of last feed remained significant after adjustment for sex and socioeconomic status.

Sleep position was not significant (only 1 of the 9 infants was in this position when left and when found). Bedsharing was only significant for the combined group (parents and other people). Illness in the last week was significant on univariate
analysis but not after adjustment. Smoking by the partner, use of an adult pillow, and outside temperature were not significant on univariate analysis (details available from the author).

**Descriptive study**—183 unexpected infant deaths were reported to the coroner during the 4-year study period and the coroner requested death reports for 52% (95/183) of these deaths. There were 94 deaths (78 with death reports and 16 without)— provisionally classified as group B, C, or D—that were reviewed in detail by the correspondence author.

The causes of death were then sub-categorised from two perspectives: from the diagnosis recorded on the death report or other coroner records; and from the diagnosis considered to be most appropriate after a detailed review (Table 3). This showed that based on the coroner diagnosis, there were 18 SIDS—but (based on the review) there were 33 potential SIDS deaths. One death could not be classified due to inadequate information. The 17 death reports that were not checked had clear diagnoses such as congenital heart disease or other illness (11), or the death report could not be located (1), or was still pending (5). There was sufficient information on 3 of the latter to confirm that the diagnosis was not SIDS.

Other coroner information was checked for the 18% (16/88) of deaths with no Death Reports but with a diagnosis that could be a potential SIDS (e.g. pneumonia, asphyxia). The 15 deaths that had not been classified by the coroner as SIDS (but were nevertheless considered to be potential SIDS upon review) had been classified as pneumonia, bronchopneumonia, aspiration pneumonia or interstitial pneumonitis (8); asphyxia due to wedging (1); and unknown, unascertained, or pending (6).

Review of 119 death certificates of deaths not reported to the coroner during the period 1 February 1999 to 31 December 1999 showed no evidence that potential SIDS deaths were bypassing the Hong Kong coronial system.

**Calculation of SIDS incidence**—During the 4-year period (1999–2002) there were 206,611 live births in Hong Kong (48,219 in 1999; 54,134 in 2000; 51,281 in 2001; 52,977 in 2002). Therefore, based on the maximum of 33 potential SIDS rather than the 18 deaths officially classified as SIDS, the incidence of SIDS in Hong Kong for the period 1999–2002 was estimated to be 0.16/1000 (95% CI: 0.11–0.22/1000). It was noted that 7 of the 33 potential SIDS infants (21%) were non-Chinese.

The 2001 Census showed that 6.2% of children under 5 years and 5.1% of the total population was non-Chinese thus indicating that the rate of SIDS in the Chinese population may be even lower. The official mortality rates (per 1000 live births) for 1999, 2000, 2001, 2002 were 3.1, 3.0, 2.6, 2.3 for infant mortality and 1.4, 1.2, 1.0, 1.0 for postneonatal mortality respectively (data from Hong Kong Census Department).
Table 2. Variables showing univariate- and sex and socioeconomic-adjusted relationship

<table>
<thead>
<tr>
<th>Variable</th>
<th>Infants who died (n=16)</th>
<th>Controls (n=223)</th>
<th>OR(u) (95% CI)†</th>
<th>OR(A) (95% CI)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleeping position on last occasion (when left)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not front</td>
<td>12 (75%)</td>
<td>217 (97.7%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Front</td>
<td>4 (25%)</td>
<td>5 (2.3%)</td>
<td>14.5 (3.4–60.9)**</td>
<td>9.1 (1.8–45.1)**</td>
</tr>
<tr>
<td>Sleeping position on last occasion (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not front</td>
<td>12 (75.0%)</td>
<td>213 (95.5%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Front</td>
<td>4 (25.0%)</td>
<td>10 (4.5%)</td>
<td>7.1 (1.9–26.0)**</td>
<td>5.0 (1.2–21.0)*</td>
</tr>
<tr>
<td>Mother smokes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before pregnancy</td>
<td>3 (19%)</td>
<td>15 (6.7%)</td>
<td>3.2 (0.8–12.5)</td>
<td>1.8 (0.4–8.4)</td>
</tr>
<tr>
<td>2nd trimester</td>
<td>3 (19%)</td>
<td>4 (1.8%)</td>
<td>12.6 (2.6–62.5)**</td>
<td>18.6 (3.0–117)**</td>
</tr>
<tr>
<td>Since birth</td>
<td>3 (19%)</td>
<td>10 (4.5%)</td>
<td>4.9 (1.2–20.1)*</td>
<td>4.6 (0.9–22.7)</td>
</tr>
<tr>
<td>Partner smokes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before pregnancy</td>
<td>10 (63%)</td>
<td>73 (33%)</td>
<td>3.4 (1.2–9.8)*</td>
<td>2.9 (0.96–8.8)</td>
</tr>
<tr>
<td>2nd trimester</td>
<td>10 (63%)</td>
<td>73 (33%)</td>
<td>3.4 (1.2–9.8)*</td>
<td>2.9 (0.96–8.8)</td>
</tr>
<tr>
<td>Since birth</td>
<td>10 (63%)</td>
<td>72 (32%)</td>
<td>3.5 (1.2–10.0)*</td>
<td>3.0 (0.99–9.1)</td>
</tr>
<tr>
<td>Who was looking after the baby (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td>6 (37.5%)</td>
<td>160 (71.7%)</td>
<td>4.2 (1.5–12.1)**</td>
<td>6.1 (1.9–20.1)**</td>
</tr>
<tr>
<td>Others</td>
<td>10 (62.5%)</td>
<td>63 (28.3%)</td>
<td>4.0 (1.4–11.3)*</td>
<td>3.6 (1.2–11)*</td>
</tr>
<tr>
<td>Bedsharing (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>7 (44%)</td>
<td>168 (76)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>9 (56%)</td>
<td>54 (24)</td>
<td>2.9 (0.9–9.5)</td>
<td>2.6 (0.7–9.5)</td>
</tr>
<tr>
<td>Bedsharing with at least one parent (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>7 (44%)</td>
<td>168 (76%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>With at least one parent</td>
<td>5 (31%)</td>
<td>42 (19%)</td>
<td>2.9 (0.9–9.5)</td>
<td>2.6 (0.7–9.5)</td>
</tr>
<tr>
<td>With others</td>
<td>4 (25%)</td>
<td>12 (5.4%)</td>
<td>8.0 (2.1–31.2)**</td>
<td>6.6 (1.5–29.3)*</td>
</tr>
<tr>
<td>Bed sharing with other people except parent(s) (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>12 (75%)</td>
<td>210 (94.6%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>4 (25%)</td>
<td>12 (5.4%)</td>
<td>5.8 (1.6–20.8)*</td>
<td>4.9 (1.2–20.2)*</td>
</tr>
<tr>
<td>Used adult pillow (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>14 (87.5%)</td>
<td>220 (98.7%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>2 (12.5%)</td>
<td>3 (1.3%)</td>
<td>10.5 (1.6–67.9)*</td>
<td>5.8 (0.7–49.2)</td>
</tr>
<tr>
<td>Used small infant’s pillow (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>10 (67%)</td>
<td>134 (60%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>5 (33%)</td>
<td>89 (40%)</td>
<td>0.8 (0.2–2.3)</td>
<td>0.7 (0.2–2.2)</td>
</tr>
<tr>
<td>Head and body totally covered (when found)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>14 (87.5%)</td>
<td>222 (99.6%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>2 (12.5%)</td>
<td>1 (0.4%)</td>
<td>31.7 (2.7–371)**</td>
<td>108 (7–1666)**</td>
</tr>
<tr>
<td>Amount of last feed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not less than usual</td>
<td>10 (62.5%)</td>
<td>218 (97.8%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Less than usual</td>
<td>6 (37.5%)</td>
<td>5 (2.2%)</td>
<td>26.2 (6.8–100.5)**</td>
<td>63 (10.7–368)**</td>
</tr>
<tr>
<td>The baby was ill in the past week</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>6 (37.5%)</td>
<td>176 (78.9%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>10 (62.5%)</td>
<td>47 (21.1%)</td>
<td>6.2 (2.2–18.1)**</td>
<td>6.1 (1.9–19.1)**</td>
</tr>
<tr>
<td>The weather was thought to be</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cold</td>
<td>4 (25.0%)</td>
<td>18 (8%)</td>
<td>2.6 (0.7–9.1)</td>
<td>2.7 (0.7–10.6)</td>
</tr>
<tr>
<td>Average</td>
<td>10 (62.5%)</td>
<td>116 (52%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Hot</td>
<td>2 (12.5%)</td>
<td>89 (40%)</td>
<td>0.3 (0.1–1.2)</td>
<td>0.3 (0.06–1.4)</td>
</tr>
<tr>
<td>Outside temperature§</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;19.5°C</td>
<td>6 (43%)</td>
<td>47 (23%)</td>
<td>4.7 (1.1–19.7)*</td>
<td>3.9 (0.9–17.8)</td>
</tr>
<tr>
<td>19.5-27.0°C</td>
<td>3 (21%)</td>
<td>111 (54%)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>&gt;27.0°C</td>
<td>5 (36%)</td>
<td>47 (23%)</td>
<td>3.9 (0.9–17.1)</td>
<td>3.2 (0.7–15.4)</td>
</tr>
</tbody>
</table>

*\(p<0.05\); **\(p<0.01\); †\(OR_u\): univariate odds ratio; ‡\(OR_A\): sex and socioeconomic status-adjusted odds ratio; §19.5 and 27°C were respectively the 25th and 75th percentiles of the temperature readings provided by the Hong Kong Observatory for the estimated time of death (cases) or nominated time (controls).
Table 3. Causes of 183 child deaths under 2 years of age reported to the Hong Kong coroner during 1 February 1999 to 31 January 2003 classified according to (1) primary diagnosis from the coroner’s death report, autopsy report or other information and (2) after detailed review of these reports by one of us (T Nelson)

<table>
<thead>
<tr>
<th>Category</th>
<th>Coroner diagnosis</th>
<th>Review diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital heart disease</td>
<td>48</td>
<td>49</td>
</tr>
<tr>
<td>Other congenital abnormality</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Inborn error of metabolism</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Injury (cause unspecified)</td>
<td>11*</td>
<td>-</td>
</tr>
<tr>
<td>Homicide</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Infanticide/unattended delivery</td>
<td>-</td>
<td>10</td>
</tr>
<tr>
<td>Accidental injury</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Possible non-accidental injury</td>
<td>2</td>
<td>7**</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>24</td>
<td>12</td>
</tr>
<tr>
<td>Gastroenteritis/bowel problem</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>CNS infection</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Related to neonatal period</td>
<td>18</td>
<td>14</td>
</tr>
<tr>
<td>Other defined illness</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>SIDS</td>
<td>18</td>
<td>33</td>
</tr>
<tr>
<td>Unknown or unascertained</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Diagnosis pending</td>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

*4/11 multiple injury; **6/7 unexplained cerebral or subdural haematomas.

Discussion

The incidence of SIDS in Hong Kong has fallen even further from the low value of 0.3/1000 live births (95% CI: 0.18–0.46/1000) in 1987, to 0.16/1000 (95% CI: 0.11–0.22/1000) in 1999–2002. Review of the coroner records showed that (when the study pathologist did not undertake the postmortem examination) forensic or hospital pathologists were much more likely to use the terms ‘unknown’ and ‘unascertained’. This reflects an inherent problem of SIDS studies, when both under-diagnosis and over-diagnosis can be potential problems. We erred on the side of over-diagnosis when estimating SIDS incidence and if stricter definitions had been used, the incidence would have been even lower.

The case-control comparison shows that, as elsewhere, prone sleeping and parental smoking are key risk factors, with high odds ratios. However, 12 of 16 babies who died had been placed on their back, emphasising that other factors must also be important. Bedsharing is a common behaviour in Hong Kong: 24% of controls did so. Bedsharing with someone other than parent had a sex and socioeconomic status-adjusted odds ratio of 4.9 (1.2–20.2), hence suggesting that this practice appeared to be hazardous.

In Hong Kong, where SIDS incidence is low but bedsharing is common, it can be assumed that methods of bedsharing are generally “safe”. However our data suggests that even within this culture, bedsharing is not always a safe practice and that parents and other caregivers need to be aware of potential risks. Finding babies with their heads covered was also common, as noted in other studies. The issue of diagnosis of
SIDS was addressed by repeating the analysis on a subgroup of “typical SIDS” and controls less than 1 year old. This showed generally similar results.

Because of the lower rates than anticipated, our study had to be extended from the originally planned 3 years to 4 years. We still obtained small numbers of cases, which reduces the power to demonstrate differences, and to conduct more extensive studies of the relationship of SIDS to risk factors.

We could only do follow-up interviews for those deaths that were initially suspected to be SIDS and reported to us by the mortuary staff. The response rate for controls was about 55% for the first control, but 90% overall. Loss of controls occurred mainly because of moving to unknown addresses or subsequent reluctance to be interviewed. It is not known to what extent failure to interview the first selected control might have influenced the results.

The time lag between the deaths and our interview was often long, much longer than for the controls. This lag usually occurred because of delays in arranging a time for interview that was acceptable to the families. However, while the “reference sleep” would be only one among many for controls, and therefore easy to forget, it was much more salient for the family of the dead infants, so details would be anticipated to more readily recalled. This is anticipated to reduce the potential for recall bias, although certain variables, such as amount of last feed and illness of the baby during the last week, could be more prone to such bias.

The rates of SIDS in Chinese populations are lower than in Western countries, but this is one of the lowest reported. Of interest was the greater proportion of non-Chinese in our cases (21%) relative to the Census data, which showed that 5.1% of the population are non-Chinese. Comparison of our attribution of cause with the official data shows that without special care it is easy to miss potential cases, and the difference may be about 50%. Especially in populations with low rates of SIDS such as Hong Kong Chinese, it is important to check for metabolic diseases (such as the ornithine carboxylase deficiency we found), since even a few such cases could distort the apparent rates.

Special attention must also be paid to those deaths diagnosed as respiratory illness, unascertained or unknown. While the latter two labels may reassure the pathologist that an unnatural cause remains an option, these terms may carry negative connotations for bereaved parents, and leave a lack of closure. By contrast, though the cause of SIDS is unknown, it leaves the understanding that the cause is natural, and not a reason for guilt.

The Hong Kong coronial system does not routinely have access to medical professionals, yet coroners decide whether a death report and investigation is required. We found several discrepancies in cause of death, and these included some with important legal and medical implications, such as suspected “shaken baby” syndrome. Therefore, a more formal process is possibly needed to review all child deaths, whether reported to the coroner or not. Such systems have been established elsewhere, such as the Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) established in 1992 in the UK, or the approach of the Victorian Institute of Preventive Medicine (VIFM) in Melbourne, Australia where a Scientific Officer investigates deaths suspected to be SIDS, and the death scene information assists the pathologists in determining the cause of death.
A recent report of the Royal College of Pathologists and Royal College of Paediatrics and Child Health on Sudden Unexpected Death in Infancy (SUDI) highlights that parents who have suffered this terrible tragedy have a right to a diagnosis, and that all SUDI postmortems should follow recommended protocols to optimise the chances that the cause of death can be identified.\textsuperscript{15}

Parents also need sensitive support and follow-up. The present study has shown that many of the flaws in the investigation and management of SUDI, identified by the United Kingdom report, are also occurring in Hong Kong. In the light of the findings of our study and publication of the working group recommendations, it will be important for Hong Kong to take steps to standardise the investigation and management of these deaths. Indeed, there is clearly a need to establish a child mortality review system in Hong Kong to systematically monitor all child deaths and to provide regular feedback to the public, health authorities, and professionals.

In the absence of good understanding of the pathology of SIDS, studies such as ours provide evidence for recommending changes in child-rearing practices. The consistent relationship between parental smoking and SIDS adds another reason to quit smoking. Pregnancy and the birth of a child may be important events that could encourage parents to stop or reduce smoking.\textsuperscript{16} While bedsharing is relatively common practice in Hong Kong, we must inform parents that this is potentially unsafe under certain circumstances. In addition, parents should be aware of the risks of bedclothing covering the baby’s head.

This estimate of the even lower rate of SIDS in Hong Kong after 10 years shows a need to monitor such rates regularly, with the hope of better understanding this disease. We cannot at this point be sure whether there has been substantial change in Hong Kong child-rearing practices, or whether this may simply be the result of improved living conditions in the interim. Our data has shown that when SIDS rates are very low, these rates may be distorted with the use of labels such as unknown or unascertained.\textsuperscript{12}

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References:


Perceptions of New Zealand adults about complementary and alternative therapies for cancer treatment

Judy Trevena, Anthony Reeder

Abstract

**Aim** To study perceptions regarding complementary and alternative medicine (CAM) treatments for cancer among adult New Zealanders.

**Methods** An anonymous telephone questionnaire that included questions to explore perceptions about CAM and cancer treatment was administered to a randomly selected sample of New Zealanders, 20 years and older.

**Results** A total of 438 New Zealand adults participated in the survey, out of 689 eligible contacts (68% participation). Less than one-third (28%) agreed with the statement *alternative therapy for cancer has an equal or better chance of curing cancer as medical treatment*, 34% disagreed, and 38% said they did not know. Most (63%) felt that complementary therapies could be beneficial to people who were also receiving conventional cancer treatment, although only 36% could name one or more such therapies. One-third (32%) said that alternative therapies could be used instead of conventional cancer treatments, but only 16% of the sample could name any alternative therapies. The CAM therapies named most often were nutrition (vitamin and mineral supplements, herbs, and diets) and psychosocial therapies (including positive thinking, spiritual therapies, and relaxation).

**Conclusions** There seems to be little consensus about the efficacy of CAM therapies for cancer. New Zealanders may lack information about CAM, or may be withholding judgment because of contradictory messages.

Many different terms are used to describe the area of healthcare that is largely external to conventional biomedicine, but complementary and alternative medicine (CAM) is the collective term recommended for New Zealand use.¹ Therapies are sometimes specifically described as *complementary* if they are used to supplement conventional biomedical treatments for cancer, *to control symptoms and improve wellbeing* (p20),² or as *alternative*, if used instead of conventional treatment. This use of the term *alternative* is often viewed as undesirable, since it defines such therapies in terms of what they are not, rather than what they are, and also defines them in contrast to an *orthodox* system.¹ Nevertheless, the term *alternative* remains in some popular usage and may convey the more radical perception that there is an effective choice other than conventional biomedical treatment.

Methods of categorising CAM therapies vary considerably. For example, some studies include spiritual practices in their definitions of CAM ³–⁶ whereas others consider spiritual practices to be a part of an outlook on life rather than a therapy.⁷ In New Zealand, an adaptation of the five group categorisation used by the US National Center for Complementary and Alternative Medicine (NCCAM) has been recommended to represent the full spectrum of modalities that exist here.¹
Although some complementary therapies have demonstrated effectiveness in improving quality of life, there is no convincing evidence that any alternative therapies can cure cancer.\(^2\) Although the effectiveness of CAM therapies is still in question, a survey of Australian oncologists found that meditation / relaxation / visualisation techniques were the CAM therapies considered most helpful, followed by hypnotherapy and acupuncture.\(^6\) CAM therapies are widely used by cancer patients, with 49% of a New Zealand sample\(^8\) and 66.7% of a Canadian sample\(^5\) reporting use of one or more CAM therapies.

Recent studies in use among the general population have reported that 23.4% of New Zealanders\(^9\) and 23.3% of Australians\(^10\) visited a CAM practitioner in the past year, while the percentages of people who used at least one type of CAM therapy in the previous year were 52.1% in Australia\(^10\) and 35.1% in America.\(^11\) Although the public profile of CAM therapies has increased dramatically over recent decades,\(^12\) this increase may have levelled off: an American study\(^11\) has indicated that the prevalence of CAM use did not increase markedly between 1997 to 2002.

CAM therapies are also widely regarded as being safe,\(^8\) despite the potential for dangerous effects, including negative interactions between CAM therapies and conventional medications. One New Zealand study found that 89% of cancer patients considered CAM to be safe, but that fewer than half of these patients discussed their usage of CAM with their doctors.\(^8\) Because of increasing public awareness, reported high use of CAM therapies, and the lack of objective information, it has been argued that there is an urgent need to make available clear and accurate information about CAM therapies, so that people can make informed decisions regarding their use.\(^1,13\)

The New Zealand Cancer Control Strategy identified the need (Goal 4, Objective 4) to ensure that those with cancer and their family and whānau have access to high-quality information on treatment and care, including complementary and alternative medicine.\(^14\) One of the proposed “broad areas of action” is to make sure that comprehensive, reliable and objective information, including that from the Ministry of Health database on CAM research, is easily accessible and understandable to patients, their families and whānau. The subsequent Action Plan, 2005-2010, identifies one outcome as ensuring that information for consumers will be comprehensive, evidence-based and reflect an integrated approach, combining self-help, CAM, and biomedical information.\(^15\)

Currently, cancer patients tend to learn about CAM therapies from friends (41%) and family (39%), rather than the Internet (3%).\(^16\) Nevertheless, the provision of trustworthy information on the Internet would be of benefit as the use of that source seems likely to increase. The exploration of current public awareness about CAM would assist in the targeting of such a service. Despite its potential importance for informing and helping in the evaluation of health promotion and cancer control programmes, the assessment of public perceptions about cancer has received little attention in New Zealand.\(^17\) The present study was designed to meet this need and contribute to current discussion about CAM in New Zealand\(^18,19\) by reporting the perceptions of a random sample of adult New Zealanders regarding CAM therapies for cancer treatment.
Methods

Sample selection and research procedures were fully described in an earlier paper. In summary, a national telephone survey was conducted in August and September 2001 among a random sample, 20 years and older, identified from telephone directory listings, supplemented with self-identified Maori from electoral rolls.

The questionnaire was designed to explore perceptions of the causes, prevention and treatment of cancer as well as provide demographic information. For questions with fixed responses (such as agree/disagree/don’t know), the interviewer read out all allowable answers and electronically recorded responses as a numerical code.

For open-ended questions, interviewers used numerical codes for the most commonly anticipated answers. All other answers were recorded verbatim and subsequently coded by one researcher, with the coding later checked by another member of the research team. After each response, participants were asked “Anything else?” until they could provide no further answers.

The CAM section of the questionnaire was introduced with the statement:

“Now I am going to ask you a few questions about other therapies which people sometimes use when they have cancer. These are therapies that are not part of the usual medical treatments of radiology, chemotherapy and surgery.”

Then two questions were asked which were worded to indicate a distinction between complementary and alternative therapies, as follows:

“Do you believe that there are any therapies that can be beneficial to people who are also receiving conventional medical treatment for their cancer? Sometimes these are called complementary therapies.” (If the respondent answered in the affirmative, they were then asked “Could you name any such therapies?”)

“Do you believe that there are any therapies that can be used instead of mainstream medical treatment to cure cancer? Sometimes these are called alternative therapies.” “Could you name any such therapies?”

Results

A total of 1565 attempts were made to perform interviews, resulting in 1130 contacts, of which 689 were deemed eligible, according to population quotas. Of these, 251 refused to participate, producing 438 completed interviews (231 females and 207 males) and 64% participation. The age, sex, and ethnicity distributions of the respondents were closely similar to those for the New Zealand population in the 1996 Census, but respondents were better educated, contained a larger proportion in full-time employment, but a smaller proportion of those permanently unable to work.

There was almost universal agreement (96%) about the benefit of early detection of cancer (S1), and most people (79%) were optimistic regarding the possibility of curing cancer (S2), although the level of fear regarding cancer treatment seemed quite high (S3)—see Table 1.

There was also considerable uncertainty regarding the effectiveness of alternative therapies when compared to conventional medical treatment (S4). Although many people (63%) felt that complementary therapies could be beneficial (Q5), only about half as many (32%) considered that alternative therapies could cure cancer (Q6).
Table 1. Perceptions about cancer and cancer treatment; percentages reporting agreement or disagreement with statements (n=438)*

<table>
<thead>
<tr>
<th>Statements</th>
<th>Agree</th>
<th>Not sure</th>
<th>Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1: Overall, survival time is much better when cancer is identified and treated early, than when it is not identified and treated until later</td>
<td>96</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>S2: Even with early detection, there is not much chance of curing cancer</td>
<td>12</td>
<td>9</td>
<td>79</td>
</tr>
<tr>
<td>S3: Most cancer treatment is so terrible, it is worse than death</td>
<td>22</td>
<td>22</td>
<td>57</td>
</tr>
<tr>
<td>S4: Alternative therapy for cancer has an equal or better chance of curing cancer as medical treatment</td>
<td>28</td>
<td>38</td>
<td>34</td>
</tr>
</tbody>
</table>

Questions

<table>
<thead>
<tr>
<th>Questions</th>
<th>Yes</th>
<th>Don’t know</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q5: Do you believe that there are any therapies that can be beneficial to people who are also receiving conventional medical treatment for their cancer? Sometimes these are called complementary therapies</td>
<td>63</td>
<td>24</td>
<td>14</td>
</tr>
<tr>
<td>Q6: Do you believe that there are any therapies that can be used instead of mainstream medical treatment to cure cancer? Sometimes these are called alternative therapies.</td>
<td>32</td>
<td>20</td>
<td>48</td>
</tr>
</tbody>
</table>

* Percentages may not add to 100, due to rounding
When asked if they could name any complementary therapies, 64% could not name any, and 21% could name only one therapy. Others mentioned two (9%), three (5%), four (1%), and even six (1%) different therapies. The therapies mentioned were coded into the five groups recommended by the Ministerial Advisory Committee on Complementary and Alternative Health (MACCAH).

As shown in Table 2, nearly 20% of the sample (more than half of those who named any complementary therapies) mentioned one or more therapy from Group 3 (Biological-based theories, including diet and dietary supplements, herbalism, and aromatherapy), as a complementary therapy. Around 15% of the sample gave one or more therapy from Group 2 (mind/body/spirit interventions, including meditation, positive thinking, relaxation and spiritual healing); nearly 10% of the sample cited one or more therapy from Group 1 (alternative medical systems, such as acupuncture, homeopathy, naturopathy, and yoga); and fewer than 5% listed any therapies from Group 5 (energy therapies: colour therapy, chi kung) or Group 4 (manipulative and body-based therapies: exercise and massage). Several of the 12 (3%) unclassifiable responses mentioned the use of clinics in America or Mexico, or specific cases reported in the media.

When asked if they could name any alternative therapies, 84% of respondents did not suggest any, and others mentioned one (11%), two (2%), or three (2%) therapies each. One person listed four therapies, and another six. As with the complementary therapies, the most commonly mentioned alternative therapies were from Group 3, followed by Group 2 and Group 1, with few people mentioning therapies from Group 4 or Group 5 (see Table 2).

Logistic regression was used to test whether attitudes and knowledge of CAM were related to the demographic variables of age (20–39, 40–59, 60+ years, with n = 183, 144 and 111 respectively), gender (207 male, 231 female), ethnicity (391 non-Maori, 47 self-identified Maori), formal educational qualifications (226 with no tertiary qualification, 200 with a tertiary qualification) and employment status (191 not employed full-time, 246 employed full-time).

The logistic regression compared people aged 20–39 and 40–59 with those aged 60 and over, as chi-squared comparisons indicated that (where differences due to age seemed to exist) the younger groups were more positive or knowledgeable about CAM than the oldest group. Odds ratios are presented in Table 3 for positive answers (yes / agree / named any therapies) rather than negative answers (no / disagree / did not name any therapies): answers of don’t know were excluded. Analysis for S1 is not reported, as few people disagreed with the statement.

Males, Maori, persons lacking a post-secondary qualification, and those not employed full-time were more pessimistic about the chance of curing cancer (S2). Being Maori was also associated with thinking cancer treatment was “worse than death” (S3) and agreeing that alternative therapy had an equal or better chance of curing cancer as medical treatment (S4). People who thought that complementary therapies could be beneficial in addition to conventional medical treatment (Q5) were more likely to be female, with a post-secondary qualification, and to be aged 40–59 rather than 60 or over.
Table 2. Numbers and percentages of participants naming complementary and alternative therapies

<table>
<thead>
<tr>
<th>MACCAH Group</th>
<th>Complementary</th>
<th>Alternative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>% (total)</td>
</tr>
<tr>
<td>Could not name any therapies</td>
<td>279</td>
<td>64</td>
</tr>
<tr>
<td>1: Alternative medical systems</td>
<td>41</td>
<td>9</td>
</tr>
<tr>
<td>2: Mind/body/spirit interventions</td>
<td>69</td>
<td>16</td>
</tr>
<tr>
<td>3: Biological-based therapies</td>
<td>86</td>
<td>20</td>
</tr>
<tr>
<td>4: Manipulative and body-based therapies</td>
<td>13</td>
<td>3</td>
</tr>
<tr>
<td>5: Energy therapies</td>
<td>15</td>
<td>3</td>
</tr>
<tr>
<td>Not classifiable</td>
<td>12</td>
<td>3</td>
</tr>
</tbody>
</table>
Table 3. Unadjusted odds ratios (confidence intervals) for all demographic predictors of CAM perceptions, and adjusted* odds ratios (confidence intervals) where these were significant

<table>
<thead>
<tr>
<th>Question</th>
<th>Younger†</th>
<th>Female</th>
<th>Maori</th>
<th>Qualified</th>
<th>Full-time</th>
</tr>
</thead>
<tbody>
<tr>
<td>S2</td>
<td>unadj</td>
<td>0.58 (0.28-1.20)</td>
<td>0.66 (0.37-1.19)</td>
<td>2.48 (1.16-5.27)</td>
<td>0.42 (0.22-0.80)</td>
</tr>
<tr>
<td>S2</td>
<td>adj</td>
<td>0.72 (0.35-1.50)</td>
<td>0.39 (0.19-0.77)</td>
<td>2.85 (1.25-6.50)</td>
<td>0.40 (0.20-0.78)</td>
</tr>
<tr>
<td>S3</td>
<td>unadj</td>
<td>0.70 (0.38-1.27)</td>
<td>0.84 (0.52-1.34)</td>
<td>2.71 (1.39-5.32)</td>
<td>0.74 (0.46-1.20)</td>
</tr>
<tr>
<td>S3</td>
<td>adj</td>
<td>0.71 (0.38-1.32)</td>
<td>2.83 (1.39-5.79)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S4</td>
<td>unadj</td>
<td>1.56 (0.86-2.83)</td>
<td>1.38 (0.85-2.22)</td>
<td>2.99 (1.31-6.84)</td>
<td>0.74 (0.46-1.20)</td>
</tr>
<tr>
<td>S4</td>
<td>adj</td>
<td>0.89 (0.46-1.70)</td>
<td>2.58 (1.11-6.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S5</td>
<td>unadj</td>
<td>2.39 (1.23-4.65)</td>
<td>1.62 (0.92-2.84)</td>
<td>1.65 (0.56-4.87)</td>
<td>2.39 (1.32-4.34)</td>
</tr>
<tr>
<td>S5</td>
<td>adj</td>
<td>3.29 (1.56-6.92)</td>
<td>2.01 (1.04-3.87)</td>
<td>2.50 (1.31-4.77)</td>
<td></td>
</tr>
<tr>
<td>NCom</td>
<td>unadj</td>
<td>3.49 (1.45-8.42)§</td>
<td>1.69 (1.14-2.52)</td>
<td>1.63 (0.89-3.00)</td>
<td>1.82 (1.23-2.72)</td>
</tr>
<tr>
<td>NCom</td>
<td>adj</td>
<td>1.53 (0.92-2.54)</td>
<td>2.09 (1.36-3.20)</td>
<td>2.09 (1.36-3.20)</td>
<td>0.55 (0.36-0.90)</td>
</tr>
<tr>
<td>S6</td>
<td>unadj</td>
<td>2.35 (1.32-4.20)</td>
<td>0.79 (0.52-1.22)</td>
<td>2.28 (1.15-4.52)</td>
<td>1.31 (0.85-2.02)</td>
</tr>
<tr>
<td>S6</td>
<td>adj</td>
<td>2.06 (1.11-3.82)</td>
<td>2.09 (1.36-3.20)</td>
<td>2.09 (1.36-3.20)</td>
<td>0.55 (0.36-0.90)</td>
</tr>
<tr>
<td>NAAlt</td>
<td>unadj</td>
<td>1.98 (1.01-3.88)‡</td>
<td>1.64 (1.05-2.54)</td>
<td>2.28 (1.15-4.52)</td>
<td>1.31 (0.85-2.02)</td>
</tr>
<tr>
<td>NAAlt</td>
<td>adj</td>
<td>1.78 (0.85-3.72)</td>
<td>2.19 (1.04-4.63)</td>
<td>2.44 (1.43-4.18)</td>
<td>1.09 (0.65-1.83)</td>
</tr>
</tbody>
</table>

* Adjusted for age, gender, ethnicity, qualification and full-time employment
† First comparison is Age 20-39 vs Age 60+: second comparison is Age 40-59 vs Age 60+
‡ Greater probability for Age 20-39 than Age 60+
§ Greater probability for Age 40-59 than Age 60+
The people who were more likely to name one or more complementary therapy rather than none were female, more qualified, not employed full-time, and aged 40–59 rather than 60 or over.

The belief that there were alternative therapies which could be used instead of mainstream medical treatment was more prevalent among people aged 20–39 than those aged 60 or over. People who could name one or more such alternative therapy were more likely to be Maori, more qualified, and aged 40–59 rather than 60 and over.

**Discussion**

This study seems to be the first to report on the awareness of (and attitudes towards) complementary and alternative therapies for cancer among a random sample of the adult New Zealand population. The response rate of 64% was comparable to rates of 57% and 76% obtained in previous postal surveys, and telephone surveys have also been shown to yield results similar to those of postal surveys.

Opinions were divided about whether or not alternative therapies for cancer were equally or more effective than conventional medical treatment, and many respondents said they did not know, indicating a need for reliable information to be made more available. New Zealanders in our study were more likely (38%) to be “not sure” (whether alternative therapies were as good at curing cancer as mainstream medical treatments) than people in an Australian sample, of whom only 5% were not sure (and a further 6% neither agreed nor disagreed). The quite large proportion of “not sure” responses in the current study may indicate a lack of knowledge about alternative methods of treatment, or there may be a reluctance to make a judgement either for or against alternative therapies, in the face of contradictory sources of evidence. In either case, this result reinforces previous urgent calls for reliable, objective information about CAM to be made more available. The Ministry of Health website (http://www.cam.org.nz) should, increasingly, help to meet this need.

It is of concern that so many people (63%) express a belief that complementary therapies can be beneficial when used alongside conventional medicine, especially given evidence that fewer than half of cancer patients who use CAM therapies discuss these therapies with their health professionals. The authors of that report discuss the potential risks involved, including adverse reactions to CAM therapies, and dangerous interactions between CAM and conventional treatments. The Cancer Society of New Zealand has also stressed the importance of cancer patients discussing CAM therapies with their physicians.

Another source of concern is the degree of pessimism regarding cancer reported by the Maori participants who were less likely than non-Maori to believe that cancer was curable, and more likely to believe that cancer treatment was worse than death. Although based on very small numbers, and therefore to be treated with caution, this greater pessimism may, in part, reflect the actual experience of cancer among the Maori population, given the increasing inequalities in outcomes now known to exist. In addition to making appropriate changes in health services, associated health promotion efforts may be needed to address perceptions.

Younger people were more likely than those over 60 years to believe that complementary and alternative therapies are efficacious. Women were more likely
than men to believe that complementary therapies could be beneficial, which is consistent with the findings of the 2002/3 New Zealand Health Survey, a recent survey which indicated that women were more likely than men to have visited a CAM practitioner in the previous year (28% of women, compared to 18% of men surveyed). 

In the NZ Health Survey, overall, 23% of the respondents had visited a CAM practitioner in the previous 12 months, and the three CAM practitioners visited were most often massage therapists, chiropractors, and osteopaths (visited in the previous year by 9%, 6%, and 5% of the sample, respectively). All three of these therapists fall into MACCAH Group 4 (Manipulative and body-based therapies), in contrast to the findings of the current study where, in the specific context of cancer treatment, Group 4 therapies were suggested least.

The plausible explanation for this may be that such therapies are, and are perceived as, less appropriate for cancer treatment. In addition, participants in the NZ Health Survey were shown a card with a number of different CAM practitioners on it (with massage therapists listed first), whereas the current study did not use any prompts, and participants may not have perceived massage as a complementary therapy.

Overall, as found in several studies from other countries, the most frequently reported CAM therapies for cancer seem to be psychosocial (including spiritual therapies, psychotherapy, relaxation, and visualisation) and nutrition (vitamin and mineral supplements, herbs, and diets).

In light of the high levels of uncertainty about the efficacy of CAM therapies found in the present study and the small proportions able to name any therapies, perhaps one of the most useful current initiatives in New Zealand is the attempt to help improve knowledge by making accessible to the public authoritative information on the Complementary and Alternative Medicine website (www.cam.org.nz) supported by the Ministry of Health.

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Attitudes of hospital medical practitioners to the mandatory reporting of professional misconduct

Sumit Raniga, Phil Hider, David Spriggs, Mike Ardagh

Abstract

Background New legislation now requires doctors to report unfit colleagues to the Medical Council of New Zealand. However, little research has examined the attitudes and willingness of doctors to report errant colleagues.

Aim To examine the attitudes of a range of hospital-based medical practitioners towards the mandatory reporting of colleagues.

Methods House staff, registrars, and consultants at two major tertiary teaching hospitals in New Zealand were surveyed using a written questionnaire. Doctors were asked to state their level of agreement with a series of statements and to make responses to three hypothetical scenarios: an alcohol impaired practitioner; a senior colleague with recent behavioural change; and a surgeon expressing racist views.

Results Responses were received from 52% of medical staff at the two hospitals. Respondents were consultants (52%), registrars (39%), and house staff (9%). Most (98%) respondents agreed that all doctors make clinical errors and there was a need for open discussion about error. The majority (80%) also accepted that doctors were responsible for the actions of colleagues and agreed that they would act if a colleague was failing to achieve professional standards. Only 45% agreed with the mandatory reporting of error. The responses to three scenarios illustrate some variability in how different groups of doctors would address the behaviour of an errant colleague.

Conclusions Although most hospital doctors accept they should act if a colleague is falling below professional standards, there is only limited support for mandatory reporting; instead, doctors may prefer to consult senior colleagues about an errant colleague or sometimes counsel the practitioner themselves.

In New Zealand and many other Western countries the medical profession exercises self-regulation under the auspices of defining legislation. Critical to the success of this approach is the willingness of doctors to report their peers. Recent events including the Cartwright Report and the Gisborne Inquiry have drawn attention to the need for the medical profession and society to consider how well the profession undertakes this key function.

Arguably a practitioner’s peers are in the best position to observe and recognise episodes of poor professional practice. Despite this, complaints by doctors about the professional competence of their colleagues are relatively uncommon. Indeed, only 45 (17%) of the 267 doctors whose competence was reviewed by the Medical Council of New Zealand between 1996-2002 were reported by their colleagues.

New legislation, the Health Practitioners Competence Assurance Act (HPCAA) 2003, now requires that all doctors must notify the Medical Council of New Zealand (MCNZ) when their own or a colleague’s mental or physical fitness is in doubt,
although it encourages rather than demands that doctors should report colleagues whose competence is in question. In addition, there also exists an ethical duty on practitioners to report colleagues who are practising below an acceptable standard.\textsuperscript{5}

The Good Medical Practice Guide of the MCNZ advises doctors that ‘you must protect patients when you believe a doctor’s or other health colleague’s health, conduct or performance is a threat to them’.\textsuperscript{6} Similarly the ethical code of the New Zealand Medical Association requires that doctors must ‘take appropriate steps to ensure unsafe or unethical practices on the part of colleagues are curtailed and/or reported to relevant authorities without delay.’\textsuperscript{7}

The Code of Health and Disability Services Consumers’ Rights requires, in Right 4(2), that providers comply with ‘ethical and other relevant standards’. Indeed, the Health and Disability Commissioner has stated that health professionals have an ethical duty, enforceable via the Code, to report concerns about a poorly performing colleague to management or a senior colleague (Tauranga Hospital Inquiry Report www.hdc.org.nz/opinions 18 Feb 2005)

The term ‘whistle-blowing’ has been used to describe any health professional who raises concerns about the performance of a colleague.\textsuperscript{3} Although it could be argued that taking concerns through the proper channels is not technically whistle-blowing it will be used here to describe any reporting of concerns by a health professional irrespective of the route taken.\textsuperscript{8}

While it can be appreciated, on one hand, that it is clearly the right thing that doctors should protect the public interest when confronted by a potentially unsafe colleague, in reality this choice is often more complicated. Whistle-blowers frequently report wrestling with an agonising ethical dilemma between personal loyalty and public safety before taking action.\textsuperscript{9}

The feeling that whistle-blowers are betraying their colleagues illustrates a societal norm that is especially strong among professionals whose collegial loyalty underpins their sense of professional practice.\textsuperscript{10} Another issue for practitioners is their difficulty in confidently recognising cases of substandard care when the incompetence is not gross or extreme. Similarly, the subjective nature of practitioners’ perceptions of professional incompetence have raised concern that some allegations could even be made by professionals who are motivated by greed, envy, or dislike.\textsuperscript{11,12}

Increasing the reservations of a potential complainant, examples exist of difficult circumstances befalling the personal and professional lives of whistle-blowers.\textsuperscript{9,13,14} In the face of these difficulties, few jurisdictions around the World have adopted mandatory reporting for medical errors or issues related to the competence of colleagues.\textsuperscript{15} Instead, many have chosen to strengthen their legislative support for whistle-blowing professionals\textsuperscript{16} whilst at the same time allowing regulating bodies to be more proactive about maintaining standards and enhancing reaccreditation requirements for medical professionals.\textsuperscript{17}

Despite the important issues and the major changes that surround whistle-blowing, relatively few surveys have documented the attitudes of doctors to this activity and their willingness to report errant colleagues.\textsuperscript{18}

The aim of the current study was to examine the attitudes of a range of hospital-based medical practitioners towards mandatory reporting of colleagues who fail to achieve
the required professional standards either due to their health, conduct, or incompetence.

**Methods**

**Questionnaire design**—The attitudes of medical practitioners towards mandatory reporting of deficient practice were assessed by means of a written questionnaire. The questionnaire was designed with the aim of providing accurate, relevant information. To improve compliance the questionnaire was made relatively short and was based on three short fictitious scenarios. The scenarios were intended to be realistic and thought-provoking. Questions examining the attitudes of doctors towards mandatory reporting were appended after each scenario.

An initial draft of the questionnaire was peer-reviewed by a panel of four senior, experienced clinicians.

The questionnaire was specifically designed to address the following issues related to deficient practice:

- **Professional competence**: including deficiencies in knowledge, skills, or attitudes.
- **Conditions affecting fitness to practice**: physical or mental conditions affecting fitness to practice including alcohol or drug dependence, other psychiatric disorders, a temporary stress reaction, an infection with a transmissible disease, declining competence due to age-related loss of motor skills or to the early stages of dementia, and certain illnesses and injuries.
- **Professional conduct**: including frequently repeated rude or disruptive behaviours, sexual harassment, and demands for “special treatment” as well as any other behaviours that may disrupt the medical environment and give rise to impaired patient safety.

In order to improve compliance and aid objective analysis, graded series of responses were provided to most questions and respondents were required to circle the most appropriate response. Doctors were asked to indicate their level of agreement with various statements related to each of the scenarios. In order to foster honest reporting, no information about the identity or clinical speciality of respondents was requested in the survey. However, all participants where given the opportunity to make any comments using free text on any aspect of the study.

**Sampling and analysis**—Questionnaires were distributed via the internal mail system to medical practitioners employed to work in two tertiary New Zealand teaching hospitals. The questionnaire was sent to all medical officers (including house staff, registrars, and consultants) in each organisation using the addresses that were currently available on the records at the payroll office at each location. A letter requesting participation and providing background information, as well as a self-addressed envelope was included in the questionnaire pack. No identifying information was recorded on the questionnaire, and personal follow-up requests to complete the survey could not be made.

The survey was conducted between December 2003 and March 2004.

**Results**

A total of 650 questionnaires where distributed, and a response was received from 339 (52%) of the doctors. Of respondents, 177 (52%) were consultants, 131 (39%) were registrars, and 31 (9%) were house officers.

Overall, most (332, 98%) respondents agreed that with the statement that *all doctors make (and will continue to make) clinical errors, thus it is important that there be an attitude in the profession that promotes open discussion of mistakes and the lessons that can be learnt*. Notably, a higher proportion of consultants (74%, 131)—compared with registrars (63%, 83) or house staff (55%, 17)—strongly agreed with the above statement.

Views on mandatory reporting were less uniform. Only 153 (45%) doctors agreed with the statement that *mandatory reporting represents an important element in the process of oversight, put in place to promote high standards of medical practice; 112 (33%) of practitioners were not sure, and 74 (22%) disagreed with the statement.*
Responses were broadly similar between the three groups. Most participants (272, 80%) consistently across the three groups of medical staff supported the view that doctors are professionally responsible for the actions of colleagues and they should be prepared to act if a colleague is failing to achieve the required professional standards. The majority of doctors (251, 74%) considered that they were unsure whether the MCNZ competence assessment process is fair and effective. A higher number and proportion of consultants (23 and 13%) relative to their colleagues (6 and 5% of registrars and 0 house staff) concluded that the process was not fair or effective.

Scenario A involved the case of an alcohol impaired and inappropriate practitioner and asked respondents to indicate their willingness to report his behaviour. Most respondents (260, 77%) indicated that they would report his activities to a senior colleague, although some (45, 13%) would try and counsel the doctor themselves. Most doctors would still report their colleague even when it was suggested that the practitioner’s behaviour was transient (262, 77%).

However, respondents were less certain about their course of action when it was suggested that their colleague had made sexually inappropriate remarks to a nurse. Only 191 (56%) indicated their intention to report their colleague, and 97 (29%) and 51 (15%) were either unsure or would not make a complaint. Most doctors (269, 79%) were aware of the process they should follow to report a colleague, but junior doctors were relatively less familiar with the steps (15 or 48% had no idea what to do compared with 39 [12%] registrars and 16 [9%] consultants).

Scenario B concerned a senior practitioner with recent behavioural change and increasing signs of confusion. Even in the absence of any patient complaints, 197 (58%) doctors indicated that they would still report their colleague to a senior team member—although a significant number (92, 27%), especially consultants (69, 39%), would themselves attempt to counsel the doctor.

Most respondents (233, 69%) disagreed with the statement that this situation lay beyond their responsibility, and the majority (246, 73%) were not reluctant to raise the issue even if it may adversely affect their relationship with senior staff. More junior staff, however, signalled their greater difficulty with this situation—as relatively more house officers (9, 29%) and registrars (39, 30%) compared with consultants (18, 10%) indicated that the issue was beyond their responsibility, while 14 (45%) house officers and 25 (19%) registrars recognised that they were unlikely to raise the matter as it would adversely impinge on their relationships with senior staff.

Finally, most (289, 85%) doctors agreed that they would seek a second opinion after an informal appeal to the doctor was met with denials about any problem.
Table 1. Scenario A with responses

You have known Dr A since your days at medical school. He joined your team around 6 months ago. In this period there have been a few complaints from some of the staff with regard to his punctuality. Staff nurses have complained that he tends to make inappropriate comments on occasions, and that they feel uncomfortable working with him. You have been informed that on one occasion, Dr A was asked to take 'sick leave' as he arrived to his 8.00 am shift with the smell of alcohol on his breath. You have spoken to Dr A and tried to address some of these issues as a friend. Two weeks later, you are close to finishing your shift, and are waiting to handover to Dr A, he arrives late and you can smell alcohol on his breath. You ask Dr A to go home.

Based on the information provided, you would:

<table>
<thead>
<tr>
<th>Position</th>
<th>Take no action</th>
<th>Counsel Dr A personally</th>
<th>Report to more senior members of the team</th>
<th>Report to hospital management</th>
<th>Report to MCNZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>3% (1)</td>
<td>29% (9)</td>
<td>65% (20)</td>
<td>–</td>
<td>3% (1)</td>
</tr>
<tr>
<td>Registrars</td>
<td>–</td>
<td>7% (9)</td>
<td>86% (112)</td>
<td>5% (7)</td>
<td>2% (3)</td>
</tr>
<tr>
<td>Consultants</td>
<td>–</td>
<td>15% (27)</td>
<td>72% (128)</td>
<td>8% (14)</td>
<td>5% (8)</td>
</tr>
<tr>
<td>Total</td>
<td>0.3% (1)</td>
<td>13% (45)</td>
<td>77% (260)</td>
<td>6% (21)</td>
<td>4% (12)</td>
</tr>
</tbody>
</table>

You have known Dr A for a long time, his behaviour is uncharacteristic and you believe it is temporary. There is no need to report Dr A, as his behaviour will change:

<table>
<thead>
<tr>
<th>Position</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Not Sure</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>3% (1)</td>
<td>10% (3)</td>
<td>26% (8)</td>
<td>58% (18)</td>
<td>3% (1)</td>
</tr>
<tr>
<td>Registrars</td>
<td>–</td>
<td>7% (9)</td>
<td>15% (19)</td>
<td>66% (86)</td>
<td>13% (17)</td>
</tr>
<tr>
<td>Consultants</td>
<td>–</td>
<td>4% (7)</td>
<td>17% (30)</td>
<td>64% (113)</td>
<td>15% (27)</td>
</tr>
<tr>
<td>Total</td>
<td>0.3% (1)</td>
<td>6% (19)</td>
<td>17% (57)</td>
<td>64% (217)</td>
<td>13% (45)</td>
</tr>
</tbody>
</table>

You would report Dr A, as a staff nurse informs you that Dr A’s inappropriate comments were of a sexual nature:

<table>
<thead>
<tr>
<th>Position</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Not Sure</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>6% (20)</td>
<td></td>
<td>17% (57)</td>
<td>64% (217)</td>
<td>13% (45)</td>
</tr>
</tbody>
</table>

Total Agreed: 6% (20)  Total Not Sure: 17% (57)  Total Disagreed: 77% (262)
<table>
<thead>
<tr>
<th></th>
<th>House Officers</th>
<th>Registrars</th>
<th>Consultants</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7% (2)</td>
<td>32% (10)</td>
<td>45% (14)</td>
<td>13% (4)</td>
</tr>
<tr>
<td></td>
<td>3% (4)</td>
<td>52% (68)</td>
<td>29% (38)</td>
<td>15% (19)</td>
</tr>
<tr>
<td></td>
<td>11% (19)</td>
<td>50% (88)</td>
<td>25% (45)</td>
<td>12% (21)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>8% (25)</strong></td>
<td><strong>49% (166)</strong></td>
<td><strong>29% (97)</strong></td>
<td><strong>13% (44)</strong></td>
</tr>
<tr>
<td><strong>Total Agreed</strong></td>
<td><strong>56% (191)</strong></td>
<td><strong>Total Not Sure: 29% (97)</strong></td>
<td><strong>Total Disagreed: 15% (51)</strong></td>
<td></td>
</tr>
</tbody>
</table>
Table 1. Continued

If you did decide to report Dr A, you are aware of the process and would know what steps to take:

<table>
<thead>
<tr>
<th>Position</th>
<th>I have no idea and wouldn’t know what to do</th>
<th>I have some idea and would know where to start</th>
<th>I am well aware and would know exactly what to do</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>48% (15)</td>
<td>52% (16)</td>
<td>–</td>
</tr>
<tr>
<td>Registrars</td>
<td>12% (39)</td>
<td>65% (85)</td>
<td>5% (7)</td>
</tr>
<tr>
<td>Consultants</td>
<td>9% (16)</td>
<td>77% (137)</td>
<td>14% (24)</td>
</tr>
<tr>
<td>Total</td>
<td>21% (70)</td>
<td>70% (238)</td>
<td>9% (31)</td>
</tr>
</tbody>
</table>
Table 2. Scenario B with responses

Dr B is a respected senior medical practitioner with a good reputation. In the last year or so, you and other members of your team have noticed some changes in Dr B's behaviour. She tends to repeat herself a lot, and on occasions has trouble remembering the names of common drugs. There are times when Dr B has trouble 'making sense'. In the last few months you have witnessed uncharacteristic outbursts from Dr B when she is stressed.

Based on the information provided and the fact that there have been no formal patient complaints or any evidence of substandard practice, you would:

<table>
<thead>
<tr>
<th>Position</th>
<th>Take no action</th>
<th>Counsel Dr B personally</th>
<th>Report to more senior members of the team</th>
<th>Report to hospital management</th>
<th>Report to MCNZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>29% (9)</td>
<td>7% (2)</td>
<td>61% (19)</td>
<td>3% (1)</td>
<td>–</td>
</tr>
<tr>
<td>Registrars</td>
<td>12% (16)</td>
<td>16% (21)</td>
<td>69% (90)</td>
<td>2% (3)</td>
<td>1% (1)</td>
</tr>
<tr>
<td>Consultants</td>
<td>3% (6)</td>
<td>39% (69)</td>
<td>50% (88)</td>
<td>5% (9)</td>
<td>3% (5)</td>
</tr>
<tr>
<td>Total</td>
<td>9% (31)</td>
<td>27% (92)</td>
<td>58% (197)</td>
<td>4% (13)</td>
<td>2% (6)</td>
</tr>
</tbody>
</table>

You would not take any action as this situation lies beyond your responsibility, and someone holding a more senior position in the medical team should address it:

<table>
<thead>
<tr>
<th>Position</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Not Sure</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>10% (3)</td>
<td>19% (6)</td>
<td>26% (8)</td>
<td>42% (13)</td>
<td>3% (1)</td>
</tr>
<tr>
<td>Registrars</td>
<td>3% (4)</td>
<td>27% (35)</td>
<td>15% (19)</td>
<td>53% (69)</td>
<td>3% (4)</td>
</tr>
<tr>
<td>Consultants</td>
<td>1% (1)</td>
<td>10% (17)</td>
<td>7% (13)</td>
<td>68% (121)</td>
<td>14% (25)</td>
</tr>
<tr>
<td>Total</td>
<td>2% (8)</td>
<td>17% (58)</td>
<td>12% (40)</td>
<td>60% (203)</td>
<td>9% (30)</td>
</tr>
</tbody>
</table>

Total Agreed: 19% (66)  Total Not Sure: 2% (40)  Total Disagreed: 69% (233)
You would be reluctant to raise this issue as it may adversely affect your relationships with other colleagues, especially the senior staff members who know and respect Dr B:

<table>
<thead>
<tr>
<th>Position</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Not Sure</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>10% (3)</td>
<td>36% (11)</td>
<td>16% (5)</td>
<td>39% (12)</td>
<td>-</td>
</tr>
<tr>
<td>Registrars</td>
<td>2% (2)</td>
<td>18% (23)</td>
<td>20% (26)</td>
<td>56% (73)</td>
<td>5% (7)</td>
</tr>
<tr>
<td>Consultants</td>
<td>1% (1)</td>
<td>7% (12)</td>
<td>6% (10)</td>
<td>76% (134)</td>
<td>11% (20)</td>
</tr>
<tr>
<td>Total</td>
<td>2% (6)</td>
<td>14% (46)</td>
<td>12% (41)</td>
<td>65% (219)</td>
<td>8% (27)</td>
</tr>
<tr>
<td></td>
<td>Total Agreed: 15% (52)</td>
<td>Total Not Sure: 12% (41)</td>
<td>Total Disagreed: 73% (246)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Continued

You take the time to speak informally to Dr B about some of these concerns. She strongly denies any difficulties, and is especially irritated at the suggestion that she should consider seeking help:

<table>
<thead>
<tr>
<th>Position</th>
<th>I would not readdress the matter any more</th>
<th>I would seek a second opinion on the matter</th>
<th>I would take steps to report Dr B</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>16% (5)</td>
<td>81% (25)</td>
<td>3% (1)</td>
</tr>
<tr>
<td>Registrars</td>
<td>12% (15)</td>
<td>82% (108)</td>
<td>6% (8)</td>
</tr>
<tr>
<td>Consultants</td>
<td>1% (2)</td>
<td>88% (156)</td>
<td>11% (19)</td>
</tr>
<tr>
<td>Total</td>
<td>7% (22)</td>
<td>85% (289)</td>
<td>8% (28)</td>
</tr>
</tbody>
</table>
Table 3. Scenario C with responses

Dr C is a renowned surgeon. You have known him for a number of years now and have a good professional relationship with him. But on several occasions, both professionally and informally, Dr C has expressed certain views about minority racial groups that have startled you. You have always assumed that these are his personal views and would not influence how he practices medicine. Last week, while speaking to a patient, a teacher by profession belonging to a minority ethnic group, you learn that she has not been fully informed about the risks of an upcoming procedure. You discover that Dr C has already obtained “informed consent”. When suggesting to Dr C that the patient was not fully aware of the risks involved, Dr C replied, “these people wouldn’t understand even if you bothered telling them”.

Dr C’s behaviour is unacceptable:

<table>
<thead>
<tr>
<th>Position</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Not Sure</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>48% (15)</td>
<td>48% (15)</td>
<td>–</td>
<td>–</td>
<td>3% (1)</td>
</tr>
<tr>
<td>Registrars</td>
<td>44% (58)</td>
<td>51% (67)</td>
<td>2% (3)</td>
<td>2% (2)</td>
<td>1% (1)</td>
</tr>
<tr>
<td>Consultants</td>
<td>44% (77)</td>
<td>52% (92)</td>
<td>2% (4)</td>
<td>1% (2)</td>
<td>1% (2)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>44% (150)</strong></td>
<td><strong>51% (174)</strong></td>
<td><strong>2% (7)</strong></td>
<td><strong>1% (4)</strong></td>
<td><strong>1% (4)</strong></td>
</tr>
<tr>
<td><strong>Total Agreed:</strong></td>
<td><strong>95% (324)</strong></td>
<td><strong>Total Not Sure: 2% (7)</strong></td>
<td><strong>Total Disagreed: 2% (8)</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Based on the information provided, you would:

<table>
<thead>
<tr>
<th>Position</th>
<th>Take no action</th>
<th>Counsel Dr C personally</th>
<th>Report to more senior members of the team</th>
<th>Report to hospital management</th>
<th>Report to MCNZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>26% (8)</td>
<td>23% (7)</td>
<td>42% (13)</td>
<td>10% (3)</td>
<td>–</td>
</tr>
<tr>
<td>Registrars</td>
<td>12% (15)</td>
<td>38% (50)</td>
<td>44% (58)</td>
<td>5% (6)</td>
<td>2% (2)</td>
</tr>
<tr>
<td>Consultants</td>
<td>5% (9)</td>
<td>48% (84)</td>
<td>40% (70)</td>
<td>7% (12)</td>
<td>1% (2)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>9% (32)</strong></td>
<td><strong>41% (141)</strong></td>
<td><strong>41% (141)</strong></td>
<td><strong>6% (21)</strong></td>
<td><strong>1% (4)</strong></td>
</tr>
</tbody>
</table>

You raise your concerns with a fellow doctor who is aware of Dr C’s views, but he warns you not to go any further with this matter, as it wouldn’t make any difference:
Position | I would not readdress the matter any more | I would seek a second opinion on the matter | I would take steps to report Dr C
---|---|---|---
House Officers | 29% (9) | 61% (19) | 10% (3)
Registrars | 17% (22) | 76% (99) | 8% (10)
Consultants | 8% (14) | 84% (148) | 9% (15)
Total | 13% (45) | 79% (266) | 8% (28)

Table 3. Continued

You would not take action as it may potentially jeopardise your position and/or future career:

<table>
<thead>
<tr>
<th>Position</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Not Sure</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>House Officers</td>
<td>7% (2)</td>
<td>19% (6)</td>
<td>42% (13)</td>
<td>29% (9)</td>
<td>3% (1)</td>
</tr>
<tr>
<td>Registrars</td>
<td>2% (2)</td>
<td>10% (13)</td>
<td>30% (39)</td>
<td>50% (66)</td>
<td>8% (11)</td>
</tr>
<tr>
<td>Consultants</td>
<td>–</td>
<td>4% (7)</td>
<td>14% (24)</td>
<td>64% (114)</td>
<td>18% (32)</td>
</tr>
<tr>
<td>Total</td>
<td>1% (4)</td>
<td>8% (26)</td>
<td>22% (76)</td>
<td>56% (189)</td>
<td>13% (44)</td>
</tr>
</tbody>
</table>

Total Agreed: 9% (30)  Total Not Sure: 22% (76)  Total % Disagreed: 69% (233)
Scenario C considered the case of a surgeon with racist views. Among the respondents and across the groups there was almost uniform agreement (324, 96%) that the behaviour of the surgeon by denying the patient proper informed consent on the basis of their race was unacceptable. Participants were equally divided though as to whether they would counsel the doctor themselves (141, 41%) or report him to a senior colleague (141, 41%).

Most (266, 79%) of the doctors who completed the questionnaire indicated that they would still seek a second opinion or take other steps to report the doctor (28, 8%) after a colleague warned that they should not proceed any further with the matter. Similarly most respondents (233, 69%) were not deterred by the suggestion that they should not take any action as it may jeopardise their future careers. A higher proportion of house officers (26%) compared with either registrars (11%) or consultants (4%) reported that they were less likely to pursue the matter in this context.

Discussion

Most (272, 80%) medical staff at two tertiary, teaching hospitals supported the view that doctors were professionally responsible for the actions of colleagues and agreed that they would act if a colleague was failing to achieve the required professional standards. This result mirrors the findings from a recent study of young doctors in the United Kingdom (UK) that examined the attitudes on a number of professional issues including whistle-blowing.

The UK study similarly concluded that most (90%) doctors believed they were corporately responsible for the actions of their colleagues, and agreed that they would act if a colleague was falling below acceptable professional standards. Furthermore, in the current study, the willingness to report colleagues whose competence was below an acceptable standard was generally expressed across all groups of doctors including consultants, registrars, and house staff. However, although the results were consistent they were not unanimous and some young doctors, in particular, expressed difficulty with reporting colleagues, particularly when the dysfunctional clinician was more senior.

This finding is consistent with the results from focus groups in the UK which have documented that young doctors were sometimes reluctant to report examples of unethical behaviour they witnessed among their peers or teachers. It is perhaps not surprising that some doctors have difficulty with reporting defective colleagues given that some cultural mores in the community still decry the actions of whistle-blowers. Furthermore, other surveys have reported that some established formal training programmes have failed to improve medical students ethical performance during their training.

Confronted by these findings, medical schools have recently reconsidered how they select their students and redefined the nature and the content of the ethical training they provide, especially in relation to whistle-blowing.

Another important finding is confirmation that most (332, 98%) doctors in the survey recognised that doctors make clinical errors and that there was therefore the need for an attitude in the medical profession that promotes ‘open discussion of mistakes’ and the lessons that can be learnt from them. The willingness of practitioners to openly
discuss medical error is a vital foundation in any efforts to identify adverse events and introduce processes to avoid them.\textsuperscript{23} This finding supports efforts by various professional, organisational, and government bodies to promote the open discussion of medical error among practitioners and to consider a system-oriented rather than an individual-blaming approach to quality improvement.\textsuperscript{24}

High rates of acceptance for the open discussion of error among consultants in this survey, and the power of medical role models to influence young doctor behaviour,\textsuperscript{25} suggests that an open approach to medical error could become the professional norm in this country.

While most (272, 80\%) respondents believed that they should be prepared to act if a colleague is failing to achieve the required professional standards, only 153 (45\%) agreed that mandatory reporting represents an important element in the process of oversight and 112 (33\%) were unsure. Several explanations exist for doctors’ reluctance to accept mandatory reporting.

Many are aware that whistle-blowing often does not leave the perpetrator unscathed and internal conflict exists between the duty to report and the fear of repercussions.\textsuperscript{14} It has also been argued that opposition to mandatory reporting represents rejection of the means of achieving an outcome rather than the outcome itself.\textsuperscript{5} The statutory duty to report to an external agency could be regarded by practitioners as creating a punitive atmosphere and a culture of fear.\textsuperscript{26} The absence of a statutory obligation may be more likely to foster the appropriate atmosphere to engender honest discussion within an organisation about error and encourage organisational interventions.\textsuperscript{5,26}

Finally, another important possible explanation is provided by the finding that 251 (74\%) doctors expressed uncertainty about whether the competence assessment process was fair. If practitioners do not believe the assessment process to be fair it is possible that they may not wish to participate. Overcoming this perception is a challenge for the MCNZ and other professional organisations. Assisting the change in this perception are recent amendments to Accident Compensation Corporation (ACC) legislation that remove the concepts of medical error and medical mishap and thereby no longer require blame to be attached to individuals for harms related to the provision of healthcare.\textsuperscript{27}

The results from scenario C exhibit the potential difficulty that some practitioners may have with knowing to whom they should report cases of unacceptable behaviour. The case highlights the particular difficulty that junior staff experience with pursuing a complaint even when it was initially discounted by another colleague. This difficulty likely relates to a feeling of powerlessness among junior staff, and is akin to the impotence sometimes expressed by other health professionals when confronted by incompetent practitioners in positions of authority.\textsuperscript{3}

The difficulties of other professionals, coupled by the inability or unwillingness of some affected doctors to seek care,\textsuperscript{28,29} increases the demands upon practitioners to take action when they are aware of an incompetent colleague.\textsuperscript{3}

This survey has several limitations. It describes the responses of practitioners to fictitious case vignettes. Although empirical evidence does support the validity and reliability of using vignettes as a reliable tool to describe the behaviour of doctors in real situations,\textsuperscript{30} the relationship between what practitioners say they would do in a
hypothetical situation and what they actually do in real-life is not clear-cut or certain.  

Secondly, there is potential for selection bias in relation to which practitioners choose to participate in the study. Although the anonymous nature of the study was designed to enhance the response rate, it also prevented personal follow-up of non-responders. Response rates varied across the three groups of doctors, and some response categories were associated with small numbers. The rate was lowest among house officers and highest among consultants. It is not possible to compare those who responded with those doctors who did not, and consequently the potential for selection bias cannot be determined. The relatively low participation rate among house staff suggests that there may be some difficulty with generalising the findings from this survey to house surgeons in general. In addition, the study did not collect personal data such as gender, age, and speciality; therefore we are unable to determine whether attitudes to mandatory reporting varied in relation to these demographic and professional characteristics.

Finally the study has not provided any details about why some practitioners may not wish to report dysfunctional doctors. Possible explanations include the belief that they would not be believed, that nothing would change, or that they would themselves be censured or harmed. To reliably address these issues, further research is needed using representative cohorts of practitioners and a wider array of questions.

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Aetiology and pathogenesis of chronic fatigue syndrome: a review

Robin Mihrshahi, Robyn Beirman

Abstract

Chronic fatigue syndrome (CFS) is a debilitating disease of uncertain aetiology that is characterised by unexplained, severe fatigue associated with a number of typical symptoms. This paper reviews the scientific literature related to current theories about the aetiology and pathogenesis of CFS by focussing on what appear to be the four most significant aspects in the development and perpetuation of this disease: the role of infectious agents as well as immunological, neuroendocrine, and psychiatric factors. A multifactorial model for the aetiology of CFS, which includes and draws together these four aspects, is proposed; and suggestions are offered regarding approaches to the diagnosis and treatment of this disease.

Chronic fatigue syndrome (CFS) is a debilitating disease of uncertain aetiology that is characterised by unexplained, persistent or relapsing, severe fatigue associated with muscle aches, weakness, pharyngitis, lymphadenopathy, headache, depression, sleep disturbance, memory difficulties, and confusion.¹,²

Over the years, many different hypotheses have been proposed with regards to the aetiology and pathogenesis of this condition. The criteria used for the diagnosis of CFS were uncertain and inconsistent for a long time, but were standardised by an international study group of the US Centers for Disease Control (CDC) in 1994. According to this CDC definition, which is now used everywhere in the world, CFS is a disease characterised by medically unexplained severe fatigue that persists or relapses for 6 months or more and is associated with at least 4 out of 8 distinctive physical symptoms.³ The CDC criteria for the diagnosis of CFS are summarised in Table 1 (adapted from ibid).

Possible medical explanations that need to be considered before CFS can be diagnosed include untreated hypothyroidism, sleep apnoea, narcolepsy, malignancies, unresolved hepatitis B or C infection, iatrogenic conditions such as side effects of medication, various psychiatric conditions, alcohol or substance abuse, and severe obesity (ibid). The CDC exclusion criteria for the diagnosis of CFS are summarised in Table 2 (adapted from ibid).

All of these, and various other conditions, are thus considered differential diagnoses of CFS.⁴ By far the most commonly mentioned differential diagnoses of CFS, however, are fibromyalgia (FM) and infectious mononucleosis. The clinical features of CFS, FM, and IM are very similar and include fatigue, muscle pain, reduced exercise tolerance, depressive complaints, and sleep disturbances.⁴,⁵
Table 1. US Centers for Disease Control (CDC) criteria for the diagnosis of chronic fatigue syndrome (CFS)

Main criteria:
- Severe fatigue that persists or relapses for at least 6 months.
- Medical explanations are excluded.

The condition will be classified as CFS if fatigue is sufficiently severe, of new or definite onset, not alleviated by rest, and results in substantial reduction in previous levels of activities and if 4 or more of the following symptoms exist:
- Impaired memory or concentration capacity.
- Recurrent sore throat.
- Tender cervical or axillary lymph nodes.
- Mild muscle pain.
- Arthralgia.
- New types of headache.
- Sleep that is not refreshing.
- Post-exertional malaise.

The condition will be classified as idiopathic chronic fatigue if fatigue severity or symptom criteria for CFS are not met.

Table 2. CDC exclusion criteria for the diagnosis of CFS

- Any active medical condition that may explain the presence of chronic fatigue, such as untreated hypothyroidism, sleep apnoea, and narcolepsy, and iatrogenic conditions such as side effects of medication.
- Any previously diagnosed medical condition whose resolution has not been documented beyond reasonable clinical doubt and whose continued activity may explain the chronic fatiguing illness. Such conditions may include previously treated malignancies and unresolved cases of hepatitis B or C virus infection.
- Any past or current diagnosis of a major depressive disorder with psychotic or melancholic features; bipolar affective disorders; schizophrenia of any subtype; delusional disorders of any subtype; dementias of any subtype; anorexia nervosa; or bulimia nervosa.
- Alcohol or other substance abuse within 2 years before the onset of the chronic fatigue and at any time afterward.
- Severe obesity as defined by a body mass index equal to or greater than 45.
Although fatigue is one of the most commonly reported non-specific clinical symptoms, the prevalence of CFS does not appear to be particularly high. Studies using the CDC definition of CFS have generally reported a prevalence of the syndrome of 0.1–0.7%. A random digit dialling study conducted in Chicago found an increased prevalence of CFS among women, minority groups, and persons with lower levels of education and occupational status.

This paper reviews the scientific literature related to current theories about the aetiology and pathogenesis of CFS. In doing so, it focuses on what currently appear to be the four most significant aspects in the development and perpetuation of this disease: the role of infectious agents, immunological, neuroendocrine, and psychiatric factors.

**The role of infectious agents**

Many CFS patients report an infectious-like onset of their illness, and much research has thus been conducted to identify a possible causative infectious agent for CFS. The fact that outbreaks of CFS have occurred in the past—and the observation that there seems to be a higher rate of onset during the cold season—also supports the hypothesis that an infectious illness can trigger the onset of CFS.

In a review of the scientific literature published before the year 2002, Evengard and Klimas conclude that various infectious agents may trigger the onset of CFS. Epstein Barr virus (EBV), the causative agent of IM and Borrelia burgdorferi (the spirochete causing Lyme disease) were most commonly cited.

Both of these agents are polyclonal immunologic activators, and could thus trigger the disease through activation of the immune system. Other possible triggering agents suggested included cytomegalovirus (CMV), human herpesvirus (HHV) type 6 and 7, Borna Disease virus (BDV), various enteroviruses, as well as Chlamydia and Mycoplasma species. Evidence for a persistent chronic infection in CFS sufferers was only found for two agents, however, HHV-6 and possibly Mycoplasma species.

Several studies published after 2002 reported results that might shed some more light on the possible role of infectious agents in the pathogenesis of CFS:

- A study involving 150 subjects with a history of IM found support for the existence of two discrete post IM chronic fatigue syndromes, one of which was still demonstrable 4 years after onset.
- Lane et al reported the detection of enterovirus sequences in muscle biopsy samples from 20.8% of CFS patients, but not in any of their control samples.
- Nicolson et al found a high prevalence of mycoplasmal (52%), Chlamydia pneumoniae (7.5%), and active HHV-6 (30.5%) infections in 200 CFS patients. Parts of those findings were in agreement with a number of previous studies that had consistently found evidence of chronic mycoplasmal infection in about 50% of CFS sufferers.
- Another study (including 22 monozygotic twin-pairs discordant for CFS), however, found no evidence of an increased prevalence of active HHV-6, HHV-7,
HHV-8, CTV, EBV, herpes simplex virus, varicella zoster virus, JC virus, BK virus, or parvovirus B19 infections in CFS sufferers.\textsuperscript{14}

- Gustaw\textsuperscript{15} reported the development of CFS in as many as 71\% of borreliosis patients and in 24\% of subjects with a history of tick-borne encephalitis.

In summary, it thus appears that the presence of chronic mycoplasmal infections might play a role in the perpetuation and/or development of CFS, while several viruses and bacteria (especially \textit{B. burgdorferi}) may trigger the onset of the disease, but do not seem to be consistently associated with its perpetuation.

**Immunological factors**

A significant number of studies have been conducted to investigate the possible presence of characteristic immunological abnormalities in CFS patients. Many of these studies have, however, produced results that were either inconclusive or in contradiction with previous findings.

A systematic review of the relevant literature published between 1966 and 2000 found no consistently present decisive differences between CFS patients and control groups in the quantity and function of T-cells, although the highest rating studies reviewed pointed to a possible increase in T-cell activity in CFS subjects.\textsuperscript{16} Clear differences could also not be demonstrated between CFS subjects and normal controls with regards to cytokine levels, B-cell quantity and function, and immunoglobulin levels. Overall, this review concluded that no consistent evidence exists for an aetiological role of immune dysfunction in CFS.

Several studies published after 2000 found additional immunological differences between CFS subjects and control groups:

- Skowera et al,\textsuperscript{17} for example, found evidence for an effector memory cell bias towards type 2 responsiveness, as well as ongoing type 0 immune activation in unstimulated cultures of peripheral blood cells, while Brunet et al.\textsuperscript{18} reported the detection of delayed-type hypersensitive responses to certain common environmental antigens in almost 50\% of CFS patients.

- Kennedy et al\textsuperscript{19} demonstrated increased neutrophil apoptosis in CFS sufferers, an immunological reaction which also occurs in patients with infection.

- A Japanese study\textsuperscript{20} found autoantibodies against type 1 muscarinic cholinergic receptors in subgroups of CFS patients, and another by Masuda et al\textsuperscript{21} reported suppressed NK-cell activity in both postinfectious and non-infectious CFS subjects.

- A Dutch study by Nijs et al\textsuperscript{22} found evidence of immune activation in patients with CFS, whereas disturbed glucocorticoid regulation of interleukin-10 was found in CFS subjects by Visser et al.\textsuperscript{23}

Far from giving any conclusive answers about the possible role of immune dysfunction in the aetiology of CFS, these more recent studies further highlight the fact that no single, consistently present immunological abnormality has yet been identified in CFS patients.
Given the multitude of positive findings in immunological studies of CFS sufferers, it does, however, seem likely that some type of immune dysfunction may play a role in the pathogenesis and/or perpetuation of this disease.

**Neuroendocrine factors**

**HPA-axis dysfunction**—One of the neuroendocrine systems that has been the subject of many CFS related studies is the hypothalamic-pituitary-adrenal (HPA) axis. Cleare has reviewed the relevant literature published up to 2002 and reports that about half of the studies reviewed in this way found evidence for lowered cortisol levels—at least at some point during the day.

Moreover, trials of replacement therapy have been able to tentatively link this hypocortisolemia to the production or perpetuation of some of the symptoms experienced by CFS sufferers.

As many factors (such as sleep disturbance, psychiatric comorbidity, medication, and chronic stress) may influence the HPA axis in CFS, Cleare suggests that the aetiology of this HPA axis disturbance is probably multifactorial. Nevertheless, evidence suggesting that the hypocortisolism frequently seen in CFS may at least partially be caused by enhanced negative feedback of corticosteroid receptors in the hypothalamus or pituitary gland was found to be fairly consistent.

Two studies also reported a reduced maximal secretory capacity of the adrenal cortex for cortisol in response to a challenge with adrenocorticotropic hormone (ACTH). This may be linked to an overall reduction in adrenal gland size which was observed in CFS patients with such a blunted cortisol response in another study.

However, Cleare reports that studies using subjects that had been suffering from CFS for a long time appeared to be more likely to find reduced basal cortisol levels than those using patients that had been ill for a shorter period.

This finding suggests that hypercortisolism might be a result rather than a cause of CFS. At least two studies published after Cleare’s review also support the hypothesis of reduced HPA axis activity in CFS indicated by lowered levels of salivary or blood cortisol.

**Autonomic nervous system dysfunction**—Orthostatic intolerance leading to neurally mediated hypotension has been found in some CFS sufferers, which suggests a disturbance of the baroreceptor reflex that controls blood pressure via the sympathetic nervous system and the parasympathetic fibres of the vagus nerve.

Abnormalities in vagal regulation of heart rate have also been observed in CFS patients. This may offer an explanation for the reduced cardiovascular response to exercise seen in some cases of CFS. Overall, there thus seems to be an autonomic imbalance with slight sympathetic predominance in CFS.

**Central sensitisation**—Several studies have found increased perception of pain (hyperalgesia) in CFS sufferers, which may be due to central sensitisation—i.e. an exaggerated response of central nervous system (CNS) neurons to peripheral noxious stimuli. Another theory for this hyperalgesia in CFS, however, is offered by Scott et al who suggest that a reduction in opioid levels that was found in CFS sufferers may cause this exaggerated sensitivity to pain.
This latter hypothesis is also supported by the decreased beta-endorphin levels that were found in CFS patients by Conti et al.\textsuperscript{39}

In conclusion, it thus appears likely that down-regulation of the HPA-axis, a disturbance of the autonomic nervous system, and certain neuroendocrine dysfunctions causing hyperalgesia may be either involved in the pathogenesis of CFS or contribute to its perpetuation.

**Psychiatric factors**

Many of the symptoms of CFS (such as fatigue, cognitive dysfunction, and sleep disorders) are also present in some nonpsychotic psychiatric disorders. For this reason, some physicians consider CFS to be a psychiatric condition.\textsuperscript{6} CFS patients, however, usually disagree with such a categorisation and complain that they are mistakenly given a psychiatric label. This impression was confirmed by a study, which found that a large proportion of CFS sufferers had, indeed, been wrongfully diagnosed with a psychiatric condition in the past.\textsuperscript{40}

Nevertheless, personality disorders are quite prevalent among CFS sufferers\textsuperscript{41} and cannot always be explained as a result of the experience of this chronic illness alone.\textsuperscript{42,43} Comorbid depression, for example, is commonly associated with CFS.\textsuperscript{44,45} Nevertheless, the depression seen in CFS appears to differ significantly from that usually associated with psychiatric disorders.

CFS patients, for example, have been found to rate their current health status lower, show a stronger illness identity and greater impairment in physical functioning, complain more of bodily pain, attribute their condition more to external factors, and show a greater reduction in vitality and social functioning than subjects suffering from major depression.\textsuperscript{46,47}

The lack of self esteem and the tendency towards self-blame often observed in patients with major depression also appears to be rare in CFS sufferers.\textsuperscript{48} Moreover, there seems to be a significant difference in the neuroendocrine aspects of CFS and major depression: While an up-regulation of the HPA axis has often been observed in major depression, CFS sufferers more often show reduced HPA axis activity.\textsuperscript{28} Given this distinct nature of the depression seen in CFS, it thus seems unlikely that this disease is simply a result of a pre-existent state of major depression.

Nevertheless, there is also a substantial amount of evidence pointing towards a possible involvement of psychiatric factors in the pathogenesis and perpetuation of CFS. A study by Hatcher and House,\textsuperscript{49} for example, found that stressful events, especially such as were characterised as dilemmas, often seem to precede the onset of CFS.

**Conclusion**

In spite of the presence of abundant research, the aetiology and pathogenesis of CFS still remains unknown. Given the fact that disturbances to a number of body systems as well as to the mental status of CFS sufferers have been found, it seems likely that the causes of this illness are multifactorial and may vary from patient to patient.\textsuperscript{50}

From the evidence reviewed above, it appears possible that an infectious agent may trigger the onset of the CFS, especially in patients that are experiencing a dilemma or
that are already suffering from a psychiatric condition which might impact on their immune status. Changes to the immune, and possibly the neuroendocrine system, might then be caused or perpetuated either by the infection becoming chronic or because of exaggerated illness worry and identification with the disease.

For the treatment of CFS, a multi-directional approach may be most likely to prove beneficial. This, however, is dependent upon improved and more thorough diagnostic procedures. Instead of simply dismissing CFS sufferers as psychiatric cases, as has often been the case in the past, the patient should be assessed to look for known medical conditions that may explain at least some of their symptoms.

An important step in this direction would be a more uniform application of the battery of clinical tests suggested by the 1994 CDC study group. The diagnostic protocol proposed by this group includes history taking; physical and mental examination; complete blood count (CBG) and urine analysis (UA); as well as tests to determine erythrocyte sedimentation rate (ESR) and levels of alanine aminotransferase (ALT), total protein, albumin, globulin, alkaline phosphatase, Ca, phosphorus (PO₄), glucose, blood urea nitrogen (BUN), electrolytes, creatinine, and thyroid stimulating hormone (TSH).

A systematic review of various approaches to the treatment of CFS suggests that cognitive behavioural therapy as well as graded exercise programmes seem to be most beneficial in the treatment of CFS. Both of these approaches do, however, appear to primarily help improve patients’ coping skills rather than eliminating or reducing the symptoms of CFS. The identification and specific treatment of possible aetiological, contributory, or predisposing factors should therefore remain an important concern of primary health care providers.

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Massive haemorrhage caused by a bone marrow aspirate and trephine (BMAT) procedure in a uraemic patient

Nicholas Gray, Geoffrey Hawson, Peter Hollett, Andrew Cluer

Bone marrow aspirate and trephine (BMAT) is generally considered a safe procedure. We report a case of massive haemorrhage following BMAT that indirectly contributed to the patient’s death.

Case report

A 60-year-old man with a history of monoclonal gammopathy of uncertain significance presented with oliguric renal failure requiring dialysis. Investigations revealed a positive urinary Kappa Bence Jones protein of 615 mg/L and a monoclonal Kappa IgG paraprotein of 27 g/L on serum protein electrophoresis. Renal biopsy showed cast nephropathy with involvement of 10% of renal tubules.

He was transferred to another hospital where a BMAT at the right posterior superior iliac spine confirmed the diagnosis of myeloma with 36% plasma cells. The procedure was not technically difficult despite the patient weighing 120 kg. Platelet count and coagulation studies (prothrombin time 12 seconds, activated partial thromboplastin time 33 seconds, fibrinogen 3.2 g/L) were normal. The patient did not have a history of a bleeding diathesis.

The next day he developed painful swelling of the right buttock and falling haemoglobin. Eighteen units of packed red cells (PRC) were transfused over 4 days. During this time he completed one cycle of VAD chemotherapy (with a reduced dose of adriamycin). He had three cycles of plasma exchange with fresh frozen plasma replacement to remove plasma light chains in an effort to improve renal function and prognosis. He was transferred back to the original hospital.

An MRI scan showed extensive haematoma involving the right thigh (Figure 1). Eleven units of PRC were transfused over the next 2 weeks. Coagulation studies and platelet count remained normal. Despite diminishing transfusion requirements, he continued to deteriorate and developed Klebsiella oxytoca septicaemia. Fine needle aspirate confirmed the focus of infection as the right thigh haematoma which was incised and drained.

He was admitted to intensive care post operatively where a total of 42 units of PRC were transfused over 10 days. Bleeding continued until a false aneurysm in a third degree branch of the right internal iliac artery was identified at angiography (Figure 2) and successfully embolised. The false aneurysm was adjacent to the anterior iliac crest, some distance from the original site of the BMAT. Platelet function testing with collagen/epinephrines, haemophilia (factor 8 and 9) screens and Von Willebrand screens were performed when the active bleeding had resolved and were normal.

Pneumocystis pneumonia and multiple episodes of dialysis access related septicaemia followed. He was discharged 112 days after admission but was never well enough to tolerate further chemotherapy. He died 4 months later.
Discussion

This case demonstrates that BMAT can cause life-threatening haemorrhage.

A retrospective survey reported 54890 marrow biopsies, of which 67% also had a trephine. There were 26 adverse events including 14 haemorrhages, 7 broken needles, 3 infections, and 2 miscellaneous events. The haemorrhages resulted in prolonged hospitalisation, 1 death, 6 cases needing PRC transfusion, and 1 case needing a platelet transfusion. Risk factors for bleeding were myeloproliferative disorders (due to platelet dysfunction), aspirin, warfarin, disseminated intravascular coagulation, and obesity. The overall serious adverse event rate was 0.05%.

A prospective study reported 13,506 bone marrow examinations, of which 3927 were aspiration alone, and 9579 were aspiration and trephine. There were 17 adverse events including 10 haemorrhages, 4 infections, 2 needle failures, and 1 of pain. Haemorrhage was managed conservatively in 4 cases, required PRC transfusion in 3 cases, and contributed to death in 1 case. Risk factors were myeloproliferative disorders, thrombocytopenia, platelet dysfunction, warfarin, and heparin.

Renal impairment is associated with an increased tendency to bleed due to platelet dysfunction. This may be reduced by correction of anaemia with red cell transfusion, correction of uraemia with dialysis, intravenous deamino-8-D-arginine vasopressin, and administration of intravenous or topical oestrogen. These measures were all attempted in this case.

BMAT is an important diagnostic investigation which can be associated with significant adverse events, particularly haemorrhage. The risk may be reduced by ceasing antiplatelet agents and anticoagulation before the procedure. Platelet dysfunction due to renal impairment possibly increases the risk of haemorrhage following BMAT.
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Post-traumatic haematoma: a red herring to something more sinister

Hisham Hurreiz, Irshad Hussain, Musa Barkeji

This case study describes the initial reporting of a post-traumatic swelling as a haematoma following a relatively minor trauma, which upon further investigation, was proven to be a high-grade liposarcoma.

Post-traumatic haematomas occur commonly but the limits to which another diagnosis should be sought needs to be clarified. We present our case study followed by a discussion on post-traumatic haematoma emphasising its natural history as well as a brief account on soft tissue sarcomas and their management.

Case report

A 48-year-old left-handed well-built information technology consultant sustained a right arm bruise when he accidentally slipped onto the edge of a workbench. This posterior mid-arm injury caused immediate pain and a bruise. Two days later, he noticed a soft painless lump which continued to increase in size. Two months later, his general practitioner referred him to the surgical department due to a slow growing non-tender lump measuring 10×10.5 cm. His only significant past medical history was of well-controlled hypertension and nasal polyps.

Initial examination suggested a haematoma but it was thought important to rule out a false aneurysm. Duplex ultrasound scan suggested a possible haematoma. MRI was the next investigation of choice but due to a delay of 5 months a CT scan was requested. No muscle defect, calcification, or intramuscular haematoma were detected on the CT scan.

Nine months after the initial injury, the patient began to experience severe pain forcing him to seek urgent help. He presented to the Accident and Emergency department complaining of a 1-month history of worsening pain and paraesthesia of the right forearm and hand. Pain was now increasing when he moved his elbow and wrist, which impeding him in his occupation; he had been on sick leave for the last 3 weeks.

Examination revealed a 12×12×10 cm stony hard circular lesion which was tender and immobile. There was decreased movement of joints due to pain and right hand grip was recorded as 4/5. A subsequent emergency MRI scan reported features which excluded the possibility of a lipoma, with areas that could represent a haematoma with no evidence of malignancy.

Exploration of the lesion revealed a 12×12×10 cm smooth-surfaced, well-circumscribed, poorly encapsulated, multi-lobulated intramuscular (triceps) mass weighing 515 grams. Histology revealed an incompletely excised high-grade liposarcoma for which he was urgently referred to a tertiary cancer specialist Unit. Plans were then being made for another operation for a wide tumour clearance. This
operation was performed the next week followed by a course of radiotherapy and the patient is now recovering well.

**Discussion**

Post-traumatic haematomas are commonly encountered in the lower limbs following athletic injuries. Their clinical presentation is variable but complete resolution usually occurs within 6 weeks of the injury.

In rare cases, the lump might persist for longer than expected becoming hard and more circumscribed and leading to diagnostic confusion with soft tissue sarcomas. There are reports in the literature of ancient haematomas (up to 20 years old) clinically and radiologically mimicking soft tissue sarcomas but there is no documented evidence to suggest that long-standing haematomas have a potential to become malignant.

The occurrence of a soft tissue sarcoma developing on an extremity following trauma in our case is probably a coincidence; usually a minor accident or trauma draws attention to a pre-existing tumour. In these cases, the diagnosis of a muscle haematoma should be considered if the swelling fails to regress in size or continues to enlarge after a period of conservative treatment; or if the history of trauma is vague and the size of the resultant swelling does not correspond to the severity of the insult. In these cases other sinister causes must be considered and excluded before assuming that the swelling is due to trauma.

Soft tissue sarcomas are uncommon tumours comprising about 1% of all malignancies with liposarcomas constituting about 25–30% of all sarcomas. They may present with diverse clinical and radiological manifestations. The prognosis of soft tissue sarcomas is generally poor with those affecting the extremities and trunk having a better prognosis than visceral and retroperitoneal sarcomas.

Early diagnosis is crucial if the tumour is to be completely resected and total cure is to be achieved.

Although ultrasound and CT scans can be used as initial radiological investigations in suspected cases of muscle haematoma, MRI is the investigation of choice, especially if contrast is used. The diagnostic yield from contrast MRI is high in cases of soft tissue sarcomas of the limbs as well as in cases of post-traumatic muscle haematoma. This investigation is very useful in the follow-up of patients with muscle injuries.

The diagnosis of soft tissue sarcomas is usually established by doing a core biopsy, although Singh et al showed that fine needle aspiration biopsy (FNAB) can give results which are comparable to core biopsy, especially when trying to differentiate between benign and malignant soft tissue tumours. The yield from FNAB can be improved by combining it with other investigations such as immunohistochemistry and electron microscopy.

Sarcomas are ideally treated in specialised tertiary referral centres by multidisciplinary teams. Treatment consists of surgical excision (in the form of compartmental resection in case of extremity sarcomas), combined with adjuvant radiotherapy and/or chemotherapy. The main aim of treatment is to prevent recurrence. The expertise of plastic surgeons is needed in cases involving the extremities to close the defect resulting after surgical excision of the tumour.
Conclusion

Soft tissue sarcomas are rare malignant tumours. Their diagnosis is often missed due to confusion with other benign swellings. The surgeon needs to keep an open mind in cases of limb swellings following trauma. Moreover, all cases should be investigated thoroughly until a diagnosis of either a benign or malignant lesion is reached.

Malignant lesions must be referred urgently to specialist centres for further management as the consequences of delayed management are usually serious with increased mortality and morbidity to patients.

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References:


Evidence, economics, and emotions: the case for temozolomide

David Hamilton

**Abstract**

Temozolomide, given as part of first line therapy in the treatment of grade IV astrocytoma, has been shown to improve survival in the short term. The financial cost of the treatment is considerable in New Zealand. This drug provides a good example in the field of oncology of a modern expensive pharmaceutical being a clear improvement over its cheaper predecessors, but it raises the question of what price should be paid to prolong survival in an incurable illness?

<table>
<thead>
<tr>
<th><strong>Drug</strong></th>
<th>Temozolomide (Temodal®) oral capsule</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class</strong></td>
<td>Cytotoxic imidazotetrazine alkylating agent</td>
</tr>
<tr>
<td><strong>Indications</strong></td>
<td>Patients with newly diagnosed glioblastoma multiforme (grade IV astrocytoma) concomitantly with radiotherapy and then as adjuvant treatment.</td>
</tr>
<tr>
<td></td>
<td>Patients with recurrent high-grade glioma, such as glioblastoma multiforme or anaplastic astrocytoma.</td>
</tr>
<tr>
<td></td>
<td>First-line treatment for patients with advanced metastatic malignant melanoma.</td>
</tr>
<tr>
<td><strong>Costs</strong>*</td>
<td>Average cost per patient for newly diagnosed regimen = NZ$53,500 (Manufacturer price = $31,000)</td>
</tr>
<tr>
<td></td>
<td>Average cost per patient per monthly cycle for recurrent high grade glioma = $6,100 (Manufacturer Price = $3,500)</td>
</tr>
<tr>
<td></td>
<td>(Costs to patient are estimated by applying MIMS’ standard factor of 1.725 to the stated manufacturer price. This may vary between pharmaceutical outlets and the actual price charged may be less. Subsequent prices in this article are based on this standard mark up.)</td>
</tr>
</tbody>
</table>


| **Subsidy** | None. Not currently included in Pharmaceutical Schedule. |
**Background**

Primary brain tumours account for 3.1% of cancer deaths in New Zealand (NZ) males and 2.6% of cancer deaths in NZ females.

In 2001, 146 males and 108 females were registered as having a primary brain tumour and 130 males and 95 females died of brain tumour.**

The majority of primary brain tumours in adults are high-grade astrocytomas of which the great majority are glioblastoma multiforme (GBM) giving a figure of approximately 100–120 patients per annum in NZ with GBM.

GBM is a tumour with a very poor prognosis, with a median survival of 9–10 months and 1-year survival of around 40%. Figures quoted in the literature may vary depending on case selection, with older patients known to experience a poorer outcome. The standard therapy is maximal safe surgical debulking followed by high-dose radiotherapy.

The use of chemotherapy with nitrosoureas remains controversial. The majority of published trials of chemotherapy in high-grade glioma include a proportion of patients with grade III astrocytoma in whom the prognosis is better.

The most recent published meta-analysis*** of chemotherapy as part of initial combined modality management of high-grade glioma reported an increase in 1-year survival for GBM from 35% to 41%, and 2-year survival from 9% to 13% with the addition of nitrosourea-based chemotherapy.

The evidence behind the use of temozolomide in newly diagnosed GBM is the large randomised study published in the *New England Journal of Medicine**** in March 2005, which only included patients with GBM, aged ≤70 and in good general condition. This reported an improvement in 1-year survival from 50.6% to 61.1% and in 2-year survival from 10.4% to 26.5% with the addition of temozolomide to postoperative radiotherapy.

This increase in 2-year survival has been hailed as the greatest improvement in outcome for GBM patients in several decades and sets the new “gold standard” for therapy. This has led to the inclusion of temozolomide on the Australian Pharmaceutical Benefits Schedule (PBS) for this indication on 1 July 2005, and the inclusion of this indication by Medsafe on 1 September 2005 in NZ.

In the relapsed situation, 20% of patients with GBM would be expected to have progression-free survival at 6 months on
Discussion

At the time of writing, temozolomide remains unfunded in NZ for any indication. This led to the recent patient protest outside parliament on 15 November 2005 calling for an accelerated process to provide funding through PHARMAC and to bring NZ into alignment with Australia.

The Pharmacology and Therapeutics Advisory Committee (PTAC) was considering the application for funding at a meeting on 17 November 2005.

The evidence for efficacy of first line temozolomide is strong, being based on a large randomised controlled phase III trial. The results reported are also similar to previously reported phase II data. The results available however extend only to 2 years from treatment. Although 26% of treated patients are alive at 2 years, only 10.7% are progression-free.

Unlike most cancers, brain tumours can affect patients of any age and are the second most common tumour in children after leukaemias. GBM however occurs in a minority of paediatric patients. It is, however, the commonest histology in adult brain tumours.

As a clinician treating patients with brain tumours I wish to be able to offer them the best standard treatment available. Aggressive treatment is not necessarily appropriate for all patients, but applying similar criteria to those used in the New England Journal of Medicine study, patients with a good functional status following surgery and a “good” quality of life are most likely to benefit from the addition of temozolomide to radiation therapy.

In discussing therapeutic options with patients, the Health & Disability Commissioner has ruled that this should include options not necessarily available in the treating centre, or even in NZ. “Standard” treatment therefore has to include world
standards. Patients confronted with this disease and its dreadful prognosis make extensive use of the Internet looking for treatments that offer hope of benefit.

In the absence of a validated, subsidised therapy, they may look at other more anecdotal and potentially harmful regimens.

This raises the economic question of what value does NZ society place on the prolongation of survival, probably of the order of magnitude of a few months for patients with an effectively incurable disease.

Assuming 100 patients diagnosed with GBM in NZ per annum, and that only 50 are fit enough to undergo combined modality therapy, the additional annual drug cost for temozolomide (not including the necessary anti-emetics and prophylactic antibiotics) would be $2,675,000.00. In clinical outcomes, this would lead to an additional 8 patients alive at 2 years, at an effective cost of $334,375 per patient.

In NZ, PHARMAC is charged with containing the cost of the pharmaceutical budget. Any new therapy has to “compete” with others for funding from a limited budget. Something else has to give to enable a new therapy to be subsidised. In the case of management of newly diagnosed GBM, this is effectively a new treatment, and does not replace any prior component of treatment. Without an increase in the overall pharmaceutical budget, it is difficult to see how temozolomide will secure funding.

Unfortunately, brain tumour therapy is only an example of a common issue in oncology practice where new drugs used in addition to existing regimens add to survival at defined time points in a statistically significant way at a cost commonly around $60,000 to $100,000 per patient treated. With absolute gains in survival of 3%–10% this may cost one million dollars per additional patient alive.

Statistics and health economics are however very difficult concepts for the individual wishing to have the best therapy possible.

Ultimately, politicians will decide where NZ stands in the world rankings of contemporary medicine, and what offers the best health return from a constrained budget.

**Conflict of interest statement:** I have not been the recipient of any funding from Schering-Plough, the manufacturer of Temodal®, and have not participated in any clinical trials of its use. I am the principal neuro-oncologist for the Wellington Region.
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NZMJ Note: Refer to http://www.nzma.org.nz/journal/118-1227/1806 in this issue of the Journal for PHARMAC’s response.
Bloodletting


Now, the list remedy that they so invariably employed is one that it behoves us to see if we employ enough—I mean venesection, or bleeding. Their object in bleeding, of course, was the outcome of the old humoral pathology, that by letting blood they would get rid of the vicious-humours from the body, and hence effect a cure.

More modern pathology has consigned that idea to the limbo of the rubbish-heap; but, though the theory is wrong, is the practice wrong in given cases? As in the old days, many lives were lost by too generous a system of venesection, I venture to think, nowadays, not a few lives have been lost by the modern practitioner being afraid to use that form of treatment.

Any one who has been a resident house surgeon in a hospital must have noticed how very much better scalp-wounds do where there has been free bleeding than where the haemorrhage has been scanty. In the latter, cellulitis is common; in the former, uncommon. Why this should be so I am unable to say; but as a fact of experience I think you will bear the statement out.

In pneumonia in a plethoric big man, bleeding, even in the early stages, will do good. In the late stages, when the right ventricle is engorged, there is no doubt that bleeding has saved life; and in such cases the physiological action is perfectly plain. I admit it requires pluck on the part of the medical practitioner where a patient’s respirations are 64, perhaps, and he is delirious, with a very high temperature, to bleed him; but if he be much cyanosed, and the right ventricle is failing, with the first sound of the heart almost gone, in most cases venesection should, if the administration of oxygen fail, be tried. Again, in certain heart cases where cyanosis and dilatation of the right ventricle are the main symptoms, bleeding should be carried out.

I was called about a week ago to see a girl of sixteen, who appeared to be at the point of death; she was extremely cyanosed, the heart was working with the utmost difficulty—the action slow and extremely irregular; the right ventricle much dilated; dulness extending 2 in. to the right of the sternum; and a loud, systolic murmur at the apex. With great difficulty I got permission to try venesection, and, had she died, no doubt I should have been held liable for her death; but, happily, the effect of removal of 16 oz. of blood was magical, and she is now convalescent. I may add that when I bleed I make a practice of giving a dose of digitalin first. There is yet one other class of case where venesection is of the utmost value, and that is where convulsions persist, and the patient is practically in the status epilepticus.

I remember seeing, when I first came here, a young man of twenty-five with mild scarlet fever. On the tenth day he developed nephritis, with blood, casts, and scanty urine. He rapidly passed into a uraemic condition, with violent convulsions. I purged and sweated him thoroughly, and in order to allay the convulsions I put him under chloroform. As long he was deeply under, the convulsions ceased; but the moment the
drug was relaxed they came on again. I then bled him freely from the right arm, taking away 25 oz. of blood. The result was that he never had another convulsion, and he made a rapid recovery.

NZMJ Note: See http://www.pbs.org/wnet/redgold/basics/bloodlettinghistory.html for the history of bloodletting.
A rare complication of port-a-cath use

Omprakash Damodaran, Girish Mallesara

A 29-year-old man with Ewing’s sarcoma of his right posterior chest wall was admitted to the Oncology Unit for his second course of chemotherapy. To facilitate chemotherapy administration, a dual lumen port-a-cath had been inserted into the right subclavian vein 3 weeks earlier, with the tip of the catheter placed in the superior vena cava.

The patient felt some pain under his right clavicle after the nurse flushed normal saline into the catheter, after not being able to draw blood from both lumens. A chest X-ray taken following this (Figures 1 and 2) revealed a fractured tip of the catheter in a branch of the left lower pulmonary artery. The retrieval of the fragment was performed successfully, using a snare catheter passed through the right femoral vein (Figure 3). The patient recovered with no complications and continued with his chemotherapy.

Figure 1. Chest X-ray (PA view)
Arrow shows port-a-cath fragment

Figure 2. Chest X-ray (lateral view)
Arrow shows port-a-cath fragment

URL: http://www.nzma.org.nz/journal/118-1227/1777/ © NZMA
Discussion

It’s been estimated that less than 1% of indwelling venous catheters fracture.\(^1\) Aetiology might be associated with the pinching effect of the catheter as it passes between the clavicle and the first rib.\(^2\) Fractures can be minimised by instructing the patient to abstain from heavy physical activities or movement of the shoulder.\(^3\) Once a fracture has occurred, the fragment should be located and removed as soon as possible to prevent life-threatening dysrhythmias and other complications.\(^3\)

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Acknowledgement: We thank Dr Fiona Able (Clinical Oncologist, Mater Hospital, Newcastle) for her assistance.

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β blockers—first choice in the treatment of hypertension?

One year ago (NZMJ 17 December 2004), we abstracted a Swedish paper that cast doubts on the role of atenolol in hypertension. Now, the same authors report on a meta-analysis involving 127,879 patients in which they review the role of all β blockers as first-line antihypertensive drugs, bearing in mind that they are commonly recommended in this role. And their conclusion is that “the effect of β blockers is less than optimum, with a raised risk of stroke. Hence, we believe that β blockers should not remain first choice in the treatment of primary hypertension and should not be used as reference drugs in future randomised controlled trials of hypertension.” Strong words. However, they say that β blockers do lower blood pressure to the same extent as other drugs—but are less effective in stroke prevention. An accompanying editorial agrees with their views but reminds us that some patients genuinely do need β blockers as their first line therapy—specifically those with coronary artery disease.

Hospitalisation before and after gastric bypass surgery

The use of Roux-en-Y gastric bypass (RYGB) has been reported to be effective in the treatment of obesity and its related comorbidities—in particular diabetes, hyperlipidemia, hypertension and obstructive sleep apnea. Amazingly, more than 100,000 such procedures are performed annually in the United States. One would suspect that vanity might also figure in the indication for surgery list. Anyway, how effective is it in terms of health, as measured by post surgical hospitalisation? A recent study from California reports on 60,077 patients who underwent RYGB. Apparently, the rate of hospitalisation in the year following RYGB was more than double the rate in the year preceding RYGB (p<.001). And the rates were similar in the second and third years. The most common reasons for admission prior to RYGB were obesity-related problems (e.g. osteoarthritis, lower extremity cellulitis), and elective operation (e.g. hysterectomy), while the most common reasons for admission after RYGB were complications often thought to be procedure related, such as ventral hernia repair and gastric revision. Swings and roundabouts.

What about warfarin after myocardial infarction?

Patients with a history of myocardial infarction are at increased risk for recurrent infarction, stroke and death. Several interventions have proven beneficial in the secondary prevention of myocardial infarction, including β blockers, angiotensin-converting enzyme inhibitors, lipid-lowering therapy, and aspirin. Although some studies have shown that addition of warfarin to aspirin decreases subsequent risk for cardiovascular events, this has not become standard management principally because of the dangers of haemorrhage. A recent meta-analysis of ten trials involving over
11,000 patients concludes that the cardiovascular benefits of warfarin outweigh the bleeding risks in patients who have a myocardial infarction or an acute coronary syndrome, provided that they have a low or intermediate risk for bleeding. A very important provision! The report did not include patients with coronary stents and the findings may not apply to them.


**Clostridium difficile colitis after colorectal surgery**

Apparently *Clostridium difficile* colitis is a known complication of colon and rectal surgery occurring in over 20% of such patients. In the USA at least, prophylactic antibiotics have become standard in elective colon and rectal surgery, but controversy persists on the ideal choice and route of antibiotics: oral, intravenous (IV), or both in combination. The authors of this study report on a cohort of 304 patients, including 107 who had pre-operative oral antibiotics. They found that the rate of post-operative *C. difficile* colitis was 4.2% in the entire study population. The rate of *C. difficile* infection was higher in patients who received oral antibiotics (7.4%) compared with patients who did not receive oral antibiotics (2.6%; P=.03). There were no *C. difficile*-related mortalities. Consequently they “recommended that oral nonabsorbable antibiotics not be used in pre-operative bowel preparation regimens since post-operative *C. difficile* infection can lead to additional morbidity, length of stay, and hospital costs.” Seems reasonable.

Arch Surg 2005;140:752–6

**Vasodilator therapy in severe aortic regurgitation**

Severe aortic regurgitation is optimally treated by aortic-valve replacement, otherwise the defect causes left ventricular volume overload, leading to progressive dilatation of the chamber and eventual deterioration in left ventricular function—and death. A suggestion was made several years ago that vasodilator therapy could preserve left ventricular function and delay the need for surgery. This theory has been tested in a recent Spanish trial. They randomly assigned 95 patients with asymptomatic severe aortic regurgitation and normal left ventricular function to receive nifedipine, enalapril, or no treatment, to identify the possible beneficial effects of vasodilator therapy on left ventricular function. After a mean of seven years of follow-up, the rate of aortic-valve replacement was similar among the groups: 39% in the control group, 50% in the enalapril group, and 41% in the nifedipine group (P=0.62). Furthermore, such therapy did not reduce the aortic regurgitant volume, decrease the size of the left ventricle, or improve left ventricular function.

Statins and myopathy

The day following a relevant television documentary (Lipex, Close-Up, TV1, 1 November 2005, compiled by Ian Sinclair), I was consulted by a patient with symptoms of angina. He was interested in protective measures to reduce risk but explanations of how treatment with a low-medium dose of a statin might reduce this by about 1 in 10 over 5 years set against a risk of myopathy of 1 in 10,000 were to no avail as his wife now considered the medication “far too dangerous”.

I am sure this situation was echoed in consulting rooms around New Zealand, despite the programme including factual information on risks and benefits. Urban myths about statins (fuelled by talkback radio) are prominent and will no doubt be further stimulated, but there are issues requiring reflection.

The major preventive benefit (in those at sufficiently high risk) and extremely low adverse event rate of statin therapy were restated in the recent review of placebo-controlled trials.1 We do have to be mindful that these trials generally used fixed low-medium doses of statins while higher doses and combination therapies increasingly being urged2 could have different benefit/risk equations. The recently published Z phase of the A-Z Trial3 documented nine cases of myopathy (CK >10 times the upper normal limit) among 2263 patient taking simvastatin 80 mg daily for 6 to 24 months (a rate of about 1 in 250).

Atorvastatin, the only other statin available in New Zealand (by Special Authority), even when used in highest recommended doses (80 mg/day) seems to be associated with low rates of myopathy.4 Additional concomitantly-used drugs (including erythromycin, calcium antagonists, cyclosporine, some antifungals and fibrates), diet (notably grapefruit juice), and other patient-related factors may interfere with metabolic breakdown (or otherwise interact with statins) and increase the risk of myopathy. Not all these factors can be reliably avoided in routine clinical practice and are more likely encountered there than in tightly controlled trials.

Perceptions of risk and benefit of medical interventions among health professionals, patients, and the public often show significant variance from what has been reliably documented.5 Coverage in the media highlighting particular problems, even when figures given are reasonably factual, is one contributor to these potential distortions. We should strive to put across a correctly balanced view of benefits of risks of proposed therapies and interventions.

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Disclosures: I have attended meetings sponsored by Pharmaceutical Companies who market statins in New Zealand (Merck, Sharp and Dohme (NZ) Ltd, Pfizer (NZ) Ltd). I am a member of the Cardiovascular Subcommittee of the Pharmacology and Therapeutics Advisory Committee to PHARMAC.

References:


Library outreach for health professionals

It is not news that New Zealand is facing a problem with retention of general practitioners (GPs) in rural areas. Janes and Dowell report that one area of concern to rural practitioners is the lack of professional support. The role of the medical library is to provide support. How can we get together?

In 2003, the Midcentral District Health Board (DHB) Clinical Library launched its Outreach Service for community and rural health professionals. The service, which was developed by Library staff and supported by the DHB, was conceived as a way of delivering quality health information and library services to health practitioners in the community.

It was designed to allow easy web access, via the Library Homepage, to a comprehensive range of library services and resources. Registration was made available to all healthcare practitioners who live and/or work in the Midcentral DHB region.

Studies analysing the information needs of rural health professionals have shown a significant diversity in the types of information sought. Whilst a need for current clinical information was expected, there was also a need for information covering nursing, health administration, allied health, and social sciences.

To this end, registrants are given desktop access to library databases such as Medline, Psychinfo, MDCConsult, and Health Business Elite and as well as online access to a number of major texts including Harrison’s Textbook of Medicine, The Merck Manual, Rakel’s Textbook of Family Practice, Nelson’s Textbook of Pediatrics, and many others. Access is also given to hundreds of full text journals which cover a broad range of topics including general medicine, sports medicine, complementary therapies, allied health, nursing, and general reference.

Library services include literature searches, photocopying, interlibrary loans, lending of books and journals, and the ever-popular Journal Contents Page service. All of these can be accessed via the Internet from office or home.

Rural GP access to, and use of, the Internet for patient care is problematic. Janes et al report that although approximately 70% of rural GPs have Internet access at work, only a small percentage of them use it more than twice a week for patient care. Access is not enough. Having the knowledge and the skills to efficiently access information is equally important. The MDHB Clinical Library offers tutorials on navigating the library webpage and in efficient use of the databases it provides.

From 2006, Outreach registrants will be able to enrol for tutorials based at Palmerston North Hospital or alternatively may arrange for Library staff to visit them at their own place of work. For those who do not have Internet access, or who do not want to do their own searching, a phone call to the library is all it takes to have a search done by a qualified librarian.

There is no need for the rural GP or practice nurse to feel disadvantaged through lack of quality information. The Outreach Service is also available to urban GPs as well as
to a wide range of nursing and allied health professionals, midwives, pharmacists, dentists, and Rest Home staff.

Midcentral DHB Clinical Library is the first in New Zealand to offer this complete package free-of-charge to its wider health community. Other DHB libraries are now considering their options.

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References:


PHARMAC’s response on temozolomide and funding costly medicines that prolong life shortly

Dr David Hamilton (http://www.nzma.org.nz/journal/118-1227/1774) accurately describes the clinical benefits of temozolomide and PHARMAC’s consideration of funding; and how this relates to what price to pay to prolong survival in incurable illnesses. We update progress with the funding of temozolomide, and agree there are dilemmas around the funding of high-cost medicines that give definite if limited survival gains.

Temozolomide

We have moved as quickly as possible to assess temozolomide. It is not usual to carry out a clinical review by PTAC and an economic analysis before a drug is registered by Medsafe and the supplier has made an application to PHARMAC; however this is what was done for temozolomide. Details of timelines can be seen in the Appendix to this letter. Our approach with temozolomide recognised the limited life expectancy for patients diagnosed with glioblastoma multiforme, and their particular needs.

Temozolomide is unfortunately a costly treatment, currently in the region of $40,000 to $50,000 per patient per year, and even with only 50 treated new patients per year this would result in an overall annual cost of around $2 million. For that sort of expenditure our analysis must be robust and the expenditure compared with other areas of need.

We consider that there are a number of reasons to support the funding of temozolomide when considered under PHARMAC’s decision criteria. We have also had constructive discussions with the supplier and have sent them a proposal for funding, and anticipate a decision early in 2006.

What value does society place on limited survival gains in incurable disease?

Currently, PHARMAC has funding available for medicines such as temozolomide and has a programme of new investments. However, that funding would not cover all the applications for new medicines that we have received, and funding for out-years is uncertain, so choices between medicines need to be made.

There will always be difficult decisions to make when funding pharmaceuticals within a constrained budget, and these decisions are not going to get any easier. This prioritisation will be particularly tested in coming years, as on the horizon are a number of new oncology drugs and other beneficial high-cost medicines.

Spending on cancer drugs funded from the community pharmaceutical budget has grown from just over $1 million three years ago to nearly $12 million in 2004/05. This makes it one of the fastest-growing areas of expenditure, and some new and expensive treatments are also being considered. Although difficult to obtain reliable data on hospital use of cancer pharmaceutical treatments, our best estimate is that
hospitals spent $35 million in the 2004/05 financial year. Growth in cancer treatment has outstripped growth in spending on other treatments.

Newer oncology drugs are often extremely expensive, often in the range (and sometimes greater than) $50,000 per year per patient—temozolomide and trastuzumab (for breast cancer) being good examples. These drugs will mean that we will all need to continue to make decisions about where New Zealand’s priorities lie. At the moment, expensive treatments, which may offer significant benefits to a small number of people, must compete for limited funds with less expensive medicines that treat large numbers and achieve greater putative population health gains\textsuperscript{2} for the same total costs. The growing number of costly new treatments makes such decisions both more common and more difficult.

Relevant to these issues, there has been recent debate in the Journal whether PHARMAC should lower the discount rate used in its economic analyses (affecting how medicines are prioritised).\textsuperscript{3–5} Such issues are important to the funding of medicines that give short-term survival gains, as lower discount rates tend to advantage long-term gains at the expense of short-term.

PHARMAC is currently reviewing its decision-making process for high-cost medicines—driven in part by having to turn down treatments for small numbers of people, who then miss out. PHARMAC’s Board should be considering the outcome of the review process next year; prior to that, any proposed changes to our decision making processes would undergo public consultation.

PHARMAC’s prioritisation process tries to allocate scarce resources in a fair and transparent way—consistent from year to year and medicine to medicine.\textsuperscript{6} Transparency in the decision-making is important, so that people understand the decision even if they don’t agree with it. While we have a fixed, albeit increasing, pharmaceutical budget, the issue of rationing—making explicit choices to fund and not fund particular medicines—remains something that New Zealand must keep doing.

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Conflict of interest: Scott Metcalfe is externally contracted to work with PHARMAC for public health advice. Steffan Crausaz, Peter Moodie and Wayne McNee declare no conflicts.

References and endnotes:

   Section 2.2 Decision Criteria


9. PTAC reviewed the application from Schering-Plough and advice received from CaTSoP at its November scheduled meeting. PTAC considered both the Stupp et al 2005 and Athanassiou et al 2005 RCTs—both being on the use of temozolomide as adjunctive therapy in combination with radiotherapy in patients with newly diagnosed glioblastoma multiforme, but with their patient populations differing in the extent of disease progression at randomisation (the patients in the study by Stupp et al had a generally higher performance status than those in Athanassiou et al). PTAC considered that the patient population in Athanassiou et al would be more representative of the patients presenting with glioblastoma multiforme in NZ.

PTAC considered that the available evidence demonstrated that some patients obtain a considerable benefit, with an additional 15% of patients surviving at 2 years compared with radiotherapy alone (median survival benefit 2.5-5.7 months). However, PTAC considered the majority of patients would obtain little benefit from treatment with temozolomide, and that it was appropriate to examine targeting of treatment to those patients likely to benefit from treatment with temozolomide. PTAC considered that, from the data provided, patients with higher performance status (Karnofsky score >80, WHO score 0 or 1) obtained significant benefit with temozolomide treatment; tumour resection (rather than biopsy with no resection) was also predictive of a response.

PTAC recommended that temozolomide should be listed on the Pharmaceutical Schedule for the adjuvant treatment of newly diagnosed glioblastoma multiforme in combination with radiotherapy. PTAC recommended that subsidy should be targeted to this patient group possibly by means of a Special Authority. PTAC considered that patients should have a good performance status (Karnofsky score >80 or WHO score 0 or 1) at diagnosis, and preferably a resectable or partially resectable tumour. PTAC gave a high priority to this recommendation.
PTAC considered that CaTSoP should review any criteria. PTAC considered that a low priority should be given to funding under criteria that included a poor performance score (Karnofsky score <80 or WHO score 2). PTAC also recommended that approvals for funding should be restricted to the initial treatment in combination with radiotherapy followed by a maximum of six cycles of temozolomide.

Appendix: Timeline to date

- 2001: Temozolomide first submitted to the Pharmaceutical and Therapeutics Advisory Committee (PTAC), for use in recurrent glioblastomas post-radiotherapy. The evidence for its effectiveness in this setting was not strong, and PTAC and then its cancer treatments subcommittee (CaTSoP) advises PHARMAC that funding should not be made available.

- March-April 2005: Evidence supporting the use of temozolomide in conjunction with radiotherapy is published in major medical journals.

- PHARMAC asks PTAC to consider temozolomide again in light of the new evidence. This is an unusual step, as temozolomide not approved for use in this way by Medsafe, and no funding application by the supplier of temozolomide (Schering-Plough) to PHARMAC.

- 18 August 2005: PTAC considers the new phase III trial7 evidence on temozolomide. Seeks specialist advice from CaTSoP, deferring any recommendation until temozolomide approved by Medsafe and the supplier applies for funding.

- 1 September 2005: Medsafe approves temozolomide for use in conjunction with radiotherapy.

- 2 September 2005: Cancer treatments sub-committee of PTAC examines the new phase III trial7 evidence.

- 18 October 2005: PHARMAC receives new funding application for temozolomide from Schering-Plough.

- October-November 2005: PHARMAC conducts rapid economic analysis of temozolomide.

- 17 November 2005: PTAC considers the new application, making a positive recommendation (viz., recommending with high priority the listing of temozolomide for newly diagnosed glioblastoma multiforme used in combination with radiotherapy, targeted to patients with good performance status at diagnosis and preferably a fully/partially resectable tumour).

- November-December 2005: Ongoing negotiations between PHARMAC and the supplier, including sending the supplier a proposal for funding, aiming for agreement in early 2006.
PHARMAC responds on TNF inhibitors for inflammatory arthritis

There are several features in the Special Series article in October on the funding of tumour necrosis factor (TNF)-alpha receptor antagonists (TNF inhibitors) for inflammatory arthritis (http://www.nzma.org.nz/journal/118-1224/1706/) that deserve clarification.

TNF inhibitors are now funded for severe treatment-resistant rheumatoid arthritis

The PHARMAC Board decided in October 2005 to list the TNF inhibitor adalimumab on the Pharmaceutical Schedule under Special Authority for patients with severe treatment-resistant rheumatoid arthritis (RA). Implementation of this decision was subject to obtaining specific advice from the Pharmacology and Therapeutics Advisory Committee (PTAC). This advice has since been given, and publicly-subsidised adalimumab will be available from 1 January 2006 (http://www.pharmac.govt.nz/pdf/051205.pdf).

Details of eligibility criteria for adalimumab can be found in the Appendix to this letter. New Zealand’s criteria will largely align to those of Australia. PHARMAC has been running a commercial process with suppliers since May 2005, and secured a confidential agreement so that a TNF inhibitor could be listed. The PHARMAC Board resolved to list adalimumab on the basis of all of PHARMAC’s nine formal decision criteria (http://www.pharmac.govt.nz/operational_policies_and_procedures.asp). These included the high health needs and lack of other treatment for patients with severe treatment-resistant RA, the effectiveness of TNF inhibitors, and PTAC’s high-priority recommendation for funding TNF inhibitors.

Cost-effectiveness of TNF inhibitors for RA

PHARMAC stands behind its previous economic analyses and the discussion document on TNF inhibitors, which was distributed to District Health Board (DHB) hospitals as part of the Hospital Pharmaceutical Assessment Process (HPAP)—detailed later.

The initial document and economic analysis underwent a thorough review process, including internal review of the economic methodology by PHARMAC staff and external specialist rheumatology input. The draft discussion document was then circulated to DHB hospitals for comment. PHARMAC comprehensively considered the responses to this consultation before it made revisions to arrive at the final document. The key elements of all of the consultation responses were also considered by PTAC when it reviewed the economic analysis in August 2004. Issues raised, and PHARMAC’s responses to these issues, are detailed in PHARMAC’s Analysis of consultation responses to the Infliximab and Etanercept Discussion Document, which is available via the link at the corresponding position of the full text version:
The authors of the *Special Series* article discuss several aspects of the economic analysis, including the target population, the comparator and outcomes used, and what savings are included. We respond as follows:

**Target population**

The calculations of quality-adjusted life years (QALYs) gained in the economic analysis were relevant to the likely high-need high-response target population in New Zealand, and indeed relate closely to the imminent eligibility (entry and exit) criteria for adalimumab. The calculations were not based on the whole intention-to-treat population in the ATTRACT trial. Rather the analysis was based on unpublished data, sourced from the supplier, on the subgroup of patients in the trial with severe treatment-resistant disease. PHARMAC then calculated the $191,000/QALY for infliximab that was specific to this more severely-affected subgroup.

**Comparator used in analysis**

Methotrexate was the correct comparator to use in PHARMAC’s economic analyses. Patients are likely to be on a cocktail of drugs (which all differ in efficacy and side-effects), but in most cases they should be co-administered methotrexate (unless methotrexate is contraindicated or intolerable). Infliximab should be given in combination with methotrexate—and concurrent methotrexate improves the long-term effectiveness of infliximab and etanercept, and for adalimumab the best results are obtained with the concurrent methotrexate.

In addition, since methotrexate was used in both arms of the economic analysis (either alone or in combination with infliximab), the cost and benefits occurred in both arms, cancelling out each outer; this in effect was equivalent to comparing infliximab monotherapy with placebo.

PTAC, when reviewing the economic analysis, considered that although methotrexate was not the ideal comparator, it was the most appropriate comparator given currently available data. PTAC members noted that methotrexate was used as the comparator treatment in the key clinical trial on infliximab (ATTRACT), and that there were no data available comparing infliximab with prednisone or leflunomide.

Even if a more expensive agent (e.g. leflunomide) was used as a comparator, it would make little difference to the results of economic analysis—because cost-effectiveness was relatively insensitive to the costs of the comparator treatment (which PHARMAC did vary in sensitivity analysis).

**Outcomes considered in analysis**

PHARMAC’s economic analysis was based on the Health Assessment Questionnaire (HAQ) index scores. HAQ scores are considered to be sensitive measures of DMARD effectiveness and correlate with disease severity (ACR scores). HAQ scores were also a pre-specified primary endpoint of the ATTRACT trial.

The use of HAQ scores in the analysis indirectly accounted for empirical reductions in joint damage, as radiological joint destruction strongly correlates with HAQ.
Therefore, any reductions in joint erosions with TNF inhibitor treatment were reflected in the decrease in HAQ scores and hence the QALY gains measured in PHARMAC’s analysis.

The long-term benefits of reducing joint erosion were not measured in the relevant clinical trials. Hence it is difficult to conclusively predict the effects of TNF inhibitors on erosive effects in economic models, other than by using proxy but relevant measures such as HAQ scores.

**Savings beyond the health sector**

Although there may be non-health sector savings from using TNF inhibitors, there are both philosophical and pragmatic reasons for limiting analyses to health sector costs alone, as outlined by PHARMAC previously in the *Journal* and in its Prescription for Pharmacoeconomics.

**Discussion documents sent to DHB hospitals**

PHARMAC did not publish the summary discussion document on TNF inhibitors. The document was written in response to a request from DHB hospitals, and made available to all DHBs as part of the HPAP—see [http://www.pharmac.govt.nz/hospital_strategy.asp](http://www.pharmac.govt.nz/hospital_strategy.asp). These documents are circulated to DHBs as confidential documents, at the request of and agreement with the pharmaceutical industry.

The advice in the discussion document should not in itself have been a barrier to DHB hospitals’ funding of medicines such as TNF inhibitors. Cost-effectiveness is only one of a number of factors considered by DHBs when making funding decisions about such medicines.

We are interested that concerns with the methods and assumptions used by the economic analysis have been highlighted by international commentators. We would be keen to see the nature or source of these concerns, although we haven’t been able to identify them yet in the literature. Being confidential to DHBs, we are unsure how the TNF inhibitors discussion document has gained international readership—beyond overseas rheumatologists working for DHBs on fixed term contracts, whose comments were part of the responses considered by PHARMAC.

**Other inflammatory arthropathies**

PTAC in November 2004 recommended listing etanercept for ankylosing spondylitis, but with low priority. PTAC did note however that it should reconsider that priority rating once longer-term data become available. In general, applications with low-priority PTAC recommendations are treated with less urgency than higher priority recommendations.

PHARMAC has yet to receive any applications for TNF inhibitors for psoriatic arthritis or other inflammatory arthropathies.

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Conflict of interest: Scott Metcalfe is externally contracted to work with PHARMAC for public health advice. Peter Moodie, Rachel Grocott, and Tommy Wilkinson declare no conflicts.

References and endnotes:


6. This subgroup of those 87 patients in the ATTRACT trial who had severe treatment-resistant disease was restricted to patients with all of the following features: rheumatoid factor positive; had failed three or more alternative DMARDs including methotrexate; had ESR ≥28 mm/hr or CRP ≥20 mg/L (ATTRACT inclusion criteria); and had ≥15 swollen or tender joints.

7. For this subgroup, 40% of patients switched treatment after 14 weeks because they failed to meet the criteria for continuing treatment (being a 50% reduction in the number of swollen and tender joints).


10. The TEMPO trial of etanercept found that the ACR responses of patients administered etanercept and methotrexate were significantly better compared with etanercept alone and methotrexate alone. At 52 weeks, 69% of patients in the combination group achieved ACR50, compared with 43% in the methotrexate group (p=0.0091) and 48% in the etanercept group
More than a third (35%) of patients receiving combination treatment had disease remission after one year, compared with 16% of patients administered etanercept only and 13% administered methotrexate only. However, a number of patients still had active inflammation. (Klareskog L, van der Heijde D, de Jager JP, et al; TEMPO (Trial of Etanercept and Methotrexate with Radiographic Patient Outcomes) study investigators. Therapeutic effect of the combination of etanercept and methotrexate compared with each treatment alone in patients with rheumatoid arthritis: double-blind randomised controlled trial. Lancet. 2004;363:675–81.)

11. Maini et al 1998 found that patients receiving 1 mg/kg of infliximab without methotrexate became unresponsive to repeat infusions of infliximab. However those who were co-administered methotrexate appeared to benefit from a synergy between the drugs, which was observed as a prolonged duration of response. (Maini RN, Breedveld FC, Kalden JR, et al. Therapeutic efficacy of multiple intravenous infusions of anti-tumor necrosis factor alpha monoclonal antibody combined with low-dose weekly methotrexate in rheumatoid arthritis. Arthritis Rheum. 1998;41:1552–63.)


14. The HAQ measures both function and pain, by assessing patients’ ability to dress, arise, eat, walk, maintain personal hygiene, reach, and grip (degrees of difficulty) and pain (visual analogue scale) (Blumenauer B, Cranney A, Clinch J, Tugwell P. Quality of life in patients with rheumatoid arthritis: which drugs might make a difference? Pharmacoeconomics. 2003;21:927–46.).


The perspective taken by PHARMAC when conducting cost effectiveness analyses is that of the health sector. This relates to PHARMAC’s primary objective of achieving the best health outcomes possible from pharmaceutical treatment within the funding available (New Zealand Public Health and Disability Act 2000 [NZPHDA], Section 47 Objectives of Pharm). This implies that any patient benefits and/or costs that accrue beyond being either healthy or unhealthy are outside the scope of PHARMAC analysis (where “health” is defined by default in the NZPHDA as amenable to health services interventions).

This means that extra economic production stemming from an individual being healthier is outside the scope of PHARMAC’s analyses. Including indirect costs, such as loss of earnings, may prejudice decisions against issues affecting the young, elderly, and less economically productive groups. This conflicts with the public priorities as stated by the Government under the New Zealand Health Strategy (http://www.moh.govt.nz/nzhs.html).

In addition, indirect costs such as patient travelling times and productivity losses are not easily measured. There is usually little available data on these issues or how to cost them across patient sub-groups. Consequently, incorporating these into analyses would mean using significant and untestable assumptions. Given the large uncertainties involved, PHARMAC has felt it best to avoid incorporating these estimates into its base case analyses.

24. HPAD analyses are undertaken for DHB hospitals as part of the Hospital Pharmaceutical Assessment Process (HPAP). HPAP was established in 2002 as part of the National Hospital Pharmaceutical Strategy, to reduce duplication of work and increase discussion on the costs and benefits of new pharmaceuticals by distributing hospital pharmaceutical assessments nationally. These assessments are distributed to DHBs as confidential documents, which is at the request of and agreement with the pharmaceutical industry.

Further information on the purpose of HPAP and PHARMAC’s role in the distribution of discussion documents can be found on the PHARMAC website—http://www.pharmac.govt.nz/hospital_strategy.asp

25. PHARMAC receives about 30 applications for funding each year. In general, of those applications that PTAC does assign priority to, about 40% have been given high or moderate priority, 30% low priority or fund only if cost-neutral, and for 30% PTAC has recommended they be declined (applications considered by PTAC during 2004 and 2005 to date). Priority ratings are used both to inform PHARMAC on the use of analyst resources in conducting technology assessments and for PHARMAC to prioritise spending.

Appendix

Special Authority criteria for adalimumab, effective from 1 January 2006:

Special Authority for Subsidy

Initial application only from a rheumatologist. Approvals valid for 6 months for applications meeting the following criteria:

All of the following

1. Patient is an adult who has had severe and active erosive Rheumatoid Arthritis for six months duration or longer; and

2. Treatment is to be used as an adjunct to methotrexate therapy or monotherapy where use of methotrexate is limited by toxicity or intolerance; and
3 Patient has tried and not responded to at least three months of oral or parenteral methotrexate at a dose of at least 20 mg weekly or a maximum tolerated dose; and

4 Patient has tried and not responded to at least three months of oral or parenteral methotrexate in combination with at least two of the following (triple therapy): sulphasalazine, prednisone at a dose of at least 7.5 mg per day, azathioprine, intramuscular gold, or hydroxychloroquine sulphate (at maximum tolerated doses); and

5 Patient has tried and not responded to at least three months therapy at the maximum tolerated dose of either:
   5.1 Cyclosporin alone or in combination with another agent; or
   5.2 Leflunomide alone or in combination with another agent; and

6 Either
   6.1 Patient has persistent symptoms of poorly-controlled and active disease in at least 20 active, swollen, tender joints; or
   6.2 Patient has persistent symptoms of poorly-controlled and active disease in at least four active joints from the following: wrist, elbow, knee, ankle, and either shoulder or hip; and

7 Either:
   7.1 Patient has a C-reactive protein level greater than 15 mg/L measured no more than one month prior to the date of this application; or
   7.2 C-reactive protein levels not measured as patient is currently receiving prednisone therapy at a dose of greater than 5 mg per day and has done so for more than three months;

8 The patient consents to details of their treatment being held on a central registry and has signed a consent form outlining the conditions of ongoing treatment.

Renewal only from a rheumatologist or general physician on the recommendation from a rheumatologist.
Approvals valid for 6 months for applications meeting the following criteria:

9 Treatment is to be used as an adjunct to methotrexate therapy or monotherapy where use of methotrexate is limited by toxicity or intolerance; and

10 Either:
   10.1 Following 4 months initial treatment, the patient has at least a 50% decrease in active joint count from baseline and a clinically significant response to treatment in the opinion of the physician; or
   10.2 On subsequent reapplications, the patient demonstrates at least a continuing 30% improvement in active joint count from baseline and a clinically significant response to treatment in the opinion of the physician.
PHARMAC responds on treatments for pulmonary arterial hypertension

Dr Ken Whyte recently wrote about the funding of medicines for rare life-threatening diseases (high-cost treatments for ‘orphan diseases’), using the example of bosentan for pulmonary arterial hypertension (PAH) (http://www.nzma.org.nz/journal/118-1226/1759). He raises difficult issues that need discussion.

Treatments for PAH

Subsidised access to high-cost treatments for PAH (such as iloprost, bosentan and high-dose sildenafil) had since 2001 been initially provided under the Community Exceptional Circumstances (CEC) scheme (http://www.pharmac.govt.nz/exceptional_circumstances.asp). Over that time applications were relatively rare, no more than a few per year.

However, the CEC scheme requires rarity, i.e. that the prevalence of a condition is limited to no more than 10 cases nationally. During 2004 it became apparent that the rarity limit would be significantly exceeded (28 patients are now funded for high-cost PAH treatments, many on sildenafil). Under the limits of the CEC scheme, PHARMAC was no longer able to approve applications for high-cost PAH treatments under CEC. PHARMAC therefore moved to find a permanent solution to the funding of PAH treatments.

The Pharmacology and Therapeutics Advisory Committee (PTAC) has noted a lack of information and a number of dilemmas with the management of PAH, and made a high priority recommendation that funding issues be resolved as soon as possible. PHARMAC is actively working on this. The relevant portions of the minutes of the two relevant PTAC meetings can be found in the Appendix to this letter.

Until a permanent solution is found, applications for new patients can still be made for subsidised treatment through the Hospital Exceptional Circumstances (HEC) scheme. Unlike CEC, HEC does not have a rarity criterion, but requires that treatment is cost-saving to the District Health Board (DHB). Since mid-2004, 19 patients have received approval for the use of high-cost PAH treatments under the HEC scheme, with more applications being received and approved every week.

Treatments for PAH are expensive, and annual treatment costs for each patient vary substantially between medications, with $90-180,000 for iloprost, $56,000 for bosentan and $20-30,000 for sildenafil. Current DHB expenditure on high-cost PAH treatments is some $600-700,000 per year, and the number of patients seeking treatment continues to grow.

The evidence for bosentan, iloprost and sildenafil also continues to grow. The recently published SUPER trial, referred to by Dr Whyte, and the SERAPH study indicate that sildenafil may be as effective as the more expensive bosentan. Neither high-dose sildenafil nor nebulised iloprost is registered in New Zealand for use in PAH.
In response to Dr Whyte’s comments about the cost-effectiveness of PAH treatments, we find it difficult to comment on the one published economic analysis on PAH (Highland et al 2003) that we and he can locate, which did have important limitations. Economic analyses for individual PHARMAC CEC funding decisions have indicated that all three treatment options may be cost-effective as a bridge to transplantation, but perhaps the only cost-effective maintenance treatment for patients ineligible for transplant is sildenafil. Funding a medicine in Australia does not necessarily mean convincing cost-effectiveness—for instance, Australia continued to fund COX-2 inhibitors despite dubious cost-effectiveness.

**Prioritisation of very high cost medicines**

In general terms, PHARMAC’s prioritisation process tries to allocate scarce resources in a fair way. There are very expensive treatments that may offer significant benefits to a small number of people. Such very expensive treatments have to compete for limited funds with less expensive medicines that treat large numbers and achieve greater population health gains for the same total costs. The growing number of costly new treatments makes such decisions both more common and more difficult.

PHARMAC is currently reviewing its decision-making process for high-cost medicines—driven in part by having to turn down treatments for small numbers of people who then miss out because there are no alternative treatments. This is where, even after assuming 100% effectiveness with large clinical benefits, the cost of these medicines is very high (at times $250,000 per patient year or more). Funding them would deny treating too many people with other diseases.

PHARMAC’s Board intends to consider the outcome of this review process next year; prior to that, any proposed changes to our decision-making processes would undergo public consultation.

That said, PHARMAC is actively working to permanently solve the funding of PAH treatments, independent of the high-cost review process.

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**Conflict of interest:** Scott Metcalfe is externally contracted to work with PHARMAC for public health advice. Dilky Rasiah and Sean Dougherty declare no conflicts.
References and endnotes:


2. As of 5 December 2005, 28 patients were funded for PAH treatment through Exceptional Circumstances—five were using iloprost, two bosentan, 20 sildenafil, and one vardenafil. Eleven of the patients had been approved sildenafil after being declined iloprost or bosentan. A further five patients had been declined any of the PAH treatments. In addition, many patients will receive iloprost for acute treatment as inpatients. Annual expenditure on high-cost PAH treatments is currently around $600-700,000, with a roughly 20:80 split between community and hospitals. Most of the expenditure is on iloprost, either as acute in-hospital use or through EC (where the injection is nebulised).


5. SERAPH is a head-to-head trial comparing sildenafil with bosentan that suggests that sildenafil is not demonstrably inferior to bosentan—although the primary measure was debatable and there was one death in the sildenafil group. High-dose sildenafil is not yet registered in New Zealand for PAH, but has recently been registered in the US for ‘the treatment of pulmonary arterial hypertension (WHO group 1) to improve exercise ability’.


7. TRIP search http://www.tripdatabase.com/ 9 December 2005 keywords pulmonary hypertension; PubMed searches 9 December 2005 keywords pulmonary hypertension AND (cost effectiveness OR quality adjusted life years OR cost utility or economic)

8. The Highland et al 2003 economic analysis compared bosentan with treprostinil and epoprostenol—not used in New Zealand—in a South Carolina cost context. They did not examine sildenafil or nebulised iloprost, and relied on indirect comparisons; there were no head-to-head comparisons, and the trial data available were sparse, compounded by quite different eligibility criteria. Useful comment on this economic analysis can be found on the NHS CRD website at http://nhscrdsyork.ac.uk/online/nhsseed/20040088.htm


Appendix: PTAC minutes relating to PAH

Relevant record from the Pharmacology and Therapeutics Advisory Committee meeting 19 August 2004

“17 Management of Pulmonary Hypertension

The Committee reviewed the literature presented by PHARMAC staff on pulmonary hypertension (PAH), which included a number of articles sourced by the discussion leader. Members agreed, in general terms, with the management course for pulmonary hypertension outlined in the PTAC paper submitted by PHARMAC staff.

The Committee discussed the strength and quality of the evidence presented for iloprost, bosentan and high-dose sildenafil for the treatment of PAH. The Committee noted that all the randomised controlled trials involving either iloprost, beraprost, bosentan or sildenafil for use in PAH were relatively small, of short duration and confined to adults. Members noted that the studies illustrated varying degrees of short-term (up to 6 month) improvements in six-minute walking test results or exercise times. Members were advised that there were two recent studies reporting long-term efficacy and survival rates with bosentan, but they did not have the chance to see the source publications to evaluate the robustness and relevance of the data. They noted that none of the medications iloprost, beraprost, sildenafil or bosentan were without adverse effects.

The Committee noted that only iloprost has a registered indication for PAH in New Zealand. Members also noted that there are other therapies being trialled overseas for PAH which include sitaxsentan and ambrisentan (selective endothelin A antagonists) and combination therapy. Members also noted the use of inhaled nitric oxide and arginine, but there have been no RCTs of their use in PAH.

The Committee considered that the patients who benefit the most from iloprost, sildenafil or bosentan are those with primary pulmonary hypertension, and considered that the evidence for secondary PAH was not as clear. Members noted that there were no documented differences in incidence and prevalence rates of PAH between Maori and Pacific people and others, and considered that overall numbers of patients were too low to detect statistically meaningful differences.

The Committee noted that Australia’s Pharmaceutical Benefits Scheme funds bosentan with restrictions, and that, if bosentan were funded on the Pharmaceutical Schedule, then similar or even stricter criteria (for instance only for primary PAH or only as a bridge to transplantation) could be used for any or all of the newer PAH treatments.

Members noted that the use of these treatments would result in additional non-medication expenditure to the health sector, as frequent investigations would need to be undertaken to monitor effects of treatment.

They also noted that the economic analyses, and the rapid cost-utility analyses (CUAs) and board papers that have been completed to assess individual patients for Community Exceptional Circumstances (CEC) funding, highlight the difficulties inherent in making decisions regarding PAH funding. These include:

- The treatments are very expensive:
A number of patients are already funded via CEC;
Patient numbers now exceed the CEC rarity criterion;
Given the cost, cost-effectiveness is likely to be very poor;
Current treatment options suggest improvement in exercise tolerance and haemodynamics, but there is no evidence to date for any improvement in survival;
Given the lack of systematic survival data, significant assumptions and extrapolations are required in any CUA analyses.

The Committee noted that, often when there is very little long-term or endpoint data, members would not consider the funding issue further until such data had been provided. However, for the following reasons, they considered that this situation needed further consideration at this time:

- Difficulty in separating the pharmaceutical treatments for PAH from heart/lung transplantation aspects of therapy that are already happening;
- CEC being asked to fund patients already started on treatment, and the ethical issues faced in withdrawing government funding in this situation;
- CEC already funding a number of patients but rarity criterion having now been exceeded;
- The significant number of case studies indicating increased survival.

The Committee considered that, as new patients no longer meet the CEC criteria, it would be unacceptable to recommend an option that did not consider applications for new patients. Similarly it would not be acceptable to defer a decision until endpoint data and registration were available.

PHARMAC staff presented various funding options to the Committee:

- the status quo;
- disease state management panel funding; and
- HEC funding.

With potentially 120-200 patients with severe PAH in New Zealand, members did not consider it was an option to have future funding via CEC from a budgetary perspective, because of delays (turnaround time) and breaching of the CEC rarity criterion. They considered that the EC panel, with the number of applications it had now considered, did have the required expertise (with expert opinion sought if required) to consider PAH treatment applications.

The Committee noted that HEC management was consistent with an approach that PAH treatment is part of an overall treatment package that may or may not include transplantation. However, given the small size of some DHB budgets (from where HEC funding would come)—and hence the risk that patients domiciled in smaller DHBs may not even be referred to HEC for consideration of funding—HEC management may not be a long-term option. However, the Committee also discussed the possibility of the lead DHB, i.e. the transplant assessment units, funding the treatment, such that HEC could be a longer-term option. There was also some discussion about the possibility of a national protocol (or similar) for these products,
formulated by experts in the field, which HEC could administer in collaboration with DHBs.

The Committee considered that the disease state management panel option might be the best option long-term. Members envisaged that such a panel would manage the patient throughout the whole process, from consideration of conventional treatments and suitability for transplantation through to use of newer treatments. The Committee had concerns about budget overruns, and that a panel consisting solely of experts in the area would cause potential conflicts of interest as they would most likely also be the clinicians managing the patients. They recommended that any such panel would need to operate within a set budget and would consist of experts in the area together with PTAC and/or EC panel members. The Committee recommended that such a panel should act within strict protocols outlining how long treatments would be tried before being abandoned. Members noted that such an overview approach is not possible currently under CEC for the reasons outlined above. They noted that, within the protocols, the PAH panel would have the flexibility to manage the patients, so that any required trade-offs could and would need to be made in order for the panel to stay within budget.

The Committee **recommended** that applications for PAH treatment be considered via HEC in the interim, while PHARMAC staff explore the possibility of other options, including funding treatments via a disease state management panel. PTAC gave this recommendation a high priority, and asked that the funding issue be resolved as soon as practicable.”

**Relevant record from the Pharmacology and Therapeutics Advisory Committee meeting 17 February 2005**

“Two applications have been received for consideration:

1) Bosentan (Tracleer), submitted by Actelion Pharmaceuticals Australia and Asia Pacific (January 2005)

2) Iloprost (Ilomedin), submitted by Schering NZ Limited (October 2004)

**14. Bosentan (Tracleer)**

The Committee noted that the PHARMAC Board had considered a paper on the management of PAH at its 15 December 2004 meeting and had directed PHARMAC staff to seek Pharmaceutical Schedule applications.

The Committee noted that bosentan and iloprost are currently funded via the Hospital Exceptional Circumstances (HEC) scheme, as the rarity threshold for Community Exceptional Circumstances (CEC) has been exceeded.

The Committee noted that there is an estimated prevalence of 120-200 patients with PAH in NZ (using UK prevalence data), of whom only 10-25% would be likely to respond to calcium channel blockers.

The Committee noted that bosentan has received provisional registration with Medsafe in December 2004, pending further information from the company.

The Committee noted that apart from the Channick et al (2001) and Rubin et al (2002) randomised controlled trials, the only other evidence of note were open-label
extension studies by Sitbon et al (2003) and Roux et al (2001), which looked at the long term safety and efficacy of bosentan, and an open-label longitudinal study by Barst et al (2003) which looked at the safety and efficacy of the drug in paediatric patients with PAH.

The Committee considered that bosentan demonstrated subjective and objective improvements, especially in terms of exercise tolerance, haemodynamic parameters and New York Heart Association (NYHA) functional class. It also considered that outcomes were likely to be better in patients with primary PAH than in those with PAH secondary to connective tissue/collagen vascular disease, although this had not been shown statistically. Members considered that bosentan did not demonstrate clear end point advantages over other unlisted treatments such as nebulised iloprost, sildenafil, or sitaxsentan, although they noted that comparative data was limited. The Committee considered that bosentan represented an advance on currently funded treatments on the Pharmaceutical Schedule such as warfarin, diuretics, and calcium channel blockers. The drug also has an advantage in being orally administered.

The Committee considered that there were significant safety concerns regarding bosentan since the drug is associated with such risks as hepatotoxicity, (effects on CYP450), and potential teratogenicity.

The Committee noted that Actelion’s cost projections may be underestimated because the company used US prevalence figures of 12.5 cases per million, whereas UK data suggests a prevalence of 30 to 50 cases per million.

The Committee considered that, in the absence of long-term observational studies, head-to-head studies, and studies using treatments in combination (eg. nebulised iloprost and sildenafil) that address efficacy, survival, safety, quality of life and costs, the approach to managing PAH would largely depend on regional experience, funding constraints, administrative regulations, clinical context and patient preference. The Committee noted that limited randomised controlled trial (RCT) data suggest that bosentan, nebulised iloprost and sildenafil have similar effects.

The Committee recommended that the option of a PAH treatment panel be pursued by PHARMAC. Based on the evidence so far supplied on bosentan, the Committee considered that the treatment could be funded through such a mechanism. Additionally, iloprost, sildenafil and other developing treatments for PAH could also be considered via this mechanism. It noted that the panel would need to operate under strict entry and exit criteria and a budgetary cap. The Committee noted that access to funding for PAH treatments, for those in whom it is appropriate, may currently be sought via Hospital Exceptional Circumstances.

On the basis of clinical evidence, the Committee recommended the listing of this treatment on the Pharmaceutical Schedule with a low priority, as the Committee was of the opinion that additional evidence on the use of this treatment in PAH, as outlined above, was required.

However, the Committee noted that there is a significant unmet need in these patients due to the severe nature of this disease, and that only a small proportion of patients can be successfully treated using standard treatments. Therefore, the Committee considered a high priority should be given to finding a method of funding treatments for PAH.
The relevant decision criteria are: (i) the health needs of all eligible people within New Zealand; (iii) the availability and suitability of existing medicines, therapeutic medical devices and related products and related things; (v) the cost-effectiveness of meeting health needs by funding pharmaceuticals rather than using other publicly funded health and disability support services; and (vi) the budgetary impact (in terms of the Pharmaceutical budget and the Government’s overall health budget) of any changes to the Pharmaceutical Schedule.

15. Iloprost (Ilomedin)

The Committee noted that the PHARMAC Board had considered a paper on the management of PAH at its 15 December 2004 meeting and had directed PHARMAC staff to seek Pharmaceutical Schedule applications.

The Committee noted that bosentan and iloprost are currently funded via the Hospital Exceptional Circumstances (HEC) scheme, as the rarity threshold for Community Exceptional Circumstances (CEC) has been exceeded.

The Committee noted that there is an estimated prevalence of 120-200 patients with PAH in NZ (using UK prevalence data), of whom only 10-25% would be likely to respond to calcium channel blockers.

The Committee noted that only iloprost IV is registered in New Zealand. This means that the use of the IV solution in a nebuliser to deliver iloprost in an inhaled form is an unregistered use.

The Committee considered that the evidence for nebulised iloprost was weak, and was no better or worse than for other treatment options in PAH. However, members considered that seriously ill patients (NYHA class 4) should probably be treated first with IV prostacyclin or nebulised iloprost or maybe sildenafil, as bosentan, beraprost and subcutaneous prostacyclins may not provide a significant clinical response for several weeks.

The Committee noted the Ghofrani et al (2002) study, looking at acute haemodynamic response, showed the combination of nebulised iloprost and sildenafil 50 mg could have synergistic effects.

The Committee considered that iloprost demonstrated subjective and objective improvements, especially in terms of exercise tolerance, haemodynamic parameters, and NYHA functional class. Members considered that nebulised iloprost did not demonstrate clear end point advantages over other unlisted treatments like bosentan, sildenafil, or sitaxsentan, although they noted that comparative data was limited. The Committee considered that iloprost represented an advance on currently funded treatments on the Pharmaceutical Schedule but noted that the frequency of nebulisations (6-9 times a day) may be inconvenient and may affect patient preference for treatment. The Committee also considered that iloprost has a few minor adverse effects but is generally well tolerated.

The Committee noted that Schering has suggested establishing a fund of $500,000/year for the treatment of PAH, to be managed by a panel of 2-3 experts in the field instead of a listing under Special Authority.

The Committee considered that, in the absence of long-term observational studies, head-to-head studies, and studies using treatments in combination (e.g. nebulised...
iloprost and sildenafil) that address efficacy, survival, safety, quality of life and costs, the approach to managing PAH would largely depend on regional experience, funding constraints, administrative regulations, clinical context and patient preference. The Committee noted that limited randomised controlled trial (RCT) data suggest that bosentan, nebulised iloprost and sildenafil have similar effects.

The Committee **recommended** that the option of a PAH treatment panel be pursued by PHARMAC. Based on the evidence so far supplied on iloprost, the Committee considered that the treatment could be funded through such a mechanism. Additionally, bosentan, sildenafil and other developing treatments for PAH could also be considered via this mechanism. It noted that the panel would need to operate under strict entry and exit criteria and a budgetary cap. The Committee noted that access to funding for PAH treatments, for those in whom it is appropriate, may currently be sought via Hospital Exceptional Circumstances.

On the basis of clinical evidence, the Committee **recommended** the listing of this treatment on the Pharmaceutical Schedule with a low priority, as the Committee was of the opinion that additional evidence on the use of this treatment in PAH, as outlined above, was required.

However, the Committee noted that there is a significant unmet need in these patients due to the severe nature of this disease, and that only a small proportion of patients can be successfully treated using standard treatments. Therefore, the Committee considered a high priority should be given to finding a method of funding treatments for PAH.

The relevant decision criteria are: (i) the health needs of all eligible people within New Zealand; (iii) the availability and suitability of existing medicines, therapeutic medical devices and related products and related things; (v) the cost-effectiveness of meeting health needs by funding pharmaceuticals rather than using other publicly funded health and disability support services; and (vi) the budgetary impact (in terms of the Pharmaceutical budget and the Government’s overall health budget) of any changes to the Pharmaceutical Schedule.”
Inappropriate Care – Professional Misconduct (02/96C)

Charge:

The Complaints Assessment Committee pursuant to s93(1)(b) of the Act charged that Robert William Robertson in the course of his management and treatment of his patient:

1. Failed to appropriately follow up on a cytology report dated 10 June 1994 in relation to the patient’s left breast which report stated that an “in-situ ductal lesion cannot be excluded with certainty”; and

2. In the period prior to 1 October 1998 failed to take appropriate clinical steps to diagnose and/or failed to adequately assess, pre-operatively or otherwise, an identified solid lesion in the patient’s left breast; and

3. Failed to provide the patient with an acceptable standard of care:
   1. Prior to performing an excision biopsy on the patient on 5 October 1998 failed to carry out an appropriate breast examination in the absence of a diagnosis and/or despite not having seen her in a consultation since 23 July 1998; and/or

2. On 9 October 1998 following diagnosis of invasive carcinoma offered the patient a wide local excision as treatment for her breast cancer when he had not taken any clinical steps to ascertain the presence or absence of further lesions in her left breast; and/or

3. Failed to take notes and/or adequate notes in the patient’s medical records of his consultations with her:
   i. In 1994 when he failed to document his actions after receiving the abnormal cytology report;
   ii. On or about 13 February 1997 and 7 August 1997 when she presented to him with a specific palpable lesion in her left breast; and
   iii. Between early 1999 and mid 2000 on the occasions he met with her after hours in his practice rooms at Surgical Associates.

4. Failed to provide sufficient information to the patient as was necessary for her to make informed decisions:
   i. Failed to tell the patient about a cytology report dated 10 June 1994 that stated that an “in-situ ductal lesion cannot be excluded with certainty”; and/or
   ii. Failed to provide the patient with accurate information about standard diagnostic tests for solid breast lumps.

4. Failed to act at all times in the best interests of his patient:
   1. Provided information to, and about, his patient which was inaccurate, misleading or wrong:
In October 1998 stated in written communications with two doctors, and in written reports on file, that diagnostic tests had been undertaken with respect to an identified solid lesion in the patient’s left breast, and that the results of these diagnostic tests had been reported as negative for carcinoma, when no such tests had been undertaken and between October 1998 and July 2000, repeated these claims in verbal communication with the patient;

In July 1998 stated verbally to the patient, and again in October 1998 to the patient and her partner that she had refused a procedure, being the removal of her identified solid left breast lesion, when he had not offered this procedure to her, but rather had counselled her against it;

Stated to the patient that early detection of breast cancer was ineffective and that his failure to remove the patient’s breast lump sooner would therefore not have affected her prognosis;

Between April 1999 and July 2000, repeatedly denied to the patient any possible relevance of the cytology report dated 10 June 1994;

In a letter dated 30 October 2000 to ACC, advised that because of the cytology report dated 10 June 1994, the patient had a further ultrasound in November 1994 which is not correct.

2. In the period early 1999 to mid 2000 failed to treat his patient in a professional, honest and respectful manner:

Invited her to meet him outside of his practice and despite her refusal, continued to make such invitations;

Encouraged ongoing contact with him when she was questioning the effect of this on her mental health;

Continued to encourage her to develop an attachment to him;

Suggested her memories of events were mistaken, and that she was in danger of losing her mind;

Made disparaging comments about her expressions of distress and her desire to live;

Failed to recommend her to seek assistance for her disclosed symptoms of emotional distress;

Attempted to discourage her from taking complaint action against him by one or more of the following:

a. failing to provide her with information about avenues for complaint action;

b. repeatedly pointing out how a complaint would impact on his personal and professional life;
c. stating she did not care for him sufficiently;

d. threatening to be dishonest if she should take complaint action in order that she should fail to achieve a result;

5. On one occasion between mid-2000 and early-2001 and again in mid-2001, procured the re-reading of slides of a substance that was taken from the patient’s left breast on 31 May 1994, without her consent.

The conduct alleged in Particulars 1, 2, 3, 4 and 5 either separately or cumulatively amount to professional misconduct.

Background:

In December 1991, the patient was referred by her general practitioner to Dr Robertson for an opinion and help in further management in respect of thickening and induration in the right breast and cystic like swelling in the left breast along with thickening and induration. She was referred as a result of her strong family history, cited as her mother having had breast cancer and dying at the age of 45 years.

From that initial appointment the patient attended at intervals of at least six months. In some instances additional referrals were made outside the normal six month follow-up period. These appointments were made when the patient was concerned about specific lumps and sought the assistance of her GP for an additional referral.

From the clinical notes, it appears that on a number of occasions notes were made regarding tenderness or swelling in the breasts and references to fine needle aspiration. The patient’s weight was taken on each occasion and on nine occasions that is the only information contained in the clinical notes. The reporting letters to the patient’s GP contain more information but were still reasonably brief.

During the period from December 1991 until the patient was advised that she had invasive cancer and DCIS in October 1998, she had attended 15 appointments at the Hospital and had had a number of routine mammograms and ultrasounds, although none had been sought as part of Dr Robertson’s management after November 1996.

On 31 May 1994 the patient saw Dr Robertson following a routine mammogram that had been done on 23 May 1994. The cytology report following that consultation is dated 10 June 1994 and stated:

“.... Although probably benign, the possibility that these clusters represent an in-situ ductal lesion cannot be excluded with certainty.”
From the hospital records it appeared that upon receipt of the report Dr Robertson wrote “for biopsy”. This was then crossed out and replaced with the words “file see again at OPD.”

The patient had another ultrasound done in November 1994 and that was discussed with her on 30 November 1994. There was no discussion about the 10 June 1994 report. At that consultation the patient was advised that the lump she had been concerned about had been a fibroadenoma.

In December 1996 the patient became aware of a small palpable solid lump in the upper outer quadrant in her left breast at 2 o’clock. Her GP referred her to another specialist, Dr E who aspirated three cysts and in respect of the lump identified by the patient stated:

“A solid nodule located laterally at 2 o’clock was also sampled yielding cohesive groups of ductal epithelial cells, bare stromal nuclei and a few connective tissue fragments. The appearance is of a BENIGN LESION.”

That report was to be copied to Dr Robertson although it was unclear at what time it came into his possession. The patient however was clear that she referred to that report at her next consultation with Dr Robertson on 13 February 1997.

From December 1997 to early 1998 the patient and her partner were overseas but were both becoming increasingly concerned about a lump in the patient’s left breast.

On 19 March 1998 the patient returned to her GP and asked for a referral to Dr Robertson because of the lump. She was seen by Dr Robertson on 23 April 1998 and in his reporting letter he stated that she had been worried about a larger lump in the left breast which had been uncomfortable and had increased in size recently.

On 23 July 1998 the patient saw Dr Robertson again and three cysts were aspirated. There was no testing of the solid lump in the left breast and Dr Robertson described it in his letter as follows:

“The solid lump remains present in the left upper outer quadrant and does not seem to have changed but it is certainly more obvious when she is lying on her side and she is more aware of it. I think it would be better to have this removed, .....

Arrangements were made for surgery to take place on 11 August 1998 but the patient was found to be pregnant and the biopsy surgery was postponed. The pregnancy was ectopic and once the patient had recovered sufficiently the surgery was rescheduled and took place on 5 October 1998.
Two days after the surgery Dr Robertson advised the patient that she had invasive cancer and DCIS. In his reporting letter to her GP following that meeting, Dr Robertson stated:

“It is somewhat disappointing that this has proved to be a cancer as originally we had considered this a fibroadenoma based on the cytology from earlier in the year.”

On 21 October Dr Robertson performed a left mastectomy on the patient. Following that surgery at a further consultation there was discussion between the patient, her partner and Dr Robertson surrounding the issue as to why the “lesion” had not been more definitively diagnosed. On 14 April 1999 the patient underwent a second prophylactic mastectomy of the right breast. On the day following surgery Dr Robertson visited the patient and the nurse noted in the hospital records that they had a “long chat.”

Between 20 April 1999 and 28 March 2000 Dr Robertson and the patient met six times at his rooms. The meetings were scheduled for 6.00 pm but did not generally begin until around 6.30 pm and each meeting was approximately 90 minutes long. At the meetings the patient and Dr Robertson discussed what had occurred in relation to her treatment. While the evidence as to the specifics of these meetings varied, the Tribunal was satisfied that these meetings were a means of explaining or understanding the sequence of events that had resulted in the patient being advised that she had invasive cancer in October 1998 and the resultant double mastectomy. There were also a number of telephone calls between Dr Robertson and the patient during October/November 1999.

The patient had obtained a copy of her file just three days after the first meeting and what she found on the file gave her cause for concern. It gave rise to a number of questions about her treatment that she wished Dr Robertson to answer.

Subsequent to the lodging of the complaint re-readings of the slide that was the subject of the report of 10 June 1994 were undertaken. It is accepted by both the CAC and Dr Robertson that those re-readings were done without the patient’s consent.

Finding:

The Tribunal found Dr Robertson guilty of professional misconduct.

The Tribunal considered the patient’s recollection of events was credible and in the event of conflict her evidence was preferred over Dr Robertson’s sometimes incomplete recollection.
In relation to particular 1 the Tribunal, by a majority, considered that Dr Robertson did appropriately follow-up on the cytology dated 10 June 1994, and therefore particular 1 was not established.

The Tribunal was satisfied particulars 2 to 5 inclusive of the charge were established.

Much of the evidence for Dr Robertson centred on the cytology report of 10 June 1994 and whether or not the report was justified in stating “an in-situ ductal lesion cannot be excluded with certainty”. This had resulted in the re-reading of the slides. The Tribunal, however, considered that the significance of the report was that there had been no mention of it and that the patient was in fact not aware of it until she obtained her medical records in 1999.

The issue of whether the lump was detectable in 1994 or in 1996 could not be answered definitively. However, the Tribunal was of the view that there did not appear to have been any management plan in place and considered it difficult to understand how there could be any effective management with the paucity of notes. The Tribunal considered that a lack of diagrams showing where cysts were located and aspirated and where lumps had been identified would have made any ongoing management very difficult. The Tribunal also considered it significant that the last ultrasound was done in November 1994 and the last mammogram was done in November 1996.

The Tribunal accepted that from the end of 1996 through to 1998 concern was being expressed by the patient about a lump and the Tribunal accepted at the consultation on 13 February 1997 the patient referred to the cytology report undertaken in December 1996. The Tribunal was concerned some follow-up or attempt to locate that report or to undertake any further tests in respect of the lump was not undertaken.

The matter of the six meetings held between the patient and Dr Robertson also raised concern for the Tribunal. It was unclear on what basis Dr Robertson entered these meetings as he was of the view that they were not in the nature of doctor/patient meetings and yet he was clear that he had been treating the patient up until July 1999. These meetings and the length of them confirmed for the Tribunal the fact that the patient was articulating her concerns and her issues and that Dr Robertson was attempting to appease those concerns and to avoid the possibility of a complaint being laid.

The Tribunal was satisfied that the management and treatment of the patient by Dr Robertson departed from accepted standards to a point of indifference on a number of counts. It considered that, as the doctor primarily responsible for the management and treatment of the patient, Dr Robertson did not discharge that responsibility in a manner that would be expected of a surgeon of his experience and expertise.
Penalty:

The Tribunal ordered Dr Robertson be censured, pay a fine of $10,000 and pay 40% of the costs in respect of the hearing.

It further ordered a notice of the hearing be published in the New Zealand Medical Journal.

Appeal:

Counsel for Dr Robertson appealed the substantive and the penalty Decisions to the District Court. The District Court upheld the Tribunal's findings of professional misconduct, but only in the following respects:

- Particular 2 which amounted to professional misconduct.
- Particulars 4.2.i (in part), 4.2.ii (in part) and 4.2.vii.b which cumulatively amounted to professional misconduct.
- Particular 2, particular 4.1.iii and particulars 4.2.i (in part), 4.2.ii (in part) and 4.2.vii.b which cumulatively amounted to professional misconduct.

The Court reduced the fine to $5,000.00 and granted leave to Counsel for Dr Robertson to make further submissions on the subject of costs in respect of the Tribunal proceedings.

The CAC cross appealed and submitted the Tribunal should have imposed conditions on Dr Robertson's practice. The Court dismissed the appeal filed on behalf of the CAC. The Court denied Dr Robertson permanent name suppression.

(R W Robertson v CAC, (District Court Christchurch, CIV-2004-009-1784, 28 November 2005, Moran J)).

The full decisions relating to the case can be found on the Tribunal website at www.mpdt.org.nz Reference No: 02/96C.
Paul Fogarty

Blenheim doctor Paul Fogarty helped bring thousands of Marlburians into the world. Dr Fogarty, 99, passed away at Bethsaida Retirement Village in Blenheim after outliving his wife, siblings and many of his friends.

He came to Blenheim in 1934, and as a respected surgeon and doctor, touched the lives of many in what was then a small town.

He played a prominent role at Holmdale Maternity Hospital, which brought more than a generation of Marlburians screaming and crying into the world, before maternity services were moved to Wairau Hospital in December, 1987.

Marlborough’s first maternity home, Holmdale was established in Litchfield St in 1918. The ante-natal unit, which was used by the hospital midwives for ante-natal classes and to book appointments, was added to the site in 1958.

In an interview in 2002 Dr Fogarty recalled fond memories of the thousands of babies he delivered at Holmdale in his 35 years on the job. “It was a good place. I had a lot of fun delivering all those babies,” he said.

Paul Fogarty was born in Greymouth, but when his father realised his son was academically gifted, he sent him to Sacred Heart boarding school in Auckland. There he enjoyed everything but the food, said his youngest son Christopher Fogarty, 62. “He reckoned it was absolutely awful.”

Dr Fogarty was dux at Sacred Heart before moving to Otago to study medicine. He went on to study in Edinburgh, becoming a Fellow of the Royal College of Surgeons.

His wife, who he met in Wellington, was a pianist with whom he shared a love of gardening and classical music. They had a successful marriage for 65 years, Mr Fogarty said. “They were very strong people and uncompromising in their principals—strong Catholics, similar interests and they just got on really well together.”

In 1934 the couple moved to Blenheim and never left. “He often used to say when we’d go for drives, ‘I never regretted coming to Marlborough.’ He really loved the place.”

He enjoyed the climate, golf, and the water and was a regular at the Blenheim Aquatic Centre, where early morning patrons remember him arriving in pyjamas and dressing gown for his daily swim, a routine he followed until he was 95. Indeed, as shown in the photo, regulars at Blenheim’s Aquatic Centre had a cake ready to celebrate Dr
Paul Fogarty’s 95th birthday when he arrived for his regular early morning swim on August 28, 2001.

Coming from Irish stock, Dr Fogarty enjoyed people, his son said. “He liked to party, he was pretty sociable. He was always very unassuming and friendly with a large range of people.”

During World War 2 Dr Fogarty was Blenheim’s sole surgeon and was sometimes called out three or four times a night. “He got a lot of satisfaction from his work as a surgeon.”

His father was a “straight shooter,” but never got off-side with anyone or upset people with his manner. And as a father he was stern, caring and inspiring, said Mr Fogarty. Dr Fogarty had four children, 13 grandchildren and 12 great-grandchildren, several of whom have entered the medical profession.

Consultant physician John Hedley said when he started working at Wairau Hospital Dr Fogarty had already retired and was only seeing a few patients, who were old friends. He treated many of Dr Fogarty’s patients, who, along with hospital staff, remembered him fondly. It is the patients and the hospital records which show the amount of great work Dr Fogarty did, he said.

“He was a grand man—a wonderful asset to Marlborough. He had a wonderful sense of humour. A lovely man, a person I admired and Marlborough was lucky to be served by him. It has been a wonderful thing he made it to 99.”

This obituary entitled Doctor gave long service originally appeared in Marlborough Express newspaper (Blenheim) on 21 November 2005 and was written by Sarah McDougall. We are also grateful to Laura Basham and Nicole Chauval of the Marlborough Express.
Graham Lelliott (Nobby) Clark

GP, mountaineer, pigeon breeder and author, and local historian

In the age when family doctors were on call 24 hours a day, “Nobby” Clark was the GP par excellence. The Christchurch doctor took surgery in his own home and made house calls at all hours for nearly 50 years, until retiring at 82.

Daughters Jenny and Nicky remember their father coming in from surgery each evening exactly at 6pm for dinner, then going back at 6.30.

A medical officer in World War 2, Clark somehow found time also for mountaineering and tramping, breeding and writing about pigeons, studying and writing local history, dabbling in painting and investments, collecting art works, playing squash and tennis, reading, gardening and appreciating music.

He died in Christchurch recently aged 91. He lived most of his life almost opposite Calvary (later Southern Cross) Hospital in Bealey Avenue and close to Christchurch Women's Hospital. There, his father, a self-made man who had emigrated from Britain and set up several wool scours in the South Island, had settled on a large section. Clark later built a Heathcote Helmore-designed house for himself and family beside the old home.

While being close to hospitals had advantages, it also led to many late-night rushes to the wards. His daughters remember the night a woman arrived with severe stomach pains. Clark diagnosed advanced pregnancy and rushed her through a neighbour's back garden to the women's hospital.

Educated at Dunelm Preparatory School and Christ's College, Clark graduated with a BSc from Canterbury University in 1935. He did medical studies at Otago University and, after graduating in 1941, became a house surgeon at Christchurch Hospital.

Clark married Erica Macfarlane, a nurse at the hospital, shortly before his conscription into the army in 1942. He was commissioned as a Lieutenant in the NZ Medical Corps and served in the Pacific. He was promoted to Captain in 1943.

An interest in respiratory disorders led him to apply for positions at the Pukeora Sanitorium in Hawkes Bay, when Tb was prevalent. He served there as assistant medical superintendent from 1945 to 1947. His research and work in respiratory health, and the thesis he wrote, gained him his MD in 1948.

He then took his family to England, where he did further studies in 1949-1950, with a view to specialising. However, he decided general practice was for him and returned...
to Christchurch. As a relic of his interests, he bought an X-ray machine and had to enlarge the house to accommodate it.

Blissful school holidays spent with friends at Tasman Downs station, near Mount Cook, sparked Clark's interest in mountaineering. He worked as a guide at Franz Josef Glacier during university holidays.

He climbed Mount Cook in 1935 and was part of a NZ Alpine Club expedition about to set off for the Himalayas to climb the world's third highest mountain, Kangchenjunga, when war broke out. Clark had done detailed research on the physiological effects of high altitude in preparation for the climb.

He continued tramping after the war and, at 65, led a party over the demanding Copland Pass in the Southern Alps. He bought a garage at Arthurs Pass and converted it into a family bach.

The kind of research Clark did for the Himalayas expedition was typical of his thoroughness and attention to detail, his daughters say. He brought this approach to pigeon fancying, where he used the study of genetics to improve his large stock of birds. Books he wrote on breeding pigeons were in demand around the world.

As a long-time resident of Bealey Avenue, he wrote a history of the properties along this street (first known as the North Belt), in 1976. The book was well received and is still used by researchers and genealogists. He followed it in 1979 with a similar history of Rolleston Avenue and Park Terrace (West Belt).

His medical practice was busy but, when Clark managed to have some time to himself, he could still be found hard at work in his surgery, his daughters say. Usually he would be researching or working on one of his many interests. When he took holidays, he and his wife loved to travel abroad and take in opera.

He was a quiet and reserved man, surprisingly shy for one of such accomplishments. He and his wife mixed with a steady group of friends, enjoying tennis parties well into their 70s.

Born Christchurch, September 14, 1914; died Christchurch, November 14, 2005. Survived by wife Erica, daughters Jenny, Susie and Nicky and seven grandchildren.

This obituary entitled Bealey Avenue doctor originally appeared in The Press newspaper (Christchurch) on 26 November 2005 and was written by Mike Crean. We are also grateful to Bruce Rennie and Carol Ashby of The Press.
Calling obituary writers

Obituaries do not write themselves, oddly enough. They require organisation, time, and respect for our departed colleagues.

Many decades ago, our medical practitioners were Otago graduates with a sprinkling of Brits. Things have changed. With the Auckland Medical School and our overseas-trained colleagues coming from a wide variety of medical schools, it is no longer a tight little club where everyone knows everyone else.

Thus we need a network to tell us who has died and who amongst family and colleagues can best give the rest of us some overview of the life, both professional and social, of our dead colleague.

For several years I have tried to rejuvenate this part of the Journal. I have been greatly helped in this task by colleagues in Auckland, Wellington, and Dunedin who scan the local newspapers. Anyone whose death notice does not appear in one of the four main dailies is liable to miss out.

Who is to write the obituary? Someone who cares. It need not be a literary masterpiece but should give something of the texture of the person’s life. Most funerals have a eulogy and the eulogist is often in the best position to help with an obituary.

About 400 words is usual but, with the electronic journal, space is no longer the problem it was in the days of hard copy.

The next time a colleague dies, ask yourself: “Who is going to do the obituary?” It could be you or someone whose arm may need only a gentle twist.

Most of the Journal belongs to the younger and brighter of us but the obituaries belong to us all. Even the old and cranky.

Roy Holmes
Coordinator of Obituaries
NZMJ

Calling book review writers

Book publishers in New Zealand and overseas regularly post us new publications in medicine and related fields. These books range from pocket-sized paperbacks to weighty tomes containing almost a thousand pages and retailing for several hundreds of dollars. Increasingly, some of these larger textbooks have electronic media attached (CDs).

Although we occasionally review books ourselves, the editor usually instructs my assistant and I to send such books to suitable reviewers throughout New Zealand, especially to our regular and reliable manuscript reviewers as a token of our appreciation (the reviewer can keep the book afterwards).

With medical professionals being very busy people and book reviews not being our core business, to date we have followed a hands-off approach of not setting deadlines nor reminding reviewers (unlike manuscript reviews).

This policy may need to change, however, as a sizeable percentage of book reviews never get written despite the best of intentions. Interestingly we seem to get many completed book reviews after the summer holidays, which suggests that this is the only time available for some people to read large books.

Therefore, if you received a book several months ago but don’t have the time to write the review, then please pass the book to a suitable colleague willing and able to undertake this task. The reviewer need not read every page of the book nor does the review have to be long or eloquent (many book reviews contain less than one full page of text). A concise, clearly written summary of the book is all that is required. To assist you, see the journal archives [http://www.nzma.org.nz/journal/archive.shtml](http://www.nzma.org.nz/journal/archive.shtml) for examples of published book reviews.

We will also consider unsolicited book reviews, and we welcome correspondence (stating your credentials and preferred subjects) from volunteers keen to write book reviews for the NZMJ in the future.

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Medical Benevolent Fund

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The 17th Hospice NZ Palliative Care and NZ Pain Society Conference: Making a Difference

The 17th Hospice NZ Palliative Care and NZ Pain Society Conference: Making a Difference will be held in October next year in Dunedin, New Zealand. The conference is scheduled for October 26–28, 2006.

For more details, contact event project manager Barry Woodland at Conference Innovators, phone +64 (0)3 379 0390 or email barry@conference.co.nz

To register your interest in presenting, please contact:

- David Jones, New Zealand Pain Society, at davidjones@healthotago.co.nz, or
- Simon Allan, Hospice NZ clinical medical adviser, Arohanui Hospice medical director and Palmerston North Hospital Regional Cancer Treatment Service clinical director, at Simon.Allan@midcentral.co.nz
GRAHAM AITKEN NUFFIELD TRUST

Graham Aitken Nuffield Medical Postgraduate Travelling Scholarship

Applications are invited from well-qualified New Zealand medical graduates in the 25–35 age group for the above Scholarship.

The purpose of the Scholarship is to provide travel funds to enable New Zealand graduates to further their clinical medical training and research interests in the United Kingdom.

The Scholarship will provide up to three return air fares to the UK, together with allowances amounting to $3000.

Candidates for the Scholarship must submit a training or research programme for approval together with the name of a person in the UK who will provide salary and facilities.

For further information please consult the Deans of the Schools of Medicine, or write to:

Professor A D Campbell, Graham Aitken Nuffield Trust, C/- Chemistry Department, University of Otago, P O Box 56, Dunedin.

Applications must be submitted to Professor Campbell by 31 March 2006
National Heart Foundation: 2006 Grant Applications

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