CONTENTS

24 October 2003

This Issue in the Journal

A summary of the original articles featured in this issue of the NZMJ

Editorials

Maori/non-Maori alcohol consumption profiles: implications for reducing health inequalities
  Kypros Kypri

Improving quality: maintaining the momentum
  Gerald Moss

Original Articles

Differences in patterns of alcohol consumption between Maori and non-Maori in Aotearoa (New Zealand)
  Dale Bramley, Joanna Broad, Ricci Harris, Papaarangi Reid, Rod Jackson, for the Alcohol Burden of Disease and Disability Group

Antidepressant poisoning deaths in New Zealand for 2001
  David Reith, John Fountain, Murray Tilyard, Rebecca McDowell

Epidemiology of slipped capital femoral epiphysis in a population with a high proportion of New Zealand Maori and Pacific children
  Susan Stott, Terri Bidwell

The Auckland Breast Cancer Register: a special project of the Auckland Breast Cancer Study Group
  Lorraine Neave, Vernon Harvey, Chelleraj Benjamin, Paul Thompson, Ora Pellett, Jeremy Whitlock, Wayne Jones, Garth Poole

Viewpoint

Replacing sugar-based soft drinks with sugar-free alternatives could slow the progress of the obesity epidemic: have your Coke® and drink it too
  Emme Chacko, Ingrid McDuff, Rod Jackson

Case Notes

New Zealand’s first fatality linked to use of 1,4-butanediol (1,4-B, Fantasy): no evidence of coingestion or comorbidity
  Lynn Theron, Karl Jansen, Adrian Skinner

Clozapine-associated polyserositis
  Allen Lim, Pathmanathan Sivakumaran, Marie Israel
100 Years Ago in the NZMJ

Diet in health and sickness

Medical Image

Mediastinal pseudo-mass

Methuselah

Selected excerpts from Methuselah

Medicolegal

Medical discipline - not guilty
Disgraceful conduct - unsafe practice

Obituaries

Heath Thompson
William Geddes Shiach

Book Review

e-Pathways: computers and the patient’s journey through care, Kathryn de Luc and Julian Todd (eds)
Gerald Moss
Differences in patterns of alcohol consumption between Maori and non-Maori in Aotearoa (New Zealand)
D Bramley, J Broad, R Harris, P Reid, R Jackson, for the Alcohol Burden of Disease and Disability Group

Average daily alcohol consumption is commonly used to measure the burden of alcohol on health-related conditions. Data from over 44 000 people in five New Zealand surveys show little difference between Maori and non-Maori daily alcohol consumption. However, when compared with Maori of the same sex and age, non-Maori are more likely to drink alcohol. They typically drink more often but much less per occasion. Pattern of drinking is important to consider when describing the impact of alcohol consumption on health.

Antidepressant poisoning deaths in New Zealand for 2001
D Reith, J Fountain, M Tilyard, R McDowell

Deaths from antidepressant overdose in New Zealand for the year 2001 were compared with the number of prescriptions of each medicine over the same year. The study used information from coronial inquiries and from the PharmHouse database. There were fewer poisoning deaths in relation to prescriptions of newer drugs, such as the selective serotonin reuptake inhibitors, than of older medicines, such as the tricyclic antidepressants.

Epidemiology of slipped capital femoral epiphysis in a population with a high proportion of New Zealand Maori and Pacific children
S Stott, T Bidwell

Slipped capital femoral epiphysis is a hip disorder that typically occurs in children aged ten to fourteen years. This epidemiologic study confirms the increased risk of slipped capital femoral epiphysis in children of NZ Maori or Pacific ethnicity and shows that children as young as eight years are now presenting with slipped capital femoral epiphysis. The study highlights the high rate of sequential slipped capital femoral epiphyses, with half of all second slips occurring within six months of the first.

The Auckland Breast Cancer Register: a special project of the Auckland Breast Cancer Study Group
L Neave, V Harvey, C Benjamin, P Thompson, O Pellett, J Whitlock, W Jones, G Poole

The Auckland Breast Cancer Register (ABCR) provides a unique opportunity to audit current clinical practice in breast cancer management for the Auckland region.
from 1204 cases recorded over two years (2000–2002) are reported from the multidisciplinary teams in both public and private practice. This audit documents demographics, diagnosis, treatments and outcomes. The ABCR provides essential information that will lead to a better understanding of breast cancer in Auckland and to more effective delivery of clinical resources in the Auckland region.
Maori/non-Maori alcohol consumption profiles: implications for reducing health inequalities

Kypros Kypri

The paper ‘Differences in patterns of alcohol consumption between Maori and non-Maori in Aotearoa (New Zealand)’ by Bramley et al appears in this issue of the NZMJ. It is an important study that includes data from five surveys – encompassing nearly 45 000 people – and a comparison of consumption patterns for Maori and non-Maori, by age and sex.

The principal finding is that while the total volume of alcohol consumed was similar in the two populations, the drinking patterns differed markedly. Relative to Maori, non-Maori drank more frequently but, on average, 40% less alcohol per drinking occasion. The findings are consistent with previous New Zealand research, and with differences between indigenous and non-indigenous people documented in other countries.

The study contributes to an emerging body of research examining patterns of alcohol consumption and their effects on health. Recent international studies of this type found that at the country level, aggregate consumption (estimated from sales or tax data) and drinking patterns (estimated from survey data) were independently related to the incidence of alcohol-related harm. Heavy episodic drinking was found to be particularly problematic. Rehm and colleagues argue that attention to both total volume and the incidence of heavy episodic drinking is important in understanding and preventing harm at the population level.

In general, the detrimental health effects of alcohol are characterised as either chronic or acute. The former, including liver cirrhosis, a range of cancers, depressive disorders, alcohol dependence, hypertensive disease, and haemorrhagic stroke, can result from repeated exposure to alcohol over many years.

In contrast, acute consequences can result from relatively brief exposures to the toxic or intoxicating effects of alcohol. They include road-traffic-crash injuries, falls, drowning, poisoning, assault, self-inflicted injury, and foetal alcohol syndrome, all of which have high attributable fractions for alcohol, particularly among the young. In contrast to chronic consequences, they do not require the victim to be a regular heavy drinker, and can be the result of another person’s drinking.

Net alcohol-related mortality is primarily attributable to the acute consequences listed above, and a disproportionate burden is borne by young people. This has particular implications for Maori health, considering the age distribution of the Maori population (69% aged under 35 years) relative to the non-Maori population (47% aged under 35 years). Furthermore, the cardio-protective benefits of moderate consumption for older individuals are enjoyed by a smaller proportion of Maori relative to non-Maori. Accordingly, if alcohol policies remain unchanged, the disparity in Maori/non-Maori life expectancy attributable to alcohol will likely increase in years to come.
Changes to the Sale of Liquor Act in 1989 and the 1999 amendments effected a substantial liberalisation of the availability of alcohol in New Zealand, with the introduction of supermarket sales, longer opening hours, and a near doubling in the number of liquor outlets in the period 1990 to 1995. Alcohol sponsorship, and brand advertising in the broadcast media, permitted for the first time in the early 1990s, continue to proliferate. Despite substantial evidence of a likely increase in youth alcohol-related harm, and opposition from numerous public health agencies and advocates, the minimum purchase age was reduced in 1999. It is too early to evaluate the effects of that change in terms of alcohol-related morbidity and mortality, but studies indicate that youth drinking levels have increased and that significant numbers of 15- to 17-year-olds can purchase alcohol from licensed premises or from friends who are of age. Given the relative youth of the Maori population, the effects of this particular change are also likely to increase health disparities.

The burden of injury and disease attributable to alcohol may be lessened by reducing aggregate consumption levels and/or by adopting less harmful patterns of drinking. Experts agree that a mix of preventive strategies is required, including measures to reduce aggregate consumption, and strategies aimed at high-risk situations and individuals. There is now a substantial literature on methods to reduce overall consumption, for example via increases in price and restrictions on availability. These approaches are, however, vigorously lobbied against by the alcohol industry and have, for some time, been out of favour politically in New Zealand. Evidence is comparatively sparse on the efficacy of methods reputed to selectively reduce high-risk consumption.

Rehm et al observe that ‘the impact of average volume of consumption on mortality or morbidity is partly moderated by the way alcohol is consumed by the individual, which in turn is influenced by the cultural context’. There is relatively little research on the way policies, economic conditions, and interventions are mediated and moderated by cultural contexts in New Zealand.

We know little about the likely reasons for the differences in drinking patterns described by Bramley et al. We know less still about what policies and interventions benefit Maori. Addressing alcohol-related health inequalities is not simply a matter of ‘doing something special for Maori’: we need to learn to be comfortable in talking about disparities, to understand their origins, and to conduct policy-relevant research on their reduction. The research described by Bramley et al is an excellent start.

**Author information:** Kypros Kypri, Research Fellow, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin

**Correspondence:** Dr Kypros Kypri, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, P O Box 913, Dunedin. Fax: (03) 479 8337; email: kypros.kypri@ipru.otago.ac.nz

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Improving quality: maintaining the momentum

Gerald Moss

The ‘Quality and patient safety’ movement in New Zealand has gained momentum through the enthusiasm resulting from the recent 3rd Asia Pacific Forum on Quality Improvement in Health Care, publication of the report ‘Improving quality’ and the announcement of many quality awards both nationally and locally. The actual implementation of quality initiatives – that is to say ‘change management’ – and demonstration of success will depend upon maintaining this momentum. This immediately raises issues around leadership and culture change in a complex adaptive system, and around effectiveness and evaluation. Quality and Safety in Healthcare – a relatively inexpensive journal also online at http://www.qshc.com – contains a wealth of interesting, informative and relevant material around these issues.

Clinical governance

In 1997 the concept of ‘clinical governance’ was introduced into the UK National Health System as an approach to quality improvement that supported ‘joined-up-ness’ of new and established quality-improvement initiatives. This systems approach not only aimed to improve effectiveness through integration of these activities but also drew attention to the interdependence of the system components – each component had effects upon the other components, and was in turn affected by what happened in the other components.

This approach to quality improvement, albeit with local adaptation, has been strongly promoted in the Australian context, and moderately so in New Zealand. The importance of clinical governance lies in the acceptance of the concept by clinical and allied health professionals and the opportunity to learn from the enormous amount of published research coming out of the UK.

A new report from the National Audit Office (NAO) on the implementation of clinical governance in the NHS is both timely and informative. It identifies significant barriers to further improvement, including lack of time and resources, cultural difficulties, lack of strategy and lack of expertise in particular components. There are also concerns over continuing professional development, largely caused by workload or organisation of working commitments, which conflicts with training. The report concludes that progress in implementing clinical governance is patchy, and varies between trusts, within trusts and between the components of clinical governance. Overall, the key features of the better-performing organisations are quality of leadership, commitment of staff, and willingness to consider doing things differently.

Leadership

There is no shortage of books on leadership and courses offering training in leadership; however, there is little information on the effectiveness of these and for whom they are targeted. This issue may be addressed in New Zealand as a result of the formation of the Leadership Institute, an initiative of the University of Auckland.
Business School, in February 2003. It will have a national focus to harness the energy, ideas and momentum of the Knowledge Wave 2003 Leadership Forum.

Clearly leadership from the top – that is from the Minister of Health and through district health board (DHB) chairs and CEOs – is of paramount importance, but ‘leadership should be researched as a process that can occur throughout organisations, and not just from people at the senior end of the hierarchy.’\textsuperscript{10} Those who feel unable to develop themselves as leaders could become excellent supporters; support is a resource that leaders require.

\textbf{Culture change}

A healthcare organisation consists of a myriad of subcultures, each unique with different histories, perspectives and collegial loyalties along with proud and protective attitudes around their respective departments. When proposing change, administrators should remember that one size does not fit all.\textsuperscript{11–14} A further consideration is the matching of hierarchical management and professional models of patient care. An inclusive approach, where there are opportunities for communication, where communication is two-way, where people’s opinions are valued and where patience is a virtue, is likely to be conducive to success.

The ‘quality manager’ is involved in leadership, the development of skills, and the provision of resource amongst other duties. The situation is New Zealand is similar to that in Australia, namely poor support on the part of organisations for these professionals and a lack of opportunities for their own professional development, education and training.\textsuperscript{15}

\textbf{Patient safety}

The boundaries between the various elements of clinical governance are poorly defined and there is a great deal of overlap; indeed, the underlying principle of clinical governance is that of integration and joined-up-ness. However, the term ‘patient safety’ draws attention to the burden of adverse events\textsuperscript{16} and the growing interest in clinical risk management. Part of the hoped-for change in healthcare is the development of ‘open disclosure’ when things go wrong and of a blame-free culture in which mistakes can be disclosed and the management of risk improved.

The current environment in respect of the law in healthcare tends to focus on the need to attribute blame before compensation can be awarded, even if simply to address a serious outcome in which negligence played no part. This tendency is a major concern as it works against the development of a blame-free culture.\textsuperscript{17}

The NAO report indicated that, although the recording, collation and review of data were performed well, performance was weak as regards training in risk management and in moving from identifying measures to improve quality to taking action.\textsuperscript{9}

\textbf{Implementation}

Much research has been conducted around the implementation of research-based evidence in clinical practice and is relevant to change management in healthcare generally. Success factors identified include strong evidence, supportive opinion leaders, integration within a committed organisation, context analysis, professional involvement, good project management, and careful understanding of the local
Implementation should be seen not as rational and linear (political model) but as a negotiated and uncertain process enacted locally within clinical groups and based on tacit knowledge.\textsuperscript{19}

The characteristics associated with the successful implementation of quality and excellence in healthcare share, in particular: strong leadership, involvement, empowerment, customer focus, teamwork, trust, effective information transfer, and organisational commitment.\textsuperscript{14}

Increasingly, healthcare professionals have been required to do more with less, and management professionals have been burdened with pressures over quantity and cost to the relative exclusion of quality and safety.\textsuperscript{20} Failure to provide adequate resources after having raised enthusiasm and commitment is likely to be counterproductive and bring momentum to a halt. Generally, staff point to the lack of time as a barrier to implementation, but it is also a barrier to reflection and evaluation. How will they know how they are doing?

The requirements for successful implementation of complex change have been enumerated along with indicators of failure associated with the lack of each one.\textsuperscript{21} For example, the requirements are vision, skills, incentive, resources and an action plan. If all are present, change will probably occur. Where there is no vision confusion reigns; where there is no skill anxiety results; if there is no incentive change will be gradual at best; lack of resources leads to frustration; and if there is no action plan false starts may be expected.

**Knowledge management**

Systems within healthcare organisations depend upon:

- information – which must be accessible in a timely manner;
- knowledge – which tends to diffuse poorly across boundaries;
- learning – which often does not appear to happen.\textsuperscript{21}

The art and science of marketing have received little attention in healthcare. Part of these are a requirement for providers to ‘know the customer’; knowing the customer is important in change management, and in successful consumer involvement.

Medical librarians and information technologists are becoming key players (as health informationists) in knowledge management and could become even more effective through opportunities for engagement with clinical and management professionals.

Organisations might be encouraged through the Wave Project to share expertise, and use economy of scale, for example in the provision of comprehensive and coordinated education and training of a high standard for quality managers.

A universal understanding of complexity in healthcare (the subject of a series of articles in the BMJ) will greatly enhance the likelihood of successful implementation of change. It is not about agreeing that healthcare is complex, it is about understanding that healthcare is a complex adaptive system in which, for example, one observes the rich interaction of components and not just the system’s structure, in which history and the environment interact in a non-linear way, and in which emergent behaviour cannot be predicted.\textsuperscript{4} Such understanding encourages inclusiveness and helps an organisation to make sense of itself.
Evaluation

‘Evaluation is an integral component of quality improvement and there is much to be learned from the evaluation of small-scale quality improvement initiatives at a local level.’ Quality improvement programmes consume considerable resources and may have significant consequences, yet little is known about whether or not they are effective and the reasons for this. It does not make any sense to plan an intervention without an intention to evaluate its effectiveness; planning the evaluation should be part of planning the intervention. ‘Those planning and reporting evaluations of quality improvement should do so in the context of a systematic review. Similarly, those planning quality improvement activities should consider the results of systematic reviews when doing so.’ Although the literature supports the development of evaluation-informed and evidence-based management in healthcare, reviews confirm that use of appropriate research is sub-optimal.

Making it happen

Progress in the development of quality improvement in healthcare has been slow and incremental. In order to build and maintain momentum, to the point where the snowball becomes an avalanche, it is necessary to reach a ‘tipping point’ or critical level, the requisites for which may be summarised below:

- key people, such as ‘connectors’ or network people, ‘mavens’ who are curious and knowledgeable, and ‘salesmen’ or super opinion leaders;
- the packaging or marketing of information – described as the ‘stickiness factor’ – which grabs attention and makes an impression;
- the power of context, which includes forming groups of a manageable size, allowing true engagement, a deep understanding of the context, and meticulous planning.

How are we doing?

Recommendations from the NAO for trusts in the UK are based on evaluations of progress so far in that context. At present, such data are not available in the New Zealand context although evaluation would indicate what has or has not worked, why this is so, and where resources might be most usefully applied. The NAO-recommended activities will be well established in some New Zealand DHBs but, just as in the UK, implementation is likely to be patchy. These recommended activities are:

- the review of information requirements;
- the development of systems of internal reporting on quality;
- the maximisation of the benefits to be derived from clinical audit;
- support for continuing professional development;
- benchmarking of key clinical governance initiatives;
- agreement on action plans, timetables and priorities.

One thing cries out above all else: leadership.
Author information: Gerald Moss, Consultant in Clinical Governance, Christchurch

Correspondence: Dr Gerald Moss, 29 Rubens Place, Christchurch. Email: gerald.moss@clear.net.nz

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Differences in patterns of alcohol consumption between Maori and non-Maori in Aotearoa (New Zealand)

Dale Bramley, Joanna Broad, Ricci Harris, Papaarangi Reid and Rod Jackson, for the Alcohol Burden of Disease and Disability Group

Abstract

Aim To describe relative differences in alcohol consumption patterns in Maori and non-Maori from all available large-scale New Zealand surveys.

Methods Data from five New Zealand surveys (national and population specific) conducted since 1988 were made available to the investigators and were re-analysed by sex and age group in Maori and non-Maori using multivariate modelling.

Results There was a total of 44 830 people in the combined study populations, of whom 6926 (15.4%) were Maori. There was significant variation in the populations sampled and instruments used for measuring alcohol; however, the relative differences in consumption patterns between Maori and non-Maori were similar across all studies. In all age groups, and in men and women, non-Maori were more likely to be drinkers. The strength of this relationship increased with age. In all age groups, frequency of alcohol consumption (days a year) was higher for non-Maori, though the relative volume drunk on a usual drinking occasion was consistently around 40% less than for Maori. The averaged daily volume of alcohol consumed was similar between Maori and non-Maori.

Conclusions Maori have markedly different alcohol consumption patterns from non-Maori, which are not apparent when averaged daily alcohol consumption is compared. Frequency of drinking and amount consumed on a typical drinking occasion should be considered when determining the relationship between Maori alcohol consumption and health-related problems.

The relationship of alcohol consumption with morbidity and mortality is well documented and is influenced by the pattern of drinking, however, no such data exist specifically for Maori.

The intent of this paper is to investigate relative differences in patterns of consumption between Maori and non-Maori rather than to document differences in the prevalence of consumption; therefore, we sought all major available studies whether or not they were based on representative population samples.

The results of this paper will inform future research that examines the association between Maori alcohol consumption patterns and health.

Methods

Surveys Data from five large New Zealand surveys conducted since 1988 were re-analysed by sex and age group to examine differences between Maori and non-Maori. Inclusion criteria for studies included access to the data sets comprising individual records, recording of self-identified ethnicity data, adequate numbers of Maori and standardised reporting of alcohol consumption. As the focus was on relative differences between Maori and non-Maori, it was unnecessary for studies to be based on
representative population samples as long as all Maori and all non-Maori in the study populations selected had equal opportunities for inclusion. Included were two national (New Zealand Health Survey 1997 and the Sleep Survey 1999) and three population-specific studies (Fletcher Challenge/University of Auckland Survey 1992, NZ Blood Donors Health Study 1998–1999 and the Workforce Diabetes Survey 1988–1990). Details of sampling, inclusion criteria, and data collection procedures are available. It is unfortunate that the National Alcohol Surveys conducted by Whariki and the Alcohol and Public Health Research Unit at University of Auckland were not available (see discussion).

**Demographic classification** All data were collected from the respondents at interview (or, in the case of the Sleep Survey, by self-completed questionnaire). Ethnicity was classified and coded according to self-identified ethnicity. Where more than one ethnic group was recorded the New Zealand Census hierarchical categorisation of ethnic group was used to classify all responses to one prioritised ethnic group. In all analyses Maori were the reference group to which non-Maori were compared.

Sex and age were recorded at interview/survey. Those aged less than 18 years (because the law does not allow people aged under 18 years to buy liquor) or over 75 years (because numbers of people aged over 75 years were low) were excluded to achieve an age range of 18 to 74 years. In two surveys this age range was further limited by the survey design: the Sleep Survey (30–60 years only) and the Workforce Diabetes Survey (40–74 years only). Results are presented by sex and age group where age groups are defined as 18–34 years, 35–49 years and 50–74 years.

**Measures of alcohol consumption** Four different measures of alcohol consumption were considered where available. Each survey asked how frequently participants drank alcohol, and provided several response categories. In general, any participant reporting drinking alcohol never or less often than monthly was regarded as a non-drinker, those drinking more often than monthly were classified as drinkers. Participants in the Sleep Survey were classified as non-drinkers if they drank never or less often than once a week, because no longer time category was available. Participants in the New Zealand Health Survey were classified as non-drinkers if they reported not drinking alcohol in the last year.

Based on the reported frequency of drinking, we calculated the number of days a year on which the respondent drank alcohol. Two methods of calculation were employed: in the first we allocated zero days for non-drinkers in order to represent frequency of consumption for all the population; in the second we excluded non-drinkers from analyses in order to represent frequency of consumption for drinkers only.

Where the survey obtained information about volume of alcohol drunk on a typical occasion, we estimated the grams of ethanol consumed per occasion. Where possible, we estimated the quantity of alcohol using listings provided by the New Zealand Department of Food and Agriculture, otherwise we used either 10 or 12 grams per standard drink according to the methods used by the investigators. Volume was not calculated for non-drinkers and they were omitted from these analyses.

Frequency of drinking and typical volume data were not available for those in the Workforce Diabetes Survey. For the few participants in the NZ Health Survey whose consumption information was missing, corresponding data from the National Nutrition Survey has been used.

Averaged daily alcohol consumption (in grams) was calculated from the product of the proportion of days on which drinking occurred and the volume consumed on a typical occasion. Again, non-drinkers were omitted.

**Statistical methods** Within each survey, the proportion of participants in each sex and age group classified as non-drinkers was reported. Logistic regression was used to estimate the relative odds that non-Maori were drinkers, compared with Maori. Ethnicity (in binary form) and age (as a continuous variable to adjust for the different distributions of age within the surveys) were the only predictors in these regressions. Summary estimates by sex and age group were obtained in similar manner, with the addition of a variable to indicate which survey the individual was from. An interaction term between survey and ethnicity was used to test for heterogeneity, then removed for reporting summary estimates for each sex–age group.

Mean frequency of drinking is reported as mean number of days a year on which alcohol is drunk. To estimate relative frequency of drinking for non-Maori, we used Poisson regression models with an offset of 365 days and scaled for the deviance to correct for slight over-dispersions where present. Summary estimates were obtained in a similar manner, again with the addition of variables to represent sex and age group, age as a continuous variable and which survey the individual was from. Again, an interaction term between survey and ethnicity was used to test for heterogeneity.
For both volume of alcohol (grams) drunk on a typical occasion, and averaged daily volume consumed, means for each group defined by survey, sex and age are shown, together with the relative volume drunk by non-Maori compared with Maori. Log transformations of alcohol measures were modelled using generalised linear regression models to obtain summary estimates, with tests of heterogeneity as above.

The model coefficients for non-Maori relative to Maori, and their 95% confidence limits, were back-transformed to obtain relative estimates of consumption for non-Maori compared with Maori.

In all models, persons with missing data for the variable of interest were excluded from analyses. Results in which the 95% confidence interval does not include 1.00 are regarded as statistically significant. In producing the summary estimates, where significant heterogeneity was found, box-plots for non-Maori and Maori were compared to determine its extent and source.

**Results**

There was a total of 44 830 people in the combined study population. Of these, 24 484 (54.6%) were males; 13 174 (29.4%) were aged 18–34 years; 18 478 (41.2%) aged 35–49 years; and 13 178 (29.4%) aged 55–74 years.

Of all participants, 6926 (15.4%) were Maori (3535 men) and 37 904 non-Maori (20 949 men). In the 50- to 74-year age group, a smaller proportion was Maori compared with non-Maori (26.2% of Maori compared with 30.0% of non-Maori). Table 1 shows the numbers included from each survey, by ethnicity, sex and age group (see appendix for tables and figures). The largest survey was the NZ Blood Donors Health Study, which contributed 17 437 study participants, 38.9% of the total. The Sleep Survey contributed the largest number of Maori participants (3194).

Figure 1 shows the percentage of participants who were classified as non-drinkers (see appendix for tables and figures). In all age groups, except men aged 18–34 years, a significantly higher proportion of Maori were non-drinkers. Table 2 reports the likelihood of non-Maori participants being drinkers relative to Maori. As age increased, so did the strength of this relationship for both men and women. Among one group at comparatively high risk of heart disease, men aged 50–74 years, non-Maori were about 90% more likely to be drinkers than Maori men (odds ratio = 1.89, 95% confidence interval (CI) 1.55–2.31).

Figure 2 shows the frequency of alcohol consumption (days a year) for Maori and non-Maori, inclusive of non-drinkers. Across all the groups examined, non-Maori were more frequent consumers of alcohol relative to Maori, reporting drinking on over 50% more days of the year. This effect was more marked with increasing age (Table 3), ranging from a low among men aged 18–34 years (relative frequency (RF) = 1.47, 95% CI 1.46–1.49) to a high among women aged 50–74 years of more than double the frequency (RF = 2.27, 95% CI 2.24–2.30).

Table 4 and Figure 3 report frequency of alcohol consumption (days a year) for Maori and non-Maori among drinkers. Again, a similar pattern was seen though there was a lower degree of association.

The volume of alcohol consumed on a typical occasion reported by non-Maori was consistently less than Maori (Table 5, Figure 4). For every study this was apparent in each age group for both sexes; summary measures consistently estimated the size of the difference at 35–42% less. Non-Maori men aged 35–49 years reported drinking about 40% less alcohol on a typical occasion than Maori men in the same age group (relative volume (RV) = 0.59, 95% CI 0.56–0.62).
Regardless of survey, age group and sex, there were few statistically significant differences in average daily consumption between Maori and non-Maori (Figure 5). Table 6 shows average daily consumption of alcohol among non-Maori drinkers relative to Maori drinkers. When all surveys were combined, only two summary estimates reached significance, both for the youngest age group: men (relative daily volume (RDV) = 0.90, 95% CI 0.82–0.98) and women (RDV = 0.73, 95% CI 0.67–0.81).

In many of the models estimating overall effects, there were significant interactions between the variables representing survey and Maori/non-Maori ethnicity, suggesting heterogeneity in the association between Maori and the various measures of alcohol in the different surveys. Generally, this heterogeneity would lead to rejection of summary estimates based on such models. However, examination of plots of alcohol consumption by sex, age group and survey by Maori/non-Maori, showed that almost all such differences were small. Given the narrow confidence intervals about the estimates for all but the final alcohol measure (averaged daily consumption) and the general consistency of the directions of effect, we concluded that large numbers and use of continuous variables were providing power to detect very small differences that could be ignored for the purpose of this paper, ie, to compare consumption between Maori and non-Maori. Consequently, all summary measures have been reported as the best estimate of the relationship between non-Maori and Maori drinking patterns.

Discussion

This research has been undertaken using a kaupapa Maori framework, whereby the study analysis was undertaken from a Maori perspective. This is distinct from other methodologies that may ‘minoritise’ Maori with insufficient data quantity or quality to undertake analyses necessary to inform Maori health development. In this framework, Maori are the reference population and are compared with non-Maori. Where appropriate, this type of analysis enables disparities to be identified and their elimination prioritised. This analytic approach is consistent with a Treaty of Waitangi framework.

This analysis is unique in that it combines data from a number of studies that have been undertaken in Aotearoa in recent years. In total, there were 6926 Maori participants in the studies used for this analysis (and 37 904 non-Maori), and this is therefore the largest published analysis of alcohol consumption in Aotearoa for Maori. Our objective was to describe relative differences in patterns of consumption between Maori and non-Maori so we included all available major studies whether or not they were based on representative population samples. Therefore, the relative differences in drinking patterns identified, but not the absolute levels of consumption, are likely to be generalisable to the population of Aotearoa.

The main findings from this research are that Maori are less likely to drink alcohol, drink less often, but drink more on a typical drinking occasion, when compared with non-Maori. These differences in drinking patterns combine such that average alcohol consumption a day among Maori and non-Maori is similar.

Between all studies, national and non-national, there is marked consistency of results. This has occurred in spite of differences in methodology between individual studies, supporting the validity of the pooled estimates.
Maori, therefore, have markedly different patterns of alcohol consumption to non-Maori. These different patterns of alcohol consumption have implications for health.

Most estimates of this relationship have used measures of average daily alcohol consumption and not pattern of alcohol consumption.\textsuperscript{12} Using the average alcohol consumption variable for Maori therefore may not produce accurate associations. Other studies have shown that specific drinking patterns may have independent effects on health that are not explained by total consumption.\textsuperscript{13} This may also be true for Maori. Further research regarding the relationship between Maori alcohol consumption patterns and health risk is therefore needed.

Discussion of alcohol consumption by ethnicity has been a sensitive topic for some years. This has in part been due to a ‘victim blaming’ interpretation\textsuperscript{14,15} of data that have been published where there is no acknowledgement that risk behaviours of individuals are socially patterned. Such an approach is generally counterproductive to dialogue and progress in reducing harm from alcohol consumption.

Historically, alcohol became readily available to Maori in the early nineteenth century.\textsuperscript{16} Maori initially showed a strong aversion to drinking alcohol. Discriminatory legislation regarding consumption of alcohol by Maori was not removed until 1948 with the passage of the Licensing Amendment Act, which repealed the previous law prohibiting consumption of alcohol by Maori in public bars.\textsuperscript{16}

While this study demonstrates the different drinking patterns between Maori and non-Maori, research from the 1996/1997 New Zealand Health Survey also indicates that Maori adults are more likely than non-Maori to have potentially ‘hazardous drinking’ patterns.\textsuperscript{17} Such patterns carry with them higher risk to physical or mental health. Furthermore, ‘hazardous drinking’ is socially patterned in that it is associated with socioeconomic gradients that disproportionately affect Maori.\textsuperscript{18} Such drinking patterns and their associated health problems are not unique to Maori and are similar among other indigenous populations that have experienced colonisation.\textsuperscript{19,20}

In recent times there have been health promotion initiatives to raise awareness among Maori about the harm associated with hazardous drinking patterns in the expectation that those ‘at risk’ will change attitudes and behaviours.\textsuperscript{21} These efforts have occurred in an environment of increasing availability of alcohol and re-introduction of multimedia advertising of alcohol. It is not yet known whether these factors are associated with increased uptake of alcohol, especially among younger and more vulnerable populations. At the same time, there has been limited policy development aimed at addressing the socioeconomic gradient that continues to marginalise Maori into the most deprived echelons of our society within which hazardous drinking is more prevalent.

To date, few published studies have compared Maori and non-Maori alcohol consumption patterns. The most comprehensive is Te Ao Waipiro.\textsuperscript{22} In that study, the median frequency of drinking amongst Maori was lower, and the median annual volume of consumption for Maori males was higher, than for all males. The median quantity consumed per occasion was much higher among Maori than for the total population. Our results are consistent with those study findings, although our analyses differ in that we compared Maori with non-Maori, whilst in Te Ao Waipiro Maori were compared with the total population, which included Maori.
Alcohol data from the National Alcohol Surveys, including the 2000 National Maori Alcohol Survey conducted by the Whariki Research Group and the Alcohol and Public Health Research Unit, were not included in this analysis as the kaupapa of these surveys were to firstly present Maori-specific analyses and then determine further analyses through a process of consultation with Maori. This process was underway, though not complete, at the time of writing this paper.

The 1997 National Nutrition Survey, which used a subset of the participants of the NZ Health Survey plus additional Maori and Pacific people, reported that Maori men had a higher mean daily alcohol intake (25 g) than European and others (19 g). Among women, Maori had similar intakes to European and others (8 g and 9 g respectively). These findings are not directly comparable as they represent alcohol intake over a single 24-hour period and include non-drinkers.

A report from Te Puni Kokiri in 2000, using data from the National Nutrition Survey, stated that Maori are considerably less likely to be moderate drinkers (as opposed to non-drinkers or hazardous drinkers) than non-Maori (46% and 66% respectively) and relatively higher proportions of Maori are either non-drinkers or hazardous drinkers. While our results are not directly comparable, they are broadly consistent.

There are a number of potential sources of bias that may occur with our study methodology. These include combining data from studies conducted during different time periods (1988–2001) and the possibility that drinking patterns may have changed over that period; the use of a mixed group of studies, only some of which were population-based; and the use of different instruments for measuring alcohol consumption. However, the similarity of our results to those from the individual studies is reassuring and suggests that these factors are likely to have caused only minimal bias.

In summary, non-Maori and Maori have markedly different alcohol consumption patterns. This must be considered when determining the relationship between Maori alcohol consumption and health risk or when applying methods of risk estimation based on average consumption levels.

Author information: Dale M Bramley (Nga Puhi), Senior Lecturer; Joanna B Broad, Research Fellow, EPIQ Group, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland; Ricci Harris (Ngati Kahungunu, Ngati Raukawa, Kai Tahu), Public Health Medicine Registrar; Papaarangi Reid (Te Rarawa), Public Health Physician, Te Ropu Rangahau Hauora a Eru Pomare, Wellington School of Medicine and Health Sciences, University of Otago, Wellington; Rod Jackson, Head of Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland. On behalf of the Alcohol Burden of Disease and Disability Group, University of Auckland: Shanthi Ameratunga, Dale Bramley, Joanna Broad, Jennie Connor, Rod Jackson, Patricia Metcalf, Robert Scragg and Sue Wells.

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MacMahon and Robyn Norton (Fletcher-Challenge University of Auckland Heart and Health Study), Ricci Harris, Papaarangi Reid and Philippa Gander (Sleep Survey), and the Ministry of Health (National Nutrition Survey). We particularly appreciate the willingness of the investigators of these studies to allow use of their data, and their advice and assistance.

We thank Jennie Connor, Robert Scragg, Shanthi Ameratunga, Elizabeth Robinson, Robyn Norton, Margaret Geddes, Mike MacAvoy and Helen Moewaka Barnes for their advice and comments on earlier drafts of this paper.

Correspondence: Dr Dale Bramley, Division of Community Health, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 441 8957; email: dale.bramley@waitematadhb.govt.nz

References:


Appendix: figures and tables

Figure 1. Percentage of non-drinkers (with 95% error bars, crude (unadjusted) data)

![Figure 1](image1.png)

Figure 2. Frequency of drinking including non-drinkers and drinkers (with 95% error bars, crude (unadjusted) data)

![Figure 2](image2.png)
Figure 3. Frequency of drinking among drinkers (with 95% error bars, crude (unadjusted) data)

![Frequency of drinking among drinkers](image1)

Figure 4. Alcohol drunk on typical occasion (with 95% error bars, crude (unadjusted) data)

![Alcohol drunk on typical occasion](image2)
Figure 5. Average volume alcohol drunk per day (with 95% error bars, crude (unadjusted) data)
Table 1. Studies used in analyses of alcohol consumption

<table>
<thead>
<tr>
<th>Year of fieldwork (approx)</th>
<th>Ref</th>
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<th>All Non-Maori†</th>
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<td></td>
<td>18–34</td>
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<tr>
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<td>Population-based studies</td>
<td></td>
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*includes all those with any self-reported Maori ethnicity
†includes all those in survey with no mention of Maori ethnicity
‡nationally representative random sample of those aged 15+ years, selected using an area-based sampling frame, face-to-face interviews; linked to data from sub-sample surveyed in nutrition survey, plus additional Maori sample; self-administered food-frequency questions
§National random sample of people aged 30–59 years selected from electoral rolls, postal survey
¦Fletcher Challenge employees aged 35+ years, interviewed at work sites in Auckland and Tokoroa
¶blood donors throughout NZ, self-administered questionnaire completed during voluntary blood donation visits
**employees aged 40–64 years from work sites in Auckland region, interviewer-administered questionnaire
Table 2. Non-drinkers (%) and the odds ratios (OR) of being a drinker, non-Maori relative to Maori, by gender and age group

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<td>All women</td>
<td>20.8</td>
<td>23.3</td>
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\*includes those with self-reported Maori ethnicity, the referent group in OR calculations
\(\hat{i}\)includes all those in survey with no mention of Maori ethnicity
\(\hat{j}\)OR = odds ratio of being a drinker for this age–sex group, vs Maori, calculated by logistic regression with adjustment for age as a continuous variable
\(\hat{k}\)95% CI = 95% confidence intervals about the estimate
Table 3. Frequency of alcohol consumption (days a year) and ratio of frequency of drinking among all respondents, non-Maori relative to Maori, by gender and age group

<table>
<thead>
<tr>
<th>Mean days a year alcohol consumed*</th>
<th>Relative frequency of drinking by non-Maori vs Maori$</th>
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<tr>
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<td>Maori†</td>
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<td></td>
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<tr>
<td>50–74</td>
<td></td>
</tr>
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</table>

Men

Population-based studies

NZ Health Survey & National Nutrition Survey 51.7 66.0 49.0 87.5 100.3 126.2 1.67 1.63–1.70 1.49 1.45–1.52 2.56 2.49–2.62
Sleep Survey 48.1 58.3 76.4 78.7 108.0 128.1 1.62 1.58–1.66 1.86 1.83–1.88 1.68 1.66–1.70

Studies in selected populations

Fletcher Challenge/Univ. of Auckland Survey 62.1 76.8 103.4 101.4 122.1 138.6 1.63 1.61–1.66 1.56 1.54–1.58 1.34 1.31–1.36
NZ Blood Donors Health Study 71.5 77.0 125.5 80.6 128.5 160.1 1.10 1.09–1.12 1.65 1.62–1.68 1.27 1.24–1.30
Workforce Diabetes Survey - - - - - - - - - - - - - -

All men 59.5 65.8 79.2 89.0 120.4 145.2 1.47 1.46–1.49 1.69 1.67–1.70 1.65 1.64–1.67

Women

Population-based studies

NZ Health Survey & National Nutrition Survey 27.7 33.0 31.0 47.0 69.4 75.2 1.70 1.67–1.74 2.09 2.04–2.14 2.45 2.37–2.53
Sleep Survey 35.2 38.5 37.5 47.1 72.9 88.2 1.34 1.30–1.37 1.90 1.87–1.92 2.35 2.32–2.39

Studies in selected populations

Fletcher Challenge/Univ. of Auckland Survey 44.4 53.4 50.5 79.4 109.2 118.7 1.78 1.73–1.84 2.04 1.97–2.12 2.36 2.19–2.56
NZ Blood Donors Health Study 48.7 57.4 76.6 70.5 105.8 126.9 1.44 1.41–1.46 1.80 1.77–1.84 1.66 1.60–1.71
Workforce Diabetes Survey - - - - - - - - - - - - - -

All women 38.0 41.0 38.8 65.9 93.8 105.7 1.51 1.50–1.53 1.91 1.89–1.93 2.27 2.24–2.30

\*means and regressions models included all responders

\†includes all those with any mention of self-reported Maori ethnicity – the reference group in modelling of frequencies

\‡includes all those in survey with no mention of Maori ethnicity

\§ratios were calculated using a Poisson regression model for each sex and age group, adjusting for age as a continuous variable within age group, and for survey in summary estimates

\§95% CI = 95% confidence intervals about the estimate
Table 4. Frequency of alcohol consumption (days a year) and ratio of frequency of drinking among drinkers, non-Maori relative to Maori, by gender and age group

<table>
<thead>
<tr>
<th></th>
<th>Mean days a year alcohol consumed*</th>
<th>Relative frequency of drinking by non-Maori vs Maori‡</th>
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<td></td>
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<td>All Non-Maori‡</td>
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<tr>
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<td>18–34 35–49 50–74</td>
<td>18–34 35–49 50–74</td>
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<td>% % %</td>
<td>% % %</td>
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<td></td>
<td>Ratio§</td>
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<td><strong>Men</strong></td>
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<tr>
<td>Population-based studies</td>
<td></td>
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<tr>
<td>NZ Health Survey &amp; National Nutrition Survey</td>
<td>58.8 79.8 75.6</td>
<td>100.4 114.6 148.0</td>
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<td>Sleep Survey</td>
<td>57.3 71.1 96.2</td>
<td>86.2 118.9 143.2</td>
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<td>Fletcher Challenge/Univ. of Auckland Survey</td>
<td>69.9 89.7 121.6</td>
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<td>Workforce Diabetes Survey</td>
<td>48.1 53.9 59.9</td>
<td>81.9 112.8 136.2</td>
</tr>
<tr>
<td>All women</td>
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</tr>
</tbody>
</table>

*means and regressions models included drinkers only
†includes all those with any mention of self-reported Maori ethnicity – the reference group in modelling of frequencies
‡includes all those in survey with no mention of Maori ethnicity
§ratios were calculated using a Poisson regression model for each sex and age group, adjusting for age as a continuous variable within age group, and for survey in summary estimates
¶95% CI = 95% confidence intervals about the estimate
Table 5. Volume of alcohol consumed on a typical occasion (mean, gm) among drinkers, and ratio of volume reportedly consumed on usual occasion, non-Maori relative to Maori, by gender and age group

<table>
<thead>
<tr>
<th></th>
<th>Mean volume consumed on typical occasion*</th>
<th>Relative volume drunk by non-Maori vs Maori‡</th>
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<tbody>
<tr>
<td></td>
<td>Maori†</td>
<td>All Non-Maori‡</td>
</tr>
<tr>
<td></td>
<td>18–34  35–49  50–74     18–34  35–49  50–74</td>
<td>18–34  35–49  50–74     18–34  35–49  50–74</td>
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<td>%     %     %     %     %     %</td>
<td>%     %     %     %     %     %</td>
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<tr>
<td></td>
<td>Mean</td>
<td>Mean</td>
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<tr>
<td>All women</td>
<td>57.0</td>
<td>39.9</td>
</tr>
</tbody>
</table>

*means and regression models included only those where recorded volume of alcohol drunk on usual occasion was >0
†includes all those with any mention of self-reported Maori ethnicity – the reference group in modelling of frequencies
‡includes all those in survey with no mention of Maori ethnicity
§regression coefficients were calculated using linear regression model with log link, for each sex and age group, adjusting for age as a continuous variable within age group, and for survey in summary estimates
¶95% CI = 95% confidence intervals about the estimate
Table 6. Averaged daily volume of alcohol consumed (mean, gm) among drinkers, and ratio of averaged daily volume reportedly consumed, non-Maori relative to Maori, by gender and age group

<table>
<thead>
<tr>
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<th>Mean volume consumed on typical occasion*</th>
<th>Relative volume drunk by non-Maori vs Maori†</th>
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<td>Fletcher Challenge/Univ. of Auckland Survey</td>
<td>18.6</td>
<td>16.3</td>
</tr>
<tr>
<td>NZ Blood Donors Health Study</td>
<td>20.0</td>
<td>14.0</td>
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<tr>
<td>Workforce Diabetes Survey</td>
<td>-</td>
<td>17.8</td>
</tr>
<tr>
<td>All men</td>
<td>15.6</td>
<td>12.7</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Population-based studies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ Health Survey &amp; National Nutrition Survey</td>
<td>6.2</td>
<td>4.8</td>
</tr>
<tr>
<td>Sleep Survey</td>
<td>5.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Studies in selected populations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fletcher Challenge/Univ. of Auckland Survey</td>
<td>8.6</td>
<td>9.5</td>
</tr>
<tr>
<td>NZ Blood Donors Health Study</td>
<td>12.7</td>
<td>9.2</td>
</tr>
<tr>
<td>Workforce Diabetes Survey</td>
<td>-</td>
<td>9.0</td>
</tr>
<tr>
<td>All women</td>
<td>8.0</td>
<td>5.9</td>
</tr>
</tbody>
</table>

*means and regression models included only those where recorded volume of alcohol drunk on usual occasion was >0
†includes all those with any mention of self-reported Maori ethnicity – the reference group in ratio calculations
‡includes all those in survey with no mention of Maori ethnicity
§regression coefficients were calculated using linear regression model with log transformation, for each sex and age group, adjusting for age as a continuous variable within age group, and for survey in summary estimates
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Antidepressant poisoning deaths in New Zealand for 2001

David Reith, John Fountain, Murray Tilyard and Rebecca McDowell

Abstract

Aim To compare the rates of death per volume of drug dispensed for antidepressants in New Zealand.

Methods Deaths from antidepressant poisonings were identified from the reports of coronial inquiries for New Zealand in 2001. Prescriptions for antidepressant medications were identified from the PharmHouse database from 1 January 2001 to 31 December 2001. The rates of deaths (95% CI) per prescription, tablet/capsule or defined daily dose were calculated for individual antidepressants and classes of antidepressant.

Results There were 200 poisoning deaths recorded in the database for New Zealand in 2001. Antidepressants were involved in 41 deaths, and death was attributed to an antidepressant in 23 cases. There were 5.52 (95% CI 3.85–7.68) deaths per 100 000 prescriptions for tricyclic antidepressants (TCAs) and 2.51 (1.57–3.79) deaths per 100 000 prescriptions for selective serotonin reuptake inhibitors (SSRIs). There was marked variability in rates of death per volume of drug dispensed between individual antidepressants.

Conclusions SSRIs have lower rates of death per volume of drug dispensed than TCAs and there is also variation in these rates within these classes of drugs. Toxicity in overdose should be considered when prescribing antidepressants.

Antidepressant medications have been associated with poisoning deaths due to the increased risk of suicide in depression and the toxicological profile of the older antidepressant medications, particularly the tricyclic antidepressants (TCAs). Within the group of antidepressant medications there is variability in the relative toxicity of individual agents, most marked in comparing the selective serotonin reuptake inhibitors (SSRIs) with the TCAs. However, even within these classes of drugs there appears to be variability in relative toxicity.

Despite the introduction of newer antidepressant medications, prescription rates for TCAs have not fallen to the degree that would be expected from the increased use of SSRIs. TCAs are relatively cheap and newer agents do not appear to offer any marked advances in efficacy. TCAs may also be effective at lower doses than those commonly used by prescribers. TCAs are also being increasingly used for indications other than depression. In this context, the continuing use of those TCAs that have greater toxicity in overdose would result in greater mortality, and therefore action is required by regulatory agencies, purchasers of medicines and individual prescribers to limit the use of those agents with greater relative toxicity.

Information about toxicity in overdose is not generally available when medications are first marketed, and such information is often not collected during post-marketing surveillance, where the emphasis is upon adverse events at normal dosing. However,
toxicity in overdose is particularly relevant with psychotropic medications. Identification of medicines with greater toxicity in overdose would enable prescribers to make more informed judgements of the risk–benefit profiles of their potential treatments.

This study aimed to compare the death rate per prescription, tablet and defined daily dose (DDD) for antidepressant medications used in New Zealand in the year 2001, separately for the overall exposure or for the primary agent involved.

Methods

Deaths from antidepressant poisonings were identified from the reports of coronial inquiries for New Zealand in 2001. The cases were identified from coronial chemical injury cases collected from the Coronal Services Office (CSO) in Wellington as of 13 January 2003. From previous experience there may be delay of over a year in the reporting of deaths from coroners and it is estimated that 90–95% of the poisoning deaths for 2001 were recorded by this date.6 Toxicology data were obtained from Institute of Environmental Science and Research (ESR) toxicology reports that were present in approximately 95% of the coroners’ files and, where the toxicology report was absent, the substances involved were extracted from the coroner’s report, the pathology report, police statement or the statement of family or friends. All the substances detected were recorded in the chemical injury database with the exceptions of ethanol (where the blood level was less than 20 mg/dl) and lignocaine (a drug commonly given in resuscitation). Intentional deaths (suicides and homicides) were separated from unintentional deaths according to the judgement of the coroner. The analysis included both intentional and accidental deaths. The primary substance involved in the fatality was determined by the coroners’ reports and the ESR reports.

Prescriptions for antidepressant medications were identified from the PharmHouse database from 1 January 2001 to 31 December 2001. The PharmHouse database is a subset of the New Zealand Health Information System database and contains records of all the claims for medicines dispensed within New Zealand. The records include the drug name, formulation and strength, the type of prescriber and the prescriber’s New Zealand Medical Council number. The data were imported into Stata® for data management to enable tabulation of the prescription numbers by drug type.7 Analyses were also performed using tablet or capsule numbers, and defined daily doses (DDD) as the denominators.8 These separate analyses were performed because the number of tablets required to treat a patient for a day varies considerably between medications, and the number of tablets per prescription also varies considerably. Rates and their 95% confidence intervals were calculated using the command ‘cii’ and the Poisson distribution in Stata®.

Results

As of 16 January 2003, there were 200 deaths due to chemical injury recorded for the year 2001 in New Zealand. There were 146 intentional and 54 unintentional deaths, and these occurred in 144 males and 56 females. There were 41 deaths involving antidepressant medications, and for 23 of these the antidepressant medication was the primary agent involved (Table 1). For the remaining deaths the primary agent was considered to be an alternative agent, such as carbon monoxide in three cases, dextropropoxyphene in three cases, ethylene glycol in two cases, and morphine in two cases. Individual antidepressants were involved in death on 57 occasions. On 29 occasions a single antidepressant was involved, on ten occasions two were involved and on two occasions four were involved.

For the corresponding time period there were 1 560 990 prescriptions for antidepressant medication dispensed in New Zealand. This represented a total of 38 472 206 DDDs, or sufficient antidepressants to treat 105 403 patients for one year (around 3% of the population of New Zealand). The most commonly prescribed
antidepressant medications in New Zealand were paroxetine, fluoxetine, amitriptyline, citalopram and dothiepin (Table 1).

### Table 1. Prescription volumes and poisoning deaths for antidepressants in New Zealand, 2001

<table>
<thead>
<tr>
<th>Antidepressants</th>
<th>P (n)</th>
<th>Deaths (C)</th>
<th>Deaths/100 000 P (95% CI)</th>
<th>Deaths (PA)</th>
<th>Deaths/100 000 P (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SSRIs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paroxetine HCl</td>
<td>408 757</td>
<td>6</td>
<td>1.47 (0.54–3.19)</td>
<td>1</td>
<td>0.24 (0.01–1.36)</td>
</tr>
<tr>
<td>Fluoxetine HCl</td>
<td>324 103</td>
<td>8</td>
<td>2.47 (1.07–4.86)</td>
<td>0</td>
<td>0 (0–1.14)</td>
</tr>
<tr>
<td>Citalopram</td>
<td>126 674</td>
<td>6</td>
<td>4.74 (1.74–10.31)</td>
<td>0</td>
<td>0 (0–2.91)</td>
</tr>
<tr>
<td>Nefazodone</td>
<td>18 474</td>
<td>1</td>
<td>5.41 (0.14–30.16)</td>
<td>0</td>
<td>0 (0–19.96)</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>63</td>
<td>1</td>
<td>1587.30 (40.19–8843.86)</td>
<td>0</td>
<td>0 (0–5853.37)</td>
</tr>
<tr>
<td>Sertraline HCl</td>
<td>17</td>
<td>0</td>
<td>0 (0–29 691.91)</td>
<td>0</td>
<td>0 (0–29 691.91)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>878 088</td>
<td>22</td>
<td>2.51 (1.57–3.79)</td>
<td>1</td>
<td>0.11 (0–0.63)</td>
</tr>
<tr>
<td><strong>TCAs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>240 753</td>
<td>10</td>
<td>4.15 (1.99–7.64)</td>
<td>5</td>
<td>2.08 (0.67–4.85)</td>
</tr>
<tr>
<td>Dothiepin HCl</td>
<td>106 415</td>
<td>12</td>
<td>11.28 (5.87–19.70)</td>
<td>7</td>
<td>6.58 (2.64–13.55)</td>
</tr>
<tr>
<td>Doxepin HCl</td>
<td>101 098</td>
<td>2</td>
<td>1.98 (0.24–7.14)</td>
<td>2</td>
<td>1.98 (0.24–7.14)</td>
</tr>
<tr>
<td>Nortriptyline HCl</td>
<td>82 912</td>
<td>5</td>
<td>6.03 (1.96–14.07)</td>
<td>5</td>
<td>6.03 (1.96–14.07)</td>
</tr>
<tr>
<td>Trimipramine</td>
<td>33 902</td>
<td>2</td>
<td>6.06 (0.73–21.88)</td>
<td>1</td>
<td>3.03 (0.08–16.88)</td>
</tr>
<tr>
<td>Imipramine HCl</td>
<td>27 914</td>
<td>1</td>
<td>3.58 (0.09–19.96)</td>
<td>1</td>
<td>3.58 (0.09–19.96)</td>
</tr>
<tr>
<td>Clomipramine HCl</td>
<td>27 719</td>
<td>1</td>
<td>3.61 (0.09–20.10)</td>
<td>1</td>
<td>3.61 (0.09–20.1)</td>
</tr>
<tr>
<td>Amoxapine</td>
<td>75 151</td>
<td>1</td>
<td>13.31 (0.34–74.14)</td>
<td>0</td>
<td>0 (0–49.07)</td>
</tr>
<tr>
<td>Maprotiline HCl</td>
<td>32 888</td>
<td>0</td>
<td>0 (0–112.25)</td>
<td>0</td>
<td>0 (0–112.25)</td>
</tr>
<tr>
<td>Desipramine HCl</td>
<td>26 515</td>
<td>1</td>
<td>37.72 (0.96–210.17)</td>
<td>0</td>
<td>0 (0–139.10)</td>
</tr>
<tr>
<td>Mianserin HCl</td>
<td>733</td>
<td>0</td>
<td>0 (0–503.09)</td>
<td>0</td>
<td>0 (0–503.09)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>634 000</td>
<td>35</td>
<td>5.52 (3.85–7.68)</td>
<td>22</td>
<td>3.47 (2.18–5.25)</td>
</tr>
<tr>
<td><strong>MAOIs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moclobemide</td>
<td>43 690</td>
<td>0</td>
<td>0 (0–8.44)</td>
<td>0</td>
<td>0 (0–8.44)</td>
</tr>
<tr>
<td>Phenelzine SO₄</td>
<td>2632</td>
<td>0</td>
<td>0 (0–140.11)</td>
<td>0</td>
<td>0 (0–140.11)</td>
</tr>
<tr>
<td>Tranylcypromine</td>
<td>2580</td>
<td>0</td>
<td>0 (0–142.93)</td>
<td>0</td>
<td>0 (0–142.93)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>48 902</td>
<td>0</td>
<td>0 (0–7.54)</td>
<td>0</td>
<td>0 (0–7.54)</td>
</tr>
</tbody>
</table>

P = prescriptions; C = combined; PA = primary agent; SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant; MAOI = monoamine oxidase inhibitor

The rate ratio (95% CI) for deaths per prescription for SSRIs versus TCAs was 0.45 (0.25–0.79) indicating a significantly decreased rate with SSRIs. The medications with the highest rates of death per volume of drug dispensed (as measured by deaths per 100 000 prescriptions) were venlafaxine, desipramine, amoxapine, dothiepin, nortriptyline and trimipramine (Table 1). However, the confidence intervals were extremely wide for the venlafaxine estimates, reflecting the low usage of this medication in New Zealand. When measured by numbers of tablets dispensed or by DDD the ranking was similar (Table 2), except that, when measured by numbers of tablets or capsules dispensed, citalopram and trimipramine had a greater rate of death than dothiepin and nortriptyline and, when measured by DDDs dispensed, dothiepin had a greater rate of death per DDD than amoxapine.
Table 2. Deaths per number of tablets/capsules prescribed and deaths per defined daily dose (DDD)

<table>
<thead>
<tr>
<th>Antidepressants</th>
<th>Deaths per 1 000 000 tablets/capsules</th>
<th>Deaths per 1 000 000 DDDs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SSRIs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paroxetine HCl</td>
<td>0.48 (0.18–1.04)</td>
<td>0.48 (0.18–1.04)</td>
</tr>
<tr>
<td>Fluoxetine HCl</td>
<td>0.78 (0.34–1.53)</td>
<td>0.78 (0.34–1.53)</td>
</tr>
<tr>
<td>Citalopram</td>
<td>1.46 (0.54–3.19)</td>
<td>1.46 (0.54–3.18)</td>
</tr>
<tr>
<td>Nefazodone</td>
<td>1.09 (0.03–6.1)</td>
<td>3.10 (0.08–17.3)</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>570.78 (14.50–3180)</td>
<td>761.04 (19.3–4240.20)</td>
</tr>
<tr>
<td>Sertraline HCl</td>
<td>0 (0–3078.2)</td>
<td>0 (0–2200.30)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>0.79 (0.50–1.20)</td>
<td>0.81 (0.51–1.22)</td>
</tr>
<tr>
<td><strong>TCAs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>0.84 (0.40–1.55)</td>
<td>2.98 (1.43–5.47)</td>
</tr>
<tr>
<td>Dothiepin HCl</td>
<td>1.14 (1.11–3.74)</td>
<td>8.53 (4.41–14.90)</td>
</tr>
<tr>
<td>Doxepin HCl</td>
<td>0.40 (0.05–1.43)</td>
<td>1.42 (0.17–5.12)</td>
</tr>
<tr>
<td>Nortriptyline HCl</td>
<td>0.96 (0.31–2.23)</td>
<td>3.32 (1.08–7.74)</td>
</tr>
<tr>
<td>Trimipramine maleate</td>
<td>1.17 (0.14–4.24)</td>
<td>5.08 (0.62–18.3)</td>
</tr>
<tr>
<td>Imipramine HCI</td>
<td>0.48 (0.01–2.66)</td>
<td>2.24 (0.06–12.5)</td>
</tr>
<tr>
<td>Clomipramine HCl</td>
<td>0.43 (0.01–2.41)</td>
<td>1.96 (0.05–10.10)</td>
</tr>
<tr>
<td>Amoxapine</td>
<td>2.56 (0.06–14.20)</td>
<td>8.02 (0.20–44.70)</td>
</tr>
<tr>
<td>Maprotiline HCl</td>
<td>0 (0–20.50)</td>
<td>0 (0–60.20)</td>
</tr>
<tr>
<td>Desipramine HCl</td>
<td>4.22 (0.11–23.50)</td>
<td>16.88 (0.43–94)</td>
</tr>
<tr>
<td>Mianserin HCl</td>
<td>0 (0–80.60)</td>
<td>0 (0–161.30)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1.01 (0.70–1.40)</td>
<td>3.76 (2.62–5.23)</td>
</tr>
<tr>
<td><strong>MAOIs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moclobemide</td>
<td>0 (0–1.24)</td>
<td>0 (0–2.21)</td>
</tr>
<tr>
<td>Phenelzine SO₄</td>
<td>0 (0–16.80)</td>
<td>0 (0–67.10)</td>
</tr>
<tr>
<td>Tranylcypromine SO₄</td>
<td>0 (0–16.50)</td>
<td>0 (0–16.50)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>0 (0–1.08)</td>
<td>0 (0–1.90)</td>
</tr>
</tbody>
</table>

SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant; MAOI = monoamine oxidase inhibitor

Discussion

The present study is an example of an ‘ecological’ study whereby characteristics of an overall population, in this case numbers of deaths in relation to numbers of prescriptions, are compared but the data cannot be related back to an individual. The main problem with this study design is that it is not possible to correct for confounders, such as degree of depression or co-medication, in the statistical analysis. On many occasions more than one antidepressant was ingested and for the multiple-agent deaths it is difficult to attribute causality to an individual agent. The indications for prescribing were unknown and may represent use for indications other than depression. Factors such as the length of time a patient was medicated and their compliance with medication were not available in the database. In addition, the late reporting of deaths could introduce some bias, although there is no indication that any systematic bias in reporting would occur. Such bias would therefore decrease the precision of our estimates of the rate of death for each medicine. The major advantage of the design is that the data are reliable, compared with other data sources, and cover an entire country. Also, when prescription volumes are used as the denominator, an assessment of relative fatal toxicity can be performed.
The main finding of the present study is that SSRIs have a lower rate of death per volume of drug dispensed than TCAs for both single-agent ingestions and multiple-agent ingestions. This is consistent with previous reports in the literature for the United Kingdom, but there are limited supporting data from other populations. Much of the information from the United Kingdom has been obtained from re-analysis of the same population, often including the same data. The rates of death per volume of drug dispensed calculated in the present study are similar to those calculated in the previous studies and, taken together, strengthen the conclusion that SSRIs are less toxic in overdose than TCAs.

The present study did not examine suicidal deaths by poisons other than antidepressants, or by means other than poisoning. It is possible that SSRIs may still be associated with an increased risk of suicide (by means other than SSRI poisoning) or of self-harm. However, it is also possible that prescribers may be treating patients with higher suicidality with SSRIs in preference to TCAs because of a perception of lesser inherent toxicity in overdose. This would bias cohort studies of suicide and self-harm in antidepressant users against SSRIs.

Differences between the TCAs in rates of death per volume of drug dispensed are well described in the literature with amoxapine, viloxazine, desipramine and dothiepin having greater toxicity than other TCAs. The toxicity of individual tricyclic antidepressants appears to relate to their individual cardiotoxicity and potency as GABA\_A antagonists rather than their relative potencies as noradrenergic or serotoninergic reuptake inhibitors. In the context of overdose, TCA toxicity is also related to the potential to induce seizures and arrhythmias rather than noradrenergic or serotoninergic reuptake inhibition. Hence, the use of TCAs with lesser toxicity would not be expected to result in reduced efficacy.

The toxic potential of SSRIs relates to their ability to induce the serotonin syndrome and seizures in overdose. There is limited evidence that some SSRIs, particularly venlafaxine, have greater toxicity in overdose than others. Unlike the TCAs, this greater potential may relate to greater potency and clinical efficacy. Clearly, a more detailed analysis is required to determine whether increased toxicity is offset by greater efficacy with the SSRIs.

The ingestion of SSRIs in combination with other drugs may lead to a more severe clinical presentation, and greater risk of death, than ingestion of SSRIs alone. The implication being that the examination of rates of death per volume of drug dispensed for multiple ingestion is relevant, in addition to examination of single-agent rates of death per volume of drug dispensed. The difficulty lies in identifying which medication has the greater risk for death. In the absence of large-scale cohort studies, it is not possible to correct for confounders such as suicidality or co-ingestion, and analysis of rates of death per volume of drug dispensed represents the best method for comparing the clinical toxicity of different drugs.

It is possible that choice of antidepressant medication may be influenced by their perceived characteristics. Patients more at risk of suicide may be prescribed particular medications in order to achieve a more rapid response. This would result in over-expression of these agents in suicides. The present study, and also previous studies reporting rates of death per volume of drug dispensed, do not correct for severity of depression, or suicidality, hence it is not possible to correct for this bias.
However, the results of the studies are consistent, both within and between different countries, and this would suggest greater risk of fatal toxicity with particular medications.

The differences between the antidepressants in rate of death are accentuated when comparisons are made using DDDs and reduced when comparisons are made using numbers of tablets or capsules dispensed. This can be explained by the fact that SSRIs require fewer tablets or capsules to make up a DDD. It is possible that the perception of SSRIs as having lesser toxicity has resulted in a greater number of DDDs being provided per prescription. In addition, their daily dosing has been developed as one rather than several tablets or capsules per day. The implication is that when antidepressants are compared on the basis of equivalent therapeutic potency, the differences in toxicity become even more apparent.

Overall, the death rate from antidepressant poisoning in New Zealand is low but could be further reduced by restricting the availability of the more toxic drugs within classes. It has been previously demonstrated that limiting the availability of medications with greater toxicity may result in a reduction in overall mortality. Limitation of pack sizes has also contributed to a reduction in ingested dose in self-poisoning. The factors influencing the availability of antidepressants should include safety in addition to efficacy, tolerability and economic considerations.

**Author information:** David Reith, Senior Lecturer, Department of Paediatrics and Child Health, Dunedin School of Medicine; John Fountain, Medical Toxicologist, New Zealand National Poisons Centre; Murray Tilyard, Elaine Gurr Professor of General Practice, Department of General Practice, Dunedin School of Medicine, University of Otago, Dunedin; Rebecca McDowell, Health Information Analyst, Population and Environmental Health, Institute of Environmental Science & Research, Porirua

**Acknowledgements:** We thank Justine Broadley of the Best Practice Advisory Centre.

**Correspondence:** Dr David Reith, Senior Lecturer, Dunedin School of Medicine, 3rd Floor Children’s Pavilion, Dunedin Hospital, Great King Street, Dunedin. Fax: (03) 474 7817; email: david.reith@stonebow.otago.ac.nz

**References:**


Epidemiology of slipped capital femoral epiphysis in a population with a high proportion of New Zealand Maori and Pacific children

Susan Stott and Terri Bidwell

Abstract

Aim To describe the epidemiology of slipped capital femoral epiphysis in NZ Maori and Pacific children residing in Auckland compared with NZ European children.

Methods The charts and radiographs of 211 children admitted with 307 slipped capital femoral epiphyses to Starship Children’s Hospital between 1988 and 2000 were reviewed.

Results The average age at first presentation was 132.6 +/-16.7 months in girls (range 95 to 170 months) and 149.5 +/- 19.3 months in boys (range 99 to 190 months), p <0.05. The age at presentation was not statistically different between the three ethnic groups. One hundred and seventy one children (81%) presented with a unilateral slipped capital femoral epiphysis. Forty children presented with bilateral simultaneous slipped capital femoral epiphyses; however, after two years of follow up, a further 56 children had been readmitted for pinning of the opposite hip, giving an overall rate of bilateral hip pinning of 45.5%. The relative racial frequency of slipped capital femoral epiphysis in the New Zealand Maori and the Pacific population was 4.2 times and 5.6 times the New Zealand European population, respectively.

Conclusions Children as young as eight years are now presenting with slipped capital femoral epiphyses. General practitioners should be aware of the possibility of this diagnosis, particularly in children of NZ Maori or Pacific ethnicity.

Slipped capital femoral epiphysis is one of the most common adolescent hip disorders. It typically occurs within 18 months of growth plate closure with bilateral involvement in 22% to 61% of children. The aetiology of slipped capital femoral epiphysis is unknown but a number of factors such as femoral retroversion, oblique slope of physis and weight of the child may all contribute to a biomechanically weakened physis at a time of rapid growth.

The prevalence of slipped capital femoral epiphysis varies between races. The lowest prevalence is reported in the Japanese population at 0.7 per 100 000, with children of European descent in America having an intermediate prevalence of 3.19 per 100 000. Children who are of Polynesian or Maori descent have been reported to have the highest prevalence of slipped capital femoral epiphysis in the world. However, this information was based on a study of only 34 children, of whom only 28 were NZ Maori and six Australian Aboriginal.

This study investigates the prevalence of first presentations with slipped capital femoral epiphysis in a population with a high proportion of NZ Maori and Pacific children to more accurately determine the epidemiology of this condition in these ethnic groups.
Methods

Admission data from Starship Children’s Hospital were collected for the period 1988 to 2000 and analysed for all diagnoses of slipped capital femoral epiphysis. Readmissions for complications or metalware removal were excluded. Children from outside Auckland were also excluded from the data collection. The hospital database was cross-checked with the handwritten operating-room record logs to ensure that all patients were identified. Radiographs were also audited to ensure that only cases of slipped capital femoral epiphysis were analysed.

The data collected included gender, age at admission, side of slip and the self-identified ethnicity of the patient based on a review of case records and radiographs. The definition of ethnicity used was the same as that used by Statistics New Zealand. The relative racial frequencies of slipped capital femoral epiphysis were calculated according to the method described by Loder.

Results

There were 218 children admitted with slipped capital femoral epiphyses in the time period selected. Of these, 211 had radiographic follow up adequate for analysis.

Analysis by age and gender Table 1 summarises the data collected, including the age at presentation, ethnicity and involved hip/s. Fifty seven children were identified as NZ European, 60 were identified as NZ Maori, and 89 were identified as Pacific. Five came from other ethnic backgrounds, including Indian and Asian.

Table 1. Average age at presentation and bilaterality by ethnic group and gender

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Total n</th>
<th>Average age (months)</th>
<th>Unilateral slips</th>
<th>Bilateral slips</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total Left hip</td>
<td>Right hip</td>
</tr>
<tr>
<td>NZ European</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>27</td>
<td>139.1 +/-14.0</td>
<td>13</td>
<td>10</td>
</tr>
<tr>
<td>Male</td>
<td>30</td>
<td>157.2 +/-16.1</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>NZ Maori</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>27</td>
<td>128.3 +/-16.5</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>Male</td>
<td>33</td>
<td>146.1 +/-18.4</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>Pacific</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>40</td>
<td>131 +/-17.5</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>Male</td>
<td>49</td>
<td>146.9 +/-20.6</td>
<td>27</td>
<td>17</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
<td>129</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Male</td>
<td>3</td>
<td>151</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>96</td>
<td>132.6 +/-16.7</td>
<td>51</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>115</td>
<td>149.5 +/-19.3</td>
<td>64</td>
<td>42</td>
</tr>
</tbody>
</table>

The average age at first presentation with a slipped capital femoral epiphysis was 149.5 +/-19.3 months in males (range 99 to 190 months) and 132.6 +/-16.7 months in females (95 to 170 months). These data are shown graphically in Figures 1 and 2. Statistical analysis of the age at first presentation for the different ethnic and gender groupings showed statistically significant differences between the groups at p <0.0001 (one-way ANOVA). A Tukey-Kramer multiple comparisons test confirmed significant differences in age at presentation between NZ European females and males (p <0.01), NZ Maori females and males (p <0.01) and Pacific females and males (p <0.001). NZ European females were an average of 18.1 months younger than NZ
European males (95% CI 4.5–31.7 months). NZ Maori females were on average 17.9 months younger than NZ Maori males (95% CI 4.5–31.3 months), and Pacific females were on average 15.8 months younger than Pacific males (95% CI 4.9–26.8 months). However, the age at presentation did not vary significantly between the three ethnic groups, for either males or females.

Figure 1. Age at first presentation with slipped capital femoral epiphysis for males of NZ European, NZ Maori and Pacific ethnicity

Figure 2. Age at first presentation with slipped capital femoral epiphysis for females of NZ European, NZ Maori and Pacific ethnicity
Analysis by site of slip Left-sided slipped capital femoral epiphyses were twice as common as right-sided slipped capital femoral epiphyses. The incidence of left- and right-sided slipped capital femoral epiphyses did not vary significantly between the different ethnic groups or between the genders. Forty children had bilateral slipped capital femoral epiphyses at the time of first presentation and 171 had unilateral slipped capital femoral epiphyses. However, two years after initial presentation, a further 56 children with unilateral slipped capital femoral epiphyses had been readmitted for pinning of the opposite hip, leading to an overall 45.5% bilaterality rate in the short term. A higher percentage of NZ Maori and Pacific children presented with bilateral slipped capital femoral epiphyses at their first presentation than did NZ European children. However, more European children presented with sequential slipped capital femoral epiphyses, leading to a similar bilaterality rate at two years. The rate of presentation with a second slipped capital femoral epiphysis showed an exponential decay (Figure 3), with a $t_{1/2}$ of 5.75 months ($r^2 = 0.99$). Of all second slipped capital femoral epiphyses, 77% presented within 12 months of the first slipped capital femoral epiphysis.

Figure 3. Time between the diagnosis of the first and second slipped capital femoral epiphyses for the 56 bilateral slipped capital femoral epiphyses with a sequential presentation. The number of hips that became bilateral every six months (mo) after the diagnosis of the first hip follows an exponential decay pattern: number of hips = $62.53.e^{-0.12 \times mo}$ ($r^2 = 0.99$).

Relative racial frequency All but 27 of the 211 children admitted to Starship Children’s Hospital with a slipped capital femoral epiphysis during 1988 to 2000

NZMJ 24 October 2003, Vol 116 No 1184
came from the north, central and west urban zones in Auckland, and were aged between 60 and 179 months. The ethnicity data for these 184 children and the 1991 and 1996 Census data for ethnicity of children of the same age from the same parts of Auckland (Table 2) were used to calculate the relative racial frequency of slipped capital femoral epiphysis.

**Table 2. Census figures for 1991 and 1996: children aged 5–14 years in north, central and west urban zones of Auckland**

<table>
<thead>
<tr>
<th></th>
<th>1991 n (%)</th>
<th>1996 n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NZ European</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>26 067 (64.5)</td>
<td>26 349 (56.8)</td>
</tr>
<tr>
<td>Female</td>
<td>25 446 (64.4)</td>
<td>25 350 (56.7)</td>
</tr>
<tr>
<td><strong>NZ Maori</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>5349 (13.2)</td>
<td>6810 (14.7)</td>
</tr>
<tr>
<td>Female</td>
<td>5280 (13.4)</td>
<td>6843 (15.3)</td>
</tr>
<tr>
<td><strong>Pacific</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>5880 (14.5)</td>
<td>6711 (14.5)</td>
</tr>
<tr>
<td>Female</td>
<td>5682 (14.4)</td>
<td>6414 (14.4)</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3096 (7.7)</td>
<td>6516 (14.1)</td>
</tr>
<tr>
<td>Female</td>
<td>2949 (7.4)</td>
<td>6135 (13.7)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>40 392</td>
<td>46 386</td>
</tr>
<tr>
<td>Female</td>
<td>39 357</td>
<td>44 742</td>
</tr>
</tbody>
</table>

During the study period, the percentage of children of Maori (14%) or Pacific (14.5%) ethnicity remained the same in the population at risk. The relative percentage of NZ European decreased from 64% to 57% largely due to an increase in children from other racial groups, predominantly Asian. Based on these figures, if the presence of slipped capital femoral epiphysis was equally distributed between all ethnicities, we would expect that, over the period 1988 to 2000, approximately 60% of the slipped capital femoral epiphyses would occur in NZ Europeans, 14% in NZ Maori and 14.5% in Pacific children. However, children identified as NZ European made up only 29% of all admissions for slipped capital femoral epiphysis, while 40% of all children admitted for slipped capital femoral epiphysis had Pacific Island ethnicity. Based on the relative population ratios, children of NZ Maori ethnicity were 4.2 times more likely to be admitted to Starship Children’s Hospital with a slipped capital femoral epiphysis than NZ European children in the same age group. The relative racial frequency was higher in Pacific children, who were 5.6 times more likely to be admitted to Starship Children’s Hospital with a slipped capital femoral epiphysis than NZ European children in the same age group (Figure 4).
Figure 4. Relative percentages of NZ European, NZ Maori and Pacific children, aged 60 to 179 months, in north, central and west urban zones of Auckland (based on 1991 and 1996 Census data) compared with relative percentages of children of the same ethnicity, age and residence who were admitted with a slipped capital femoral epiphysis (SCFE) to Starship Children’s Hospital from 1988 to 2000.

Discussion

Relative racial frequencies are a way to estimate the frequency of a condition in a subgroup of the population when the exact denominator, the population numbers, cannot be defined with precision. Our data suggest that NZ Maori and Pacific children have a frequency of slipped capital femoral epiphysis that is 3 to 5 times greater than that of NZ European children. These figures can only be an estimate, due to difficulties in interpretation of self-identified ethnicity and also in determining the ethnic characteristics of the local population. However, the results do support the anecdotal impression of an increased prevalence of slipped capital femoral epiphysis in the NZ Maori and Pacific population.2

The predisposition of NZ Maori and Pacific children to slipped capital femoral epiphysis may be related to the tendency of such children to be larger and physically more developed than their NZ European counterparts at the same age, placing greater stresses on a susceptible growth plate. In New Zealand, the mean body weight and height of Pacific Island children aged 5–12 years is close to the 95 percentile of the National Center for Health Statistics (USA) standards for height and weight.7 Both males and females reach their maximum height one to two years earlier than the USA standard population, a pattern typical of early maturation. As this study is retrospective, we could not determine the body mass index8 or bone age for individual children with a slipped capital femoral epiphysis. Collection of such data prospectively could help determine whether the ethnic variation in prevalence of slipped capital femoral epiphysis found in this study is a reflection of differences in obesity or different patterns of maturation in the three ethnic groups.
The age at first presentation with a slipped capital femoral epiphysis was 11.5 years in females and 12.5 years in males. These figures are one year lower than those reported for the American children of European descent in the study by Loder. At Starship Children’s Hospital, we are now seeing slipped capital femoral epiphyses in children as young as eight or nine years of age without any underlying endocrine problem. Body weight has been inversely correlated to the age at presentation with a slipped capital femoral epiphysis and may be a contributor to the earlier presentation in our patient population. Obesity in childhood is increasing in Auckland and is a greater problem in NZ Maori and Pacific Island children than NZ European children. The relationship between increasing childhood obesity and the incidence of slipped capital femoral epiphysis in New Zealand is not known but must be of concern.

The gender ratio of slipped capital femoral epiphysis in the Auckland population is close to 1:1, with a slight male predominance. The male predominance in slipped capital femoral epiphysis has decreased over the twentieth century. Data from Sweden show that at the turn of the twentieth century, 88% of slipped capital femoral epiphyses were in males. However, by the 1970–1980s, the percentage of males had decreased to only 60%. In the study by Loder, the male predominance persists in Indo-Mediterranean children and similar data have been reported in Japan, where slipped capital femoral epiphysis is very rare. However, in other populations, the ratio is closer to 1:1 male to female.

The overall short-term incidence of bilateral slipped capital femoral epiphysis in our population was higher than that reported previously and did not vary across the racial groups or with gender. Half of all second slipped capital femoral epiphyses had occurred by six months, with the earliest presentation being within one month of the previous surgery. Thus, there must be a high suspicion of a second slipped capital femoral epiphysis when a child with a unilateral slipped capital femoral epiphysis presents with pain in the opposite hip within a few months of hip pinning. Although children of Maori or Pacific descent in our study had the same incidence of bilaterality as NZ Europeans, they were more likely to present with bilateral slipped capital femoral epiphyses and less likely to develop sequential slipped capital femoral epiphyses. Only a small percentage of NZ Europeans presented with bilateral hip involvement but more had a sequential slipped capital femoral epiphysis. The explanation for this difference is not clear from this retrospective study but may reflect differences in access to healthcare or in parental awareness of the condition.

Author information: N Susan Stott, Associate Professor of Paediatric Orthopaedic Surgery, Discipline of Orthopaedics, Faculty of Medicine and Health Sciences, University of Auckland, Auckland; Terri A Bidwell, Senior Orthopaedic Registrar, Starship Children’s Hospital, Auckland

Acknowledgements: We thank Joy O’Connell (Statistics New Zealand analyst) who provided the census data for the different ethnic groups in the age range 5 to 14 years in north, central and west urban zones of Auckland.

Correspondence: Associate Professor N S Stott, Discipline of Orthopaedics, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 367 7159; email: s.stott@auckland.ac.nz
References:


The Auckland Breast Cancer Register: a special project of the Auckland Breast Cancer Study Group

Lorraine Neave, Vernon Harvey, Chelleraj Benjamin, Paul Thompson, Ora Pellett, Jeremy Whitlock, Wayne Jones, and Garth Poole

Abstract

Aims The Auckland Breast Cancer Register (ABCR) has been established in response to the need for a comprehensive database of breast cancer cases from the Auckland area.

Methods The database records patient demographics, diagnosis, treatment options, prognosis and long-term outcome (annual follow up). Data from 1204 cases, recorded between June 2000 and June 2002 are reported.

Results The major findings are that 34% of women had breast cancer detected by screening only (47% in the group eligible for free screening within the Breast Screen Aotearoa screening programme); 84% of patients had invasive carcinoma; 13% had ductal carcinoma in situ (DCIS); and 3% fine needle aspiration only. Forty nine per cent of invasive tumours were =2cm. Grade 3 tumours were found in 53% of patients under 40 years old compared with 26.8% 40 years or older. Mastectomy was performed in 56% of patients with invasive cancer and 33% of those with DCIS. Axillary surgery was performed in 94% of patients with invasive cancer and 39% had involved nodes. Seventy nine per cent of patients were referred for an opinion from an oncologist. Radiotherapy was given to 77% of these patients, chemotherapy to 33%, and hormone therapy to 57%.

Conclusions The ABCR will provide essential healthcare information that will lead to better understanding of breast cancer in Auckland and more effective delivery of the clinical resources available in the Auckland region.

The Auckland Breast Cancer Study Group (ABCSG), established in 1976, brought together a multidisciplinary group of clinicians with a particular interest in breast cancer management and research. As such, the membership includes representatives from both the public and private sectors in the fields of radiology, surgery, pathology, breast-care nursing, medical and radiation oncology, and biostatistics.

Between 1976 and 1985 the study group established a comprehensive database of 2700 cases of breast cancer in the Auckland region. This computerised database, with continued follow up, has provided important information on the incidence, pattern and management of breast cancer in a mixed ethnicity community and it has provided the resource for some 30 publications. The register was discontinued in 1985 following concerns expressed over privacy issues.

In 1996 the members of ABCSG agreed unanimously that a new breast cancer register should be established throughout the Auckland region. Against a background of important advances in all areas of breast cancer, including genetics, detection,
conservative surgery and chemo/endocrine adjuvant therapy, there was a need for a new, comprehensive database as a resource for ongoing audit and research.

In New Zealand there is clearly a need for data on breast cancer incidence, and analysis of survival by multiple presenting factors including clinical stage of disease. The Auckland region, particularly, presents a unique opportunity to accrue the details of clinical presentation and management in Maori, Pacific Island, and other ethnic groups, which will lead to a better understanding of why outcomes are worse in some groups than others. Information about current practice plays a key role in supporting evidence-based care, and allows multidisciplinary teams to provide high-quality care. Each of the specialty groups within the team uses internationally recognised guidelines and protocols to provide high-quality care. The ABCSG is not attempting to establish guidelines or recommended practice documents.

The Auckland region, particularly, presents a unique opportunity to accrue the details of clinical presentation and management in Maori, Pacific Island, and other ethnic groups, which will lead to a better understanding of why outcomes are worse in some groups than others. Information about current practice plays a key role in supporting evidence-based care, and allows multidisciplinary teams to provide high-quality care. Each of the specialty groups within the team uses internationally recognised guidelines and protocols to provide high-quality care. The ABCSG is not attempting to establish guidelines or recommended practice documents.

The group has links to the Australia–New Zealand Breast Cancer Trials Group and the Swiss-based International Breast Cancer Study Group. It has worked for almost 20 years with both these organisations in the promotion and data management of a range of ethically approved clinical trials in both early and advanced breast cancers. The Secretariat for ABCSG is situated in the Oncology Department at Auckland Hospital but is independently administered and funded solely by charitable donations.

A subcommittee of the ABCSG met regularly to determine aims and develop a new breast cancer register. In June 2000 the Auckland Breast Cancer Register (ABCR) commenced accrual having received approval from the Auckland Ethics Committee. The Register was also declared a Quality Assurance Activity under Part VI of the Medical Practitioners Act 1995.

The aims of the Register are to collect in a timely, accurate and confidential manner a predetermined set of data. These data will:

1. document the patients being diagnosed and treated;
2. determine risk factors and prognostic variables for disease relapse;
3. update individual patient progress annually to assess recurrence-free and overall survival;
4. allow review of the patterns of care and the multidisciplinary aspects of breast cancer management;
5. allow review of defined patient groups and their outcomes compared with predicted outcomes;
6. allow appropriate comparative analysis with other similar overseas studies;
7. allow comparison of patient outcomes within and outside trials to assess how representative of the overall population outcomes in trials are;
8. direct further research.

**Methods**

All patients who are New Zealand residents residing in the greater Auckland region and have a diagnosis of breast cancer after 1 June 2000 are eligible to be on the Register. These patients are identified by pathology reports sent from the National Cancer Registry. All clinicians involved with the care of patients with breast cancer were invited to participate. Participating clinicians agreed to approach all their patients presenting with newly diagnosed breast cancer and provide them with a
patient information sheet and consent form. Detailed information on the initial diagnosis and treatment is recorded, then follow-up forms are sent annually to the clinician and information on the diagnosis and treatment of any loco-regional and or metastatic disease is collected. Confidentiality of the information collected for the Register is maintained at all times. A summary of aggregated data is generated annually and the data will be analysed and the results offered for publication in peer-reviewed journals.

Results

Patient accrual Data accrued between June 2000 and June 2002 are summarised in this article. Of 1497 patients identified as eligible, 1204 (including 10 men, 0.83%), have given consent to be registered. Eighteen (1.2%) patients refused consent and 18 patients died before consent could be sought. The remaining 257 (17.2%) patients were not approached by their clinicians.

These figures demonstrate that approximately 80% of all the cases of breast cancer diagnosed in the Auckland region between June 2000 and June 2002 are represented on the ABCR database. All clinicians, public and private, who treat breast cancer cases in the Auckland region have patients registered on the ABCR. However, the main limiting factor for 100% representation is the requirement from the Health Information Privacy Code 1994 to seek individual informed consent. Those patients easily treated ‘disappear’ from the system very quickly, thus their consent becomes difficult or impossible to obtain.

Identified breast cancer cases were distributed between public (60.6%) and private services (39.4%). These figures are based upon initial diagnosis and surgical treatment because some treatment options, such as radiotherapy, are offered only within the public service and therefore many patients are treated by both public and private sectors.

Age distribution At the time of diagnosis, 71.9% of the patients were aged 50 and over, with 28.1% being over 65 years old (Table 1). It is of interest to note that seven of the ten male patients were over 70.

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Under 40</td>
<td>86</td>
</tr>
<tr>
<td>40–49</td>
<td>252</td>
</tr>
<tr>
<td>50–64</td>
<td>528</td>
</tr>
<tr>
<td>65–69</td>
<td>94</td>
</tr>
<tr>
<td>70–79</td>
<td>156</td>
</tr>
<tr>
<td>80+</td>
<td>88</td>
</tr>
<tr>
<td>Total</td>
<td>1204</td>
</tr>
</tbody>
</table>

Ethnicity Ethnicity was determined from the National Health Index (NHI). The majority of patients identified themselves as European (62.0%), with 5.3% identifying as NZ Maori, 5.4% Pacific Island, and 4.2% Asian. For 23.1% of patients ethnicity was indicated as ‘Other’ or ‘Not stated’.
Family history One hundred and forty eight (12.3%) patients gave a history of a first-degree relative (mother, sister or daughter) who had breast cancer; of these, 13 (8.8%) patients reported a second-degree relative also. Twenty one (1.7%) patients had more than one first-degree relative with breast cancer.

Clinical presentation Data for clinical presentation were available for 1181 (98.1%) cases. Six hundred and ninety four women (58.8%) presented with clinical signs or symptoms, such as a lump, pain, nipple change or skin abnormality, while 487 (41.2%) patients had a screen-detected cancer. However, 83 (17.0%) of these screen-detected patients were also found to have a clinically evident abnormality, and some may have attended screening because of this. It is of interest to note that 289 (24.5%) women had undergone a previous mammogram. However, of the 509 women in the age group eligible for inclusion in the Breast Screen Aotearoa programme, only 169 (33.2%) had undergone a previous mammogram. Detection rate by age group is seen in Table 2.

Table 2. Age groups of patients presenting with clinically evident or screen-detected breast cancer (n = 1181)

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Screen detection alone n (%)</th>
<th>Clinical detection alone n (%)</th>
<th>Screen detection and clinical evidence n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 40</td>
<td>5 (0.4)</td>
<td>78 (6.6)</td>
<td>1 (0.1)</td>
</tr>
<tr>
<td>40–49</td>
<td>54 (4.6)</td>
<td>177 (16.3)</td>
<td>14 (1.2)</td>
</tr>
<tr>
<td>50–64</td>
<td>274 (23.2)</td>
<td>192 (16.2)</td>
<td>52 (4.4)</td>
</tr>
<tr>
<td>65+</td>
<td>71 (6.0)</td>
<td>247 (20.9)</td>
<td>16 (1.4)</td>
</tr>
<tr>
<td>Total</td>
<td>404 (34.2)</td>
<td>694 (58.8)</td>
<td>83 (7.0)</td>
</tr>
</tbody>
</table>

Radiology Mammographic findings were analysed for a total of 1135 patients, with 1042 (91.8%) having mammographic features of carcinoma. Breast ultrasound was performed for 922 patients, with 821 (89.0%) having ultrasound features of carcinoma.

Of 910 patients on whom both mammography and breast ultrasound were performed, 877 (96.4%) had malignant lesions detected by either mammography, ultrasound or both, with only 33 cases (3.6%) not identified by either of these imaging techniques.

Definitive diagnosis Data for definitive diagnosis were available for 1185 (98.4%) patients. A definitive diagnosis was confirmed by core biopsy alone in 632 (53.3%) patients and fine needle aspiration (FNA) alone in 292 (24.6%). A small number of patients (181, 15.3%) required both FNA and core biopsy for confirmation and a further 80 (6.7%) patients had an excision biopsy for definitive diagnosis. The procedure used for core biopsy was described in 67.3% of cases; of these, 77.9% were ultrasound-guided biopsies.

Type of cancer Invasive carcinoma with or without an in situ component was diagnosed in 1014 (84.2%) of the 1204 cases, ductal carcinoma in situ (DCIS) alone was diagnosed in 154 (12.8%) cases. The other 36 (3.0%) patients had an FNA to determine a malignant breast carcinoma, but did not proceed with further surgical intervention because of comorbidity condition or patient refusal to undergo surgery.
**Tumour size (TStage)** Tumour size, as reported from the pathology results, is shown in Table 3. In 48.7% of patients with invasive cancer the tumour was ≤2 cm. Patients with TX staging had neo-adjuvant therapy, therefore true tumour size could not be assessed, or declined surgery (Table 3).

**Table 3. Tumour size (TStage) in Auckland Breast Cancer Registry patients, June 2000 to June 2003**

<table>
<thead>
<tr>
<th>TStage</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Tis (DCIS)</td>
<td>151</td>
</tr>
<tr>
<td>T1 (≤2.0 cm)</td>
<td>586</td>
</tr>
<tr>
<td>T2 (2.1-5.0 cm)</td>
<td>325</td>
</tr>
<tr>
<td>T3 ≥5.0 cm</td>
<td>63</td>
</tr>
<tr>
<td>T4</td>
<td>13</td>
</tr>
<tr>
<td>TX</td>
<td>66</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1204</td>
</tr>
</tbody>
</table>

**Grade of cancer** Tumour grade was reported for 967 (97.4%) of 993 surgical patients with invasive tumours. Grade 1 tumour was diagnosed in 205 (21.2%) patients, grade 2 in 483 (49.9%) patients, and grade 3 in 279 (28.9%) patients. However, 53% of patients under 40 presented with grade 3 tumours compared with 26.8% of patients over 40 (Figure 1).

**Figure 1. Percentage of patients in each age group with a grade 1, 2 or 3 tumour (data labels = number of patients)**

**Nottingham Prognostic Index (NPI)** The NPI, a model of prognosis developed from tumour size, grade and nodal involvement (tumour size (cm) x 0.2 + grade + nodes (0 = 1, 1–3 = 2, ≥4 = 3)) could be calculated for 905 (89.3%) of 1014 patients.
diagnosed with invasive breast cancer. Patients for whom NPI could not be calculated had no surgical intervention, did not have an axillary dissection, or had neo-adjuvant treatment (chemotherapy/hormone therapy and/or radiotherapy prior to surgical intervention) (Table 4).

Table 4. Range of Nottingham Prognostic Index (NPI) scores for patients with invasive breast cancer

<table>
<thead>
<tr>
<th>NPI range</th>
<th>Patients</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not known</td>
<td></td>
<td>109</td>
<td>10.7</td>
</tr>
<tr>
<td>=2.40</td>
<td></td>
<td>124</td>
<td>12.2</td>
</tr>
<tr>
<td>2.41–3.40</td>
<td></td>
<td>219</td>
<td>21.6</td>
</tr>
<tr>
<td>3.41–4.40</td>
<td></td>
<td>207</td>
<td>20.4</td>
</tr>
<tr>
<td>4.41–5.39</td>
<td></td>
<td>200</td>
<td>19.7</td>
</tr>
<tr>
<td>&gt;5.40</td>
<td></td>
<td>155</td>
<td>15.3</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>1014</td>
<td>100.0</td>
</tr>
</tbody>
</table>

**Hormone receptor status** The pathologist reported receptor status for 962 (79.9%) patients. Oestrogen-positive tumours were found in 722 (75.1%) patients and progesterone-positive tumours in 640 (66.5%) patients. Both receptors were positive for 606 (63.0%) patients. One hundred and sixty five patients were also tested for Her2 status, and 52 patients were status 2+ or 3+ using immunostaining techniques.

**Figure 2. Type of definitive surgery by type of cancer (n = 1204)**

Type of definitive surgery by type of cancer Five hundred and fifty four patients (55.8%) with invasive cancer had mastectomy and 439 (44.2%) had a partial
mastectomy. In patients diagnosed with in situ cancer alone, 49 (32.7%) had mastectomy, 101 (67.3%) a partial mastectomy, but 28 (27.7%) of these patients had only a diagnostic lumpectomy and no further surgery. Sixty one patients did not have primary surgery (Figure 2).

**Breast reconstruction** One hundred and eleven women (18.4%) treated by mastectomy also chose to have breast reconstruction. Sixteen had an implant, 19 latissimus dorsi reconstruction, and 76 underwent a TRAM (transverse rectus abdominus myocutaneous) flap reconstruction. However, these data do not account for the number of women who may be on the waiting list for reconstruction.

**Axillary surgery** Axillary node dissection was performed for 934 (94.1%) of the 993 patients who underwent surgery for invasive cancer. Axillary nodes were involved in 367 (39.3%) patients and 567 (60.7%) patients had negative nodes. Of the 150 patients with in situ disease only, 28 (18.7%) had axillary surgery and none of these patients had nodal involvement.

**Oncology referral** Of the 1204 patients registered with the ABCR between June 2000 and June 2002, 949 (78.8%) were referred to a medical and/or radiation oncologist for consideration of local and/or systemic treatment. Of these, 846 (89.1%) patients had adjuvant treatment (Table 5), 36 (3.8%) had neo-adjuvant treatment alone, and 38 (4.0%) had both neo-adjuvant and adjuvant treatments. Adjuvant radiotherapy treatment was given to 81.3% of patients who had a partial mastectomy, and 35.0% who had mastectomy.

**Table 5. Adjuvant oncology treatment given to patients referred to medical and/or radiation oncologists (n = 846)**

<table>
<thead>
<tr>
<th>Adjuvant treatment</th>
<th>Patients n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiotherapy</td>
<td>650 (76.8)</td>
</tr>
<tr>
<td>Mastectomy</td>
<td>211 (35.0)</td>
</tr>
<tr>
<td>Partial mastectomy</td>
<td>439 (81.3)</td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>282 (33.3)</td>
</tr>
<tr>
<td>Hormone therapy</td>
<td>485 (57.3)</td>
</tr>
<tr>
<td>Chemotherapy and hormone therapy</td>
<td>148 (17.5)</td>
</tr>
</tbody>
</table>

**Recurrence and deaths** At this early stage of data collection, 102 (8.5%) patients have been diagnosed with a recurrence: 66 with metastatic disease, 18 with loco-regional recurrence, and 18 with both. Six patients had another primary breast cancer diagnosed in the contralateral breast.

A total of 65 patients have died, 40 from breast cancer and 25 from other causes (Table 6).
Table 6. Patient outcome within a maximum of two years from initial presentation

<table>
<thead>
<tr>
<th>Outcome</th>
<th>n</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loco-regional recurrence</td>
<td>18</td>
<td>9 within six months post-diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 less than six months post-diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>16 at presentation</td>
</tr>
<tr>
<td>Metastatic recurrence</td>
<td>66</td>
<td>13 within six months post-diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>37 less than six months post-diagnosis</td>
</tr>
<tr>
<td>Loco-regional and metastatic recurrence</td>
<td>18</td>
<td>7 within six months post-diagnosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11 less than six months post-diagnosis</td>
</tr>
<tr>
<td>Second primary breast cancer</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Deceased</td>
<td>65</td>
<td>40 breast cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>25 other causes</td>
</tr>
</tbody>
</table>

**Discussion**

Given that the data presented in this paper are from the first two years of data collection, detailed analyses would be premature. Of the 1497 patients identified as eligible for inclusion in the Register, only 18 (1.2%) patients have refused consent (8 of these refusing all treatment). We would hope that with continued development and acceptance of the Register this figure would fall, as would the number of patients who have not been asked to participate by their clinicians. We recognise that the data collection generates extra work, but believe that the need to document current practice justifies this.

Most women present with clinical symptoms, such as a lump. In the 50- to 64-year age group, when women are eligible for free screening through the Breast Screen Aotearoa programme, 62% of tumours were diagnosed at screening; however, 15.9% of these were also clinically evident. It appears that women and doctors may be using the screening programme for diagnostic mammography of clinical abnormalities. This will distort the screening figures. It is worrying that overall more than 50% of women are still presenting with clinical disease. There is clearly a need to increase recruitment to screening within the target group, particularly Maori and Polynesian women. Of the 509 women who would have been eligible for a breast screen since screening became free in 1999 only 169 (33.2%) had undergone a previous mammogram. However, these figures may be merely reflecting the relative newness of free breast screening. With greater public awareness and acceptance of the breast screen programme, it is hoped and expected that these figures will improve.

Mammography remains the primary screening technique in the diagnosis of breast cancer, with a mammographic detection rate of 92% in this patient group. This is comparable to detection rates reported in the literature. However, breast ultrasound, in conjunction with mammography, has become an integral part of the diagnostic work up for patients with clinical symptoms or mammographically detected abnormalities. Ultrasound was also the method of choice in 77.9% of patients who underwent image-guided needle biopsy.

Core biopsy, which allows differentiation between DCIS and invasive cancer, is clearly preferred over FNA for definitive diagnosis, as demonstrated in this patient...
Pleasingly, only a small number of patients required excision biopsy to confirm the diagnosis.

The pathological characteristics of the tumours at this early stage of the Register are in line with those reported in the literature. Morrow et al report that 92% of tumours were infiltrating ductal, or lobular, 50.4% T1, and 75% N0 (T1 and T2 tumours only were included in this study). In the present study, 18.5% of patients who underwent breast-conserving surgery for invasive cancer had nodal involvement, which is consistent with Morrow et al; however, 44.3% of patients requiring mastectomy had nodal involvement, which is high compared with other studies. There was also a trend for younger patients to present with more aggressive disease, with 53% of patients under 40 years old having a grade 3 tumour compared with 26.8% over 40 years.

The incidence of a family history of breast cancer was higher than that reported in the literature. However, these data are self-reported and may not reflect a true familial rate. It would be interesting to further investigate these reports, but privacy regulations makes this event unlikely in the near future (consent would have to be sought from every relative for their records to be reviewed).

According to the guidelines the rate of breast-conserving surgery (44.8%) might be considered low. However, these same guidelines would exclude 6.6% of the present population because of T3 and T4 tumours. In addition, some patients with T2 tumours may have been advised to have a mastectomy by their surgeons because the size of the tumour relative to the breast size may not have allowed clear margins to be obtained with an acceptable cosmetic result. Furthermore, the present study includes 11% of New Zealand Maori and Polynesian patients, who as a group seem to present with a more advanced stage of breast cancer than other ethnic groups.

Many women also choose to have a mastectomy even though breast-conserving surgery is feasible. Barriers to adjuvant treatment include transport difficulties, distance from radiotherapy unit, obligations at home, and fear of radiotherapy. Some women are also anxious about the possibility of recurrence even though it is now well accepted that the two forms of local treatment are equivalent for outcome. In addition, some women may opt for mastectomy and immediate reconstruction, instead of breast-conserving surgery.

Morrow et al report rates of breast-conserving surgery ranging from 54% in the Northeast and Pacific regions, to 32% in Southern and Midwest regions of the USA, with an overall rate of 42.6% which is lower than the present report. However, cases with T1 and T2 tumours only were included. There is growing evidence to suggest that the rate of mastectomy is dropping, albeit more slowly than guidelines recommend. A further decrease might be encouraged by targeting improved participation in screening programmes and improvement in information and education of both patients and physicians.

Referral to medical or radiation oncology for additional therapy is common and possibly reflects the widespread involvement of multidisciplinary groups in the identification of patients who may benefit from oncology treatment. Additionally, it may also reflect the high number of clinical research trials coordinated through the Oncology Department. We have not analysed oncology treatment practices or
outcome measures, as numbers for individual therapies remain small and follow up short.

Despite the brevity of the annual follow ups, 102 (8.5%) patients had further disease diagnosed within two years of initial presentation. Sixteen of these patients had metastatic disease diagnosed at presentation and nine patients had a loco-regional recurrence diagnosed within six months of initial diagnosis. The latter may in part represent progression of undiagnosed primary disease rather than recurrence.

The database is providing detailed information on breast cancer in Auckland for the first time since the previous database had to be discontinued in 1985. The rapid acceptance of the Register has already led to an expanding workload. After only two years staffing requirements for documentation have increased from 0.5 FTE to 2.0 FTE and are anticipated to grow further. While this has significant funding implications we remain convinced that the value of the Register will justify the costs. Once established, wider coverage might be considered.

All members of the ABCSG are directly involved in the diagnosis and treatment of breast cancer patients, and the ABCR was established so that the best possible outcomes could be achieved for individual patients. The value of cancer registries and high-quality audit or surveillance in cancer control is well documented. To fully achieve the aims of the ABCR, 100% accrual is necessary, but, as stated earlier, the primary limiting factor is the requirement for individual informed consent. Even though all the participating clinicians have the highest regard for patient privacy and confidentiality, it is recognised that there are situations in which patients can not be approached for their individual informed consent and indeed in many audit tools such consent is not required.

The ABCSG is currently trying to address this issue and has made an application to the Auckland Ethics Committee to review the national and international guidelines for a waiver of individual informed consent for this audit of information already in the medical record. At the time of writing no decision had been reached. However, this issue is one that also needs to be addressed by the New Zealand public; as Jocelyn Chamberlain stated, in a review of breast cancer screening in New Zealand, ‘If the popular feeling remains “Privacy at all costs” then it must be recognised that one of those costs is ineffective and inefficient public health systems.’

There is an extensive literature supporting the association between process of care and outcomes in breast cancer. Aspects of detection, diagnostic evaluation and therapy are known to have an important effect on quality of life and mortality. There is a broad consensus on screening, diagnosis and treatment strategies for breast cancer, and many studies on the patterns of care in oncology focus on breast cancer. As such, we would propose that breast cancer is an ideal condition (common, protocol driven, managed by multidisciplinary teams) to act as an audit tool for cancer therapy per se.

Author information: Lorraine Neave, Breast Care Nurse, North Shore Hospital, Auckland; Vernon Harvey, Clinical Director Oncology Department; Chelleraj S Benjamin, Clinical Director Radiation Oncology; Paul Thompson, Consultant Medical Oncologist; Ora Pellett, Project Coordinator ABCR, Oncology Department; Jeremy Whitlock, Radiologist, Radiology Department; Wayne Jones, Clinical Director General Surgery, Department of Surgery, Auckland City Hospital, Auckland;
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Correspondence: Dr C S Benjamin (Chairman ABCSG), Clinical Director Radiation Oncology, Oncology Department, Auckland Hospital, Private Bag 92019, Auckland. Fax: (09) 623 4161; email CSBenjamin@adhb.govt.nz

References:


Replacing sugar-based soft drinks with sugar-free alternatives could slow the progress of the obesity epidemic: have your Coke® and drink it too

Emme Chacko, Ingrid McDuff and Rod Jackson

The average New Zealand adult is now officially overweight. Mean body mass index for both men and women is more than 26 kg/m$^2$, one unit above the normal weight cut-off of 25 kg/m$^2$. Obesity, usually defined as a body mass index greater than 30 units, increased by over 50% between 1989 and 1997 and now afflicts nearly one in five adult New Zealanders. Average adult body weight increased by 3.2 kg between 1989 and 1997, paralleling an increase in energy intake; among men daily energy intakes are estimated to have increased from 11.2 MJ in 1989 to 12 MJ in 1997, and in women from 7.2 MJ to 8 MJ. Local information on children is limited but international data suggest similar trends to those for adults. Excessive body weight is now considered one of the most important paediatric medical problems in the United States.

Sources of energy intake have changed in recent years, with total fat contributing just under 35% of total energy in 1997, down from about 38% in 1989. In contrast, carbohydrate intake has increased from almost 44% to over 46% over the same period. Increasing consumption of sugar-sweetened beverages has contributed to the increased consumption of carbohydrate and to the increased total energy intake. While the replacement of energy-dense fat with less-energy-dense carbohydrate has been encouraged as one way to reduce total energy intake, New Zealanders appear to have overcompensated. The causes of obesity are multifactorial; however, the increased availability and consumption of highly palatable, sugar-based soft drinks may be an important contributing factor. Despite being fat free, many of these drinks are surprisingly energy dense (up to 10 teaspoons of sugar in a standard 330 ml can) and relatively less satiating than solid foods with the same energy content, leading to excessive consumption.

Carbonated-beverage (ie, soft-drink) consumption in New Zealand has increased by about 45% in the last five years and we are now the 11th highest consumers per capita worldwide. An unsubstantiated local report suggested that up to 20% of some children’s energy intake is derived from soft drinks but there are no published national data on consumption levels in children. The recently completed National Nutrition Survey in Children will provide us with better information later in the year. However, it is known that males aged 15 to 24 years are the highest adult consumers of soft drinks in New Zealand. Non-alcoholic beverages (including fruit juice, coffee, tea) provide this subgroup of the population with about 260 kcal a day, about 10% of their daily energy intake and about 20% of their daily carbohydrate intake. Well over half these calories are believed to come from soft drinks alone.

Prospective studies have demonstrated an association between soft-drink consumption and weight gain. In a study by Ludwig et al each additional regular serving of soft drink was associated with an increase in body mass index of 0.24 kg/m$^2$ (95% CI
In contrast, drinking diet soft drink was negatively associated with obesity. Other adverse effects of regular soft-drink consumption include the well-known cariogenic effect of sugar on tooth enamel.

Replacing sugar-sweetened soft drinks with artificially sweetened equivalents nationwide could have modest but measurable effects. Many people drink these beverages out of thirst or social conditioning, rather than for energy. Diet drinks using artificial sweeteners would provide the same amount of fluid replenishment and possibly palatability but virtually no energy (about 3 kcal per can). As a first step we suggest that schools and hospitals substitute current ‘sugar-saturated’ soft drinks with sugar-free alternatives in cafeterias and vending machines. There are few New Zealand data on vending machine use but American reports suggest that seven out of ten people use them daily, so the impact of substitution on consumption could be substantial. There has been some debate recently suggesting that schools ban soft drinks from vending machines; however, substitution would allow schools to continue to gain revenue from vending machine sales without adding more sugar to young New Zealanders’ diets.

There is considerable public misconception about the possible hazards of artificial sweeteners used in diet drinks. The most prevalent sweeteners in diet drinks are aspartame and acesulfame potassium. Both have undergone rigorous toxicological study and have been shown to be safe for consumption in humans including pregnant women, children and the ill. Regulatory groups in over 100 countries, including the Joint Food and Agricultural Organization/World Health Organization Expert Committee on Food Additives (JECFA), have approved the use of these sweeteners and the use of low-calorie artificially sweetened products worldwide tripled in the last two decades of the twentieth century. In the United States alone, there are 150 million regular consumers of such products and the use of artificial sweeteners pervades all types of foods, beverages and pharmaceutical products.

Many soft drinks contain caffeine and there is some concern about the potential effects of caffeine consumption particularly with regards to the increase in urinary excretion of calcium. However, human studies suggest that moderate intakes have little or no deleterious effect in young women because of compensatory mechanisms to increase calcium absorption. Older women do not appear to compensate adequately, especially in circumstances of inadequate intake of calcium. Nevertheless, diet soft drinks contain less caffeine than tea and less than one third of the caffeine of equivalent amounts of brewed coffee, both of which beverages are consumed much more frequently than soft drinks in older adults.

Data from the 1997 National Nutrition Survey suggest that young New Zealand men, aged 15–24 years, consume on average almost 300 ml of regular soft drinks daily. By extrapolating from the Ludwig study the effect of soft-drink consumption on body mass index we estimated that an increase in body mass of approximately 0.2 kg/m² (equivalent to a weight gain of 0.6 kg) could potentially be attributed to consumption of soft drinks in young New Zealand men. The potential impact of this increased body mass on blood pressure has been estimated indirectly from the international INTERSALT study, as has been done in other modelling studies. This degree of weight gain was associated with an increase in mean systolic blood pressure of about 0.2 mmHg, and in the long term could potentially be associated with an increase in
stroke of about 0.5% per annum, and an increase in coronary events of about 0.3% per annum.\textsuperscript{11}

If this soft-drink associated increase in body mass and blood pressure affected the total adult population, based on national morbidity and mortality data from 1999,\textsuperscript{13} there would be an annual excess of about 50 hospitalisations and 15 deaths from stroke and about 90 hospitalisations and almost 25 deaths from coronary disease; an annual total of about 140 preventable hospitalisations and about 40 preventable deaths. The morbidity and mortality attributable to the consumption of all non-alcoholic beverages would be approximately twice that of soft drinks alone and these calculations only consider the effects of increasing weight on blood pressure and not on other weight-related disease, particularly diabetes. Moreover, it is recognised that the data from nutrition surveys generally under-report consumption making our projections conservative.

The obesity-related harms of sugar-sweetened soft drinks described above are likely to far outweigh any theoretical harm of the artificial sweeteners found in diet soft drinks, providing sufficient evidence to justify policies limiting their consumption. The substitution of sugar-based soft drinks with diet soft drinks in vending machines in schools and hospitals would be one small but achievable step in the right direction. Schools and hospitals were the first smoke-free zones in New Zealand and could now take a leadership role in tackling the obesity epidemic.

\textbf{Author information:} Emme Chacko, Final Year Medical Student; Ingrid McDuff, Postgraduate Student in Public Health; Rod Jackson, Head of Section, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland

\textbf{Correspondence:} Professor Rod Jackson, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 373 7494; email: rt.jackson@auckland.ac.nz

\textbf{References:}


New Zealand’s first fatality linked to use of 1,4-butanediol (1,4-B, Fantasy): no evidence of coingestion or comorbidity

Lynn Theron, Karl Jansen and Adrian Skinner

1,4-butanediol (1,4-B) is a precursor of gamma hydroxybutyrate (GHB), a drug to which it is rapidly converted following ingestion. Thus, both anaesthetic euphoriants are referred to as ‘GHB’ and ‘Fantasy’. Both are leading causes of drug-induced coma, with 21 overdoses seen at Auckland Hospital in 1999, rising to 162 in 2002 (unpublished data derived from Auckland Hospital Emergency Department Overdose Database). Internationally, coingestion of alcohol and other drugs, comorbidity and/or use in high-risk settings have been common features in those fatalities that have been reported, as with non-medical use of other anaesthetics such as ketamine.3 Some have claimed that these drugs are safe if there is no coingestion or comorbidity, and that coma does not require emergency services.4,5 We describe the first confirmed fatality in New Zealand linked to the use of 1,4-B. This case demonstrates that death from 1,4-B can definitely occur when it is taken at home without coingestion or comorbidity. Advice that emergency services are not required in cases of coma is unsafe. There has now been a further death in Wellington that has been possibly linked with use of 1,4-B.

Case report

A healthy, 22-year-old male and his girlfriend both lost consciousness following ingestion of 15 ml of 1,4-B at a flat where others were present. He told his companions that he had taken too much, had a seizure and went to bed. He was found three hours later, not breathing, by friends. Neither he nor his girlfriend had ingested alcohol or any other drugs except 1,4-B. An ambulance was called and he was found to be in cardiac arrest. No initial bystander CPR had been administered. He was intubated, ventilated, resuscitated and defibrillated. Spontaneous circulation occurred after 30 minutes.

On arrival at the Emergency Department of Auckland Hospital, observations and biochemistry were typical of 1,4-B overdose (unpublished data, Auckland Hospital Emergency Department Overdose Database).1 The local analytical method used for biological samples measures only GHB. The blood level of GHB on admission was 220 mg/l. A chest X-ray indicated aspiration pneumonia. Brain death was confirmed the next day. The patient’s girlfriend recovered after six hours of ventilation.

1,4-B was detected in bottles, vomit and towels taken from the scene. Blood samples taken on admission were analysed for alcohol, most psychotropic medicines that affect the mind, and morphine, heroin, cocaine and amphetamines. Urine was analysed for alcohol and cannabis. There was no evidence for the use of any drugs except 1,4-B. Conclusions from the post-mortem examination were that death resulted from complications due to a 1,4-B overdose. Vomitus was inhaled resulting in pneumonia, shock, cardiac/respiratory arrest and brain damage.
Discussion

Some promotional web sites and ‘user guides’ have stated that taking too much 1,4-B or GHB results in a deep sleep from which the person recovers in a few hours, and that there is no need to call emergency services unless the person has also used drugs such as alcohol. A pamphlet distributed in Auckland at the time of this death stated that ‘there have been instances where people have been inappropriately taken to an emergency room when their friends found them unconscious and unrousable, and assumed they were in danger. These individuals invariably woke up about three hours later, wondering where they were and why all these strange people were doing things to them. Unless other drugs or alcohol have been consumed with these substances, the only treatment necessary is to allow the sleeping person to wake up naturally.’ This case illustrates that such advice can be dangerous. The case is also relevant to statements such as: ‘a significant part of the Government’s strategy to demonise GHB has been to encourage reporters and coroners to allege GHB as a cause of death…Of those twelve deaths, none were caused by any toxicity of GHB. They were either caused by pre-existing medical conditions (cirrhosis), other drugs, or traffic accidents.” The case reported here correctly attributes the death to taking 1,4-B.

Author information: Lynn Theron, Emergency Medicine Specialist, Emergency Department; Karl LR Jansen, Consultant Psychiatrist, Te Whetu Tawera; Adrian M Skinner, Emergency Medicine Registrar, Emergency Department, Auckland Hospital, Auckland

Correspondence: Dr Karl Jansen, Te Whetu Tawera, Auckland Hospital, PO Box 92024, Auckland. Fax: (09) 302 3058; email: K@BTInternet.com

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4. Anonymous. 1,4-butanediol. Drug Education Trust 2001. (No address supplied. For a copy of this pamphlet contact the author.)
Clozapine-associated polyserositis

Allen Lim, Pathmanathan Sivakumaran and Marie Israel

Clozapine, an atypical antipsychotic, is increasingly used in treating refractory schizophrenia. So far, two cases of clozapine-induced polyserositis \(^1,2\) and three cases of isolated pleural effusions \(^3\)–\(^5\) have been reported. We document here the first New Zealand case of clozapine-associated pleuropericardial effusion.

**Case report**

A 74-year-old Caucasian man with schizoaffective disorder was admitted for management of acute psychosis. Failure of risperidone and olanzapine necessitated the use of clozapine, introduced at 25 mg daily with weekly increments of 12.5 mg. This treatment resulted in significant psychological improvement. Twenty days later, whilst on clozapine 50 mg daily, the patient developed a dry cough, chills and rigors, with fever of 38.2 °C. Physical examination, blood investigations and radiology failed to identify an underlying cause. Amoxycillin/clavulanate was commenced empirically, for presumed chest infection.

Despite this, he experienced worsening respiratory symptoms. Blood tests now revealed ESR 90mm/hr, CRP 127mg/L and platelet count 538 x 10^9/l. Other haematological indices, including eosinophil count and biochemistry, were normal. Repeat chest radiograph showed an enlarged cardiac silhouette with small bilateral pleural effusions. Transthoracic echocardiography confirmed a moderate-sized pericardial effusion without tamponade, and normal ventricular size and function.

Pericardiocentesis resulted in removal of 400 ml of blood-stained fluid and analysis of the aspirate was not possible due to specimen clotting. Culture did not grow any organism. The following day, a large, right pleural effusion developed. A total of 1600 ml of straw-coloured fluid was aspirated. Microscopy revealed 2833 x 10^6/l red cells and 233 x 10^6/l white cells with 74% monocytes but no eosinophils. Aspirate protein was 24 g/l with pleural fluid:serum protein ratio of 37%, suggesting this to be a transudate. Aspirate pH was 7.7 and LDH 88 U/l. Microbiological examination and cytology were all negative. CT scanning two days later revealed a large, left pleural effusion and excluded other thoracic or abdominal pathology. Pleurocentesis yielded 1000 ml of straw-coloured fluid with 1017 x 10^6/l white cells, 16 917 x 10^6/l red cells, LDH 149 U/l and pH 8.0. Aspirate protein was 34g/l and the pleural fluid:serum protein ratio was 52%. Rheumatological screen and serial blood cultures were all negative. Mantoux test was non-reactive.

In the absence of a definitive aetiology, clozapine toxicity was considered and the drug withdrawn. The patient’s systemic symptoms rapidly resolved in a week, with the inflammatory markers normalising within a month. At outpatient follow up two months later, he was well with no clinical or radiological evidence of recurrence.
Discussion

Our diagnosis of clozapine-associated polyserositis was one of exclusion, supported by its temporal relationship of onset with clozapine initiation and remission upon drug cessation. Fever and agranulocytosis are well-documented side effects of clozapine. However, there have also been several reports of unusual adverse reactions, such as pleural and pericardial inflammation/effusions.

Review of published case reports suggests pleuropericardial effusions can develop between 7 and 18 days after initiation on 50 to 400 mg of clozapine a day.1–5 Rapid clinical resolution, within 7 to 10 days, is the expected outcome upon withdrawal of therapy. The pleural effusions were either a transudate or an exudate, with monocytic or neutrophilic predominance. Pleural eosinophilia, seen in various drug-related effusions, is not a feature of clozapine-induced effusions.6 There were no obvious common factors between cases to suggest clinical predisposition.

The pathophysiology of clozapine-induced serositis is unknown. The pericardial aspirate from our patient was suggestive of haemorrhagic pericarditis. It is difficult to speculate a mechanism for the development of bilateral pleural effusion. It is plausible the right pleural effusion, due to its rapidity of onset (under 24 hours), was related to left ventricular failure associated with cardiac tamponade. Presence of leukocytosis and raised protein in the left pleural effusion suggests an inflammatory process, possibly an autoimmune-type reaction, as the aetiology. Furthermore, a cumulative dose effect as a significant contributor in the development of serositis cannot be discounted.

Our case highlights the importance of recognising clozapine as a rare cause of pleuropericardial effusion.

Author information: Allen BS Lim, Registrar; Pathmanathan Sivakumaran, Consultant Respiratory Physician, Division of Medicine; Marie Israel, Psychiatrist for Older People, Middlemore Hospital, Otahuhu, Auckland

Correspondence: Dr Pathmanathan Sivakumaran, Division of Medicine, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland. Fax: (09) 276 0282; email: Psivakumaran@middlemore.co.nz

References:

Dr. Dewey, in a little book called “The True Science of Living,” says that temporary complete starvation until there is once more a healthy appetite is the best cure for a host of dyspepsias, debilities, bodily and mental depressions, headaches, &c. It is quite conceivable that persons may be in a state of starvation not from any want of food, but from the fact that the digestive capacity is constantly overpowered by excess of food. Such patients should be treated on the Haig plan: order two meals a day to be taken, at 8 a.m. and at 2 p.m., and at 8 p.m., instead of another dinner, have the stomach thoroughly washed out, and let the patient go to bed on a perfectly empty, clean stomach. The organ then gets twelve hours of absolute rest to store up its physiological energy. The only danger of this treatment is of over-eating, for the man who before had no appetite because his stomach never had any rest now gets so hungry that he eats too much at his two meals. I am not sure that the two-meals-a-day plan would suit us all, but I am sure that the third meal, if taken at all, should be a very small light one – say, some biscuits and fruit with a glass of milk, or a little milk-pudding. If a good breakfast has been eaten at 8, nothing more than what I have indicated is required until a 6 or 7 o’clock dinner. The usual practice of a meat luncheon with afternoon tea consisting of tea and several rather rich and indigestible cakes, followed by a late dinner, the only exercise meantime being a walk of half a mile or a mile to the office and back or, in the case of ladies, to pay a call, must be wrong. In the above I am speaking only of middle-aged men and women. Children and young adults who are taking a great deal of exercise can take an almost unlimited amount of food with advantage; it is after forty years of age that the danger of over-feeding comes in.
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Mediastinal pseudo-mass

A two-month-old boy was referred by his GP with cough and a fever. On examination he was tachypnoeic with decreased air entry over the right upper lobe and widespread crepitations. Chest radiograph revealed a homogenous area in the right hemithorax (Figure 1), suggesting pneumonia. Splaying of the ribs posteriorly raised the possibility of a neurogenic tumour. MRI scan subsequently confirmed the mediastinal mass was a normal thymus (Figure 2). The rib anomalies were incidental and the child’s symptoms resolved with conservative treatment. The normal thymus is large in children and young infants, and may mimic mediastinal pathology. In cases of doubt further imaging may be warranted.

Figure 1. Supine chest radiograph

![Figure 1](image1)

Figure 2. Magnetic resonance image scan

![Figure 2](image2)

We are grateful to Simon Janes of the Department of General Surgery, Christchurch Public Hospital, Christchurch, NZ, and Katharine Halliday of the Department of Paediatric Radiology, Queen’s Medical Centre, Nottingham, UK, for this issue’s Medical Image.
Drugs are for beauty too

Some might dismiss it as vanity, but society’s increasing preoccupation with looks is fuelling a booming business in cosmetic drugs, or ‘cosmeceuticals’, worth $3.4 billion last year in the United States alone.

Cosmeceuticals lurk in the shadowy ground between drugs and cosmetics. Allergan’s Botox, which flattens furrowed foreheads, is one example. Merck’s Propecia for balding pates is another, as are off-the-shelf skin creams with active biological ingredients.

Those who add a prescription cosmeceutical to their morning routine can have real hope of seeing results. But the enticing skincare aisles of your local drugstore tell a different story: dermatologists confess that some 90% of ingredients in anti-ageing creams are little more than overpriced petroleum jelly.

Cosmeceutical manufacturers must shoulder much of the blame, for trying to sidestep drug regulations. The US Food and Drug Administration (FDA) defines drugs as agents intended for use in the diagnosis, cure, mitigation, treatment or prevention of disease, or that affect the structure or function of the body. Cosmetics escape the rigorous trials demanded of a drug because they are assumed only to alter our appearance. But the system falls down for cosmeceuticals, because it is the manufacturers – not the FDA – who decide whether a product is classed as drug or cosmetic. A cream that claims to cure eczema is a drug; if it claims to promote healthy skin, it is a cosmetic. This bizarre situation means that, although some cosmetics companies have excellent R&D arms, many are dissuaded from finding genuinely active ingredients, or advertising their properties if they do, for fear of having to undergo expensive drug trials.

A vaccine against malaria?

Bill Gates, Microsoft’s founder, travelled to Mozambique to announce the donation of $168m to fight malaria, ushering in what some call a new era of philanthropy.

He almost doubled what the rest of the world – governments, the UN and charities – spend on a disease that kills a million people every year, 90% of them in Africa.

Some of the money will boost research on malaria prevention and new drugs to fight drug-resistant strains. Most will go into a quest for a vaccine which, if successful, could transform the continent. ‘It is time to treat Africa’s malaria epidemic like the crisis it is,’ Mr Gates said. ‘It is unacceptable that 3000 African children die every day from a largely preventable and treatable disease.’

The World Health Organization and the government of Mozambique hailed the donation as a humanitarian gesture that partly filled a huge gap in funding for malaria research.
The announcement came just days after Forbes Magazine published a rich list topped for the tenth consecutive year by Mr Gates and his $45bn fortune. Mr Gates is fascinated with biology and scientific advances such as mapping the genome of the mosquito, believing that the insights can benefit the poor and sick.

Guardian Weekly, 25 September – 1 October 2003

**Anaesthesia in groin hernia repair**

For many years, groin hernia repair has been one of the most common operations worldwide. Yet, there is still no consensus about the best choice of anaesthesia. The present day surgeon faces almost the same choice as did his or her predecessors – the choice between local, regional, or general anaesthesia. Local anaesthesia is preferred at most centres where there is a special interest in hernia repair, whereas in other settings, such as general surgical units, regional or general anaesthesia is more often used.

In a recently reported trial, 616 patients at 10 hospitals in Sweden were randomly assigned to have either local, regional, or general anaesthesia. Primary endpoints were early and late post-operative complications. Secondary endpoints were duration of surgery and anaesthesia, length of post-operative hospital stay, and time to normal activity.

Intra-operative tolerance of local anaesthesia was high. In the early post-operative period, local anaesthesia was superior to the other two types with respect to almost all endpoints. At 8 days’ and 30 days’ follow up, there were no significant differences between the three groups. Local anaesthesia has substantial advantages compared with regional or general anaesthesia, such as shorter duration of admission, less post-operative pain, and fewer micturition difficulties. The favourable results obtained with local anaesthesia in specialised hernia centres can, to a great extent, be reproduced by general surgeons in routine surgical practice.


**Helmet design flaw may put cyclists at risk**

Bicycle helmet standards have a design flaw that could leave cyclists vulnerable to serious head injuries, say researchers in Belgium. The current standards fail to protect one of the most vulnerable parts of the human head – the temple. Yet the researchers have shown that the temple is a common impact site.

Bart Depreitere, Carl Van Lierde and their colleagues at the biomechanics lab of the University Hospital Gasthuisberg Leuven and the Catholic University of Leuven studied head injuries in 86 cyclists who had been involved in accidents. They found that 57% of them had suffered impacts to the side of the head, and a further 27% had suffered impacts to the front (*Accident Analysis and Prevention*, in press).

Helmets do protect against some of these injuries, but most current helmet designs leave the temple unprotected, says Depreitere. ‘The temple area is a critical site, because the bone is thin. There is a high incidence of brain injuries from impacts in this area.’

*Accident Analysis and Prevention*, in press
A simple tweak to helmet design would do the trick, says Depreitere. ‘I think that an extra piece just in front of the ear, a couple of centimetres or so, would protect the temple.’

New Scientist, 9 August 2003
Medical discipline – not guilty

Charge: A Complaints Assessment Committee charged that Dr Zauka was convicted by the District Court of the following offence, being an offence punishable by imprisonment for a term of three months or longer:

- drove a motor vehicle under the influence of drink or a drug or both to such an extent as to be incapable of having proper control of that vehicle (section 58 (1) and (2) Land Transport Act 1998);

- and the circumstances of the offence reflected adversely on Dr Zauka’s fitness to practise medicine.

Offence: The incident on 7 January 2002 which led to Dr Zauka being apprehended by the police was a dangerous and erratic piece of driving. It was very fortunate no-one was killed or injured by Dr Zauka’s conduct. When he appeared in the District Court he pleaded guilty. He was convicted, fined $600 and disqualified from holding or obtaining a motor vehicle driver’s licence for six months.

Background: Dr Zauka was recruited by an employment agency to come to New Zealand. He arrived in New Zealand on 5 November 2001. His wife and six children were still living in South Africa. Dr Zauka understood he was to work in Auckland. Last minute changes resulted in him being placed with the Kapiti Accident and Medical Centre in Paraparaumu. The arrangements at the Kapiti Accident and Medical Centre were in a state of flux when Dr Zauka arrived. The Centre was poorly managed and eventually changed its management in mid 2002.

At the time Dr Zauka started work Dr Krivan was his ‘supervisor’. Dr Krivan was not aware of his new responsibility until ‘the last minute’. He had never supervised a doctor before and despite his best intentions and efforts he readily acknowledged that his supervision did not accord with the Medical Council’s comprehensive ‘Guidelines for Supervision and Induction of Temporary Registrants’. Dr Zauka was required to work extensive hours. He worked six days a week including statutory holidays. At this time he was living in a motel unit. Although Dr Zauka had daily contact with his wife and children he was clearly very lonely and struggled to adjust to his new circumstances. Dr Zauka is a practising Anglican. His faith is important to him and he greatly missed having the support of his church while he was working at Paraparaumu.

Dr Zauka’s difficulties were further compounded when he developed a viral infection in early January 2002. He developed severe stomach cramps and had a nurse at the practice administer buscopan injections (20 mg). These were administered once a day for the three-day period preceding his arrest. In addition to the buscopan injections Dr Zauka self-medicated buscopan tablets. He had also self-medicated valium tablets to assist with a bad dental abscess approximately two weeks before the driving offence. Dr Zauka accepted it was wrong for him to self-medicate these medicines. He said that he took valium over three nights and that he took only one 5 mg tablet each night.
On 7 January 2002 Dr Zauka went to a restaurant by himself. He drank wine with his meal. He recalls leaving the restaurant in his car. Thereafter he has no recollection of what occurred until he woke up in hospital.

**Finding:** The Tribunal dismissed the charge. It was not persuaded the circumstances of Dr Zauka’s conviction reflected adversely on his fitness to practise medicine.

The Tribunal’s decision to dismiss the charge was influenced by the following factors:

1. The conviction in the District Court at Porirua on 16 April 2002 was the only conviction Dr Zauka has incurred in New Zealand or elsewhere.
2. The punishment imposed by the District Court comprised a modest fine and the minimum period of disqualification from driving. The District Court did not regard the offending as a serious case of breaching the ‘drink drive’ provisions of the Land Transport Act 1998.
3. The offending occurred when Dr Zauka was ‘off duty’ and did not impact on his discharge of his professional responsibilities.
4. The medical evidence acquired soon after the offence revealed the offence was not part of a previous or ongoing pattern of alcohol or drug abuse. The Tribunal accepted Dr Zauka had not consumed alcohol on a frequent basis before the incident and it also accepted he had not drunk alcohol since 7 January 2002. The Tribunal also accepted Dr Zauka had not self-prescribed any medication since early January 2002.
5. A factor which played a significant role in generating the stress which Dr Zauka suffered from prior to 7 January 2002 was the inadequate support and help he had when he started working in New Zealand.
6. Dr Zauka no longer suffered from the stress which affected him at the time of the offence.

The Tribunal wished to place on record its grave concern about Dr Zauka’s gross lack of judgment on the night of 7 January 2002. In addition it considered Dr Zauka made a grave error in judgment in prescribing buscopan and valium for himself. The Tribunal accepted he appreciated his error.

The Tribunal ordered publication of a summary its findings in the *New Zealand Medical Journal*.

The full decisions relating to the case can be found on the Tribunal web site at [www.mpdt.org.nz](http://www.mpdt.org.nz) Reference No: 03/103C.
Disgraceful conduct – unsafe practice

Charge: A Complaints Assessment Committee charged that Dr Chan acted in a way that amounted to disgraceful conduct in a professional respect in that:

1. He continued to consult with and make arrangements for further surgery with the patient, regarding his previously performed liposuction while suspended from practising medicine; and/or

2. He failed to inform the patient that he was suspended from practising medicine while continuing to consult with her and to make arrangements for further remedial surgery; and/or

3. He failed to inform the patient that he was not a vocationally registered plastic surgeon in New Zealand; and/or

4. He failed to carry out an adequate pre-operative patient assessment, including a clinical examination; and/or

5. He failed to exercise appropriate professional judgment in offering liposculpture to the patient in view of her history of anorexia nervosa, chronic benzodiazepine use and her recommended weight for her height based on body mass index.

6. He failed to obtain the patient’s informed consent to his proposed treatment including the anaesthesia and surgical procedure in that:

   (a) He did not adequately inform the patient of the anaesthesia process, the surgical procedure and the risks and complications associated with the procedure and the post-operative care that was required.

   (b) The consent forms for anaesthesia and for surgery were given to the patient to sign after she had been given the pre-operative oral sedation.

7. There were serious deficiencies in Dr Chan’s anaesthetic practice, namely:

   (a) He failed to provide adequate information to the patient about the nature and/or effects of the anaesthetic that she was to receive; and/or

   (b) There was no anaesthetist present during the patient’s surgery and drugs were administered in a dosage and combination contrary to the accepted guidelines laid down by the Australian and New Zealand College of Anaesthetists, which state that unless an anaesthetist is present, only conscious sedation may be used. The dosage of drugs and combination of drugs administered to the patient could reasonably be expected to result in loss of consciousness.

   (c) He failed to monitor the patient’s condition adequately during the surgical procedure; and/or

   (d) He failed to monitor the patient’s condition adequately post-operatively.

8. He discharged the patient without any of the usual discharge criteria being met thereby potentially compromising her safety.
9. He failed, post-operatively, to adequately acknowledge or address the patient’s concerns arising from her dissatisfaction with the cosmetic result of the surgery.

**Background:** On 21 January 2001 the patient consulted Dr Chan at his clinic. She weighed approximately 52 kilograms and was 155cm tall. She wished to discuss a large liposuction on her hips, arms, bottom, thighs and stomach.

The patient said she had suffered from anorexia and bulimia. At the time she first consulted Dr Chan she said she was still suffering from various eating disorders. She had also been addicted to sleeping pills on and off for about five years and was currently taking diazepam for this. She had not undergone liposuction or any similar procedure before.

Dr Chan told her he was the most experienced cosmetic surgeon in Australasia and that he had performed more operations than any other doctor in either country. He did not tell the patient what his actual qualifications were, he indicated that he had all the necessary qualifications and was reliable, talented and very good at what he did.

Dr Chan told her the procedure would be painless and that she would be in a twilight state where she would not be fully anaesthetised but nor would she feel anything. Other than this, she said Dr Chan did not tell her anything about the risks associated with the anaesthetic she would have if she were to undergo the liposuction. She asked what could go wrong. She said he did not respond but brushed off her questions.

The only form dated 21 January 2001 produced to the Tribunal provided for information relating to the patient’s address, occupation, date of birth, height and weight. General health is ticked as excellent, smoking and alcohol intake as moderate. It provided for ‘Current Medication’ which was recorded as ‘occasionally diazepam’.

The Tribunal found the patient received an unsigned copy of ‘Neurolept Anaesthetic Information Sheet’ or a document to like effect on 21 January 2001.

There was a further document ‘Operation Sheet’ with the date of 27 January 2001 typewritten on it. There was a section at the bottom “Medical History” which the patient signed. It had thirteen boxes providing for a yes/no answer and a provision for allergies and current medications. The patient wrote ‘Diazepam’ and someone else wrote ‘5 mg every 2–3 days’. The Tribunal concluded that this document was not presented to the patient until the day of surgery.

The patient said Dr Chan did not tell her anything about the post-operative period, but the nurse told her she would have massage sessions and be a little sore for the first month and quite swollen, and that some people return to work a few days later while others, depending on the degree of surgery, might need to take a few extra days off work.

The patient attended Dr Chan’s clinic on 27 January. He did not give her any explanation about the procedure. He checked the areas the patient wanted treated. She didn’t think they spoke more than two or three sentences at that time.

She was given a consent form by the nurse to sign. She was given no explanation concerning it. She read it and signed it. It provided consent for liposuction of ‘Arms, Hips, Butt, outer thigh, U and L abdomen, inner thighs’. The words ‘inner thighs’ were added in handwriting which the patient said was not her handwriting.
The patient recalled waking about three times during the procedure and she felt significant pain on each occasion.

The patient did not recall seeing Dr Chan while she was in recovery. She thought she remained in the clinic for about an hour before driving to a friend’s house. The nurses were aware that she had driven herself as they told her at a subsequent visit. She had told them her friend was collecting her.

While at her friend’s house blood started to ooze from the incisions, soaking the body garment and onto the carpet. Her friend wrapped her in towels for about an hour and gave her a drink but she was unable to take more than a little sip as she felt nauseated. She was quite shaky and still very affected by the drugs she had been given during the operation. She believed she stayed with her friend for about an hour and a half and that by the time she left there it would have been about three hours after the operation had finished.

It then took her a further hour to drive to where she was staying. During the drive she tried to concentrate on driving and not get blood all over the car. Once she arrived, she became more shaky and nauseated and felt very weak. She was staying by herself. She stayed in bed for the following two days as she could barely move due to the pain. She passed out on the first occasion when she got out of bed and tried to stand up and would have fainted in all on two or three occasions. She was nauseous and in incredible agony even though she was taking the pain killers she had been given. The patient had understood that the recovery was going to be straightforward and had not expected this outcome. After two days she was able to get up and move about a little and later, at the end of the second day, she was able to eat a small amount and take some fluids and go to the toilet with a little more ease without passing out.

The patient said the day after the procedure she started leaving many messages for Dr Chan to call her. She said she telephoned both clinics several times only to get the message paging service. She was eventually telephoned by a nurse and was told that her symptoms were normal and to rest. She made further phone calls to the nurses and on occasions could not get through as there was no-one available. She wanted to speak directly to Dr Chan but the nurses would always say that he was too busy or not available or in surgery or doing something. On the occasions she did make contact she said she understood from what they said that he was aware of her messages.

The patient described the following five weeks as being very difficult. She subsequently attended Dr Chan’s clinic on 3, 10, 15 and 22 February for massage sessions with Dr Chan’s nurse at further cost. She did not see Dr Chan on any of these occasions.

At one of those sessions the patient expressed concern about the unevenness of her hips.

The other doctor who assisted Dr Chan with the surgery agreed there was a difference in her hips and suggested a little of the excess fat could still be trimmed from her stomach and upper arms for a more satisfactory result.

A date was booked for the corrective procedure for Monday 9 July at 11 am. The patient made all the necessary domestic and work-related arrangements in anticipation of the surgery and, on the day prior to it, telephoned the clinic to confirm. She was astonished and distressed when she was told there was a problem and that there was
no booking for her surgery. A further booking was made for 17 August for the corrective procedure.

On 17 August she telephoned the clinic at around 9 am to confirm she was on her way to the clinic. During this call she was put on hold and, eventually, she was told in a very unsympathetic manner, that the surgery was off, that the patient could not have the procedure, and that she had no idea when it would be possible.

Later that same evening the doctor who assisted Dr Chan telephoned the patient. He told her that Dr Chan had been suspended from practice and nothing else could therefore be done. The patient repeatedly called Dr Chan’s number in Australia until he finally answered. She explained her plight but he refused to discuss how he could help her.

On 6 September 2001 the patient wrote a three-page letter to Dr Chan setting out the history of events and the remedy she was seeking. By any account, it was a plaintive and desperate letter suggesting either a refund of the moneys paid so she could pay to go to another cosmetic surgeon or obtain a quote and undergo the procedure for which Dr Chan could pay. Dr Chan did not respond to that letter.

In October 2001, the patient telephoned Dr Chan again. She asked when he was returning to New Zealand. He said he had no plans to return and that she would have to phone the Australasian Cosmetic Surgery Centre to book the procedure. She told him she had been leaving messages there for two months but no-one had called her back. She asked if he could help her contact the person who had taken over the clinic and organise the corrective procedure but at that point he said that the telephone reception was bad and he could not hear her. She asked if he could refund some of the money she had paid to him and he replied he would have nothing more to do with the matter.

**Finding:** The Tribunal found Dr Chan guilty of disgraceful conduct in a professional respect.

The Tribunal was not satisfied the allegation contained in Particular 1 was established. Dr Chan was not suspended until 27 April 2001 and there was no evidence presented to the Tribunal that the patient saw Dr Chan after that date apart from when she saw him for a few seconds when she had some photographs taken ‘about’ three months after the surgery, which would put the date around the time of suspension.

The Tribunal was not satisfied the allegation contained in Particular 2 was established. It considered it was not clear whether the booking for the corrective procedure for 9 July 2001 was made prior to Dr Chan’s suspension. Even if it were, it was also not clear whether the booking was made with Dr Chan’s knowledge and consent, and similarly with the subsequent booking of 17 August 2001.

The Tribunal was not satisfied Particular 3 was established and considered that even if it was provided, such a failure would not amount to a disciplinary matter in this case.

The Tribunal was satisfied Particular 4 was established and the allegation in Particular 5 was established in relation to the patient’s history of anorexia nervosa, but was not established in relation to her history of chronic benzodiazepine use and her recommended weight for her height based on body mass index.
The Tribunal was satisfied on the facts that Particulars 6 and 7 were established. The Tribunal was satisfied Particular 8 was established and that same-day discharge should only be contemplated after a prolonged period of observation with a minimum of four hours and with evidence of full recovery from the effects of the sedative drugs, evidence that the patient had adequate pain relief and that there was no evidence of significant ooze from the wounds.

The Tribunal was satisfied on the facts that Particular 9 was established.

**Penalty:** The Tribunal wished to make it plain that it was of the firm view that Dr Chan is an unsafe practitioner.

It ordered as follows:

1. Dr Chan’s name be removed from the register of medical practitioners pursuant to section 110(a) of the Act.
2. Dr Chan be censured.
3. Dr Chan pay a fine of $15 000.
4. Dr Chan pay $23 913.99, which represents 60% of the costs of the CAC investigation and prosecution and the Tribunal’s hearing.
5. A report of the Tribunal’s decisions be published in the New Zealand Medical Journal.
6. The Tribunal requested that the Medical Council consider notifying the content of the decision to the Registration Board in the particular State in Australia where Dr Chan may be currently employed and/or currently practises.

The full decisions relating to the case can be found on the Tribunal web site at [www.mpdt.org.nz](http://www.mpdt.org.nz) Reference No: 02/93C.
Heath Thompson

The Canterbury Hospital Board’s first thoracic surgeon in 1954, Heath Thompson performed ground-breaking cardiac and vascular operations. He died at his Christchurch home on 30 August 2003, aged 83.

Dr Thompson was a pioneer and a ‘very significant figure’ in Canterbury’s medical history. He was an outstanding doctor and an honourable person.

Born in London on 3 May 1920, Dr Thompson moved with his family to New Zealand in 1926. They lived briefly in the North Island before moving to Christchurch, where he attended Fendalton Primary School and Christ’s College.

Dr Thompson excelled at athletics and rowing and continued these activities at university. As a scholar he achieved highly by dint of hard work and gritty determination.

He did his medical intermediate at Canterbury College in 1938 and completed his studies for MB ChB at Otago, graduating in 1943. Under World War II ‘manpower’ regulations, he was then posted to Greymouth Hospital as a house surgeon.

Also on the staff at Greymouth was former Dunedin physiotherapist Bernice (Bunny) Alldred, whom he married in 1944.

After the war, the Thompsons volunteered for work in China. They served for three years with the Friends’ Ambulance Unit (a Quaker organisation) during the civil war. Based first at Anjang, north of the Yellow River, they treated battle casualties from both the communist and nationalist sides. Then they moved to Wuhan, where Dr Thompson performed general surgery and Mrs Thompson started a school of physiotherapy.

Dr Thompson said the range of work he had encountered at Greymouth was good preparation for his work in China. When the couple revisited China in 1978, they met many former patients and found the physiotherapy school still functioning.

Postgraduate studies and practice in thoracic surgery followed, in England and Wales, from 1949 to 1953. Then the Thompsons returned to Christchurch.

Tuberculosis was a major health problem in Canterbury then, before new antibiotics were found to control it. Dr Thompson performed many operations on Tb patients in primitive facilities at the Cashmere sanatorium. He also pioneered vascular and cardiac surgery, before the use of bypass pumps and the development of new expertise shifted much of this work to Auckland’s Greenlane Hospital.

Dr Thompson carried out early asthma treatments, clearing out lungs in the days before bronchial-dilating inhalers became available. He and his wife worked together to improve asthma treatment and were largely responsible for the establishment of the Asthma Society. They produced three films on respiratory diseases that were widely acclaimed and used. Dr Thompson had many papers on his work published.
In 1959 Dr Thompson became Head of the Department of Cardiothoracic Surgery for the hospital board. In 1980 he became Medical Superintendent of The Princess Margaret Hospital, where he remained until his retirement in 1985.

Dr Thompson established a respiratory intensive care unit at Princess Margaret. He campaigned for many years for a heart surgery unit in Christchurch and was a longtime patron of advocacy group Cardiac Companions.

Dr Thompson was held in high esteem by his colleagues. On his retirement, fellow surgeon Alan Chimside spoke of his ‘enormous dedication to his work and the welfare of his patients, and department, and staff’.

His colleagues commissioned a portrait of him, which was painted by Dr John Gillies and hangs in the cardiac surgery ward at Christchurch Hospital.

Dr Thompson was unassuming but always worked hard for what he wanted.

He continued to play tennis regularly into his 70s. He was a keen gardener and lover of roses, and enjoyed classical music. This enjoyment was not simply passive as for several years he was manager of the Risingholme Centre orchestra. He discharged this task with his customary quiet efficiency. He was committed to the peace movement and actively opposed nuclear weapons.

This obituary is almost entirely based on one written by Mike Crean for the Christchurch Press (20 September 2003)
Mr William Geddes Shiach, MB ChB (Aberdeen) FRACS, a long-serving surgeon in Gisborne, died suddenly on 24 August 2003.

He was born in Dufftown, Scotland, in 1921, was educated there, and due to World War II had abbreviated undergraduate medical studies at the University of Aberdeen, where he was Surgical prizewinner. On graduation he began service in the Royal Navy as Surgeon Lieutenant RNR and served on the frigate *HMS Locust* at the D-day Normandy landings.

After the War he joined the professorial surgical unit at Aberdeen, but he decided in 1950 to join the Cook Hospital staff as Senior Surgical Registrar.

He then began a long association with Gisborne surgical services in full-time and part-time posts. He spent a period from 1958 to 1963 as Surgeon Superintendent, and he retired in 1986 as Senior Surgeon. He provided visiting consultative services at Wairoa and Opotiki for several years.

Surgical practice in small, isolated districts such as the East Coast requires a diverse range of skills, not only in the broadest spectrum of general surgery, but also acute trauma and childbirth emergencies. Support is also deficient – no registrars – so call means immediate involvement in each new situation. Mr Shiach was unstinting in his commitment to a substantial, structured surgical load and a call commitment never better than one in three nights and weekends. Slender orthopaedic staffing saw him acquire an important orthopaedic trauma attachment. His broad and versatile surgical experience led to a sixteen-year attachment to ACC assessment after retiring from the hospital staff.

He maintained enthusiastic support for continuing education and he was a consistent attendant at postgraduate activities.

His cool appraisal and detached judgement made him an influential voice in hospital policy.

He received great support from his Gisborne-born wife, Nancy, and had a very robust family of three daughters, one of whom, Dr Helen Shiach, is medically qualified.

Recreationally, he was an ambitious and indefatigable DIY exponent to the advantage of the beautiful home he created, and in which he was a most generous host. The Gisborne district is indeed fortunate that he forsook his native Scotland for the East Coast of New Zealand.

We are grateful to Dr J D Frankish for this obituary.
e-Pathways: computers and the patient’s journey through care


The development of care pathways (clinical pathways, integrated care pathways, etc) and other patient care processes has been widespread,\(^1,2\) with many examples of both good practice and considerable variability, as well as a lack of consistency and lack of opportunities for clinicians to learn from each other and to engage with management.

This excellent book is about work in progress to address these issues, but care-pathway beginners should look elsewhere.\(^3\) Healthcare workers who understand pathways, believe in the multidisciplinary approach, and want to work smarter not harder, will not be disappointed.

Care plans may be mapped or flowcharted to facilitate a common understanding between participants. The logic of the plan should be underpinned by the best available evidence, which must be kept up to date. The care plan should be a flexible and dynamic record of planned activities and variances from the plan, should have a beginning and an end, and be accessible by all participants.

Easy interrogation of captured information and enhanced communication require use of a common language, agreed definitions, and the practical application of information technology. Whether developed as part of an electronic patient record or migrated to via a paper-based record, agreement upon the basics will facilitate the necessary knowledge management.

Although set against a background of practice within the NHS, the book provides useful signposts and action points for antipodean readers. The authors propose establishing a ‘Community of Practice’ – an online community of people interested in the topic of (in this instance) care pathways to support more conventional means of education, training and communication.

A glossary and over 60 web sites are listed in the book, with more provided as links in the included CD-ROM. The material is clearly and attractively set out but, as the later chapters may be heavy going for some readers, a summary of chapters 5 and 6 is provided.

Gerald Moss
Christchurch

References:
