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This Issue in the Journal

Alcohol consumption and its contribution to the burden of coronary heart disease in middle-aged and older New Zealanders: a population-based case-control study
S Wells, J Broad, R Jackson

Alcohol consumption has long been associated with increased risk of diseases (such as liver disorders and some cancers) and increased risk of sustaining injuries. International studies have also demonstrated that alcohol consumption may reduce the risk of coronary heart disease. An Auckland-based case-control study of non-Maori, non-Pacific Island residents has found that middle-aged and older people who regularly drink small amounts of alcohol more than once per month have a significantly lower risk of coronary heart disease than non-drinkers.

Smoking in a New Zealand university student sample
K Kypri, J Baxter

The purpose of this study was to estimate the prevalence of smoking in a New Zealand university student population—for comparison with students in other countries, and with similarly aged youth in New Zealand. In 2002, a random sample of 1564 University of Otago students completed an Internet survey (82% response). Daily smoking (10%) was uncommon relative to the general population aged 15–24 years, and to students in other developed countries. Smoking among Maori women remains a priority area for intervention.

Reduction in crime by drug users on a methadone maintenance therapy programme in New Zealand
I Sheerin, T Green, D Sellman, S Adamson, D Deering

A survey of 85 drug users on methadone maintenance therapy (MMT) in Christchurch found there was a large reduction in drug use and crime during a mean time of 57 months when participants had been on MMT. Sixty percent of participants said they had been committing crime daily before MMT, compared with only 1% after 57 months on MMT. There was a large reduction in costs of crime and imprisonment to society, which was conservatively estimated at $55 386 per drug user per year. These reductions were similar for both Maori and non-Maori participants.

Patterns of amphetamine use in New Zealand: findings from the 2001 National Drug Survey
C Wilkins, M Pledger, K Bhatta, S Casswell

This paper examines the level of heavy amphetamine use, poly drug use, and intravenous drug use by amphetamine users in New Zealand. Over 10% of New Zealand men aged 18–29 had used amphetamines in the previous year. About one-

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fifth of users used amounts of amphetamine in a single session that have been identified in research elsewhere as being hazardous. Poly drug use was common within the amphetamine-using population, with the use of a range of illicit drug types at levels many times higher than the general population. Of particular concern were the relatively high levels of LSD, ecstasy, cocaine, homebake heroin, and intravenous drug use among amphetamine users (compared to the general population). Ongoing monitoring is required to identify whether increased amphetamine use is a source of increased intravenous drug use.

Changes in stroke care at Auckland Hospital between 1996 and 2001
A Barber, A Charleston, N Anderson, D Spriggs, D Bennett, P Bennett, K Thomas, Y Baker

This study shows an overall improvement in the care of people with stroke at Auckland Hospital between 1996 and 2001. These improvements are likely to be the result of greater organisation of medical, nursing, and allied health staff into a specialised stroke team. The authors conclude that greater improvements in care could be gained with the development of a stroke unit where people with stroke are cared for in a specialised hospital ward.

The Acute Stroke Unit at Middlemore Hospital: an evaluation in its first year of operation
M Di Matteo, C Anderson, Y Ratnasabapathy, G Green, K Tryon

Despite high quality, overseas research evidence, health services have been slow to develop specialist stroke services in New Zealand. This study reports an evaluation of New Zealand's first comprehensive (that is, combined acute care and early rehabilitation) Acute Stroke Unit (ASU), established at Middlemore Hospital in Counties Manukau District Health Board, Auckland in 2001. The results provide important 'set-up' information and show several indicators of a better process of care, rehabilitation, and outcome for patients after introduction of the ASU.

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Is it time for treatment trials in addiction?

Roger Mulder

This issue of the journal focuses on addictions. Four studies survey the New Zealand population in various ways. Wells and colleagues report that regular moderate drinking of alcohol is good for middle-aged citizens,[1] at least with regard to coronary artery disease. They also point out that younger people do not receive similar alcohol-related benefits; instead they suffer a net harm from alcohol consumption. Sheerin and colleagues report a substantial reduction in illegal opioid use and crime once subjects were stabilised on methadone maintenance therapy (MMT); the effect appears substantial, with the upper estimate reduction in societal costs of $131 950 per injecting drug user.[2]

The other two studies follow a long-standing tradition of surveying patterns of drug use. Kypri and Baxter report that approximately 10% of University of Otago students smoke cigarettes daily—these rates are low compared with national peer norms and similar international samples.[3] Wilkins and colleagues report findings on amphetamine use from the National Drug Survey.[4] They note that 5% of people aged 15–45 had used stimulants in the previous year, and less than 5% of these (ie, 0.25% of the whole sample) reported using stimulants weekly or more, suggesting that media reports of a highly addictive drug rampaging through the New Zealand population are somewhat premature (as most people appear to use stimulants intermittently).

The latter two surveys have something worthwhile to say but the question arises of why there is a virtual absence of trials studying the effectiveness of treatments in New Zealand patients with addictions. The apparently endless surveys have clearly established that a substantial population of New Zealanders are addicted to a variety of substances. A fair conclusion is that the rates in the use of various substances vary over time and in different populations, but that the big two are alcohol and nicotine, followed by cannabis. We surely know this by now. Why then, are there so few studies assessing the effectiveness of treatments for individuals addicted to these three substances?

It could be argued that public education may ameliorate the significant public health consequences of milder alcohol, nicotine, and cannabis use disorders. Education about self-help strategies for quitting, or cutting down, may obviate the need for more professional assistance. But those whose problems resist self-help (and present for treatment) need more effective forms of treatment, more efficiently delivered than is usually the case at present.[5] In my opinion, the best way to develop such services is to give patients systematically evaluated treatments that are designed to test their efficacy in a New Zealand population setting—ie, randomised controlled trials.

To my knowledge, there are only two randomised controlled trials carried out in New Zealand that have ever been published in peer-reviewed journals.[6,7] In contrast, there are dozens of surveys of the prevalence and patterns of alcohol and drug use, and reviews of what actions could be necessary. Why is this? Surveys are certainly easier and cheaper to carry out that controlled treatment trials. Perhaps they are more likely

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to receive funding. However, if we really believe that addiction is the serious and widespread problem that the repeated surveys report that it is, then learning more and more about changes in prevalence and risk factors is of limited value. One might argue that knowing about risk factors may lead to measures to help modify them. But it seems to be at least as important to study their impact on the uptake and effectiveness of treatment. If Maori women have the highest rates of smoking of University of Otago students, as the Kypri and Baxter study reports,[3] then any treatment would have to be attractive and acceptable to them, and tested to see if it actually was. The best way to test the effectiveness of treatments is via randomised controlled trials. At present, often we have insufficient data to even reassure ourselves that some of our interventions may not be harmful.

The Sheerin (and colleagues) study is a start.[2] It systematically assesses the outcome of a treatment (MMT) currently used in New Zealand and suggests that it is effective with regard to opioid use and crime—but it looks at the highly aberrant minority of intravenous drug users. We need similar systematic assessments of currently used treatments for alcohol, nicotine, and cannabis addiction. We also need trials of new and innovative treatments for these disorders, which systematically compare treatments with each other or against no treatment.

It is little wonder that many clinicians steer clear of the area. They see it as riddled with politics, prejudices, and conflicting beliefs. If we are serious about helping people with addictions, then it is surely time to stop going on about how common the problem is, who it affects, why certain groups are more vulnerable—a sort of public wringing of hands—and begin funding carefully considered, properly designed trials of what treatments work for New Zealand populations with alcohol and drug addictions.

Author Information: Roger Mulder, Professor of Psychological Medicine, Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences, University of Otago, Christchurch

Correspondence: Professor Roger Mulder, Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences, University of Otago, PO Box 4345, Christchurch. Fax: (03) 372 0407; email: roger.mulder@chmeds.ac.nz

References:

1. Wells S, Broad J, Jackson R. Alcohol consumption and its contribution to the burden of coronary heart disease in middle-aged and older New Zealanders: a population-based case-control study. NZ Med J. 2004;117(1190). URL: http://www.nzma.org.nz/journal/117-1190/793/
2. Sheerin I, Green T, Sellman D, et al Reduction in crime by drug users on a methadone maintenance therapy programme in New Zealand. NZ Med J. 2004;117(1190). URL: http://www.nzma.org.nz/journal/117-1190/795/
3. Kypri K, Baxter J. Smoking in a New Zealand university student sample. NZ Med J. 2004;117(1190). URL: http://www.nzma.org.nz/journal/117-1190/794/
4. Wilkins C, Pledger M, Bhatta K, Casswell S. Patterns of amphetamine use in New Zealand: findings from the 2001 National Drug Survey. NZ Med J. 2004;117(1190). URL: http://www.nzma.org.nz/journal/117-1190/796/
5. Hall W, Teesion M. Alcohol use disorders: who should be treated and how? In: Andrews G, Henderson S, editors. Unmet need in psychiatry. Cambridge, UK: Cambridge University Press; 2000.

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6. Sellman JD, Sullivan P, Dore GM, et al. A randomized controlled trial of motivational enhancement therapy (MET) for mild to moderate alcohol dependence. J Stud Alcohol. 2001; 62:389–96.

7. Chapman P, Huygens I. An evaluation of three treatment programmes for alcoholism: An experimental study with 6- and 8-month follow-ups. Br J Addict. 1988;83:67–81.

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Stroke care in New Zealand: a team game where everyone needs to run with the ball

John Gommans

Stroke is a leading cause of death and disability in New Zealand (NZ). Every day, 20 New Zealanders will suffer a stroke, but less than half of them will be alive and independent after 1 year. The average lifetime cost of each stroke for acute care, rehabilitation, support services, and institutional care is $50 000–$100 000.[1]

Thankfully, strokes can be prevented,[2] and mortality and morbidity from acute stroke reduced.[1] The need to establish 'organised stroke services' in each District Health Board (DHB) is the most important recommendation of the new stroke management guidelines. Organised stroke care usually includes; acute admission to (and early rehabilitation in) a stroke unit, care coordinated by a multidisciplinary team, staff with expertise in stroke and rehabilitation, use of written protocols, involvement of patients' families and caregivers, and access to community stroke teams. It is no surprise to health professionals that patients do better if they are admitted to an area dedicated to their problems, and are cared for by people with appropriate expertise and interest.

If all people admitted with stroke are managed in a stroke unit (number-needed-to-treat [NNT] 18 to prevent death or dependency), if most are also rapidly given aspirin (NNT 83), and if a selected few are treated with intravenous thrombolysis (NNT 16)—then significantly more will live, regain their independence, and avoid the need to enter institutional care.[2] Recent publications, however, confirm that stroke care in NZ is disorganised and not consistent with guideline recommendations.[3,4] This implies that (every day) one or two of these 20 new stroke patients will remain dependent or die when this could otherwise have been avoided. Reasons for this astounding neglect of an important, feared, expensive, and disabling condition (ie. stroke) were addressed in a previous editorial,[5] and include both health professional and health management attitudes to stroke.

Is there hope for change? The answer is probably yes. The recent guidelines provide a clear blueprint for what we should be doing, although the information has been available in international and local guidelines for several years. The real challenge, as always with guidelines, will be in their implementation. The Ministry of Health (MoH) has made a start by providing some funding to the Stroke Foundation of NZ to assist with implementation of stroke guidelines.

We now also have a growing pool of clinicians with expertise in stroke who can provide leadership, facilitate education, and promote evidence-based practice. While no DHB approaches the full recommendations for an organised stroke service, some centres have implemented components of it. Hanger previously discussed the benefits of a new stroke rehabilitation unit[6]—and, in this issue, we have similar papers about a mobile stroke team,[7] and NZ's first comprehensive acute stroke unit.[8] These papers also demonstrate positive impacts on quality of care and some outcome measures, but one senses a degree of frustration from the latest researchers. The Middlemore unit is

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only large enough to manage one third of their stroke patients and appears to lack back-up by a dedicated stroke rehabilitation unit, the most important feature of the published stroke unit trials. The Auckland stroke team managed to reach 88% of acute stroke patients in their hospital—although, as they acknowledge, stroke teams on general wards are not as effective as admission to a stroke unit. Again, no dedicated stroke rehabilitation service exists, and their subsequent new stroke unit also only reaches one third of the target population.

Audits are important to ensure that new units and processes are consistent with best practice guidelines and so that resources are used appropriately. Audits may also identify unexpected findings requiring further investigation—such as the reduction in referrals from the Middlemore unit to rehabilitation and community services. Local stroke services, however, should not be expected to justify the benefits of implementing best practice.

As individual health professionals, we can usually practice evidence-based medicine—eg, 'should I lower this person's blood pressure and, if so, how?' Alone, we cannot practice 'organised stroke care' because this requires a system. Health management, therefore, is an essential member of the stroke team.

The MoH has at least 'caught the ball' by convening a stroke advisory group, supporting development of the new guidelines, and providing some funding for their implementation. However, whether their proposed stroke service specifications will be mandated within DHB contracts, or whether funding levels will be adequate, remains to be seen.

In hospitals, the need to aggregate patients with staff (and to keep appropriate beds free) clashes with the lean, multi-skilled or de-skilled, high-occupancy service beloved of modern cost accountants. When 'any bed will do', and efficiency is judged by minimal costs and maximal occupancy rates rather than patient outcomes, then people with stroke will suffer. The recent devolution of institutional care costs to DHBs now gives them a major financial incentive to address this issue—because 20% of stroke survivors will require this care, and this number is expected to treble over the next 20 years as NZ's population ages. Stroke units can reduce this need for institutional care (NNT 16), therefore investment in acute and rehabilitation care should provide long-term financial benefits for DHBs.

In summary, organised stroke care is a team game with clear tactics that produces winning results for patients, staff, and health systems—at least when played overseas. Our home game, however, remains in some disarray! The MoH has agreed to the rules but they have yet to provide the promised playing field. Some enthusiastic health professionals have already kicked off, while many others are still on the training field. Indeed, without DHB players willing and able to run with the ball, the ultimate goal of best patient outcomes will continue to escape us.

Author Information: John Gommans, General Physician and Geriatrician, Hawke's Bay DHB; Honorary Medical Director (Central Region), Stroke Foundation of NZ; member of NZ Stroke Guidelines Development Team; and member of Ministry of Health's Stroke Advisory Group.

Correspondence: Dr John Gommans, Hawke's Bay Hospital, Private Bag 9014, Hastings. Fax: (06) 878 1319; email: john.gommans@hawkesbaydhb.govt.nz

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References:

1. New Zealand Guidelines Group. Life after stroke: New Zealand guideline for management of stroke. November; 2003. Available online. URL: http://www.nzgg.org.nz/guidelines/0037/Stroke_Summary.pdf Accessed March 2004.
2. New Zealand Guidelines Group. The assessment and management of cardiovascular risk. December; 2003. Available online. URL: http://www.nzgg.org.nz/guidelines/0035/CVD_Risk_Summary.pdf Accessed March 2004.
3. Barber PA, Anderson N, Bennett P, Gommans J. Acute stroke services in New Zealand. NZ Med J. 2002;115:3–6
4. Gommans J, Barber PA, McNaughton H, et al. Stroke rehabilitation services in New Zealand. NZ Med J. 2003;116(1174). URL: http://www.nzma.org.nz/journal/116-1174/435/
5. Wilkinson T, Sainsbury R. Stroke services high priority, but neither coordinated nor timely (Editorial) NZ Med J 16 May 2003, Vol 116(1174). URL: http://www.nzma.org.nz/journal/116-1174/433/
6. Hanger HC. Implementing a stroke rehabilitation area: the first six months. NZ Med J. 2002;115(1158). URL: http://www.nzma.org.nz/journal/115-1158/110/
7. Barber PA, Charleston A, Anderson N, et al. Changes in stroke care at Auckland Hospital between 1996 and 2001. NZ Med J. 2004;117(1190). URL http://www.nzma.org.nz/journal/117-1190/797/
8. Di Matteo M, Anderson C, Ratnasabapathy Y, et al. The acute stroke unit at Middlemore Hospital: an evaluation of its first year of operation. NZ Med J. 2004;117(1190). URL http://www.nzma.org.nz/journal/117-1190/798/

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Code of Conduct for expert witnesses giving evidence before the Medical Practitioners Disciplinary Tribunal

Jonathan Coates

In recent years, medical practitioners have become increasingly familiar with the legal processes that regulate medical practice. For some, this has included being requested to comment in a formal disciplinary hearing, as an expert, on the acceptability of a colleague's practice. Although experienced lawyers will (almost always) be available to guide practitioners in such circumstances, many practitioners (who are asked to give evidence as experts) will do so with a degree of uncertainty as to what the process entails. To assist in guiding medical practitioners who give evidence before the Medical Practitioners Disciplinary Tribunal ('the Tribunal'), the Tribunal has recently published a Code of Conduct for Expert Witnesses ('the Code'). The Code is the same as that used by the High Court, and stipulates the conduct required of expert witnesses when they appear before the Tribunal. The Code is published in full in this edition of the Journal, and is self-explanatory (see http://www.nzma.org.nz/journal/117-1190/807/); it is not detailed, and needs to be read in its entirety. The Code is broken into three parts, which are briefly addressed below.

The first part emphasises that the expert witness' overriding duty is to the Tribunal. In particular, the expert witness is not an advocate for the party who has engaged the witness. Furthermore, the witness must assist the Tribunal impartially on relevant matters within the expert's area of expertise.

The second part of the Code sets out several matters that the expert witness' evidence must address. This includes (but is not limited to) acknowledging that the witness has read the Code and will comply with it, confirming that the evidence is within the expert's area of expertise, stating the expert's qualifications as an expert, giving reasons for the opinions, and specifying any literature (or other material used or relied on) in support of the opinions expressed. The expert witness also has an obligation to inform the Tribunal if he or she has knowledge or information that would qualify (or otherwise limit) the completeness or accuracy of his or her evidence. The Code will need to be read to ascertain all the matters which the expert must disclose in evidence.

The third part of the Code gives the Tribunal authority to direct an expert witness to confer with another expert witness. In addition, the Tribunal can direct an expert witness to try to reach an agreement with another expert witness on matters within the field of expertise of the expert witnesses. The Tribunal can require expert witnesses to prepare and sign a joint statement setting out the matters on which the experts agree and the matters on which there is disagreement (including the reasons for disagreement). When conferring with another expert witness, an expert witness must exercise independent and professional judgement and must not act on the instructions or directions of any person to withhold or avoid agreement.

The Tribunal's adoption of the Code brings the Tribunal into line with the High Court's processes concerning expert witnesses, and is to be welcomed. Moreover, the

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implementation of the Code should result in the narrowing of issues that are in dispute and which require the Tribunal's adjudication; this, in turn, should assist in expediting disciplinary proceedings. Whilst the Code (at this stage) just applies to the Medical Practitioners Disciplinary Tribunal, it is likely that the Health Practitioners Disciplinary Tribunal (which will be established this year under the Health Practitioners Competence Assurance Act 2003) will adopt a similar code.

Author information: Jonathan Coates, Senior Associate, Buddle Findlay, Wellington

Correspondence: Jonathan Coates, Buddle Findlay, PO Box 2694, Wellington. Fax: (04) 462 0432; email: jonathan.coates@buddlefindlay.com

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Alcohol consumption and its contribution to the burden of coronary heart disease in middle-aged and older New Zealanders: a population-based case-control study

Susan Wells, Joanna Broad, and Rodney Jackson

Abstract

Aims To quantify the effect of alcohol consumption on risk of coronary heart disease (CHD) and to estimate the contribution of alcohol to the burden of coronary heart disease mortality and morbidity in New Zealand.

Methods A population-based case-control study was conducted. Cases were all fatal and major non-fatal coronary events in non-Maori, non-Pacific-Island Auckland residents aged 35–74 years who were identified between 1 January and 31 December 1992 from the Auckland Region Coronary Study. The controls were an age-stratified random sample from the same population. Trained nurse interviewers administered a standard questionnaire to cases (or their next-of-kin) and controls.

Results The study included 1381 cases of acute fatal or non-fatal hospitalised myocardial infarction or coronary heart disease death, and 1892 controls. Compared to non-drinking men, the coronary heart disease risk in men reporting that they regularly drank alcohol more than once per month, was approximately halved. For women, the coronary risk in regular drinkers was approximately one-third lower than in non-drinkers, although the protective association appeared to be stronger among light-to-moderate drinkers.

It was estimated that if all non-drinkers and those drinking less than once per month had the same coronary disease risk as regular drinkers, there would be approximately 15% fewer coronary disease events in men and 21% fewer events in women aged 35–74 years in Auckland. Conversely, if regular drinkers had the same risk as non-regular drinkers, there would be 50% more events in men and 16% more events in women.

Conclusions Middle-aged and older people who regularly drink alcohol (more than once per month) have a significantly lower risk of coronary heart disease than non-drinkers. The magnitude of the attributable risks calculated here suggest that light drinking could have a substantial beneficial effect on coronary disease rates in this age group in New Zealand.

The relationship between alcohol consumption and risk of death from all causes is best described by a J or U-shaped curve,[1,2] with abstainers and heavy drinkers having higher mortality risks than light-to-moderate drinkers. The observed benefit for light-to-moderate drinkers is largely due to a reduction in coronary disease.[1,3–8] While most studies suggest that any regular drinking, whether light or heavy, reduces the risk of coronary disease, in heavy drinkers any coronary-related benefit is overwhelmed by alcohol-related harm.

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There are large social and medical consequences of alcohol consumption at a population level. Alcohol consumption can significantly increase an individual's risk of developing some diseases (eg, alcoholic psychoses, pancreatitis, liver cirrhosis, breast and large bowel cancer), and sustaining injuries (road traffic crash, suicides, falls, drownings, assaults, and fire injuries).[9] The major burden of death and disability related to alcohol in younger age groups (particularly less than about 35 years) is due to injury. Indeed, younger people gain little or no benefit from alcohol consumption because their coronary risk is negligible.[9]

The impact of alcohol varies between countries and regions due to different levels of its use by populations, and differences in population age structures and disease patterns.[10] The World Health Report (2002) estimated that alcohol consumption caused 4% of the global disease and disability burden, and ranked it third (in terms of percentage of all lost healthy-life years) among the leading risk factors in the developed world (behind tobacco and high blood pressure).[11] The burden of disease and disability attributable to alcohol in New Zealand, accommodating both the beneficial and harmful effects of drinking, has recently been estimated as part of a major report on the health of New Zealanders.[9] The alcohol-attributable burden estimates were based on alcohol consumption prevalence data from the 1996-7 New Zealand Health Survey AUDIT questionnaire, and on international data on alcohol-related risk [(7)]. In the New Zealand survey, it was estimated that if non-drinking New Zealanders aged 40–74 years were regular drinkers, coronary heart disease event rates could fall by about 9% for women and 13% for men.

Our study was undertaken to determine the magnitude of the risk of coronary disease in New Zealand drinkers and non-drinkers, and to investigate the validity of previous estimates of population-attributable risk related to alcohol and coronary disease (using local rather than international data).

Methods

Study design and procedures Between January 1992 and March 1993, we conducted a population-based case-control study of coronary heart disease in the Auckland region.

Cases The cases were all coronary events in men and women aged 35 to 74 years for the 12-month period from 1 January 1992, who were registered in the Auckland Regional Coronary or Stroke (ARCOS) Study. The ARCOS Study was a 10-year community-based cardiovascular disease register and was one of the World Health Organisation (WHO) international MONICA Project study sites.[12] The register identified people who met the WHO criteria for a coronary event and who lived in the greater Auckland region. A coronary event was defined as a coronary death (definite fatal myocardial infarction, possible fatal myocardial infarction, and unclassifiable coronary death) or a hospitalised non-fatal definite myocardial infarction event. Definitions and data collection methods in the ARCOS study have been described elsewhere.[14] In brief, participants who met the inclusion criteria and who were admitted to hospital were interviewed by trained nurses using standard questionnaires. Information on those persons who had died was gathered from relatives, necropsy records, and death certificates using the same interviewer-administered questionnaire.

Controls Controls were an age-stratified random sample of men and women 35–74 years of age, living in the same geographic area as the cases. The general electoral roll for the region was used as the sampling frame, and approximately 250 people in each 10-year age/sex band between 35 and 74 years were selected. An initial letter of invitation with a reply-paid consent form was sent, followed up by two further letters sent at two-weekly intervals if necessary. Participants attended study centres between June 1992 and March 1993. A standard questionnaire was administered by a trained interviewer. The same questions on sociodemographic characteristics, cardiovascular disease risk factors, and alcohol consumption were asked to both cases and controls, and several study staff

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interviewed both the cases and controls. Following the control interview, risk-factor measurements were made (including blood pressure, height and weight).

Alcohol consumption measures A non-drinker was defined as a lifetime non-drinker; an ex-drinker was defined as a person who used to drink at least once per month but at the time of survey drank less than once per month or not at all. An occasional drinker was a person who drinks alcohol less than once per month. Frequency of alcohol drinking was categorised by the number of occasions alcohol was consumed per day, per week, per fortnight or per month. Types of alcoholic beverages drunk on the most-common or usual drinking occasion were classified as beer, spirits/liqueurs, ordinary table wine, fortified wine, and 'other'. Consumption on a usual occasion was recorded by type of alcohol and container size, and then servings were converted to number of standard (10 grams of alcohol) drinks.

Potential confounding variables A wide range of potential confounding variables were collected in this study and were considered in the analyses—and (according to Rose's questionnaire)[14] included age; sex; socioeconomic status; income; education; family history of premature cardiovascular disease; personal history of diabetes, myocardial infarction, or angina;[14] currently taking aspirin, nitrate, or antihypertensive medication; tobacco smoking; leisure-time physical activity; 'overweight'; and obesity. Lipid profile data was not included as this was mostly unrecorded for the cases. Measured blood pressure was not included because blood pressure often falls post-myocardial infarction and was also not available for fatal cases. All data were self-reported except for body mass index (BMI) that was calculated mainly from measured height and weight. Where measurement was not physically possible, reported estimates of height and weight were used. 'Overweight' was defined as body mass index (BMI) greater than 25 and less than or equal to 30 kg/m^2, and obesity was defined as BMI greater than 30 kg/m^2. A family history of premature cardiovascular disease was considered positive if the father had a myocardial infarction (MI) or stroke before the age of 55 (for the mother, before the age of 65). As the questionnaire did not differentiate the sibling gender, family history of premature cardiovascular disease in a sibling was considered positive if an event occurred prior to the age of 60. An active smoker was defined as a person who currently smoked at least one cigarette per day on average. Self-reported leisure time physical activity was categorised as either moderate (20 minutes or more of moderate activity at least three times a week), vigorous (the same duration and frequency as moderate activity but of an intensity causing sweating or hard breathing), or sedentary (if otherwise classified).

Socioeconomic status was classified as high, medium, or low on the NZSEI scale—in which groups rating 9–40 were denoted low socioeconomic status, 41–59 denoted medium socioeconomic status, and greater than 60 denoted high socioeconomic status.[15]

Total household income was categorised into three categories in 1992 dollars; low (less than $15,000/year), medium (more than $15,000/year but less than $50,000/year), and high (more than $50,000/year). To assess educational attainment, participants were asked their age on leaving school, and whether they received further education after they left school.

Exclusions Maori and Pacific Islanders were excluded from the analyses—as the sampling frame for controls did not include the Maori electoral roll, and the general electoral roll significantly under-represented the true proportions of Maori and Pacific Islanders in the general population.

Statistical analyses All analyses were conducted using SAS statistical software,[16] and odds ratios (together with 95% confidence intervals) were calculated using unconditional logistic regression. As the study outcome (ie, an acute coronary event) is rare, the odds ratios calculated will be very similar to relative risks. Two gender-specific models were developed to examine the association between each alcohol consumption measure and CHD event. The first model was age-adjusted only. The second model was adjusted for age and other factors—including previous medical history of CHD (previous MI or angina or currently on nitrate medication), diabetes, premature family history of cardiovascular disease, tobacco smoking, current antihypertensive drug treatment, BMI, leisure-time physical activity, and socioeconomic factors (household income, educational attainment, and socioeconomic status).

Population-attributable risk estimates were calculated according to methods developed by Greenland and described in Rockhill et al.[17] The formula uses relative-risk estimates (adjusted odds ratios were used as estimates of relative risk) and the proportion of cases exposed.

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Results

There were 1318 coronary events (668 fatal events) identified in people aged 35–74 years who met the study case criteria during the 12-month study period. To identify these events, 1228 (93.2%) patients or next-of-kin completed interviews, and complete alcohol data was available for 92% of the events. In the control sample (selected from the electoral roll), 1892 people agreed to participate—with an overall response rate of 72%. The response rate was higher in men than in women and declined with increasing age. Of those persons who agreed to participate, all provided answers to the questionnaire (answering, on average, 98% of items).

Sociodemographic and other characteristics of cases and controls are shown in Tables 1 and 2. These tables show that the cases were more likely to be over 55 years; have lower household income, educational attainment, and socioeconomic status; have a previous history of MI and angina; be on cardiovascular medications; have a higher proportion of cardiovascular risk factors (such as diabetes, family history of premature CVD, tobacco smoking); and be more sedentary.

Table 1. Sociodemographic characteristics of coronary heart disease (CHD) cases and population controls

	Men				**Women**			
	CHD cases		**Population controls**		**CHD cases**		**Population controls**	
	n	%	n	%	n	%	n	%
All	**961**	**100.0**	**957**	**100.0**	**357**	**100.0**	**935**	**100.0**
Age (years)								
35–44	57	6.0	227	23.7	5	1.4	212	22.7
45–54	166	17.3	230	24.0	32	9.0	236	25.2
55–64	337	35.1	256	26.8	74	20.7	237	25.3
65–74	401	41.7	244	25.5	246	68.9	250	26.7
Ethnicity (after exclusion of Maori and Pacific Island people)								
Asian /other	34	3.5	24	2.5	10	2.8	26	2.8
European	927	96.5	933	97.5	347	97.2	909	97.2
Education								
Left school <15 years	214	22.3	185	19.3	197	55.2	680	72.7
Further education after schooling	193	20.1	284	29.7	46	12.9	329	35.2
Income of household								
Income <$15 000	157	16.3	53	5.5	100	28.0	106	11.3
Income $50 000+	125	13.0	368	38.5	19	5.3	252	27.0
Income missing	151	15.7	25	2.6	56	15.7	84	9.0
Socioeconomic status								
Low (NZSEI <40)	259	27.0	108	11.3	91	25.5	134	14.3
High (NZSEI >60)	153	15.9	284	29.7	32	9.0	279	29.8
Missing	90	9.4	2	0.2	110	30.8	97	10.4

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Table 2. Selected characteristics of coronary heart disease cases and population controls

	Men				Women			
	CHD cases		Population controls		CHD cases		Population controls	
	n	%	n	%	n	%	n	%
All	**961**	**100.0**	**957**	**100.0**	**357**	**100.0**	**935**	**100.0**
Health and medical history								
Self-reported angina	221	23.0	50	5.2	88	24.6	74	7.9
Self-reported diabetic	107	11.1	37	3.9	57	16.0	36	3.9
Self-report prior MI	233	24.2	31	3.2	65	18.2	15	1.6
On anti-hypertensives	433	45.1	157	16.4	211	59.1	171	18.3
On nitrates	196	20.4	24	2.5	106	29.7	26	2.8
On aspirin	236	24.6	92	9.6	99	27.7	115	12.3
Sedentary	512	53.3	237	24.8	233	65.3	237	25.3
Family history of premature CVD	198	20.6	118	12.3	66	18.5	133	14.2
Body mass index								
Obese (>30)	119	12.4	104	10.9	46	12.9	103	11.0
Overweight (25–30)	406	42.2	405	42.3	88	24.6	254	27.2
Missing	68	7.1	51	5.3	37	10.4	84	9.0
Tobacco smoking								
Current smoker	304	31.6	178	18.6	91	25.5	125	13.4
Previous smoker	411	42.8	429	44.8	93	26.1	281	30.1

Tables 3 and 4 show gender-specific adjusted odds ratios of coronary disease by frequency of drinking, volume of drinking on usual drinking occasion (grams of alcohol consumed), and average daily volume of alcohol consumed (grams/day). Risk estimates for two multivariate models are shown; adjusted for age-group only, and age-group with known risk factors and socioeconomic factors.

With regard to frequency of drinking in men, statistically significant lower coronary risks were observed for all categories of regular drinking, compared to lifetime non-drinkers, ex-drinkers, and those drinking occasionally. For men, similar patterns of risk were observed for ‘usual volume per occasion’ and ‘average daily consumption’. The coronary risk was approximately halved (range 48%-59%) for all categories of persons regular drinking more than once per month, and the risk estimates were similar in all models.

For women, the observed protective association between drinking and coronary risk was more variable over the different alcohol intake measures (probably due to smaller numbers of events), and was generally smaller in magnitude than men. With regard to frequency of drinking, compared to lifetime non-drinking women, only those women who normally drink at least once per week (or more) demonstrated a protective association for coronary disease, which almost reached statistical significance after adjustment for potential confounding factors (OR 0.62; 0.37–1.04). Considering the volume of alcohol drunk on a usual drinking occasion, compared to non-drinking women, only the categories of women drinking 5 to 20 grams (one-half to two standard drinks) of alcohol were associated with statistically significant coronary disease risk reduction (OR 0.36; 0.21–0.61). Similarly, compared to non-drinking women, a statistically significant reduction in coronary risk was only observed for women whose average daily volume of alcohol drinking was one quarter to less than

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two standard drinks per day. In general, coronary disease risk in women drinkers was between 20 to 40% lower than in non-drinkers.

For both men and women, alcohol consumption at any level or frequency was associated with lower risk of CHD.

Table 3. Adjusted odds ratios (95% CI) of coronary heart disease associated with alcohol intake, men aged 35–74 years

	Cases (n = 961)		Controls (n = 957)		Model M1: Adjusted for age group only		Model M2: Also adjusted for other factors*	
	n	%	n	%	OR	95% CI	OR	95% CI
Frequency of drinking								
Lifetime non-drinker	71	7.4	42	4.4	1.00	-	1.00	-
Ex-drinker	117	12.2	65	6.8	1.03	0.62–1.71	0.70	0.38–1.27
Occasional drinker (<1/mth)	140	14.6	84	8.8	0.97	0.59–1.57	0.98	0.55–1.73
Drinks:								
Monthly but not weekly	81	8.4	96	10.0	0.58	0.35–0.97	0.52	0.29–0.95
Weekly	88	9.2	139	14.5	0.43	0.26–0.70	0.41	0.23–0.73
More than weekly but not daily	158	16.4	241	25.2	0.48	0.30–0.75	0.51	0.30–0.87
At least daily	243	25.3	290	30.3	0.49	0.32–0.76	0.50	0.30–0.84
Missing data	63	6.6	0	0.0				
Volume of alcohol on usual drinking occasion								
Non-drinker	326	33.9	191	20.0			1.00	-
<1.5 gms alcohol	76	7.9	84	8.8	0.59	0.41–0.86	0.60	0.38–0.95
1.5 – <5	98	10.2	158	16.5	0.40	0.29–0.55	0.47	0.32–0.68
5 – <10	85	8.8	135	14.1	0.44	0.32–0.62	0.57	0.38–0.85
10 – <20	114	11.9	161	16.8	0.47	0.34–0.64	0.56	0.38–0.81
20 – <30	69	7.2	96	10.0	0.45	0.31–0.66	0.60	0.39–0.94
30+	117	12.2	132	13.8	0.59	0.43–0.81	0.58	0.40–0.86
Missing data	76	7.9	0	0.0				
Average daily volume of alcohol								
Non-drinker	326	33.9	191	20.0	1.00	-	1.00	-
<5 gms alcohol	56	5.8	73	7.6	0.43	0.29–0.64	0.47	0.29–0.77
5 – <10	157	16.3	213	22.3	0.45	0.34–0.60	0.55	0.39–0.77
10 – <20	131	13.6	207	21.6	0.42	0.32–0.57	0.59	0.42–0.84
20 – <30	78	8.1	124	13.0	0.45	0.32–0.64	0.51	0.34–0.78
30+	137	14.3	149	15.6	0.69	0.51–0.95	0.60	0.41–0.87
Missing data	76	7.9	0	0.0				

*****Model M2 adjusted for age group, history of CHD, tobacco smoking, leisure-time physical activity, current antihypertensive drug treatment, family history of premature cardiovascular disease, BMI, diabetes, socioeconomic status, income and low education.

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Table 4. Adjusted Odds Ratios (95% CI) of coronary heart disease associated with alcohol intake, women aged 35–74

	Cases (n = 357)		Controls (n = 935)		Model W1: Adjusted for age group only		Model W2: Also adjusted for other factors*	
	n	%	n	%	OR	95% CI	OR	95% CI
Frequency of drinking								
Lifetime non-drinker	82	23.0	136	14.5	1.00	-	1.00	-
Ex-drinker	37	10.4	56	6.0	1.29	0.75–2.22	0.86	0.42–1.77
Occasional drinker (<1/mth)	96	26.9	176	18.8	1.06	0.71–1.59	1.15	0.69–1.89
Drinks monthly but not weekly	44	12.3	229	24.5	0.52	0.33–0.83	0.76	0.43–1.33
Drinks once or more weekly	66	18.5	338	36.1	0.45	0.30–0.67	0.62	0.37–1.04
Missing data	32	9.0	0	0.0				
Volume of alcohol on usual drinking occasion								
Non-drinker	215	60.2	368	39.4	1.00	-	1.00	-
<1.5 gms alcohol	31	8.7	107	11.4	0.67	0.42–1.08	0.93	0.52–1.65
1.5 – <5	26	7.3	146	15.6	0.45	0.28–0.73	0.83	0.45–1.52
5 – <20	28	7.8	240	25.7	0.26	0.17–0.41	0.36	0.21–0.61
20+	22	6.2	74	7.9	0.62	0.36–1.07	0.80	0.41–1.56
Missing data	35	9.8	0	0.0				
Average daily volume of alcohol								
Non-drinker	215	60.2	368	39.4	1.00	-	1.00	
<1.5 gms alcohol	38	10.6	70	7.	0.88	0.55–1.39	1.10	-0.62–1.98
1.5 – <5	36	10.1	258	27.6	0.31	0.20–0.46	0.54	0.33–0.88
5 – <20	18	5.0	150	16.0	0.32	0.19–0.56	0.42	0.22–0.83
20+	15	4.2	84	9.0	0.54	0.29–1.01	0.58	0.27–1.26
Missing data	35	9.8	5	0.5				

*Model M2 adjusted for age group, history of CHD, tobacco smoking, leisure-time physical activity, current antihypertensive drug treatment, family history of premature cardiovascular disease, BMI, diabetes, socioeconomic status and income.

In Table 5, estimates of population-attributable risks demonstrate the potential proportional reduction in coronary events that would occur if lifetime non-drinkers, ex-drinkers, and occasional drinkers had the same coronary disease risk as regular drinkers. These estimates assume that the odds ratios described are causal and unconfounded. In men aged 35 to 74 years, it is estimated that there would be approximately 15% fewer major-incident coronary disease events if all non-drinkers, ex-drinkers, and occasional drinkers had the same alcohol-related coronary risk as regular drinkers. The equivalent proportions for women aged 35 to 74 years were higher (approximately 21%) because a greater proportion of female cases were non-drinkers or occasional drinkers. Conversely, CHD events would be substantially increased in this population (50% for men, 16% for women) if regular drinkers had the same alcohol-related coronary disease risk as lifetime non-drinkers.

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Table 5. Population-attributable risk (PAR) of Coronary Heart Disease, related to alcohol consumption

	Men				Women			
	OR*	**95% CI**	**Prev. (%)**	**PAR**	**OR***	**95% CI**	**Prev. (%)**	**PAR**
Regular drinker[†]	1.00	-	59.3	0.0	1.00	-	30.8	0.0
All non-drinkers	1.86	(1.43–2.42)	34.1	-15.8	1.55	(1.08, 1.22)	60.2	-21.4
Missing data			6.6				9.0	
				-15.8				-21.4
All non-drinkers[‡]	1.00	-	34.1	0.0	1.00	-	60.2	0.0
Regular drinker	0.54	(0.41–0.70)	59.3	50.5	0.65	(0.45, 0.93)	30.8	16.6
Missing data			6.6				9.0	
				50.5				16.6

Prev.= prevalence in cases

*Estimates of relative risks based on odds ratios adjusted for previous history of age, physical activity, history of CHD, tobacco smoking, leisure time physical activity, current antihypertensive drug treatment, BMI, family history of premature cardiovascular disease, socio-economic status, income, and low education.

[†]PARs are the proportions of CHD events in the population that would be averted if those people not currently drinking regularly had the same alcohol-related coronary disease risk as regular drinkers.

[‡]PARs are the increased proportions of CHD events in the population that would occur if regular drinkers ex-drinkers and occasional drinkers had the same alcohol-related coronary disease risk as lifetime non-drinkers.

Discussion

This large population-based case-control Auckland study shows a strong and statistically significant protective association between alcohol consumption and coronary heart disease risk for men aged 35–74 years. The coronary risk for men regularly drinking alcohol (more than once per month) was approximately half that of non-drinkers. However, for women, statistically significant protective effects were seen only in the light-to-moderate drinkers, and the magnitude of the protective association was smaller. If non-drinkers and occasional drinkers in this Auckland population had the same coronary risk as regular drinkers, it was estimated that there would be about 15% fewer major coronary events among men and about 21% fewer in women.

We have presented gender-specific results—as women develop alcohol-related health problems at lower levels of consumption than men, possibly due to their lower total body-water volume and differences in how they metabolise ethanol compared to men.[18] The protective effect of regular alcohol consumption appeared to be less in women than men in this study. However there was greater variability in the effect estimates for women, and poorer precision due to smaller numbers of cases, so any apparent differences in effect by gender may not be real.

Unfortunately it was not possible to collect information on a representative sample of Maori and Pacific Island adults. Therefore, significant gaps remain in our understanding of the attributable burden of alcohol on coronary heart disease in these ethnic groups. Approximately one third of all New Zealanders live in the greater

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Auckland region, so these findings are likely to be generalisable to European New Zealanders during the study period.

As approximately 50% of the cases were fatal, their interviews were completed by their next-of-kin or other proxy respondent. This may have influenced the accuracy of the information collected. In an earlier Auckland case-control study of coronary heart disease, a validation study of the alcohol consumption data was done. This was based on the same questionnaire as this study, and obtained from either primary respondents or their next-of-kin.[19] The findings indicated that use of proxy sources of data to assess alcohol drinking were unlikely to produce biased estimates of alcohol consumption at the aggregate level.[19]

The lower response rates in controls (compared with cases) may have caused some bias in the risk estimates. Generally, people in poorer health are less likely to participate in epidemiological studies, so the non-response bias may lead to an over-estimate of the protective effect of drinking alcohol. This would be consistent with the international literature as our findings suggest a greater protective effect of alcohol than some other studies.[3,4,6] However, our findings are within the range observed in other epidemiological studies that vary from no clear evidence of a protective effect from alcohol consumption,[20] to as much as 70% relative risk reduction.[21] Comprehensive case-identifying procedures were followed and response rates in cases were high, so there is unlikely to be significant bias related to missing cases. Although the absolute number of coronary events based on the WHO MONICA definitions of a fatal and non-fatal coronary event would be less than estimated using newer troponin-based definitions, there is no reason to believe that the relative effect of alcohol on coronary events would be different.

We attempted to control for confounding by undertaking gender-specific analyses (adjusted for age-group, CHD risk factors, and socioeconomic factors). However, there is still potential for residual confounding, particularly in relation to measures of socioeconomic status that were missing in a proportion of cases. Furthermore socioeconomic indices collected may not adequately control for socioeconomic confounding, especially in women.[22] It is also possible that more health-conscious people were aware of the benefits of regular drinking in the early 1990s, which could have introduced confounding. Furthermore, perceived health benefits may have led to greater social acceptability of drinking and thereby more accurate reporting of alcohol consumption in health-conscious people than in previous studies. Commentators on alcohol studies (demonstrating protective associations between drinking and coronary risk) have suggested the effect may have been an artefact due to drinkers in poor health giving up drinking. However, in our study, we were able to separate lifetime non-drinkers from ex-drinkers and, in general, ex-drinkers were not at excess risk compared with lifetime non-drinkers.

Alcohol consumption patterns for individuals may vary over time. The 1996/7 New Zealand Health Survey found that older people tended to drink more regularly than younger people, but tended to drink less on a single occasion.[23] In this study, we have used several measures of alcohol consumption collected from individuals at one point in time over a period of a year and as such we are unable to assess long-term alcohol exposure. Furthermore, the biological effects of alcohol on individuals may vary with ageing. An interaction term was used to assess possible interaction between age and frequency of alcohol consumption but no effect-measure modification was evident.

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While the observed protective association between regular drinking and coronary risk should be interpreted cautiously because of the potential for residual confounding, the association is well supported by experimental studies that have assessed the effects of moderate alcohol intake on levels of high density lipoprotein (HDL) cholesterol, apolipoprotein A I, triglycerides, fibrinogen, and other biological markers. A recent meta-analysis has summarised the findings of 42 studies.[24] The authors estimated that an intake of 30 grams of alcohol per day would cause a 25% reduction in risk of coronary disease mediated through changes in lipids (mainly increases in HDL levels) and haemostatic factors (mainly lowered fibrinogen levels).[25] However, other factors such as nutrient profile, insulin sensitivity, platelet aggregation, endothelial function, and inflammation may also be beneficially affected by alcohol consumption.[24–26] Furthermore, the relative risk estimates in our study are reasonably consistent with estimates from case-control cohort studies and several reviews.[1,3,5,7,27-30] The international literature suggests that consumption of 1-3 drinks per day is associated with a 20-50% lower risk of coronary disease after adjustment for potential confounding factors. This protective association has been observed for up to six standard drinks per day,[3] although higher intake levels are also associated with increased risk of other health problems. This risk reduction is as large as that associated with many cardioprotective drugs (eg, aspirin, beta-blockers, or cholesterol-lowering drugs).[31]

The observation of a significant protective effect in people who report drinking more than monthly (but less than weekly) is somewhat surprising as it seems unlikely that this amount of alcohol would have a substantive biological effect. While part of the apparent protective effect may be due to residual confounding, there is considerable evidence that people underestimate their alcohol consumption,[32] and it is likely that many people in this drinking category have under-reported their true drinking patterns.

When calculating population attributable risk, the definition of the risk categories should correspond (as closely as possible) with the exposure categories used to measure risk factor prevalence.[9] Furthermore, the patterns and type of exposure in the populations from which the relative risks are derived should be similar to those of the population to which the relative risk will be applied.[33] This population-based case-control study was able to satisfy both these methodological principles. This study, supports a recent New Zealand report's[9] estimation of population-attributable risk related to alcohol and coronary disease for men—but we suggest that benefits to women have been significantly underestimated. The differences in estimates are likely to be due to the use of different exposure measures (the previous work used the AUDIT score), the level of exposure associated with minimum risk, and the use of the same relative risk estimate for men and women (RR=0.8), which may have impacted more on women than in men due to the different alcohol consumption pattern of women.

In conclusion, a strong protective association was observed between alcohol consumption and coronary disease risk. However, any coronary-related benefit from drinking alcohol for the individual will vary in direct proportion to their absolute risk of a coronary event. Those persons most likely to benefit from regular drinking are middle-aged and elderly people who are at higher absolute risk of a coronary event whilst, conversely, the health risk of individuals at low risk of CHD is potentially

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worsened by alcohol intake because the adverse effects will overwhelm any small coronary-related benefits.[34–36] Moreover, the benefits of alcohol consumption were apparent at quite low levels of self-reported consumption and there are significant adverse effects caused by heavy drinking. Therefore, any population-based advice on the benefits of alcohol consumption is only relevant for middle-aged (and older) people, and for light drinking.

Author information: Susan Wells, Senior Lecturer in Clinical Epidemiology; Joanna Broad, Epidemiologist/Biostatistician; Rodney Jackson, Professor of Epidemiology, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland

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Correspondence: Dr Susan Wells, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Private Bag 92019, Auckland 1. Fax: (09) 3737 624; email: s.wells@auckland.ac.nz

References:

1. Doll R, Peto R, Hall E, et al. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. BMJ. 1994;309:911–8.
2. Duffy JC. Alcohol consumption and all-cause mortality. Int J Epidemiol. 1995;24:100–5.
3. Corrao G, Rubbiati L, Bagnardi V, et al. Alcohol and coronary heart disease: a meta-analysis. Addiction. 2000;95:1505–23.
4. Maclure M. Demonstration of deductive meta-analysis: ethanol intake and risk of myocardial infarction. Epidemiol Rev. 1993;15:328–51.
5. Marmot MG. Alcohol and coronary heart disease. Int J Epidemiol. 2001;30:724–9.
6. Holman CD, English DR, Milne E, Winter MG. Meta-analysis of alcohol and all-cause mortality: a validation of NHMRC recommendations. Med J Aust. 1996;164:141–5.
7. Thun MJ, Peto R, Lopez AD, et al. Alcohol consumption and mortality among middle-aged and elderly US adults. N Engl J Med. 1997;337:1705–14.
8. Jackson R, Scragg R, Beaglehole R. Does recent alcohol consumption reduce the risk of acute myocardial infarction and coronary death in regular drinkers? Am J Epidemiol. 1992;136:819–24.
9. Ministry of Health. Our health, our future. The health of New Zealanders 1999. Wellington, New Zealand: Ministry of Health; 1999.
10. Murray C, Lopez A. The global burden of disease and injury series. Cambridge, MA: Harvard University; 2002. Available online. URL: http://www.hsph.harvard.edu/organizations/bdu/ Accessed March 2004.
11. Ezzati M, Lopez AD, Rodgers A, et al; Comparative Risk Assessment Collaborating Group. Selected major risk factors and global and regional burden of disease. Lancet. 2002;360:1347–60.
12. Tunstall-Pedoe H, Kuulasmaa K, Amouyel P, et al. Myocardial infarction and coronary deaths in the World Health Organisation MONICA Project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents. Circulation. 1994;90:583–612.

 © NZMA

13. Doggen C, van der Palen J, Beaglehole R. Trends in medical management of acute myocardial infarction, Auckland 1983–90. NZ Med J. 1993;106:278–81.

14. Rose G, Blackburn H, Gillum R, Prineas R. Cardiovascular survey methods. Geneva: World Health Organisation; 1982.

15. Davis P, McLeod K, Ransom M, Ongley P. The New Zealand Socioeconomic Index of Occupational Status (NZSEI). Report No.: Research Report #2. Wellington: Statistics New Zealand; 1997.

16. SAS Institute Inc. SAS System for Windows. In. Version 8e ed. North Carolina: SAS Institute; 1999.

17. Rockhill B, Newman B, Weinberg C. Use and misuse of population attributable fractions. Am J Public Health. 1998;88:15–9.

18. Bradley KA, Badrinath S, Bush K, et al. Medical risks for women who drink alcohol. J Gen Intern Med. 1998;13:627–39.

19. Graham P, Jackson R. Primary versus proxy respondents: comparability of questionnaire data on alcohol consumption. Am J Epidemiol. 1993;138:443–52.

20. Hart CL, Smith GD, Hole DJ, Hawthorne VM. Alcohol consumption and mortality from all causes, coronary heart disease, and stroke: results from a prospective cohort of Scottish men with 21 years of follow up. BMJ. 1999;318:1725–9.

21. McElduff P, Dobson AJ. How much alcohol and how often? Population based case-control study of alcohol consumption and risk of a major coronary event. BMJ. 1997;314:1159–64.

22. Long JA, Ickovics JR, Gill TM, Horwitz RI. Social class and mortality in older women. J Clin Epidemiol. 2002;55:952–8.

23. Ministry of Health. Taking the pulse. The 1996–97 New Zealand Health Survey. Wellington: Ministry of Health; 1999.

24. Rimm EB, Williams P, Fosher K, et al. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. BMJ. 1999;319:1523–8.

25. Rimm E. Commentary: alcohol and coronary heart disease – laying the foundation for future work. Int J Epidemiol. 2001;30:738–9.

26. Goldberg DM, Soleas GJ, Levesque M. Moderate alcohol consumption: the gentle face of Janus. Clin Biochem. 1999;32:505–18.

27. Jackson R, Scragg R, Beaglehole R. Alcohol consumption and the risk of coronary heart disease. BMJ. 1991;303:221–6.

28. Klatsky AL. Moderate drinking and reduced risk of heart disease. Alcohol Res Health. 1999;23:15–23.

29. Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine or spirits. BMJ. 1996;312:731–6.

30. Brenner H, Rothenbacher D, Bode G, et al. Coronary heart disease risk reduction in a predominantly beer-drinking population. Epidemiology. 2001;12:390–5.

31. Yusuf S. Two decades of progress preventing vascular disease. Lancet. 2002;360:2–3.

32. Lemmans PH. The alcohol content of self-report and ‘standard’ drinks. Addiction. 1994;89:593–601.

33. English DR, Holman CD, Milne E, et al. The quantification of drug caused morbidity and mortality in Australia. Canberra: Commonwealth Department of Human Services and Health; 1995.

 © NZMA

34. Seddon M. Alcohol. An evidence-based nutrition statement from the National Heart Foundation of New Zealand's Nutrition Advisory Committee. Auckland: The National Heart Foundation of New Zealand; 1999.

35. NHMRC. Australian Alcohol Guidelines: health risks and benefits. Canberra: National Health and Medical Research Council, Commonwealth of Australia; 2001.

36. Jackson R, Beaglehole R. Alcohol consumption guidelines: relative safety vs absolute risks and benefits. Lancet. 1995;346:716.

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Smoking in a New Zealand university student sample

Kypros Kypri and Joanne Baxter

Abstract

Aims The aims of this study were to estimate the prevalence of smoking in the University of Otago student population, and to compare estimates with those for university students in other countries, and with other similarly aged youth in New Zealand.

Methods In 2002, 1910 randomly selected University of Otago students were invited to complete an Internet survey, and 1564 (82%) of these students responded.

Results Daily smoking was reported by 10% of both women and men. A further 10% of women and 9% of men reported occasional smoking. Daily smoking was reported by 16% of Maori women and 9% of Maori men.

Conclusions Daily smoking was uncommon among University of Otago students relative to the general New Zealand population aged 15–24 years. Moreover, rates were lower than those recorded for students in other developed countries and appear to have decreased over the last decade. Smoking among Maori women, in particular, is a priority area for intervention.

New Zealand, like several other developed countries, has witnessed a period of decline in smoking and related mortality. Smoking rates, however, remain at levels sufficient to cause health problems for a significant portion of the population, and for Maori in particular. There are considerable human and economic costs in the treatment and care of smokers, as well as persons affected by second-hand smoke.[1] In New Zealand, it is estimated that 24% of females and 26% of males smoke daily.[2]

A recent study examined smoking among university students in 23 countries, covering Europe, the Americas, Asia, and Africa, but not Australasia.[3] For women, the age-adjusted prevalence (and 95% confidence interval [CI]) of smoking (daily plus occasional) ranged from 2% (-2%–6%) in Thailand to 46% (40%–51%) in Spain. For men, the range was 14% (9%–19%) in Thailand to 47% (43%–51%) in Portugal.

In an anonymous survey of health habits among tertiary students (mean age 18.3 years) living in residential halls in New Zealand,[4] daily smoking was reported by 10% of women and 10% of men. The rates found in the halls-based study were considered low compared with national survey data, which suggest that among youth aged 15–24 years, 29% of women and 25% of men smoke daily.[2] The low prevalence of daily smoking was assumed to be attributable, at least in part, to the characteristics of the study sample—students living in halls of residence—who may have been unrepresentative of the wider student population, and of New Zealand youth in general.

The aims of this study were to estimate the prevalence of smoking, by gender and ethnicity, in a university student population; and to compare estimates with those for

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university students in other countries, and with other similarly aged youth in New Zealand.

Methods

The sampling frame for the survey was the University of Otago enrolment list as at 15 April 2002. In 2002, 7.3% of University of Otago students identified themselves as Maori on their enrolment form. We used the University's classification of ethnicity, which closely resembles that used by Statistics New Zealand.[5] When enrolling, students were asked to 'indicate the group or group(s) with which you identify', from the following list: 'European/Pakeha or New Zealand European, New Zealand Maori, Samoan, Cook Island Maori, Tongan, Niuean, Tokelauan, Chinese, Indian, Fijian, Other Pacific Island (please specify), Other Asian, or Other (please specify).' Where multiple categories were selected, we used the Statistics New Zealand algorithm to form three level 1 categories: Maori, European, and Other.[5]

Funding was available to conduct a survey with 1500 respondents. For the purpose of increasing the precision of prevalence estimates for Maori, we sought to recruit 218 Maori students (double the number expected from a simple random process); with the balance of the sample being non-Maori. Assuming a 79% response rate (the lower bound of a confidence interval from a pilot study estimate) we invited random samples of 276 Maori and 1634 non-Maori students (total n = 1910), to complete an Internet-based survey of their alcohol use and related behaviours. A more detailed description of the study population, sampling, recruitment, questionnaire, resulting coverage, and non-response error, is available elsewhere.[6,7]

In a section of the survey headed 'Tobacco Smoking', students were asked: 'Which of the following best describes your use of cigarettes?'. Responses were 'Never smoked cigarettes at all, or never smoked regularly', 'Do not smoke now but used to smoke regularly, 1+ (cigarettes) per day', 'Occasionally smoke, on average, less than one (cigarette) per day', and 'Currently smoke cigarettes regularly, 1+ per day'. The last three groups are hereafter referred to as 'Ex-smokers', 'Occasional smokers' and 'Daily smokers' respectively.

For the purpose of international comparisons, the last two categories were collapsed to produce the category 'Current smokers'. The international study by Steptoe et al included a response category for cigar or pipe smoking, which they collapsed into the category 'Current smoking'.[3] This was not separately specified in our present study, so it is possible that, in estimates of prevalence, we failed to include individuals who smoke only cigars or pipe tobacco.

In a separate study,[7] in which we examined possible non-response biases in relation to drinking prevalence rates, we compared the drinking levels of late responders (ie, those students who participated after the commencement of telephone reminders, day 20 of the survey administration) with early responders (ie, those students who responded prior to day 20), and found that late responders were, on average, heavier drinkers. However, when this was taken into account in the estimation of alcohol consumption for the entire sample (ie, inclusive of non-respondents), it was concluded that non-response bias was negligible.[7] We applied the same analysis in the present study, in which we computed the prevalence of smoking for late respondents, applied it to the non-respondents, and produced an adjusted prevalence estimate for the entire sample (n = 1910).

Results

Responses were received from 1564 students (response rate 82%), of whom 219 were Maori (response rate 79%). The mean ages (standard deviation) of women and men were 20.4 (2.4) and 20.7 (2.6) respectively. For both genders, 14% were Maori, 70% were European, and 16% were of other ethnicities. Students reported the following residence types: house/flat sharing (63%), residential hall (20%), living with parents (9%), and 'other' (8%).

There were 19 women (2%) and 19 men (3%) who did not complete the section of smoking, leaving a sample of 883 women and 643 men for analysis. These missing cases were not included in the calculation of prevalence estimates.

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Table 1 presents smoking prevalence rates (and 95% confidence intervals) by gender. Differences in smoking rates did not vary significantly by gender ($?^2 = 2.28$, $p = 0.68$).

Table 1. Smoking prevalence rates by gender

	Women (n = 883) % (95% CI)	**Men (n = 643)** % (95% CI)
Never smoked	73 (70–76)	74 (71–78)
Ex-smoker	7 (5–9)	6 (5–9)
Occasional smoker (<1 cigarette per day)	10 (8–13)	9 (7–11)
Daily smoker (1+ cigarette per day)	10 (8–12)	10 (8–13)

CI = confidence interval

For the purpose of international comparison, the 'occasional' (<1 cigarette per day) and 'daily smoking' (1+ cigarette per day) categories were combined. Table 2 presents prevalence rates of any smoking (and 95% confidence intervals) by gender and ethnicity, and shows Maori women as the group most likely to smoke, with almost 1 in 3 either an occasional smoker (n = 21, 16%) or a daily smoker (n = 20, 16%).

Table 2. Smoking (occasional or daily) by gender and ethnicity

	Women (n = 883) % (95% CI)	**Men (n = 643)** % (95% CI)
European	20 (17–23)	20 (17–25)
Maori	32 (24–41)	14 (8–24)
Other	8 (4–14)	16 (10–25)
All	**20 (17–23)**	**19 (16–23)**

CI = confidence interval

Steptoe et al aggregated data for countries in Western Europe and the USA.[8] The prevalence of smoking (occasional or daily) for women and men was 28% (23%–33%) and 31% (27%–34%), respectively. The overall rates in the present study (20% and 19%, respectively) fall well below the lower bounds of the confidence intervals for those estimates.

For the purpose of comparison with New Zealand data, only the daily smoking category can be used. The daily smoking rates for University of Otago students (10% for both females and males) are considerably lower than rates for youth aged 15–24 years in the general New Zealand population (29% and 25%, respectively).[2] In a study of a birth cohort aged 21 years in 1993,[9] daily smoking was reported by 15% of tertiary students [n = 351] (personal communication, Associate Professor Rob McGee, Dunedin Multidisciplinary Health and Development Study, 1980–94).

Table 3 presents smoking prevalence rates as a function of response latency: whether the respondent completed the survey before (early) or after (late) the commencement of telephone reminders. A chi-square ($?^2$) analysis showed that response latency

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varied as a function of smoking status ($?^2$ = 9.84, p = 0.02). This appeared to be due to the preponderance of daily smokers in the late response category. If the prevalence of daily smoking among late respondents applied to the non-respondents (n = 346), the prevalence of daily smoking in the entire sample (n = 1910) would have been 10.6% [((346*14.8%)+152)/1910], which is less than one percentage point higher than the estimate presented in Table 1.

Table 3. Smoking prevalence rates by survey response latency

	Response latency			
	Early		Late	
	n	(%)	n	(%)
Never smoked	928	(74.7)	195	(68.9)
Ex-smoker	82	(6.6)	21	(7.4)
Occasional smoker (<1 cigarette per day)	123	(9.9)	25	(8.8)
Daily smoker (1+ cigarettes per day)	110	(8.8)	42	(14.8)

Discussion

In this random sample of university students, 20% of survey respondents reported current smoking (occasional or daily), while 10% reported daily smoking. These rates are low in comparison with national peer norms and the prevalence of smoking among university students in other developed countries. Comparison with a cohort of 21 year-olds in 1993, suggests that the prevalence of smoking among students decreased by approximately one third in the last 10 years. Of concern, however, are the rates of smoking among subgroups of students; in particular, Maori women, who are 60% more likely to smoke than female students of New Zealand European ethnicity. Notably, a different pattern was evident for Maori men, whose smoking prevalence was less than half that of Maori women.

Strengths of this study include the use of random sampling, the high response rate, the likely low non-response bias, and use of a computerised survey—a format shown to increase reporting of high risk behaviours.[10] Small differences in the way smoking behaviour was assessed across studies limit confidence in estimates of differences in prevalence estimates across time and country.

A New Zealand study of 18-year-olds involved in a longitudinal study found that interview-based self-reported smoking status was highly correlated with saliva cotinine levels (r = 0.75).[11] Moreover, research suggests that respondents are more likely to report stigmatised behaviours via computerised survey instruments than by pen-and-paper surveys.[10] Thus it is likely that the estimates reported here do not significantly under-estimate the true smoking prevalence among University of Otago students.

The study sample was drawn from a single university (University of Otago), although it should be noted that University of Otago students come from all over New Zealand. In 2002, 78% of enrolled students gave a non-Dunedin address as their usual residence.[12] Relative to all New Zealand universities in 2003, the University of Otago has a higher percentage of European students (70.0% versus 56.4%), a slightly lower

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percentage of Maori students (7.0% versus 8.3%), and lower percentages of Pacific (1.7% versus 3.7%) and Asian students (9.5% versus 12.7%).[13] The generalisability of the overall smoking prevalence rates to the university sector as a whole is limited by these differences in the distribution of ethnicity. However, the smoking prevalence rates for European and Maori students can be used separately as estimates of the prevalence in these groups of university students across New Zealand.

The comparison of smoking prevalence by gender alone, masked important variation by ethnicity. While Maori men did not differ significantly from non-Maori men, Maori women had a significantly higher prevalence of smoking than women of European (or other) ethnicity. This pattern mirrors that for young people in the New Zealand population.[1] These differences may contribute to the 10-year disparity in life expectancy between Maori and non-Maori identified in a recent report,[14] and they highlight the need for interventions to reduce inequalities in health. Given the evidence for a range of smoking cessation interventions,[1] efforts are required to ensure that such services are actively promoted among Maori women in particular. A recent evaluation supports the accessibility and effectiveness of Aukati Kai Papa, a culturally appropriate smoking cessation program for Maori.[15]

Author information: Dr Kypros Kypri, Research Fellow, Injury Prevention Research Unit; Joanne Baxter, Senior Lecturer, Department of Preventive and Social Medicine, University of Otago, Dunedin

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Correspondence: Dr Kypros Kypri, School of Medical Practice and Population Health, University of Newcastle, c/o Hunter Population Health, Locked Bag 10, Wallsend, NSW 2287, Australia. Fax: +61 2 4924 6215; email: Kypros.Kypri@hunter.health.nsw.gov.au

References:

1. National Health Committee. Guidelines for smoking cessation: revised literature review and background information. Wellington: Ministry of Health; 2002.
2. Ministry of Health. Taking the pulse: the 1996/97 New Zealand Health Survey. Wellington: Ministry of Health; 1999.
3. Steptoe A, Wardle J, Cui W, et al. An international comparison of tobacco smoking, beliefs and risk awareness in university students from 23 countries. Addiction. 2002;97:1561–71.
4. Kypri K, Langley JD, McGee R, et al. High prevalence, persistent hazardous drinking in New Zealand tertiary students. Alcohol Alcohol. 2002;37:457–64.
5. Statistics New Zealand. Ethnicity – standard classification. Wellington: Statistics New Zealand; 1996.
6. Kypri K, Gallagher SJ. Incentives to increase participation in an Internet survey of alcohol use: a controlled experiment. Alcohol Alcohol. 2003;38:437–41.
7. Kypri K, Stephenson SCR, Langley JD. Assessment of non-response error in an Internet survey of university student alcohol use. Alcoholism Clinical and Experimental Research. In press 2004.
8. Steptoe A, Wardle J, Cui W, et al. Trends in smoking, diet, physical exercise, and attitudes toward health in European university students from 13 countries, 1990–2000. Prev Med. 2002;35:97–104.

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9. Silva PA, Stanton WR, editors. From child to adult. Dunedin: Oxford University Press; 1996.

10. Turner CF, Ku L, Rogers SM, et al. Adolescent sexual behaviour, drug use, and violence: increased reporting with computer survey technology. Science. 1998;280:867–73.

11. Stanton WR, McClelland M, Elwood C, et al. Prevalence, reliability and bias of adolescents' reports of smoking and quitting. Addiction. 1996;91:1705–14.

12. University of Otago Financial Services Division. Statistical tables 2002. Dunedin: University of Otago; 2003.

13. Ministry of Education. Tertiary education statistics 2002. Wellington: Ministry of Education; 2002. Available online. URL: http://www.minedu.govt.nz Accessed February 2004.

14. Ajwani S, Blakely T, Robson B, et al. Decades of disparity: ethnic mortality trends in New Zealand 1980–1999. Wellington: Ministry of Health and University of Otago; 2003. p. 130.

15. Ministry of Health. Evaluation of Culturally Appropriate Smoking Cessation Programme for Maori women and their Whanau: Aukati Kai Paipa. Wellington: Ministry of Health; 2003. Available online. URL: http://www.moh.govt.nz/moh.nsf/0/50be7bea182bcb5bcc256d6c000c5408/$FILE/Aukati%20Kai%20Paipa%202000.pdf Accessed February 2004.

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Reduction in crime by drug users on a methadone maintenance therapy programme in New Zealand

Ian Sheerin, Terri Green, Douglas Sellman, Simon Adamson, and Daryle Deering

Abstract

Aim The study aimed to estimate changes in drug use, crime, imprisonment and societal costs among a sample of Maori and non-Maori injecting drug users (IDUs) on a methadone maintenance therapy (MMT) programme in Christchurch, New Zealand.

Methods Fifty-one non-Maori and 34 Maori IDUs were interviewed to obtain a self-reported history of drug use, crime, imprisonment, and effects on personal health. Information was obtained on drug use and crime before starting MMT and also after stabilisation on MMT. Follow-up interviews were conducted 18 months (mean) after the first interview.

Results Considerable reductions in the frequency of crime occurred—with 60% of participants reporting they committed crimes every day before MMT, compared with only 1% at interview. Large reductions were reported in both expenditure on illicit drugs and income from illegal activities. Reductions in opioid use and crime were similar for both Maori and non-Maori. A significant minority of participants reported continuing some form of crime while on MMT; 29% reported committing at least one offence during the week prior to interview.

Conclusions MMT is associated with a large reduction in the costs of crime and imprisonment among IDUs. This reduction in crime is similar for both Maori and non-Maori.

Drug-related crime is a major cost for the community—incurring insurance, law enforcement, and imprisonment costs, and also involving loss of property. Reduction in crime and imprisonment has been used in many studies as a measure of treatment outcome.[1] There is strong evidence from other Western countries, which shows that high rates of crime are associated with the injecting of illicit opioids.[2,3] Furthermore, it has been postulated that low rates of employment among IDUs, combined with the high costs of illicit drugs, result in many IDUs turning to crime as a way of funding their drug habits.[1,4] Reduction in property crimes have been demonstrated among injecting drug users (IDUs) retained in methadone maintenance therapy (MMT) in North America[1,3] and Australia[5]. In New Zealand, one of the aims of the national methadone protocol is to reduce crime associated with illegal opioid use.[6]

Limited evidence is available on changes in criminal activity associated with MMT for opioid addiction in New Zealand. It has been suggested that reductions in the cost of crime and imprisonment in New Zealand could more than offset the costs of MMT,[7] and that it may help to justify funding of more methadone services to address waiting lists for MMT. A New Zealand study showed high rates of crime among IDUs on the waiting list for MMT in Christchurch, but did not investigate changes in crime after IDUs started MMT.[8] A study of IDUs on MMT in Otago found evidence

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of reductions in drug-related convictions,[9] but did not provide information on types of criminal activities or on costs of crime to society.

In New Zealand, there is concern about ethnic differentials, with Maori (the indigenous people) having lower health status, making up half of the national prison population, and being over-represented in admissions to alcohol and drug services.[10] In 1998/99, 16% of admissions to MMT in Christchurch were Maori (Canterbury District Health Board, personal communication, 1999). However, there is scant information on reduction of crime for Maori IDUs in MMT.

Resource limitations have resulted in waiting lists for MMT. Waiting times have been found to vary by location,[11] with a mean waiting time of 12.7 months in Christchurch (research in progress). There has been a gradual increase in funding for MMT with 3774 patients receiving MMT nationally in 2001.[11] However, there has also been opposition to funding services for drug addiction with a proportion of the community remaining sceptical about the benefits of MMT.[11] Therefore, in order to inform this debate and decisions on resource allocation, it is necessary to research the effects of MMT on changing criminal behaviour and the use of illicit drugs, in addition to health outcomes in the New Zealand setting. This paper reports the findings on changes in crime associated with MMT.

Methods

A preliminary survey (hereafter termed Interview 1) was undertaken of IDUs on MMT in Canterbury with interviews of approximately 1 hour occurring between September, 1999 and November, 2002. Eighty-five randomly selected participants who had been prescribed MMT for at least 3 weeks were recruited (51 non-Maori and 34 Maori) to ensure sufficient Maori participants to examine ethnic differences. At interview, data was obtained on demographics, current drug use, history of the costs of drug use, and criminal activity. A follow-up interview (18 months after Interview 1) was administered to obtain longitudinal data on outcomes of continued treatment, drug use, and criminal activity. The study was approved by the Canterbury Ethics Committee.

At the first interviews (Interview 1), self-reported data were obtained on drug use and criminal activity during the previous week. Information on drug use and crime before MMT was also obtained by self-report. Average illegal earnings before MMT were estimated from self-reported expenditure on drugs before MMT, less $100 per week, which (it was assumed) could have been funded from legitimate income. This was calculated as a conservative estimate of illegal income—given that Adamson and Sellman,[8] in their methadone waiting list sample, found that average illegal earnings per week exceeded the total value of drugs used by approximately $200 per week. In addition, illegal earnings were assumed to be zero if participants reported that they did not commit any crime before MMT or were supplied free-of-charge by another person.

Costs of losses to society due to criminal activity at Interview 1 were estimated in two ways. The first method summed the illegal income from the crimes. The second method used a mixed approach, attributing a value to goods stolen or received based on the open market value of the goods. Other crimes (eg, fraud, prostitution, drug dealing) were valued as in the first method, at the amounts earned by the offender. In neither method was a value assigned to crimes such as breach of parole, disorderly behaviour, wilful damage, or firearms offences.

Information on the details of crimes committed before MMT was not available. Costs of the societal losses of crime before MMT were estimated using only the first method (ie, by the illegal income of participants).

Costs of imprisonment were based on information supplied from the Department of Corrections (2000), which shows an average cost of $41 462 per prisoner per annum in the year 2000 (averaged over all levels of security.) The estimated annual cost of imprisonment before MMT is in year-2000 dollars and was calculated by dividing the average cost of imprisonment per person by the mean number of years since participants started injecting opioids (15.6 years). This approach provides an estimate of the annual costs of imprisonment associated with injecting drug use. Because the ethnic differences were

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not statistically significant in regard to history of imprisonment and time since starting injecting, the overall mean values were used for the purposes of calculating costs of imprisonment.

Data were analysed using 'Statistical Package for the Social Sciences' (SPSS). For statistical tests of significance of changes (in proportions over time), we used McNemar's test. T tests were used to test for differences between mean values.

Results

The mean duration of the current episode of MMT, from stabilising on MMT until Interview 1, was 57 months per participant (52 months for non-Maori and 64 months for Maori; range 2–276 months). The mean number of years between starting to inject and admission to MMT was 15.6 years. Fifty-three percent of the participants were men. The mean age was 35 years for Maori and 36 years for non-Maori. The mean age of starting to inject opioids was 20 years. No ethnic differences in these variables were found. Only 31% of participants reported that they had full-time work before MMT and (at Interview 1) most were on welfare benefits (38% on sickness benefits and 20% on invalids' benefits). Participation in full-time employment reduced from 31% before MMT to 12% at both Interview 1 and at follow-up (significant, $p < 0.01$). During this period, participants who said they were unemployed or on welfare benefits increased from 45% before MMT to 64% at Interview 1 (significant, $p < 0.05$).

There was evidence of widespread criminal activity before participants started MMT. Eighty-nine percent reported that they had criminal convictions, and 61% reported having convictions for crimes that were committed to earn money for drugs. For those with convictions, the mean number of convictions related to drugs and alcohol was 24. Fifty-five percent had a history of imprisonment. For those who had been in prison, the mean total weeks of imprisonment was 108.3, which was spread over a mean of 17 years since these participants were aged 16. The mean number of prison sentences was 3.5. There were no significant ethnic differences in these variables. These data indicate high levels of criminal activity before MMT.

Participants described committing many types of crime before starting MMT in order to get money to pay for illicit drugs. For women, these activities include prostitution, drug dealing, and property crime. For men, the main activities included drug dealing and property crime.

Use of illicit drugs At Interview 1, participants reported that their use of both opioids and benzodiazipines had reduced compared with the 6-month period before they started MMT. All participants reported using opioids (with a mean of 6.8 days per week) before starting MMT. However, at Interview 1, only 10% of non-Maori and 9% of Maori reported that they had used opioids during the previous week ($p < 0.001$, using McNemar's test); see Table 1.

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Table 1. Reductions in opioid use, expenditure on drugs, illegal income, and crime since starting methadone maintenance therapy (MMT)

	Statistic	Non-Maori (n = 51)		Maori (n = 34)		All participants (n = 85)	
		Before MMT	At Int 1*	Before MMT	At Int 1*	Before MMT	At Int 1*
Using opioids†	%	100	10	100	9	100	10
Expenditure on drugs† (per week per person)	Mean $	1144	39	1532	62	1299	48
	SD	1325	76	1584	171	1438	123
Illegal income† (per week per person)	Mean $	1000	111	1405	123	1162	116
	SD	1350	440	1602	469	1461	449
Involved in any crime†‡	%	86	43	91	26	88	36
Involved in daily crime†	%	61	0	59	3	60	1
Days per week spent committing crime†	Mean	4.9	0.7	5.0	0.7	4.9	0.7
	SD	2.8	1.1	2.6	1.5	2.7	1.3

*At Interview 1, participants had been on MMT for a mean time of 57 months; †significant at p <0.001; ‡includes possession and smoking of cannabis, and traffic offences.

Use of illicit benzodiazipines reduced from 48% of IDUs (in the 6-month period before MMT) to 13% in the previous week at Interview 1 (p <0.001). However, participants reported a non-significant increase in use of cannabis with 59% using it before MMT compared with 65% using at Interview 1. There were no significant ethnic differences in changes in use of these substances.

Changes in expenditure on drugs can also be viewed as an indicator of changes in drug use. The reduction in expenditure on illicit drugs was dramatic and was statistically significant (p <0.001) for both Maori and non-Maori (Table 1). Non-Maori reported spending a mean of $1144 per week per person before MMT, which had reduced to $39 per week per person at Interview 1 (p <0.001). Expenditure on drugs by Maori reduced from a mean of $1532 per week per person before MMT to $62 per person per week at Interview 1 (p <0.001). The ethnic difference in expenditure on drugs was not statistically significant. Among all participants, in the 6-month period before starting MMT, mean expenditure on drugs per week per participant was $1299, which reduced to $48 at Interview 1 (p <0.001).

There was no significant gender difference in expenditure on illicit drugs before MMT. The main variable associated with expenditure on drugs was involvement in crime. Participants who reported criminal offending also reported significantly higher expenditure on drugs, with a mean of $1441 per week compared with a mean of $580 per week for people who reported no crime before MMT (p <0.001).

Illegal income Before starting MMT, the mean illegal income for all participants was $1162 per week per person, which reduced to $116 per week per person at Interview 1 (Table 1, significant at p <0.001). There were some non-significant differences by ethnicity in mean illegal income before MMT. However, there were wide variances in

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this population, with estimated illegal income before MMT ranging from $0 to $6900 per week per person. Hence, we conclude that illegal incomes were similar for both Maori and non-Maori IDUs and that the reduction in illegal income while they were on MMT was similar for both groups.

Participants who reported receiving illegal income reduced dramatically from 86% before MMT to 12% at Interview 1 ($p < 0.001$). This reduction was similar for both Maori and non-Maori. In most cases, the illegal income for the week prior to Interview 1was below $400. But there were five people who reported earning more ($500, $637, $1170, $2500, and $3030 respectively), and they had been on MMT for times ranging from 3 to 12 years. The two people with the highest illegal earnings had earned their money mainly from drug dealing. The two women who earned $630 and $1170 made their money mainly from prostitution.

Level of criminal activity Eighty percent of non-Maori and 88% of Maori participants reported that their involvement in crime had reduced considerably since they had been on MMT. Only 14% of non-Maori and 9% of Maori said their involvement was unchanged or increased. Over 90% said their involvement with drug dealers or people committing crimes had reduced.

There was a large reduction in the frequency of crime reported by participants (Table 1). Eighty-eight percent of participants said that they had been involved in crime before MMT, compared with 36% in the previous week at Interview 1 (significant at $p < 0.001$). However, the reduction in the level of crime is perhaps better illustrated by the finding that before MMT, 60% said they were committing crime on a daily basis, compared with only 1% in the previous week at Interview 1 (Table 1, significant at $p < 0.001$). There were no significant ethnic differences in these changes.

Reduction in crime is also indicated by the finding that before MMT, participants spent a mean of 4.9 days per week committing crimes, compared with 0.7 days per week at Interview 1 (Table 1, $p < 0.001$). There were no significant ethnic differences.

Arrests The pattern of arrests since starting MMT is an indicator of the proportion of methadone patients who continue crime. At Interview 1, 33% of non-Maori and 47% of Maori reported they had been arrested for crimes committed since starting MMT (42% of all participants). For those who had been arrested, the mean number of arrests was 3.2 for both Maori and non-Maori. Only two of these arrests resulted in the persons being released without charge. All other arrests resulted in convictions, most commonly with fines and/or periodic detention. There were no differences evident between Maori and non-Maori in the pattern of convictions. This pattern of arrests occurred over a mean time of 57 months on MMT, a long period during which it is possible that the rate of arrests may have reduced.

At follow-up, 18 months (mean) after Interview 1, 17 participants (20%) said they had been arrested in the previous year. Only one of these arrests resulted in release without charge. This indicates that a proportion of IDUs continue criminal activity, even after a mean time of almost 5 years on MMT.

Types of criminal offences at Interview 1 At Interview 1, the three types of offences that were most frequently reported by participants were drug dealing (11%), benefit fraud (13%), and traffic offences (12%) (see Table 2). Five percent of participants reported either cultivation of cannabis, breach of supervision, or breach of parole,

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respectively. Property crimes, such as thefts and receiving stolen goods, were reported by few people. There were no differences evident between Maori and non-Maori in the types of offences reported. Excluding traffic offences and possession of cannabis, 29% of participants reported committing at least one offence in the week prior to interview. However, 37% of non-Maori and 18% of Maori reported committing at least one offence (Table 2); the difference by ethnicity was significant at p = 0.05. At Interview 1, most of the people who reported supplying drugs said they were doing so as a favour to other people (at little or no financial gain). Two people reported financial gains from dealing ($2500 and $3000 respectively).

Table 2. Types of criminal offences reported by participants on MMT in the previous week (at Interview 1)

Criminal Offence	Non-Maori (n = 51)	Maori (n = 34)	All participants (n = 85)
	%	%	%
Supplied and/or manufactured drugs	12	9	11
Cultivation	6	3	5
Supplied liquor to an under age person	0	3	1
Theft of drugs	2	0	1
Other thefts	4	0	2
Received stolen goods	0	6	2
Fraud involving welfare benefits and/or ACC	16	9	13
Breach of supervision or parole	8	0	5
Wilful damage	2	0	1
Disorderly behaviour	2	0	1
Breach of non-molestation order	2	0	1
Firearms offences	2	0	1
Operating a brothel	0	1	1
Prostitution (soliciting)	2	3	2
Traffic offences	12	12	12
At least one of the above offences (excluding traffic offences)*	37	18	29

Note: Some participants said they committed more than one crime. A total of 81 offences were reported.

*The difference by ethnicity was significant at chi-square = 3.78, p = 0.05, degrees of freedom = 1

There was a large reduction in involvement in prostitution for both Maori and non-Maori (p <0.001) For Maori women, 89% said they earned money from prostitution before MMT, compared with only 6% at Interview 1. Sixty-eight percent of non-Maori women said they had been involved in prostitution before MMT, while only 5% remained involved at Interview 1. The ethnic difference was not statistically significant.

Reduction in costs of crime Two components of the societal costs of crime can be estimated from this research—the cost of imprisonment and the cost of losses due to criminal activity.

Before MMT, average costs of imprisonment across the whole sample were $3067 per person per year compared with $2073 at Interview 1. The reduction in cost averaged $994 per participant per year and applied to both Maori and non-Maori.

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Information was not available on the level of offending according to time on MMT, but the follow-up interviews, at 18 months (mean) after Interview 1, revealed that six participants had been imprisoned between Interview 1 and follow-up. This indicates that, in any given year, approximately 5% of these participants will be imprisoned while they are on MMT.

The illegal income of IDUs can be used as an estimate of the cost to society of losses incurred through theft, fraud, etc. The mean illegal income per IDU dropped from $1162 per week before MMT to $116 per week at Interview 1—a reduction of $1046 per week or $54 392 per year (Table 3). This reduction in societal costs was similar for both Maori and non-Maori. This is likely to be an underestimate of the reduction since the value to society of the loss both before MMT and at Interview 1 is likely to be much higher than the amount of illegal income. For example, the study found that the value to society of the losses associated with the crime reported at Interview 1 totalled $13 180 for the 81 offences that were identified in the week prior to interview. This is an average of $155 per IDU per week, compared to the $116 generated in illegal income.

Table 3. Reduction in costs of crime to society (per IDU per year)

	Cost to society ($) Before MMT	Cost to society ($) At Interview 1	Reduction in societal costs ($)
Illegal income	60 424	6032	54 392
Imprisonment	3067	2073	994
Total*	**63 491**	**8105**	**55 386**

*This does not include costs of law enforcement or the judicial system

Discussion

This study provides data showing substantial reduction in crime among IDUs who are retained in MMT. High rates of criminal activity before MMT were followed by reductions in crime after stabilisation on MMT.

The findings in this study of high crime rates before MMT is supported by an earlier study of IDUs on the waiting list for MMT in Christchurch. Adamson and Sellman found that 88% of people on the waiting list for MMT in Christchurch reported receiving illegal income in the week preceding interview.[8] The main types of illegal activity they recorded were drug dealing, property crime, and prostitution. Adamson and Sellman found that 61% reported committing property crimes in the 7 days before interview (a mean of 8.4 crimes). Seventy-two percent reported drug-related crimes, including supply and cultivation.

Most people cannot sustain, for any length of time, a drug habit that costs an average $1299 per week by financing it from normal paid work. Although, some people reported selling possessions, borrowing, and using savings to buy drugs, this was usually sustained for only short periods of time before participants turned to other ways of financing their habits. Research (in progress) has shown that, before MMT, most participants financed their drug habits from sources of income other than from normal paid work—notably from property crime, drug dealing, and prostitution.

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Adamson and Sellman found a mean illegal income of $1079 per week per person among people on the waiting list for MMT.[8] This figure is similar to the mean estimated illegal income before MMT of $1162 per week for participants in this sample.

This current study has demonstrated that IDUs being on MMT in New Zealand is associated with substantial reductions in expenditure on drugs, crime, and imprisonment. The data indicate that most of these IDUs have stopped crime since being on MMT. However, approximately 29% continue significant criminal offending regardless of the length of time on MMT.

This study indicates reduced societal costs of crime per participant of $55 386 per year per IDU. However, this estimated reduction in costs is low, because costs of property crime to victims are higher than the amount earned illegally by offenders who (for example) sell stolen goods at lower than market value. Adamson and Sellman[8] found that the market value of crime was 2.3 times the amount earned by the offenders who were IDUs on a waiting list for MMT in Christchurch. Similarly, studies of crime committed by IDUs in the USA, estimated the legal market value of stolen goods was three times the illegal income of IDUs.[2,13]

By following the methods of Adamson and Sellman,[8] and valuing the losses from crime at 2.3 times the illegal income, the societal cost of the loss due to criminal activity before MMT is estimated at $2673 per week per IDU. Comparing this with the $155 per week at Interview 1, an upper estimate of the reduction in societal costs of losses from criminal activity is $2518 per IDU per week (or $130 936 per annum). Including the cost reduction for imprisonment yields, an upper estimate of the reduction in societal costs of crime is $131 930 per IDU on MMT.

Both the lower and upper estimates of costs of crime to society presented here are conservative since they exclude the costs of law enforcement and the judicial system. Also, no monetary value has been placed on losses due to crimes such as assault, offensive behaviour, illegal possession of firearms, or breach of probation.

We found no major ethnic differences in drug use and crime. Research (in progress) is finding that there are ethnic differences that are related to family issues and to employment. However, the large reductions in illicit drug use and crime were similar for both Maori and non-Maori.

This study had sufficient power to detect moderate-to-large ethnic differences that may be significant from a clinical point of view. However, it is possible that a larger sample size may have detected smaller ethnic differences that may be of interest from a population health viewpoint (such as in expenditure on illicit drugs before MMT, or in involvement in prostitution).

Different studies have used different measures of crime, including official statistics.[5] However, official arrests are not necessarily an accurate indicator of crime because many offences may go either unreported or unsolved. Self-report is a different method of measuring crime that may be more sensitive as it includes unreported and unsolved crimes. There is evidence that under safe and confidential conditions (as in this study), methadone patients give accurate reports about their drug use and crime.[9]

There is evidence from other countries that crime rates are reduced among IDUs in MMT.[1] However, it has also been found that retention in treatment and patient

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outcomes vary according to the practices of different methadone programmes.[1,5] Hence, the findings of this current research should only be generalised to other settings with caution.

A potential methodological weakness of this study is that it relies on participants' memory recall of their drug use and crime before they started MMT. However, the validity of these data is supported by a close similarity to the findings of Adamson and Sellman.[8] Furthermore, the reductions in crime and illicit opioid use are of such magnitude that pre-MMT drug use and crime would have to be grossly over-estimated to alter the fundamental conclusion that significant reductions in societal costs occur following commencement of MMT.

In summary. our results indicate substantial quantifiable societal benefits of MMT that exceed the costs of MMT (estimated at $4497 per person per annum.) From a societal standpoint, there is an argument to improve access to MMT and to reduce waiting lists. These results also raise a resource allocation issue in so far as the costs of MMT fall on the health budget while the benefits in terms of reduced crime are accrued in other sectors such as the justice system, private firms, and households. If such benefits could be explicitly linked to health-sector funding decisions, the wider societal effects of improving access to MMT could be included in decisions on the level of resourcing of MMT programmes.

Author information: Ian Sheerin, Health Economist and Lecturer, Department of Public Health and General Practice; Terri Green, Senior Lecturer; Douglas Sellman, Lecturer; Simon Adamson, Lecturer; and Daryle Deering, Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences, University of Otago, Christchurch

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Correspondence: Ian Sheerin, Department of Public Health and General Practice, Christchurch School of Medicine and Health Sciences, PO Box 4345, Christchurch. Fax: (03) 364 3614; email ian.sheerin@chmeds.ac.nz

References:

1. Ward J, Mattick RP, Hall W (eds). Methadone maintenance treatment and other opioid replacement therapies. Amsterdam: Harwood Academic; 1998.
2. McGlothlin WH, Anglin MD. Shutting off methadone. Costs and benefits. Arch Gen Psychiatry 1981;38:885–92.
3. Hubbard RL, Marsden ME, Rachal JV, et al. Drug abuse treatment – a national study of effectiveness. Chapel Hill: University of North Carolina Press; 1989.
4. Gerstein DR, Harwood HJ, editors. Treating drug problems: a study of the evolution, effectiveness and financing of public and private drug treatment systems. Washington DC: National Academy Press; 1990.
5. Bell J, Hall W, Byth K. Changes in criminal activity after entering methadone maintenance. Br J Addiction. 1992;87;2:251–8.

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6. Ministry of Health. National protocol for methadone treatment in New Zealand. Wellington; 1996.

7. Sellman JD, Hannifin J, Deering D, Borren P. Delivery of treatment for people with opioid dependence in New Zealand: options and recommendations. A commissioned paper for the Ministry of Health, 1996 Available online. URL: http://www.moh.govt.nz/moh.nsf/0/f42340536703d2a24c25667a0072aef6/$FILE/opiod.pdf Accessed February 2004.

8. Adamson SJ, Sellman JD. The pattern of intravenous drug use and associated criminal activity in patients on a methadone treatment waiting list. Drug Alcohol Rev. 1998;17:159–66.

9. Dore GM, Walker JD, Paice JR, Clarkson S. Methadone maintenance treatment: outcomes from the Otago methadone programme. NZ Med J. 1999;112:442–5.

10. Durie M. Mauri ora: the dynamics of Maori Health. Auckland: Oxford University Press; 1998.

11. Sellman JD, Harvey M, Deering DE. South Island methadone project report. New Zealand: a commissioned paper for the Ministry of Health, 2001.

12. Department of Corrections. Forecast report 1 July 1999–30 June 2000. Wellington: Department of Corrections; 2000.

13. Anglin MD, Speckart GR, Booth MW, et al. Consequences and costs of shutting off methadone. Addict Behav. 1989;14:307–26.

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Patterns of amphetamine use in New Zealand: findings from the 2001 National Drug Survey

Chris Wilkins, Megan Pledger, Krishna Bhatta, and Sally Casswell

Abstract

Aims To measure the level of heavy amphetamine use, poly drug use, and intravenous drug use by amphetamine users in New Zealand.

Methods Using a Computer Assisted Telephone Interview (CATI) system, a national sample of approximately 5500 people (aged 15–45 years) were interviewed about their recreational drug use.

Results Five percent of the sample (4.3–5.7) had used stimulants (uppers, speed, amphetamine, methamphetamine) in the last year. Eighty-one percent of these users used stimulants once-a-month or less frequently. Twenty-two percent used half a gram of stimulants or more on a typical occasion. Stimulant users used an average of 6.4 drug types in the last year (range 1–17, standard deviation [SD] 2.8) and 4.2 drug types in the last month (range 0–13, SD 2.1). Three percent of last-year stimulant users (0.7–4.6) had used a needle to inject drugs in the previous year.

Conclusions About one in five amphetamine users used quantities of amphetamine in a single session that have been identified in previous research as being hazardous levels. High levels of poly drug use among amphetamine users indicate users may be at risk of problems from a range of drug types and combinations of drug types, and not just from amphetamine alone. Ongoing monitoring is required to identify if increased amphetamine use is a source of increased intravenous drug use.

New Zealand has recently experienced a rapid rise in the use and manufacture of powerful amphetamines, such as methamphetamine.[1–3] Anecdotally, the rise in amphetamine use in New Zealand has been implicated in increases in hospital admissions for drug-induced psychosis,[4] increases in street robbery and car conversion,[5] and increases in violent crime.[5] While these consequences are consistent with the experience of increased amphetamine use elsewhere,[6,7] statistics on criminal offending and hospital admissions in New Zealand do not routinely record the drug type involved in an incident and so it has been impossible to precisely measure the impact of increased amphetamine use. Understanding the implications of growing amphetamine use is further complicated by the time lag of 12 to 18 months, which is commonly experienced by users between initial use and progression to problematic use.[8,9]

Overseas, clinical research on amphetamines,[9,10] and studies of amphetamine users,[11–14] have identified the central role that the route and pattern of amphetamine-use plays in the risk of users experiencing serious problems. Hall and Hando found that amphetamine users reporting intravenous administration,[11] using twice-a-week or more, and using more than half a street gram in a single session were more likely to experience adverse psychological effects, dependency, and report violent offences. Other studies have noted a link between the increased use of amphetamines by young

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people, and rises in intravenous drug use by these age groups, suggesting this is a response by some amphetamine users to growing tolerance.[15–18] Studies elsewhere have also found amphetamine users to be extensive poly drug users.[11,14,16,19,20] Other stimulant type drugs, such as cocaine, were commonly used in combination with amphetamines, while opioids and tranquillisers were used to self medicate against adverse side effects. This poly drug use increased the likelihood of users experiencing problems.[19,21,22]

This paper presents findings from the 2001 National Drug Survey on the patterns of amphetamine use in New Zealand. Data is presented on the frequency and amount of amphetamine used, and the extent of poly drug use and intravenous drug use by amphetamine users.

Methods

In 2001, using a Computer Assisted Telephone Interview (CATI) system, a national sample of approximately 5500 people aged 15–45 were interviewed about their recreational drug use. Telephone numbers were selected using a stratified random digit dialling method so that each household (of a particular stratum) nationwide had an equal chance of being called. In order to represent the different socioeconomic characteristics of the population, the country was divided into 33 strata. A proportionate sample from each stratum was then taken. Within each household, one person was randomly selected for an interview. The sample was weighted to adjust for household size. Interviewers received intensive training at the beginning of the survey, and a supervisor was present at each shift to monitor the quality and consistency of interviewing and to handle any problems. Each telephone was tried at least 10 times in an effort to reach those persons seldom at home. An 80% response rate was achieved. Further details of the methodology can be found in Wilkins et al.[23]

Respondents were asked about their use of alcohol, tobacco, cannabis—and 22 other drug types, such as cocaine, crack, heroin, and LSD. The amphetamine drugs were referred to by the broad term 'stimulants', which the interviewer described as meaning 'uppers, speed, amphetamine, and methamphetamine'. Respondents were asked about use of other stimulant-type drugs, such as cocaine, crack cocaine, and ice (ie, crystal methamphetamine), in separate questions of the interview.

Those persons who had used stimulants in the last year were asked a range of additional questions about their patterns of use, including how many times they had used stimulants in the last year, and the quantity of stimulant they used on a 'typical occasion'. Anecdotally, the quantities of amphetamine most commonly used in New Zealand were lines, points (approximately 0.1 gram) and grams of powder. The quantity question included coded amounts from 0.1 gram up to 28 grams. The equivalent amount in lines and points was included in brackets next to the appropriate quantity to facilitate the identification of the amount typically used. Respondents could also indicate if they only used 'pills/tablets' or 'liquid' amphetamine.

In a separate section of the interview, respondents were asked if they had ever used a needle to inject drugs for recreational purposes and how many times they had done so in the last year. Respondents were not asked directly what drug types they had injected. However, the drug types used by a respondent could be identified from other parts of the interview, and the injectable drugs are essentially limited to the opioids and amphetamines. What is reported is intravenous drug use by amphetamine users rather than intravenous amphetamine use. The confidence levels reported are at the 95% level.

Results

Prevalence Overall, 5.0% (4.3–5.7) of the sample had used stimulants (uppers, speed, amphetamine, methamphetamine) in the last year (n = 275). Users were overwhelming male (70%) and in the 18–29 year old age groups (Figure 1). For males, 5.7% (2.4–9.0) aged 15–17 years, 15.0% (8.7–21.2) aged 18–19 years, 12.3% (8.8–15.9) aged 20–24 years, and 11.3% (7.8–14.7) aged 25–29 years had used stimulants in the last year.

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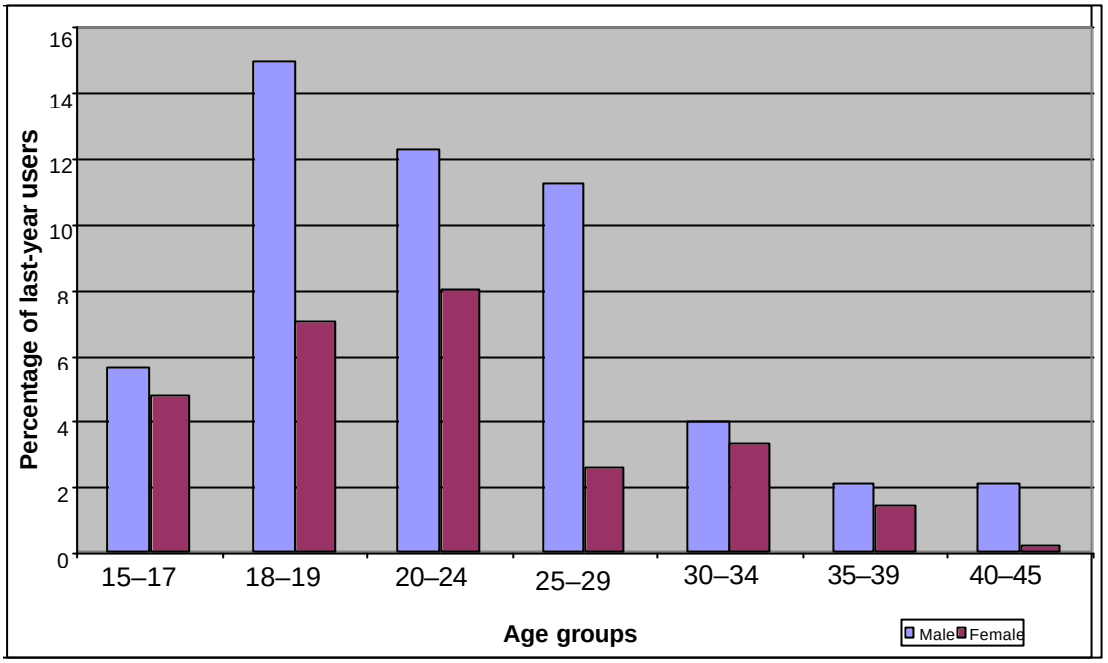
Figure 1. Last-year use of stimulants (by gender and age group) 2001



Frequency of use Fifty-three percent of last-year stimulant users had used these drugs 1-2 times in the last year (Figure 2). A further 20.1% had used them 3-5 times in the last year. The other frequencies of use reported in the last year were 7.7% using 10-19 times (about once every month), 3.1% using 50-59 times (about once-a-week), and 1.0% using 100-109 times (about twice-a-week). Only 0.2% of last-year users said they used stimulants 350-359 times in the last year (about daily).

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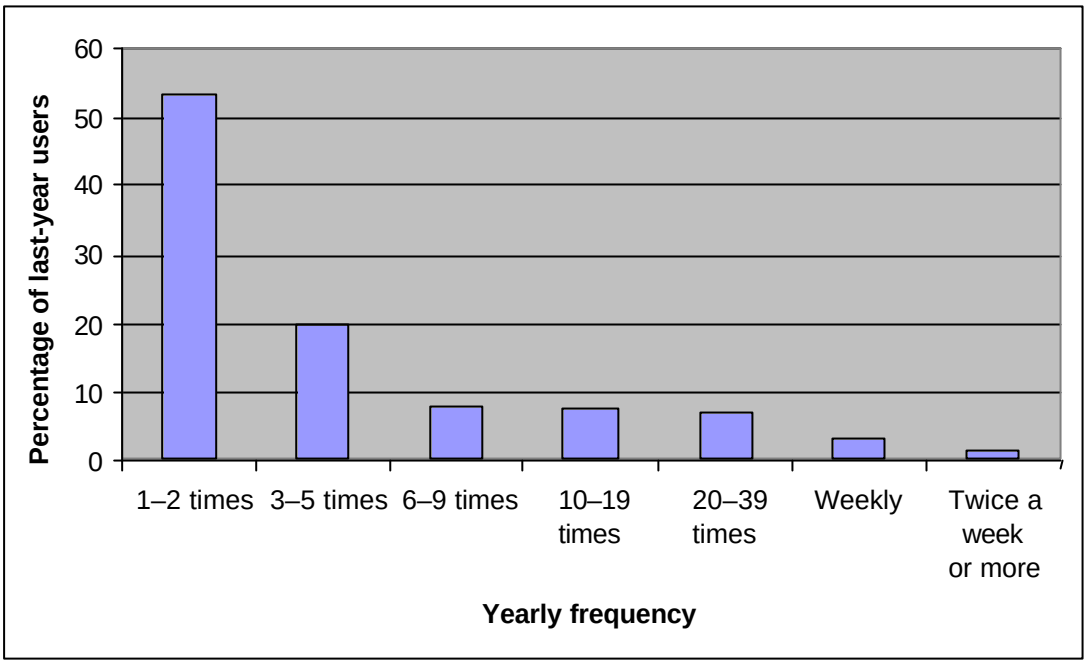
Figure 2. Frequency of use of stimulants in the last 12 months, 2001



Quantity used About 7% (6.8%) of last-year stimulant users indicated they only used pills/tablets of amphetamine. No respondents reported only using liquid amphetamine.

Of those users who used powder amphetamine (93.2% of all users), the most popular amount used on a typical occasion was one line (0.1 gram) [32.3%], followed by two lines (0.2 gram) [27.7%], and five lines (0.5 gram) [13.3%], respectively (Figure 3). Five percent of last-year users used 1 gram of stimulants on a typical occasion.

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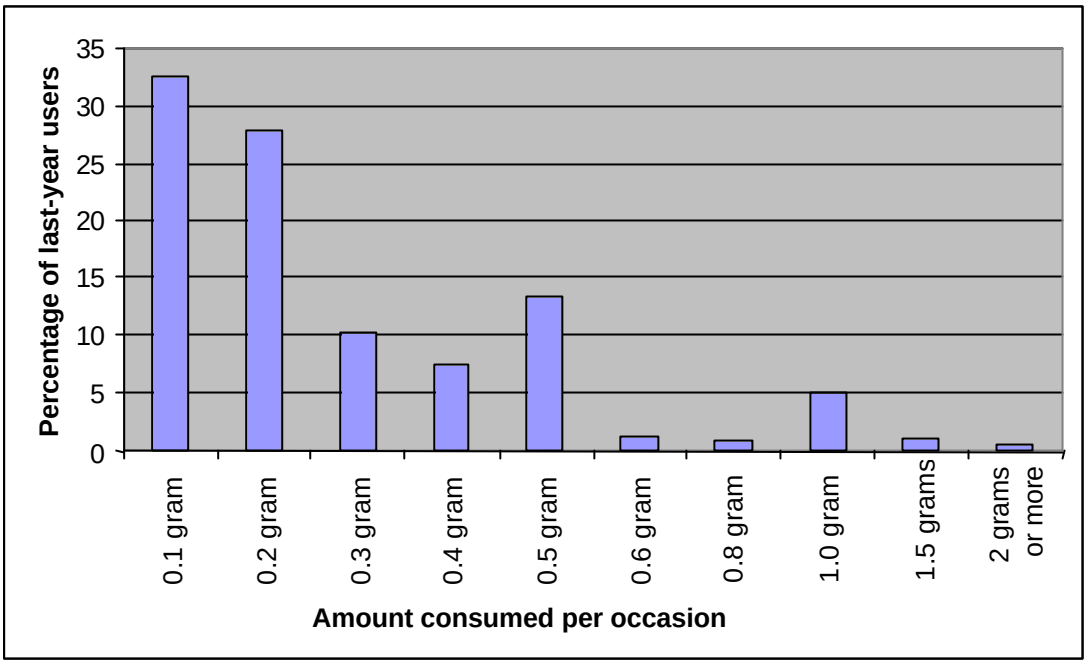
Figure 3. Amount* of stimulant used on a typical occasion, 2001



*For those users who did not consume pills or liquid

Poly drug use The other drug types most commonly used in the last year by stimulant users were alcohol (92.5%), cannabis (85.7%), tobacco (72.6%), skunkweed [hydroponic cannabis] (65.8%), LSD (44.7%), ecstasy (43.4%), magic mushrooms (26.5%), ice [crystal methamphetamine] (15.0%), rush [amyl nitrate, butyl nitrate] (14.3%), kava (12.8%), GHB [gamma-hydroxybuterate] (10.6%), cocaine (9.3%), and ketamine (9.1%); see Table 1. Last-year stimulant users had tried an average of 8.5 drug types ever (range 1–20, SD 3.3), used an average of 6.4 drug types in the last year (range 1–17, SD 2.8), and used an average of 4.2 drug types in the last 30 days (range 0–13, SD 2.1).

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Table 1. Percentage of last-year users of stimulants who used other drugs in the last 12 months, 2001

	Ever used				Used in the last 12 months				Used in the last 30 days			
	Last-year stimulant users		Whole sample		Last-year stimulant users		Whole sample		Last-year stimulant users		Whole sample	
Stimulants (amphetamine/ methamphetamine)	100.0		11.0	(10.1–11.9)	100.0		5.0	(4.3–5.7)	32.4	(25.8–39.1)	1.6	(1.2–2.0)
Alcohol	93.0	(89.1–96.9)	86.5	(85.5–87.6)	92.5	(88.6–96.4)	85.4	(84.3–86.5)	88.0	(83.3–92.8)	77.3	(76.0–78.6)
Tobacco	91.4	(87.4–95.4)	64.1	(62.7– 65.5)	72.6	(66.1–79.1)	34.6	(33.1–36.0)	65.9	(59.1–72.6)	28.1	(26.8–29.5)
Ice (crystal methamphetamine)	18.2	(12.6–23.7)	1.3	(1.0–1.7)	15.0	(9.7–20.2)	0.9	(0.6–1.2)	3.8	(1.1–6.4)	0.2	(0.1–0.4)
Cannabis	97.9	(96.1–99.8)	52.1	(50.6–53.6)	85.7	(81.2–90.2)	20.3	(19.1–21.5)	65.1	(58.7–71.5)	10.5	(9.6–11.5)
Ecstasy	49.2	(42.3–56.1)	5.4	(4.7–6.0)	43.4	(36.6–50.2)	3.4	(2.8–4.0)	15.2	(10.3–20.2)	0.9	(0.6–1.2)
Cocaine	19.0	(13.8–24.2)	3.2	(2.7–3.7)	9.3	(5.5–13.1)	0.6	(0.4–0.9)	0.7	(0.0–1.7)	0.0	(0.0–0.1)
Crack cocaine	3.8	(1.3–6.3)	0.3	(0.1–0.4)	1.5	(0.0–3.3)	0.1	(0.0–0.2)	0.2	(0.0–0.5)	0.0	(0.0–0.0)
Heroin	3.2	(1.3–5.1)	0.7	(0.5–1.0)	0.5	(0.0–1.2)	0.1	(0.0–0.1)	0.0		0.0	(0.0–0.0)
Ketamine	11.5	(7.1–15.8)	0.7	(0.5–1.0)	9.1	(5.1–13.1)	0.5	(0.3–0.7)	2.0	(0.0–4.1)	0.1	(0.0–0.2)
LSD	69.7	(63.3–76.1)	9.6	(8.8–10.5)	44.7	(37.8–51.6)	3.2	(2.6–3.7)	8.2	(4.2–12.2)	0.5	(0.3–0.7)
Mushrooms	51.2	(44.4–58.1)	8.9	(8.1–9.8)	26.5	(19.9–33.1)	2.4	(1.8–2.9)	7.3	(3.3–11.3)	0.6	(0.4–0.9)
Tranquillisers	18.6	(13.6–23.7)	2.6	(2.2–3.1)	6.9	(3.7–10.1)	0.4	(0.3–0.6)	3.1	(1.0–5.2)	0.2	(0.1–0.3)
Needle	5.9	(2.4–9.3)	0.8	(0.5–1.0)	2.6	(0.7–4.6)	0.2	(0.1–0.3)	0.8	(0.0–1.7)	0.1	(0.0–0.2)
GHB (gamma-hydroxybuterate)	14.9	(10.2–19.7)	1.1	(0.8–1.5)	10.6	(6.5–14.6)	0.8	(0.5–1.0)	1.7	(0.1–3.2)	0.1	(0.0–0.2)
Homebake heroin	11.1	(6.9–15.4)	1.5	(1.1–1.8)	6.8	(3.1–10.4)	0.5	(0.3–0.8)	2.2	(0.0–4.5)	0.2	(0.0–0.3)
Morphine	7.3	(4.1–10.4)	1.0	(0.7–1.3)	3.1	(1.0–5.2)	0.2	(0.1–0.4)	0.7	(0.0–1.5)	0.1	(0.0–0.1)
Poppies	14.5	(9.5–19.4)	2.4	(1.9–2.9)	3.5	(1.0–6.0)	0.3	(0.2–0.5)	1.0	(0.0–2.2)	0.1	(0.0–0.2)
Rush (amyl nitrate–butyl nitrate)	34.9	(28.2–41.6)	4.7	(4.1–5.3)	14.3	(9.5–19.2)	0.9	(0.6–1.2)	4.2	(1.6–6.8)	0.3	(0.1–0.4)

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Skunkweed cannabis	77.5	(72.0–82.9)	14.1	(13.0–15.1)	65.8	(59.5–72.1)	9.4	(8.5–10.3)	45.6	(38.7–52.6)	5.1	(4.4–5.8)
Solvents	11.4	(6.8–16.1)	2.2	(1.7–2.6)	1.1	(0.0–2.4)	0.2	(0.1–0.4)	0.9	(0.0–2.2)	0.1	(0.0–0.2)
Other opiates	7.9	(4.6–11.2)	1.0	(0.7–1.3)	4.6	(2.0–7.2)	0.3	(0.2–0.5)	1.2	(0.0–2.4)	0.1	(0.0–0.2)
Kava	29.5	(23.1– 35.9)	9.6	(8.7–10.5)	12.8	(8.1–17.5)	3.2	(2.6–3.7)	1.9	(0.2–3.5)	0.4	(0.2–0.5)
Other hallucinogenic	84.6	(80.1–89.1)	15.0	(14.0–16.1)	67.9	(61.7–74.1)	6.1	(5.3–6.8)	23.1	(17.1–29.2)	1.6	(1.2–2.0)
Average number of other drugs ever used:					**8.4**							
Average number of other drugs used in the last year:					**6.4**							
Average number of other drugs used in the last 30 days:					**4.2**							

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Last-year needle use A total of 0.2% (0.1– 0.3) of the whole sample (11 people in the weighted survey but 13 respondents) had used a needle to inject a drug in the last year. Seventy-seven percent of these last-year needle users also used one of the opioid drugs in the last year (ie, heroin, homebake, morphine, poppies, other opiates). Sixty-eight percent of last-year needle users also used stimulants.

Discussion

Due to the difficulties of surveying illicit drug users,[24] particularly heavy drug users,[25] the National Drug Survey is likely to under estimate the true number of users. However, well designed CATI surveys with high response rates have been found to produce similar results to other population survey methodologies.[26] The findings presented here are best thought of as providing reliable but conservative estimates of illicit drug use in New Zealand.

The National Drug Survey provides a broad representative picture of the amphetamine using population in New Zealand, including experimental and occasional users. However, the household sample frame may mean that some heavy problematic users who are living on the streets or living particularly erratic lifestyles are missed. This limitation is likely to be particularly relevant with respect to reaching intravenous drug users who are often heavy drug users.

Amphetamine drugs come in varying levels of potency and purity and the strength of amphetamine plays an important role in the risk of experiencing problems. At present the understanding of the strength of the amphetamines being used in New Zealand is largely anecdotal. Seizures of amphetamine in New Zealand are not routinely tested by the authorities for purity levels as this information is not generally central to achieving a prosecution. Approximating the strength of the amphetamines used is made difficult by the different slang names which are developed to identify different types of amphetamine, such as the term ‘pure’ in New Zealand, the loose way these street terms are used by drug dealers and drug users, and the varying ability of users to accurately assess the potency of the drugs they are using depending on their level of knowledge and experience.

Drawing on existing sources, including social histories of amphetamine use,[27] recent analysis of the amphetamine situation in Australia[28] and reports of amphetamine seizures in New Zealand,[1–2,29] it is possible to identify four broad types of amphetamine being used in New Zealand: amphetamine sulphate; methamphetamine powder; ‘pure’ methamphetamine; and ice or crystal methamphetamine. Amphetamine sulphates include diet pills and common prescription medicines, which may have been illegally obtained from legitimate dispensing sources. Methamphetamine is a particularly powerful type of amphetamine.[28] In the powder form, it is usually heavily cut with adulterants. The New Zealand Customs Service reports the normal purity of methamphetamine powder at street level in New Zealand is between 5%–15%.[29] Methamphetamine powder is purchased by the gram or ounce and is consumed in lines of powder. ‘Pure’ is high-potency uncut methamphetamine and is sold by the point (0.1 gram). A point of ‘pure’ is sufficient for a number of doses. Ice or crystal methamphetamine is high-potency crystallised methamphetamine and is generally manufactured and imported from Asia. It is not entirely clear (at present) how different the New Zealand manufactured ‘pure’ is from the Asian crystal methamphetamine.

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Several limitations with the National Drug Survey data from the perspective of estimating the level of heavy amphetamine use in New Zealand must be acknowledged The questions in the National Drug Survey about the quantity of stimulants used referred to use on a 'typical occasion' only. This may not fully capture amphetamine consumption patterns that can sometimes include binge use, where a user consumes large amounts of the drug over several hours or days.[10] This type of use greatly increases the risk of problems such as psychosis.

Second, stimulant users in the National Drug Survey were not asked directly whether they used a needle to inject stimulants and this would have provided a clearer picture concerning the level of intravenous amphetamine use as opposed to merely amphetamine use by intravenous drug users. However, the fact that an amphetamine user is also using a needle to inject other drugs suggests that the injection of amphetamine, if not already occurring, may be a future option as tolerance develops.

With these limitations in mind, several key points about patterns of amphetamine use in New Zealand can be drawn from the data. Over 10% of New Zealand men aged 18–29, the highest using group, had used amphetamines in the previous year in 2001. Many last-year users used amphetamines fairly infrequently—ie, 73% used them five times or less in the previous year. However, while many users also used fairly low doses, 22% used 0.5 gram (or more) of amphetamine on a typical occasion. Poly drug use was common within the amphetamine-using population with the use of a range of illicit drug types at levels many times higher than that of the general population. Of particular concern were the relatively high levels of the use of LSD, ecstasy, cocaine, homebake heroin and intravenous drug use among amphetamine users compared to the general population.

Most of the needle-using amphetamine users also used opioids. It may be the case they are primarily opioid users. Australian research has found that opioid users will switch to other illicit drug types such as cocaine and amphetamines when heroin is in short supply.[30] Opioid users in New Zealand may be simply responding to the recent greater availability of high potency amphetamine relative to the traditional supply of opioids. The small number of intravenous drug users in the National Drug Survey sample makes further analysis problematic. Close monitoring of intravenous drug use within the amphetamine-using population is required in New Zealand to ensure increased amphetamine use is not fuelling increases in intravenous drug use. Other research methodologies than the household population approach used in the National Drug Survey may be more suited to achieving this task.

The infrequency of amphetamine use in New Zealand could be explained by several factors—including the cultural context of its use (ie, it is still being limited to infrequent large dance party events), the 'newness' of the drug, the immaturity of domestic production and supply networks, and/or the effectiveness of police enforcement. Exploring these reasons is beyond the scope of this present paper. However, it interesting to note that the price of amphetamine in New Zealand is higher than in Australia with 1 gram selling for $100-$180 in New Zealand compared to $59-$118 in Australia (based on prices reported in the Illicit Drug Reporting System of $50-$100,[20] and a 'New Zealand dollar to Australian dollar' exchange rate of 0.85).[31] Unfortunately, the price in New Zealand is believed to have fallen dramatically since the establishment of large-scale domestic manufacture in the late 1990s (from $250–$300 per gram in mid-1999 to $100–$180 after that time).[31]

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Ongoing competition between domestic producers may cause this trend to continue (with implications for frequency of use).

It is also important not to overstate the role that the route of administration and the pattern of amphetamine-use play in users experiencing adverse effects. As with all drugs, effects and harms are also dependent on the user's physical condition, psychological state of mind, context of use (eg, at home, at a club, while driving, etc), and whether the drug was used in combination with other substances.[32] In the case of psycho-stimulants, heavy long-term use has been associated with increased sensitivity to dosage.[9] Users who have experienced methamphetamine-induced psychosis have been found to experience relapses of psychosis after only a small subsequent dose of the drug, or even after exposure to a stressful situation.[33,34]

In conclusion, the findings from the 2001 National Drug Survey indicate there is a substantial minority of amphetamine users in New Zealand who use quantities of amphetamine in a single session that have been identified in research elsewhere as hazardous.[11]. This is of great concern as high dosage has been described in the literature as the 'first stage' to other hazardous using patterns, such as intravenous administration and high frequency use.[35] High levels of poly drug use by amphetamine users indicates users may be at risk of problems from other drug types or drug types used in combination with amphetamines, and not solely from amphetamines alone. Ongoing monitoring is required to identify if increased amphetamine use is a source of increased intravenous drug use.

Author information: Chris Wilkins, Researcher; Megan Pledger, Statistician; Krishna Bhatta, Statistician; and Professor Sally Casswell, Director, SHORE (Centre for Social and Health Outcomes Research and Evaluation), Massey University, Auckland

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Correspondence: Dr Chris Wilkins, SHORE (Centre for Social and Health Outcomes Research and Evaluation), PO Box 6137, Wellesley Street, Auckland. Fax: (09) 366 5149; email: c.wilkins@massey.ac.nz

References:

1. Horne B. Policing the illicit use of amphetamine related drugs in New Zealand. Wellington: Wellington Regional Drug Squad, New Zealand Police; 1997.
2. New Zealand Police. Preliminary Assessment of Substance Proposed for Classification or reclassification Under Sections 4-4B of the Misuse of Drugs Act 1975 ("the Act") (Confidential). Wellington: New Zealand Police; 2002.

 © NZMA

3. Wilkins C, Bhatta K, Casswell S. The emergence of amphetamine use in New Zealand: findings from the 1998 and 2001 National Drug Surveys. NZ Med J 2002;115(1166). URL: http://www.nzma.org.nz/journal/115-1166/256/

4. Kay M. Warning on big rise in drug-psychosis cases. Dominion Post 2002 8th August.

5. New Zealand Police. Crime statistics highlight unique issues facing Auckland City Police. Press Release 2002 30th August

6. Joe-Laidler K, Morgan P. Kinship and community: the 'ice' crisis in Hawaii. In: Klee H, editor. Amphetamine Misuse: International Perspectives on Current Trends. Amsterdam: Harwood Academic Publishers; 1997. p. 163–79.

7. Morgan P, Beck J. The legacy and the paradox: hidden contexts of methamphetamine misuse in the United States. In: Klee H, editor. Amphetamine Misuse: International Perspectives on Current Trends. Amsterdam: Harwood Academic Publishers; 1997. p. 135–62.

8. Darke S, Kaye S, Topp L. New South Wales Drug Trends 2001: Findings from the Illicit Drug Reporting System (IDRS). Sydney: National Drug and Alcohol Research Centre, University of New South Wales; 2002. Report No.: NDARC Technical Report No.125.

9. Gawin F, Ellinwood E. Cocaine and other stimulants: actions, abuse and treatment. New England Journal of Medicine 1988;318:1173–82.

10. Chesher G. Pharmacology of the sympathomimetic psychostimulants. In: Burrows D, Flaherty B, MacAvoy M, editors. Illicit Psychostimulant Use in Australia. Canberra: Australian Government Publishing Service; 1993. p. 9–30.

11. Hall W, Hando J. Route of administration and adverse effects of amphetamine use among young adults in Sydney, Australia. Drug Alcohol Rev. 1994;13:277–84.

12. Hall W, Hando J, Darke S, Ross J. Psychological morbidity and route of administration among amphetamine users in Sydney, Australia. Addiction. 1996;91:81–7.

13. Klee H, Morris J. Factors that lead young amphetamine misusers to seek help: implications for drug prevention and harm. Drugs: Education, Prevention and Policy 1994;1:289–97.

14. Hando J, Topp L, Hall W. Amphetamine-related harms and treatment preferences of regular amphetamine users in Sydney, Australia. Drug Alcohol Depend. 1997;46:105–13.

15. McAllister I, Makkai T. The prevalence and characteristics of injecting drug users in Australia. Drug Alcohol Rev. 2001;20:29–36.

16. Peters A, Davies T, Richardson A. Increasing popularity of injection as the route of administration of amphetamine in Edinburgh. Drug Alcohol Depend. 1997;48:227–34.

17. Klee H. A new target for behavioural research - amphetamine misuse. Br J Addict. 1992;87:439–46.

18. Hando J, Hall W. HIV risk-taking behaviour among amphetamine users in Sydney, Australia. Addiction. 1994;89:79–85.

19. Darke S, Hall W. Levels and correlates of polydrug use among heroin users and regular amphetamine users. Drug Alcohol Depend. 1995;39:231–5.

20. Baker A, Boggs T, Lewin T. Characteristics of regular amphetamine users and implications for treatment. Drug Alcohol Rev. 2001;20:49–56.

21. Darke S, Ross J, J C. The use of benzodiazepines among regular amphetamine users. Addiction 1994;89:1683–90.

22. Williamson S, Gossop M, Powis B, Griffiths P, Fountain J, Strang J. Adverse effects of stimulant drugs in a community sample of drug users. Drug Alcohol Depend. 1997;44:87–94.

23. Wilkins C, Casswell C, Bhatta K, Pledger M. Drug Use in New Zealand : National Surveys Comparison 1998 & 2001. Auckland: Alcohol and Public Health Research Unit, University of Auckland; 2002.

 © NZMA

24. Griffiths P, Vingoe L, Hunt N, Mountenay J, Hartnoll R. Drug information systems, early warning, and new drug trends: can drug monitoring systems become more sensitive to emerging trends in drug consumption? Substance Use & Misuse 2000;35:811–44.

25. MacCoun R, Reuter P. Drug War Heresies: Learning from Other Vices, Times, and Places. New York: Cambridge University Press; 2001.

26. McAuliffe W, Geller S, La Brie R, et al. Are telephone surveys suitable for studying substance abuse? Cost, administration, coverage and response rate issues. Journal of Drug Issues 1998;28:455–82.

27. Kuhn C, Swartzwelder S, Wilson W. Buzzed: the straight facts about the most used and abused drugs from alcohol to ecstacy. New York: WW Norton & Co; 1998.

28. Topp L, Degenhardt L, Kaye S, Darke S. The emergence of potent forms of methamphetamine in Sydney, Australia: a case study of the IDRS as a strategic early warning system. Drug Alcohol Rev 2002;21:341–8.

29. New Zealand Customs Service. Review of Customs Drug Enforcement Strategies 2002. Project Horizon Outcome Report. Wellington: New Zealand Customs Service; 2002.

30. Weatherburn D, Jones C, Freeman K, Makkai T. Supply control and harm reduction: lessons from the Australian heroin 'drought'. Addiction. 2003,98:83–91.

31. Australian Bureau of Criminal Intelligence. Illicit drugs in New Zealand: an overview. In: Australian Illicit Drug Report 1999-2000. Canberra: Australian Bureau of Criminal Intelligence; 2001.

32. Zinberg N. The use and misuse of intoxicants: factors in the development of controlled use. In: Hamowy R, editor. Dealing with Drugs: Consequences of Government Control. Lexington Books: Lexington, Mass; 1987.

33. Sato M, Chen C, Akiyama K, Otsuki S. Acute exacerbation of paranoid psychotic state after long-term abstinence in patients with previous methamphetamine psychosis. Biol Psychiatry. 1983;18:429–40.

34. Yui K, Goto K, Ikemoto S, et al. Susceptibility to subsequent episodes of spontaneous recurrence of methamphetamine psychosis. Drug Alcohol Depend. 2001;64:133–42.

35. Chesher G. Hazardous amphetamine use: the need to consider dosage [Editorial]. Drug Alcohol Rev. 1991;10:299–303.

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Changes in stroke care at Auckland Hospital between 1996 and 2001

Alan Barber, Alison Charleston, Neil Anderson, David Spriggs, Derek Bennett, Patricia Bennett, Kirsty Thomas, and Yvette Baker

Abstract

Aims In 1996, we performed a descriptive audit of stroke care in Auckland Hospital. Since then, a mobile stroke team has been established. We have repeated the 1996 audit to assess changes in stroke management.

Methods From 1 June to 30 September 2001, information was prospectively recorded for all patients with stroke.

Results There were 177 strokes in 175 patients (92 men, mean age 70.9, standard deviation [SD] 14.9 years). Ninety-seven percent of patients had cerebral imaging (median 4.5 hours; interquartile range [IQR] 2.7–11.6). Acute aspirin was given to 78% of patients in 2001 and 40% in 1996 ($p < 0.001$). Twenty-four percent of patients were kept 'nil by mouth' for at least 24 hours (46% in 1996, $p < 0.001$). At discharge, 73% of patients were taking antiplatelet or anticoagulant therapy (61% in 1996, $p < 0.001$). Only 50% of the patients with elevated discharge blood pressures were taking antihypertensives. There had been a reduction in the mean length of hospital stay to 16 days (21 days in 1996) but no significant change in mortality (14% compared with 17% in 1996).

Conclusion A stroke service may increase the attention to the 'processes' of stroke care and use of therapies, which are shown to be of benefit in randomised controlled trials.

In 1996, we published an audit of stroke care in Auckland Hospital.[1] At that time there was no organised inpatient stroke care in our hospital. However, there has since been increasing evidence that organised stroke care results in improved outcome compared with conventional care.[2] In 2000, a mobile stroke team was established at Auckland Hospital. The stroke team includes neurologists, geriatricians, and general physicians; a stroke nurse coordinator; and members from each of the allied health disciplines.

The majority of stroke patients are admitted to general medical wards where they are managed by a general physician. A stroke team physician reviews the stroke patients and makes recommendations about acute management and investigation, and secondary stroke prevention. The specialist stroke nurse and allied health staff have regular rounds and coordinate the multidisciplinary care of patients. Early assessment and referral for inpatient rehabilitation (as well as early discharge planning) are encouraged. There is a weekly multidisciplinary meeting where all aspects of patient care (and the service as a whole) are reviewed. The stroke nurse provides patient/family and staff education. Some of the more complex patients are referred to the Stroke Clinic for follow-up.

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There have been a number of other developments in stroke management since 1996. Recombinant tissue plasminogen activator (rt-PA) within 3 hours,[3,4] and aspirin 150–300 mg within 48 hours, have been shown to reduce death and dependency following ischaemic stroke.[5] Furthermore, combined angiotensin converting enzyme (ACE) inhibitor and diuretic therapy,[6] and the 3-hydroxy-3 methylgluturyl coenzyme A reductase inhibitors, or 'statins',[7,8] have been shown to be beneficial in secondary stroke prevention.

We have repeated the 1996 stroke audit to assess the changes in stroke care at Auckland Hospital over a 5-year period. The aim was to determine the uptake of recent developments in stroke care and the impact of the stroke service on patient management.

Methods

Auckland Hospital has the Auckland isthmus as its catchment area. It also provides the neurology service for Auckland and Northland and, as such, accepts complex and often younger patients from these regions. From 1 June to 30 September 2001, all patients over the age of 15 years presenting to Auckland Hospital with symptoms and signs consistent with an acute stroke were identified by daily checks of emergency department (ED), acute admitting ward, intensive care unit (ICU), geriatric, and vascular surgery databases, as well as attendance at the medical handover meetings. A specialist stroke nurse with one of the stroke team physicians prospectively recorded information from patients, their family, or hospital notes.

The timing of the audit (June to September) and the method of identification of patients were the same as those used in 1996. The form used to record data was the same as that used in 1996 with the addition of a more comprehensive checklist of admission medications, location of inpatient rehabilitation, and differentiation between current and past cigarette smoking.

We used the World Health Organisation definition of stroke,[9] but did not include patients with subarachnoid haemorrhage, subdural or extradural haematoma, haemorrhage into a cerebral tumour, or post-traumatic intracerebral haemorrhage (ICH). We also excluded patients with a transient ischaemic attack (TIA), or patients transferred from other hospitals for tertiary neurological or neurosurgical care. Strokes were classified into three subtypes; ischaemic stroke, ICH, and unspecified stroke type in those patients who did not have cerebral imaging or an autopsy.

All results are presented as the medians and interquartile range (IQR) unless stated. Comparisons of categorical variables between the two audits were made using the chi-square ($?^2$) test. Other comparisons were made using the Wilcoxon rank-sum test. Results were considered significant at the 5% level.

Results

There were 177 stroke events in 175 patients (92 men, mean age 70.9 ± standard deviation 14.9 years) during the 122 days of the audit. Two patients had two admissions for stroke during the period of the study. Four patients had strokes while in hospital. Patients were admitted under the general medicine (83%), neurology (6%), geriatric (3%), neurosurgical (2%) or vascular surgical (1%) service. The stroke or neurology services reviewed 155 of 177 (88%) patients.

There has been an increase in the proportion of patients taking aspirin or warfarin at the time of admission with stroke—from 34% in 1996 to 61% in 2001 (Table 1). There has also been a increase in the proportion of patients with atrial fibrillation taking warfarin, from 12% in 1996 to 32% in 2001 ($?^2 = 4.203$, $p = 0.040$).

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Table 1. Comparison of stroke patient characteristics and management: 2001 and 1996

	2001 n/total (%)	1996 n/total (%)	c^2 / p =
Stroke type			
Ischaemic	146/177 (82)	135/184 (73)	0.006
Intracerebral haemorrhage	25/177 (14)	26/184 (14)	
Unspecified	6/177 (3)	23/184 (13)	
Admission medications			
Aspirin	98/177 (55)	49/167 (29)	<0.001
Warfarin	11/177 (6)	8/167 (5)	
Neither	68/177 (38)	110/167 (66)	
Onset to arrival			
Within 3 hours	35/177 (20)	57/184 (31)	<0.001
Within 6 hours	50/177 (28)	70/184 (38)	
Neither	92/177 (52)	57/184 (31)	
CT scan			
	167/177 (94)	161/184 (88)	0.024
Nutrition			
Patients 'nil by mouth'	42/177 (24)	85/184 (46)	<0.001
NG feeding	21/177 (12)	23/184 (13)	
PEG insertion	5/177 (3)	6/184 (3)	
Neither	109/177 (61)	70/177 (38)	
Acute medications			
Aspirin	107/138 (78)	54/135 (40)	<0.001
Discharge medications			
Aspirin	44/177 (25)	81/184 (44)	<0.001
Warfarin	27/177 (15)	29/184 (16)	
Aspirin & Warfarin	12/177 (7)	0/184 (0)	
Aspirin & Dipyridamole	46/177 (26)	2/184 (1)	
Neither	48/177 (27)	72/184 (39)	
Mortality			
Ischaemic stroke	14/177 (8)	13/184 (7)	0.9488
Intracerebral haemorrhage	10/177 (6)	10/184 (5)	
Neither	153/177 (86)	161/184 (88)	

NG = nasogastric; PEG = percuataneous endoscopic gastrostomy; CT = computerised tomography

Time to arrival and medical assessment Compared with 1996, there has been a reduction in the number of patients arriving in hospital within the first 3 to 6 hours of symptom onset (Table 1). The time of symptom onset was available for 93 of 177 (53%) stroke events. In the remaining 84 events, a patient had woken with symptoms or was unable to give a time of symptom onset and there was no reliable witness. Of all 177 patients, only 20% had a known time of onset and had arrived at hospital within 3 hours.

General practitioners (GPs) assessed 63 (36%) patients prior to hospital arrival. Patients assessed by a GP arrived at hospital later (median 18.5 hours from symptom onset, IQR 6.2–24) than those not assessed by a GP (3.9 hours, IQR 1.1–11.1; Wilcoxon rank-sum test, p <0.0001).

Emergency department (ED) physicians assessed 79 (45%) patients (median time from arrival to assessment 50 minutes, IQR 20–75). The remaining patients were first assessed by the general medicine, neurology, neurosurgical or geriatric services (2.8

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hours, IQR 1.4–4.3). The median time from arrival in hospital to first medical assessment was 68 minutes (IQR 35– 115).

Investigation Almost all patients (97%) had some form of cerebral imaging (Table 1). Computerised tomography (CT) scans were performed in 167 (94%) patients compared with 88% in 1996 ($?^2 = 5.097$, $p = 0.024$). Magnetic resonance imaging (MRI) was obtained in 25 (14%) patients; of whom 21 had also had CT scans. The median time from arrival of the patient in hospital to cerebral imaging was 4.5 hours (IQR 2.7–11.6); down from a median of 10 hours in 1996.

Cerebral angiography was performed in three patients. Thirty-two of 146 (22%) ischaemic stroke patients had a carotid duplex ultrasound (US) study (median time to US study 31 days, IQR 6–42). One patient had a carotid duplex US study more than 6 months after their stroke. A further six patients were still awaiting carotid studies more than 6 months after their stroke. Transthoracic echocardiography (TTE) was performed in 56 (34%) patients, transoesophageal echocardiography (TOE) in nine (5%) patients, and Holter monitoring in seven (4%) patients.

Acute management Aspirin was given (within 48 hours) to 107 of 138 (78%) ischaemic stroke patients (where this information was available), compared to 40% of patients in 1996 ($?^2 = 39.741$, $p < 0.001$). Two patients were treated with rt-PA. Only 5 of 146 (3%) ischaemic stroke patients had a known time of symptom onset and cerebral imaging within 3 hours, and could therefore be considered for rt-PA.

There has been a reduction in the number of patients kept ‘nil by mouth’ for more than 24 hours—from 46% in 1996 to 24% in 2001. Of the 31 ischaemic stroke patients who did not receive early aspirin, 15 had been kept ‘nil by mouth’ for at least 24 hours.

Twenty of 146 (14%) ischaemic stroke patients were treated with heparin, either intravenously (12) or subcutaneously (8). The indication for heparin (where it could be determined) was arterial dissection in four patients, posterior circulation stroke in two patients, and myocardial infarction in one patient. Heparin is not routinely used for deep vein thrombosis (DVT) prophylaxis at our institution—where aspirin (ischaemic stroke patients only), full-length compression stockings, and early mobilisation are recommended.

Complications Thirteen of 177 (7%) patients developed pneumonia during their admission (13% in 1996). Seven of 177 (4%) patients had neurological deterioration (4% in 1996), one patient (0.6%) had a gastrointestinal bleed (5% in 1996), myocardial infarction (2% in 1996), or DVT.

Inpatient rehabilitation Inpatient rehabilitation was carried out in 66 of 177 (37%) patients compared with 54 of 184 patients (29%) in 1996 ($?^2 = 2.56$, $p = 0.109$). Thirty-six of 115 (31%) patients aged 65 years or older were transferred for inpatient rehabilitation at a median of 9 days (IQR 5–13) from admission. Rehabilitation for patients less than 65 years is carried out at a separate inpatient facility. Fourteen of 62 (23%) patients less than 65 years-of-age were transferred for ‘younger-patient’ rehabilitation at a median of 20 days (IQR 13–28) from admission. Sixteen of 177 (9%) patients received rehabilitation in other hospitals.

Secondary stroke prevention Compared with 1996, there has been an increase in the number of patients discharged on either anti-platelet (aspirin with or without

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dipyridamole) or anti-coagulant (warfarin with or without aspirin) therapy (Table 1). Almost all surviving ischaemic stroke patients were discharged on antiplatelet or anticoagulant therapy. Only six of 30 (20%) ischaemic stroke patients with atrial fibrillation on admission ECG were prescribed warfarin. The reasons for the low use of warfarin as secondary stroke prevention in patients with atrial fibrillation were not specifically identified.

The median admission systolic blood pressure (BP) was 160 mmHg (IQR 145–188) and the diastolic BP was 90 mmHg (IQR 80–100). At discharge, the systolic BP was 138 mmHg (IQR 122–150) and the diastolic BP was 76 mmHg (IQR 70–85). Sixty-six of 144 (46%) surviving stroke patients were taking antihypertensive therapy at discharge. Eleven of 20 patients with discharge systolic BPs equal to or greater than 165 mmHg, and five of 10 patients with discharge diastolic BPs equal or greater than 95 mmHg had not been prescribed antihypertensive therapy.

At admission, 46 of 177 patients (26%) gave a history of hyperlipidaemia, and 23 of 177 (13%) patients were using lipid-lowering medication. At discharge, lipid-lowering medication had been prescribed to 30 of 152 (20%) surviving patients.

Stroke outcome and length of stay There has been a 5-day reduction in the mean length of stay at Auckland Hospital to 16.3 days (SD 17.9, median 9 days; IQR 4–22) since 1996. There has been no significant change in hospital mortality between 1996 (17% in hospital mortality) and 2001 (14%).

Discussion

This study provides a description of stroke care in a New Zealand hospital. Previous studies of stroke in New Zealand hospitals have been reported.[10–14] However, this audit has enabled an examination of changes in stroke management at the same institution over a 5-year period.

Almost all stroke patients now have cerebral imaging. In 1991/1992, 40% of stroke patients had cerebral imaging,[15,16] increasing to 88% in 1996 and 97% in 2001. This is the likely reason for the reduction in the proportion of strokes classified as being of 'unspecified' type, and reflects recognition of the need for imaging to exclude stroke mimics and to differentiate between cerebral infarction and ICH. Three quarters of stroke patients now have brain imaging within 12 hours.

It is concerning that there has been a reduction (from 31% in 1996 to 20% in 2001) in the number of patients reaching hospital within three hours of stroke onset. The cause of this increased delay to hospital arrival (between 1996 and 2001 audits) has not been assessed, and any discussion as to the reason is speculative. Specifically, general practitioners and the community may perceive greater pressures on hospital beds—leading to a delay in seeking admission, greater stresses on the Ambulance Service, and worsening Auckland traffic. There continues to be a delay between hospital arrival, medical assessment, and brain imaging. These findings may account for the low use of rt-PA. It is not clear why three of five potentially eligible patients were not treated.

Compared with 1996, almost twice as many patients are now treated with aspirin within 48 hours of symptom onset. This probably reflects greater awareness of the benefits of early aspirin therapy and earlier imaging to exclude cerebral haemorrhage. Aspirin 160–300 mg daily (started within 48 hours) reduces the risk of death or

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dependency from 47.0% to 45.8%.[5] Therefore, assuming 550 stroke patients are admitted to Auckland Hospital per year, the increased use of aspirin acutely has resulted in two to three fewer dead or disabled patients in 2001 compared with 1996.

Fewer patients were kept 'nil by mouth' compared to 1996. This is due to the encouragement of the use of dysphagia assessments and simple bedside swallow tests by the Stroke Service. The reduction of patients kept nil by mouth may partly explain the increased use of aspirin acutely. The number of patients managed with enteral feeding has remained constant since 1996.

There has been an increase in the use of antiplatelet and anticoagulant therapy for secondary stroke prevention. There has also been a dramatic increase in the use of dipyridamole in conjunction with aspirin. These changes reflect greater awareness of the benefits of antiplatelet and anticoagulant therapy in secondary stroke prevention. Other antiplatelet medications (such as Clopidogrel) are not subsidised by the New Zealand government so no patients were prescribed these medications during the time of the audit.

Just over half of the patients in the present study were discharged on no antihypertensive therapy. The Perindopril Protection Against Recurrent Stroke Study (PROGRESS) trial found that (in patients with stroke) combined angiotensin converting enzyme inhibitor and diuretic therapy reduced the risk of recurrent stroke or major vascular event.[6] The benefits of combined therapy were even seen in patients who were 'normotensive' at baseline. The PROGRESS results were published towards the end of recruitment. It is of concern that many patients are discharged with blood pressure measurements still in the hypertensive range.

One in five patients were taking lipid-lowering therapy at discharge. In contrast, almost no patients were taking lipid-lowering therapy in 1996. This increase reflects the dissemination of the results of studies demonstrating the benefit of statins in patients with ischaemic heart disease (IHD).[7] Since this audit was completed, the MRC/BHF heart protection study has shown that simvastatin given to patients with cerebrovascular disease, with or without a history of IHD, significantly reduces the risk of a major vascular event.[8] Furthermore, this reduction in risk did not appear to be influenced by pre-treatment cholesterol or triglyceride concentrations. It is likely that the use of such therapy will increase dramatically in response to this trial and the recent relaxation of the PHARMAC restrictions on the use of statins following stroke.

There has been a 5-day reduction in the length of stay at Auckland Hospital between 1996 and 2001. It is tempting to suggest that the reduced length of stay is due to improved stroke outcome. However, there is greater pressure on hospital beds in general, and there has been no reduction in mortality. There may also have been an apparent reduction in stay as a result of the more accurate classification of patients with mild symptoms as stroke and not TIA.

There has been an improvement in stroke care since 1996. We believe this is probably due (in part) to the direction provided by the Stroke Service, but we acknowledge that this study does not specifically examine this question. In 1996, a neurologist saw only 13% of patients. In 2001, a Stroke Service physician or neurologist had seen 88% of patients. The Stroke Service has formulated guidelines for the investigation and management of stroke patients and has ensured the more rapid and even implementation of stroke therapies across the hospital. Coordinated multidisciplinary

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care and the encouragement of early discharge planning and referral for inpatient rehabilitation may have led to shorter lengths of stay. The Stroke Service, in conjunction with the Radiology Department, has encouraged the more appropriate use of magnetic resonance imaging and carotid ultrasound scanning. Furthermore, there is now formal staff and patient/family stroke education.

However, in response to recent studies, many of the changes seen may have occurred anyway. We did not audit changes in allied health practices. We did not specifically determine what proportion of subjects with stroke were not identified by the Stroke Service. We accept some patients may not have been identified and included in the audit, but believe these numbers were small and of a similar proportion to those in 1996. While the Stroke Service provides support to the general medical teams, most patients are seen by a Stroke Service physician only once. This may explain why some patients with blood pressure measurements in the hypertensive range are discharged on no antihypertensive medication and why some ischaemic stroke patients in atrial fibrillation are not prescribed warfarin.

Patients in geographical stroke units have substantial improvements in management processes, fewer complications, and improved outcome (compared to patients managed on general wards with stroke team support).[17,18] In the latter half of 2002, a small acute stroke unit (with shared care between the general medical and neurology services) was opened at Auckland Hospital. This unit has close links with the geriatric and rehabilitation services, and received 32% of all stroke admissions in the first half of 2003. It is hoped that this is the forerunner of a larger, comprehensive stroke unit.

Author information: P Alan Barber, Neurologist and Director of Auckland City Hospital Stroke Service and University of Auckland; Alison Charleston, Geriatrician, Auckland City Hospital; Neil Anderson, Neurologist, Auckland City Hospital; David Spriggs, General Physician and Geriatrician, Director of General Medicine, Auckland City Hospital; Derek Bennett, Statistician, Clinical Trials Research Unit, University of Auckland; Patricia Bennett, Stroke Research Nurse, Auckland City Hosptial; Kirsty Thomas, Stroke Nurse Specialist, Auckland City Hospital; Yvette Baker, Stroke Nurse Specialist; Auckland City Hospital, Auckland

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Correspondence: Dr P Alan Barber, Director of Auckland Hospital Stroke Service, Park Rd, Grafton, Auckland 1001. Fax: (09) 375 4309; email: abarber@adhb.govt.nz

References:

1. Charleston AJ, Barber PA, Bennett P, et al. Management of acute stroke in Auckland Hospital in 1996. NZ Med J. 1999;112:71–4.
2. Stroke Unit Trialists Collaboration. Organised inpatient (stroke unit) care for stroke (Cochrane Review). Oxford: The Cochrane Library; 2001.
3. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med. 1995;333:1581–7.
4. Kwiatkowski TG, Libman RB, Frankel M, et al. Effects of tissue plasminogen activator for acute is chemic stroke at one year. National Institute of Neurological Disorders and Stroke Recombinant Tissue Plasminogen Activator Stroke Study Group. N Engl J Med. 1999;340:1781–7.
5. Pathansali R, Bath PM. IST and CAST: some answers but more questions. J Hum Hypertens. 1998;12:73-4.

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6. PROGRESS Collaborative Group. Randomised trial of a perindopril-based blood-pressure-lowering regimen among 6,105 individuals with previous stroke or transient ischaemic attack. Lancet. 2001;358:1033–41.

7. White HD, Simes RJ, Anderson NE, et al. Pravastatin therapy and the risk of stroke. N Engl J Med. 2000;343:317–26.

8. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet. 2002;360:7–22.

9. Hatano S. Experience from a multicentre stroke register: a preliminary report. Bulletin of the World Health Organisation. 1976;54:451–553.

10. McNaughton H. Stroke audit in a New Zealand hospital. NZ Med J. 1996;109:257–60.

11. Friedman PJ. Ischaemic stroke and intracerebral haematoma in the Waikato Stroke Registry. NZ Med J. 1992;105:171–3.

12. Hanger HC. Implementing a stroke rehabilitation area: the first six months. NZ Med J. 2002;115(1158). URL; http://www.nzma.org.nz/journal/115-1158/110/

13. Barber PA, Anderson NE, Bennett P, Gommans J. Acute stroke services in New Zealand. NZ Med J. 2002;115:3–6.

14. Gommans J, Barber PA, McNaughton H, et al. Stroke rehabilitation services in New Zealand. NZ Med J. 2003;116(1174). URL: http://www.nzma.org.nz/journal/116-1174/435/

15. Anderson NE, Broad JB, Bonita R. Delays in hospital admission and investigation in acute stroke. BMJ. 1995;311:162.

16. Anderson NE, Bonita R, Broad JB. Early management and outcome of acute stroke in Auckland. Aust N Z J Med. 1997;27:561–7.

17. Kalra L, Evans A, Perez I, et al. Alternative strategies for stroke care: a prospective randomised controlled trial. Lancet. 2000;356:894–9.

18. Evans A, Perez I, Harraf F, et al. Can differences in management processes explain different outcomes between stroke unit and stroke-team care? Lancet. 2001;358:1586–92.

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The Acute Stroke Unit at Middlemore Hospital: an evaluation in its first year of operation

Maria Di Matteo, Craig Anderson, Yogini Ratnasabapathy, Geoff Green, and Kath Tryon

Abstract

Aim Despite strong evidence of benefit, few stroke units exist in New Zealand. In this paper, we describe the process and outcome for the country's first, comprehensive Acute Stroke Unit (ASU), established at Middlemore Hospital in 2001.

Methods The evaluation comprised: (a) two independent 'before and after' audits of medical records of a random selection of patients (2 x n=100) identified from Diagnostic Related Group (DRG) discharge codes for stroke in 1999 (12 months) and 2001–02 (9 months); (b) a review of all DRG stroke outcome data and internal cost analyses for the study periods; and (c) a 'time-in-motion' study of nursing care requirements.

Results The DRG data showed an increase in separations (538 vs 613); stable re-admissions (8% vs 7%); and declines in average length of stay (6.1 vs 5.4 days), deaths (14.0% vs 8.8%), and referrals for rehabilitation (127 vs 67); while the audit indicated shorter times from admission to brain imaging, and swallow and allied health assessments, for stroke from 1999 to 2001–02. A 1:4 nurse:patient ratio seems to provide an optimum level of care for costs.

Conclusions The introduction of the ASU has been associated with improvements in several key indicators of quality of care for patients with stroke.

Specialised Acute Stroke Units (ASUs) have been shown to improve the use of health care resources and the chances of patients surviving free of dependency and/or institutional care after stroke.[1] Moreover, the benefits appear consistent across major patient subgroups: such as the young and old; those with mild, moderate, or severe grades of disability; and in those admitted either early or late after onset. Several distinct features of ASUs that appear important are better coordination of multidisciplinary team (MDT) rehabilitation, integration of nurses and carers in the rehabilitation process, staff with an interest and special skills in stroke care, and the education and training of staff, patients, and carers.[2] However, despite the strong evidence base, and the recommendations of local experts and organisations,[3,4] most New Zealanders do not have access to well organised stroke care,[5,6] due (in part) to the reluctance of leaders to re-configure services, and a lack of clinical expertise and interest in stroke.

As part of the re-organisation of medical services and the opening of a new building, a 12-bed ASU was established within the Division of Medicine at Middlemore Hospital, Counties Manukau District Health Board (CMDHB), in September 2001. This geographically defined unit, located within a 23-bed general medical ward, includes a dedicated MDT of medical, nursing, and allied health staff, and has a policy of accepting direct admissions (or referrals) from the Emergency Department

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and medical or surgical teams during standard working hours. Given its potential to benefit a wide range of patients with stroke, entry into the ASU is based on the availability of beds rather than particular stroke syndromes or patient characteristics. Its aims are to provide both specialist coordinated acute care and early rehabilitation for patients with acute stroke, to implement a structured plan of care within 48 hours, and for discharge or transfer for further inpatient rehabilitation to occur within 1 week of admission.

At the request of the CMDHB, and as a final stage to implementation, we undertook an assessment of the impact of the ASU in its first year of operation.

Methods

The assessment involved: (a) a 'before and after' comparison study of process and outcome data obtained from two independent audits of medical records for a random selection of patients with stroke; (b) analyses of outcomes and costs for stroke using the hospital separations database and other information from systems in the organisation; and (c) a 'time-in-motion' evaluation of nursing care requirements. A medical registrar (MDM), who was independent of the ASU during the study period, extracted data from the case notes of 100 patients who had died or were discharged with a diagnosis of stroke during the experimental 'after' period (9 months; September 2001 to May 2002). These patients had been randomly selected by a clerk in the Medical Records Office who had been provided with a list from the computerised separations database of DRG codes 37 and 38 for stroke and transient ischaemic attack (TIA), only as a primary diagnosis. For an assessment of variation in care, the clerk was also instructed to select approximately equal proportions of patients who had been managed within and outside the ASU. Pre-coded data extraction forms were used to obtain information on several key items of case mix, process of care, and outcome, including the time-dependent variables (admission to assessment, days) of brain imaging, swallow assessment, and allied health assessment. These data were compared with a similar audit of 100 randomly selected case notes pertaining to stroke-related DRGs during a 'before' period (12 months; January to December, 1999), undertaken by another independent medical practitioner, and used in a report recommending establishment of the ASU to CMDHB in December, 2000.

In addition, the outcomes of total number of admissions, total average length of stay (ALOS), re-admissions (within one month), and deaths for all cases of stroke/TIA (only as a primary diagnosis) were obtained from the hospital DRG database for the defined 'before' and 'after' study periods. The average unit cost related to an ALOS for stroke/TIA were then calculated by dividing the total costs by the number of cases in each of the study periods. Direct and indirect costs for stroke/TIA, included both fixed and unfixed costs, and covered expenses such as investigations, therapists, nutritional services, medical services, pharmacy, and bed costs, as well as overhead charges for depreciation, administration, and support services. These data were analysed using the software and systems of the Decision Support Unit of Middlemore Hospital.[7] Finally, the optimal nurse:patient ratio was tested in a 'time and motion' study. This involved an independent registered nurse being assigned to the ASU to directly assess the type and amount of nursing care undertaken by five nurses, at varying levels in their clinical career pathway, over four morning and one afternoon duty rosters during a 'typical' week in June 2002. The costs for nursing were then identified by the Decision Support Unit.

Statistical evaluations were conducted with 'Software Package for the Social Services' (SPSS) for Windows software.[8] Descriptive statistics including mean, median, minimum, and maximum values are presented in tables, with the Mann-Whitney U test used to compare groups with continuous variables and skewed distribution, and the chi-square ($?^2$) test statistic used to compare groups with categorical variables. All tests were two-tailed and the level of significance was $p < 0.05$.

Results

Process and outcome audit Table 1 presents the characteristics of the 100 patients in the control 'before' group and 100 patients in the intervention 'after' group, with the latter group further subdivided into 51 patients admitted to the ASU and 49 patients who received all their care in other medical wards.

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Table 1. Demographic and clinical characteristics, by group

Characteristic	1999 group	2001–02 group, overall		2001–2002, by unit		
	(n = 100)	(n = 100)	p value	Other medical unit (n = 49)	Acute Stroke Unit (n = 51)	p value
Demography						
Mean age, yrs (range)	70 (31–89)	70 (33–96)		71 (41–96) 24	(33–89) 29	
Female	51	53	n/s	35	49	0.001
Definite stroke*	90	84	n/s	27	25	
Married	64	52	n/s			
Ethnicity						
Caucasian	62	49	n/s	22	27	n/s
Maori/Pacific Islander	22	27		16	11	
Other	16	24		11	13	
Pre-morbid dependency	21	11	n/s	7	4	n/s
Co-morbidity (≥ 2 risk factors)†	37	54	n/s	27	27	n/s
Mode of referral						
GP	47	41	0.003	19	22	n/s
Self	39	56		29	27	
Other	13	1		-	1	
Unknown	1	2		1	1	

*Excludes transient ischaemic attack (TIA) and other cerebral pathologies such as subdural haematoma

†Includes two or more of diabetes, cardiac disease, renal failure, hypertension, cigarette smoking

The profile of patients was broadly similar in the 'before' and 'after' groups, although there were slightly fewer patients with a diagnosis of stroke in the 'after' group due to the inclusion of several patients with TIA and subdural haematoma who had been retrieved by the DRG codes. This was pertinent to the 'medical ward' subgroup, as patients with TIA were not routinely admitted to the ASU. Furthermore, patients in the 'after' group had less pre-morbid dependency but more co-morbidity (eg, hypertension, diabetes) than the 'before' group.

Table 2 shows data on the process of care. The proportion of patients who underwent brain imaging was high (approximately 95%) in both the 'before' and 'after' groups, and there was a trend for a greater proportion of patients being imaged on the ASU compared to other medical units. Compared to the 'before' group, swallow assessments were undertaken in a higher proportion of patients closer to the time of admission in the 'after' group, and were performed more often in patients on the ASU than in other medical units. Although the proportion of patients who received allied health assessments was high in both groups, overall, the trends were for reductions in the time from admission to this assessment, and for more patients in the ASU to receive the assessment compared to those in other medical units. Although the proportion of patients who had a formal functional assessment performed on them was low (about one fifth in the 'before' and 'after' groups), there was an increase in the proportion of patients with completed discharge summaries.

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Table 2. Process of care, by group

Variable	1999 group	2001–02 group, overall		2001–02 group, by unit		
	(n = 100)	(n = 100)	p value	Other medical unit (n = 49)	Acute Stroke Unit (n = 51)	p value
CT use (includes MRI)	95	93		43	50	0.02
Admission to test (days)						
- Mean	1.6	0.9	n/s			
- Median (range)	1.0 (0–23)	1.0 (0–4)	n/s			
Swallow assessment	35	41	0.001	10	31	<0.001
Admission to assessment (days)						
- Mean	4.7	3.1	n/s			
- Median (range)	3.0 (0–19)	3.0 (1–11)	n/s			
Allied health assessment	86	84	n/s	36	48	0.01
Admission to assessment (days)						
- Mean	2.0	1.7				
- Median (range)	1.0 (1–17)	1.0 (0–9)	n/s			
Functional assessment recorded	26	14	n/s	2	12	0.01
Discharge letter completed*	68	98	<0.001	49	49	

*Includes transfer summary

Table 3 shows measures of patient outcomes. Overall, there was a decline in the number of admissions associated with complications in the ‘after’ study period, with the ASU having more patients with complicated hospital stays and higher mortality than other medical units, yet a greater proportion of patients discharged home. In addition, from ‘before’ to ‘after’, there were declines in the proportion of patients referred to the Assessment, Treatment and Rehabilitation (AT&R) Unit (17% vs 35%) and to Home Health Care Services (13% vs 49%), although the number of referrals to outpatient medical clinics for follow-up increased.

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Table 3. Outcome measures

Variable	1999 group	2001–02 group		2001–02 group, by unit		
	(n = 100)	(n = 100)	p value	Other medical unit (n = 49)	Acute Stroke Unit (n = 51)	p value
Hospital stay with complications*	43	32	n/s	11	21	
Discharge disposition from Medicine						
Death	5	9	0.02	3	6	0.01
Home	44	58		38	20	
AT&R†	35	16		2	14	
Private hospital or other	16	17		6	11	
Follow-up						
Follow-up plan identified	35	41	n/s			
Medical outpatient clinic	31	52	<0.001	19	33	0.01
Home health care referral	49	13	<0.001	6	7	
NASC referral‡	N/A	14	-	5	9	
Stroke Foundation referral†	N/A	11	-	2	9	0.03
Evidence of delay in discharge§	28	9	<0.001	1	8	0.06
Mean (range) time waiting		6.4 (2–12)	-			

*Includes any of the following: falls, pressure sores, infections, confusion, venous thrombosis, embolism, myocardial infarction, and seizure

†AT&R = Assessment, Treatment and Rehabilitation Unit for older people

‡NASC = Needs Assessment and Service Coordination service for older people; data only included in the 2001–02 audit

§In most cases, delay in discharge was related to an unexpected illness (e.g. pneumonia) in the patient or the wait of an available bed in a private hospital, but it also included investigations such as gastrostomy and videofluroscopy procedures.

Review of DRGs for stroke/TIA The DRG data showed that the number of separations for stroke/TIA had increased from 538 in 1999 to 613 in 2001–02, with 33% of all cases in the later period being managed within the ASU. The ALOS for stroke had decreased from 6.1 days to 5.4 days, and the ALOS in ASU ranged from 5.8 to 9.1 days (mean 7.1 days) compared to 3.0 to 6.1 days (mean 4.2 days) in the rest of general medicine. For the two time periods, transfers to AT&R had decreased (127 to 67) and re-admissions to hospital had declined (8% to 7%), with the ASU cases having a lower re-admission (4.2%) compared to cases from the rest of the hospital (5.5%). Overall, deaths had decreased between the two study periods, but were higher (11.4%) for the ASU compared to other units (9%) during 2001–02.

Cost analyses The average cost per stroke/TIA (relative to ALOS) had increased from $3560 in 1999 to $4464 in 2001–02. The total cost of care in the ASU was $759 473 during the ‘after’ period, which was approximately $70 000 higher than that for

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other medical wards—the result of an actual 1:3 nurse-to-patient ratio of care together with the use of special, watch and bureau nurses. An annualised figure of $84 000 for the ASU was derived by deducting the average cost of the 12 beds from the total cost for all beds on the ward. Based on the 'time and motion' study, which showed that a 1:4 nurse-to-patient ratio was adequate for patient care and the safety of staff, the cost of the ASU was estimated at $718 179 (without special and watch nurses)—a difference of $41 000 above that of other medical wards.

Discussion

We undertook an evaluation of the effectiveness of the ASU, by comparing various measures of the new model of care against an historical standard. In general, the data show that the introduction of the ASU has been associated with improvement in the quality of care outcomes for patients with acute stroke admitted to Middlemore Hospital. These effects occurred against a background of rising patient numbers, as well as higher costs of care for patients with stroke.

Increasing demand and economic pressures on the health care system have intensified the need for evidence-based approaches, cost-effective analyses, and better strategies to improve patient and consumer outcomes. Quality of care is increasingly being judged on process and outcome data, although this is a complex task, with the deficiencies in valid and reliable measures being widely acknowledged.[9] In our audit of medical records, we chose several measures that captured aspects of the chain of care for patients with stroke; that of diagnosis, functional (disability) assessment, acute and rehabilitation management, discharge, and follow-up. The data show a consistently high level of brain imaging being undertaken to confirm the diagnosis of stroke in both study periods, but the trend was for earlier imaging to be undertaken in 2001–02 compared to 1999. Arguably more important, though, was the finding of an increase in swallow assessments, and a trend for them to be done closer to admission following introduction of the ASU.

Moreover, compared to other medical wards, this assessment was more likely to be done on patients admitted to the ASU, possibly due to the training of Dysphagia Link Nurses to improve clinical management and reduce the time that patients are kept 'nil by mouth'. Allied health assessments, including those by occupational health, physiotherapy, speech-language therapy, and social workers, was high in both study groups, but the trend was for earlier assessments to be undertaken in 2001–02.

Overall, the number of hospital admissions complicated by medical illness, such as falls and infections, was lower in 2001–02. However, the greater proportion of complicated cases on the ASU, as compared to other medical wards, can largely be attributed to case selection—whereby patients with TIA were excluded (and the more severe or complicated cases included) in the ASU. Use of functional assessment measures, such as the Barthel Index,[10] were low in both study periods, in part because none of the staff on the ASU were instructed in their use; a situation that has now been corrected. There was a large increase in the proportion of completed patient discharge letters, but this probably reflects the introduction of electronic discharge summaries after 1999.

While the audit of medical records showed a trend towards an increase in the number of deaths after stroke, this was not supported by the DRG data for all patients with

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stroke/TIA during the two study periods. Deaths from stroke had declined by over one-third since 1999, but the higher number of deaths on the ASU compared to other medical units during 2001–02 may again be attributable to the admission of more severe cases on that ward. This explanation is further supported by the higher number of patients referred to AT&R and private hospitals from the ASU—as compared to other medical units, where a greater proportion were discharged home. Overall, the number of patients discharged home after stroke had increased since 1999.

The overall decline in referrals to AT&R in 2001–02 is striking. One explanation is that the ASU is providing early rehabilitation, including effective discharge planning, that was not possible on other medical wards, resulting in an earlier and direct discharge to home for some patients who would otherwise have received further inpatient rehabilitation. As the overall ALOS for patients with stroke had declined from 1999 to 2001–02, the ASU does not appear to be retaining patients longer than was necessary, and (in line with other studies) shows that patients were usually discharged within 7 days of admission.[1] A decline in ALOS was also seen after the introduction of a dedicated stroke rehabilitation ward for older people at the Princess Margaret Hospital, Christchurch, in 2001.[11] Moreover, the higher number of patients discharged home, the increased referral for follow-up in a medical outpatient clinic, and the low re-admission, provide further support that the ASU offers an improved and effective discharge process. While the low referral to Needs Assessment Service Coordination (NASC) and to Home Health Care services may be due to patients being more independent and having less need of home help, it could also represent changes in documentation of referrals, and so requires further investigation.

The relative and absolute costs of stroke are high, and increasing, as evident by the DRG data. However, this trend should be viewed within the context of an overall increase in the costs of medical care as well as inflation, rather than being necessarily due to the use of MDTs on the ASU and other medical units. The ‘time in motion’ study showed that a 1:4 (as opposed to the 1:3) nurse:patient ratio, initially employed on the ASU (with a health care assistant employed for tasks such as tidying rooms, making beds and emptying indwelling catheters), is adequate for care and safety, and provides an appropriate budget for nursing care.

There are several limitations to this study that must be mentioned. Firstly, the quality of the data is highly dependent on the accuracy and completeness of information included in the DRG codes and medical records, cross-sectionally and over time. However, we used only primary stroke/TIA DRG codes that have been shown to be valid,[12] and any bias is likely to have been balanced across the two study periods. Secondly, the small number of charts included in the audits raises the possibility of error due to chance or random variation, particularly due to differences in periods of observation (12 months in 1999 versus 9 months in 2001–02). Finally, it is not only difficult to draw conclusions regarding ‘cause-and-effect’ relationships (that is, effects of the ASU), but it is also important to consider the data in the context of ongoing changes in the health care environment, both within the hospital and the community as a whole. Public health strategies and new community initiatives, especially chronic disease management projects, could have had an impact on stroke outcomes independent of the ASU.

Despite its limitations, though, we believe our study has shown that the introduction of the ASU has been associated with a better process of care, as evidenced by an

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increase in swallow assessments and speed of input from allied health, as well as better discharge planning and medical follow-up. As with any new service, there were set-up problems, some of which could have been avoided with better planning. However, the findings from the audits complement the DRG data in indicating favourable trends in stroke outcome, possibly due to the benefits of the MDT model of care and rehabilitation, as indicated by the randomised evidence.[2] However, the declines in referrals for inpatient rehabilitation and to community services, requires further investigation to ensure that patients' re-settlement back home after stroke is effective.

Author information: Maria Di Matteo, Medical Registrar, Middlemore Hospital; Craig Anderson, Clinical Director, Acute Stroke Unit, Middlemore Hospital, and Professor of Geriatric Medicine and Co-Director, Clinical Trials Research Unit of the University of Auckland; Yogini Ratnasabapathy, Physician and Senior Lecturer in Geriatrics, Middlemore Hospital; Geoff Green, Geriatrician, AT&R Unit, Middlemore Hospital; Kath Tryon, Service Coordinator, Medicine, Counties Manukau District Health Board, Auckland

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Correspondence: Professor Craig Anderson, Co-Director, Clinical Trials Research Unit of the University of Auckland, Private Bag 93311, Otahuhu, Auckland. Fax: (09) 373 1710; email: c.anderson@ctru.auckland.ac.nz

References:

1. Collaborative systematic review of the randomised trials of organised inpatient (stroke unit) care with stroke. Stroke Unit Trialists' Collaboration. BMJ. 1997;314:1151–9.
2. How do stroke units improve patient outcomes? A collaborative systematic review randomized trials. Stroke Unit Trialists' Collaboration. Stroke. 1997;28:2139–44.
3. Wilkinson T, Sainsbury R. Stroke services high priority, but neither coordinated nor timely (editorial). NZ Med J 2003;116(1174). URL: http://www.nzma.org.nz/journal/116-1174/433/

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4. Life after stroke: guidelines for best practice in rehabilitation after stroke. Auckland: Adis International, 1996.

5. Barber A, Anderson NE, Bennett P, Gommans J. Acute stroke services in New Zealand. NZ Med J 2002;115:3–6.

6. Gommans J, Barber A, McNaughton H, et al. Stroke rehabilitation services in New Zealand. NZ Med J 2003;116(1174). URL: http://www.nzma.org.nz/journal/116-1174/435/

7. Sunrise Decision Support Manager. Eclipsys Technologies Corporation, FL, USA, 2002. Available online. URL: http://www.eclipsys.com/News/PressReleases/04-01-13_KLAS_top_20_03.asp Accessed March 2004.

8. SPSS for Windows, V10.1 Chicago, Ill: SPSS Inc; 2000. Available online. URL: http://www.spss.com/ Accessed March 2004.

9. McNaughton H, McPherson K, Taylor W, Weatherall M. Relationship between process and outcome in stroke care. Stroke 2003;34:713–7.

10. Wade DT, Collin C. The Barthel ADL Index: a standard measure of physical disability? Int Disabil Stud 1988; 10: 64–7.

11. Hanger HC. Implementing a Stroke Rehabilitation Area: the first six months. NZ Med J 2002; 115(1158). URL: http://www.nzma.org.nz/journal/115-1158/110/

12. Mayo NE, Neville D, Kirkland S, et al. Hospitalization and case-fatality rates for stroke in Canada from 1982 through 1991. The Canadian Collaborative Study Group of Stroke Hospitalizations. Stroke 1996;27:1215–20.

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A case of human poisoning by salinomycin, an agricultural antibiotic

Phillipa Story and Alan Doube

Ionophore antibiotics are used in farming for the prevention of coccidiodomycosis in poultry and to alter gut flora in order to improve nutrient absorption in ruminants. However, this class of antibiotics affects both animal and bacterial cell physiology. Their mechanism of action at the cellular level is to selectively bind certain ions creating intra- and extracellular biochemical disturbances. The ions bound vary with different members of this class of drug, with salinomycin preferentially binding potassium. This interferes with potassium transport across mitchondrial membranes, resulting in low intracellular energy production. The Na^{+}/Ca^{2+} exchange mechanism may also be disrupted allowing a fatal accumulation of intracellular calcium.[1] This mechanism particularly affects skeletal muscle in all animals, and cardiac muscle in a few (eg, cattle).

In porcine studies, salinomycin has been demonstrated shortly after ingestion to have a sympathomimetic effect in animals up to three times more powerful in dose-response testing than adrenaline, with positive chronotropic, inotropic, and pressor effects.[2]

Animal poisonings with ionophore antibiotics are widely described in veterinary and related journals. There is considerable interspecies variation in morbidity and mortality that cannot simply be attributed to body mass. Horses seem to be particularly susceptible to skeletal rhabdomyolysis and early death.[1,3,6] The clinical features that are common to most species are an early period of weakness and ataxia followed by a progressive muscle weakness with hind-limb predominance. There have also been cases of cardiac muscle involvement in cattle during chronic poisoning resulting in cardiomyopathy and death from congestive cardiac failure.[4] The typical onset of rhabdomyolysis in dogs was the fifth day post-ingestion, with death, when it occurred, delayed as long as 14 days,[1] and survivors affected for up to 50 days.[5]

The clinical course of the human patient in this case fairly closely resembles that described for dogs with a progressive, bilateral, symmetrical, leg weakness ascending to the forearms and chest with absent reflexes, preserved sensation, smooth-muscle activity, and mental function. Other causes of rhabdomyolysis including trauma and infection were considered but were not present in this case.

Case report

A previously healthy, 35-year-old male was working in a factory making animal feed mixes. One of his tasks was to add salinomycin granules into a ‘worm’ screw as chicken grain feed flowed past. An accidental blowback of the salinomycin granules occurred resulting in inhalation and swallowing of a small amount despite washing out of the mouth. A few minutes later he became acutely unwell with nausea, shortness of breath, and dizziness. He arrived in the emergency department 30 minutes after exposure, where he was found to be agitated and complaining of leg

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weakness, nausea, and photophobia. The patient was alert and orientated despite his emotional distress and appeared markedly pale and diaphoretic, with a bounding pulse of 110/min, blood pressure 156/76, oxygen saturation 100%, and temperature 36.2°C. Examination of the chest, abdomen and skin was unremarkable, and neurological examination revealed bilateral leg weakness and moderate photophobia. It was difficult to make a detailed neurological assessment because of the patient's agitation, but he was orientated and there were no obvious localising lesions of either the cranial nerves or limbs. On contacting the National Poisons Centre (University of Otago Medical School, P O Box 913, Dunedin, tel 0800 POISON, www.toxinz.com) for advice, we discovered that there were no human data available for poisoning with this substance. Animal data suggested that the patient might have a transient hypernatraemia and weakness. No specific antidotes were known to exist and the patient was treated supportively with oral charcoal 50 g, oxygen 10 L/min by mask, and intravenous fluids (1 L normal saline); and admitted for observation.

Over the next 48 hours, the patient complained of limb weakness. He was found to be areflexic with 2/5 power in his legs and 4/5 in his arms. Initial investigations showed normal electrolytes and urea, a transient hyperglycaemia of 16.6 mmol/L, and a transient leucocytosis of 16.4 x 10^9. Over the next two days, the patient made a significant recovery of muscle power, with only mild weakness detectable in the knee extensors, and all further blood tests (CBC, electrolytes, glucose) were normal. The tachycardia of 100–110/min settled over the first day with no pharmacological intervention, and he remained haemodynamically stable at all times. He received a further 2 L of intravenous fluid over the 24 hours as well as oral intake. There is no record of urine volume, but the patient was passing urine, and ward urinalysis revealed ketones only. On Day 6 post-exposure, he complained of pain in his left calf and a small, below-knee deep venous thrombosis was diagnosed on ultrasound and D-dimer assay. He was commenced on enoxaparin and warfarin and continued to show good return of muscle power with normal observations. On Day 9, the patient was discharged to his family with clinic follow up arranged. His medication on discharge consisted of warfarin and diclofenac.

On Day 14, the patient re-presented to the emergency department complaining of increasingly severe pain in both legs. Examination revealed normal tone in his legs with decreased reflexes and marked tenderness of the calf muscles. Biochemistry revealed a raised creatine kinase (CK) of 25 000 U/L with mild elevations of alanine aminotransferase 294 U/L (normal 0–45U/L) and gamma-glutamyl transpeptidase 75 U/L (normal 0–60 U/L). Cardiac isomers of CK were normal, as was the echocardiogram (ECG). Haematology showed Hb144 g/L, WCC 12.6, platelets 323, and INR of 2.2, consistent with warfarin treatment.The urine was positive for myoglobin, but renal function and serum calcium remained normal. It was not clear at this stage whether the rhabdomyolysis was related to the patient's recent exposure or some other pathology.

The patient experienced ongoing muscle pain and weakness that progressed from his legs to his arm and chest over a 4-day period. He remained alert and had no cranial nerve lesions or respiratory difficulty. After initial treatment with IV normal saline and urinary alkalisation (8.4% $NaHCO_3$), the patient was managed with oral fluids and bed rest while evidence of ongoing muscle injury remained. The CK remained high (peak 38 000 U/L) for 7 days before beginning to decline, and reached almost

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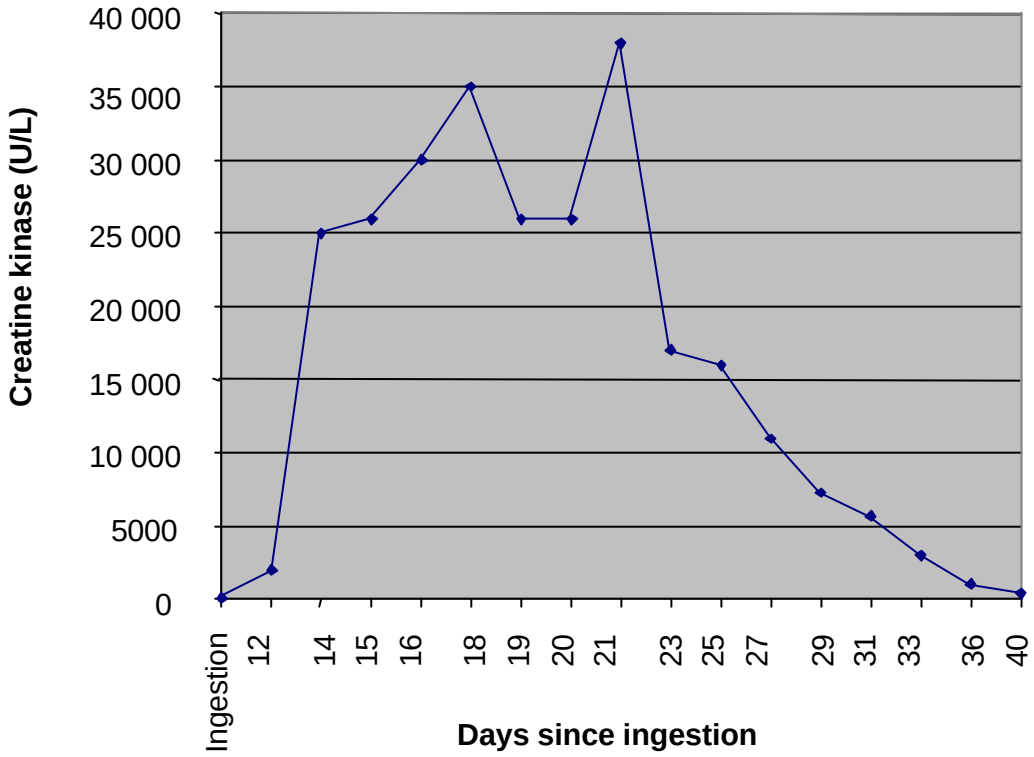
normal levels on Day 40 post-ingestion (435 U/L). Urine myoglobin became undetectable by Day 35 with normal renal function throughout. A muscle biopsy was not performed. Due to the description of cardiomyopathy in cattle,[4] a baseline ECG was performed which was normal. The patient suffered from several episodes of chest pain, but repeated ECGs and troponin measurements were normal. The patient made a slow and uneventful recovery and was finally discharged 40 days after the original exposure. At that time, he still had very limited exercise tolerance—eg, climbing a single flight of stairs was difficult and painful.

Figure 1. Progress of serum creatine kinase (U/L) measurements in patient over recovery period



Discussion

It is clear from this case that humans may be vulnerable to the toxic effects of ionophore antibiotics. This patient ingested an estimated 1 mg/kg of salinomycin resulting in a 6-week hospital admission with prolonged rhabdomyolysis, pain, and disability. The patient had no other discernable causes for rhabdomyolysis. It is likely that his initial presentation with diaphoresis and tachycardia is attributable to the sympathomimetic properties of salinomycin.[2] The subsequent clinical course most closely resembled that of the dog in both symptom progression and duration.[3,5] Animal research into potential antidote treatments, including administration of selenium and vitamin E have not shown any impact on toxicity,[7] and supportive measures (charcoal, oxygen, IV fluids) remain the only current treatment method.[8] It

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would appear from this, and animal cases, that any patient making an initial full recovery will need to be closely followed for the advent of rhabdomyolysis, which may be delayed. From our experience, we recommend aggressive management of myoglobinuria (urinary alkalisation) and prolonged bed rest to minimise metabolic demand on ATP-depleted muscle. Although this patient did not suffer any detectable myocardial damage, it is not possible to rule this out in a future human case and an early baseline cardiac ECG is suggested.

This is the first published description of salinomycin poisoning in humans. It was clear from the patient and from product literature that the agricultural community does not widely appreciate the serious consequences that may arise from exposure to ionophore antibiotics. The authors hope that this paper will provide an important safety warning in the agricultural industry, and provide a guide for treatment and monitoring for physicians.

Author information: Phillipa A Story, Emergency Physician; Alan Doube, Consultant in General Medicine and Rheumatology, Waikato Hospital, Hamilton

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Correspondence: Dr Phillipa Story, Emergency Department, Waikato Hospital, Private Bag 3200, Hamilton. Fax: (07) 839 8772; email: kiwidoc02@hotmail.com

References:

1. Parton K, Bruere AN, Chambers JP. Veterinary clinical toxicology. 2nd edition. Palmerston North: Veterinary Continuing Education, Massey University; 2001.
2. Fahim M, del Valle G, Pressman BC. Comparison of the effects of the ionophore salinomycin and adrenaline on the haemodynamics and work efficiency of the dog heart. Cardiovasc Res 1986;20:145–52.
3. McKellar Q, Lawrence K. Ionophores. In Practice, British Veterinary Association. 1996;(Sept): 385–6.
4. Bastianello SS, McGregor HL, Penrith ML, Fourie N. A chronic cardiomyopathy in feedlot cattle attributed to toxic levels of salinomycin in the feed. J S Afr Vet Assoc. 1996;67:38–41.
5. Safran N, Aizenberg I, Bark H. Paralytic syndrome attributed to lasalocid residues in a commercial ration fed to dogs. J Am Vet Med Assoc. 1993;202:1273–5.
6. Nicpon J, Czerw P, Harps O, Deegen E. Salinomycin poisoning in a Polish stud horse. Tierarztl Prax Ausg G Grosstiere Nutztiere. 1997;25:438–41.
7. Wendt M, Busing S, Bollwahn W. Toxicity of the combination of salinomycin and tiamulin in swine. Dtsch Tzt Wscht. 1997;104:405–10.
8. Novilla MN. The veterinary importance of the toxic syndrome induced by ionophores. Vet Hum Toxicol. 1992;34:66–70.

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Accidental subcutaneous copper salt injection: toxic effects and management

David Atkinson, Michael Beasley, and Peter Dryburgh

This report advises on findings from a case of accidental copper glycinate injection into the leg of a healthy, 27-year-old, male agricultural worker who has hepatitis B carrier status.

Case report

The patient presented to his general practitioner on a Friday evening, 2 hours after accidental self-injection of approximately 120 mg (2 ml) of copper glycinate solution into the left lower leg. The injury had been sustained in the hazardous process of administering this supplement to cattle. The injection site was close to the path of the common peroneal nerve as it curves around the proximal fibula. The patient was suffering considerable pain. He experienced mild tingling in the distribution of the nerve; however, he had no motor deficits.

The needle-entry site was evident; and approximately 2.5 cm away, a 2 cm (in-diameter) circle of green-blue subcutaneous discolouration was seen.

The general practitioner called the National Poisons Centre and was advised of the potential risk for systemic toxicity based on cattle studies, as well as local reaction. Discussion with a local veterinarian further reinforced these dangers. Acute referral was made to the surgical service of Kaitaia Hospital and the patient presented next morning for surgical debridement.

The injection site was obvious with an area of 3 x 3 cm of central dark necrosis surrounded by an area of erythema and oedema approximately 10 cm in diameter.

Debridement of the injection site on the left leg overlying the head of the fibula and lateral aspect of the upper calf was performed. This revealed extensive, blackish-grey, chemical discolouration of skin, subcutaneous fat and the deep fascia. All the discoloured tissues, which were clearly necrotic and did not exhibit any arterial bleeding, were debrided. The common peroneal nerve was not encountered. The wound was packed open.

On discussion with the National Poisons Centre, intravenous post-operative treatment with the (most readily available) chelating agent, calcium disodium edetate, was recommended, as an additional precaution. Several doses were given, until further information became available suggesting that it was unlikely to be therapeutic and was potentially toxic.

A serum copper level taken the day after injury was reported as 14 umol/L (normal range 13–23 umol/L). During this time, renal and hepatic function remained normal.

A further minor debridement was performed 3 days later, followed by split skin grafting of an apparently healthy granulating wound 5 days later. During the hospital stay, pain was a prominent feature, radiating up the anterior and lateral aspects of the

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thigh and requiring analgesia with Tramadol and Paracetamol. The patient was discharged 2 days after the skin graft.

The recipient graft site became infected with *Staphylococcus aureus*, with only a 50% take. After a further period of dressings another split skin graft of the remaining granulating areas was performed. This grafting procedure was covered with antibiotic prophylaxis for 1 week, but despite no detectable infection, these grafts also sloughed peripherally. It was felt that residual copper in the tissues, on the periphery of the granulating area, was responsible for the difficulty in obtaining successful skin grafting. The portion of the graft that had taken, slowly expanded outwards to cover the area. Healing was nearly complete about 11 weeks from the time of injury. Troublesome neurogenic pain, radiating up the thigh and in the region of the injection site remained a problem and was eased with a small dose of Amitriptyline at night.

Prominent features of this case were the difficulty in obtaining successful grafting of the granulating areas, especially at their peripheries, and the persistence of significant neurogenic pain.

Discussion

It is well established that inorganic copper salts are significantly toxic to humans via ingestion. As well as significant gastrointestinal symptoms, including haematemesis and melaena, effects can include intravascular haemolysis, acute hepatic necrosis, hypotension and renal failure. Methaemoglobinaemia, myoglobinuria and acute pancreatitis have also been observed in some patients.[1]

In contrast, there is very little information on the effects of parenteral doses of copper formulations in humans. For example, one manufacturer has received no reports of inadvertent self-injection resulting in toxicity (Parnell Laboratories, Sydney; Australia. Personal communication, 2002) and the New Zealand Poisons Centre can recall just one anecdotal report of apparently deliberate self-injection resulting in major toxicity; however, details of this case are sparse.

In animal experiments it is shown that copper injections have relatively low safety margins,[2] thus veterinarians often warn against imprudent double dosing of animals, particularly young calves and sheep, because this is potentially fatal.

Copper edetate has more rapid absorption and can be more toxic whereas copper glycinate causes more reactions at the injection site.[3] The depot copper is rapidly transported to the liver, where it is stored and then continuously released, maintaining serum copper levels.[3]

Regardless of the form of these preparations, plasma copper levels are maximal 2 to 4 hours post-injection,[3] although peak plasma concentration following copper glycinate administration is significantly lower than that of an equivalent copper edetate dose.[4] This is an important consideration, since haemolysis occurs as a direct effect of free copper ions on erythrocytes. This has been seen in calves, weighing approximately 55 kg, receiving 240 mg of copper edetate (four times the recommended dose).[3] However, it is stated that at no stage following injection of the copper glycinate formulation is there circulation of free ionised copper, and that there is minimal potential for copper toxicity.[4]

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The liver is also vulnerable. A study using 480 mg (four times the recommended dose) of injectable copper edetate in mature cows, weighing approximately 380 kg, demonstrated that in approximately 24 hours three quarters of the available copper had made its way to the liver. This caused rapid, severe hepatic necrosis, leading to death in several animals.[5] Those animals receiving a similar dose of copper glycinate survived.[5]

One computer model for cattle indicates that liver copper following copper glycinate injection is likely to be maximal roughly 10 days after injection.[6]

Those receiving copper glycinate are shown to develop large subcutaneous lumps, swellings and sterile abscesses in about 25% to 50% of cases, lasting up to 3 months. Copper edetate, in contrast, has a local reaction rate of approximately 5%.[7]

The management of systemic copper poisoning can be problematic. Chelating agents have been recommended in severe poisoning, although there are few pharmacokinetic data by which to evaluate their effectiveness.[8] While some blood level data hint at a role (such as for a combination of dimercaptopropanol and $CaNa_2$ EDTA),[9] this is not entirely convincing, and other cases do not. In occupationally exposed workers treated with $CaNa_2$EDTA (20 mg/kg IV), the 24-hour urinary copper excretion was increased only 1.3 times that of background, compared with lead (13-fold increase), zinc (11-fold), manganese (3.8 fold), and cadmium (3.4 fold).[10] Thus, at least with mildly elevated plasma and/or erythrocyte concentrations, $CaNa_2$EDTA did not significantly increase the urinary excretion of copper compared with some other metals. In lead-poisoned children undergoing 5-day courses of $CaNa_2$EDTA (1000 mg/m^2/day), urinary excretion and, more importantly, blood-copper concentration were unaffected even though significant reductions in plasma zinc (and lead) were noted.[11]

Normally, however, urinary excretion is a very minor pathway of copper elimination, so that even a moderate percentage increase may not have much impact. The great majority of copper is eliminated via the bile, or passes more directly through the bowel wall.[12] A very significant fraction of the chelators—dimercaptopropanol (BAL), d-penicillamine, and their metal chelates—are excreted in the bile, and one source suggests that these agents can enhance copper elimination.[13] They are arguably the current agents of choice. However, in one combined copper and zinc ingestion, BAL (4 mg/kg; 6-hourly; IM) and d-penicillamine (250 mg; 6-hourly; via nasogastric tube), when commenced within 4 hours, had little apparent effect on plasma copper levels.[14] On the other hand, DMPS (2,3-dimercapto-1-propane sulphonate), an analogue of BAL, has shown some promise as a copper chelator.[15]In one case of acute poisoning, arising from excess dermal absorption, benefit was claimed for penicillamine.[16] It may also be beneficial in chronic copper over-exposures,[17] and it has been associated with improvements in chronic copper toxicity.[18] A therapeutic benefit has also been claimed in Wilson's disease,[19] attributed to effects on removing copper but also possibly detoxifying hepatic stores.[20]

Overall, however, the available data do not suggest a reliable, marked benefit with chelating agents for large acute doses of copper. Furthermore their usage can be complicated by the development of renal failure (to which they could potentially contribute), and BAL is not recommended in the presence of hepatotoxicity.[21]

Copper is a dialysable substance, and haemodialysis may be indicated in severe cases. It is likely to be most effective in the early stages of poisoning, when the metal (in

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some formulations) is still present in significant quantities as free copper.[1] However, with non-ingestion routes, apart from local injury, the patient may be initially asymptomatic, and copper analyses may take days to obtain, delaying indicators for the use of this modality. Certainly, with copper ingestions, haemodialysis has not prevented some fatalities when commenced 6 to 7 days after the event.[22]

Urgent surgical exploration of the wound site, with removal of visible injection products and debridement of adversely affected tissues is recommended for all poisoning by injectable copper salts, except tiny amounts. Injections of copper glycinate must be explored due to the risks of rapid, severe, soft-tissue necrosis and local reaction. Copper EDTA, which is more rapidly absorbed, poses additional systemic risks including hepatotoxicity and haemolysis. This is particularly the case with injections estimated over 0.25 mg/kg. Intervention should be prompt, ideally within 4 hours of injury and certainly 8 hours, especially with large doses, as significant absorption of most forms of copper can occur within this time.

Author information: David Atkinson, General Practitioner, Te Aroha Noa Medical Centre, Whangarei; Michael Beasley, Medical Toxicologist, National Poisons Centre, Dunedin; Peter Dryburgh, Senior Surgeon, Clinical Advisor, Far North Area Health Services, Kaitaia

Correspondence: Dr David Atkinson, Te Aroha Noa Medical Centre, 73A Bank St, Whangarei. Fax: (09) 438 3297; email: dratkinson@clear.net.nz

References:

1. Chugh KS, Sharma BK, Singhal PC, et al. Acute renal failure following copper sulphate intoxication, Postgrad Med J. 1977;53:18–23.
2. Copper Chloride, Copper Gluconate, Copper Heptanoate, Copper Oxide, Copper Methionate, Copper Sulphate and Dicopper Oxide Summary Report. Committee for Veterinary Medicinal Products, London: European Agency for the Evaluation of Medicinal Products (EMEA); May 1998.
3. Bohman VR, Drake EL, Behrens WC. Injectable copper and tissue composition of cattle, J Dairy Sci 1984;67:1468–73.
4. Parnell Laboratories NZ Ltd. Copperex Injection. Index of Veterinary Specialties. 2001 Annual, Auckland.
5. Bohman VR, Poole SC, Kvasnicka WG, et al. The toxicology and composition of bovine tissues after parenteral administration of high levels of copper salts. Vet Hum Toxicol. 1987;29:307–12.
6. Blincoe C. Computer simulation of copper pharmaceuticals in cattle, Journal of Controlled Release 1993;26:31–8.
7. Boila RJ, Devlin TJ, Drysdale RA, Lillie LE. Injectable Cu complexes as supplementary Cu for grazing cattle, Canadian Journal of Animal Science. 1984;64:365–78.
8. Barceloux DG. Copper. J Clin Toxicol. 1999;37:217–30.
9. Walsh FM, Crosson FJ, Bayley M, McReynolds J. Acute copper intoxication. Pathophysiology and therapy with a case report. Am J Dis Child. 1977;131:149–51.
10. Araki S, Aono H, Murata K. Mobilisation of heavy metals into urine by CaEDTA: relation to erythrocyte and plasma concentrations and exposure indicators. Br J Ind Med. 1986;43:636–41.

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11. Thomas DJ, Chisolm JJ. Lead, zinc and copper decorporation during calcium disodium ethylenediamine tetraacetate treatment of lead-poisoned children. J Pharmacol Exp Ther 1986;239:829–35.

12. Baselt RC. Disposition of toxic drugs and chemicals in man. 5th ed. Foster City, CA: Chemical Toxicology Institute; 1995.

13. Haddad LM, Shannon MW, Winchester JF, editors. Clinical management of poisoning and drug overdose. 3rd ed. Philadelphia: WB Saunders Company; 1998.

14. Hantson P, Lievens M, Mahieu P. Accidental ingestion of a zinc and copper sulfate preparation. J Clin Toxicol 1996;34:725–30.

15. Hruby K, Donner A. 2,3-dimercapto-1-propane sulphonate in heavy metal poisoning. Med Toxicol Adverse Drug Exp. 1987;2:317–23.

16. Holtzman NA, Elliott DA, Heller RH. Copper intoxication. Report of a case with observations on ceruloplasmin. N Engl J Med. 1966;275:347–52.

17. Salmon MA, Wright T. Chronic copper poisoning presenting as pink disease. Arch Dis Child. 1974;46:108–10.

18. Bhusnurmath SR, Walia BN, Singh S, et al. Sequential histopathologic alterations in Indian childhood cirrhosis treated with d-penicillamine. Hum Pathol 1991; 22: 653–8.

19. Schilsky ML, Scheinberg IH, Sternlieb I. Prognosis of Wilsonian chronic active hepatitis. Gastroenterology. 1991;100:762–7.

20. Scheinberg IH, Sternlieb I, Schilsky M, Stockert RJ. Penicillamine may detoxify copper in Wilson's disease. Lancet. 1987;2:95.

21. Goldfrank LR, Flomenbaum NE, Lewin NA, et al, editors. Goldfrank's Toxicologic Emergencies. 6th edition. Stamford, Connecticut: Appleton & Lange; 1998.

22. Akintonwa A, Mabadeje AF, Odutola TA, et al. Fatal poisonings by copper sulfate ingested from 'spiritual water'. Vet Hum Toxicol. 1989;31:453–4.

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Time for major roadworks on the tobacco road?

Julian Crane, Tony Blakely, and Sarah Hill

Nicotine replacement

Inhaling tobacco smoke is a remarkable and exquisitely refined mechanism for delivering nicotine to the central nervous system. Remarkable for its acute safety and chronic catastrophe, and unique because it is tobacco (not nicotine) that causes the damage. The failure to make this crucial distinction is a tragedy.

Alternative methods of delivering nicotine via tobacco have been available for centuries in the form of chewing tobacco, snuff, drinks, enemas, and percutaneous administration—all developed by the aboriginal peoples of North and South America.[1,2]

In 1942, Johnston administered nicotine intravenously to himself and 34 volunteers thus altering smoking behaviour in recipients.[3] Further development of alternative nicotine delivery took another 30 years with the development of nicotine chewing gum. This was followed by transdermal, nasal, and oral nicotine vapour inhalers. These Alternative Nicotine Delivery Systems (ANDS) were developed to decrease the craving of tobacco withdrawal and improve abstinence.

Many studies confirm the ability of these systems to improve 'quit rates' but their impact on long-term tobacco abstinence is modest.[4] Generally, their pharmacokinetics are designed to provide low doses of nicotine over a prolonged period rather than the high-dose burst from smoking. Currently, the addicted smoker only has tobacco to provide this nicotine burst, and only gains access to alternative nicotine in the context of quitting smoking. Paradoxically, access to the safer forms of nicotine is often highly restricted, a point that has been previously emphasised.[5,6] Indeed, the relationships between tobacco and nicotine (in the context of the public's health) have only recently been explored.[7]

Tobacco control

In the last 30 years, tobacco control strategies have considerably reduced smoking in some countries. The most successful have employed multiple approaches, including mandatory packet warnings, price increases, a ban on tobacco advertising, smoke-free environment legislation, health education, the provision of quit programmes, and litigation against the tobacco industry.

New Zealand has employed all of these strategies and, between 1970 and 2001, per-capita tobacco consumption has reduced by 60%. Adult smoking prevalence has decreased from 36% in 1976 to 28% in 1990. But since the 1990s, smoking prevalence has remained static. For Maori adults, the reductions have been much less dramatic (from 58% in 1976 to 51% in 2001). Furthermore, for Pacific Island adults, there has been an even smaller decrease (from 35% to 31%).

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Nowadays, socioeconomic disparity is even greater, with smoking prevalence three-fold higher for those from families with annual incomes below $20,000 compared to those with annual incomes above $120,000.[8] In New Zealand, the decline in smoking prevalence has stalled, and for Maori and Pacific people has been negligible. This socioeconomic and ethnic disparity in smoking prevalence clearly illustrates that in New Zealand, at least, tobacco control has largely benefited the more affluent. In fact, smoking prevalence amongst the poorest members of New Zealand's society was higher in 2001 than the overall smoking prevalence in 1976.

Such disparity is evident elsewhere, for example in the UK, where a similar three-fold disparity in smoking prevalence exists between the most and least advantaged groups.[9] In the UK, this disparity is beginning to be recognised for the targeting of smoking cessation services (with some evidence of benefit).[10]

Perversely, the very success of tobacco control has left the remaining smokers and most of the world's developing economies in the unfettered embrace of a demonised tobacco industry. The outrage from public health at the tobacco industry's intransigence and tactics has clouded the entirely separate issues of tobacco and nicotine, rendering the idea of developing recreational or long-term replacement nicotine, a heresy. The introduction of alternative forms of nicotine as abstinence-promoting therapies have been tightly regulated, initially by prescription and latterly by restriction to pharmacies. The fundamental flaw has been the failure to separate nicotine from tobacco, both literally and metaphorically.

New approaches to nicotine replacement are required as Bates has suggested.[11] There are now a large number of Alternative Nicotine Delivery Systems available. A first step would be to make them as widely available as tobacco and significantly cheaper. Specifically, nicotine needs to be taken out of the pharmacy to openly compete with tobacco at every outlet. Moreover, the role of ANDS needs to be redefined—from improving abstinence rates to long-term replacement for tobacco for those smokers unable (or unwilling) to quit.

Studies of ANDS as long-term replacement will be required to define the most useful therapies singly and in combination—particularly among low-income and marginalised groups. However to implement a comprehensive nicotine replacement strategy, an effective inhaled nicotine delivery system (designed to deliver cigarette-like doses safely) will be needed.

Inhaled nicotine

Since the development of the metered dose inhaler in the 1960s, the pharmaceutical industry has gradually refined and improved the pulmonary delivery of drugs, principally for the management of asthma. The recent need to reformulate asthma treatments (such as beclomethasone) as liquids rather than solid particles in CFC-free carriers has led to smaller particle sizes and a doubling of potency (12). The goal of these therapies is to provide high doses locally at the airway mucosa. The aim of pulmonary nicotine delivery will be to deliver nicotine to the brain via the lung. Such inhaled nicotine delivery systems are not without risk.

First, a focus on them may distract policy-makers and the health-promotion workforce from other aspects of ongoing comprehensive tobacco control. Second, nicotine itself is not without adverse health effect, although (without doubt) nicotine is much less

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dangerous to health than tobacco. Third, the availability of high-dose nicotine may dissuade people from quitting, and encourage initiation of a new nicotine habit among youth who would not have commenced smoking tobacco otherwise.

Several key elements would need to accompany any serious programme to introduce inhaled nicotine, however the devices and their effects must be acceptable to smokers. Specifically, they must be able to approximate the nicotine bolus obtained from smoking, and there would be an inevitable trade-off between sufficient appeal to smokers and insufficient appeal to experimental adolescents.

Nicotine at some mucosal surfaces is painful. As the tobacco industry was well aware of this early on, it introduced mentholated cigarettes (menthol being a weak local anaesthetic) to ease neophytes into their addiction. The development and marketing of inhaled nicotine would require close cooperation between state and enterprise to ensure a balance with tobacco abstinence strategies. The financial and legislative barriers to developing, and then marketing, the appropriate technology are considerable. Without support, and a carefully crafted strategic approach from governments, public health, and the anti-tobacco lobbies, the risks for any industry far outweigh the benefits. But in an appropriate regulatory climate, in which a long-term strategy for marketing had been agreed, there could be sufficient incentives for development of inhaled nicotine and extension of nicotine-delivery programmes.

New Zealand has some characteristics that make it an ideal country to pioneer such an approach. New Zealand has a strong long-standing commitment to public health and has pioneered smoke-free legislation, mechanisms to control tobacco, and the provision of alternative forms of nicotine. Despite these efforts, continuing reductions in smoking prevalence have slowed considerably and have largely benefited the more affluent sectors of society. Regarding extended nicotine programmes, New Zealand is a small isolated country separated by thousands of kilometres of ocean in all directions frustrating smuggling and a black market in tobacco. There are also precedents for partnerships between government and the pharmaceutical industry.

For example, the New Zealand government is currently investing $200 million developing a vaccine for hyperendemic meningococcal disease. Meningococcaemia kills approximately 20 people per year in New Zealand, whereas tobacco kills close to 5000 per year. Currently, the New Zealand government collects $880 million of revenue from tobacco annually. A small proportion of this revenue could be used to help develop a comprehensive nicotine-replacement programme.

There is an urgent need for new approaches to tobacco. The failure to separate tobacco from nicotine is a major barrier to further progress in preventing tobacco-related disease. Once separated, there is every reason to expect that, with an appropriate mix of incentive and regulation, a replacement nicotine strategy (including inhaled forms), could be developed and successfully introduced.

Governments need to be reassured that it will be considerably less harmful than tobacco and that recruitment to a new addiction industry is minimised. Regular monitoring will be required. Industry must be satisfied that it is financially viable and that there is an appropriate legislative framework in place to allow effective market entry. Essentially ‘Big Pharma’ needs to compete with ‘Big Tobacco’. Most importantly, it must satisfy the addicted smoker who will need to be encouraged to switch from tobacco to nicotine with a mixture of marketing and financial incentives.

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Furthermore, it must be readily available, and sit in a new niche between recreation and therapy. Once established and acceptable, tobacco as a nicotine delivery system will gradually disappear, and with it the whole issue of environmental tobacco-smoke exposure. None of this is likely to be easy, but neither is it impossible, and the potential gains are enormous.

The use of tobacco is part of almost every culture, and despite the best efforts of health professionals and regulatory authorities over the last 30–40 years, it is still readily available in every country and used by approximately one sixth of the World's population. In New Zealand, tobacco control has taken us a considerable way down the road to smoking abstinence, but the reductions have been inequitable and have lost momentum. While we need to retain many of the current tobacco control strategies, we urgently need new approaches and one of these is alternative nicotine replacement.

Author information: Julian Crane, Professor, Department of Medicine; Tony Blakely, Senior Research Fellow, Department of Public Health; Sarah Hill, Registrar in Public Health, Department of Public Health, Welllington

Correspondence: Professor Julian Crane, Wellington School of Medicine, University of Otago, PO Box 7343, Wellington, New Zealand. Fax: (04) 389 5427; email: crane@wnmeds.ac.nz

References:

1. Robicsek F. The smoking gods: tobacco in Maya art, history and religion: Norman, OK: University of Oklahoma Press; 1978.
2. Wilbert J. Tobacco and shamanism in South America. New Haven, Conn: Yale University Press; 1987.
3. Johnston LM. Tobacco smoking and nicotine. Lancet. 1942;2:742.
4. Blondal T, Gudmundsson LJ, Olafsdottir I, et al. Nicotine nasal spray with nicotine patch for smoking cessation: randomised trial with six year follow up. BMJ. 1999;318:285–8.
5. Kawachi I. Levelling the playing field for regulation of nicotine. BMJ. 2003;326:115–6.
6. Sweanor D. Regulatory imbalance between medicinal and non-medicinal nicotine. Addiction. 2000;95:S25–8.
7. Ferrance R, Slade J, Room R, Pope M, editors. Nicotine and public health: Washington, DC: American Public Health Association; 2000.
8. Ministry of Health. Tobacco Facts. Wellington; 2002. Available online. URL: http://www.moh.govt.nz/moh.nsf/c43c7844c94e08cd4c2566d300838b43/2da018a2eb73c257cc256bc600076cb5/$FILE/TobaccoFacts2002.pdf Accessed March 2004
9. Walker A, Maher J, Coulthard M, et al. Results from the 2000/01 General Household Survey. London: Stationery Office; 2001.
10. Lowey H, Tocque K, Bellis MA, Fullard B. Smoking cessation services are reducing inequalities. J Epidemiol Comm Health. 2003;57:579–80.
11. Bates C. What is the future for the tobacco industry? Tob Control 2000;9:237–8.
12. Fireman P, Prenner BM, Vincken W, et al. Long-term safety and efficacy of a chlorofluorocarbon-free beclomethasone dipropionate extrafine aerosol. Ann Allergy Asthma Immunol 2001;86:557–65.

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The contribution of the wealthy towards hospital services

This extract is taken from an editorial published in the New Zealand Medical Journal 1904, Volume 3 (12), p422–3

Speaking generally, there can be no question that radical alteration in the constitution of all Hospital Boards is a clamant necessity. Hospitals are in this colony on quite a different footing from all such institutions in the Old Country. Here all hospitals are financed by the State, and, with the exception of a few "homes," nothing is left to private charity. At Home all hospitals, with the exception of those in connection with poorhouses, are supported entirely by voluntary contributions. It can easily be seen, therefore, that little or nothing is to be gained by comparing the methods of management. At Home the hospitals were erected and are maintained for the reception and treatment of the indigent sick; here, by reason of the fact that all – rich and poor – are called upon to contribute to the cost of their upkeep, all are entitled to treatment in these institutions. Great abuses have crept in at Home – where there is little excuse – and out here the profession have for many years felt that certain classes of the community have by their entering the public hospitals evaded their just debts. As the law stands, however, no equitable exception can be taken to the entrance of a rich man to the wards of a general hospital. He, in common with all other ratepayers, pays for the support of such institutions, and therefore is just as much entitled as the poor man to treatment therein. We do not see how this can in justice be altered, but surely it should not be difficult to so arrange the tariff that the wealthy man should pay a sum more commensurate with the value he receives than he does at present. To suggest that by the payment of £1 8s. per week, *plus* the ordinary hospital-rate charges, the rich man has resolved his debt to the community is ridiculous, and yet it cannot be denied that many deserving poor have been denied admission because the beds were occupied by those well able to pay the outside charges.

Were the honorary staff paid for their work at the hospitals the problem from a professional point of view would be simplified. It might then be assumed that the medical men who took the positions of surgeons and physicians to hospitals had decided that they were receiving a sufficient reward for their work. But, as a matter of fact, 90 per cent. of the work done is performed by a staff who receive no monetary reward. It is not to be wondered at, therefore, if a surgeon sees a wealthy patient entering a general hospital to have an operation done for which he was well able to pay the ordinary fee, that he should feel aggrieved.

The remedy for this is for Hospital Boards to charge at least the ordinary fees to those able to pay. At attendance on the poor or indigent the profession has never grumbled, and the best services of its members have always been at their command.

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Pseudocyesis

Figure 1 is the MRI of the pelvis of a 28-year-old Afro-Caribbean woman.

Figure 1

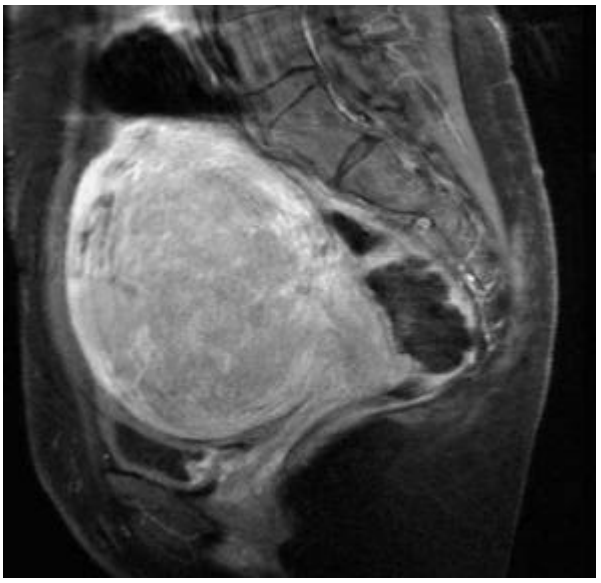

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Figure 1 is a sagittal MRI of the uterus, showing a large, heterogeneous mass consistent with a massive fibroid. The size of the fibroid is equivalent to a 28-week fetus.

Figure 2 demonstrates a selective catheter placed via the right common femoral artery into the left uterine artery. Note the large tortuous nature of this vessel. Similar appearances were seen on selective angiography of the right uterine artery. These arteries were embolised using polyvinyl alcohol (PVA) microspherules.

Figure 3 is an MRI at one month showing liquefied fibroid lying within the uterine canal (yellow arrow) and extending out through the vagina (blue arrow). Note the preservation of the normal myometrium (red arrow).

Figure 2

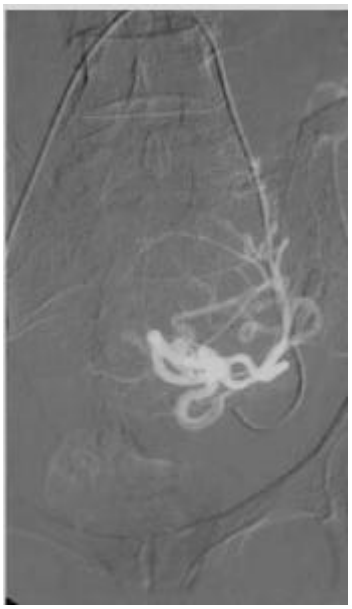
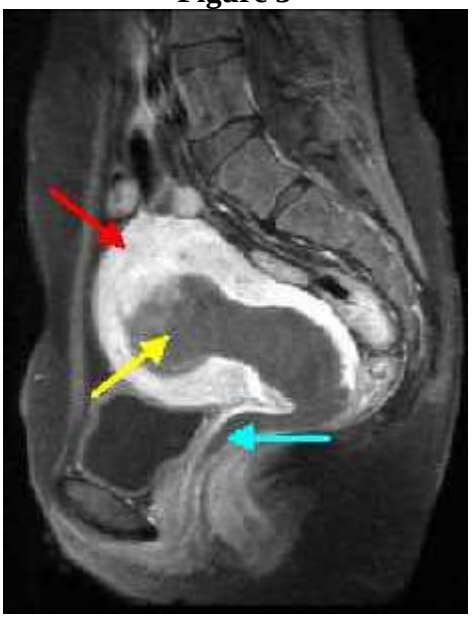


Figure 3



Comment

Percutaneous embolotherapy for symptomatic fibroids is now an accepted treatment. It is uterus preserving and probably fertility sparing. The procedure, however, does have a significant radiation burden and requires the skills and attention of a multidisciplinary team.

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Roll on, July 2005

Recent agreements between Australia and New Zealand will result in big changes to the advertising and sale of therapeutic products. NZ will join Australia in banning direct consumer advertising of prescription drugs. And a joint scheme to regulate all therapeutic products in both countries, including complementary medicines and over-the-counter items, will come into force next year.

NZ is the only developed country, apart from the US, to allow advertising of prescription medicines. Now that will change. But the new trans-Tasman agency to regulate therapeutic products is an even bigger step. It will come into force in July 2005. Announcing the move, NZ health minister Annette King noted that at present food is more strictly regulated than alternative therapies.

New Scientist, 24 January 2004

Business 1, Ethics 0

A leading nephrology journal has rejected a guest editorial questioning the efficacy of epoetin in end stage renal disease, despite favourable peer reviews, apparently because it feared losing advertising. In a letter to the author of the proposed editorial, the executive editor of the California based journal *Transplantation and Dialysis* said he had been 'overruled by our marketing department.'

The editorial was written by Dennis Cotter, president of the non-profit making Medical Technology and Practice Patterns Institute. In it, he argued that the US National Kidney Foundation's existing guidelines on end stage renal disease early on flawed logic in claiming a survival benefit associated with higher packed cell volume (haematocrit) achieved through epoetin treatment.

Joseph Herman, editor of the journal, told Mr Cotter in a letter that: 'I have now heard back from a third reviewer of your EPO editorial, who also recommended that it be published…Unfortunately, I have been overruled by our marketing department with regard to publishing your editorial.'

Arthur Caplan, chair of the Department of Medical Ethics at the University of Pennsylvania, said: 'It is completely unethical for a marketing or business related part of a journal to have any say over the content of a journal.'

BMJ. 2004;328:244

Rehabilitation in the community for stroke patients

Patients returning home after a stroke sometimes lose their independence; however, there is not much consensus about the effects of rehabilitation services for stroke patients once they have left hospital. The Outpatient Service Trialists did a Cochrane systematic review of randomised trials of outpatients' services to determine how rehabilitation services could affect stroke recovery.

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This review was based on a study of 14 trials involving 1617 patients. The authors concluded that therapy-based rehabilitation services for individuals living at home after stroke can reduce risk of deterioration in ability to undertake activities of daily living. The investigators conclude that debate should move from whether services are effective to how to make the most of their benefits.

Lancet. 2004;363:352–6

Breast implants and accuracy of mammography

Breast augmentation is the third most common type of plastic surgery performed for cosmetic reasons in the United States, with 268 888 procedures in 2002. Although breast implants have not been found to be associated with an increased risk of breast cancer, implants may interfere with routine mammography evaluation; therefore, women with breast augmentation may be more likely to be diagnosed with advanced disease.

In an analysis of data from seven US mammography registries, involving almost one million women over seven years, Miglioretti and colleagues found that among asymptomatic women, the sensitivity of screening mammography was lower in women with breast implants than in those without implants. Prognostic characteristics of tumours in women with breast cancer, however, were not significantly different in women with breast augmentation compared with those without augmentation.

JAMA. 2004;291:442–50

PHARMAC, USA?

The need to manage escalating healthcare costs while maintaining reasonable access to care is becoming the salient challenge in US healthcare policy. Insurance coverage is patchy and incomplete, and serious problems with access persist. The healthcare system has not succeeded in controlling expenditures. The cost pressures resulting from technological advances and an ageing population are likely to exacerbate the problem of access.

Nowhere is concern about cost and access more pressing than in the provision of prescription drugs through state Medicaid programmes. Prescription drugs are the fastest-growing component of healthcare spending. Medicaid has been hit hard – its spending on drugs soared from $4.8 billion in 1990 to $21.0 billion in 2000.

A variety of state initiatives have emerged for reducing expenditures on prescription drugs. These initiatives have been informed in their design by experience in other countries and facilitated by the Medicaid programme’s waiver provisions, which afford states latitude to engage in policy experiments. The initiatives have met with vigorous opposition from the drug industry.

N Engl J Med. 2004;350:608–13

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Withholding of annual practising certificates

I am responding to a recent editorial in the NZMJ (http://www.nzma.org.nz/journal/117-1188/741/)[1] and would like to correct the statement that the Medical Council withholds doctors' annual practising certificates (APCs) when they have yet to appear before the Medical Practitioners Disciplinary Tribunal.

I am not aware of any situation where a doctor has had his or her APC withheld because of pending disciplinary proceedings. In these circumstances the legislation requires the Registrar to refer the application to the next full Council meeting for consideration and a decision on whether the APC can be issued.

In my experience the Council has never declined to issue an APC in these circumstances.

The Council Registrar can issue a 'certificate of good standing' (CGS) if a doctor is seeking registration to work in another country. This certificate is intended to show that there is no legal barrier to the doctor being currently registered on disciplinary, competence, criminal or health grounds.

If it is not possible for the Registrar to issue a CGS because there is a current or intended investigation or proceedings before the Council or the Tribunal, a 'letter of standing' is issued instead including a brief statement on the reason for non-issue of a CGS.

I hope this clarifies the issue for your readers.

Sue Ineson
Chief Executive
Medical Council of New Zealand

Reference:

1. Frizelle FA. Going from bad to worse? NZ Med J. 2004;117(1188). URL: http://www.nzma.org.nz/journal/117-1188/741/

Response

My editorial of 30 January did contain an error, as pointed out by Sue Ineson in her letter above. I stated that the New Zealand Medical Council at times withheld doctors' annual practising certificates when they had yet to appear before the Medical Practitioners Disciplinary Tribunal.

What I should have said is that the Medical Council has withheld a doctor's APC after appearing before the MPDT when the findings of this Tribunal did not suggest or recommend that the doctor's APC be withdrawn, or that there be any conditions on his practice of medicine. Indeed, in one case I know of the Tribunal made no restriction on the doctor's ability to practise medicine, even though this was sought by

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the prosecution, and yet still the Medical Council withheld, for a time, the issue of the APC.

Frank A Frizelle
Editor, NZMJ

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Analysis of element content in scalp hair of healthy people and breast-cancer patients with SEM/EDX method

Scanning electron microscopy (SEM) with energy-dispersive X-ray microanalysis (EDX) is frequently used for morphological and qualitative chemical characterisation of different materials.[1]

Hair analysis may be a promising tool for routine clinical screening and diagnosis of patients with breast cancer.[2] We tried to avoid possible variance by choosing hair samples from patients of the same sex and similar age groups (patients 53 ± 9, controls 55 ± 7 years) and by using uncoloured hair. In addition, sampling of hair was performed with maximum care according to the recommendations of the Hair Analysis Standardisation Board.[3] Therefore, all the samples were from people of the same ethnic and geographic origin and thus having similar dietary habits etc.

A Leo 440 Computer-Controlled Digital Scanning Electron Microscope was used for analysis.

The characteristic dispersive X-ray spectra of the elements is indicated in Table 1.

Table 1. Distribution of the element contents in patients with breast cancer (stage III) and healthy people analysed by SEM/EDX method

Element	Patient group	Control group
C	+	+
O	+	+
Al	+	+
Si	+	+
S	+	+
K	+	+
Ca	+	+
Hg	+	+
Re	–	+
Mg	+	+
Na	+	+
Zn	–	+
Sr	–	+
Cl	+	+
Ba	–	+
Pb	+	+
In	+	+
Cu	+	–
Rb	+	–
Fe	+	–
To	+	–

+ = spectra available; – = spectra unavailable

Pb and Fe have been reported to act as mitogen and carcinogen,[4] and tumour cells have greater need for Fe.[5] In accordance with this we observed dispersive X-ray

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spectra of Fe in cancer patients but not controls. However, dispersive X-ray spectra of Pb were seen in both breast-cancer patients and the control group. The X-ray spectra of Cu were also clearly observed in breast-cancer patients but not the control group in accordance with results reported by other investigators.[6]

A clear negative correlation between Re, Zn, Sr, and Ba content and breast cancer was observed; Zn is consistent with reports in the literature.[7] Interestingly, no dispersive X-ray spectra of Re, Sr and Ba were observed in the hair samples of breast-cancer patients. These results show that Re, Sr and Ba have similar anti-carcinogenic properties to Zn.

In conclusion, it should be noted that this is the first report on the analysis of dispersive X-ray spectra of these elements in patients with breast cancer compared with healthy people. The clear absence or overlap of some elements in either group proves that hair analysis could be a useful tool for providing reliable individual diagnostic statements of breast cancer.

Eser Kilic
Department of Biochemistry

Sema Ozturk
Department of Physics
Erciyes University, Kayseri, Turkey

Asuman Demiroglu
Department of Biology
Gebze Institute of Technology, Gebze, Turkey

Zeki Yilmaz
Department of General Surgery

Oguz Yildiz
Department of Radiation Oncology

Mehmet Akkurt
Department of Physics

Recep Saraymen
Department of Biochemistry

Engin Ok
Department of General Surgery
Erciyes University, Kayseri, Turkey

References:

1. Seta S, Sato H, Yoshino M, Miyasaka S. SEM/EDX analysis of inorganic elements in human scalp hairs with special reference to the variation with different locations on the head. Scan Electron Microsc. 1982;1:127–40.
2. Sukumar A. Factors influencing levels of trace elements in human hair. Rev Environ Contam Toxicol. 2002;175:47–78.
3. Passwater RA, Cranton EM. Trace elements, hair analysis and nutrition. New Canaan, CT: Keats Publishing Inc.; 1983.
4. Wingren G, Axelson O. Epidemiologic studies of occupational cancer as related to complex mixtures of trace elements in the art glass industry. Scand J Work Environ Health. 1993;19 Suppl 1:95–100.

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5. Hann HW, Stahlhut MW, Menduke H. Iron enhances tumor growth. Observation on spontaneous mammary tumors in mice. Cancer. 1991;68:2407–10.

6. Coates RJ, Weiss NS, Daling JR, et al. Cancer risk in relation to serum copper levels. Cancer Res. 1989;49:4353–6.

7. Piccinini L, Borella P, Bargellini A, et al. A case-control study on selenium, zinc, and copper in plasma and hair of subjects affected by breast and lung cancer. Biol Trace Elem Res. 1996;51:23–30.

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MPDT Practice Note Number Three

THE TRIBUNAL wishes to GIVE NOTICE to all parties who may appear before it that as at 5 February 2004 it has adopted the High Court Code of Conduct for Expert Witnesses.

SCHEDULE 1

PROCEDURES to be followed when giving expert evidence to the Tribunal:

Duty to the Tribunal

1. An expert witness has an overriding duty to assist the Tribunal impartially on relevant matters within the expert's area of expertise.
2. An expert witness is not an advocate for the party who engages the witness.

Evidence of expert witness

3. In any evidence given by an expert witness, the expert witness must –
   (a) acknowledge that the expert witness has read this Code of Conduct and agrees to comply with it.
   (b) state the expert witness' qualifications as an expert.
   (c) state the issues the evidence of the expert witness addresses and that the evidence is within the expert's area of expertise.
   (d) state the facts and assumptions on which the opinions of the expert witness are based.
   (e) state the reasons for the opinions given by the expert witness.
   (f) specify any literature or other material used or relied on in support of the opinions expressed by the expert witness.
   (g) describe any examinations, tests, or other investigations on which the expert witness has relied and identify, and give details of the qualifications of, any person who carried them out.
4. If an expert witness believes that his or her evidence or any part of it may be incomplete or inaccurate without some qualification, that qualification must be stated in his or her evidence.
5. If an expert witness believes that his or her opinion is not a concluded opinion because of insufficient research or data or for any other reason, this must be stated in his or her evidence.

Duty to confer

6. An expert witness must comply with any direction of the Court to –
   (a) confer with another expert witness.
   (b) try to reach agreement with the other expert witness on matters within the field of expertise of the expert witnesses.

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(c) prepare and sign a joint witness statement stating the matters on which the expert witnesses agree and the matters on which they do not agree, including the reasons for their disagreement.

7. In conferring with another expert witness, the expert witness must exercise independent and professional judgment and must not act on the instructions or directions of any person to withhold or avoid agreement.

DATED at Wellington this 5th day of February 2004

D B Collins, QC
Chair
Medical Practitioners Disciplinary Tribunal

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James Macalister Bremner

Jim Bremner died on 7 December 2003 after 12 months of increasing disability, which he bore with remarkable stoicism.

He was born on 9 April 1930 in London, where his father, the late Walton Bremner, was training in surgery, and arrived in New Zealand at the age of two. He always retained his British passport. He was educated at the Normal School and Elmwood Primary School and entered Christs College in 1943, where he was a Junior Somes Scholar. After a distinguished academic career, which followed an entirely classical pathway, he obtained a Junior University Scholarship. His sporting activities were limited by extreme short-sightedness, but he was a useful, medium-paced bowler and played in the second XI.

When Jim started Medical Intermediate at Canterbury University College he had never studied any science. High marks were required for entry into the second year but he obtained them and in 1949 he took up residence at Knox College, where he stayed for four years and made lifelong friends. He qualified MB ChB (NZ) in 1953.

After one year as a house surgeon at Christchurch Hospital Jim left to undertake surgical training in the UK. He was a houseman in surgery from September 1956 at the Royal Postgraduate Hospital, Hammersmith, for six months followed by a further six months there as Casualty SHO. He took the FRCS England in 1957.

In December of the same year he married Beverley Eade, a theatre nurse from Christchurch, in the same Pont Street church in which his father and mother, Gladys Macalister, were married 29 years before. The marriage was a lasting and happy union productive of three children.

Soon after his wedding he started work as a registrar at Addenbrooke's Hospital, Cambridge, and after 15 months there he returned to Christchurch Hospital as Resident Assistant Surgeon. This was a post that entailed doing most of the acute surgery for the city, before the opening of the Princess Margaret Hospital. He became FRACS in 1960.

In 1961 Jim was appointed Visiting Surgeon to the then North Canterbury Hospital Board and commenced private surgical practice, sharing consultation rooms in 'Harley' with his father. In 1964 the late Heath Thompson needed an assistant in the Thoracic Surgical Unit at the newly opened Princess Margaret Hospital and Jim spent six months training at the Cardio-Thoracic Unit at Greenlane Hospital. For the next eight years he was offsider for Mr Thompson in addition to his general surgical workload and his private practice. He developed special interests in thyroid and oncological surgery and conducted combined clinics with the isotope and oncological physicians. With acute call in both disciplines and two general and one thoracic 'arranged' list weekly his public hospital workload was onerous. This was quite apart

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from his private work, which grew steadily, especially after his father's untimely death.

The unexpected death of JW Ardagh in 1983 presented an opportunity for full-time hospital practice and Jim applied for and was appointed Director of Surgical Services and Head of the Department of General Surgery to the Canterbury Hospital Board. He relinquished the post in 1991 as the Health Reforms loomed and his long-standing problem of cardiac arrhythmia worsened. This was four years before his retirement.

From 1984 to 1990 Jim was Staff Representative on the Christchurch School of Medicine Council and a member of its Subcommittee on Postgraduate Education. He had long been on the Executive Committee of the Canterbury Postgraduate Society and Chairman of the same from 1978 to 1982.

At the commencement of the School in 1973 Jim was appointed Clinical Lecturer in Surgery; he went on to become Reader in 1985. He examined in surgery for the Final MB ChB on five occasions from 1973. He was a good teacher, drawing on an encyclopaedic memory; he brooked no nonsense and there were students who thought him gruff – but they remembered what he taught them!

From 1984 he directed his energies into RACS matters. He served on the NZ Committee of the Board in General Surgery for eight years, the first four as Honorary Secretary. Two years later he was elected to the NZ Committee and appointed to the Court of Examiners. He presented a few papers and co-authored some articles in surgical journals, but he was essentially a 'doer' rather than an author or speaker (for which he had no great aptitude or liking) and at his busiest period he had not too much time.

Outside his professional sphere, family life was important to Jim. After his father died he took over his large property, 'Memsie' in St Albans, added a swimming pool and kept up the neighbours' tennis court. It was here that his family grew up. A holiday house in Queenstown was well used; the trees provided plenty of highly dangerous chain-saw recreation and jet-boat expeditions produced equal parts of thrills and mechanical frustrations. When the children left home Bev and Jim moved to a new 'Memsie' – a 10-acre block at Clarkville – where they developed a beautiful formal garden in which Jim could indulge his liking for order by trimming many metres of hedge.

He had an abiding interest in the classics and history and read widely. Despite his remarkable memory he would often revisit books or chapters of books that interested him. He was an animal lover and always had a black Labrador dog named after some figure of Roman history. He was fond of music, particularly Mozart, Schubert and Handel, and the sound system he installed in his last home gave him great pleasure when his mobility became limited.

Late in 2002 Jim became tired and short of breath and was found to have a metastatic renal carcinoma. He was given a prognosis of weeks only and he moved swiftly to sell his Clarkville and Queenstown properties and to buy a house in Christchurch. All this activity seemed to stimulate him and, with the dedicated help of his doctor, his oncological colleagues, district nurses and especially Beverley and his children, he fought a valiant rearguard action against his disease. He was able to attend the 50th reunion of his 'Class of 53' in Dunedin in February and his '60 years on' reunion

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dinner at Christs College in September. He outlived his grim prognosis by 12 months – always realistic and philosophical and uncomplaining – and died peacefully at home.

Jim Bremner was essentially a shy person and always an honest one (sometimes brutally so). He had a wry sense of humour. His life was dedicated to doing his best for his patients and if he did not always gain their love, he certainly earned their respect and gratitude.

He will be greatly missed by his colleagues, his ex-patients and his family – his wife Beverley and children, Sally, Alister and Lizzie – to whom our deepest sympathy is extended.

We are grateful to Dr JRM Davidson for this obituary

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Graham Aitken Nuffield Medical Postgraduate Travelling Scholarship

Applications are invited from well qualified New Zealand medical graduates in the 25–35 age group for the above Scholarship.

The purpose of the Scholarship is to provide travel funds to enable New Zealand graduates to further their clinical medical training and research interests in the United Kingdom.

The Scholarship will provide up to three return air fares to the UK, together with allowances amounting to $3000.

Candidates for the Scholarship must submit a training or research programme for approval together with the name of a person in the UK who will provide salary and facilities.

For further information please consult the Deans of the Schools of Medicine or write to Professor AD Campbell, Honorary Secretary, Managing Trustees, Graham Aitken Nuffield Trust, C/- Department of Chemistry, University of Otago, PO Box 56, Dunedin. Email: adc@otago.net.nz

Applications must be submitted to Professor Campbell by 31 March 2004

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Medical Benevolent Fund

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

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
Tel: 0800 656161

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