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This Issue in the Journal

Ventolin to Salamol—a crossover study in New Zealand
S Reti

In July 1 2005, PHARMAC (the New Zealand body responsible for government-funded pharmaceutical subsidies) removed the subsidy on the Ventolin® metered dose inhaler (MDI) (salbutamol – GlaxoSmithKline) in favour of a chlorofluorocarbon (CFC)-free equivalent, Salamol® MDI (salbutamol – Baker Norton). This study utilised a validated Asthma Control Questionnaire to examine the effect on asthma stability of converting 36 asthma patients from Ventolin to Salamol for a period of 4 weeks. Six of the 36 had to withdraw prematurely, 15/36 returned to Ventolin at some point, and of the remaining patients, 15/36 93% had worse asthma stability.

Asthmatics: too drunk to drive? The time curve of exhaled ethanol levels after use of Salamol in normal subjects
O’Connell, L Beckert

This study measures the effects of the alcohol-containing metered dose salbutamol inhaler, Salamol®, on roadside breath testing available in New Zealand. The authors confirm that this inhaler affects the alcohol readings on the breath tests, sometimes giving a failed result (over the current legal limit of 400 micrograms (mcg)/L). However this effect is very transient in a normal population; all readings return to normal after 3 minutes. To reduce the likelihood of a failed roadside alcohol breath test due to the Salamol inhaler, the authors recommend that the asthma patient waits at least 5 minutes after 2 puffs of Salamol, although this time may need to be increased if an incorrect inhaler technique is used or more than 2 puffs are taken.

Complete reference ranges for pulmonary function tests from a single New Zealand population
S Marsh, S Aldington, M Williams, M Weatherall, P Shirtcliffe, A McNaughton, A Pritchard, R Beasley

This study provides a complete set of lung function reference equations for a New Zealand population. These equations are long overdue as those in current use are not valid, having been produced over 25 years ago. Implementation of the new reference equations will allow for more accurate interpretation of lung function tests which are important in the diagnosis, assessment and management of a range of respiratory conditions.
Defensive changes in medical practice and the complaints process: a qualitative study of New Zealand doctors
W Cunningham, S Dovey

A survey of doctors and interviews with doctors who have received a complaint are examined to identify patterns of change in their practice in response to complaints. Results indicate that doctors change their practice in response to complaints in ways that both improve and harm patient care. Improved care results from doctors meeting professional and societal expectations; from increased attention to the doctor-patient relationship; and from changes to remedy systemic error. However, there is evidence of defensive medicine, with doctors reporting that they over-investigate, refer, and admit patients to hospital in an attempt to reduce their risk of a complaint (or to improve their ability to defend one). Doctors also reported removing themselves from patient care and from communities to reduce their risk of complaint. Complaints focussing on individual doctors encourage defensive medicine, whereas complaints focussing on systems of care can improve sub-standard practice. The responsibility for changing doctors’ behaviour appropriately lies with both the profession and society; to achieve this, the authors call for an improved educational process linked to complaints.

Alcohol and drug treatment population profile: a comparison of 1998 and 2004 data in New Zealand
S Adamson, D Sellman, D Deering, P Robertson, K de Zwart

Nearly 300 randomly selected alcohol and drug workers in New Zealand were interviewed by telephone. A profile of clients presenting to services was obtained and comparisons were made with a very similar survey in 1998 undertaken. Two main findings related to drugs used and ethnicity. Although alcohol, cannabis, and opioid drugs (such as heroin) still predominate, amphetamine use in presenting clients has escalated. Secondly, a significant disparity between Maori and non-Maori follow-up rates was found.

C Wilkins, P Sweetsur, Sally Casswell

Increasing detections of amphetamine laboratories and seizures of amphetamine precursor chemicals in New Zealand (NZ) in recent years suggest that ‘amphetamine use’ is continuing to increase in this country. However, this ongoing success may merely reflect greater focus on amphetamine and better law enforcement against amphetamine offending. The aim of this paper is to track the level of amphetamine use in the NZ population from the three most recent national household drug surveys conducted in 1998, 2001, and 2003. We found that amphetamine use had increased in 2001 compared to 1998, but had levelled out in 2003 compared to 2001. While the perception of the price of amphetamine did not change between 2003 and 2001, there appeared to be some relative decline in the availability of amphetamine in 2003 compared to 2001.
We suggest that two environmental factors have contributed to the stabilisation of the amphetamine situation in New Zealand in 2003: (1) growing awareness of the mental health risks of methamphetamine use among young people, and ;(2) the legislative and law enforcement response to methamphetamine after 2001. We caution that methamphetamine remains a drug of serious concern in New Zealand and that future increases in is use can be expected based on other countries’ experiences.
Salomol and inequalities in New Zealand

Innes Asher

The paper by Reti in this issue of the Journal\(^1\) raises important issues for New Zealanders with asthma who have been unable to obtain Ventolin\(^\oplus\) as a fully subsidised metered dose inhaler (MDI) since 1 July 2005.

In this first study of the clinical effectiveness of Salomol\(^\oplus\) MDI versus Ventolin MDI in New Zealanders with asthma, adults using Ventolin MDI as their usual reliever changed to Salomol MDI for 1 month, and most found their asthma stability worsened, or they could not maintain asthma control on Salomol MDI alone, or they withdrew because they believed the Salomol MDI was ineffective.

The weaknesses of this study design are obvious: the small number of subjects; neither subjects nor investigator blinded to the treatment thus introducing bias; no crossover back to Ventolin; the short duration; and the study undertaken after a lot of adverse media publicity about Salomol MDI. Reti has mentioned most of these aspects in the discussion however. His findings add to the concern about the clinical effectiveness of Salomol MDI in the New Zealand situation,\(^2\) and further investigation is warranted.

There are several reasons why Salomol MDI may be perceived as less effective than Ventolin MDI.

Firstly, Salomol MDI is more likely to block than the Ventolin MDI. The dispensing port of Salomol MDI is smaller than Ventolin MDI, on average 250 microns compared with 580 microns respectively (measured on 20 Salomol MDIs and Ventolin MDIs by the Centre for Advanced Composite Chemicals, Department of Engineering, The University of Auckland June 2005; data available on request).

Secondly, Salomol MDI requires more pressure to push down the canister to activate the dose compared to Ventolin MDI presumably because of the smaller size of the dispensing port. The physical dimensions of the plastic casing and canister are also different and therefore the hand-hold and mechanics of finger/thumb actuation may feel different.

Thirdly, Salomol MDI requires regular washing and rinsing in real life usage to prevent blocking,\(^3\) whereas lack of washing of Ventolin MDI results in no reduction in delivered dose (although repeated use of Ventolin without washing can lower the particle size).\(^4\) The instructions for cleaning include taking the canister out of the plastic holder, washing and rinsing so that all detergent is rinsed off, drip dry overnight, and putting the canister back in the plastic casing.
Such cleaning may be impractical in certain situations as noted by members of the Medical Adverse Reactions Committee:

“...the nature of acute asthma attacks is such that patients may not be able to clean the inhaler if it blocks in an emergency. Acute asthma attacks may occur in isolated locations, or in other locations where cleaning the inhaler is not practical...patients commonly keep multiple inhalers in multiple locations, and therefore weekly cleaning of all these inhalers is not realistic.”

In addition for people with asthma using spacers (most children), there is potential for confusion as these instructions for rinsing the MDI are the opposite of those for spacers which must be washed but not rinsed (to leave on a coating of detergent which removes static charge) then left to drip dry.

Fourthly, brand switching favours the known product in which the patient has confidence. This is a particular factor in asthma where worry and anxiety (in this case about the efficacy of the inhaler) can adversely affect asthma control.6

Between March and June 2005, Medsafe’s Centre for Adverse Reactions Monitoring (CARM) received 773 reports of reduced therapeutic effect, clogging, or blocking of the device, as well as other complaints.5

Medsafe responded by commissioning independent in vitro studies of Salamol MDI. These studies confirmed blockage in about 40% of the inhalers which were reported to be blocked after regular use. However after cleaning, all Salamol MDIs met manufacturer’s specifications. So confidence in the in vitro performance of Salamol has been restored. Medsafe found that blocking was due to inadequate cleaning of the device, and that there was lack of patient awareness of the need for cleaning.3

What happens in real life? Despite package inserts for both Salamol MDI and Ventolin MDI specifying regular cleaning, many people do not clean their inhalers according to instructions.4,7 Those using Ventolin MDI appear to get away without washing them (and may not even need to4) whereas clearly some Salamol users do not.1–5

Even if all Salamol MDI users were to wash their devices according to the instructions, would the problem of less perceived efficacy disappear? This is unlikely because of the other factors mentioned earlier in this editorial. But to answer the question definitively, well-designed studies of clinical effectiveness need to be undertaken that demonstrate whether Salamol transfers well to real-world populations.

It is vital to examine the hypothesis: “Salamol MDI is as clinically effective as Ventolin MDI in asthma” with randomised double blind double dummy placebo controlled studies adequately powered in the key age groups of people using salbutamol MDI — children, adults and the elderly—to establish with greater confidence the similarity or difference between the two devices in clinical practice. Medsafe should commission such studies.

All New Zealanders with asthma need to have access to an effective β2-agonist MDI. No country in the World has undertaken the experiment of providing Salamol MDI as the sole supply of salbutamol MDI as PHARMAC had planned.

The good experience of Salamol MDI in the United Kingdom is in an environment where Ventolin MDI is freely available, and the characteristics of successful Salamol
MDI users in the UK are not known (they might be more conscientious device cleaners!).

When PHARMAC listed Salamol MDI as fully subsidised from 1 February 2005 they announced that it “will be the sole subsidised brand of salbutamol aerosol inhalers from 1 July 2005, and that all other brands will be delisted from 1 July 2005.”

Medsafe is critical of the lack of information given at that time to adequately inform patients and health professionals about the importance of cleaning. In response to public pressure, PHARMAC changed the decision so that both Salamol and Ventolin chlorofluorocarbon (CFC)-free inhalers were retained in the Pharmaceutical Schedule from 1 July 2005, with Ventolin reference-priced to Salamol. This has resulted in a part charge for Ventolin of NZ$2.00, augmented by pharmacists’ charges.

Such a small cost may seem inconsequential to many New Zealanders who already pay high prescription costs. However it may be an unaffordable cost for those on low incomes, the most severely affected being children.

About 200,000 New Zealand children live in poverty, having insufficient family income to afford all the necessities. Moreover, income is a major determinant of health, and especially affects the occurrence or severity of respiratory diseases including asthma.

As it stands, a parent on low income whose child benefits less from Salamol than Ventolin may have to make a choice between a Ventolin MDI and other competing costs for food, clothing, housing, education, or a doctor’s visit, or accept poorer asthma control.

This injustice hits the poor—disproportionately Māori and Pacific children who already have other obstacles in accessing healthy lifestyles and adequate asthma care. PHARMAC’s Māori responsiveness strategy states that “cost was a significant barrier to Māori accessing their prescriptions. The $3 part charge was considered as a barrier to many whānau [families], particularly when they have other competing priorities for their often low incomes, such as food and rent.”

PHARMAC’s decision-making in regard to Salamol has not met PHARMAC’s own strategy of identifying and removing health inequalities.

In conclusion, Reti’s study raises serious issues. Until the clinical effectiveness of Salamol MDI is properly studied in all age groups in “real life” situations, Salamol should not be the sole subsidised salbutamol in New Zealand and Ventolin MDI should be fully subsidised.

Conflict of interest: Professor Asher was a member of the Board of the Asthma and Respiratory Foundation from 2003–2005. She has received funding support for research from GlaxoSmithKline, AstraZeneca, Merck Sharp and Dohme, and Neolab (UK).

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5. Medicines Adverse Reactions Committee 22nd meeting minutes; 9 June 2005 4.3.3 http://www.medsafe.govt.nz/profs.htm


Lung function: normal for New Zealand?

Robert Hancox, Joanne Baxter

Like most laboratory measurements, interpreting the results of lung function tests requires reference values—what would be “normal” for the person being tested? Unfortunately, to a much greater extent than most laboratory tests, values of lung function depend on sex, age, body size, and racial or ethnic differences. Hence laboratories use reference equations derived from normal populations (i.e. non-smokers with no history of respiratory disease) to predict what an individual’s lung function should be based on these measures. Ideally these reference equations should be generated from the local population. Moreover, the reference equations should be checked and updated every few years to take into account changes in equipment and testing protocols and possible changes in lung health.

To this end, the new reference equations developed by Marsh et al, and published in this issue of the Journal, are a valuable contribution to pulmonary function testing in New Zealand. However, the development of these new equations raises several issues.

The first of these is whether they will be taken up by New Zealand laboratories. A comprehensive set of prediction formulae for New Zealand Europeans has been available for more than 25 years, but is used by very few lung function laboratories. One reason for this may be that new lung function equipment usually arrives programmed with European and/or American reference equations. Changing these to local reference values is not always easy. Maybe there is also a sense that prediction formulae developed by large American or European studies will be better than home-grown equations. If so, this is certainly wrong—the existing New Zealand equations predicted the lung function healthy participants in Wellington Respiratory Survey at least as well as any overseas formulae and were substantially better than the widely used European (ECCS/ERS) equations.

A bigger problem is that we have no reference equations at all for Māori. The community-based survey by Marsh et al had insufficient non-Europeans to make meaningful equations. However, their study makes it clear that we cannot assume that prediction formulae for Europeans will adequately represent Māori: if formulae derived from Europeans living in Europe underestimate the lung function of Europeans living in New Zealand how much difference might there be between ethnic groups within New Zealand?

Studies in other countries suggest that those of African, Japanese, Indian, and other non-European ethnicities have lower lung function for their body size than Europeans. In these countries, predicted values for non-whites are sometimes adjusted by subtracting an “ethnic factor” of 12% from the predicted values for Europeans. This is clearly unsatisfactory.

This issue is particularly important since Māori are disproportionately affected by the most common respiratory diseases. In other words, we have no normal values for the New Zealanders who are most likely to require the services of a lung-function
laboratory. The situation is a little better for Pacific Island people living in New Zealand for whom spirometric values have been validated against standard prediction formulae. Perhaps surprisingly, the ECCS/ERS values performed very well. Thus while the “European” equations do not accurately predict the lung function of New Zealand Europeans, they may be adequate for New Zealand Pacific Islanders.

The importance of developing local values was discussed earlier this year at the Australia and New Zealand Society of Respiratory Scientists/Thoracic Society of Australia and New Zealand meeting in Canberra, Australia. The data presented by Marsh et al highlight the importance of using reference equations matched to the local population. For New Zealand, the priority is now to develop prediction equations for Māori.

The work of recruiting and testing normal volunteers could be shared among several laboratories. The task would be considerably less demanding than the current laboratory accreditation procedures and would be more worthwhile—after all there is little point in producing accurate measurements if we don’t know how to interpret them.

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Alcohol and other drug treatment in New Zealand—one size doesn’t fit all

Ross McCormick, Chris Kalin, Terry Huriwai

Treatment services for those with alcohol and other drug problems change over the years, sometimes due to the whim of bureaucrats, sometimes due to political pressure, but we always hope to be informed by evidence.

Adamson et al’s paper, 1 in this issue of the Journal, builds on the evidence needed to inform alcohol and other drug treatment policy, service, and workforce development. Between 1998 and 2004, the average alcohol and drug treatment-seeking client has aged and is less likely to be Caucasian, although Māori are over-represented in both years.

Māori clients are younger than other clients, more often use cannabis, and are less likely to live in a large city. Amphetamine users are younger and domiciled mainly in the North Island. In 2004, people were seen more often in community-based clinics and fewer were admitted to residential care. Those retained in follow-up treatment following an initial assessment are more likely to be female, non-Māori, and opioid users, although ‘kaupapa Māori’ services retain Māori clients better.

Since 1998, treatment-oriented policy has favoured community-based treatment over residential treatment services, and several residential treatment services have closed. Even so, the number of residential places has now returned to almost the levels seen around the year 2000. The 1980s and 1990s saw an increase in ‘kaupapa Māori’ services, but there has been little development in recent times.

District Health Boards are responsible for the planning and funding of alcohol and other drug treatment services in their areas, with direction and monitoring by central government.

New Zealand’s alcohol and other drug treatment strategy should take into account the findings of papers such as Adamson et al. The evidence in this paper helps build a clearer picture of the number, location, and types of services needed. It also has several implications for workforce development.

What does Adamson’s paper suggest to us?

First, where have the young, Caucasian, non–opioid users gone? (Pacific and Asian people were not mentioned in the survey). Māori treatment attendees were younger, as were amphetamine users. Problem use of substances usually starts when young, and New Zealanders use a variety of drugs if only because of our inconsistent supply of opioids.

What attracts someone to treatment for alcohol and other drug-dependency or problem use? To seek treatment, an individual needs to be aware that they have a problem and to consider that the benefits of continued substance use are outweighed by the problems they are encountering. Coming to this awareness can occur as someone ages, but is enabled by good point of first contact health and legal services
that can detect the reasons behind a presentation and assist those with problematic substance misuse to think about the issue.

These services obviously need to be acceptable to youth. It could be that our services have not fully adjusted to changing youth culture and drug use patterns. Youth consumer networks, Pacific groups, Asian leaders, and recovering drug users may well be able to offer advice.

Second, substance-dependent clients come from a variety of backgrounds—some have never worked, some have little education, and some have a criminal history. Their needs have to be analysed and appropriate habilitation offered. Those with complex presentations need more intensive treatment than community-based outpatient services can offer. The sooner there is an intervention in the course of development of a substance-misuse problem, the more likely a positive outcome can be achieved. Also, the longer a substance misuser attends a treatment process, the better the outcome. Follow-up or after-care is a core component to sustaining recovery.

Despite the welcome recent decision of the prison service to continue to offer opioid treatment services in prison, and a wider interest by Government in reducing prison numbers and re-offending by addressing substance misuse, good habilitation will have less chance of success in a prison than in a residential treatment service designed for that propose. Indeed, it could be that more residential treatment habilitation centres are needed as an alternative to prison.

Third, Māori are over-represented in the statistics Adamson describes. It is worrying to see that fewer Māori engage in the treatment process after the initial contact—the reasons need to be explored urgently. However, a success signalled in Adamson’s paper is the better retention rate of Māori in ‘kaupapa Māori’ services. That fact needs to be celebrated and considered alongside the recent political arguments suggesting less “special” treatment for Māori. This finding alone would suggest that the choice of a Māori alcohol and other drug habilitation service should be readily accessible or at the very least there should be increased Māori responsiveness in non dedicated Māori services. Indeed, further development of ‘kaupapa Māori’ services should be encouraged and supported.

Fourth, over the last few years we have seen a rise in amphetamine use. Amphetamines are not a new drug in New Zealand; they have been abused for at least the last 35 years. A fascinating finding in Adamson’s paper is that treatment-seeking amphetamine users mostly live in the North Island. This, together with the needs of under-represented groups in treatment such as the young and over-represented groups such as Māori, suggests that it is time for a rethink of the ideal service mix for alcohol and other drug treatment services in each region in collaboration with District Health Boards and local and regional advisors.

We congratulate Adamson and his colleagues on their research, at the very least it suggests that a “one size fits all” treatment service is not the way of the future.
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Asian Health Chart Book 2006: foundation for a new health agenda in New Zealand?

Max Abbott, Wilson Young

One-in-five New Zealanders are foreign born, a high figure by international standards. Moreover, since the late-1980s, many recent migrants have been of Asian ethnicity. Indeed, migrant and long-resident Asian New Zealanders together make up almost 10% of the total population. This figure is expected to reach 15% by 2020, more than double the current percentage for Pacific peoples.¹

Despite demographic reality and the *New Zealand Health Strategy*² goal to monitor the health of all New Zealanders, little has been done to determine Asian health status. Applications from independent researchers confront other funding priorities and official surveys typically lack focus and statistical power. The recent national mental health survey, which over-sampled major ethnic groups other than Asians, is a case in point.³ There is no mention in the survey report of either Asian or migrant mental health. In Ministry of Health and other government reports, Asian and European data are often merged, or the former consigned to a residual ‘other’ (non-Maori, non-Pacific, non-European) category.

The paucity of information concerning Asian health has been partly addressed by the recent publication of Asian-specific findings from the 2002–03 National Health Survey and an earlier survey of secondary schools.⁴,⁵ The Ministry of Health’s *Asian Health Chart Book 2006*⁶ incorporates information from these and other sources to provide more extensive coverage of the health status of New Zealand’s Asian population.

The article in this issue of the Journal entitled *Asian health in New Zealand—progress and challenges*⁷ provides a timely overview of these reports and other relevant studies and briefly considers their implications for future research, health policy, and services. It also sets a challenge for government and all of us in the health sector.

In terms of crude and avoidable mortality, Asian New Zealanders (especially Chinese) compare favourably with the rest of the population. This is also the case for many other health indicators. However, there is considerable variation between different Asian peoples, with those of Indian ethnicity having very high rates of low birth weight, obesity, type 2 diabetes, and cardiovascular disease.

On some indices, sectors of the Asian population experience greater morbidity than any other ethnic group in this country. These differences are not unexpected given the enormous diversity encompassed by the term ‘Asian’—including differences in culture, pre-migration experience (including refugee status), language use, education, time resident, acculturation, socioeconomic deprivation, and exposure to prejudice and discrimination.

Substantial heterogeneity can be obscured when data are ‘averaged.’ This is not unique to Asian health. For example, a recent survey of postnatal depression among Pacific mothers in Auckland found that while overall prevalence (16%) was...
somewhat higher than in previous New Zealand general population studies, Samoans had one of the lowest rates reported internationally (8%) and Tongans one of the highest (31%). Such findings are not unusual and caution against over-reliance on broad ethnic groupings in research and health policy.

While there are differences between Asian peoples, there are also similarities. These include minority status, barriers to employment, mental health issues among young people, and low levels of access to health services including primary health care and screening for some specific disorders.

Relative to Europeans, Chinese, Indian, and other Asian New Zealanders also share significantly lower rates of alternative or complementary healthcare utilisation. These three Asian groupings are also alike in that for almost all health indicators first-generation and recent migrants do better than those who are New Zealand-born or have lived in the country longer.

Although migrants often experience early adaptation difficulties and can be at risk for some health problems (particularly mental health problems), the ‘healthy migrant effect’ is well documented. It is largely a consequence of selective migration. The Asian Health Chart Book notes that this advantage is likely to be short-lived “as the selection effect wears off and acculturation progresses.”

The relative invisibility of Asian health in New Zealand research and past Ministry reports has been matched by a vacuum in public and personal health services policy and practice. New Zealand Health Strategy principles include “good health and wellbeing for all,” “timely and equitable access for all New Zealanders to a comprehensive range of health and disability services,” and “active involvement of consumers and communities at all levels.”

For Asian peoples in New Zealand, reality, at ground level, falls short of the rhetoric. This will need to change if we are to address current specific morbidities and counter the more pervasive chronic disease burden that will result when the ‘healthy migrant effect’ dissipates with prolonged exposure to life in New Zealand.

Pioneering work by The Asian Network Inc, university research centres, individual officials and health professionals, Waitemata and Auckland District Health Boards, and various others has been strengthened by publication of the Asian Health Chart Book.

We commend the Ministry for this initiative. An Asian Health Agenda is now required to provide a focus for further research, workforce development, greater engagement in mainstream and targeted health services, and the development of ethnic-specific health protection and promotion initiatives that will enable Asian New Zealanders to maintain healthy lifestyles, prevent disease, and minimise disability.

Disclaimer: The views expressed here are those of the authors and not necessarily those of their employing bodies.

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Ventolin to Salamol—a crossover study in New Zealand

Shane Reti

Abstract

**Aims** To assess asthma stability in adults converted from Ventolin® to Salamol®.

**Methods** Thirty-six general practice adults with documented asthma and using Ventolin at least weekly in the previous 12 months, changed their Ventolin for Salamol for a period of 4 weeks. The validated Asthma Control Questionnaire was applied at the beginning and end of the study period.

**Results** Of the 36 adults, 6/36 (17%; 95%CI 4–29%) prematurely withdrew mainly due to Salamol ineffectiveness. A further 15/36 (42%; 95%CI 25–58%) could not maintain Salamol alone and returned to Ventolin at some time during the study period with 10/15 (67%; 95% CI 42–91%) citing Salamol ineffectiveness. Of the remaining 15/36 who maintained the study design, nearly all had worse asthma stability 14/15 (93%; 95% CI 80–100%).

**Conclusions** Asthma stability was significantly worse with Salamol compared to Ventolin. Psychological features related to changing inhalers, different physical aspects of Salamol inhalers, and pharmacological ineffectiveness are possible explanations.

In July 1 2005, PHARMAC (the New Zealand body responsible for government-funded pharmaceutical subsidies) removed the subsidy on the Ventolin® metered dose inhaler (MDI) (salbutamol – GlaxoSmithKline) in favour of a chlorofluorocarbon (CFC)-free equivalent, Salamol® MDI (salbutamol – Baker Norton).

From February 2005, the government agency responsible for monitoring adverse drug reactions—Centre for Adverse Reactions Monitoring (CARM)—noted increasing reports relating to patients crossing over from Ventolin to Salamol, even “exceeding the normal capacity of CARM’s processing systems, and exceeding the usual reporting rate for brand switching complaints”.

The three main complaints were decreased therapeutic effect, blockage, and taste. Paediatricians also reported particular concerns for children converting to Salamol, and questioned the overall cost effectiveness of the crossover.

A formal investigation was undertaken by Medsafe, the government agency responsible for registering pharmaceuticals, which primarily examining the functionality of both new (16 inhalers) and returned faulty inhalers (33 inhalers) against such measures as dose deposition, content uniformity, and average dose per actuation.

Medsafe’s report was published in December 2005 with the main finding pointing to device blockage as the likely main cause for decreased therapeutic effect. Increased patient education and adherence to the manufacturer’s weekly cleaning recommendations was the suggested solution. Under these conditions, the testing of
Salamol appeared to pass appropriate laboratory tests of functionality, however it is a completely different issue to then discuss the clinical implications for asthmatics and the overall effects on their asthma stability”.

Several studies have demonstrated clinical equivalence between various salbutamol-containing MDIs with and without CFCs.\(^3\text{-}^8\) One question is whether these findings can be generalised to all CFC-free salbutamol-containing MDIs, or whether company specific formulations and MDI design have differences that matter for asthma stability. Lumry et al drew attention to CFC-free salbutamol MDIs variably containing “excipients such as oleic acid, lecithin, or alcohol”\(^9\).

Lee has suggested that patient familiarity with the physical aspects of salbutamol inhalers is important for asthmatics.\(^10\) To this effect, there are few studies that specifically compare brand name Salamol with Ventolin as is being introduced in New Zealand. A 1996 randomised crossover study of 10 patients found both to be equally effective,\(^11\) as did a 1994 crossover study of 11 patients.\(^12\)

Comparing Salamol and Ventolin in the current New Zealand setting is significant in that New Zealand has the one of the highest asthma rates in the world (15% of adults, 20% of children\(^13\)), the range of available asthma inhalers is less than in larger countries, and medication choice is mostly driven by government subsidies.

Ventolin has been the main stay of beta-agonist treatment for many years. In this context, this study examines the effects of converting asthma patients from Ventolin to Salamol.

**Method**

Subjects were computer selected from the general practice of the author with the following criteria:

- Registered patients aged 17 years and over at 1 April 2005.
- An existing diagnosis of asthma.
- Prescribed Ventolin in the previous 12 months.
- Using Ventolin at least weekly.

Subjects then met with the author (SR) who explained that the study was exploring the conversion from Ventolin to Salamol, that the author had faith in both, and safety measures associated with the study. Signed patient consent to participate was then obtained.

The author then applied the validated New Zealand version of the Asthma Control Questionnaire which is a combination of validated questions on asthma symptoms, limitations, inhaler usage, and a peak flow measurement.\(^14\) Demographic questions on age, gender and ethnicity were also asked.

Subjects were then given a prescription for Salamol, and instructed to completely replace Ventolin with Salamol for a period of 4 weeks. All existing medications, asthma and non-asthma related, were to remain unchanged.

Four weeks later, the patients were recalled for follow-up and the Asthma Control Questionnaire reapplied. During the study, any subjects who were unable to complete the study, or who had to return to Ventolin, were returned for consultation, and appropriate questioning made with responses recorded.

As per the questionnaire validation, a change in asthma stability was accepted as being a change of > 0.5 points on the questionnaire scale. The statistical means were calculated for all categories, with the standard error of the mean reported as 95% confidence intervals.

**Results**

Thirty-six subjects were initially enrolled in the study, 21 women and 15 men with an age range of between 17–76, and an average age of 48.83 years. None had hospital
admissions for asthma in the previous year; 18/36 (50%) were using a preventer daily, and the average percentage predicted peak expiratory flow rate was 70.11%.

Of the 36, 6/36 (17%; 95% CI 4–29%) withdrew during the month-long study period; 5 of the 6 (83%, 95% CI 53 – 100%) withdrew due to Salamol ineffectiveness, and the remaining subject withdrew citing the unpleasant taste of Salamol.

A further 15/36 of the original subjects (42%; 95% CI 25–58%) could not maintain Salamol alone, and had to return to Ventolin at some point during the month.

Of the 15 who returned to Ventolin, 10/15 (67%, 95% CI 42–91%) cited Salamol ineffectiveness, 1/15 device blockage, 3/15 the convenience of having Ventolin handy, and 1/15 gave no reason.

This left 15/36 (42%; 95% CI 25–58%) of the original subjects successfully maintaining ‘Salamol only’ as per the study design. Figure 1 shows a flow diagram of participant outcomes.

Assessment of asthma stability with the asthma control questionnaire showed 14/15 (93%; 95% CI 80–100%) had worse asthma stability, and the remaining subject had unchanged asthma stability; 2 of the 15 (13%; 95% CI 0–31%), who maintained Salamol only, gave possible influences on their asthma over the study period—both citing chest infections.

**Figure 1. Flow diagram of participant outcomes**

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Discussion

Only 15 subjects out of 36 in this study were able to maintain the study design using Salamol; and in this group of 15, 93% has worse asthma stability.

For all study participants, Salamol ineffectiveness was a significant factor. However these findings need to be considered in the context of several potential limitations. Firstly, study numbers are small (this particularly limits demographic analysis), although consistent in size with several other asthma inhaler crossover studies.\textsuperscript{11,12,15}

Secondly, there are external variables such as the weather, pollen counts, winter chest infections, change in home or work environment or lifestyle, change in preventer use, and negative media that could have had an influence over the month-long study period. Most of these variables are discussed under the heading “Peripheral Related Factors”, and to summarise here, are not considered to be significant influences in this study.

Self directed changes in steroid usage could influence the findings, however none of the subjects completing the study reported changed their steroid inhaler usage. Furthermore, Bleecker et al demonstrated the non-significance of inhaled steroid use in a similar crossover study comparing Airomir\textsuperscript{®} CFC-free (another CFC-free formulation of salbutamol) and Ventolin.\textsuperscript{16}

There are several explanations for the findings of apparent ineffectiveness and deteriorated asthma stability on the crossover to Salamol. These can be considered under the headings patient-related, peripheral-related, or product-related factors.

**Patient-related factors**—Patient related factors primarily relate to how change alone, and not Salamol ineffectiveness, may contribute to the findings.

Indeed, anxiety, apprehension, and caution are likely to be the norm in a crossover study of this type, possibly precipitating an early return to a previous trusted inhaler. This uncertainty under change was demonstrated in a crossover study of 29 patients using either branded salbutamol, generic salbutamol, or their usual salbutamol (blinded).

Juniper concluded “patients’ own assessment of their relief inhaler seems to be influenced by factors other than efficacy”.\textsuperscript{15} If change is accepted as an operative factor, then with these two products the effect was further magnified by Salamol being significantly smaller in size than Ventolin, having a less forceful particle actuation, and having a different taste.

Lee has suggested that patient familiarity with the physical aspects of salbutamol inhalers is important for asthmatics.\textsuperscript{10} While change-related patient factors may well have contributed to subjects returning to Ventolin and reporting ineffectiveness, these factors cannot fully account for the 93% who maintained Salamol only, and who by ACQ testing had worse asthma stability.

**Peripheral-related factors**—These factors are outside the control of the study design and are known to have an influence in asthma, and could possibly have contributed to Salamol ineffectiveness. These factors include weather, pollen, chest infections, change in home or work environment or lifestyle, and negative media.
To assess the effect of these external factors, subjects were asked if they were aware of anything that might have altered their asthma over the study period. This form of subjective self-analysis is more blunt than definitive, however it did contribute some useful information suggesting a small influence particularly from chest infections.

External effects were also minimised by choosing a month that was not a recognised change of season month, nor overtly mid-winter.

Salamol had also received mostly negative reporting in the media up to 5 months prior to the study onset. The effect of this on this study is difficult to assess, however it was mitigated as far as possible through patient education in the consent process, and the unbiased support for Salamol from the author as their family doctor. Overall, the influence of these peripheral factors is considered to be minimal.

**Product-related factors**—The apparent ineffectiveness of Salamol in this crossover study is in contrast to the few previously mentioned crossover studies from Ventolin to Salamol.

In this study, subjects complained about the taste, device blockage, and the uncertainty of whether they were getting anything in their mouth after using Salamol.

Other studies with similar products e.g. Ventolin and Aeromir (another CFC-free formulation of salbutamol) demonstrate a slower particle speed for the CFC-free formulation that may account for the delivery uncertainty. Bamber reported blockage difficulties with CFC-free inhalers and Chew confirmed blockage-induced reductions in fine particle mass requiring weekly mouthpiece washing. These device-related factors may certainly account for the observed poor asthma stability, and to this must be added the final possibility that the absolute effectiveness of CFC-free salbutamol in Salamol is simply not as effective as that in Ventolin.

It is possible that inter-company preferences for non-active ingredients such as excipients eg alcohol, alters the effectiveness of Salbutamol itself.

**Conclusions**

While promoted as being pharmaceutically similar, Salamol was less effective than Ventolin in this study. This could be due to several factors, including true differences in active ingredient efficacy, physical differences in inhaler devices, and subject-related change anxiety. Further work needs to be done to identify these individual factors with greater recognition especially, of the “change-related” contributions to asthma.

If the physical delivery features of a device are different, and patients are not adequately reassured, educated, and safely trialled, then it is highly likely any new asthma inhaler introduction will face difficulties no matter how bioequivalent it may pharmaceutically turn out to be.

**Disclosures:** The author of this article is an independent researcher and has not been the recipient of any funding from GlaxoSmithKline (the manufacturer of Ventolin), or funding from any other source.

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Acknowledgments: I thank Allen Liang for peer review and Mike Mullany for statistical support.

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Asthmatics: too drunk to drive? The time curve of exhaled ethanol levels after use of Salamol in normal subjects

Oisin O’Connell, Lutz Beckert

Abstract

Salamol®, a new chlorofluorocarbon-free metered dose inhaler (MDI) containing salbutamol with small amounts of ethanol as a co-solvent, has been introduced in New Zealand. We evaluated the effect of breath alcohol levels after two inhalations from this MDI, and plotted a time-response curve. We tested 16 healthy volunteers. The breath alcohol levels rose to 1932 mcg/L immediately after inhalation with a standardised technique. We concluded that in all normal subjects their breath test was negative after 3 minutes, and at least a 5 minute wait post Salamol inhalation is necessary before roadside breath testing should be undertaken, otherwise a failed breath test is likely.

Until recently, chlorofluorocarbons (CFCs) have been used as propellants in metered dose inhalers (MDIs). CFCs react with UV light to form oxygen radicals, which react with ozone. The damage to the ozone layer has had wide attention and is particularly relevant for New Zealand.

The Montreal Protocol demands the complete substitution of “Substances that deplete the ozone layer”.¹ Under the Protocol, the production of CFCs was to be phased out by 1996, although there was an exemption for limited production of CFCs for use in MDIs for patients with conditions such as asthma and chronic obstructive pulmonary disease until the end of 2005.

CFC concentrations in the stratosphere are still increasing because CFCs emitted in earlier years continue to rise to the stratosphere (the most widely used CFCs remain in the atmosphere for between 50–100 years). Scientists predict that ozone depletion will reach its worst point during the next few years and then gradually decline until the ozone layer returns to normal around 2050.

It is acknowledged that the transition to CFC-free MDIs would take several years while manufacturers replaced the CFCs with other propellants. Regarding the use of CFCs in asthma aerosol inhalers, hydrofluoroalkanes (HFCs), sometimes referred to as HFCs (hydrofluorocarbons), are the preferred replacements because they contain neither chlorine nor bromine and therefore have no detrimental effect on stratospheric ozone. The most widely used HFA is 1,1,1,2-tetrafluoroethane/HFA-134a such as used in Norflurane®, which has only one-third of the ‘greenhouse’ (global warming) effect of the CFCs it replaces. HFA-134a is used as it is a good solvent but requires ethanol to keep the active compound dispersed.

HFA, along with sulphur hexafluoride and perfluorocarbons, have a large heat generating capacity. However as they account for only a small percentage (for example 1.8% in the USA) of the greenhouse effect gases, their contribution to global warming is negligible. So although HFA has no effect on the ozone layer, it does have...
an effect on greenhouse warming.\textsuperscript{2} The Kyoto Protocol devised mechanisms for the management of climate changes, especially with regard to the reduction in the production of gases that can cause global warming. The six greenhouse effect gases (carbon dioxide, methane, nitric oxide, hydrofluoroalkane, perfluorocarbon, and sulphur hexafluoride) were gathered into a single group. This approach gives countries more flexibility to choose the percentage of reduction of each gas, so that they can achieve the total reduction goal established by the protocol.

A recent case report\textsuperscript{3} in the \textit{New Zealand Medical Journal} reported on a positive breath alcohol test in a non-drinking patient with asthma following inhalation of a Salamol\textsuperscript{®} inhaler. The report led us to measure the actual levels of breath alcohol post inhalation and to plot these levels over a time period.

In New Zealand, up to four steps are currently involved in roadside breath alcohol testing of motorists:

- Firstly, a baseline screening roadside breath test (RBT) is undertaken by speaking in to an Alotech\textsuperscript{®} AR1005—this establishes whether alcohol is present.
- Secondly, a blowing test gives a pass/fail result as measured again on an Alotech.
- Thirdly, a quantitative assessment is measured using an Ethylometer\textsuperscript{®} (679T). The current legal limit in New Zealand is less than 400 micrograms (mcg)/L.
- Fourthly, blood test sampling can be requested.

**Methods**

Sixteen healthy doctors (aged 22–43 years) participated in this study. Baseline measurements included height, weight, age, and a screening for breath alcohol levels measured on an Alotech. None of the subjects had a history of asthma or any other respiratory disorder. None of the subjects were taking any medications and none had taken alcohol in the preceding 12 hours.

The participants were instructed to take two breaths from the Salamol inhaler without a spacer. The doctors used a standardised technique involving shaking the MDI, then inhaling and holding for 10 seconds and repeated immediately afterwards.

Participants were then instructed to breath in to an Ethylometer to measure breath alcohol levels at 0, 1.5, and 3 minutes. Ten doctors repeated the test on a subsequent day with levels measured 1 and 2.5 minutes post inhalation. The time interval was necessary because the Ethylometer had a calibration time of 1.5 minutes between measurements. All doctors were also tested on the roadside using an Alotech to determine how long it took for the person to reach a pass screening test at 1, 2, and 3 minutes. See Table 1.

**Table 1. Number of subjects performing each measurement at each interval**

<table>
<thead>
<tr>
<th>Time (minutes)</th>
<th>Alotech (number of participants)</th>
<th>Ethylometer (number of participants)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>1.5</td>
<td>0</td>
<td>10 (on day 2)</td>
</tr>
<tr>
<td>2</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>2.5</td>
<td>0</td>
<td>10 (on day 2)</td>
</tr>
<tr>
<td>3</td>
<td>16</td>
<td>16</td>
</tr>
</tbody>
</table>
We used Pearson’s correlation coefficient to test the associations between peak ethanol level, age, height, and weight.

**Results**

All subjects had a negative baseline measured breath alcohol screening level. All of our subjects had a “fail” reading on breath testing using the Alotech ‘Random Breath Tester’ after 1 minute, 44% after 2 minutes, and all subjects passed after 3 minutes.

The use of the Salmol inhaler transiently increased breath alcohol levels above the legal limit for driving as measured on the Ethylometer. The results are shown in Figure 1. The highest level measured after immediate inhalation is 1932 mcg/L, with a mean measurement of 982 mcg.

**Figure 1. Time response curve for breath alcohol level using Ethylometer (679T) after Salamol inhalation for 16 normal volunteers. The mean and standard deviation of the volumes are shown**

In New Zealand, the legal limit for driving is 400 mcg/L. All 16 of our subjects recorded a reading above this level immediately after Salamol inhalation on the Ethylometer (679T). An average level of 913 mcg/L was reached, and a maximum of 1932 mcg/L was observed immediately after inhalation—see Table 2. We did not find any correlation between height, weight, or age against alcohol level reached.
Table 2. Results of subjects’ breath testing on Alotech and Ethylometer testers

<table>
<thead>
<tr>
<th>Time (minutes)</th>
<th>Alotech</th>
<th>Ethylometer (mcg/L; mean reading)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Not tested</td>
<td>Not measured</td>
</tr>
<tr>
<td>Immediately post-inhalation</td>
<td>No alcohol detected in subjects</td>
<td>913</td>
</tr>
<tr>
<td>1</td>
<td>16 failed (100%)</td>
<td>154</td>
</tr>
<tr>
<td>1.5</td>
<td>Not tested</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>7 failed (44%)</td>
<td>Not measured</td>
</tr>
<tr>
<td>2.5</td>
<td>Not tested</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>All passed (100%)</td>
<td>0</td>
</tr>
</tbody>
</table>

Discussion

The results demonstrate that Salamol increases alcohol breath levels to almost three times above the current New Zealand legal driving limit immediately post inhalation. These levels return rapidly to baseline level within 3 minutes in our cohort. Our results are for two actuations of the inhaler, however current guidelines suggest up to 20 repeated actuations may be given in an acute asthma exacerbations,4 and in this case, breath alcohol levels are likely to be even higher as seen in previous studies.5

Our study obviously has several limitations, including small cohort, all young and healthy doctors, and there may be different pharmacokinetics in an asthmatic and COPD population (possibly prolonging the ethanol elimination from the lung).

Other studies have previously shown that subjects suffering from asthma/COPD can occasionally experience difficulty in providing adequate breath samples for evidential breath alcohol testing devices due to inadequate Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV₁). They may, therefore, resort to the use of bronchodilators such as salbutamol to improve their respiration—this may again have implications with the new generation of inhalers using ethanol as a solvent.6,7

Previously, studies have also demonstrated that ethanol levels post inhaler can be as much as double if a poor inhaler technique rather than a standardised-trained technique is used.8 One study in the asthmatic population recommended waiting between 10–15 minutes after certain ethanol MDIs be fore undertaking evidential breath testing.9

Ignacio-Garcia et al’s study of an asthmatic population gave ethanol readings of 450 mcg/L at 1 minute following 2 puffs of Butoasma® (salbutamol with ethanol) and 80 mcg/L after 3 minutes, interestingly they also measured alcohol levels in patients using Ventolin® which does not use alcohol as a propellant and found the levels to be 350 mcg/L at 1 minute post inhalation.5 This compares with a mean level of 154 mcg/L at 1 minute and 0 mcg/L after 3 minutes in our population group.

We recommend allowing at least 5 minutes post 2 puffs of Salamol before breath testing, although this time may need to be increased if a incorrect inhaler technique is used or more than 2 puffs are taken.

Conflict of interest statement: Neither of the authors has received research funding from any other manufacturer of asthma medications.
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References:


Complete reference ranges for pulmonary function tests from a single New Zealand population

Suzanne Marsh, Sarah Aldington, Mathew Williams, Mark Weatherall, Philippa Shirtcliffe, Amanda McNaughton, Alison Pritchard, Richard Beasley

Abstract

Aim Reference equations are prerequisites for interpretation of pulmonary function tests and are important in diagnosis, assessment, and management of a range of respiratory conditions. Such equations should be derived from populations who are closely ethnically and anthropomorphically matched to those in whom the equations will be used. This paper uses measurements from a single cohort of New Zealand adults to derive reference equations for all major pulmonary function tests.

Methods Detailed pulmonary function test results including measurement of FEV$_6$ and airway resistance were obtained from a cohort of 212 adult New Zealanders of European origin, who were never smokers with no respiratory disease or symptoms. Equations were developed by linear regression, including sex and other candidate variables based on prior univariate analysis. Comparisons between measured and predicted values using the reference equations of the European Respiratory Society (ERS) were made.

Results Reference equations were produced with high values of explained variance ($R^2$) for many commonly used clinical parameters. When compared with ERS equations, measured values for spirometry and most lung volumes were significantly higher than predicted (mean difference FEV$_1$: male 0.48 L, female 0.36 L, mean difference TLC male 1.14 L, female 0.89 L, SVC male and female 0.66L).

Conclusions This study provides a complete set of contemporary pulmonary function reference equations for a New Zealand population of European origin.

Reference ranges for pulmonary function tests are a prerequisite for the accurate interpretation of results. They are also important in a wide range of respiratory disorders in which guidelines concerning diagnosis, assessment of severity and management rely on lung function measurements expressed in relation to reference values. The combined American Thoracic Society (ATS) and European Respiratory Society (ERS) guidelines encourage regular review of reference equations suggesting 10 years as an appropriate equation lifespan.

Reference equations should be derived from study populations closely anthropomorphically and ethnically matched to the populations in whom they are used. This implies it would be beneficial to use reference equations derived from the same study population for all parameters. However, few authors have supplied such data, and although numerous equations have been produced for spirometry, fewer are available for static lung volumes and gas transfer. The most recently derived reference equations from a New Zealand population were produced over 25 years ago.
The widely used equations of the European Community for Steel and Coal (ECSC), first published in 1983 and adopted by the ERS in 1993, provide a single source of reference ranges for many commonly measured respiratory parameters. These equations have frequently been used in studies of respiratory function, including the European Community Respiratory Health Survey (ECRHS) in which New Zealand played a significant part. However, although these equations are presented as a single source, they are composite equations derived from many separate studies conducted in different populations.

Although validated at the time of their original publication, more recent data suggests that at least some of these equations, particularly for spirometry, are now out of date in European populations. In addition, ECSC reference ranges are not available for newer parameters such as the forced expiratory volume in 6 seconds (FEV$_6$), which may become increasingly used as a substitute for FVC in the detection of airflow obstruction.

To address these issues, we obtained a complete set of pulmonary function tests from a single population of non-smoking New Zealand adults of European origin, free from respiratory disease and symptoms. Reference equations were derived, using linear regression techniques, for all commonly measured respiratory function parameters. Pulmonary function measurements were compared to those predicted by both the ECSC/ERS equations and more recently published equations for static lung volumes.

**Methods**

**Study participants**—Study participants, forming part of the Wellington Respiratory Survey (WRS), were recruited using a postal questionnaire sent to 3500 individuals, aged 25 to 75 years, randomly selected from the electoral register. Subjects returning completed surveys were invited to undertake a more detailed, interviewer-administered, questionnaire followed by pulmonary function tests. To increase the number of normal participants, healthy subjects were also recruited from a concurrent study investigating the pulmonary effects of marijuana smoking. This represented a convenience sample of adults aged between 18 and 70 years, recruited through newspaper and radio advertisements and informal contacts.

Normal subjects from both studies were defined using ATS guidelines, and for this study, were required to self-identify as “New Zealand European”, and be never smokers with no diagnosis of respiratory disease, no recent respiratory symptoms, and no use of inhaled medication (Table 1). Both studies were approved by Wellington Ethics Committee, and written informed consent was obtained from each subject.

**Pulmonary function testing**—Pulmonary function tests undertaken as part of the WRS have been described in detail. In brief, tests were carried out by one of three trained operators (SA, SM, MVW) at one site using two Jaeger Master Screen Body volume constant plethysmography units with pneumotachograph and diffusion unit (Masterlab 4.5 and 4.6 Erich-Jaegar, Wurzberg, Germany). Prior to testing, equipment was calibrated daily in accordance with ERS and ATS guidelines.

Subjects were not tested within 3 weeks of an upper or lower respiratory tract infection (cough, sputum production, sore throat, or nasal congestion). Subjects were weighed (wearing indoor clothing without shoes) to the nearest kg and height was measured, without shoes, using a vertical backboard, to the nearest 0.5 cm. Subjects greater than 125 kg were excluded due to simultaneous involvement in a study involving CT scanning in which a weight limit was determined by the limits of scanner equipment.

Airway resistance, static lung volumes, and slow spirometric measurements were made prior to forced manoeuvres and gas transfer measurements followed these. A nose clip was worn for all tests.

Airway resistance was measured during relaxed breathing at a rate of approximately 0.5 Hz. Following attainment of a stable baseline, thoracic gas volume representing functional residual capacity (FRC)
was calculated using plethysmography. The subject was then instructed to breathe out comfortably, maximally inspire, and expire slowly to measure slow vital capacity (SVC). Expiratory reserve volume (ERV) was measured from FRC to the point of maximum expiration. Inspiratory capacity (IC) was calculated from SVC – ERV and residual volume (RV) was calculated from FRC – ERV. The total lung capacity (TLC) was calculated as SVC (measured) + RV. FRC and airway resistance measurements were used to calculate specific airway resistance (sRaw) and conductance (sGaw).

Spirometry measurements were carried out in accordance with ATS criteria. The largest FEV₁ and FVC from a minimum of three acceptable manoeuvres were used with all other flow volume parameters taken from the manoeuvre with the largest combination of FEV₁ and FVC. FEV₆ and FEV₁/FEV₆ measurements were obtained by analysis of stored data. Gas transfer was measured in accordance with ATS criteria, and a minimum of three measurements of diffusion capacity (DL_{CO}) were made with 5-minute intervals. The average DL_{CO} of tests meeting recommended reproducibility criteria were reported. Alveolar volume (VA) was simultaneously measured, using helium as a tracer gas, and used to calculate carbon monoxide diffusing capacity per unit of alveolar volume (DL_{CO}/VA). A blood sample was taken to adjust results for haemoglobin concentration using the formula recommended by the ATS. Results for pulmonary function tests except those measuring gas transfer were corrected to BTPS units.

Table 1. Selection criteria for the normal population

<table>
<thead>
<tr>
<th>Exclusion criteria</th>
<th>Number excluded</th>
<th>Number remaining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil</td>
<td>~</td>
<td>1025</td>
</tr>
<tr>
<td>Tobacco smokers (ever)</td>
<td>535</td>
<td>490</td>
</tr>
<tr>
<td>Pipe or cigar smokers (ever) or &gt;20 marijuana cigarettes (ever)</td>
<td>99</td>
<td>391</td>
</tr>
<tr>
<td>Doctor diagnosis of CB, E, A, COPD, or B†</td>
<td>110</td>
<td>281</td>
</tr>
<tr>
<td>Wheeze last 12 months</td>
<td>18</td>
<td>263</td>
</tr>
<tr>
<td>Sputum production 3 months*</td>
<td>11</td>
<td>252</td>
</tr>
<tr>
<td>Cough 3 months¶</td>
<td>16</td>
<td>236</td>
</tr>
<tr>
<td>Inhaler use last 12 months</td>
<td>10</td>
<td>226</td>
</tr>
<tr>
<td>Non-Caucasian#</td>
<td>14</td>
<td>212</td>
</tr>
</tbody>
</table>

†CB: Chronic bronchitis, E: Emphysema, A: Asthma, COPD: Chronic Obstructive Pulmonary Disease, B: Bronchiectasis; *Production of sputum on most days for as much as three months each year; ¶Cough on most days for as much as three months each year; # Did not self-identify as New Zealand European.

Statistical analysis—Reference equations were developed by linear regression. Sex was included as an explanatory variable in each model and for each lung function a further group of candidate explanatory variables was explored; height, height², age, age², weight, and body mass index (BMI)—plus interaction terms between sex and age and sex and height. The best regression equation for each parameter was developed with regard to the amount of variability explained, the number of explanatory variables, and consistency between parameters measuring similar physiological aspects of lung function. R-squared and Mallow’s Cp were used to examine different predictor equations. Mallow’s Cp adjusts R-squared by a penalty term that takes account of the number of predictors in an equation. Normality assumptions were tested by examination of the distribution of residuals from the regression equations, by histograms, QQ-plots, and formal tests of normality of the residuals. Agreement between the newly developed equations and the ECSC/ERS [known henceforth as European] equations was by plots of the difference between the actual value of a particular lung function minus its predicted value using each equation (difference plots). If height or age were important predictors of bias (actual value minus predicted value) then the estimates of it were calculated at the mean age or height. Bias between measured and predicted values was also examined for the static lung volume equations produced by Roca et al.
SAS (version 8.2) software was used for all statistical analyses.

**Results**

Initial recruitment from the WRS study resulted in 2319 responses from 3500 screening questionnaires. Of these subjects, 758 completed the detailed questionnaire and pulmonary function tests. The secondary recruitment resulted in 267 subjects completing the detailed questionnaire and pulmonary function testing thus giving a total population group of 1025 subjects.

Exclusion of subjects with previous smoking; diagnosis of a respiratory disorder; symptoms suggestive of COPD, asthma, or bronchiectasis; recent inhaler use; and those who did not self-identify as New Zealand European resulted in 212 subjects in the reference population (Table 1).

The final normal cohort consisted of 180 subjects from the WRS study and 32 from the secondary recruitment. Subject characteristics are shown in Table 2.

**Table 2. Subject characteristics**

<table>
<thead>
<tr>
<th>Male sex</th>
<th>Number (%)</th>
<th>110 (51.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Mean (SD)</td>
<td>Median (IQR)</td>
</tr>
<tr>
<td>25–34</td>
<td>53.2 (12.8)</td>
<td>54 (44–63)</td>
</tr>
<tr>
<td>35–44</td>
<td>1.69 (0.09)</td>
<td>1.70 (1.62–1.75)</td>
</tr>
<tr>
<td>45–54</td>
<td>74.5 (12.6)</td>
<td>74 (66–82)</td>
</tr>
<tr>
<td>55–64</td>
<td>26.1 (4.0)</td>
<td>25.5 (23.4–28.5)</td>
</tr>
<tr>
<td>65–75</td>
<td>Mean (SD)</td>
<td>Median (IQR)</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3 shows the lung function reference equations derived from the WRS data. Different intercept terms were created for males and females except for the equations for $FEV_1/FVC$, $FEV_1/FEV_6$, $FRC$, and $DLCO/VA$ in which no significant sex effect was noted. The values for $s_{Raw}$ and $s_{Gaw}$ are presented as single expected values for males and females since sex was the only important predictor in this model.

The $R^2$ values represent the percentage of explained variance in each measurement accounted for by the equation.
Table 3. Wellington Respiratory Survey (WRS) prediction equations for lung function

<table>
<thead>
<tr>
<th>Lung function</th>
<th>Predictor values</th>
<th>$R^2$ † (%)</th>
<th>RMSE‡</th>
<th>1.645 × RMSE‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intercept</td>
<td>Sex</td>
<td>Age (x)</td>
<td>Height (y)</td>
</tr>
<tr>
<td>FEV₁</td>
<td>-2.73</td>
<td>0.57</td>
<td>-0.031</td>
<td>4.47</td>
</tr>
<tr>
<td>Log PEF</td>
<td>5.43</td>
<td>0.30</td>
<td>-0.0053</td>
<td>0.056</td>
</tr>
<tr>
<td>MMEF</td>
<td>342.2</td>
<td>52.8</td>
<td>-3.50</td>
<td></td>
</tr>
<tr>
<td>FEF₂₅₋₅₀</td>
<td>530.4</td>
<td>124.9</td>
<td>-3.17</td>
<td></td>
</tr>
<tr>
<td>FEF₂₅₋₇₅</td>
<td>376.2</td>
<td>56.8</td>
<td>-3.40</td>
<td></td>
</tr>
<tr>
<td>FEF₇₅₋₁₀₀</td>
<td>46.9</td>
<td>12.0</td>
<td>-1.82</td>
<td>68.2</td>
</tr>
<tr>
<td>FEV₁</td>
<td>-5.32</td>
<td>0.66</td>
<td>-0.03</td>
<td>6.46</td>
</tr>
<tr>
<td>FVC</td>
<td>-5.87</td>
<td>0.65</td>
<td>-0.03</td>
<td>6.73</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>108.1</td>
<td></td>
<td>-0.24</td>
<td>-10.6</td>
</tr>
<tr>
<td>FEV₁/FEV₆</td>
<td>107.1</td>
<td></td>
<td>-0.18</td>
<td>-14.3</td>
</tr>
<tr>
<td>sGaw</td>
<td>1.30</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sRaw</td>
<td>0.83</td>
<td>-0.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SVC</td>
<td>-5.49</td>
<td>0.80</td>
<td>-0.025</td>
<td>6.44</td>
</tr>
<tr>
<td>IC</td>
<td>-1.37</td>
<td>0.54</td>
<td>-0.014</td>
<td>2.83</td>
</tr>
<tr>
<td>FRC</td>
<td>-7.02</td>
<td></td>
<td></td>
<td>6.30</td>
</tr>
<tr>
<td>TLC</td>
<td>-9.67</td>
<td>0.59</td>
<td></td>
<td>9.45</td>
</tr>
<tr>
<td>RV</td>
<td>-3.76</td>
<td>-0.16</td>
<td>0.024</td>
<td>2.75</td>
</tr>
<tr>
<td>ERV</td>
<td>-4.59</td>
<td>0.18</td>
<td>-0.012</td>
<td>3.95</td>
</tr>
<tr>
<td>RV/TLC</td>
<td>14.5</td>
<td></td>
<td>-5.86</td>
<td>0.39</td>
</tr>
<tr>
<td>DL₃₋₅₀</td>
<td>-4.91</td>
<td>1.14</td>
<td>-0.06</td>
<td>9.58</td>
</tr>
<tr>
<td>VA</td>
<td>-9.21</td>
<td>0.61</td>
<td></td>
<td>8.8</td>
</tr>
<tr>
<td>DL₃₋₅₀/VA</td>
<td>2.49</td>
<td></td>
<td>-0.009</td>
<td>-0.32</td>
</tr>
<tr>
<td>DL₃₋₅₀Adj</td>
<td>-4.59</td>
<td>1.09</td>
<td>-0.06</td>
<td>9.43</td>
</tr>
<tr>
<td>DL₃₋₅₀Adj/VA</td>
<td>2.63</td>
<td></td>
<td>-0.009</td>
<td>-0.04</td>
</tr>
</tbody>
</table>

Prediction equations take the form:

- Female: Predicted value = Intercept + (x × Age) + (y × Height) + (z × Weight).
- Male: Predicted value = Intercept + Sex + (x × Age) + (y × Height) + (z × Weight).

† $R^2$: Explained variance.
‡ RMSE: Root mean square error.
§ The equation for DL₃₋₅₀ is converted to conventional units of ml/min/mmHg by multiplying by 2.986.²⁴

Abbreviations: FEVₙ: Forced expiratory volume in n seconds (L); FVC: Forced vital capacity (L); PEF: Peak expiratory flow (L min⁻¹); MMEF: Maximal mid expiratory flow (L min⁻¹); FEFₙ: Forced expiratory flow rate when n% of FVC expired (L min⁻¹); sGaw: Airway conductance (s⁻¹·kPa¹); sRaw: Airway resistance (kPa·s); SVC: Slow vital capacity (L); IC: Inspiratory capacity (L); FRC: Functional residual capacity (L); TLC: Total lung capacity (L); RV: Residual volume (L); ERV: Expiratory reserve volume (L); DL₃₋₅₀: Gas transfer (mmol/min/kPa); VA: Alveolar volume (L).
Most lung function parameters met normality assumptions and did not require transformation. A natural logarithmic transformation appeared to give a better statistical model for PEF (which did not meet normality assumptions) suggesting that the relationship between age, sex, and height was multiplicative for this variable.

Results for maximal mid expiratory flow (MMEF or \( \text{FEF}_{25-75} \)), \( s_{Raw} \), and \( s_{Gaw} \) did not meet normality assumptions as well as other lung function measurements but were not improved with logarithmic or other transformations.

The majority of the equations use sex, height, and age as explanatory variables. Residual plots did not suggest that the addition of squared or higher power terms to model curvature improved the fit of any of the equations.

In univariate analyses of the individual variables, weight accounted for a moderate amount of explained variance (data not shown) but was probably acting as a substitute for body size, since, in the multivariate analysis, addition of weight added little to the predictive value of the equations. The only exception to this was for \( \text{FEV}_1/\text{FEV}_6 \) where weight had a weak effect in the multivariate analysis. Adding BMI was of marginal statistical significance and did not substantially improve the model fit for any equation. The addition of sex by age or height interaction terms was not helpful in developing better prediction equations.

A standard method of calculating normal limits for measured parameters in individual subjects is to add or subtract \( 1.645 \times \text{RMSE} \) from the predicted values and these values are given in the final columns of Table 3.

Comparisons of the WRS equations with the European equations for \( \text{FEV}_1 \), TLC, RV, and \( \text{DL}_{CO} \) are shown as difference plots in Figure 1. Table 4 shows a summary of mean bias (measured minus predicted values) by sex.

Predicted values for spirometry using the European equations were significantly lower than measured values particularly for \( \text{FEV}_1 \) (mean bias, male 0.48 L; female 0.36 L) and FVC (mean bias male and female 0.77 L). In many of the spirometry variables, the difference was greater than 12% of the mean lung measurements of the cohort (Table 4). For \( \text{FEV}_1 \), bias was weakly dependent on age decreasing by 0.05 L per decade.

For lung volumes large differences were seen between measured and predicted values using the European equations (Figure 1, Table 4). The greatest consistent difference, of around 15% of the mean predicted measurement, was seen in TLC with a mean bias (measured minus predicted value) of 1.14 L for males, 0.89 L for females. Bias was height dependent, being greater in taller subjects (increase in bias (L) per 10 cm increase in height: SVC 0.084, TLC 0.22, FRC 0.23, RV 0.12). The bias for RV was also weakly dependent on age (increased bias of 0.05 L per decade). Measured lung volumes were also compared to those predicted by the equations of Roca et al\(^8\) derived using measurements obtained with a plethysmographic technique (Table 5). The bias in these predicted values was significantly less than that seen with the European equations.\(^{10,11}\)
Figure 1. Plot of the differences between actual and predicted measurements using the European (ECSC/ERS) and WRS equations (Fig 1A = FEV$_1$; Fig 1B = TLC; Fig 1C = RV; Fig 1D = DLCO)

Filled circles represent males and open squares females and horizontal lines show the bias (difference between actual value and predicted values). Where age or height were predictors of the bias, this bias is at the mean age and/or height. By definition the mean bias for the equations of the WRS is zero).

Figure 1A. Difference plots for FEV$_1$: European versus WRS
Figure 1B. Difference plots for TLC: European versus WRS

![Graph showing the difference plots for TLC: European versus WRS.](image)
Figure 1C. Difference plots for RV: European versus WRS
Figure 1D. Difference plots for DLCO: European versus WRS

![Graph showing difference plots for DLCO: European versus WRS](image)
Table 4. Comparison between measured values of WRS study and predicted values using the equations of the ECSC/ERS

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimated bias in measured units</th>
<th>Bias as % of mean measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>FEV1 (L)</td>
<td>0.48</td>
<td>0.36</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>0.77</td>
<td>0.77</td>
</tr>
<tr>
<td>PEF (L/min)</td>
<td>110.5</td>
<td>60.8</td>
</tr>
<tr>
<td>FEV1/FVC (%)</td>
<td>-1.0</td>
<td>-1.0</td>
</tr>
<tr>
<td>MMEF (L/min)</td>
<td>27.7</td>
<td>27.7</td>
</tr>
<tr>
<td>FEF25 (L/min)</td>
<td>28.9</td>
<td>28.9</td>
</tr>
<tr>
<td>FEF50 (L/min)</td>
<td>29.9</td>
<td>29.9</td>
</tr>
<tr>
<td>FEF75 (L/min)</td>
<td>32.6</td>
<td>32.6</td>
</tr>
<tr>
<td>SVC (L)</td>
<td>0.66</td>
<td>0.66</td>
</tr>
<tr>
<td>FRC (L)</td>
<td>0.32</td>
<td>0.75</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>1.14</td>
<td>0.89</td>
</tr>
<tr>
<td>RV (L)</td>
<td>-0.13</td>
<td>0.27</td>
</tr>
<tr>
<td>RV/TLC (%)</td>
<td>-5.5</td>
<td>-1.9</td>
</tr>
<tr>
<td>DlCO (mmol/min/kPa)</td>
<td>0</td>
<td>-0.48</td>
</tr>
<tr>
<td>DLCO/VA 1983</td>
<td>-0.39</td>
<td>-0.56</td>
</tr>
<tr>
<td>DLCO/VA 1993</td>
<td>-0.14</td>
<td>-0.14</td>
</tr>
</tbody>
</table>

The columns show the estimated bias (measured minus predicted value) for each parameter when the equations of the ECSC are compared to measured values obtained in the WRS study. Results are shown as mean difference in measured units and as a percentage of the mean measurement for all male and female subjects in the cohort. A positive figure means that the measured value was greater than the predicted value (under-prediction) and a negative value that the measured value was less than the predicted value (over-prediction).

Abbreviations: The 1983 [10] and 1993 [11] publications of the formulae for DLCO/VA (also known as KCO) are different giving different values for the mean bias depending on which equation is used.

Table 5: Comparison between measured values of WRS study and predicted values using the equations of Roca et al[8]

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimated bias in measured units</th>
<th>Bias as % of mean measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>-0.19</td>
<td>0.25</td>
</tr>
<tr>
<td>RV (L)</td>
<td>-0.26</td>
<td>0.16</td>
</tr>
<tr>
<td>SVC (L)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>FRC (L)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>IC (L)</td>
<td>-0.27</td>
<td>0</td>
</tr>
<tr>
<td>RV/TLC (%)</td>
<td>-2.8</td>
<td>0</td>
</tr>
</tbody>
</table>

See Tables 3 and 4 for abbreviations and explanation of terminology

Measured results for gas transfer variables show close agreement with those predicted by European equations,[10,12] with the only exception being the results for DLCO/VA using the 1983 ECSC formulae.[10] The difference plots (Figure 1) show that the variability and spread of the differences between actual and predicted values are similar for both the European equations[10–12] and those of the WRS. This suggests that the precision of the equations, reflecting the

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variability of the sample with respect to predicted values, was similar whichever equation was used.

**Discussion**

This study provides reference equations for all commonly used measurements of static and dynamic lung function and new reference values for specific resistance and conductance from a single adult New Zealand population of European origin. In addition, we have produced reference equations for FEV\(_6\) and FEV\(_1\)/FEV\(_6\). To the best of our knowledge, this is the first time that reference equations have been derived for this complete set of respiratory function measurements on the same well characterised Caucasian reference population.

**Methodological issues**—A number of methodological issues relevant to the reporting of these results were identified. Whilst this sample size of 212 subjects was adequate for validation of pre-existing equations,\(^4\) it is smaller than that used to derive reference equations in several recently published studies.\(^8,26,27\) However other published reference equations dealing with a wide range of testing modalities were obtained using a similar sample size.\(^5\)

Subjects who did not self-identify as “New Zealand European” were excluded since a low response rate resulted in this group representing less than 7% of those completing testing despite approximately 20% of the local population being non-European in origin.\(^28\) The priority will now be to develop comparable reference equations for Maori and Pacific Island people.

The age range of the participants in this study also has implications for the clinical use of the reference equations, being restricted to adult subjects aged 25–75 years. Our equations incorporated sex and standard variables such as age and height. We found that use of polynomial expressions did not improve the fit of our equations.

Although other studies have produced equations incorporating such terms,\(^17,29,30\) we have taken a pragmatic approach under the simple assumptions of linear relationships, when they exist, in an attempt not to ‘over fit’ the data. This should make our equations more likely to be appropriate in other populations similar to our own.

Sex is a major determinant of lung function,\(^20\) although it is not typically included as an explanatory variable in reference equations. We decided *a priori* to include this variable since inclusion of a factor accounting for such significant variation in pulmonary function should improve the explained variance of resultant equations.

In addition, analysis of data separately by sex implicitly states that males and females have a different estimate of variation. Incorporating sex as a variable also results in smaller standard errors, since the sample size is that for both groups combined. Despite postulating that the inclusion of sex might require age/sex or height/sex interaction terms to be included, the addition of these added little to the fit of any individual model.

A further methodological issue relates to the technique used for measuring static lung volumes. Whilst the most recent ERS/ATS guidelines on measurement of lung volumes by body plethysmography\(^31\) recommend a ‘panting’ technique, this method was not available using our equipment at the start of the study (Masterlab 4.5 Erich-Jaeger, Wurzburg, Germany). However in a comparison of the two techniques, Roca
et al. found no significant difference in results for FRC obtained using either panting or tidal breathing methods.

**Spirometry and static lung volumes**—In keeping with a previous study incorporating sex into a single equation, our equations for FEV$_1$ and FVC had high values for $R^2$ with 80% of population variability being explained by the equation. This compares favourably with typical $R^2$ values for spirometric data of the order of 16–85%. Lower values for $R^2$ are often seen for static lung volumes—but using our equations, similarly high values of explained variance were seen for TLC and SVC. $R^2$ values higher than previously reported using plethysmography were also found for other static lung volumes.

At the time of their original publication, the reference equations for the ECSC represented a major effort in standardisation of pulmonary function testing. However, our findings that measured FEV$_1$ values were on average 0.36 L (female) and 0.48 L (male) higher than those predicted by the ECSC equations, with FVC values showing an even greater discrepancy, suggest that the spirometry equations from this source can no longer be considered suitable for contemporary use in New Zealand.

These results are consistent with previous European and Australian findings and are not unexpected since the ECSC equations were compiled from a series of studies conducted between 1954–1980. The under prediction seen with these equations is likely to be due to both anthropomorphic population changes as well as changes in and standardisation of lung function measurement.

Whilst the equations of NHANES have recently been shown to be suitable for use for spirometry in New Zealand these do not extend to cover the measurement of static lung volumes or gas transfer. Indeed, fewer reference equations are available for static lung volumes than for spirometry.

We found that the European equations, which were mostly derived from studies using gas dilution methods, under-predicted measured values for FRC and TLC in our cohort.

In contrast, a direct comparison between our equations and those of Roca et al. showed much smaller differences - these equations were also produced using a plethysmographic technique. As a result, it is likely that the differences between the European and WRS equations for static lung volumes can be attributed to both measurement techniques and historical anthropomorphic changes. This means that the European equations are probably no longer suitable for predicting static lung volumes in contemporary New Zealand subjects of European origin, particularly since in New Zealand such measurements are typically made using plethysmography.

**Newer spirometric variables**—The measurement of FEV$_6$ has recently been recognised as an important addition in the spirometric assessment of airflow obstruction and, with FEV$_1$/FEV$_6$, offers particular advantages in screening for and assessment of COPD.

Since these are ‘new’ parameters, fewer reference equations are available for their prediction. Our results provide further reference values applicable across a wide age range.
Gas transfer—Traditionally it has been recognised that measurement of $D_L^{CO}$ between different laboratories is subject to wide variation. The results for $D_L^{CO}$ from our cohort show good agreement with the predicted values derived using the European equations of the ECSC/ERS. The explained variance of 71% for our new equation for $D_L^{CO}$ was significantly higher than that reported in other studies possibly due to the incorporation of sex into the prediction equation.

Summary

We have presented a complete set of reference equations for a comprehensive range of lung function measurements suitable for use in an adult New Zealand population of European origin.

The development of comparable reference equations for Maori and Pacific Island people is a priority.

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References:


Defensive changes in medical practice and the complaints process: a qualitative study of New Zealand doctors

Wayne Cunningham, Susan Dovey

Abstract

Aim To characterise doctors’ responses to complaints.

Method Survey of a systematic sample of New Zealand doctors, and indepth interviews with 12 doctors who recently received complaints.

Results 714 written survey responses and 12 indepth interviews revealed changes consistent with positive and negative defensive medicine as well as changes in the direction of “good practice”. Positive defensive medicine changes were increased investigation and referral rates, active identification of potential problem patients, over-documentation and consenting, and altered approaches to time and workload. Negative defensive medicine changes involved withdrawal from the doctor-patient relationship and particular fields of practice. Good practice changes included reflective practice, greater sensitivity to societal and professional expectations, and initiating systemic change.

Conclusions The complaints process in New Zealand has the potential to improve healthcare delivery at a systemic level and to reinforce appropriate standards of professional behaviour, but it may cause individual doctors to practice defensively. Unless an appropriate educational process is allied to the complaints process, defensive medicine may compromise patient care and constrain potential improvements in healthcare delivery overall.

One of the ways society interacts with the medical profession is through complaints processes. Implicit in this interaction is an expectation that complaints will aid the recognition and remedy of substandard practice, ultimately improving healthcare delivery by individual doctors and healthcare institutions. This assumption is not well tested despite its wide acceptance.

In New Zealand, only about 4% of serious adverse events suffered by patients result in complaints to the Office of the Health and Disability Commissioner—a rate consistent with other studies of the incidence and patient responses to adverse events in hospitals. Using adverse events as an indicator of occurrence of appropriate complaints, Bismark et al suggest that complaints processes are underused for improving patient safety and the quality of healthcare.

Conversely, we have found that the annual rate of complaints experience among New Zealand doctors is high: in 2001, our research found that 5.7% of doctors received a complaint annually. In addition, the negative impact of complaints on the person of the doctor and on doctor-patient relationships may diminish the quality of care, counter to the intended outcome of complaints processes.
Regardless of whether the rate of complaints is high or low, the outcomes of complaints processes need to be better researched to justify the view that they promote healthcare quality and safety.

Defensive medicine has been defined as: “deviations from what the physician believes is … sound medical practice”\textsuperscript{7} and “medical practice decisions predicated on a desire to avoid malpractice liability, rather than a consideration of medical risk benefit analysis”\textsuperscript{8}. These definitions require that “sound medical practice” can be defined and that risk-benefit analyses can be undertaken.

If complaints prompt medical risk versus benefits analyses, better medical care may result. More likely, however, much defensive medicine is motivated not by concerns to benefit patients, but by concerns to avoid malpractice liability.

A product of complaints is an increase in defensive medical practice.\textsuperscript{7,9} Defensive medicine may be either positive or negative.\textsuperscript{10} Positive defensive medicine is expressed in increased use of resources, both to reduce the risk of receiving a complaint and to increase doctors’ ability to defend one. Negative defensive medicine relates to the withdrawal of medical services in situations of perceived increased litigation risk. Doctors may cease providing care where they believe that particular areas of work, kinds of patients, or diseases place them at greater risk of receiving a complaint.

Malpractice insurers refer to “defensible practice”\textsuperscript{11,12} when doctors stay within the limits of their expertise and keep up-to-date. Furthermore, administration is effective; there is good communication with patients, carers, and colleagues; and medical records include all salient facts relating to the patient. Indeed, defensible practice is good, humane practice.

This study aimed to identify types of change New Zealand doctors make in response to complaints or the perceived threat of complaints. We categorised changes according to whether they contribute to improved healthcare quality and safety. This study also creates a vantage point for critiquing the role of complaints in improving the quality and safety of healthcare in New Zealand.

**Methods**

We used a mixed method study employing postal surveys and telephone interviews, designed to provide both generalisable and indepth data about reactions to medical complaints. The details of the postal survey completed in June 2001 have previously been reported.\textsuperscript{4} In the study sample we included 13.8% of all 8715 medical practitioners registered in New Zealand in 2001. Each selected doctor was mailed an invitation to participate in the study, a consent form, and two sealed envelopes—one to be opened if the recipient had never received a complaint and the other to be used by those who had ever received a complaint. While we did not define “complaint” (thus leaving participants free to decide if their experience of a particular event should be called a complaint), we collected data on the agency (HDC, ACC, MPDC, or other) that processed the complaint—78.6% of complaint events were processed by HDC, MPDC, and ACC. A further 11.4% were dealt with “in-house”, leaving only 10% that were made to other significant bodies (such as the Coroner’s Court) or unaccounted for.

Survey forms collected demographic and practice characteristic data. The form in the never envelope asked how recipients’ day-to-day practice of medicine may have been changed by awareness of other colleagues’ complaints experiences.

The form in the ever envelope collected reasons for the complaint, time of the complaint, information about the agency processing the complaint, and sections about the complaint’s impact on themselves and their practice of medicine.
In free-text format, participants:

- Described changes in their performance of technical tasks after receiving a complaint;
- Identified changes in the range of services they offered patients and in their consulting; and
- Specified strategies they used to reduce the likelihood of receiving another complaint.

To determine whether doctors completing the survey were representative of the New Zealand doctor population, non-responders to the initial postal survey were re-surveyed with a questionnaire asking if they had ever or never received a complaint.

The quantitative analysis of doctor characteristics and complaints experience has been reported elsewhere. In this paper, we report the qualitative analysis of free-text responses to survey questions about changes in practice. Written responses were analysed using line-by-line inductive analysis to develop themes indicating the types of changes reported in each section.

Additionally, 12 indepth semi-structured interviews were conducted with hospital-based specialists who had received a complaint. A letter inviting study participation was forwarded by the Medical Protection Society to 40 hospital-based specialists for whom they had provided medicolegal advice in the previous 5 years.

Twenty-five doctors subsequently contacted the lead author (Wayne Cunningham) who conducted taped, semi-structured telephone interviews (each lasting approximately 1½ hours) with them. During the interviews, they were guided through their stories about their complaints experiences; and prompted to consider specific changes in practice they had made following (and because of) these experiences.

Interviews were taped, transcribed verbatim, and the analysis was commenced immediately so that each new interview was informed by the analysis of previous interviews. After 12 interviews, data saturation was reached—no new findings emerged so no further interviews were conducted. The qualitative analysis of interview transcripts used the same methods as the analysis of free text in survey responses.

The University of Otago Ethics Committee approved the study.

Results

Of the 598 initial responders and 373 responding to the re-survey, 330 (34.0%) indicated they had ever received a complaint. Of the 201 respondents who had ever received a complaint and completed the written questionnaire, 537 written comments were provided (259 [49.2%] from general practitioners, 161 [30.6%] from hospital-based specialists, and 117 [22.2%] from general registrants).

The 397 respondents who had never received a complaint provided a further 177 written comments (62 [35.0%] from general practitioners, 50 [28.3%] from hospital-based specialists, and 65 [36.7%] from general registrants).

Table 1 shows the characteristics of the 12 interviewed hospital-based doctors as well as the main features of complaints they were involved in.

Positive defensive medicine changes

Most positive defensive medicine changes were not situation-specific. That is, participants mostly did not relate changes in practice to a specific complaint but instead reported global changes made to either reduce the likelihood of a complaint or increase their ability to defend one.

Participants who had never received a complaint demonstrated awareness of the potential for receiving one and described changes they made to minimise that risk.
### Table 1. Characteristics of interviewed hospital-based doctors and their complaints

<table>
<thead>
<tr>
<th>ID</th>
<th>Medical specialty</th>
<th>Type of hospital</th>
<th>Characteristics of complaints</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Paediatrics</td>
<td>Peripheral</td>
<td>• Care of a baby who died from the effects of extreme prematurity.</td>
</tr>
</tbody>
</table>
| 2  | Sub-specialty surgery             | Base and peripheral | • A patient undergoing a plastic surgical procedure complained about her care.  
• A patient who had been prosecuted for insurance fraud sought damages from this doctor, who had testified in the fraud case. |
| 3  | Sub-specialty surgery             | Base             | • An operation was performed on the basis of an incorrect pathology report.  
• A patient who had an adverse outcome complained about management of care.  
• Accusation of an inappropriate internal examination of a female patient. |
| 4  | Sub-specialty surgery             | Base             | • Wrong-side surgery.                                                                                                                                       |
| 5  | Accident & Emergency              | Peripheral       | • Delayed diagnosis of infection after a surgical intervention.  
• A patient died following a medication overdose.  
*This interview raised new issues about the impact of police investigations in a small town setting.* |
| 6  | Paediatrics                       | Peripheral       | • The doctor’s assessment of a sexually abused child was refuted by the child’s family.  
• Complaint was about standard of care for a chronically ill child in a dysfunctional family, where the complained-about doctor had to continue providing care.  
• Care of a child with an intractable chronic condition in a situation where there was little opportunity for specialist support. |
| 7  | Sub-specialty surgery             | Base             | • A patient complained about postoperative scarring.  
• Consenting procedures were challenged.  
• Adverse outcomes following postoperative complications. |
| 8  | Sub-specialty surgery             | Base             | • A patient died of a well-recognised but unpredictable postoperative complication.                                                                   |
| 9  | Obstetrics & Gynaecology          | Base             | • Postoperative complications and inadequate consenting procedures for a procedure performed by a visiting expert (for whom this interviewee provided oversight on behalf of the Medical Council of New Zealand). |
| 10 | Sub-specialty surgery             | Base             | • Alleged inappropriate sexual behaviour.                                                                                                                |
| 11 | General surgery                   | Base             | • Fraudulent activity in a major hospital had (falsely) implicated this doctor in wrongdoing.                                                               |
| 12 | General surgery                   | Peripheral       | • Surgical complications and allegations of incompetence.                                                                                               |
Non-situation-specific positive defensive changes related to the following:

- **Investigations**—Increased rates of investigation were linked to perceived reduction in confidence, reduced ability to make decisions, and patient pressure. Doctors interpreted this form of positive defensive practice as disadvantageous to patients and the health system generally, but were aware of the utility of over-investigating as a response to societal pressure for certainty, and as a defence mechanism, should a complaint occur:

  I order more tests. I will often agree to tests or treatments if patients are demanding, although medically I feel these are not justified. (General Registrant 22)

  I think I actually expose kids to risk more. I’m less willing to say ‘in my clinical judgment I do not believe it is worth this investigation or that test’. In other words not only will I spend money, health dollars, on testing, but I will also put kids through painful and potentially risky procedures in order to satisfy parental concern. (Paediatrician, interview)

- **Referrals and admissions**—General practitioners and general registrants used this strategy. No hospital specialists indicated an increased rate of referral to colleagues for second opinions.

  I continue to admit people to hospital just in case I get sued…I refuse to sit on mild cases overnight. (General practitioner complaint 026)

- **Identification of problem patients**—Respondents indicated actively attempting to identify likely complainants, based on their sense (and that of their staff) of the quality of the doctor-patient relationship. Having identified such patients, they tried to minimise their responsibility for patient care by referral, or if this was not possible, by over-investigation, over-documentation, or over-consenting. Wariness extended to colleagues and reflected a reduction in trust.

  I now look at everybody that comes through my door as a potential complainant. You get to the point where you just know that you can’t trust anyone… (General surgeon, interview)

- **Documentation and consenting**—Two perceptions related to the consultation process were articulated: (1) Patients have difficulty retaining information and documentation is needed to prove that appropriate information had been passed to them; and (2) Consultation notes are the only evidence of doctor-patient relationships and the quality of medical practice, and that they may be later used in defending a complaint.

  Respondents recognised the uselessness of excessive note-keeping in some situations where, for instance, complaints were about a diagnosis they did not even suspect. Telephone consultations were particularly problematic and some doctors kept phone logs so that they could later refute claims they had not contacted patients.

  Hospital-based proceduralists were particularly concerned about obtaining informed consent and changed their consenting processes because of complaints. Several respondents mentioned doubly consenting patients at separate times and places. Changes in consenting and documentation were linked to efforts to identify and protect against likely complainants.

  If there’s anybody I feel suspicious of, I send them two copies of my consultation notes, and ask them to sign one to say that it’s a true and accurate record of the consultation and have it returned. (Subspecialty surgeon, interview)
• **Time and workload issues**—Some respondents extended their consultation times and tried to reduce their workloads to reduce their complaints risk. However, closer supervision of junior colleagues actually resulted in increased workloads for some working in hospitals. Some respondents viewed taking increased time as a way to enhance doctor-patient relationships, but were challenged to manage the demands of attending to multiple patients in limited time frames.

No situation-specific themes emerged from this analysis although there were many examples of changes in practice related to specific complaints:

Every patient now gets an X-ray (1 hour trip) prior to steroid injections to feet and tendons. (General registrant, complaint 346)

If I’m not happy I’m more likely to perform a caesarian section rather than trying for a vaginal birth. (General registrant, complaint 020)

**Negative defensive medicine changes**

Most negative defensive medicine changes were specific to each complaint’s circumstances. Respondents indicated stopping practice in fields such as obstetrics and intensive care, and shifting from either rural or urban practice. They reported withdrawal from working with patients with conditions such as drug addictions or needing therapies such as vasectomy or psychotherapy.

I stopped seeing children who had been sexually abused over that time, and it left an absolute sour taste in my mouth in relation to continuing to look after children with that issue. I haven’t assessed children with those issues since. (Paediatrician, interview)

The main non-specific negative defensive response was to withdraw from caring for patients who the doctor thought posed increased complaints risks:

My experience…seems to show that the very ones you walk the extra mile for are the ones who are more likely to serve notice on you when things don’t go well. To avoid that, you become more mechanistic, more stuck to protocol—you’re also less likely to establish a therapeutic relationship.” (Paediatrician, interview)

With increasing emphasis on team responsibilities, the potential for complaints to affect a wider group of health workers (especially in hospitals) was also observed.

**Changes in the direction of good practice**

Some responses to complaints reflected good practice. Indeed, complaints encouraged reflection on practice, closer professional adherence to societal expectations and detection, and remedy of systemic error.

Reflection focused on increased awareness of the doctor-patient relationship. This is consistent with good practice and patient-centred clinical care.\(^\text{14}\)

A change in my consultation practice to make sure that all patients were asked whether they were satisfied with the advice or treatment proposed. (General registrant, complaint 222)

Closer adherence to societal expectations was exemplified by increased use of chaperones, even in busy outpatient clinics where accessing female staff was problematic for the male doctors.
Two changes to detect and remedy systemic error were reported in detail during the indepth interviews: improved communication and relationship between pathologists and surgeons prompted by a complaint into surgery performed on the basis of incorrect pathology diagnosis; and initiating “time out” in operating room practice to reduce risk of wrong-side surgery.

The case enforced a closer relationship with one’s pathology colleagues…it’s made it a little bit more robust in terms of shared decision making. That I think has been a very good outcome. (Sub-specialist surgeon, interview)

Discussion

This study has characterised types of defensive medicine practiced by New Zealand doctors in what is regarded internationally as a litigation-free environment, in response to either receiving a complaint or being aware of the complaints experiences of colleagues.

This study found that complaints have the potential to change medical practice for better and worse. Positive changes included increased reflective practice, greater attention to meeting societal and professional expectations and procedural changes to remedy systemic error. However, we also found evidence of defensive practice (both positive and negative) that was not in patients’ best interests.

Doctors investigated, referred, and admitted patients to hospital when they did not expect any tangible benefit to patients or society. The complaints process in New Zealand motivates doctors to practice medicine in a way that will reduce risks of receiving complaints and/or increase their ability to defend one.

Our interpretation is that complaints focusing on individual doctors encourage defensive medicine, and this may reduce the overall quality of care because it devalues pre-test probabilities, exposes patients to harm, and misuses and depletes scarce healthcare resources. Conversely, complaints that focus on systems of care have the potential to identify and remedy sub-standard practice.

No respondents mentioned increased medical education to remedy deficient practice as a response to complaints. The best way to reduce the incidence of defensive practice may be to formally integrate an appropriate educational process into the experience of receiving a complaint. The responsibility for instituting such change lies with the medical profession but requires a coordinated and collaborative approach between the profession and complaints authorities. Furthermore, we fear that failure to explicitly recognise defensive practice may allow it to become normalised into everyday practice.

This study relies on respondents’ self-reported changes not validated by direct observation. However, the study’s sample size and methods allowed exploration of both breadth and depth of responses. This study confirms and extends the findings of Studdert et al\textsuperscript{10} by defining fields of practice change amongst New Zealand doctors.

In spite of New Zealand’s comprehensive “no fault” public injury insurance system, and having the Office of the Health and Disability Commissioner with statutory responsibility for resolving complaints, doctors have developed similar tactics to those found in countries with tort-based remedies for patient harm.
Our findings suggest that before advocating increased use of complaints to reduce preventable adverse outcomes of care, society must carefully differentiate between the outcome of complaints on individual doctors’ practices and systemic outcomes. If complaints processes are to alter medical practice in a way that enhances patient care, society must balance the deleterious effects of defensive medicine against the opportunity for systemic improvement.

Discussion between the medical profession and society is needed to balance outcomes of complaints and to determine how to use complaints to enhance healthcare delivery. Until this is done, systemic improvements may be publicly lauded while defensive practice is privately encouraged.

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References:


Alcohol and drug treatment population profile: a comparison of 1998 and 2004 data in New Zealand

Simon Adamson, Doug Sellman, Daryle Deering, Paul Robertson, Karen de Zwart

Abstract

Aims To describe the profile of clients attending dedicated alcohol and other drug (AOD) treatment services in New Zealand, and to compare this profile with data from a 1998 survey.

Methods 288 randomly selected AOD treatment workers in New Zealand were interviewed by telephone regarding their most recent assessment and follow-up clients, yielding a randomly selected sample of 383 clients. Workers were asked to identify the age, gender, ethnicity, main substance use problem, and geographical location of clients.

Results 65% of clients were male; 32% were Māori. The mean age was 34 years and the largest group of clients were seen for alcohol-related issues (47%), followed by cannabis (24%) and opioids (15%). Amphetamines had risen as the main substance used—from near zero (in 1998) to 10% of the sample (in 2004). In 2004, surveyed clients were older and less likely to be Caucasian (European ethnicity), and were more likely to be seen in district health board (DHB) and non-residential settings. Compared to clients attending assessment appointments, those attending for follow-up were older, less likely to be female or Māori, and more likely to use opioids. Overall, Māori clients were younger, used cannabis more, and were less likely to live in a large city. Amphetamine users were younger and almost exclusively living in the North Island.

Conclusions Several substantial changes have occurred in the profile of AOD clients over the 6 years (1998–2004). Although the traditional substances of alcohol, cannabis, and opioids continue to predominate, the rise in amphetamine use in the community is reflected in a corresponding rise in amphetamine presentations to AOD services, particularly amongst younger clients and those in the North Island. While other observed changes might have been predicted from broader demographic trends and service reconfiguration, the emergence of a substantial disparity in treatment follow-up rates between Māori and non-Māori is not so easily explained.

In 1998, the National Addiction Centre (NAC), then known as the National Centre for Treatment Development, or NCTD, conducted a national survey of the alcohol and other drug treatment workforce. This survey revealed for the first time a representative profile of AOD clients across New Zealand.¹

Since that time, patterns of substance of use have changed, with a marked increase in the use of amphetamine-type stimulants in the community,² and the reduction of the minimum legal alcohol purchase age with the 1999 Sale of Liquor Amendment Act. In addition, the health system has undergone substantial restructuring, and the AOD
workforce has evolved into a more qualified and more experienced group working more predominantly in district health board (DHB)-funded services. As part of the establishment of Matua Raki (the National Addiction Treatment Sector Workforce Development Programme), the NAC repeated the 1998 national survey, with data collection again including details of recent client contacts. This survey provides the opportunity to characterise the changing profile of the AOD treatment population, and to examine the distribution of different client groups across settings. Both sets of analyses can assist in guiding future workforce development initiatives.

Methods

Dedicated Alcohol and Drug Treatment Workers (ADTWs) were defined as paid staff (full time or part time) at least 70% of whose clinical time is spent working with people who have alcohol and drug problems. ADTWs were randomly selected from a list of approximately 800 ADTWs maintained and regularly updated by the NAC and supplemented by the membership register of the Drug and Alcohol Practitioners Association of Aotearoa New Zealand (DAPAANZ). Identified ADTWs were phoned by a research assistant who had their first name, initial of last name, and workplace details only.

The intention for the 2004 survey was to interview a total of 275 ADTWs. In total, 410 names were randomly selected from the full list, using random number assignment overseen by an independent research assistant, to be contacted for interview.

All ADTWs taking part in the survey were first asked a number of questions relating to the most recent client they had assessed in the 2 weeks preceding their interview, and then for the most recent client they had seen for a follow-up session during the same period. Workers were asked the age, gender, ethnicity, and main substance use problem of clients.

Information pertaining to the geographic location and nature of the treatment service was also gathered. Location was identified as either North Island or South Island and whether in a city (subsequently categorised as one of New Zealand’s five main cities, i.e. Auckland, Hamilton, Wellington, Christchurch, or Dunedin). Services were defined as DHB or non-DHB services, and residential or non-residential. Residential services were defined as primarily detoxification or post-detoxification.

Clients identified by ADTWs had been seen between 0 and 10 working days prior to the research interview. Clients attending at services with higher numbers of clients per ADTW have the potential to be under-represented. The time delay since client contact was recorded and used to create a weighting which corrected for this bias.

Comparisons with data from the 1998 survey results were also made. The methodology employed for both surveys was equivalent, except that the weightings described above have not previously been applied to this earlier dataset. The 1998 data have now been transformed to allow direct comparison with the current data. Given the large number of comparisons made only differences with a p value of less than 0.01 are reported as significant.

Results

Of the 410 names selected for contact, 85 proved to be ineligible (37 were not clinical staff or were unknown to the service, 36 had departed, one was deceased, and 11 were duplicate names). Of the remaining 325, a total of 288 were interviewed, a response rate of 89%, with 19 declining to participate and 18 not successfully recruited despite being confirmed as currently working clinically at the identified service—as a result of always being busy/unavailable at the times they were called. The response rate for the 1998 survey was 97%.

Of the 288 participants, 169 reported conducting an initial assessment in the 10 working days prior to the interview, and 231 conducted an individual follow-up session. For 17 interviews, the assessment and follow-up session were for the same client, and therefore there were a total of 383 unique clients. For these 17 seen for both assessment and follow-up the number of days since assessment was used to
calculate the weighting described above. The slightly longer average time lapse since assessment compared to follow-up session meant that once weightings were applied, the total of 383 clients was divided into 161 assessment clients and 221 follow-up clients (i.e. a total of 382 clients).

Table 1 shows the profile of the full 2004 client sample, and indicates several significant changes since 1998. The 2004 client sample is older and less Caucasian. The main substance of use is more likely to be amphetamines and less likely to be benzodiazepines. The setting in which clients are being seen is now more likely to be a DHB service and less likely to be a post-detoxification residential service. The increase in DHB status was significant for residential clients (26%, 50%, \(\chi^2=9.91, p=0.002\)), with no significant change for non-residential clients (74%, 77%, not significant [ns]).

Table 1. Characteristics of clients seen for treatment at listed dedicated alcohol and drug treatment services (seen in New Zealand in 1998 and 2004)

<table>
<thead>
<tr>
<th>Variable</th>
<th>1998 Total (n=291)</th>
<th>2004 Total (n=383)</th>
<th>Assessment (n=161)</th>
<th>Follow-up (n=221)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age: Mean (SD), in years</td>
<td>30.8 (10.1)</td>
<td>33.7 (11.1)**</td>
<td>31.9 (10.8)</td>
<td>35.1 (11.2)*</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>58.8%</td>
<td>64.5%</td>
<td>72.3%</td>
<td>58.8%*</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>69.6%</td>
<td>60.8%*</td>
<td>49.7%</td>
<td>68.9%**</td>
</tr>
<tr>
<td>New Zealand Māori</td>
<td>27.3%</td>
<td>32.4%</td>
<td>42.9%</td>
<td>24.8%**</td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>2.9%</td>
<td>5.3%</td>
<td>6.2%</td>
<td>5.0%</td>
</tr>
<tr>
<td>Asian</td>
<td>0%</td>
<td>0.5%</td>
<td>0.6%</td>
<td>0.5%</td>
</tr>
<tr>
<td>Other</td>
<td>0.3%</td>
<td>0.9%</td>
<td>0.6%</td>
<td>1.9%</td>
</tr>
<tr>
<td>Main substance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol Only</td>
<td>27.1%</td>
<td>27.1%</td>
<td>27.3%</td>
<td>27.3%</td>
</tr>
<tr>
<td>Mainly Alcohol</td>
<td>18.7%</td>
<td>20.2%</td>
<td>23.0%</td>
<td>18.2%</td>
</tr>
<tr>
<td>Alcohol &amp; Cannabis</td>
<td>10.9%</td>
<td>9.5%</td>
<td>13.0%</td>
<td>6.8%</td>
</tr>
<tr>
<td>Mainly Cannabis</td>
<td>15.7%</td>
<td>14.3%</td>
<td>16.8%</td>
<td>12.7%</td>
</tr>
<tr>
<td>Mainly Opioids</td>
<td>17.1%</td>
<td>14.8%</td>
<td>7.5%</td>
<td>20.0%**</td>
</tr>
<tr>
<td>Mainly Amphetamines</td>
<td>0.3%</td>
<td>9.7%**</td>
<td>8.1%</td>
<td>10.9%</td>
</tr>
<tr>
<td>Mainly Benzodiazepines</td>
<td>6.0%</td>
<td>2.0%*</td>
<td>1.9%</td>
<td>1.8%</td>
</tr>
<tr>
<td>Other</td>
<td>4.3%</td>
<td>2.3%</td>
<td>2.5%</td>
<td>2.3%</td>
</tr>
<tr>
<td>Setting</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>North Island</td>
<td>74.0%</td>
<td>72.0%</td>
<td>71.0%</td>
<td>72.9%</td>
</tr>
<tr>
<td>One of Five Main Cities</td>
<td>64.6%</td>
<td>61.5%</td>
<td>57.1%</td>
<td>64.7%</td>
</tr>
<tr>
<td>District Health Board</td>
<td>60.4%¹</td>
<td>71.8%*</td>
<td>67.9%</td>
<td>74.7%</td>
</tr>
<tr>
<td>Residential</td>
<td>28.0%</td>
<td>19.3%*</td>
<td>17.4%</td>
<td>20.7%</td>
</tr>
<tr>
<td>- Detoxification</td>
<td>4.8%</td>
<td>6.5%²</td>
<td>6.8%</td>
<td>6.3%</td>
</tr>
<tr>
<td>- Post-Detoxification</td>
<td>22.4%</td>
<td>13.4%*</td>
<td>10.6%</td>
<td>14.4%</td>
</tr>
<tr>
<td>Kaupapa Māori Service</td>
<td>-3</td>
<td>16.4%</td>
<td>19.9%</td>
<td>14.0%</td>
</tr>
</tbody>
</table>

¹Services were known as Crown Health Enterprises in 1998; ²Includes two clients seen at services described as both; ³Data not collected in 1998; ⁴Auckland, Hamilton, Wellington, Christchurch, Dunedin; *p<0.01, **p<0.001.

Significant differences between the profile of assessment and follow-up clients are also evident in Table 1. In 2004, compared to assessment clients, those attending for follow-up appointments were older, less predominantly male, less likely to be Māori.
and more likely to be Caucasian, and were more likely to be opioid users (main substance).

In contrast, in 1998 there were no significant differences between assessment and follow-up clients, including for gender (63% versus 57% male), and Māori (30% versus 25%), with age (29.3 years versus 31.9 years, t=2.17, p=0.031) and opioid use also not reaching the chosen level of significance (11% versus 22%, \( \chi^2 = 6.18 \), p=0.013).

The five variables (age, gender, Caucasian ethnicity, Māori ethnicity, and opioid use as main drug) significantly distinguishing assessment and follow-up clients were entered into a forward conditional binary logistic regression. The three variables retained in the model as predictors of follow-up rather than assessment status were Caucasian ethnicity (Wald=10.54, p=0.001), opioid use as main drug (Wald=7.52, p=0.008), and female gender (Wald = 5.96, p=0.015).

The regression analysis was repeated with the addition of an interaction variable of ‘gender x Caucasian’. In this equation, gender was removed but the interaction effect remained (Wald=7.40, p=0.007), in addition to Caucasian ethnicity (Wald=17.51, p=0.00003) and opioid use as main drug (Wald = 8.80, p=0.003). Further examination of this interaction effect revealed that although Caucasian follow-up clients were significantly less likely to be men (59% versus 75%; \( \chi^2 = 7.42, p=0.006 \)), there was no significant effect for non-Caucasians (64% versus 70%; \( \chi^2 = 0.74, ns \)).

The reduced proportion of Māori in the follow-up group could not be explained by any of the variables found to covary with Māori ethnicity (see Table 2), with Māori ethnicity remaining a predictor in regression analysis (Wald =7.57, p=0.006) when all significant covariates were controlled for.

Table 2 is an intercorrelation matrix of the variables displayed in Table 1, with ethnicity transformed into two dichotomous variables representing the two dominant ethnicities, and substance use transformed to four dichotomised variables representing the four dominant main drugs. This set of analyses reveals that Māori clients are (on average) younger, more likely to use cannabis, less likely to use opioids as their main drug, less likely to be seen at services in one of the five main cities, and more likely to attend Kaupapa Māori services.

The pattern for Caucasian clients is essentially the reverse of Māori, although no significant association was found for cannabis use. The strong association between ethnicity and Kaupapa Māori services is as expected, although it should be noted that Caucasian clients nevertheless constituted 23% of those identified as attending such services. The feature of greatest note for Māori clients was the high rate attending services outside the five main cities (59%) compared to non-Māori (29%).

Table 2 also reveals that those presenting primarily for their alcohol use tended to be older, while the cannabis group were younger and more likely to live outside the five main cities.

The amphetamine group was younger. Amphetamines were identified as the main substance for 13% of North Island clients but only 1% of South Island clients, while there was a non-significant trend for the amphetamine group to be larger in the five main cities than elsewhere (12% versus 5%; \( \chi^2 = 4.92, p=0.027 \)). Residential treatment
was predominantly provided in one of the five main cities. Non-DHB services were more likely to be residential or described as Kaupapa Māori.
Table 2. Associations between client and setting characteristics

<table>
<thead>
<tr>
<th></th>
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1Chi-squared values displayed except for age (t value); *p<0.01, **p<0.001; detox=detoxification; DHB=district health board; ns=not significant.
Discussion

The data from this national telephone survey of a large, representative sample of the AOD workforce show that the 2004 client profile is broadly similar to that found in 1998. Alcohol remains the primary substance of misuse, the mean age remains the early 30s, males predominate, and Māori are disproportionately represented. Services are primarily government-funded, outpatient, and urban-based. However, despite these similarities, several clear changes have occurred over the 6 years between the two surveys (1998–2004).

The increased age of the sample may in part reflect a broader demographic shift, with the New Zealand population as a whole aging. The reduction in Caucasian clients may also reflect a broader demographic shift for the adult population across the survey years, with non-significant increases observed for all other ethnicity categories.

The dramatic increase in amphetamine-type stimulants as the main substance of misuse is consistent with the rise in use of these substances amongst the wider community. Such an increase is very concerning given the known harms associated with such use in the areas of crime, mental health, and a range of high risk behaviours.

The clear contrast between rates of amphetamines as the main presenting substance between North Island and South Island services underlines the fact that (nationally) treatment services cannot be treated as a unitary group. Given that the group of amphetamine users identified here—people using amphetamines to a sufficient extent to warrant treatment for this as their main substance—represent the heavier using end of amphetamine users, there would appear to be good reason to primarily focus any attempts to specifically address this clinical population on North Island services at this point.

The setting in which clients were seen has also changed, with an increase in the proportion of DHB services and a decrease in post-detoxification residential treatment. The latter reflects the closure of a number of residential services since the release of the Mental Health Commission’s Mental Health Blueprint, which directed a shifting of funding from post-detoxification residential services to outpatient, methadone, and detoxification services. The reduction in non-DHB services was also a reflection of this same change, with all of the reduction in non-DHB clients occurring amongst residential clients.

The 2004 data were further analysed to explore differences between clients undergoing assessments, and those retained in follow-up treatment. This showed that clients attending follow-up were typically older, more likely to be female, non-Māori, and opioid users. The higher number of opioid users in the follow-up sample is easily interpreted as a reflection of the long-term nature of opioid substitution treatment, while age appears to have been a consequence of its association with these other factors, since it was no longer significant in a regression analysis.

Understanding the role of gender and ethnicity in the assessment-to-follow-up ratio is not so straightforward. Broadly speaking, a reduced rate of a given group in the follow-up sample may be due either to failure to retain these clients in treatment, or due to planned treatment of a shorter duration.
The largest difference between assessment and follow-up samples is for Māori /Caucasian. Justice system referrals would be likely to entail less follow-up where pre-sentence or pre-parole board hearing assessments were undertaken. Māori clients were likely to be proportionately over-represented in justice referrals, given the high proportion of Māori in the justice system compared to Caucasians, and this is also the case for males. Another possible explanation that must be considered is that AOD services are for some reason less able to retain Māori and male clients.

Failure to retain Māori clients in drug treatment programmes is supported as an explanation given the better ratio between assessment and follow-up figures for Kaupapa Māori services, which treat a predominantly Māori population, compared to the overall ratio for Māori clients. The reduced follow-up rate for Māori in 2004 is in contrast to 1998 when no significant difference was found.

Both surveys have also asked AOD workers whether they thought Māori clients should be treated differently to non-Māori clients. The proportion responding ‘yes’ reduced from 86% in 1998 to 77% in 2004 (unpublished data), a statistically significant reduction ($\chi^2 = 6.77, p=0.009$). This has occurred in the context of a recent backlash against perceived “special” treatment of Māori, at least within the political arena. To what extent this factor may have had an impact on treatment delivery and utilisation remains unclear however.

Failure to retain male clients may reflect similar issues to those which lead to a lower rate of treatment-seeking amongst males with substance use problems. Such assertions, in relation to both Māori and male treatment utilisation, are speculative however as the current data do not provide information as to the referral source, treatment needs, treatment plans, or actual reason for treatment termination of different client groups.

We believe that these findings, particularly those relating to treatment utilisation by Māori, warrant further investigation, but potentially they do suggest the need for improvements (at staff and service level) in treatment provision to these client groups.

The fact that data for this study were collected through interviews with workers rather than clients means that variables such as ethnicity and main substance used are not defined by the clients themselves. Data collected were also retrospective in nature. Despite these considerations, this sample can be viewed as broadly representative of clients seen within the dedicated alcohol and drug treatment field in New Zealand at this time. A major strength of the data reported here is that they are gathered from two surveys employing the same methodology and therefore we are able to identify trends that can be interpreted as reflecting real change in client profile or treatment service configuration.

A further survey, repeating the current methodology, is planned for 2007. It will be enhanced in several ways, including the collection of referral source information.

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Acknowledgements: This study was undertaken with the assistance of funding from the Ministry of Health; it also benefited from the excellent statistical advice of Associate Professor Chris Frampton (Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences).

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References:


Chris Wilkins, Paul Sweetsur, Sally Casswell

Abstract

Aim To track recent trends in the population prevalence, availability, price, and harm of amphetamine in New Zealand.

Design National household drug surveys were conducted in 1998, 2001, and 2003 using the same Computer Assisted Telephone Interview (CATI) methodology. General population random digit dial samples of 15–45 year olds were compared between the three survey waves (n=5475 in 1998; n=5504 in 2001; n=3042 in 2003).

Findings The proportion of the sample who had ever used amphetamine increased in 2001 compared to 1998 (11.0% versus 7.6%, p<0.0001) and then decreased in 2003 compared to 2001 (9.0% versus 11.0%, p=0.0066). The last year use of amphetamine increased in 2001 compared to 1998 (5.0% versus 2.9%, p<0.0001) and then did not significantly change in 2003 compared to 2001 (4.0% versus 5.0%, p=0.0466). The proportion of last year amphetamine users who said that the availability of amphetamine had become ‘harder’ compared to 12 months ago was higher in 2003 compared to 2001 (24.5% versus 12.4%, p=0.0284). Approximately 3 out of 10 amphetamine users reported harm to at least one area of their life from amphetamine use in 2001 and 2003. Harm to ‘energy and vitality’, ‘financial position’, and ‘health’ were the areas of life most commonly reported harmed from amphetamine use in both 2001 and 2003.

Conclusions Amphetamine remains a drug of serious concern in New Zealand. There is evidence of a levelling out in the prevalence of use and some evidence of a relative decline in availability. After 2001, wider public awareness of the health risks associated with methamphetamine use, and increasing law enforcement and legislative focus on methamphetamine, may have contributed to the stabilisation of the situation by 2003.

Since the late 1990s, New Zealand has experienced dramatic increases in detections of potent amphetamines, such as methamphetamine. The number of clandestine amphetamine laboratories detected each year by the New Zealand Police increased from just 2 in 1998, to 41 in 2001, and to 202 in 2003. Related to the increase in clandestine amphetamine manufacture, there were increased seizures of ephedrine tablets which are used to synthesis amphetamine, made by the New Zealand Customs Service from 10,308 tablets in 2000, to 32,653 tablets in 2001, to 830,320 tablets in 2003. Trends in drug seizures, however, can be influenced by the resources and attention dedicated to a drug type by law enforcement agencies, and consequently they are not necessarily an accurate measure of population trends in the use of a drug.
International studies have shown that chronic or high-dose use of amphetamines can cause hostility, violence, audio and visual hallucinations, and a paranoid psychosis resembling schizophrenia. In addition they can cause damage to cardiac, vascular, and neurological systems.\textsuperscript{5–10}

In New Zealand, the growing use of amphetamine has been linked to a range of public health and social problems including mental illness, drug dependence, intravenous drug use, family break-down, violence, and property crime.\textsuperscript{2,11,12,13}

The continued rise in detections of local amphetamine laboratories and related precursor chemicals since 2001 has led to considerable public anxiety about the eventual level of amphetamine use in New Zealand. For example, some sources quoted by the popular media have suggested that amphetamine use may continue to increase to a point where it replaced cannabis as New Zealand’s most widely used illicit drug.\textsuperscript{19}

In 2002, the government and law enforcement agencies initiated several legislative and strategic responses to the rise in amphetamine use, with a particular focus on domestic methamphetamine manufacture and use.\textsuperscript{3}

The aim of this paper is to track recent trends in the population prevalence, availability, price, and harm of amphetamine in New Zealand. The analysis is carried out using data from the three most recent waves of New Zealand national household drug surveying conducted in 1998, 2001, and 2003 respectively.

Method

National household drug surveying was conducted in New Zealand in 1998, 2001, and 2003. The 1998 and 2001 surveys were funded from contestable research funds and were known as the National Drug Survey (NDS). The 2003 wave of surveying was directly funded by the Ministry of Health and was renamed the Health Behaviours Survey-Drug Use (2003 HBS-Drug Use). The 2003 HBS-Drug Use retained the same Computer Assisted Telephone Interview (CATI) survey methodology and core sections of the questionnaire from the NDS. The age range of the 2003 HBS-Drug Use sample was extended from the age ranges surveyed by the NDS (i.e. 15–45 years in 1998 and 13–45 years in 2001) to include 13–65 year olds.

To allow valid comparisons back to the 1998 NDS, the age range of the 2003 HBS-Drug Use sample and 2001 NDS sample were truncated to those aged 15–45 years old. The 1998 NDS and 2003 HBS-Drug Use included extended samples of Maori to allow detailed comparisons of Maori and non-Maori.

To ensure valid comparisons between all three survey waves, only the general population random digit dial (RDD) samples from each survey wave were compared. Interviewing for the RDD sampling was conducted between April and September in each survey wave to replicate any seasonal variation in drug use.

All three waves of RDD sampling employed the same CATI sampling methodology. 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. The country was divided into a number of strata based on telephone exchanges to represent the different socioeconomic characteristics of the population. A proportionate sample from each stratum was then taken.

Within each household, one person was randomly selected for an interview. Each telephone number was tried at least 10 times on different dates and times of the day in an effort to reach those seldom at home. Respondents were informed that the study was being conducted on behalf of the Ministry of Health and that everything they said would be confidential.

In each survey wave, participants were asked the same questions concerning whether they had ever used amphetamine and whether they had used amphetamine in the last 12 months. The questions about amphetamine referred to the broad class of the amphetamines which the interviewer described to the respondent as ‘amphetamines, uppers, speed, methamphetamine’.
Additional questions on amphetamine were included in the 2001 and 2003 waves of surveying, including questions on whether the availability and price of amphetamine had changed compared to 12 months ago, and whether the use of amphetamine had harmed eight areas of a user’s life in the previous 12 months. The respective sample sizes for each survey wave were: 5475 in 1998; 5504 in 2001 and 3042 in 2003. The response rates for the survey waves were 79% in 1998, 80% in 2001, and 68% in 2003. (This is the response rate for the general sample of 2003 HBS-Drugs which was collected in the age range 13–65 years old. It was not possible to recalculate the response rate for the truncated age range as we could not distinguish the non-response by age.)

**Analysis**

All three samples were weighted by the number of people within the household who were eligible for each survey to adjust for the selection of only one person per household. Prevalence levels and confidence intervals were calculated using logistic regression, accounting for the effects of weighting and stratification.

Two sample t-tests were used, based on the logistic regression summary statistics, to test for differences between the samples. There were three t-tests per prevalence level; one for each two-way combination of the three samples. When comparing more than two groups, the probability of making a type 1 error increases if not adjusted for. Consequently when comparisons were made between the 3 years, values are only reported as significant when the p value <0.017. This value was calculating using the Sidak-Bonferroni method.

\[
1 - \left(1 - \alpha\right)^{\frac{1}{n}}
\]

where \(\alpha\) is the desired overall alpha level and \(n\) is the number of groups

In this case \(1 - \left(1 - 0.05\right)^{\frac{1}{3}} = 0.01695\)

Where comparisons are only made between two survey years, differences have been reported if the p value <0.05. The error bars on the graphs indicate the 95% confidence intervals. All analysis was run in the SAS and SUDAAN statistical software packages.

**Results**

**Prevalence of amphetamine use**—The proportion of the sample who had ever used amphetamine increased in 2001 compared to 1998 (11.0% versus 7.6%, \(p<0.0001\)) and then decreased in 2003 compared to 2001 (9.0% versus 11.0%, \(p=0.0066\)). By age group, there was an increase in those who had ever tried amphetamine in 2001 compared to 1998 among those aged 20–24 years old (from 16.2% versus 8.5%, \(p<0.0001\)).

The last-year use of amphetamine increased in 2001 compared to 1998 (5.0% versus 2.9%, \(p<0.0001\)) and then did not change in 2003 compared to 2001 (4.0% versus 5.0%, \(p=0.0466\)). The last-year use of amphetamine increased between 1998 and 2001 for those aged 15–19 years old (4.0% versus 7.5%, \(p=0.0078\)), 20–24 years old (5.8% versus 10.5%, \(p=0.004\)), and 35-45 years old (0.6% versus 1.5%, \(p=0.0084\)) (Figure 1).

**Change in the availability of amphetamine**—The proportion of those who had used amphetamine in the last year who said that the availability of amphetamine had become ‘harder’ compared to 12 months ago was higher in 2003 compared to 2001 (24.5% versus 12.4%, \(p=0.0284\)) (Figure 2). In 2003, as in 2001, nearly half of the last-year amphetamine users reported the availability of amphetamine was ‘easier’ compared to a year ago. In 2003, 3 out of 10 (29.0%) last-year amphetamine users reported the availability of amphetamine was the ‘same’ as a year ago.
Figure 1. Last year use of amphetamine by age, 1998, 2001, and 2003

![Graph showing the percentage of last-year amphetamine users by age group (15-19, 20-24, 25-29, 30-34, 35-45) for 1998, 2001, and 2003.]

Figure 2. Change in the availability of amphetamine compared to a year ago, 2001, and 2003

![Graph showing the percentage of last-year amphetamine users perceiving the availability as easier, harder, or the same in 2001 and 2003.]

Change in price of amphetamine—There was no change in the perceptions of the price of amphetamine by last-year amphetamine users in 2003 compared to 2001. In 2003, 6 out of 10 last-year amphetamine users (61.7%) described the price of amphetamine as the ‘same’ compared to a year ago; 1 in 4 (24.8%) last-year amphetamine users said the price was ‘lower’ and 1 in 8 (13.4%) said the price was ‘higher’ compared to a year ago.

Harms from amphetamine use—There was no difference in the proportion of last-year amphetamine users reporting harm in different areas of their life from amphetamine use in 2003 compared to 2001 (Table 1).
Table 1. Last-year amphetamine users who reported areas of their life harmed from amphetamine use in the last 12 months

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<td>Financial position</td>
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<tr>
<td>At least one area</td>
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In 2003, as in 2001, harm to ‘energy and vitality’, ‘financial position’, and ‘health’ were the areas of life most commonly reported harmed from amphetamine use. Three out of 10 of those who had used amphetamine in the last year had experienced harm to at least one of the areas of life asked about in both 2001 and 2003.

Discussion

The comparisons of the three waves of New Zealand national household drug surveying indicate a general rise in the initiation of (and last-year use of) amphetamine use in New Zealand in 2001 compared to 1998. This was followed by a levelling out in levels of last-year use of amphetamine in 2003 compared to 2001.

In 2003, more last-year amphetamine users reported that the availability of amphetamine had become more difficult compared to the preceding 12 months, than in 2001. However, in 2003, as in 2001, nearly half of the users reported that the availability of amphetamine had become ‘easier’ compared to a year ago.

Approximately 6 out of 10 last-year users in 2001 and 2003 reported the price of amphetamine was the ‘same’ compared to 12 months ago. Similar proportions of last-year amphetamine users reported harm in different areas of their life in 2001 and 2003 from their amphetamine use. In both survey waves, approximately 1 in 5 last-year users reported harm to ‘energy and vitality’ (and 1 in 10 reported harm to ‘health’) due to their amphetamine use in the last 12 months.

The validity of the comparisons between the waves of national household drug surveying relies on the exact replication of the survey methodology for each wave. This ensures that any differences found between the waves represent real changes in drug use and are not due to any change in the way the survey was conducted.

As detailed in the methodology section, all three RDD general population samples employed the same CATI survey methodology, asked the same questions about amphetamine use, and were collected during the same months of the year. The extended age ranges of the 2003 HBS-Drug Use and 2001 NDS were truncated to match the 15–45 year olds surveyed in the 1998 NDS.

While replication of the CATI survey methodology allows valid comparisons between the survey waves, we acknowledge that household drug surveys in general are likely
to underestimate the true extent of drug use to some extent. This is largely due to the difficulties of reaching heavier drug users who are more likely to be homeless, incarcerated, or be difficult to contact in general.\textsuperscript{31}

There were some differences between the survey waves in regard to the performance of the survey methodology. The response rate of the survey fell in 2003 compared to previous years. One factor in the decline in the response rate of the survey may have been the increase in private telephone market surveying in New Zealand over this time, which inadvertently competes with social science surveying for respondents’ time and patience. It might be speculated that drug users may be less likely to want to participate in the survey and consequently are more likely to be non-responders.

Increasing stigmatisation of methamphetamine as result of a number of high-profile violent crimes attributed to methamphetamine use and psychosis, and the reclassification of methamphetamine to Class A, may have also contributed to declining response rates and willingness to admit amphetamine use. The higher non-response in 2003 may have caused some underestimation of drug use in 2003 compared to other years. However, given the magnitude of the difference in non-response between 2003 and 2001 (i.e. 12\%), even assuming a much higher prevalence of amphetamine use among the non-responders compared to the survey sample, it would be unlikely to change the outcome of the statistical tests. The last-year use of amphetamine among the non-responders in 2003 would have to be over 15\% (compared to 4\% within the responders) to result in a statistically significant increase in amphetamine use in 2003 compared to 2001.

In each wave of the survey, the age, gender, and ethnicity of the survey sample was compared with respective Census population figures or (where appropriate) Census population estimates.\textsuperscript{14,15} In the 1998 and 2001 waves, the weighted survey sample had a slightly higher proportion of males than the Census population figures. About two-thirds of amphetamine users in each survey wave were male,\textsuperscript{12,16,17} so the lower proportion of males in the weighted sample in 2003 compared to previous waves may have caused a very small underestimation of amphetamine use. Again these differences were not considered large enough to make a difference to the results of the statistical tests completed.

Two types of environmental change may have played a part in the stabilisation of use (and the relative decline in levels of availability) of amphetamine in New Zealand in 2003 compared to 2001. The first is a growing awareness among the youth and drug-using population concerning the health risks and disutility of methamphetamine use over the medium and long term. Chronic heavy methamphetamine users are at high risk of experiencing serious psychological problems from their drug use, such as acute psychosis, extreme paranoia, aggression, and dependence.\textsuperscript{8,20}

A recent convenience sample of frequent methamphetamine users (n=78) interviewed from five centres in New Zealand found that 58\% reported ‘short temper’, 56\% ‘paranoia’, 51\% ‘anxiety’, 43\% ‘depression’, and 22\% ‘suicidal thoughts’ related to their methamphetamine use in the last 6 months.\textsuperscript{32}

In 2002, methamphetamine use was implicated in a series of rather bizarre and extremely violent crimes in New Zealand committed by individuals under the influence of methamphetamine or suffering methamphetamine-induced psychosis. These crimes included a multiple homicide, unprovoked stranger murder, and samurai
sword attack and murder. These criminal incidents and their link with the newly emerging methamphetamine received extensive coverage in the local media.  

Studies of amphetamine users in other countries have found that the negative mental health effects of amphetamine-use (such as aggression, paranoia, and depression), rather than physical harms from use, were the problems that caused the greatest concern among users and were most likely to cause them to seek help for their drug use.  

This sensitivity was thought to be related to the value young people placed on acceptance by their peer group and the effect that behavioural abnormalities could have on these relationships.  

The highly publicised mental health effects of methamphetamine use may have damaged the reputation of methamphetamine as a manageable risk among some drug users and young people in New Zealand after 2001. A drug’s reputation or image is considered to be central to fuelling a drug epidemic by encouraging the curious to start use, and reinforcing current users desire to continue and escalate their use.  

Accounts of amphetamine trends in Japan, Australia, and the United States have identified the relative rapidity with which new amphetamine users discover the negative mental health effects of amphetamine use, and the often shocking criminal incidents associated with amphetamine-induced psychosis, as factors which have tended to contribute to the relative short time span of amphetamine epidemics.  

Secondly, following the rise in amphetamine use in 2001 there was a concerted law enforcement and legislative response to this drug type in New Zealand, with a particular focus on the locally manufactured methamphetamine.  

New Zealand Police negotiated a series of protocols with the Pharmaceutical Guild to control and monitor the over-the-counter sale of ephedrine-based flu medicines from pharmacies, as these products were been used as sources of ephedrine to synthesis methamphetamine.  

Three specialised police teams were established in 2002 to detect and dismantle clandestine methamphetamine laboratories. With the emergence of large-scale domestic methamphetamine manufacture, New Zealand Customs paid greater attention to the importation of ephedrine, products containing ephedrine, and other chemical precursors used in methamphetamine manufacture.  

In early 2003, the New Zealand Parliament reclassified methamphetamine (from a Class B to a Class A drug offence) under the Misuse of Drugs Act 1975. A Class A drug offence is the highest offence class; it carries a maximum penalty of life imprisonment for trafficking and manufacture.  

Changes to the Misuse of Drugs Act 1975 were also enacted to increase the powers of the police and customs to search and seize unlicensed imports of ephedrine and other chemicals used to synthesis methamphetamine. A strong law enforcement and legislative response to a sudden rise in amphetamine use has been discussed as a factor which has assisted in the control of outbreaks of amphetamine use in other countries at other times.  

Amphetamine remains a drug of serious concern in New Zealand. As noted here, chronic heavy methamphetamine use is associated with aggression and serious psychological problems with implications for violence, crime, and neglect of responsibility.
One in 10 New Zealanders aged 20–24 years old had used amphetamine in the last year in 2003, and approximately 3 out 10 last-year users experience harm in at least one area of their life in the preceding 12 months. There is evidence of a levelling out in the prevalence of amphetamine use and some evidence of a relative decline in the availability of amphetamine in New Zealand. This may reflect greater awareness of the health risks of methamphetamine use and a greater law enforcement focus on methamphetamine in recent years. The levelling out of amphetamine use in 2003 may well be only a temporary phenomenon, however.

The impact of the shocking criminal incidents and stiffer regulatory controls related to amphetamine may fade over the coming years. A number of countries which have historically had problems with amphetamine use have experienced recurring amphetamine epidemics as new generations of young people rediscover the attractive features of amphetamine while having little sub-cultural memory of the hazards of use.23–25,28,30

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Data collection was managed by Chris Wilkins, Rachael Lane, Joe Morley, and Mary Blade. The 2001 New Zealand National Drug Survey was a project of the Alcohol & Public Health Research Unit (APHRU) at the University of Auckland, and was funded by the Health Research Council (HRC) and the Alcohol Advisory Council of New Zealand.

The funding to conduct the survey was awarded to Sally Casswell as an investigator-initiated research grant. The 2001 survey was led by Chris Wilkins with Rachael Lane, Mary Blade, and Heather Seal. The data management and weighting for the 2001 survey were carried out by Krishna Bhatta and Megan Pledger (assisted by Michael Ford and Alistair Stewart).

The 1998 New Zealand National Drug Survey was a project of the APHRU at the University of Auckland, and was funded as a core programme of the HRC and the Alcohol Advisory Council. The funding to conduct the survey was provided in part by the HRC as an investigator-initiated research grant to Sally Casswell, and in part by the Ministry of Health.

The 1998 survey was led by Adrian Field with Brendon Dacey and Francesa Holibar. The data management and weighting for the 1998 survey were carried out by Jia-fang Zhang, Michael Ford, and Krishna Bhatta (assisted by Allan Wyllie).

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Coma in an alcoholic: Marchiafava-Bignami disease

Lenneke Haas, David Tjan, Jos Van Die, Anton Vos, Arthur van Zanten

Coma can be a challenging diagnosis for the critical care doctor, especially in alcoholic patients.

We report a case of a 55-year-old male patient in whom the diagnosis of the coma was initially unclear and only discovered with magnetic resonance imaging (MRI).

Case report

A 55-year-old man with chronic alcohol abuse was found at home with altered consciousness and dysarthria.

Initially he was considered to be “just drunk again”. However, the emergency medical service was eventually called in after 24 hours because the patient did not wake up.

His medical history revealed a non-insulin dependent diabetes mellitus and hypertension. Alcohol abuse was known for 12 years. He used to drink several litres of beer a day. He was on the following medications: losartan 100 mg once daily (OD) and glimepiride 2 mg OD.

On admission, neurological examination showed a Glasgow Coma Scale (GCS) of E1M1V1, pupil reactions were symmetrical. Slight diverging strabismus was noticed. The oculo-cephalic reflex was normal. There was no lateralisation or pathological reflex present and no neck stiffness was found. Vital signs were as follows: temperature 37.4°C, blood pressure 120/80 mmHg, pulse 100 beats per minute, and oxygen saturation 97% on room air.

Further physical examination was unremarkable.

Laboratory results showed a Hb of 7.4 mmol/L (8.5–11.0 mmol/L), MCV 108 fL (80–100 fL), leucocytes 9.7/nL (4–11/nL), platelets 41/nL (150–400/nL), γ-glutamyltransferase 562 U/L (0–50 U/L), ASAT 113 U/L (0–45 U/L), ALAT 68 U/L (0–45 U/L), LD 942 U/L (0–450 U/L), ammonia 40 µmol/L (0–35 µmol/L), Ca 1.74 mmol/L (2.20–2.65 mmol/L), Mg 0.64 mmol/L (0.7–1.2 mmol/L), glucose 11.2 mmol/L (4–10 mmol/L). Serum thiamine concentration was 43 mmol/L (70–185 mmol/L) and folic acid level 15.2 nmol/l (5–55 nmol /L). The ethanol level was <0.1 promille. Toxicological screening proved to be negative.

A lumbar puncture yielded clear colourless cerebrospinal fluid that contained no red cells and six leucocytes per cubic millimetre (3–15/mm³). Glucose level was 6.0 mmol/L and total protein level 0.55 g/L (0.29–0.67 g/L). A stain smear showed no micro-organisms and cultures did not show any growth.

Computer tomography (CT) of the brain, which was performed immediately on the emergency department, showed no significant abnormalities. The patient was intubated, ventilated, and transferred to the ICU.
Electroencephalogram (EEG) revealed slow background activity with minimal irregularities and abundance of theta-activity occipito-temporal and no seizure activity suggesting a metabolic cause of coma.

MRI of the brain showed a high signal lesion in the corpus callosum and internal capsule in the T2-weighted sagittal (Figure 1) and axial view (Figure 2), as a sign of demyelinisation and oedema.

**Figure 1. T2-weighted sagittal image in Marchiafava-Bignami disease demonstrating a small, well-defined, and hyperdense lesion in the genu of the corpus callosum (arrowed)**
Figure 2. T2-weighted axial image in Marchiafava-Bignami disease showing a high signal lesion in the corpus callosum and internal capsule (arrowed)

Intravenous thiamine (100mg OD) was started and in 2 days the neurological condition showed gradual neurological improvement (E$_4$M$_4$V$_1$). After 72 hours, patient was successfully extubated and discharged from the ICU.

**Discussion**

Extrapontine myelinolysis in chronic alcoholism are typical findings of Marchiafava-Bignami disease (MBD). It is characterised by demyelinisation or necrosis of the corpus callosum and adjacent subcortical white matter. Necrosis of the corpus callosum is pathognomic for MBD.
MBD is a rare, severe, and usually fatal neurological disorder associated with chronic alcoholism. It is first described by Carducci in 1898 in Italian red wine drinkers and by Marchiafava and Bignami in 1903.\(^1\),\(^2\) It occurs predominantly in malnourished alcoholics and is reported more often in male than female drinkers.\(^3\),\(^4\) About 250 cases have been reported in the medical literature, but it is likely that its incidence is higher, since this diagnosis might easily be missed.

The underlying mechanism of the disease is still not understood. It is probably caused by the combination of alcohol abuse and malnutrition, leading to metabolic, toxic and vascular disturbances.\(^3\)

Brion observed that the disease occurred in patients who consumed at least 2 litres of red wine for more than 20 years.\(^5\) Cases of MBD in non-alcoholic, but malnourished patients have also been reported but are extremely rare, thus suggesting a causative relation with alcohol toxicity.\(^6\)

Patients with severe alcoholism who have this syndrome frequently have other problems such as alcoholic intoxication and hallucinosis, Wernicke encephalopathy, alcoholic liver disease, and sometimes subdural haemorrhage. Therefore, the diagnosis is often unclear.

Until recently, the definite diagnosis was confirmed at autopsy. However, in the era of modern imaging technology, diagnosis could be based on clinical profiles, history of alcoholism, and specific localisations of pathological lesions in the corpus callosum demonstrated by CT and MRI.\(^7\)

Findings on CT scan may confirm the diagnosis. However, if callosal damage is mild or the lesion is small, it may not be obvious and easily missed on CT, as in our patient. MRI is currently the most sensitive diagnostic tool. It also has the advantage of sagittal imaging. Lesions appear as hypodense areas in portions of the corpus callosum on CT and as discrete or confluent areas of decreased T1 signal and increased T2 signal on MRI.

In a review of acute and chronic cases, Heinrich observed that the worst case had the most significant MRI lesions suggesting a prognostic role for MRI. CT and MRI lesions seemed to regress in patients who improved.

There are no characteristic clinical presentations of MBD. However, involvement of the corpus callosum may lead to various clinical symptoms, such as: altered mentation, depression, mania, paranoia, or dementia. It may progress into seizures, hemiparesis, aphasia, ataxia, tremor, dysarthria, or dyskinesia and spasticity.

The course of the disease may be acute, subacute or chronic and may lead to death within weeks to months.\(^8\) Death usually results from cardiorespiratory failure or from the complications of alcohol abuse. Patients typically have severe neuropsychological deficits before they die. Some patients survive for many years in a demented condition or occasionally even show some recovery.\(^9\) An interhemispheric disconnection syndrome has been reported in survivors.\(^10\)

Because the aetiology of the disease is uncertain, a specific therapy is not available. Cessation of alcohol intake is mandatory and early supplementation of thiamine and folic acid might favourably improve the outcome. Seizures and coma are treated symptomatically.
Patients who survive should receive rehabilitation and, if appropriate, alcohol and nutritional counselling. A favourable response has been reported after the use of corticosteroids in some cases.

Conclusion

We presented a case of MBD in a comatose alcoholic.

Neurological examination and radiological imaging did not reveal an initial diagnosis, nor did laboratory and toxicological screening. Only MRI revealed the high signal lesion in the corpus callosum and internal capsule, which are typical for MBD.

Our patient improved after thiamine administration and supportive care.

Although rare, this case suggests that MBD should be considered in patients with chronic alcoholism and unexplained neurological deterioration, and MRI might be warranted in the absence of other causes for coma. The diagnosis might otherwise easily be missed if not considered.

In patients with chronic alcoholism and mental confusion, this uncommon diagnosis should be considered.

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Inferior vena cava thrombosis complicating tuberculosis

Mithun Raj, Aparna Agrawal

Thrombosis of the inferior vena cava (IVC) is one of the rarest manifestations of tuberculosis with only two cases reported in the literature. We describe the case of a 32-year-old male with disseminated tuberculosis and IVC thrombosis causing a diagnostic dilemma.

Case report

A 32-year-old male labourer presented with a history of fever, productive cough, and weight loss of 6 months duration and abdominal distension lasting 1 month. There was no past history of deep vein thrombosis (DVT), recent long journey, surgery, or prolonged immobilisation. Furthermore, there was no history suggestive of any connective tissue disorder.

Examination revealed a poorly built, malnourished male with stable vitals. He was febrile with a temperature of 101°F. Respiratory examination was notable for the presence of diminished breath sounds over the right lung base with stony dullness on percussion. There were coarse crepitations heard over the right infracavicular area. The abdomen was distended with shifting dullness and there were dilated tortuous veins visible over the lateral abdominal wall with cephalad flow (Figure 1).

Figure 1. Dilated, tortuous abdominal wall veins
Investigations revealed a haemoglobin level of 10 g/dL, total leukocyte count 18,000/mm$^3$, differential—polymorphs 40%, lymphocytes 60%, platelets 320,000/mm$^3$. Peripheral blood smear was normal. Renal and liver function tests were normal.

Chest X-ray showed massive right-sided pleural effusion with fluffy infiltrates over the right upper zone. Sputum examination revealed numerous acid fast bacilli (AFB) in three consecutive early morning samples.

Pleural fluid analysis showed an exudate with protein 4.6 g/dL and sugar 54 mg/dL with 80% lymphocytes on microscopy. Pleural fluid adenosine deaminase (ADA) was positive at 80 U/L.

Ultrasound of the abdomen showed mild hepatomegaly, paraaortic lymphadenopathy, and free fluid, which on testing was an exudate with predominance of lymphocytes and positive ADA. HIV serology was negative.

A diagnosis of disseminated tuberculosis was made and the patient was started on antitubercular treatment (ATT) with four drugs: isoniazid, rifampicin, pyrazinamide, and ethambutol. On ATT, he became afebrile with improvement in constitutional symptoms, dyspnoea, and cough but his abdominal distension progressively worsened over the next week.

In view of dilated veins over the abdomen and worsening abdominal symptoms, a Doppler ultrasonogram (Duplex Scan) was performed which showed IVC obstruction with thrombus in the intra- and retrohepatic portion of IVC extending to just above the renal vein.

A computed tomogram later confirmed the findings. His rheumatoid factor, serum C-reactive protein, anti-nuclear antibody (ANA), and anticardiolipin antibody tests were negative.

Serum homocysteine was within normal limits. The patient was started on unfractionated heparin (12,500 U – subcutaneous) twice a day along with warfarin 5 mg once daily, and the INR was adjusted to 2.4.

He improved on ATT with significant weight gain and regression of ascites. Right pleural effusion cleared completely and repeat chest films were normal. He was subsequently discharged with advice regarding continuation of ATT and long-term anticoagulation but was later lost to follow-up due to non-attendance.

**Discussion**

Tuberculosis is a disorder of protean manifestations and is considered to cause a hypercoagulable state. Severe pulmonary tuberculosis (PTB) is sometimes complicated by DVT and there are isolated reports of thrombosis occurring in unusual sites like portal veins and cerebral venous sinuses.$^{1,2}$

Hypercoagulability in tuberculosis can be attributed to several factors like decreased antithrombin 3 and protein C, elevated plasma fibrinogen levels, and increased platelet aggregation.$^{3,4}$ In addition, the systemic inflammatory state prevalent in tuberculosis causes endothelial cell damage predisposing to local thrombosis.
Thrombosis of the inferior vena cava is a rare manifestation of tuberculosis with only two cases reported in the literature. The diagnostic implications of IVC thrombosis in tuberculosis is well summarised in our case. The fact that the patient’s ascites worsened, despite ATT, made us search for alternative causes, and Doppler ultrasound subsequently proved IVC obstruction.

Additional tests for primary thrombophilic states could not be obtained in the case presented as the patient was commenced on anticoagulation prior to testing. The hypercoagulable state seen in tuberculosis has therapeutic implications as well. In a case of tuberculosis there is a strong case for prophylactic anticoagulation with heparin and avoiding central venous catheters. Anticoagulant therapy in tuberculosis is also problematic as the main antitubercular drugs (INH and rifampicin) are strong enzyme inducers and can interfere with warfarin levels.

In conclusion, DVT may be one of the atypical presentations of tuberculosis and the possibility of IVC obstruction should be considered in a tuberculous ascites resistant to ATT.

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Asian health in New Zealand—progress and challenges
Kumanan Rasanathan, Shanthi Ameratunga, Samson Tse

Abstract
The recent increase in the population classed as ‘Asian’ in New Zealand (now 9.5%) has seen the establishment of an ‘Asian health’ platform with activities by academic institutions, service providers, and community organisations. However, Asian health remains outside the frames of reference for most health professionals. Three recent reports provide the first large-scale systematic data about the health of Asian peoples in New Zealand. These reports identify the problem of ‘averaging’ if the whole Asian category is used.

Key health concerns include access to health services; cardiovascular disease, and diabetes for Indian peoples; levels of physical activity; and mental health, particularly in young people. Asian peoples born in New Zealand are less healthy than recent migrants classified as Asian. This ‘healthy immigrant effect’ abates with length of settlement in New Zealand. Despite these identified issues, there is a policy void for the health of Asian peoples in New Zealand, with no clear mandate to consider or monitor Asian peoples when undertaking research or formulating policy. Explicit engagement, policy, and service development for this significant and diverse part of the population should build on the agenda laid out by the recent advances in knowledge about Asian health in New Zealand.

Recently, the Ministry of Health released the Asian Health Chart Book 2006—
a comprehensive profile of the health of ‘Asian’ peoples in New Zealand, utilising data from Statistics New Zealand, the New Zealand Health Information Service, the New Zealand Health Survey, and the National Screening Unit. This report follows similar chart books for Māori and Pacific peoples and offers both implicit and explicit recognition of New Zealand’s growing Asian population and the importance of considering this population’s particular health needs.

The Ministry’s report is the culmination of almost a decade of work establishing an ‘Asian health’ platform and consciousness in the New Zealand health sector. Despite this progress, Asian health arguably occupies a marginal space in the health system, outside the frames of reference for the majority of professionals providing public and personal health services. This paper reviews the current status of Asian health, both in terms of the health of Asian New Zealanders and the responsiveness of the New Zealand health sector, and considers the challenges to further work in this field.

Who is ‘Asian’ in New Zealand?
Statistics New Zealand estimates that 9.5% of New Zealand’s current population is Asian, compared to only 3% in 1991. This proportion is expected to grow to almost 15% of the national population by 2020. In some areas, the projected increases are much higher. For example, it is estimated that 34% of the population served by the Auckland District Health Board will be Asian by 2016.

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1 Asian Health Chart Book 2006
2 Statistics New Zealand estimates that 9.5% of New Zealand’s current population is Asian, compared to only 3% in 1991. This proportion is expected to grow to almost 15% of the national population by 2020. In some areas, the projected increases are much higher. For example, it is estimated that 34% of the population served by the Auckland District Health Board will be Asian by 2016.
However, who gets defined as Asian in New Zealand raises complex issues. Statistics New Zealand and the state sector use a definition of Asian that is unique to New Zealand,\(^5\) differing from that used in many other western countries, especially the United Kingdom.\(^6\)

The Statistics New Zealand definition includes peoples as Asian if they have origins in the Asian continent from east and south of Afghanistan (inclusive). It does not consider peoples from the Middle East, such as Iranians and Iraqis, and Central Asia as Asian.

In contrast to the state sector definition, colloquial usage of the term Asian in New Zealand—as reflected in the media\(^5\)—often specifically describes Chinese and other East and Southeast Asian peoples. In this usage, Asian does not include Indian or other South Asian New Zealanders.\(^7\)

In this paper, the Statistics New Zealand definition of Asian is followed.

The Asian grouping in New Zealand is thus very heterogeneous as it includes half the peoples of the World. Beyond this diversity in ethnicity, the Asian grouping differs along many other axes, including settlement history, socioeconomic status, English language ability, and acculturation. For example, the grouping includes recent migrants together with Indian and Chinese New Zealanders whose ancestors arrived in New Zealand over a 100 years ago. The socioeconomic profile of the Asian grouping roughly mirrors that of the total New Zealand population, with an even distribution across the deciles of the New Zealand Deprivation Index.\(^8\)

The health of Asian New Zealanders and the development of an Asian health sector in New Zealand

Following the rapid increase of the Asian population in New Zealand during the 1990s, the first major reports reflecting health issues relating to these peoples began to appear.\(^9-11\) Small studies revealed concerns around access to health services, mental health, and settlement difficulties.\(^12-14\) However, in the absence of large-scale data, it was difficult to identify whether these concerns applied to the whole population or specific Asian sub-groups.

The Asian Public Health Project report\(^15\) published by the Ministry of Health in 2003 and the first Asian health conference\(^16\) held in Auckland in 2004 served as important steps forward, signalling the need for a more systematic appraisal of the health of Asian peoples in New Zealand.

Alongside these developments, services and organisations aimed at improving Asian health began to emerge in the context of healthcare institutions (such as the Asian Health Support Service at Waitemata District Health Board and the Asian Health Website hosted by Auckland Regional Public Health Service), academic institutions (including university research centres) and community groups.
During the past year, the first large-scale reports about the health of Asian New Zealanders appeared:

- *Asian Health in Aotearoa: an Analysis of the 2002–2003 New Zealand Health Survey* (The Asian Network Inc.);\(^{17}\)
- *A Health Profile of Young Asian New Zealanders who attend Secondary School: Findings from Youth2000* (The Youth2000 project at the University of Auckland);\(^{18}\) and
- *Asian Health Chart Book 2006* (Ministry of Health).\(^{1}\)

These three recent reports have recognised that Asian health is a useful banner for organising and facilitating health research and services for Asian peoples in New Zealand—whilst at the same time identifying that Asian is a problematic category for analysis due to the diversity of peoples collected under this grouping.\(^{5,19}\)

As such, all three reports have focused on smaller sub-groupings within the Asian category and have shown differences between these sub-groupings. Interestingly, however, the three reports differ in the manner in which they do this, although they all follow the principles of considering Indian and Chinese peoples separately and attempting to consider the effect of duration of residence in New Zealand.

The three reports identify significant concerns that require action. Key issues include access to health services, in particular for Chinese peoples; cardiovascular disease and diabetes for Indian peoples; levels of physical activity; and mental health, particularly in young people. The reports also indicate that whilst Asian peoples in New Zealand are relatively healthy overall, much of this effect is due to the high health status of recent migrants—the ‘healthy immigrant effect’.\(^{20}\)

Asian New Zealanders born in this country are in general less healthy than recent migrants across a range of indicators including cardiovascular disease mortality, cancer mortality, and prevalence of health promoting behaviours.\(^{1,18}\) This is not surprising given that most migrants to New Zealand need to be in good health to be allowed to emigrate and many have high socioeconomic status in their countries of origin. These migrant groups also have high levels of education which are correlated with better health status.\(^{21}\) This positive effect on health abates with increased length of settlement in New Zealand.\(^{1,18}\)

The high levels of cardiovascular disease and diabetes in Indian New Zealanders illuminate many of the challenges in considering Asian health in New Zealand. If the Asian grouping is considered as a whole, levels of diabetes and cardiovascular disease do not seem especially high. However, when Indian peoples are considered on their own, they show the highest rates of self-reported diabetes of any ethnic group in New Zealand,\(^{12}\) a finding supported by other surveys.\(^{22}\) They also show high levels of cardiovascular disease, similar to Māori.

The obscuring of this finding when considering the whole Asian grouping shows the clear potential for the diversity of the category to mask areas of need through ‘averaging’.\(^{5}\) In this case, the relatively low levels of diabetes and cardiovascular disease currently in Chinese New Zealanders averages out the high levels in Indian New Zealanders.\(^{19}\)
Despite high levels of disease, Indian New Zealanders rarely figure as a priority group in current diabetes strategies. For example, the otherwise excellent Let's Beat Diabetes Strategy by Counties Manukau District Health Board fails to mention Indian peoples, only considering Asian peoples in a relatively undefined way—despite a range of studies (some based in South Auckland) confirming Indian peoples’ high levels of diabetes with low levels of general practitioner consultations. In contrast, Indian peoples in New Zealand are identified as a high risk group for cardiovascular disease in New Zealand screening guidelines.

The pattern of low levels of healthcare service utilisation is seen across most areas for Asian peoples in New Zealand, particularly for Chinese New Zealanders. The Ministry of Health chart book shows particular concerns around primary healthcare and cancer screening, with no evidence that this gap is filled by traditional practitioners. In the Youth2000 study, 15% of young Chinese New Zealanders reported accessing no healthcare at all—over three times the rate reported by their European peers.

Factors outside the traditional boundaries of the health sector, but of important relevance to the health and wellbeing of Asian New Zealanders, are difficulties in finding employment and experiences of racism. The importance of these associations is not unique to this population but there are important nuances in the experience.

Asian peoples in New Zealand are more likely than non-Asian New Zealanders to have tertiary qualifications, but have higher levels of unemployment and lower incomes as a group. This is partly due to a lack of effective settlement strategies for migrant Asians to New Zealand as well as failure to appropriately utilise these migrants’ potential. Lack of (or under-) employment and difficulties settling into the host community are associated with negative health effects, particularly in terms of mental health—with Chinese migrants appearing to fare worse than other migrants to New Zealand.

Recent evidence shows that Asian New Zealanders are less likely to be interviewed for vacancies than other New Zealanders (despite similar qualifications and experience, and regardless of duration of residence in New Zealand) if they have non-European names. Indeed, other studies note that the experience of racism by Asian New Zealanders is common, with particularly high levels in the employment sector.

**Challenges in advancing Asian health in New Zealand**

At a time when several reports and an emerging research literature have identified specific health issues for Asian populations in New Zealand, the apparent policy void for Asian peoples in New Zealand is concerning. Furthermore, no clear mandate exists to consider or monitor Asian peoples when undertaking health research or formulating health policy.

Indeed, operational capacity and clear policies to address the health of Asian peoples are yet to be developed by the Ministry of Health. Other government agencies have made less progress in engaging the concerns of Asian peoples. For example, the recent study on New Zealand living standards did not report findings for Asian peoples.

Further research, including qualitative analyses, is also required to consider the contexts and drivers of the health needs and inequalities identified and provide targets.
for action. It is, however, important to consider whether there is a case for considering
the health of Asian peoples in New Zealand separately to the ‘mainstream’.

The New Zealand health sector has made significant progress over the past decade in
considering specific health issues for Māori and Pacific peoples, especially in
describing inequalities. This approach has been justified on several grounds including
the relatively poor health status of these groups, and (for Māori) their constitutional
status as tangata whenua (people of the land). Despite this progress, much remains to
be done and wide inequalities remain. However, there is now an explicit recognition
of these groups in policy together with the acknowledgement that the health sector
must improve services and access for Māori and Pacific peoples in order to reduce
inequalities. These are important milestones.

For Asian peoples, in terms of crude mortality, no such inequalities exist however. In
fact, the broad Asian groups appear to have similar or higher life expectancy to the
rest of the New Zealand population.\(^1\) If this is the case, is there then any need or
utility in attempting to cater for Asian populations beyond what is available in the
‘mainstream’ health sector? And is there any need for specific health policy and
services aimed at Asian peoples in New Zealand, given the apparent success of the
health sector in ensuring their health?

Based on the recent reports, the current good health status of Asian populations in
New Zealand would appear to have little relation to services provided by the health
sector. As discussed above, over a range of services, Asian populations show low
utilisation from primary care\(^1,17,18\) to cancer screening\(^1,17\) to Accident Compensation
Corporation services\(^32\) and the favourable health indices appear to primarily reflect
the high health status of the recent migrant constituencies of Asian populations.

As the ‘healthy immigrant effect’ wanes with increased duration of settlement,\(^33\) it is
predicted that as migrant communities acculturate, they will begin to resemble other
New Zealanders’ risk status for major chronic illness (as shown by New Zealand-born
Asian populations).

Asian populations also show particular risk factors for chronic illness, with low levels
of physical activity and insufficient fruit and vegetable consumption.\(^1\) Combined with
low levels of health services utilisation, the chronic disease burden in Asian
populations in New Zealand could thus increase dramatically. Such an increase may
be seen not just in Indian groups (whose elevated risk for cardiovascular disease and
diabetes has been demonstrated in many countries\(^34–36\)), but also in Chinese groups
who may mirror the increasing prevalence of obesity and chronic disease in China.\(^37–39\)

Improvements in health policy and service responsiveness for Asian populations in
New Zealand thus appear warranted by these risks which could result in increasing
morbidity and mortality, and furthermore, increased health expenditure, for a large
and growing proportion of the population.

It seems untenable for policy and services to continue to broadly ignore one-tenth of
the New Zealand population especially in the context of inequities in access to the
‘mainstream’ health system for large parts of this population. These inequalities are
particularly challenging issues for agencies serving areas with larger Asian
populations, such as district health boards in the Auckland region and some primary health organisations.

**Conclusion**

Asian New Zealanders now constitute a significant part of New Zealand society. The recent advances in knowledge about health issues faced by this diverse population provide an agenda for progress in building capacity and policy to address these concerns. This agenda must build on the existing work already achieved by a range of service providers, academic institutions, and community groups. However, for this sector to make progress, greater recognition of Asian populations by central government and large ‘mainstream’ organisations (such as district health boards) is needed.

The challenge posed by the *Asian Health Chart Book 2006* and other recent reports is whether there is sufficient will to recognise the health needs of Asian peoples in New Zealand. If so, explicit engagement, policy and service development are required to address these needs in this significant and diverse part of the New Zealand population.

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1906 in review—including bubonic plague in Sydney

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As another Annual Meeting of the New Zealand Branch of the B. M.A. will be in full swing about the time this number of the Journal will appear, the occasion seems a suitable one to attempt some slight sketch of the work and progress of the profession since the last meeting.

Undoubtedly the year has been a quiet one from a medical and surgical point of view, no epoch-making discovery has been made, no startling innovation in surgery; and even the science of bacteriology, to which we look for the constant publication of fresh marvels, has suffered something of an eclipse.

It is true, the theory of opsonins has made some stir; it has led a member to ask a question in the New Zealand Parliament and it has provoked that ‘prince of advertisers; Bernard Shaw, to honour the profession by attempting to hold the “licensed murderers” up to ridicule in one of his plays, but beyond this it does not seem to have produced any very startling results. Still the theory is most ingenious and may some day lead to important discoveries.

No serious epidemic has invaded our shores, the few cases of Scarlet Fever and Diphtheria that have manifested themselves have been for the most part mild, and we believe have nowhere assumed the nature of an epidemic. Influenza, has, as usual, caused much inconvenience and loss, but in most parts of New Zealand the cases have been of a less severe type than usual.

As we write, the bubonic plague is again serious in Sydney, but, while we must omit no precautions, we still feel but little fear of its ever getting a real hold in New Zealand.

We believe the members of the profession are working well and harmoniously together; probably at no time in our history has there been such strict and general observance of the laws of professional ethics; these laws being, not as the public are apt to suppose, something mysterious and unintelligible, but in reality only a code of honour which should invariably, in all works of life, govern the relations between one gentleman and another.

With regard to the Journal, we can point with pride to the fact that at any rate it has come out regularly: papers of interest have been forthcoming, but there is still cause for regret that so many of the leading men refrain from writing, and that of the vast stores of material which our Hospitals afford, so little ever reaches the hands of the Editor.

We believe it would add largely to the interest of the Journal, if men who have questions to ask or suggestions to make, would do so in the form of letters to the Editor: these will at all times receive attention and if possible, publication.
In Loving Memory: The Nurses’ Memorial Chapel, Christchurch, New Zealand

Anna Rogers

On the morning of 23 October 1915, in the cold, grey Aegean Sea, the British transport ship Marquette was 5 days into her journey from Alexandria to Salonika (now Thessaloniki) where the British and French had been fighting since September.

On board were New Zealanders of No. 1 Stationary Hospital—36 nurses led by Australian-born Matron Marie Cameron, 8 officers, 9 NCOs, and 77 orderlies. But the New Zealanders, under the command of Lieutenant-Colonel DJ McGavin, were a small minority on the large vessel. For the Marquette was not a hospital ship but a troop carrier, transporting the 500 men and officers of the ammunition column of the British 29th Division, plus 500 mules, some horses, and a large number of wagons loaded with ammunition. With the crew included, there were well over 741 people on board.

Photograph kindly provided by Kippenberger Military Archive, Army Museum Waiouru (http://www.armymuseum.co.nz)

A number of nurses were enjoying a brisk, warming walk on the deck just after 9 o’clock that morning when their world changed forever. On the upper deck, Jeannie Sinclair saw a green line flashing through the water and her friend Mary Grigor remarked, ‘I wonder if it’s a torpedo.’ It was: the Marquette had been attacked by a German U-boat.

A time of terrible confusion followed. Although the nurses did not panic and followed the drill they had practised, assembling quietly on deck, there were problems with lowering the lifeboats and many of the women were hurled into the sea. Then, as her
former passengers and crew struggled to stay afloat or lay dead and dying in the cold water, the Marquette sank, bow first: it took only minutes for what had seemed a mighty ship to disappear beneath the sea.

For the next 7 or 8 hours, before rescue vessels arrived, the men and women from the Marquette clung to rafts and wreckage, encouraging each other, but sometimes slipping away and drowning.

Ten New Zealand nurses lost their lives in the tragedy: Marion Brown, Isabel Clark, Catherine Fox, Mary Gorman, Nona Hildyard, Helena Isdell, Mabel Jamieson, Mary Rae, Lorna Rattray, and Margaret Rogers. The only bodies recovered were those of Rogers, who had been a district nurse with Nurse Maude in Christchurch before joining up, and Isdell, from the little West Coast town of Kumara. Also dead were 22 New Zealand men: 19 members of the NZMC, plus 3 infantry privates attached to the No. 1 Stationary Hospital. In all, 167 of those on the Marquette perished.

The idea for a memorial in the dead nurses’ honour came soon after the tragedy. On 13 December, Lieutenant Colonel Percival Fenwick, the Assistant Director of Medical Services for the Australian and New Zealand Training Depot in Zeitoun, wrote to the chairman of the Christchurch Hospital Board to ask if anything had yet been done ‘to commemorate the bravery of the New Zealand Nurses who were drowned on the transport “Marquette”…In Egypt we are all tremendously proud of the splendid way in which these Nurses died. I feel that it would be only right to perpetuate their memory in our Hospital.’

Fenwick’s suggestion was a brass plaque placed in the hospital hall and ‘a bed in the Women’s Ward…named after each Nurse who came from our hospital’: 3 of the 10 who died—Nona Hildyard, Lorna Rattray, and Margaret Rogers—had belonged to the staff there. He was sure the Medical Corps at the front, and ‘those doing their duty in New Zealand’, would be more than ready to contribute.2

Back in 1914, Christchurch Hospital Matron Mabel Thurston had suggested the need for a chapel in the hospital grounds where nurses and patients could attend services; after the Marquette sinking, she felt such a building could become a memorial to the women who had died. On 4 April 1916, 4 days before she left to take over as matron of the New Zealand military hospital at Walton-on-Thames outside London, she reminded the board of her support for the idea, stressing that such a building was also the hope of all her nurses.3

The January 1916 issue of the nurses’ magazine Kai Tiaki had reported that a decision to ‘erect a chapel at the Christchurch Hospital’ in memory of the nurses lost in the Marquette sinking. A collection had begun on 9 November 1915 at a memorial service held in Christchurch’s St Michael and All Angels at which the bishop, Churchill Julius, spoke of the nurses, ‘who had gone out on active service at the front, with their lives suddenly cut short. They stood as an example to all, as to what our lives should be.’4 More than 200 nurses in uniform were among the many who attended.

Acting Christchurch Hospital matron Rose Muir wrote to the hospital board during 1917, stressing again the urgent need for a chapel. Staff had given more money and donations had been collected, but until the board granted permission to build, there
could be no canvassing for public subscriptions or asking for an estimate of the chapel’s cost. The board agreed to bear the cost of the foundations and basement and in September a meeting was held to choose a site for the chapel.

Christchurch district nursing pioneer Sybilla Maude was also behind the chapel idea—and not just for her city. As she wrote to *Kai Tiaki* on 27 September 1916, ‘What more fitting memorial can be suggested than a chapel attached at each hospital, which could be at the disposal of all denominations, and when peace is declared, we could combine our thank offerings with our memorial.’ She saw ‘a great need’ for hospital chapels, which were a feature of all big English nurse training schools.

‘A nurse’s life is a trying and busy one, and to be able to spend a short time for prayer or silence in a place set apart for that purpose would be very helpful. There are many friends who would be glad to help us in raising funds, and no difficulty should stand in the way of building a lasting memorial to those who have freely given their lives for our country.’

But progress continued to be slow. The Nurses’ Memorial Chapel Committee was not formed until August 1925. The public responded so generously, however, that there was money left over to fund furnishings for the chapel.

The beautiful little building was designed free of charge by architect John Goddard Collins and built by William Williamson. Made of reinforced concrete, Oamaru stone, and terracotta bricks, it has a slate roof. Timber is a feature of the fine, beautifully decorated interior. The arched roof beams and wall panelling are oregon, the window and door frames are matai, the sarking is redwood, and the parquet floor is blackwood and oak. Two local men, Frederick Gurnsey, who taught at the Canterbury College School of Art, and Jake Vivian, carved the elaborate oak reredos and altar rails. Much later, a handsome new porch was added, incorporating bricks and slate from adjacent hospital buildings pulled down in 1991.

Opened on 15 March 1927, the chapel was handed over to the North Canterbury Hospital Board the following year. The foundation stone bears the name of the then Duchess of York, later Her Majesty the Queen Mother, but because she was ill it was actually laid by her husband, the future George VI.

Although there are other memorials to the Marquette nurses, this is the only dedicated chapel of its kind in New Zealand and possibly in the world. As well as
commemorating the three Christchurch *Marquette* nurses, the chapel remembers nurses Grace Beswick and Hilda Hooker, who died after working in Christchurch Hospital during the 1918 influenza epidemic, and many other distinguished medical men and women, such as Nurse Maude, and Canterbury surgeon and *Marquette* survivor Sir Hugh Acland.

The interdenominational chapel rapidly became an integral part of the hospital’s existence, especially for trainee nurses, who were required to take patients to and from Sunday services, sometimes in beds or wheelchairs, before having the rest of the day off—their only free time during the week. Relatives of the sick were grateful for the solace the chapel offered.

The building’s life has been threatened twice. In the mid-1970s there was talk of demolition when the Hospital Board wished to erect temporary operating theatres on the site; the proposal was withdrawn. Some years later, in the 1980s, the Board decided to pull down the building and make its interior part of a chapel to be included in the new hospital block. There was strong community opposition to this plan, particularly from the newly formed Friends of the Chapel, and in August 1989 the chapel was given an official reprieve when a protection notice was issued.

The Canterbury Area Health Board agreed to lease the chapel and its land to the Christchurch City Council. The site is now classified as a historic reserve under the Reserves Act and the chapel, subleased to the Nurses’ Memorial Chapel Trust Board, is secure under the protection of an Historic Places Trust heritage order: it has a B classification. The Trust Board administers the building and ensures its preservation, and the Friends of the Nurses’ Chapel care for it lovingly.

The chapel boasts 11 lovely stained windows, 4 of which are by the noted English glass artist Veronica Whall. A more recent and very arresting window, by Stephen Belanger-Taylor, shows nurses in First and Second World War uniform, and features the *Marquette* and a number of nursing medals. The colourful aisle carpet, entitled ‘The Tree of Life’, was created by Dunedin artist Nicola Jackson.

The chapel’s little museum in the right-hand vestry contains fascinating nursing memorabilia and historical photographs; the families of nurses associated with the chapel often donate cherished items. A 9-minute video tells the story of the chapel. Each rose in the peaceful garden that surrounds the chapel has been given in memory of a friend or relative associated with the building.

Now used regularly for weddings and christenings, and such events as concerts, poetry readings, and floral displays, the chapel is open to the public every afternoon except Christmas and New Year’s Day. It is also still available to the patients, staff, and visitors of Christchurch Hospital.
This beautiful and much-loved building is an integral and unique part of Christchurch’s and New Zealand’s medical history.

NZMJ Note: To commemorate the 90th anniversary of the Marquette sinking, a special service was held at the chapel on 26 October 2005. The Governor General, Dame Silvia Cartwright, representatives of the New Zealand Defence Force, and relatives of those who died or survived the tragedy attended. For more information on the Marquette sinking and the chapel, see http://www.rootsweb.com/~nzlscant/marquette.htm

Author information: Anna Rogers, Author of While You're Away: New Zealand Nurses at War 1899–1948 (Auckland University Press; 2003), Christchurch

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2. Letter – Lieutenant Colonel Percival Fenwick to Christchurch Hospital Board, CH701 9/30, Archives New Zealand.

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Deep vein thrombosis due to absence of inferior vena cava
Charalabos Stratopoulos, Constantinos Pitsios, Panayota Valenti, George Anagnostopoulos, Epaminondas Kotronis

A 21-year-old male was admitted to hospital complaining of a 1-week history of pain and swelling in his left leg. He had previously been in good health. Tests for thrombophilia (including antithrombine III, homocysteine, protein C and S, and antiphospholipid antibodies) were normal.

Abdominal magnetic resonance imaging (MRI) revealed a total congenital absence of the inferior vena cava (IVC). Azygous and especially hemiazygos veins were greatly dilated (Figure 1). The hepatic veins formed a common trunk, which drained directly to the right atrium (Figure 2).

Discussion
Absence of the IVC is a rare congenital abnormality that may cause no symptoms and may not be diagnosed before adolescence. It consists of an absent suprarenal segment of the IVC but it is also designated as an absent or atretic intrahepatic portion of the IVC with drainage via azygos and hemiazygos collaterals into the superior vena cava.¹

Anomalies of the IVC may coexist with other cardiac and visceral anomalies such as congenital defect of pericardium, atrophic kidney, biliary atresia, the polysplenia.
syndrome, situs inversus, anomalous pulmonary venous drainage, dysgenesis of the lung, or a combination of these abnormalities.²

Absence of the IVC may exist without producing any symptoms—although haemoptysis, venous hypertension, and leg oedema with pretibial ulceration have been attributed to absent IVC accompanied by an abnormal venous drainage.

In summary, in patients with deep vein thrombosis not associated with classic predisposing factors, the diagnostic work up should include a duplex sonography, an abdominal MRI, or CT scan to exclude developmental abnormalities of the IVC.

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Radioactive patients and airport security

A case report from England discusses the experience of a thyrotoxic patient treated with radioiodine (400 MBq of $^{131}\text{I}$). Six weeks later he went to the United States for a holiday. At Orlando Airport he set off the security alarm at check-in. He was immediately detained and strip-searched. Sniffer dogs were also used. A prolonged period of interrogation ensued. Luckily, he was carrying his radionuclide card with him. He was finally released after a long delay and much embarrassment.

Obviously the radioactive threshold at security was lower in the US. In discussion and in an editorial this annoying matter is reviewed. Apparently in the UK those treated with radiiodine are advised to avoid public transport for two weeks so that they do not expose nearby passengers to radioactivity, but, to date, advice regarding air travel and radiation detectors have been overlooked in many centres.

We wonder what happens with such patients in New Zealand?

BMJ 2006;333:293–4 & 271–2

HIV/AIDS update

In retrospect, HIV/AIDS was first seen in 1981—a report on a mysterious outbreak of Kaposi’s sarcoma and *Pneumocystis pneumonia* among homosexual men. And since then, AIDS has grown to pandemic proportions resulting in 25 million deaths and 40 million persons living with human immunodeficiency virus (HIV) worldwide by the end of 2005.

On the 25th anniversary of this grim event, JAMA has devoted a whole issue to the subject. Is there any good news? Three notable points noted by your scribe. Firstly, there many be an effective vaginal microbicide on the way. Secondly, routine circumcision of all men in Africa could prevent 2 million new HIV infections and avert 300,000 deaths over the next 10 years, according to an analysis published by an international team of researchers in July. A good, but unrealistic, idea.

And then the question—would 4 antiretroviral drugs be better than 3 in new cases? No. In a recent trial there were no significant differences between the 3-drug and 4-drug antiretroviral regimens, adding abacavir as a fourth drug provided no additional benefit.

JAMA 2006;296:753–55, 769–81 & 827–43
Clopidogrel and generic competition

Those who treat patients with coronary or cerebrovascular disease will be pleased that PHARMAC now allows appropriate prescribing of clopidogrel (brand: Plavix) for such patients—one 75 mg tablet daily at a cost of $6/day.

Methuselah notes with interest that Plavix is the world’s second-best-selling drug—48 million Americans use it on a daily basis—and with sales of more than US$6 billion last year, it accounts for about 30% of Bristol-Myer’s total earnings.

Recently, August in fact, the generic manufacturer, Apotex, caused a stir by selling generic clopidogrel at US$4/tablet. And, would you believe it, in an effort to keep the generic version off the market, Bristol-Myers and Sanofi had agreed to pay its Canadian manufacturer, Apotex, $40 million not to release it until 2011. This dubious deal fell through and subsequently a court injunction ruled against Apotex, but not before Apotex had done a lot of selling.

One wonders whether it is just a coincidence that the PHARMAC price of NZ$6 per tablet is approximately the same as the Apotex generic (US$4)—both 20% lower than the Plavix cost in the US.


Accuracy and cost-effectiveness of B-type natriuretic peptide tests in the management of heart failure

B-type (brain) natriuretic peptide (BNP) is a neurohormone that is secreted in response to volume expansion and pressure overload of cardiac ventricles. Two tests measuring BNP in plasma have been developed, an enzyme-linked immunosorbent assay (ELISA) and a radioimmunosorbent assay (RIA). The ELISA test is a bedside test and would be particularly suitable to assist rapid diagnosis on site in primary and emergency care settings. However, it is 5 times more expensive than the RIA test. In the first paper in this journal, an overview of 19 studies involving over 9000 patients concludes that ELISA and RIA are both accurate but the advantages of rapid ELISA test need to be balanced against the higher cost.

The other paper reports on a prospective randomised study that demonstrates BNP testing is cost-effective in the management of dyspnoea in the emergency department. Why—less hospital admissions, less intensive care, and shortened hospital stay for those in the BNP group.

Hyponatraemia

The American Journal of Medicine has devoted a 100-page supplement to this very important topic. It is the most common electrolyte abnormality encountered in clinical practice and is reported to occur in up to 30% of hospitalised patients with acute or chronic conditions.

The 12 papers in this supplement range from physiology through a variety of aspects including advances in management. So if you want to be up with the play, this supplement is for you. Your scribe was particularly interested in three points.

Firstly, is asymptomatic hyponatraemia really asymptomatic? Apparently, not always and Guy Decaux elaborates and believes it to be a cause of falls in the elderly. Secondly, the development and imminent clinical availability of AVP receptor antagonists as aquaretic agents for treating hyponatraemia makes interesting reading. And, finally, I was surprised that none of the papers discussed sodium loss associated with prescribed drug usage as a factor of importance.

The American Journal of Medicine 2006;119(7A):S1–S100
Highly hazardous air quality associated with smoking in cars: New Zealand pilot study

Secondhand smoke (SHS) is a complex mixture of over 3800 gaseous and particulate components, exposure to which causes a range of serious adverse health effects in children, adults, and pregnant women. The 2003 Smokefree Environments Amendment Act resulted in most indoor workplaces being smokefree. However, an area of concern in New Zealand is the persisting exposure of non-smokers, particularly children, to SHS in homes, cars, and other settings. This is particularly a problem for Māori and low socioeconomic status populations.

A recent New Zealand study found that in cars where smoking was occurring, it was commonly (24%) in the presence of other occupants. The proportion of cars with smoking occupants was higher in more deprived areas. A marker that is commonly used to measure SHS levels is fine particulates (PM$_{2.5}$). Methods for measuring air quality in cars using portable real time monitors have recently been reported. These studies recorded very high levels of PM$_{2.5}$ during and after smoking, particularly when the windows were closed. We carried out a pilot study to investigate levels of PM$_{2.5}$ in cars in New Zealand under different conditions of smoking, ventilation and speed of travel. The study had Category B Ethical Approval through the University of Otago review process.

The principal investigator (RE) drove the car, (a Honda Odyssey station wagon, in which no smoking had previously occurred for at least 10 months) while another investigator (NP) smoked cigarettes (‘lights’ brand) under specified conditions. Data was collected using a TSI SidePak AM510 (TSI, Inc, St Paul, USA) portable real time air quality monitor to record average levels of respirable particulates (PM$_{2.5}$) over 1-minute periods. The instrument was used according to a protocol modified from one developed for a US study, and as used in a UK study by RE (further details of the data collection methods are available in these publications). The SidePak was located on a child’s booster seat in the rear of the car with a length of Tygon™ tubing attached to the inlet and the other end left protruding at approximately the height of the nose of a small child sitting in the back of the car.

We began by monitoring ambient air for 10 minutes at a busy traffic intersection (Basin Reserve, Wellington) at 5pm (rush hour) in September 2006. We then drove the car around suburban areas of Wellington at a mean speed of 50 kph. The route was chosen to minimise stops at intersections and traffic lights. The fan and air-conditioning was switched off throughout the monitoring period.

Whilst driving, three cigarettes were smoked, the first with the passenger’s window fully open and cigarette held outside car between puffs; the second with the passenger window open half way and cigarette held inside the car in between puffs; and the third was smoked with all windows closed. The weather was sunny throughout, with a light to moderate breeze.
Figure 1. shows the levels of fine particulates (PM$_{2.5}$) while the first two cigarettes were smoked. Figure 2 shows the particulate levels while the third cigarette was smoked with the windows closed, and the subsequent levels over the next hour.

**Figure 1. Particulate (PM$_{2.5}$) levels (µg/m$^3$) during smoking of cigarettes in a moving car with passenger window wholly or half open**

Mean levels during smoking the smoking first cigarette with the window fully down were 168.5 µg/m$^3$, and were 143 µg/m$^3$ during smoking of the second cigarette with the window half down.

Mean PM$_{2.5}$ levels during smoking of the first cigarette were 199 µg/m$^3$ (peak 217 µg/m$^3$), during the second cigarette 162 µg/m$^3$ (peak 181 µg/m$^3$), and during the third 2926 µg/m$^3$ (peak 3645 µg/m$^3$).

Fifteen minutes after the third cigarette was extinguished, PM$_{2.5}$ levels were 631 µg/m$^3$, and did not return to the baseline level until almost 40 minutes after the cigarette had been put out.

PM$_{2.5}$ levels observed during smoking were many times higher than in the ambient air (3-4 µg/m$^3$), even next to a busy traffic roundabout. For comparison, the mean daily PM$_{2.5}$ levels in Auckland during 1998–2001 were 11.0 µg/m (range 2.1 to 37.6 µg/m)$^9$. Compared to the poorest air quality days in Auckland, PM$_{2.5}$ levels in the car during smoking were about five times worse with a window wholly or partially open, and up to 100 times worse with the windows closed.
Figure 2. Particulate (PM$_{2.5}$) levels ($\mu$g/m$^3$) during smoking of a cigarette in a moving car with all windows closed (and no other ventilation operating)

The World Health Organization guidelines for annual mean and 24 hour mean PM$_{2.5}$ levels are 10 $\mu$g/m$^3$ and 25 $\mu$g/m$^3$ respectively. Some of the highest indoor levels of particulates due to SHS are found in pubs and bars where smoking is allowed. For example, a UK study found mean PM$_{2.5}$ levels 285 $\mu$g/m$^3$ over 30 minutes of monitoring in the evening (maximum 1400 $\mu$g/m$^3$) in 64 pubs across north-west England.

Air quality in the car with the window partially or wholly down was therefore similar to that found in a typical smoky pub, whereas when smoking occurred with the window closed it was at least twice as bad as even the smokiest pub.

The results confirm that unacceptably high levels of air pollution result from smoking in cars, and show that non-smokers are heavily exposed to SHS in this setting. The findings validate the public health rationale for the current Health Sponsorship Council mass media campaign in New Zealand that encourages smokers to protect their children from the harms of secondhand smoke by not smoking in their car, even when they are alone (http://www.secondhandsmoke.co.nz/media/cars.shtml).

The findings also suggest that laws to make cars smokefree, particularly when children are present (as have been adopted in other jurisdictions such as in Arkansas, Louisiana, and Puerto Rico) should be explored.

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Should New Zealand’s Commerce Commission act on cigarette brand name deception?

The New Zealand Commerce Commission has begun an investigation into the misleading use of the terms “light” and “mild” on cigarette packets in New Zealand.\(^1\) Such action is consistent with New Zealand’s treaty responsibilities, since part of the treaty *Framework Convention for Tobacco Control* (that New Zealand has signed) requires action on misleading descriptors.\(^2\)

However, some brand names themselves can also be considered misleading. An example from New Zealand is the “Freedom” brand, because of the connotations of the name, and the flying bird images on the pack (Figure 1). Even the cigarette stems have the word “Freedom” on them (Figure 2). Given the near universal regret that smokers have about starting smoking\(^3\) and the highly addictive nature of nicotine, the use of the term “freedom” would appear to be extraordinarily inappropriate. It is also highly misleading, and by creating positive associations with tobacco smoking contributes to the promotion of tobacco.

Cigarette packs in New Zealand require health warnings in text and one of these covers “addiction” (Figure 1). However, in this case, the brand name and accompanying images are likely to seriously compromise the effect of the warning. The key word in the warning (“addiction”) is also smaller than the brand name (“Freedom”) with a font size ratio between the words of approximately 1.5 to 1 (Figure 1). And if a picture is “worth a 1000 words” the ratio of the “flying birds picture” to that of the complete warning text is over 71 to 1. For the back of the pack these ratios are over 4 to 1 and over 15 to 1, respectively in favour of the pro-tobacco message.

*Figure 1. A pack of the “Freedom” brand of cigarettes marketed in New Zealand (front side)*

*Figure 2. Stem of a “Freedom” brand cigarette*
This concern about the name of the “Freedom” brand is not new, with adverse comment on it in Australia in 1995.4 There are also other cigarette brand names with problematic positive associations in New Zealand (e.g. “Holiday” and “Lucky Strike”). What is new, however, is the neuroscience evidence base from experiments with functional magnetic resonance imaging (fMRI).5 This provides biomedical mechanisms for the powerful effect of brand names and imagery on human beliefs and behaviour.

The Commerce Commission should consider expanding its current investigation to also address deceptive branding by the tobacco industry.

In addition to fines they may impose on the industry, the policy options they could recommend to Government include:

- Making the brand name a very small part of the pack cover (perhaps 5% and on one side only);
- Not allowing certain brand names where these have positive associations and attributes; or
- Banning brand names and branding altogether and allowing only plain packs with health warnings.

Ultimately, however, there is an urgent need to adopt a new regulatory framework that removes the tobacco industry from all aspects of tobacco marketing.6–8 Such a framework could also remove the profit motivation from cigarette production and distribution by making this the responsibility of a not-for-profit agency with a public health mandate.7

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Voluntary laboratory surveillance of sexually transmitted infections is under threat

Jane Morgan, Geoff Spencer

Boswell and Tie, in their editorial *All change for the New Zealand laboratories*, raise concerns about New Zealand laboratory contracts.¹ We suggest that these contractual reviews may also pose a threat to voluntary laboratory surveillance.

New Zealand has a strong history of ensuring improved surveillance of emergent infectious diseases such as HIV, SARS, and avian influenza whereas surveillance of sexually transmitted infections (STIs) still needs to improve.²³ Traditionally, STI surveillance in New Zealand was based on the British model of sexual health clinic data. Since mid-1998, this expanded to include other clinical settings and voluntary laboratory-based surveillance of *Chlamydia trachomatis* (chlamydia) and *Neisseria gonorrhoea* (gonorrhoea), which began in the central North Island and gradually extended to other laboratories.

In areas where comprehensive laboratory and clinic-based STI surveillance coexist, laboratories reported three times as many chlamydia cases and twice as many gonorrhoea cases compared with clinics in 2005.⁴ Laboratory STI surveillance is important because many access sexual health care through primary practitioners. However, in 2006, nationwide laboratory participation remains incomplete. Public health legislation is under review which may provide an opportunity to review STI-reporting requirements. In the meantime, it is crucial that existing voluntary reporting is not undermined.

In 2004, we undertook a nationwide survey of chlamydia testing and related issues amongst all New Zealand laboratories, including those not yet participating in voluntary reporting. Whilst 94% of laboratories responded, only 32 supplied testing data, of which 20/24 were hospital-based and 12/22 were community-based. All hospital-based laboratories offered to supply data, but a few were unable to within our 3-month timeline because leave had exacerbated staff shortages and workload issues or a shortage of staff familiar with data extraction systems.

Another difficulty seemed to be ineffective information technology (IT) systems. This was evidenced by difficulties in generating unique or new reports within 3 months and that not all laboratories’ IT systems were able to de-duplicate data, that is, distinguish multiple tests from the same individual, which in turn may contribute to inconsistent data. Further, there were concerns about any future change to reporting, such as collecting ethnicity data, in part because of IT software limitations.

Some community-based laboratories cited insufficient funding for reports whilst others expressed concerns about how chlamydia testing volumes would be reported, about the “commercially sensitive” nature of the information and either did not provide data or provided only ‘estimate’ test volume data.
Differing levels of reimbursement for chlamydia tests was an issue, with cost determining the testing method used by some community laboratories. At least one has introduced a testing algorithm to allow greater use of the more sensitive Nucleic Acid Amplification Testing (NAAT) tests within their funding constraints.\textsuperscript{5} Aside from evidence supporting the use of NAAT tests,\textsuperscript{6} any ongoing use of less sensitive testing methods limits meaningful regional comparisons of any reported chlamydia data.

The current laboratory services’ contracting round seems an ideal opportunity for a structured overhaul as is planned for laboratory services in the UK,\textsuperscript{7} where similar system issues have been identified, but even a national review of laboratory service provision seems unlikely in New Zealand. Rather, laboratory rationalisation is occurring at a local or regional level with a focus on cost-savings within each DHB.

It has been argued that New Zealand’s competitive contracting model and funding methods tend to drive laboratory test volumes and discourage the full exploitation of potential economies-of-scale, resulting in excessive costs.\textsuperscript{8} Not surprisingly, negotiated contracting arrangements with new providers are being promoted as beneficial to the health system through improved cost-efficiencies. However concerns abound—and the New Zealand Medical Association, the Association of Salaried Medical Specialists, and the New Zealand Institute of Medical Laboratory Science have voiced anxieties, particularly about workforce implications.

What does this mean for effective voluntary surveillance? It seems unlikely, in the absence of contractual reporting obligations, that increased laboratory privatisation and anticipated cost savings will also lead to an improvement in the system issues that currently hinder nationwide reporting. It also seems unlikely that regional inconsistencies around reimbursement and hence use of NAAT testing for chlamydia will be resolved. Further, voluntary laboratory STI surveillance is only possible because of the ongoing enthusiasm and commitment of key individuals. If increased laboratory privatisation threatens the employment of ‘STI enthusiasts’ in the laboratory workforce, then voluntary STI surveillance is also under threat.

New Zealand needs laboratory services of consistently high quality that are also affordable. However, the possible impacts of rationalising laboratory services may be far-reaching and need to be considered. The promised public health legislation review may appease those of us interested in STI surveillance. However we should not be complacent. If it is too late for a national review of laboratory services, there is an immediate need to include data provision in all re-negotiated laboratory contracts. This opportunity should not be overlooked.

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Lengthy ethics application process hinders medical research in New Zealand: time for review?

In years past, the greatest impediment to medical research in New Zealand was a perception that research funding was meagre and hard to access. But in the 21st century, the greatest impediment must be the growth of the ethics application process.

In the last two decades of the 20th century, ethics committees were regional and generally helpful to researchers. Certainly in my time on the Wellington Ethics Committee in the mid-1980s our application requirements and review process (while robust) required only a small amount of the researchers’ time.

Now the research application process is voluminous and extremely time-consuming, sufficiently so to put many keen researchers off and to pose the question “Are the best interests of the patients AND the potential researchers being served by the current process?”

Once in the hands of a governmental organisation there is a tendency for processes, such as the ethics application, to grow without any thought as to the primary aim of the process—much to the detriment of all parties (and in this case the possible reduction in the amount of quality medical research in New Zealand).

I believe it is time for the whole ethics process to be reviewed and downsized.

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Getting the point right: a response to Methuselah on acupuncture for knee osteoarthritis

In the previous issue of the NZMJ I note that Methuselah reports on an article on acupuncture for knee osteoarthritis (OA)—http://www.nzma.org.nz/journal/119-1243/2261

The flippant conclusion seems to forget that the study showed a highly significant effect for acupuncture but did bring up the issue of the methodological difficulties of using sham comparison groups. For a better understanding of this issue, see:


However, a more recent and large systematic review and meta-analysis has found acupuncture to be significantly effective in benefiting patients with peripheral joint OA, as compared to sham acupuncture:


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Warrick Nelson, Ben Harris

Baker et al (2006)\(^1\) claim overwhelming evidence that fresh chicken is the dominant source of human infection. Unfortunately, they are unable to demonstrate this. We agree there is very considerable circumstantial evidence that increasing chicken consumption is correlated with increasing rates of campylobacteriosis, but even their own graphs indicate a very erratic progression in campylobacteriosis rates, while chicken production is a smoothly graded annual increase.

In our earlier paper,\(^2\) we attempted to provide a credible transmission route to account for the marked seasonal variation in disease incidence, a previous suggestion that flies could be involved, and the common implication of chicken consumption. In response to their letter to the editor\(^3\) we further showed a correlation between increasing campylobacteriosis cases and dairy cow numbers.

A further interesting correlation, possibly more compelling than the chicken production correlation, is that between short term visitor arrivals and campylobacteriosis cases (Figure 1). The summer seasonal peak in visitor arrivals coincides with or just precedes the annual summer peak in campylobacteriosis cases. Furthermore, the annual August peak in visitor numbers also precedes, although less clearly, a smaller late winter/early spring peak in campylobacteriosis cases.

Tourism and travel are a strong risk factor for campylobacteriosis. For New Zealand, only 6-12% of cases have been thought to be travel-related.\(^4\) Norway and Iceland, for example, separate campylobacteriosis cases into domestically and foreign-acquired cases. For these two countries, travel is now clearly a major risk factor for campylobacteriosis.\(^5\)\(^6\) Sadly, even our most common tourist guide books now recognise and clearly warn travellers against the risk of infections by drinking or swimming in water in most parts of New Zealand.

There is increasing new evidence casting doubt on chicken itself as the dominant source of human campylobacteriosis, in spite of the undoubted high contamination rate of chicken meat. For example, a large Danish investigation found the most common subtypes from both humans and cattle were identical over six subtyping methods.\(^7\)

Specific comparison of human and poultry isolates from Ireland showed significant differences in percentages at the species level (\textit{Campylobacter jejuni} and \textit{C. coli}), as well as chicken isolates expressing more antibiotic resistance than the human isolates.\(^8\) Welsh human and chicken peaks in \textit{Campylobacter} seasonal peaks indicate a human peak some 2 weeks before chickens.\(^9\) Polish isolates from children and chickens showed little overlap at the gene level.\(^10\)
Elimination of *Campylobacter* in chicken meat production, while an admirable end in itself and likely worth pursuing, will not resolve the campylobacteriosis problem. The evidence shows a significant number of human cases arise from a likely combination of more complex factors and transmission routes. Insistence on a single source pursuit narrows the focus too much and draws attention away from other, equally likely, transmission routes. There is still far too little known about *Campylobacter* microbiology and epidemiology to focus only on risk reduction as suggested.

The arguments presented here are sound evidence to doubt the primacy of chicken-as-sole-source for campylobacteriosis and it would be a mistake to shut down other avenues of enquiry.

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References:


Walter Stewart James Tongue

1937–2006

Walter (Wally) Tongue was born into a prominent Auckland family, he was one of the city’s characters and died aged 68 at the start of a new phase in his life just after retirement from clinical practice in obstetrics and gynaecology.

He was educated at Kings School and Kings College in Auckland then went on to Otago University graduating MB ChB in 1962 followed by postgraduate training at National Women’s Hospital, St Thomas’s in London, and the Hillingdon Hospital.

Wally left Auckland with the Diploma in Obstetrics and returned in 1970, after completing the MRCOG in London.

He was appointed Tutor Specialist at National Women's Hospital which was a 3-year appointment but, during his third year he was chosen for the senior lecturer post in the Post Graduate School. From there, in 1973, he went into private practice by taking up a part-time position at St Helen’s Hospital which he held until 1990 when the hospital was disestablished then became a member of the part-time visiting staff of National Women’s and the North Shore Hospital.

Wally had a busy private practice in central Auckland, he provided services at the Mater Maternity, and was Chairman of the Attending Staff from 1982–1985. Also, he worked at the smaller maternity units in greater Auckland and at the Thames Hospital.

He took his share of professional responsibilities—he was Clinical Director of the Community Maternity Services at National Women's for 5 years until his retirement in 2004, Chairman of the Auckland O&G Society, and specialist representative on Maternity Benefits Negotiating Committee for the NZMA. Wally was very good at clinical teaching, bringing commonsense and a vast experience which was appreciated by the trainees whom he taught the value of heads and hands rather than machines.

He had a long association with the Royal New Zealand Navy Volunteer Reserve rising to the position of Surgeon Commander and from 1977 to 1983, and he was Honorary Physician to three Governor Generals in New Zealand. He had many interests outside medicine, he was very proud of the achievements of Joy whom he married in 1966 as well as their sons—Nicholas who represented New Zealand in Commonwealth and Olympic Games in swimming, and Charles who has excelled in Arts Administration.

Wally played a good round of golf and was a stalwart of the Auckland Golf Club. However, he was cruelly deprived of his ability to play golf and he was restricted in his life’s work in O&G by a freak complication of a heart attack which left him paraplegic at the age of 66.
Very recently, whilst establishing a holiday home in Noosa, he was struck down by legionnaires’ disease. The zest for life, which was his defining quality, never abandoned him despite being confined to a wheelchair, he was unstoppable in his determination to do all that was possible and he remained incredibly cheerful through all adversity. He learned to drive his car again, and he was invited to join the Board of the Laura Fergusson Trust having been through their programme for rehabilitation.

St Mary’s Church in Parnell was packed for his funeral, a testament to the high regard in which he was held by people from all walks of life in Auckland, and no-one who met Wally will ever forget his exuberance, his generous spirit, and good company.

Tony Baird (Auckland Gynaecology Group, Parnell, Auckland) wrote this obituary.
Alexander Vincent Kurta

16 September 1924–4 September 2006

Born and educated in New Plymouth, Dr Alex Kurta qualified in medicine at Otago University.

He spent his house surgeon years in Wellington, Christchurch, and Gisborne, and left on the last coal-burning steamer for England in 1953, later joined there by his fiancée, Laura.

He trained at east London’s Whipp’s Cross Hospital, and in Edinburgh, and gained his Membership of the Royal College of Physicians, later being elected a Fellow.

The first of their children was born in London.

Alex returned to New Zealand in 1959 and took up the post of Visiting Physician, specialising in Respiratory Medicine.

At the time, tuberculosis was very common, and his expertise in battling this disease throughout Hawke’s Bay was welcomed. Like most other hospital specialists of the time, Alex commenced general practice simultaneously in rooms in King Street North, which he continued until 1994. He also served as Physician to the Little Sisters of the Poor Holy Family Home and to Woodford House.

He was an enthusiastic teacher and often gave lectures to student nurses training at Hastings Hospital, and was fully involved in the education of young doctors.

Alex was never much given to high technology or medical gimmickry, but had been well trained in the traditional arts of history-taking and careful physical examination, which he combined with shrewd clinical judgement and a very humane and caring approach which made him well loved by his patients. He also won the respect and affection of his colleagues.

Alex was an avid collector of art and of antique wine glasses, amassing a large number of items, many more than 200 years old. He also had a keen interest in history and a quirky attachment to topiary.

Alex was a fine physician and will be sadly missed by his patients, colleagues, and many friends. He is survived by his wife, Laura, their 6 children, and 14 grandchildren.

Paddy Twigg (GP, Hastings) wrote this obituary.
Erratum


The above paper was published with an error in the Results Section. The number of smokers listed as “n=788” in Table 1 should read: n=180. The authors apologise for this error.

Please refer to the above URL to view the corrected copy of the article.
Violence and Aggression in the Workplace


This book is a practical guide for dealing with violence and aggression in the health workplace. This book would be useful for anybody working in the medical profession with patient contact. Dealing with violent and aggressive patients can occur in any specialty, at any time, and sometimes unpredictably.

The book discusses the problem and theories of workplace aggression. It then gives good practical solutions for defusing and managing these difficult situations. It is comprehensive and informative.

Workplace Bullying in the NHS


This book is written about bullying in the medical workforce. Workplace bullying has only recently become subject to scholarly research.

The book uses the recent large volume of research and puts it together in a textbook fashion. It starts with an overview of workplace bullying and then looks at the causes behind it.

It has a useful practical approach using case scenarios and then has a thorough approach to dealing with the problem.

The book is edited by Jacqueline Randle who is an Associate Professor of Nursing from the University of Nottingham. The book chapters are written by a number of different experts.

This book would be useful to people who want to know more about workplace bullying in the health sector from an indepth academic viewpoint. Managers who deal with workplace bullying complaints should also read this book. This book will give them the tools to effectively deal with the problem.

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