THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

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THE NEW ZEALAND MEDICAL JOURNAL
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This Issue in the Journal

Role of alcohol in maxillofacial fractures
Kai H Lee, Leslie Snape

According to data gathered at Christchurch Hospital over 11 years, alcohol-related trauma is a major social and economical burden requiring effective use of hospital resources and patient interventional strategies. Young men in the 15 to 29 year age group are the main victims. Most alcohol-related facial fractures were due to interpersonal violence. The high prevalence of alcohol as a contributing factor to facial fractures highlights the need for community awareness and public education on the harmful effects of alcohol.

Under-18 year old callers to New Zealand's Quitline
Maria Poynter, Chris Bullen, Robyn Whittaker, Michele Grigg

This paper aimed to investigate the characteristics of under-18 callers to New Zealand's Quitline (a telephone counselling service for people wanting to stop smoking). We analysed information collected about first-time Quitline callers in 2004 and 2005, using all (2371) callers under 18 years of age, and also a sample of 2000 18-and-over callers for comparison. We found that females and teens in their older teen years called more often, and although Māori and Pacific callers were better represented than we had expected, they were still under-represented compared to the proportion of under-18 smokers in the general population who are Māori or Pacific. Despite similar levels of nicotine dependence in under-18 and 18-and-over callers, under-18s were issued nicotine replacement therapy only half as often as adult callers.

"If everyone does it, it's not a big deal." Young people talk about chlamydia testing
Sally B Rose, M Camille Smith, Beverley A Lawton

Young people's attitudes to chlamydia testing were studied among 16–24 year olds. Interviews discussed barriers to chlamydia testing, methods of accessing testing, communicating information about *Chlamydia*, and ideas about ways to encourage testing. Reasons for not seeking testing included fear, stigma, denial of personal risk, and a lack of knowledge about chlamydia and about testing procedures. Better education and a need to 'normalise' testing were suggested as ways to increase test-uptake. All groups supported routinely offered chlamydia testing when visiting the doctor for other reasons. Participants also favoured the concept of home-testing. These findings can be used to inform the development of much needed new initiatives to control chlamydia in New Zealand.

Herpes simplex type 1 versus *Herpes simplex* type 2 in anogenital herpes; a 10 year study from the Waikato region of New Zealand

Erana Gray, Jane Morgan, Jennifer Lindeman

Anogenital herpes is herpes of the genitals and surrounding areas including around the anus and on the thighs. It can be caused by 2 types of herpes viruses—knowing which one is important, because the outlook is different for each. Type 1 is much less likely to recur than type 2, which is significant, because it is the recurrences (especially if frequent) that distresses people the most. Type 1 also causes 'cold sores' on the mouth and it may be that genital infection comes from oral sex. We found very high levels of type 1 overall, and found that it is commoner in younger people and in females. We discussed some of the reasons why that might be. We think that other centres should consider changing to the specific test used here, so that the patient can find out which sort of herpes they have.

Walking to school: frequency and predictors among primary school children in Dunedin, New Zealand

Sofie Yelavich, Cindy Towns, Richard Burt, Kent Chow, Roana Donohue, Haji S H Sani, Keryn Taylor, Andrew Gray, Jason Eberhart-Phillips, Anthony I Reeder

Using an in-class survey and a take-home caregivers' questionnaire, we found how many Dunedin primary school children walked to school and identified some factors associated with this walking. Key potentially modifiable predictors of walking were living 3 or less kilometres from school and not having a car in the household. These findings have potential implications for health, transport, and educational policies.

Rheumatic fever diagnosis, management, and secondary prevention: a New Zealand guideline

Polly Atatoa-Carr, Diana Lennon, Nigel Wilson; on behalf of the New Zealand Rheumatic Fever Guidelines Writing Group

Acute rheumatic fever is a disease caused by a bacterial throat infection and it can result in rheumatic heart disease. Rheumatic heart disease is a significant cause of early death, illness, and inability to work or learn for many New Zealanders, particularly for Māori and Pacific communities. Severity of rheumatic heart disease can be controlled by appropriate diagnosis, treatment, and management of acute rheumatic fever patients. Diagnosis and management of acute rheumatic fever cases often varies. In addition, implementation of effective prevention strategies (to avoid further attacks of rheumatic fever) is not always consistent. This paper summarises a peer-reviewed national guideline for medical practitioners in the diagnosis, management, and secondary prevention of acute rheumatic fever. The intention is that improved consistency in the diagnosis and management of this disease will reduce health inequalities and also reduce the effect that acute rheumatic fever and rheumatic heart disease has on the lives of many New Zealanders. Data from the New Zealand Child & Youth Epidemiology Service shows acute rheumatic fever to be highly likely to appear in areas of socioeconomic disadvantage. It is now very rare in any populations in Western Europe or North America.

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Children in New Zealand: their health and human rights

Nikki Turner, Karen J Hoare, Tony Dowell

> …Many things we need can wait. The child cannot. Now is the time his bones are being formed; his blood is being made; his mind is being developed. To him we cannot say tomorrow. His name is today. (Gabriela Mistral)

Eighteen years ago, World leaders gathered at the United Nations to attend the World Summit for children. The meeting culminated in the launch of the United Nations Convention on the Rights of the Child (UNCROC)—the World's most widespread human rights treaty.

Of the World's 193 nation states, only Somalia and the USA did not commit to its ratification. New Zealand's ratification committed the country to enshrine the 54 articles in the UNCROC in all of its policies. The convention enshrines for children, the right to life, survival and development, the right to an opinion and for that opinion to be heard in all contexts, protection from discrimination, and that the best interests of the child should be the primary consideration in all matters.

In 2003 the United Nations Committee met to consider how well New Zealand had implemented UNCROC.[1] Several comments and recommendations were made by the Committee including the need to prioritise children in the Government budget and improve the health of children and young people by taking action in a range of health initiatives. They emphasised the need to focus on children from poor families and address the disparities between ethnic groups, in particular Māori.

What is the status of New Zealand children 18 years on from UNCROC?

Despite the 2003 United Nations Committee recommendations, in 2007 UNICEF drew the World's attention to New Zealand's poor performance in the health of our children: more children die from injuries than any other of the 24 OECD countries providing statistics, along with comparatively high infant mortality rates and low immunisation rates.[2]

The recent report by the New Zealand Child and Youth Epidemiology Service[3] highlighted a broad range of areas with poor child health outcomes. The outstanding feature of this report is the significant socioeconomic and ethnic disparities observed in the outcomes across almost every health indicator.

For example, the relative risk of dying from sudden infant death syndrome is 10.6 times higher for an infant in NZDep Index decile 9–10 over decile 1–2;[3] the relative risk of being hospitalised for a serious skin infection is 5.2 times higher in children from decile 10 compared to those from decile 1.

For many child health measures there has been a consistent pattern of significant increases in hospitalisations since the early 1990s,[3] which although levelling off in the past few years, remains at higher levels than prior to the 1990s. This has occurred at a time when New Zealand has been showing increasing inequities[4] and a rise in relative and absolute child poverty.[5]

Moreover, in youth and adolescence, high levels of psychological disorders, suicide, teenage pregnancy, and substance abuse are creating a context in which many young people are unable to reach adult life with security and confidence.

The Ministry of Social Development has been producing research since 2000 focusing on living standards for all New Zealanders. The outstanding feature of this research is the fact that children are disproportionately more likely to be living in severe or significant hardship than any other sector of New Zealand society.

Table 1. Percentage of the New Zealand population living in severe/significant hardship as measured by the Living Standards Reports 2000 and 2004

Variables	% in severe/significant hardship 2000	% in severe/ significant hardship 2004
Children	**18%**	**26%**
Adults 25-44 yrs	12%	15%
Adults 45–64 yrs	8%	10%
Adults 65+ yrs	2%	4%

Source: Summarised data from Figure 44, The Living Standards Report, MSD 2004.[6]

Poor health outcomes for children have significant short-term and long-term consequences. There is abundant evidence that experiences *in utero* and early childhood can have profound effects on long-term health and social outcomes. To focus on achieving good health for children is a sensible societal and economic decision and complies with New Zealand's obligation to implement the UNCROC.

While the challenges to improving child health may appear formidable, there are feasible solutions. Economic disparities, the strongest driver for poor child health outcomes, have been tackled in many countries through a range of macro-economic solutions.

The recent leveling off of hospitalisation rates in this country is likely to be related in part to current strong economic growth and higher employment rates. Also the effects of recent government policies focusing on supporting those on lower incomes particularly the *Working for Families* package will be expected to have a positive contribution,[7] although it offers little to the children of beneficiary parents, who are likely to be disproportionately more represented in poor health outcomes.

To enhance these economic contributions to improving child health, the restoration of a universal child benefit could be the greatest single preventive health measure we could devise.

All complex health issues have complex and multi-factorial underlying aetiology and child health is no exception. Beyond macro-economics it is important that we acknowledge other challenges and paradoxes that may contribute to our poor child health outcomes.

There is increasing evidence that ill-health and other social problems are linked to relative deprivation and income inequality rather than absolute levels of income.[8] There is no obvious strong relationship between Gross Domestic Product (GDP) per capita and child well-being; poorer countries than us (Cuba is a good example) have much better child health statistics.

Appreciation of relative deprivation, rather than absolute, and the corrosive impact of increasing income inequality means that we must all engage in the debate about our children's future.

This debate should include a fundamental question about how we, as individuals, communities, and society in New Zealand view children.

There continues to be widespread belief that New Zealand is a child-focused and "child-friendly" society. The reality, however, is that for many children it is a harsh and brutal environment with high levels of stress, illness, anger, and violence. In addition many New Zealand institutions, and traditions, are tolerant of children rather than engaging directly with them.[9]

We need to confront the paradox that while New Zealand is an enviable physical environment for children there are few indications of a truly positive child centred society.

At a broad policy level, working together more effectively across sectors is achievable and effective. For instance, the UK has shown improvements in child outcomes with top-level commitment to children, with strategy and funding integrated across education, welfare, health, housing, and local government.[10]

There are also a wide range of health and education specific initiatives known to help improve children's' health. These include initiatives such as improving access to quality primary health care, resourcing to early childhood programmes, parenting programmes, and home visiting to families in need.[10] However a number of current New Zealand health policies affecting young people, such as those affecting alcohol consumption and tolerance of morbidity and mortality caused by immature road use, are increasingly out of step with other OECD countries.

The Ministry of Health's *Primary Health Care Strategy*, with a greater focus on population health, could further resource systems able to enhance enrolment and tracking of children. There are many children who are still not enrolled with a regular provider, despite evidence that early enrolment and regular relationships with primary care providers do improve health outcomes such as immunisation coverage rates.[11]

There is evidence to show that a systematic approach to child health in primary care can produce positive change.[12] Good enrolment and tracking systems have worked to protect children in other OECD countries,[10] and well organised primary health care compensates for substantial social disadvantage.[13]

In summary, while one-third of New Zealand's population is made up of children, and while children are disproportionately more likely to be living in hardship than other members of our society, our child health statistics will remain a shameful fact of life in this country, and New Zealand will continue to fail its international commitment to UNCROC. However there are known, effective strategies that can affect these statistics.

To achieve change requires individual and community-wide acceptance that we have a significant problem, and a willingness to make the necessary economic and societal efforts to bring about change.

New Zealand has both the tools and ability to improve child health; we have taken some effective steps in this direction. However if there is a genuine commitment to improving the future of our children now is the time for bigger bolder action.

> …An aging society that does not take care of its young has a death wish. (Dame Anne Salmond, Knowledge Wave conference, 2003)

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The knock-on effects of unrestrained drinking

Jennie Connor

The paper by Lee and Snape in this issue of the *Journal* entitled *Role of alcohol in maxillofacial fractures*[1] uses 11 years of data from Christchurch Hospital to provide a robust description of this problem in New Zealand. It goes on to urge surgeons working in their specialty to get involved in advocacy for effective public health policy to reduce such injuries.

While the research is new, most readers won't be very surprised by the findings. They illustrate a number of sentinel issues about alcohol and public health. Much alcohol-related harm is due to injury, both intentional (e.g. assault) and unintentional (e.g. traffic crashes), occurring when at least one of the parties is impaired by alcohol. Men are more commonly involved, particularly the young. It is not just the drinkers who are harmed, but those around them. There is a substantial impact on the health care resources of communities, and there will be mental and social trauma to accompany the physical impact.

Maxillofacial fractures represent just one of many adverse effects of unrestrained drinking. While this study provides a first assessment of the incidence of this problem and size of alcohol's contribution, there are many other impacts of drinking that are unmeasured or not widely acknowledged. Along with the physical health conditions that are measurable from within our health systems, and that make up the basis of our estimates of alcohol's health impact,[2] there are other physical, psychological, and social harms to the hazardous drinker. An emerging area of concern that involves all of these dimensions is the effect on the drinker's sexual behaviour and sexual health.

However, the most neglected area of research about the impacts of alcohol on the community is the effect of alcohol misuse on people other than the drinker. There are many ways that the impact is felt, from the loss of amenity due to noisy parties in residential streets, to the violent deaths of partners and children. We know that heavy episodic drinking is common but we know remarkably little about the size of this burden in our homes and neighbourhoods. As a consequence, we fail to account sufficiently for alcohol's harm to others when we consider our options to reduce hazardous drinking.

Referring to the situation in the United Kingdom, the leading alcohol researcher Thomas Babor recently asserted that "Nations, like people, can develop a pathological pattern of alcohol misuse", and linked the UK's pattern of alcohol misuse with the liberalisation of the drinking environment and the development of alcohol-control policy in "partnership" with the alcohol industry.[3]

The recent history of alcohol policy in New Zealand has been somewhat similar to that in the UK. Changes to the Sale of Liquor Act in 1989 and the subsequent amendments have brought us supermarket sales, a rapid escalation in the number of liquor outlets, and longer opening hours. The constraints were removed from alcohol sponsorship and broadcast advertising, and then in the face of evidence that harm to

young people would result,[4–6] the minimum purchase age for alcohol was lowered (from 20 to 18) in 1999.

There seems to have been a shift from consumption that was demand-driven but constrained by regulation, to a saturated market where increases in consumption are being achieved by initiatives from the suppliers. The emergence of ready-to-drink mixtures (RTDs or alcopops) has been a marketing coup, and these are now the preferred drinks of heavy drinking young women.[7]

There has been considerable harm, as expected, and some aspects of it have been rigorously measured. Hazardous drinking amongst young people has increased, and amongst 12–17 year olds there is no significant difference in the prevalence of heavy drinking between boys and girls.[8] Car crashes involving young drinking drivers have increased significantly, including in those under the new purchase age of 18, and this has been more marked in women.[9] However this evidence, and the opinion of the Select Committee that considered all the evidence, did not prevail when the decision was made in November 2006 not to raise the minimum purchase age back to 20.

One of the arguments against raising the purchase age again was that the changes in drinking culture are in some way irreversible. This suggests that we don't have good evidence about what might be effective interventions to reduce alcohol-related harm. In fact there is a wealth of evidence. Documented authoritatively in the 2003 book *Alcohol: no ordinary commodity*,[10] and summarised in the 2005 Lancet review *Alcohol and Public Health*,[11] this evidence base has now been expounded with great clarity by the British Medical Association's Board of Science in their report *Alcohol misuse: tackling the UK epidemic* released in February 2008.[12]

The BMA's recommendations include measures to control access to alcohol, promotion of responsible industry practices, measures to reduce drink driving, promotion of early intervention and treatment for problem drinkers, education and health promotion, and international cooperation on alcohol control. While these sound very general, the recommended measures themselves are actually very specific and evidence-based.

Controlling access to alcohol includes reducing hours of sale and the density of liquor outlets, and increasing taxes on alcohol in real terms. "Promoting responsibility" means strict enforcement of licensing laws so that underage drinkers and those who are intoxicated don't get served. It also means putting a stop to irresponsible promotion of drinking to young people, such as with the marketing of RTDs. Effective interventions to reduce alcohol-related car crashes include lowering the legal allowable BAC limit from 80 to 50 mg/100 ml. The widespread use of screening for alcohol problems in medical settings and the use of brief interventions, as well as expanded services for problem drinkers, are recommended because there is evidence that they work.

Educational initiatives and public information campaigns are always popular with politicians and with the alcohol industry. These interventions are appealing and easy to implement, but ineffective in reducing alcohol-related harm.[11] They have only an adjuvant role in building public support for other interventions. Specifically mentioned in the BMA's recommendations for health promotion is the inclusion of

the UK safe drinking limits on all advertising material and product labels to aid a shift in public awareness.

Finally, the BMA is calling for an international framework for alcohol control, as has been developed by the WHO for smoking (The Framework Convention on Tobacco Control). A powerful component of the discourse on tobacco control in the last few decades has been the documentation of the harm to others, and this must be a priority in alcohol research as well. Perhaps New Zealanders would be surprised to know that an estimated 40% of all deaths and almost half of all other injuries from alcohol-related car crashes are to "innocent victims", and that nearly half of these fatalities are under 20 years old.[13]

In New Zealand, people have many different ways of drinking and some choose not to drink at all. Many drink in a low-risk manner and reap the social benefits. However, for a large sector of the population there is a dominant pattern of heavy intermittent drinking episodes, the worst pattern for the drinker's own health outcomes, and the worst for damage to those around them.

Population-based approaches—such as increasing the price of alcohol, reducing access, and enforcing licensing laws—reduce hazardous drinking, and have their most marked effect on young people and heavy drinkers. They have little impact on the moderate drinker, except to reduce their risk of harm from the drinking of others.

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Role of alcohol in maxillofacial fractures

Kai H Lee, Leslie Snape

Abstract

Background Excessive consumption of alcohol results in impaired judgement and inappropriate behaviour, and is often a major contributor to interpersonal violence and motor vehicle accidents. This study examines the experience of a tertiary centre in alcohol-related facial fractures.

Methods A retrospective database of patients presenting to the Oral and Maxillofacial Surgery Service at Christchurch Hospital (New Zealand) during an 11-year period was reviewed. Variables examined include demographics, type of fractures, mode of injury, and treatment delivered.

Results 2581 patients presented with facial fractures during the study period, 49% of these being alcohol-related. Males accounted for 88% of alcohol-related fractures and 59% were males in the 15 to 29 year age group; 78% of alcohol-related fractures were due to interpersonal violence and 13% to motor vehicle accidents; 65% required hospital admission and 58% underwent surgery.

Conclusion The majority of alcohol-related facial fractures were due to interpersonal violence, with young men in the 15 to 29 year age group being predominantly affected. Alcohol-related fractures were associated with an increase in the incidence of hospitalisation and surgery. The high prevalence of alcohol as a contributing factor to facial fractures indicates a need to push for community awareness and public education on the harmful effects of alcohol.

Excessive consumption of alcohol is strongly associated with facial injuries.[1–4] Alcohol impairs judgement, brings out aggression, often leads to inter-personal violence (IPV), and is also a major factor in motor vehicle accident (MVA).[5]

There has been strong pressure by governments through advertisement and television to outlaw drunk drivers and enforce regulation. One such campaign is the *If you drink then drive, you're a bloody idiot* advertisement that was run effectively in Australia and New Zealand. However, no nationwide campaign has addressed the close relationship between social alcohol drinking in young adults and the escalation of IPV.

The incidence of complex pan-facial fractures has decreased over past decades, partly due to a decrease in MVA-related facial fractures.[6,7] A British survey reported a decrease of 34% of patients presenting to hospital with MVA-related facial injuries and a drop of facial bone fracture from 6.2 to 4.1 per 100,000.[2]

Seat belts and enforcement of no drink-driving has had a significant impact on reducing MVA-related maxillofacial trauma and this is further reduced by other safety mechanisms such as airbags.[2] However, the overall incidence of facial injuries has increased, partly explained by a steady increase in assault cases.[4,8]

Alcohol-related facial injuries affect mainly young adults. Sixty-one percent of patients in one study were young adults in the 15 to 25 year age group; almost half of these facial injuries were sustained in assaults (usually in bars or nearby streets), with 40% requiring specialist treatment.[2]

Facial trauma is not only functionally debilitating, with the majority requiring short-term time off work and experiencing limitations in certain tasks such as chewing and physical activities, but also has a particularly severe impact on the patient's emotion, as it may be cosmetically disfiguring and psychologically devastating.[9,10]

To date, there has not been a study on the role of alcohol at time of injury on the pattern and severity of facial fractures in New Zealand. Such data is useful not only for the purpose of raising public awareness, but also for implementation of government strategies and education of other health providers. It also outlines the social burden of such injuries and is useful for planning effective use of hospital resources.

Materials and methods

This study reviews patients who were referred to a tertiary hospital Oral and Maxillofacial Surgery (OMS) Service during an 11-year period from January 1996 to December 2006, with particular emphasis on alcohol-related facial fractures.

The details were reviewed retrospectively via a trauma database at the OMS Unit at Christchurch Hospital, where all facial fractures except isolated nasal fractures are managed by the team. The OMS Unit has maintained a paper-based database of all patients who have presented for consultation and treatment to the service since 1979.

The database is based on trauma patients reviewed by the on-call house surgeons and verified by the on-call Oral and Maxillofacial consultant during their on-call week. The house surgeons are advised to update the trauma book on a daily basis.

Alcohol involvement was noted if a patient had two standard drinks or more within an hour of the injury or when a patient was injured by another under similar influence. Patients with facial lacerations without fracture are not included in this study. In New Zealand, law does not allow routine check of blood alcohol level. Therefore, determining alcohol involvement often depends on patients self-reporting or clinical documentation by the emergency physician who usually consulted the patients first before referral to the OMS Service.

Variables analysed included demographics, diagnosis, mode of injury, sites of fractures, and treatment approach. A database was created using Microsoft Excel software, then results analysed and tested for statistical significance using SPSS 13.0 for Windows (Statistical Package for the Social Sciences; SPSS Inc, Chicago, Illinois, USA) software.

Statistics performed include descriptive statistics and Chi-squared test for bivariate associations.

Results

Demographics—Of a total of 2581 patients seen in the 11-year period, 1264 (49%) patients had alcohol-related facial fractures. In the alcohol group, a total of 47 patients (3.7%) had incomplete data, but were included in the data analysis. In this group, 88% of patients were males and 12% females.

There were 65% of patients in the 16 to 30 year age group, 23% in the 31 to 45 year age group, and 8% in the 46 to 60 year age group (Table 1). This differs from the non-alcohol group, in which males accounted for 75%, the 16 to 30 year age group accounting for 41% of fractures, and the 31 to 45 year age group 21% (Figure 1). The

differences in gender and age group between the alcohol-related and non-alcohol related fractures are statistically significant ($p<0.05$).

Figure 1. Age group distribution and presence of alcohol involvement

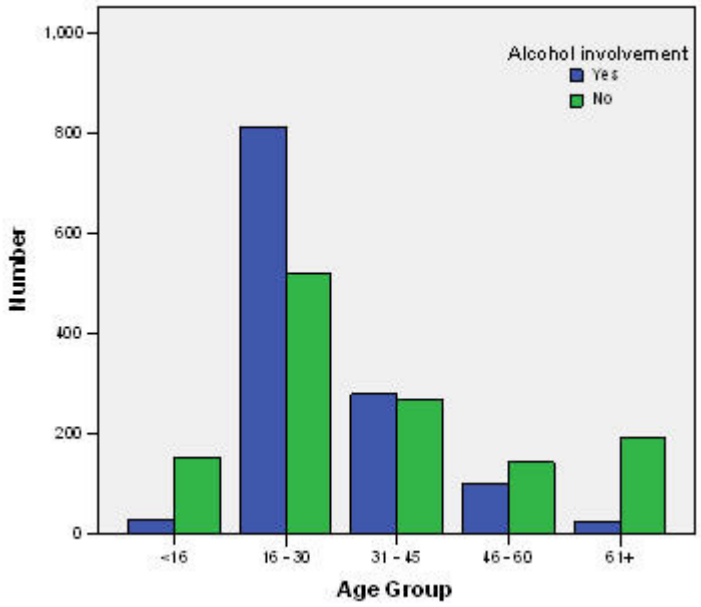


Table 1. Distribution of fractures according to age and gender in patients with alcohol involvement

Age (years)	Male numbers (%)	Female number (%)
<16	19 (1.5%)	7 (0.6%)
16–30	729 (59.2%)	75 (6.1%)
31–45	243 (19.7%)	35 (2.8%)
46–60	79 (6.4%)	22 (1.8%)
61–75	18 (1.5%)	5 (0.4%)
Total	**1088 (88.3%)**	**144 (11.7%)**

Table 2. Distribution of fracture site in patients with alcohol involvement

Site of fracture	Total (%)
Frontal	44 (3.5%)
Midface	
Zygomatic	438 (34.7%)
Orbital wall	246 (19.5%)
Maxilla	137 (10.8%)
Mandible	579 (45.8%)

Aetiology—IPV and MVAs were the main causes of facial fracture in the alcohol group (Figure 2), accounting for 78% and 13% of all fractures respectively. In the non-alcohol group, sport, fall, and IPV were the three main causes of injuries, responsible for 36%, 22%, and 12% of the injuries respectively (Figure 3).

Figure 2. Causes of fracture in the alcohol group

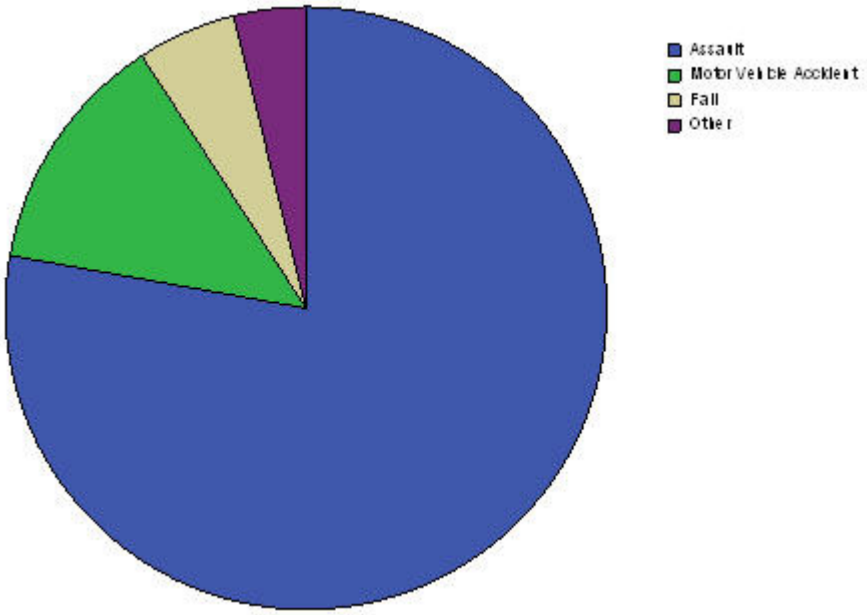
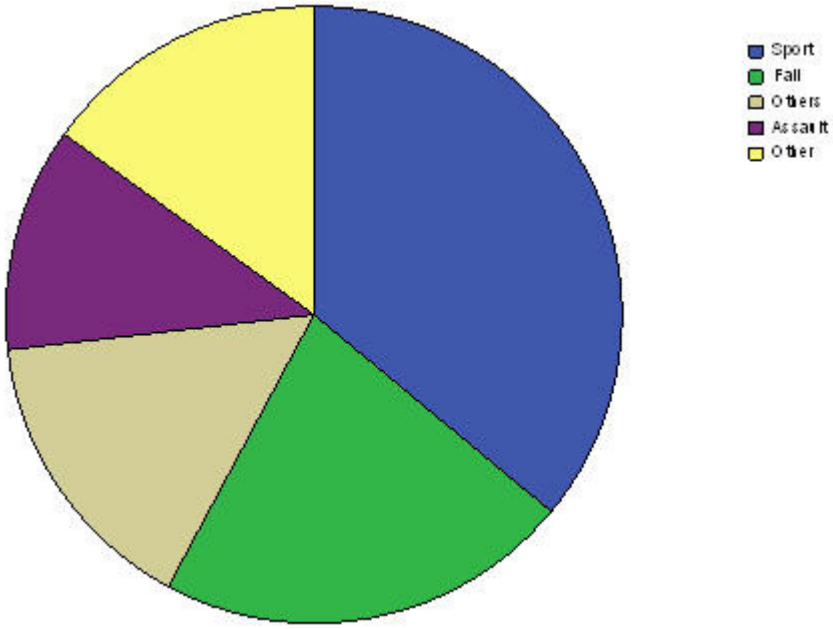


Figure 3. Causes of fractures in the non-alcohol group



Site of injury—Distribution of fracture site is illustrated in Table 2, with some patients suffering fracture at more than one site. There was a similar proportion of fractures involving the upper third and middle third of the face between the alcohol and non-alcohol groups, although mandibular fractures were more prevalent in the alcohol group (46% versus 36%).

Management—65% of patients with alcohol-related fractures required hospital admission. Of all patients in the alcohol group, 58% underwent surgery, with 44% requiring internal fixation of their fractures (Table 3).

Table 3. Methods of treatment for patients with alcohol involvement

Method of treatment	Total (%)
Conservative	492 (39.4%)
Surgical	
Open reduction & internal fixation (ORIF)	561 (44.4%)
Other method	175 (13.8%)
Others*	36 (2.8%)
Total	**1264 (100%)**

*Including non-attenders, no follow-up data, or missing data.

A high number of young male adults were hospitalised (Figure 4). On the other hand, only 55% of patients in the non-alcohol group required hospitalisation and 45% required surgery (Table 4 and 5), both of these variables being statistically significant ($p<0.05$).

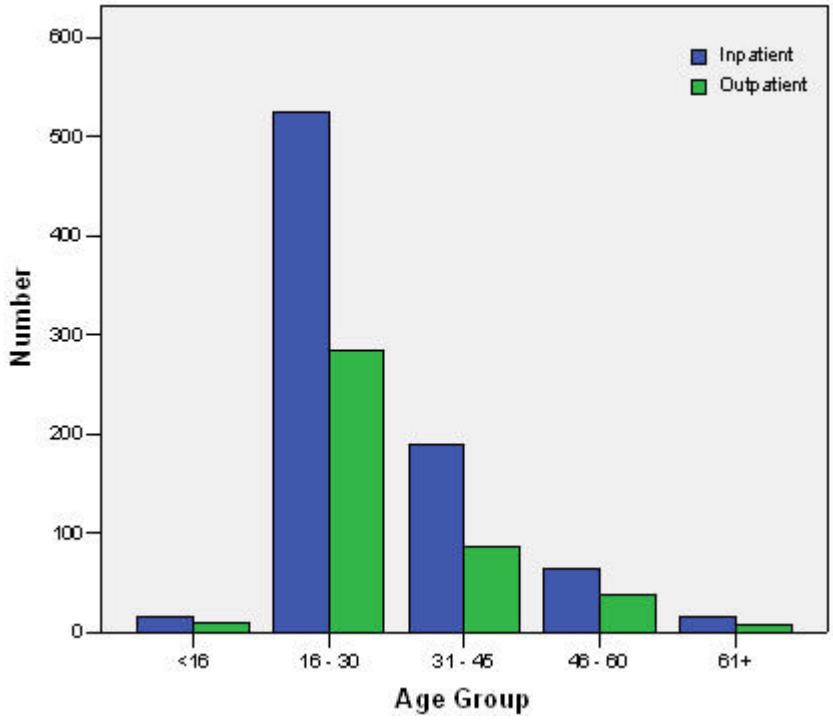
Table 4. Hospitalisation

Age (years)	Alcohol group		Non-alcohol group	
	Hospitalised	Non-hospitalised	Hospitalised	Non-hospitalised
<16	16 (1.3%)	10 (0.8%)	67 (5.3%)	84 (6.6%)
16–30	524 (42.4%)	284 (23.0%)	283 (22.4%)	234 (18.5%)
31–45	190 (15.4%)	87 (7.0%)	155 (12.2%)	111 (8.8%)
46–60	64 (5.2%)	37 (3.0%)	89 (7.0%)	53 (4.2%)
>60	15 (1.2%)	8 (0.6%)	97 (7.7%)	93 (7.3%)
Total	**809 (65.5%)**	**426 (34.5%)**	**691 (54.6%)**	**575 (45.4%)**

Table 5. Surgery

Age (years)	Alcohol group		Non-alcohol group	
	Surgery	No surgery	Surgery	No surgery
<16	13 (1.1%)	12 (1.0%)	44 (3.5%)	105 (8.4%)
16–30	478 (39.0%)	312 (25.4%)	273 (21.8%)	228 (18.2%)
31–45	169 (13.8%)	101 (8.2%)	133 (10.6%)	121 (9.7%)
46–60	52 (4.2%)	47 (3.8%)	72 (5.7%)	64 (5.1%)
>60	10 (0.8%)	11 (0.9%)	41 (3.3%)	138 (11.0%)
Total	**722 (58.8%)**	**483 (39.4)**	**563 (44.9%)**	**656 (52.4%)**

Figure 4. Hospitalisation in alcohol group



Discussion

This study shows a high level of alcohol involvement in facial fractures, comparable to overseas studies[2,3,11] which also show a similar level in alcohol-involved facial fractures over more than a decade. According to our study, alcohol was involved in half of the patients presenting with facial fractures, with a significant proportion of these attributable to young adult males involved in IPV.

There was a difference in the fracture pattern between the alcohol and non-alcohol group, with the mandible more commonly targeted, possibly due to its prominent anatomical position. Mechanism of injuries was also different for the two groups, with IPV the most frequent cause of alcohol-related fractures and sports implicated as the major cause of non-alcohol related fractures.

Overall, more than half of patients required hospitalisation. There is a statistically significant difference in the proportion of hospitalisation between the alcohol and non-alcohol group. Hospital stay is usually brief due to a combination of the changing demographics of patients (young, healthy males presenting with simple fractures due to interpersonal violence) and swift return to function after open reduction and internal fixation of the fractures using direct miniplates, rather than previously longer convalescence with the use of intermaxillary fixation.

Studies have previously shown a strong link between excess alcohol consumption, IPV, MVAs, and serious facial injury,[1–8] with a third of these patients having evidence of legal intoxication at the time of MVA or assault.[2]

Alcohol not only alters perception and has an important role in aggressive behaviour; it also makes the person vulnerable to attack.[12] A proportion may be sober and innocent bystanders who are caught up in the actions of others.

In New Zealand, the minimum age required to purchase and consume alcohol in public was lowered from age 20 to age 18 in 1999. There have been various groups such as the Salvation Army and Alcohol Healthwatch strongly lobbying government to reinstate the older minimum drinking age.

The lowering of legal drinking age was originally aimed to improve binge-drinking culture among the young, with the ultimate goal to encourage mature drinking.[13] However, according to the Salvation Army, a major provider of support service for alcohol and drug addiction, this regulation has led to wider accessibility of alcohol, worsened the binge drinking culture, and has resulted in (more) physical and emotional harm to teenagers.[13]

Oral and Maxillofacial Surgery as a leading specialty treating maxillofacial trauma should also be helping to educate the public on alcohol related facial trauma. An example of a public awareness forum in prevention led by Oral and Maxillofacial Surgeons was the BAOMS (British Association of Oral and Maxillofacial Surgeons) UK Facial Injuries Awareness Week.[2] This forum targeted the younger population through lectures, brochures, and other educational aids. A total of 200 Oral and Maxillofacial surgeons were involved in visiting schools bearing the message “Save your face – drink sensibly”. Targeting 13-14 year olds and educating them on the relationship between excess alcohol consumption and interpersonal violence through the long-term effects of facial injuries, proved to be most effective strategies.

Other campaigns may be effective in educating the public such as television advertisements discouraging drink driving. Facial injuries are the most common trauma in MVA, with facial bone fractures in 22% of these patients,[5] and such interventional strategies may be effective in reducing MVA-related facial fractures. In this study, the proportion of alcohol-related fractures secondary to MVAs (13%) is far less than the proportion secondary to IPV (78%).

Facial injuries not only may cause permanent physical disfigurement but also chronic psychological sequelae such as long-term post-traumatic stress disorder. About 30% of adults with a facial fracture or a facial laceration more than 3-cm long develop post-traumatic stress disorder.[14] Such facial injuries can have profound effect on the person’s psychological and emotional well being. It can seriously impair a person’s social and emotional functioning and render loss in economic terms.[10]

Alcohol is an important element in urban violence and health professionals, such as Oral and Maxillofacial Surgeons, have been involved in educating the public.[15–17] Any programme aimed to reduce excessive drinking will be difficult, due to social and cultural barriers, especially as alcohol is so socially accessible and acceptable.

Various interventional programmes have been recommended, for instance a proportion of young men changing their alcohol-drinking pattern following nurse-led

intervention after alcohol-related facial injuries.[18] Such programmes should involve health professionals, government, and community groups, but will rely on acceptance by affected individuals and their families.

Oral and Maxillofacial Surgeons as a professional body can be involved in pushing for certain changes of legislations to minimise alcohol-related facial fractures. One such strategy may be to change taxes to raise the price of alcoholic beverages which can decrease the level of alcohol consumption.[19] Another strategy may be to target pub and restaurant operators to be vigilant in not serving alcohol to intoxicated or underage people.[19]

Reinstating the minimum purchasing age for alcohol to 20 years may reduce the number of facial fractures as one study has shown an increase in alcohol-related traffic crashes in the younger age group after 1999.[20]

Conclusion

Alcohol-related trauma is a major social and economical burden that requires effective use of hospital resources and patient interventional strategies. The maxillofacial region is the most common target in assault-related injuries and there is a strong link between alcohol consumption and these injuries. Indeed, the noted decrease in motor vehicle accident-related facial injuries has been offset by the large increase in alcohol-related interpersonal violence.

The majority of alcohol-related facial fractures were due to interpersonal violence in our Unit. The demographics reflect that young men are mainly affected and there is a statistically significant correlation between alcohol-related trauma and hospitalisation. Oral and Maxillofacial surgeons have an important role in preventive education as well as treatment of alcohol-related facial injuries.

Competing interests: None known.

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Under-18 year old callers to New Zealand's Quitline

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Aim To investigate the characteristics of under-18 year old callers to New Zealand's Quitline (smoking-cessation telephone counselling service).

Methods Analysis of routinely collected demographic and smoking history characteristics of under-18 year old Quitline callers in 2004 and 2005.

Results In the 24 months of 2004–2005, 2371 under-18s called Quitline (for the first time) seeking smoking cessation support. Females (58.9%) and teens in their older teen years called most often. Compared with adult callers, there were significantly higher proportions of Māori (32.9% vs 19.6%) and Pacific (5% vs 3.6%) under-18 callers, and fewer European (64.0% vs 74.6%) and 'Other' (6.0% vs 7.1%) callers. Despite similar levels of nicotine dependence in under-18 and adult callers (70.1% vs 71.4% reported smoking within 30 minutes of waking), under-18s were issued nicotine replacement therapy (NRT) half as often (RR=0.51). Under-18s were more likely than adults to register a mobile phone number (48.9% vs 44.4%).

Conclusions Under-18 year old smokers are under-represented in the Quitline calling population. Māori and Pacific under-18s require further cessation support to avoid exacerbating existing disparities in smoking. Awareness that under-18 nicotine dependence is equivalent to that of adults should lead to improved provision of NRT for adolescents. Initiatives involving mobile phone technology are particularly appropriate for improving access to information and treatment for under-18s. Adolescent tobacco cessation should be accorded greater priority in tobacco control policy, practice, and research.

Tobacco smoking continues to be a major public health problem in New Zealand (NZ); almost a quarter of adults (15–64 years) are smokers,[1] and half of those who smoke will die prematurely from smoking-related conditions.[2]

Preventing tobacco smoking initiation at an early age is a key strategy in addressing tobacco use because over 80% of smokers begin smoking before the age of 18 years.[3] Between one-third and one-half of those who experiment with tobacco become regular smokers.[4] Currently 26.8% of 15–19 year old NZers smoke[1] and most want to quit: over half report making a cessation attempt in the past year.[5,6]

If they could start their lives again, 72.3% of young NZ smokers surveyed would not smoke.[7] However, young people's quit attempts are frequently unsuccessful, with the few experimental trials published yielding unassisted 3- to 6-month quit rates of around 0–11%.[8]

Cessation interventions specific to young people were largely overlooked in the literature until the mid-1990s due to a focus on preventing smoking initiation in this age demographic, and a recent Cochrane review concluded that evidence around tobacco cessation interventions for young people is still lacking.[9]

Cessation initiatives require better information about the characteristics of adolescents who seek help, so that appropriate interventions can be developed and adolescents not accessing cessation services identified. In this study we analysed the demographic and smoking characteristics of Quitline callers under the age of 18 years. Quitline is New Zealand's largest smoking cessation service, providing free and comprehensive information and advice on smoking cessation 6 days a week to over 30,000 callers each year, with a call-back service where Quit advisors provide ongoing support. In addition, Quit advisors send out exchange cards to eligible callers that enable heavily subsidised nicotine replacement therapy (NRT) patches and/or gum to be purchased.

Methods

Quitline routinely collects demographic and basic smoking data from all registered callers. We extracted data from Quitline for all first-time under-18 callers in 2004 and 2005 as part of a larger study analysing the effect on Quitline of the *Smokefree Environments Amendment Act* which came into force in December 2004. To enable comparative analysis, a population of 2000 randomly-selected 18-and-over (adult) first-time callers to Quitline was generated using a computerised random number list from the total of 61,387 18-and-over callers in the same time period.

The variables used for analysis were identified by examining the Quitline database fields for personal or programme factors that have been shown in the literature to affect cessation. Anonymity was maintained by excluding variables with identifying information. Three fields relating to previous cessation attempts were not in an extractable format for the time period chosen, and therefore could not be used.

Final variables analysed for this paper were: age, sex, ethnicity, type of contact phone number, time from waking to first tobacco (an indication of nicotine dependence[10]), and whether a NRT exchange card was issued.

Ethnicity total response coding[11] was used, with a combined variable 'Others' for Asian, Latin American, Middle Eastern, African, Other and Refused/Don't know responses, due to the small number of individuals in these categories.

Data were analysed using Intercooled Stata (version 9) software.[12] Measures of precision were not calculable for the under-18 observations because the dataset represents the full population of under-18 Quitline callers in the time period. Where possible, 95% confidence intervals (CIs) were calculated for adult callers and assessment of statistically significant differences between under-18 and adult callers determined by comparing the under-18 figure with the 95% CI for the adult population. Chi-squared results are noted as a test of independence, where applicable. Where there were missing values, the observation was excluded from analyses involving that variable, and this accounts for small differences in denominators between variables.

Results

Demographic characteristics—From 1 January 2004 to 31 December 2005, 2371 under-18s called Quitline. Callers ranged in age from 10 to 17 years, with more callers with each additional year of age (Figure 1).

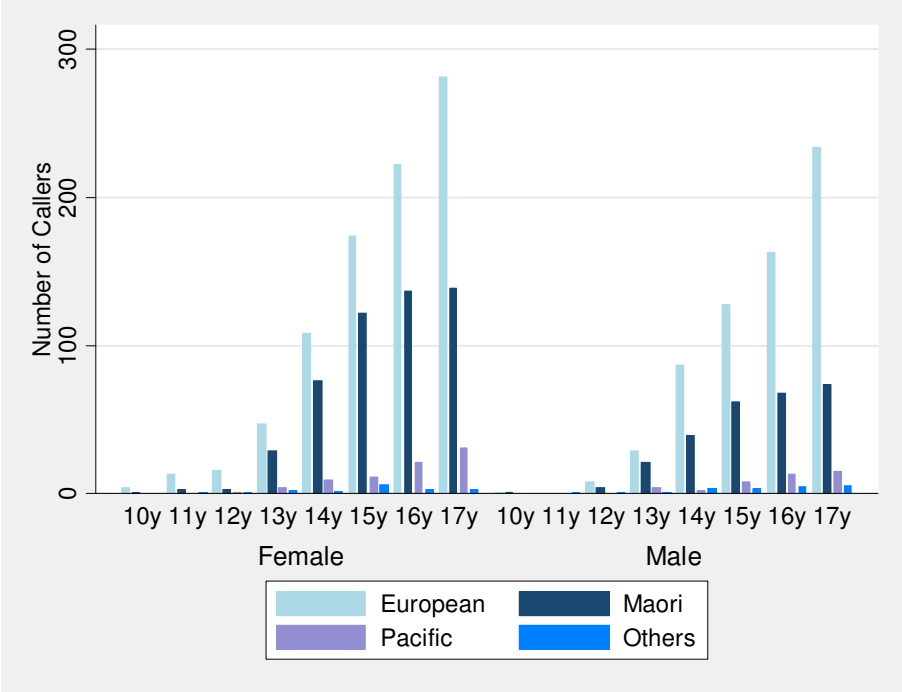
More females than males called Quitline in both under-18 and adult populations although there were proportionately more female callers among under-18s (58.9%) than adult (52.6%) callers (Chi-squared 17.37, $p<0.001$) (Table 1).

Table 1. Callers to Quitline, by age group and gender

Gender	Under 18s (%)	Adults (%; 95% CI)	Total (%)
Male	969 (40.2)	941 (47.5; 45.2–49.7)	1910 (44.0)
Female	1386 (58.9)	1042 (52.6; 50.3–54.7)	2428 (56.0)
Total	**2355**	**1983**	**4338**

Compared to adult callers, under-18 callers were more likely to be of Māori or Pacific ethnicity (Table 2). Within the under-18 calling population, male and female European callers increased with each year of age. This trend attenuated with age among Māori callers, even though the absolute number of Māori male and female callers increased with each year of age, and began a lot earlier for Māori males than females (Figure 1).

Figure 1. Under-18 Quitline callers 2004–2005 by age, gender, and ethnic group



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callers increased with each year of age, and began a lot earlier for Māori males than females (Figure 1).

Table 2 Callers to Quitline, by age group and ethnicity

Ethnicity	Under-18s (%)	Adults (%; 95% CI)	Total (%)
European	1518 (64.0)	1491 (74.6; 72.6-76.4)	3009 (68.8)
Māori	781 (32.9)	392 (19.6; 17.9-21.5)	1173 (26.8)
Pacific*	119 (5.0)	72 (3.6; 2.8-4.5)	191 (4.4)
Other	143 (6.0)	141 (7.1; 6.0-8.3)	284 (6.5)
Total	**2371**	**2000**	**4371**

*Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Dependence—The proportion of under-18 callers who smoked tobacco within 30 minutes of waking was similar to that of adult callers (Chi-squared 0.59, p=0.44) (Table 3).

Table 3. Callers to Quitline, by age group and nicotine dependence

Time from waking to first cigarette	Under-18s (%)	Adults (%; 95% CI)	Total (%)
<30 minutes	975 (70.1)	1110 (71.4; 69.1–73.7)	2085 (70.8)
>30 minutes	415 (29.9)	444 (28.6; 26.3–30.9)	859 (29.2)
Total	**1390**	**1554**	**2944**

NRT exchange cards issued—Under-18s were half as likely to be issued an exchange card for NRT (RR=0.51). Under-18s who were issued NRT cards were significantly less likely to be issued a second card than adults (RR=0.27) (Table 4).

Table 4. NRT exchange cards issued, by age group

Number of NRT exchange cards issued	Under-18s (%)	Adults (%)	Total (%)
None	1710 (72.1)	520 (26.0)	2230 (51.0)
One	364 (15.4)	559 (30.0)	923 (21.1)
Two	297 (12.5)	921 (46.1)	1218 (27.9)
Total	**2371**	**2000**	**4371**

Mobile phone use—Nearly 49% of under-18 callers gave a mobile phone number as a contact number, compared to 44.4% of adult callers (Chi-squared=8.95, p=0.003).

Discussion

This is the first New Zealand study to focus on young smokers who want to quit. A comprehensive literature review did not identify any international studies that have researched the teenage subset of smokers who call quit lines.

Several findings were as expected: that older teens called Quitline more frequently than younger teens mirrors the increasing smoking rate with increasing age in teenagers,[13] and our finding that females called more than males reflects the trend seen in adult quit lines.[14,15]

Contrary to expectations, we found that Māori and Pacific callers comprised a greater proportion of the under-18 calling population than the proportions seen in the adult calling population. This is partly explained by the fact that Māori and Pacific people comprise greater proportions of the youth population (19.5% and 9.2% of 15–19 year olds respectively) than the total population (14.0% and 6.6% respectively).[16] However, we had hypothesised that under-18 Māori and Pacific callers would be under-represented compared to their adult counterparts because Quitline's Maori/Pacific-focused advertising campaigns are targeted at adults only (25–44 year olds).

Māori and Pacific under-18 callers were still under-represented compared to their proportions in the adolescent smoking population: recent Action on Smoking and Health (ASH) surveys of 14–15 year olds, show that 37.8% of respondents who smoked at least weekly were Māori, and 12.8% were of Pacific ethnic origin.[17]

The 2371 under-18s who called Quitline in 2004 and 2005 represent 3.9% of the total of 61,387 Quitline callers in this period, a not insignificant increase on the 1.6% of callers who were under 18 in an analysis of 2001–2004 calls.[18]

Li and Grigg's study of Quitline callers over a longer period shows a 67% increase in the proportion of callers under 25 years between 2001–2005.[19] There are several possible explanations for this increase.

Firstly, under-18s may have become more aware of Quitline through advertising and word-of-mouth since its launch in 1999. Although no advertising is targeted specifically at the teen age group, there is evidence from elsewhere that adult-focused anti-tobacco advertising has some impact on adolescents.[20]

Secondly, some evidence suggests that under-18s have experienced increasing difficulty with tobacco purchasing in more recent years.[21]

Thirdly, legislative change prevented smoking in most workplaces in 2004 (the New Zealand school leaving age is 16 years and more than half of school students aged 16 years have part-time jobs[22]). These latter factors may have contributed to an increasing number of young smokers wishing to quit.

While it is encouraging from a public health viewpoint that young smokers are increasingly calling Quitline, they are under-represented among first-time Quitline callers compared to the proportion of adult smokers who want to quit. Using 2006 Census[16] and Tobacco Use Survey[1] data we estimate that there are around 80,450 adolescent smokers in NZ (26.8% of the 300,198 15–19 year olds) and 742,680 adult smokers (23.5% of 3,160,371 adults).

Quitline recorded a yearly average of 1185 under-18 and 30,694 total first-time callers in 2004 and 2005, suggesting that only about 1.5% of adolescent smokers called Quitline in that period, compared to 4.1% of adult smokers.

The fact that young smokers are less likely to call Quitline than their adult counterparts partly reflects a lack of attention to the promotion of quitting in adolescents in policy, practice, and research. Tobacco control policies concerning adolescents almost exclusively pertain to preventing tobacco initiation. Regarding practice, one US survey reported that only a third of young people were counselled about the dangers of tobacco use when visiting a doctor, and just 16.4% of young smokers were given advice to quit.[23]

Brief advice from a physician is an effective cessation strategy in adults,[24] and combined with support, such as referral to Quitline or provision of NRT, may prompt a serious quit attempt in a young person.

Our research suggests that young smokers wanting to quit were not receiving equitable access to treatment such as NRT, despite having levels of nicotine dependence equivalent to that of adults. The reason for this may be licensing and national guideline restrictions that have, until recently, discouraged NRT-use in under-18s.

Accordingly Quitline policy required parental consent for exchange card issues to under-18s. Revised cessation guidelines support the consideration of NRT-use in 12–18 year old smokers[25] without parental consent, and training providers about this new guidance may go some way to overcoming the treatment gap. A further possible explanation of the difference in NRT provision between adults and under-18s may be a mistrust of young callers' reports of their dependence; or conversely, mistrust by young callers of the efficacy of NRT.

Publicity highlighting the efficacy of NRT[26] and the early onset of nicotine addiction[27] may help overcome this. Scragg et al's recent analysis of NZ ASH Year 10 Survey data[28] suggests that diminished autonomy can occur as early as after one cigarette.

Focus group research with adolescent smokers in North America has concluded that teen smokers are often not aware of cessation programmes, and that those who are aware have generally negative and false perceptions about them.[29]

To increase the number of young people successfully quitting smoking, priority should be given to increasing the number accessing cessation services for help with quit attempts.[30] Adolescents want cessation programmes to be voluntary, free of charge, confidential, and of proven efficacy[31] all characteristics of the Quitline that could be promoted specifically to teens to encourage them to make contact.

Imparting information about the early progression to nicotine dependence may help to increase the urgency[32] of tobacco cessation efforts, so that young people prioritise quit attempts. Recent research points to using TV and cigarette packets as the primary marketing methods for such messages.[17] The use of online marketing, through YouTube[33] or advertising space on other websites popular with young people, might also be effective although this remains understudied.

In addition to novel marketing ideas, innovative cessation initiatives show particular promise for young people. Groundbreaking New Zealand research showed that a mobile phone-based text message service improved quit rates.[34]

Young people are high users of mobile phones (over 90% of 15–24 year olds according to the 2006 Census[35]) and our research shows under-18s are more likely than adults to register a mobile phone number with Quitline. The reach and scope of any adolescent-specific cessation programmes based on mobile or web technology could be much broader, and alternatives to any face-to-face interventions warrant further research.

This study was limited in a number of ways. First, we used data collected for another purpose, thus some factors, such as socioeconomic position and quit rates, could not be studied. Second, missing observations prevented analysis of some variables. Finally, the generalisability of our findings to all young smokers may not be appropriate as those calling Quitline may differ in some way to other young smokers who want to quit.[36,37]

In summary, the adolescent smoking rate may be decreasing[1] but young adults have the highest smoking rate of any age group. Unless cessation treatments become more accessible and effective for young people, the current adolescent smoking population may become yet another generation of adult smokers. A focus on preventing tobacco initiation to the neglect of cessation interventions has indirectly marginalised those teens who are already smokers. To correct this imbalance, and prevent widening disparities arising as the population smoking rate decreases, adolescent tobacco cessation should be accorded greater priority in tobacco control policy, practice, and research.

Competing interests: None known.

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"If everyone does it, it's not a big deal." Young people talk about chlamydia testing

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Abstract

Aims This study aimed to explore young people's attitudes to chlamydia testing. Data were gathered to inform the development of a clinical trial aimed at increasing chlamydia testing among 16–24 year olds.

Methods Four single sex focus groups were conducted with 16–24 year old males and females (n=28), and one with health professionals working with this age-group (n=7). A semi-structured interview schedule was used to discuss barriers to chlamydia testing, methods of accessing testing, communicating information about chlamydia and ideas about ways to encourage testing.

Results Reasons for not seeking testing included fear, stigma, denial of personal risk, and a lack of knowledge about chlamydia and about testing procedures. Better education and a need to 'normalise' testing were suggested as ways to increase test-uptake. Preferences for places to seek testing varied among participants, but all groups supported routinely offered chlamydia testing when visiting the doctor for other reasons. Participants also favoured the concept of home-testing.

Conclusions Young people identified a number of barriers to chlamydia testing, as well as ways to increase testing. These findings can be used to inform the development of much needed new initiatives to control chlamydia in New Zealand.

Chlamydia trachomatis is the most commonly diagnosed sexually transmitted infection (STI) in New Zealand, and over 70% of cases are diagnosed in under 25 year olds.[1] Of those with chlamydia, an estimated 75% of females and 50% of males will have no symptoms.[2] Untreated chlamydia has serious public health consequences including pelvic inflammatory disease, ectopic pregnancy, and infertility.[3]

Screening apparently healthy, asymptomatic individuals is therefore required to detect and reduce the reservoir of infection. Opportunistic chlamydia testing involves offering tests to those already attending healthcare.[4] In New Zealand, the Sexual Health Society recommends testing for all under-25 year olds accessing healthcare,[5] and the Ministry of Health recommends opportunistic chlamydia testing for sexually active under-25 year-olds as well as women presenting for pregnancy testing, attending antenatal clinics, or seeking termination of pregnancy.[6]

In the absence of a national STI surveillance system, it is difficult to determine the extent to which these recommendations are implemented. Figures that are available suggest that testing rates are generally low: an audit of antenatal testing showed only 37.5% of pregnant women were tested between 1999 and 2003,[7] and selected laboratory surveillance data showed 71 cases of chlamydia were diagnosed in infants (<1 year old) in 2006.[1] In 2004, only 15% of women and 5% of men aged 15–24

attending Wellington general practices were tested for chlamydia (unpublished laboratory data).

Advances in laboratory techniques for chlamydia testing have allowed for greater flexibility in sample collection—pelvic examinations performed by trained clinicians and urethral swabs are no longer required. Urine and vaginal swabs (that can be self-collected) are suitable for the detection of chlamydia using nucleic acid amplification tests such as PCR.[8,9] These samples are non-invasive and easily collected by patients themselves,[10] so are ideally suited for use in screening asymptomatic patients.

An effective chlamydia control program is essential to prevent the serious health consequences of untreated chlamydia in New Zealand. Before any such program can be developed, it is important to consider the views of those most at risk for chlamydia—16 to 24 year olds. Focus groups were conducted to explore young people's views about the barriers and acceptability of chlamydia testing.

Method

Setting—Five focus groups were held in Wellington in July and August 2006. All focus groups were conducted in a single sex environment, lead by an experienced female member of the research team (the facilitator). Food and soft-drinks were provided at each of the one hour long meetings, and 16–24 year olds were given a music voucher for their participation. This study was approved by the Central Regional Ethics Committee (CEN/06/02/007).

Participants—One group of health professionals experienced in working with sexually active young people in high-risk settings (termination of pregnancy clinics, sexual health clinics, and school health clinics) were recruited through professional contacts of the research team. Two groups of 20–24 year olds were recruited via notices posted in public places around the city centre including cafes, university libraries, and student centres, and a public hospital. Notices had an email address, freephone number, and a mobile phone number that potential participants could text to receive a call from the study team. Meetings were held in the early evening at an after hours medical centre.

Two groups of 16–18 year olds were recruited from a decile-8 coeducational secondary school in cooperation with school administrators. Decile-1 schools are the 10% of schools with the highest proportion of students from low socioeconomic communities, whereas decile-10 schools are the 10% of schools with the lowest proportion of these students.[11]

Meetings were held during lunchtimes on consecutive days at the school. Posters were placed around the school several days before each of the two meetings. Four schools were approached regarding participation by their students in this study. A decile-10 boys school declined participation due to 'time constraints', and two coeducational (decile 1 and 2) schools while interested, did not reach a decision about participation within the time constraints of this project.

Interview guide—The research team developed a semi-structured set of interview questions to illicit information about:

- Perceived barriers to chlamydia testing,
- Factors that encourage testing,
- Places to access testing,
- Ways to communicate information about STIs, and
- Incentives for increasing testing and new ideas about how to encourage testing.

Participants were told they did not have to discuss their own experiences but were asked to think about the questions in relation to people in their age-group. Health professionals were asked to think about chlamydia from their experience working with young people in high-risk settings.

Data collection—Participants were greeted by the facilitator and another member of the research team and directed to read the two-page information sheet, sign the consent form, and complete the demographics questionnaire (age, 2001 NZ census ethnicity question, occupation). The information sheet outlined the purpose of the study, provided key facts about chlamydia, and included details of

local clinics should any participants wish to seek health advice or services following participation in the meeting.

The facilitator explained that the audiotapes would be kept completely confidential and that names or other personally identifying information would not be used in the transcription.

Analysis—Focus groups were transcribed verbatim by the facilitator. The transcribed data were entered into a Microsoft Excel spreadsheet and comments were coded by another member of the research team. Initial categories were based on the original interview topics. These were further categorised into sub-topics based on participants' responses, and then again into more defined descriptors of the content of statements. Coding of the data was jointly discussed by two members of the research team and consensus reached regarding the interpretation of participants comments. Quotes illustrating agreement and/or disagreement about themes and ideas raised were then jointly selected.

Results

Table 1 presents the demographic characteristics of participants. The median age of young people was 18 years (SD 2.3). Key themes and illustrative quotes are presented in tables 2-5. Saturation of ideas occurred quickly; similar themes emerged from the four groups of young people.

Table 1. Characteristics of participants in five focus groups

Group	n	Median age (range)	Ethnicity	Occupation
Males (n=14)	8	17 (16–18)	7 NZ European (NZEu)	7 Secondary school students
	6	22 (20–22)	3 NZEu 1 NZEu & Maori 1 Indian 1 Chinese	5 Full-time tertiary students 1 Unemployed
Females (n=14)	7	17 (17–18)	7 NZEu	7 Secondary school students
	7	21 (20–24)	6 NZEu 1 NZEu & Maori	4 Full-time tertiary students 1 Full-time employment 1 Part-time tertiary & employed 1 Unemployed
Female health professionals (HP)	7	n/a	n/a	3 Social workers 3 Nurses 1 Nurse practitioner

Barriers to testing (Table 2)—Table 2 presents the barriers to testing identified by participants with illustrative quotes. All groups identified lack of awareness and understanding about chlamydia as a major reason for not actively seeking testing, including lack of knowledge about the prevalence, testing procedures, and asymptomatic presentation of chlamydia.

The teenage male participants admitted they were unaware of the urine test for chlamydia (they thought it involved a swab from the penis), and one male thought a blood test was required. Discomfort discussing sexuality was suggested as a factor that prevents both patients and providers from discussing STIs. Stigma, fear, denial,

or feelings of invincibility were also cited as reasons for not getting tested (particularly for males).

Participants in both male and female groups raised 'being male' as a factor for not being tested, and suggested that males are less likely to see a healthcare provider, and more likely to deny they are at risk. Most factors identified by health professionals as barriers to testing among young people were also raised by young people themselves. Health professionals raised additional factors such as access to clinics (convenience, location, closeness to bus routes), lighting, and opening hours that were not identified by young people as potential barriers to testing.

Table 2: Barriers to testing for young people with illustrative quotes

Barriers	Quotes
Lack of information and education	"It's partly because of lack of information. I reckon. Cause there's not much about it around school and stuff. Or you learn about it in health class in third form but … who actually listens." (F:17–18) "I also think that people don't realise the long term impacts of getting an STD, for example, chlamydia. Like as <name> was saying…. pelvic disease and stuff. But I mean, the general population, I don't think they know that at all." (M:20–24) "A lot have never heard of chlamydia that I see. It gets picked up when they're tested, when they have their swabs done and they are absolutely shocked. 'I've never heard of it.' And then they think they've got AIDS or it's some other thing so you have to work back from that. But it's lack of information and I think that risk taking." (HP)
Testing and treatment procedures	"You think STD, and you think the test is going to be nasty." (M:16–18) "Yeah like needles and stuff" (M:16–18) *On testing for males* "I know someone who's gone through that and it's apparently very painful." (F:20–24) "…there's a big misconception about treatment. Some people think it's invasive procedures involved when treating STI's and stuff and that's not true at all." (M:20–24)
Costs	"Cost to the doctor, embarrassment, stigma." (M:20–24) "….like young ones who are on a tight budget, working part time and partying. They would much rather spend that money on alcohol than they are to go to the doctors to get tested for something because they slept with a random person..." (F:20–24).
Stigma **Denial** **Fear**	"I think people are just shy. They don't want to go to their doctor and go and say 'I possibly have chlamydia' because then they will think that the doctor's thinking something about them." (F:17–18) "It's not something that's gonna happen to you because you're awesome. And only not-awesome people get chlamydia." (M:16–18) "One is the fear of it, in case it is. You know? And if you don't know about it then you…you don't have to worry about it." (F:20–24) "I'm bullet proof, it won't happen to me." (HP)
Gender	"Sexual health seems always, it's always seen as a really female domain." (F:20–24) "Being a male. Don't worry about it, it might go away." (M:20-24) "I think young 16 year-old girls are very scared to go to any male doctors. So I don't know, school clinics, having a female nurse. I don't think a 16 year old will happily go to a male GP." (HP)

Mechanisms for encouraging testing among young people (Table 3)—Table 3 presents the suggestions about factors that encourage testing with illustrative quotes. All groups suggested routine testing by GPs during check-ups or smear tests as an ideal way to normalise testing. It was suggested that the stigma attached to having a test could be removed if doctors took the initiative to offer testing, rather than requiring the patient to ask.

Participants in two groups (one male, one female) suggested that chlamydia testing be made "mandatory," and other members of the groups agreed with this. Analogies were made to routine vaccinations and visits to the dentist where reminders are sent out to say "it's time for your check-up."

Holding a competition or offering incentives to get tested were ideas suggested by the facilitator that were not generally well supported by participants. Overall, males (in both age groups) were more likely to favour incentives (competitions, prizes) than females. Male and female participants suggested engaging popular athletes as spokesmen, offering education in single-sex classes and promoting competitions with prizes (males suggested a car) as strategies to increase testing among males.

Table 3. Suggestions about how to encourage testing with illustrative quotes

Mechanism	Quotes
Offer testing routinely as part of primary care **'Normalise' it**	"People should go and get tested routinely because if everyone does it it's not a big deal" (M:16–19) "Like if they just said everyone will do a basic health checkup once a year. And that would involve tests for everything, then they don't have to even say that I'm sexually active or I'm this. Cause you know everyone's getting done." (F:17–18) "… and also the health professionals saying 'should we just do this too while you're here?' (F:20–24) "At the medical centre, if you put a notice, like chlamydia is a normal test. Just normalise it and put a notice on the notice board or on the counter. We suggest they have the test, direct communication." (HP)
Ease	"I think probably the most important thing that needs to be got out is the whole it's easy just pee in a cup." (F:20–24)
Education	"I think it's gotta be education. Like, you know, just everywhere and also making it, like, everyone gets tested. Not just a few people." (F:17–18)
Incentives Competitions	*Agree:* "I'd get it done, if I got a prize." (F:17–18) "Instead of using advertising and TV, which is going to cost you loads, you buy one car. And you'll get every male I guarantee you. (M:20–24) *Disagree:* "I don't think so. I think in a way that's almost, like, kind of glamorizing it." (F:20–24) "Personally I'd hope that people would take their sexual health more seriously than 'oh if I do it I can win a competition'." (F:20–24)
Advertising (TV, magazines)	"TV's probably the best medium to get across as long as it's not disgusting because people will just change the channel or look away." (M:16–18) "Yeah. You could do it (advertising) during the rugby for the guys." (F:20–24) "Maybe we should write a chlamydia ad for Shortland Street." (F:20–24) "What about advertising in like Girlfriend magazine?" (F:20–24)

Accessing testing (Table 4)—Views about appropriate settings for STI testing and counselling are presented in Table 4. Other people's perceptions or knowledge about why you're attending a clinic was a concern raised by many. For this reason there was agreement that GPs were a good place because "you could be going about anything."

Table 4. Ideas about places to access testing with illustrative quotes

Places	Quotes
General Practitioner	*Agree:* "I think doctors should say something to you. You know? Actually give you the idea to do it. Like remind you like they do for dentist checks. I mean, you know, like they send you a postcard telling you and I feel bad". (F:20-24) *Disagree:* "I don't know. I'd rather go to a sexual health clinic. I've known my GP forever. It would be quite weird." (M:16-18)
Sexual Health Clinic	*Agree:* "Like if you went to a STI clinic or something like that, you wouldn't feel as bad about it. Because you know that they see people with this stuff all the time." (M:16-18) "I like the STD clinic because I think kids will get more information, they'll feel more comfortable. I think they'll learn more from that setting." (HP) *Disagree:* "Nobody likes the idea of the stigma of walking into a sexual health clinic. Everyone who sees you walk in there will know why you're going in there." (M:20-24)
Youth Clinic	"I think that's supposed to be good, I haven't been there but. And it's not only sexual health but I think that's what a lot of young people go there for." (M:20-24)
Student Health	"You go into the room as if you going for a cough or a cold. You're going for a sore knee. But nobody knows what the hell you're going in for." (M:20-24)
School	"I think, yeah, to raise awareness it should be done at school" (F:17-18)
Home tests	"You would revolutionise STI testing if you could get a home test." (M:20-24) "And you can do it in your own time as well. You don't have to schedule an appointment or anything." (F:20-24)
Obtained from: Pharmacies	*Agree:* "The other thing is the stigma. Like, you go into a pharmacy, women usually are working at a pharmacy, like young girls, and you ask for a pot that's embarrassing, you're not going to do it." (M:20-24) *Disagree:* "I think people would rather pay money from the pharmacy than go ask their doctor." (F:17-18)
Obtained from: Website Telephone (hotline or texting)	"I think the website's quite a good idea. It's quite impersonal; you don't have to do anything. Just, this is my address, send me a test." (M:16-18) "And like the phones are way more, it's more, the web's not that trusted. Like you, I don't know, there's tons of dodgy stuff goes on the web so you wouldn't really." (F:17-18) "Like text into this number if you want one." (F:17-18) "90 percent of the young people have got a mobile so would get the message…. "…'chlamydia testing is free, get yours done now'…" (HP)

Some participants felt a family planning clinic or sexual health would be more private; others disagreed and said going to a sexual health clinic had the potential to make people feel stigmatised.

Participants responded favourably to the idea of home-test kits (whereby a person would mail in a urine or swab sample to a lab and receive results later). Males and females had different ideas about good places to obtain home-tests. Females liked the idea of obtaining a test from pharmacies, but males did not, especially if they cost

money and some admitted embarrassment would put them off asking pharmacy staff for a test.

Ordering a test via an online website was very popular with the 20–24 year olds, particularly the males. Females thought that more young people have mobile phones than computers, so thought accessing a test by texting was a good idea. The younger groups were concerned about having limited access to computers and lack of privacy regarding mail received at home. Younger females thought a telephone hotline was a better avenue for ordering tests.

Methods for communicating information about chlamydia testing (Table 5)— Table 5 presents the suggested methods of communicating information about STIs to young people. Most of the young people agreed that current sexual health education programmes in schools were inadequate, particularly with regard to the timing of information. Participants expressed the need for more follow-up sessions for older teenagers (16 and older).

Table 5. Communicating information about chlamydia testing and illustrative quotes

Method	Comments
Using a straightforward approach	"Don't try to try to make it cool. It's never going to be cool." (F:17–18) "But like scare people but without gruesome pictures or anything. Just saying maybe some facts about it." (M:16–18) "You do have to respect their ability to understand, and not necessarily play into how you perceive kids to be. I think they do appreciate concrete information." (HP)
Education at school	"I just think, you know, they should spend more time educating us in school. Cause I've forgotten all the STIs and I've forgotten what their symptoms are." (M:16–18)
Pamphlets	"I think having someone coming in and giving pamphlets and stuff out." (F:17–18)
TV, radio	"Yeah, like through schools is always good just to teach people about you know, STIs and whatever. But also like a sensible, tasteful ad campaign on TV or radio." (M:16–18)
Website	"If there was a website, it would just be good to have all information on clinics and stuff and where they are." (M:16–18)

A straightforward approach was favoured to inform young people about STIs, their consequences, and how to get tested. Advertising on television, radio, or magazines were also suggested as ways in reach young people with information. One of the health professionals commented on the lack of information in the general media about safe sex and said "the other message that's out there all the time is that people in television and in the movies are having sex constantly and nobody, nobody is talking about condoms and STDs."

At the end of sessions with 16–24 year olds, participants were asked to each say what they thought the best way to reach people for testing was. Both groups of school-aged participants thought more education and publicity about chlamydia were essential. The older females thought being offered tests by the GP and access to tests from

pharmacies or via websites were good ideas. The older males thought getting the message across that chlamydia is often asymptomatic was key (television was suggested as the best mode of dissemination), and that access to free tests was also critical.

Discussion

Key findings in this study were:

- Young people want more information and education about chlamydia and STIs,
- Chlamydia testing should be 'normalised' and would be well received by young people if routinely offered by general practitioners, and
- Distributing tests that can be used at home may be a way to reach young people.

These findings might not reflect the views of all 16-24 year olds or health professionals in New Zealand. Within the focus groups, there was an under-representation of non-European participants and an over-representation of individuals in full-time tertiary study. Despite these limitations, many of the themes and ideas generated by participants in this study were similar to those raised by young people in previous studies overseas.[12–16]

The main barriers identified by young people were lack of information and education about STIs and chlamydia; misinformation about what getting tested involves; cost of seeking healthcare (and being unaware that free sexual health services exist); stigma, denial of risk, and fear of results; and a perception that sexual health is a female domain.

Greater efforts need to be made to disseminate the simple facts about chlamydia to young people: chlamydia is common, often asymptomatic, and perhaps most importantly, is easy to detect from non-invasive, self-collected samples. Free and low-cost sexual health services also need to be promoted to under 25-year olds, as many participants were unaware that sexual health consultations are free in some settings. The opportunity to obtain home-test kits appealed to participants in this study. Indeed, this approach has been trialled in overseas studies with some success,[17–19] and is currently being piloted in a small Wellington study.

To be effective, a chlamydia control strategy must have high participation rates;[20] capture the at-risk population in a suitable setting (e.g. clinic, school or home);[14] include males as well as females;[21,22] use the best possible test; and have systems in place for treatment of patients and their partners.[23,24]

Participants in this study agreed that being offered routine checks at the doctors (i.e. opportunistic testing) would be well received. But for this to succeed, the obstacles facing healthcare providers must also be addressed. Prior to the roll-out of a chlamydia screening program in the United Kingdom, researchers conducted focus groups with primary care doctors and nurses to seek their views on opportunistic testing.

Lack of time; financial incentives; knowledge about the benefits of chlamydia screening; knowledge about when and how to take specimens; worries about discussing chlamydia during an unrelated consultation; implications of positive results; and issues around contact tracing were all raised as factors prohibiting testing.[25] Similar issues are likely to face primary healthcare professionals in New Zealand.

New initiatives are urgently needed to address the chlamydia problem in New Zealand. The results of this study have been used to inform the development of a pilot study aimed at increasing testing among 16–24 year olds attending primary care. Further research is warranted to identify ways of overcoming barriers to testing. A resourced, multi-faceted approach to chlamydia control will have the greatest chance of success in New Zealand, and will depend on the extent to which barriers for patients and healthcare professionals can be overcome.

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Herpes simplex type 1 versus *Herpes simplex* type 2 in anogenital herpes; a 10 year study from the Waikato region of New Zealand

Erana Gray, Jane Morgan, Jennifer Lindeman

Abstract

Aim To determine the proportion of *Herpes simplex* type 1 vs *Herpes simplex* type 2, as a cause of anogenital herpes in the Waikato region. We specifically looked for changes in the proportions over time, and for gender or age group associations.

Method We undertook a retrospective data-analysis of all anogenital isolates positive for *Herpes simplex* types 1 or 2 in those ≥14 years of age, received at Waikato Hospital Laboratory (Hamilton, New Zealand) over a 10-year period. Around half way through the study period, the test type changed from viral culture to HSV nucleic acid amplification assay.

Results Between 1997 and 2006, 3933 anogenital isolates were positive for either *Herpes simplex* types 1 or 2. The proportion of type 1 vs type 2 oscillated around 30 to 40% over the 10 years, with no particular trend. Overall, 71% of positive isolates were from females, with 80% of the type 1 isolates from females. Presence of type 1 was inversely proportional to age, accounting for 53% of positive isolates in the <25 year age group, 30% in the 25–35 year group, and 26% in the >35 year group.

Conclusion *Herpes simplex* type 1 is a major contributor to anogenital herpes in the Waikato. Females accounted for the majority of both types and there was a much higher proportion of type 1 seen in the younger age groups. These findings have been reported overseas but this is the first reported New Zealand data.

Anogenital herpes is a common condition in those who are sexually active and may cause serious physical and psychological morbidity.[1,2] Worldwide, evidence varies as to relative proportions of anogenital *Herpes simplex* type 1 (HSV-1) and type 2 (HSV-2) infections but most report a high[3–5] or an increasing[6–8] proportion of HSV-1, with few reporting very low rates.[9]

Reasons for the seemingly increasing prevalence of anogenital HSV-1 are unclear. It is suggested to reflect lower rates of childhood HSV-1 infections or possibly changing sexual practices in regard to orogenital contact. There may even be publishing bias, given the traditional role of HSV-2.

The proportion of either *Herpes simplex* type 1 or type 2 is important as this impacts on the clinical manifestations of symptomatic disease, on acquisition of further infection and also on the provision of a protective vaccine. New Zealand (NZ) HSV-1 seroprevalence data is lacking and there is no NZ viral genotype data on the relevant proportions of diagnosed anogenital herpes.

The aim of this study was to assess the relative proportions of HSV-1 vs HSV-2 amongst anogenital isolates over a 10-year period in the Waikato region and to look

specifically for changes in the proportions over time, and for gender or age group associations.

Methods

Data was obtained from all anogenital isolates positive for *Herpes simplex* between 1 January 1997 and 31 December 2006 at the Waikato Hospital Laboratory from patients aged 14 years or older. Waikato Hospital laboratory processes all the genotype (and serology) samples for the whole of Waikato District Health Board (WDHB), including hospital and community settings, although direct immuno-fluorescence of smears is available at community-based laboratories.

Lesions were sampled using commercial viral transport swabs, mainly Virocult® (Medical Wire and Equipment Ltd, Bath, England). The swab was transported to the lab as soon as possible or stored at 4°C. Samples were then cultured by inoculation into each of two cell lines—HF (Human Fibroblasts from foreskin) and A549 (Human Lung Carcinoma). The cell cultures were incubated at 37°C for 5–7 days and examined daily for typical HSV cytopathic effects.

Positive isolates were typed using type-specific monoclonal direct fluorescent antibody (Syva Microtrak HSV 1 and 2 IF reagent). From 20 September 2002, samples were obtained and transported in the same way, but analysed by HSV nucleic acid amplification (NAA) assay. DNA was extracted using an ultra-centrifugation/lysis method followed by Real-time PCR and genotyping using melting curve analysis on the Light cycler 1.2.

The year the sample was obtained, the genotype, and the patient age and gender were all recorded. Samples were excluded if any of this data was missing or if a genotype was not determined. Data was de-duplicated using identity so that multiple swabs or any later recurrences of the same virus type were excluded.

Statistics New Zealand census population data for WDHB from 1996 and 2006 was obtained.

Microsoft Excel pivot tables and charts were used to analyse the proportion of HSV-1 vs HSV-2 overall and to look for trends over time and for gender and age group associations. P values were obtained using GraphPad Software.[10]

Ethical approval was obtained from the NZ Health and Disability Ethics Committee.

Results

During the study period, 4387 samples were positive for *Herpes simplex* virus (HSV). A total of 182 were excluded because of missing data; 21 because of missing demographics (16 had no date of birth, 5 had no gender); 11 because the swab site was not given; and 150 because the virus was not typed. These 150 specimens had all been analysed by viral culture, rather than NAA assay. A further 272 isolates were duplicates and also excluded.

3933 positive anogenital samples remained, comprising 1649 culture-positive and 2284 NAA-positive. The yearly number of positive isolates increased markedly over the 10 years (Figure 1). Based on NZ census data, the resident WDHB population increased by only 8.5% over a similar 10-year time period, i.e. between the 1996 and 2006 censuses.[11] The laboratory data was available as positive test results only, the proportion of people with a positive test could not therefore be calculated.

Overall, HSV-1 accounted for a total of 37% of the positive isolates, over the 10-year study period. The proportion of HSV-1 did not alter when testing methodologies changed in 2002, with 35% HSV-1 isolates by viral culture and 38% HSV-1 isolates by NAA (p value=0.12, not significant). The annual proportion of HSV-1 oscillated between 25% and 42% of the total isolates with no particular trend over time. (Figure 2)

Figure 1. Total HSV isolates by year and gender

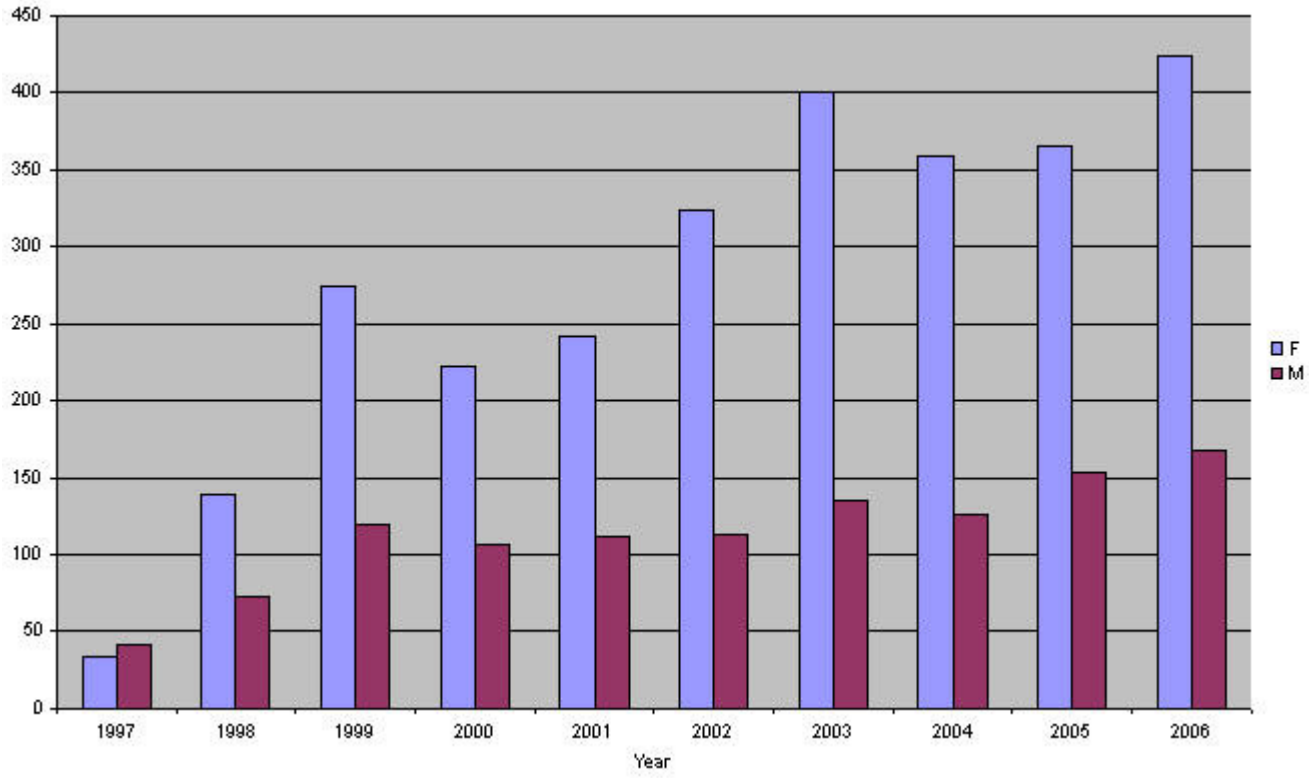
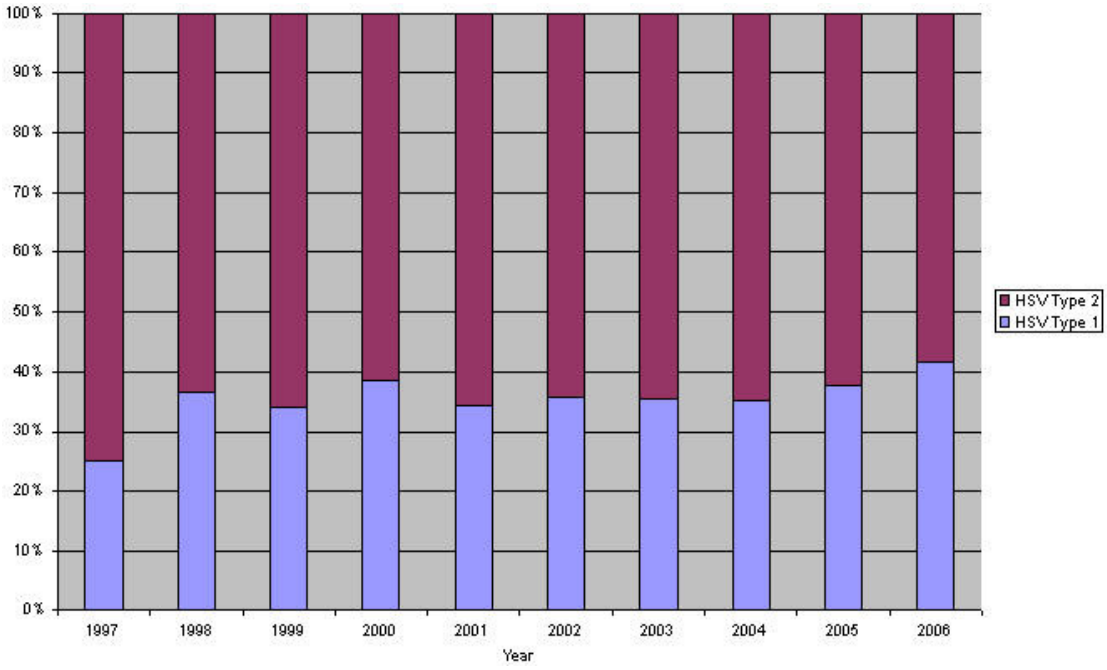


Figure 2. Proportion of HSV type by year



More HSV positive isolates were from those who were younger and female. Women accounted for 71% of positive isolates overall, with 80% of the HSV-1 isolates and 65% of HSV-2. Those under 25 years accounted for 34% of all positive isolates and cumulatively 82% of positive isolates were found in those under 45 years of age. The proportion of HSV-1 isolates was inversely related to age, making up more than half (53%) of the HSV in the under 25 year age group, decreasing to around a quarter (26%) in the over 35 year group. All p-values comparing the proportion of HSV-1 vs HSV-2 between the age groups were <0.01 (Table 1).

Table 1. Isolation of HSV by age and genotype

	AGE GROUP, *n* (%)			
	<25	25-35	35 and over	Totals
HSV Type 1, *n* (%)	717 (53)	374 (30)	345 (26)	1436
HSV Type 2, *n* (%)	635 (47)	858 (70)	1004 (74)	2497
Totals	1352	1232	1349	3933

<25yrs vs 25-35yrs, p value <0.0001

<25yrs vs 35 and over, p value <0.0001

25-35yrs vs 35 and over, p value <0.007

Discussion

Our study shows that for at least a decade, HSV-1 has accounted for a substantial proportion of diagnosed anogenital herpes in the Waikato region. Young age was strongly related to likelihood of HSV-1 and, although the numbers are small, this effect persisted over the study period (numbers not presented). Most of the diagnosed anogenital herpes in WDHB occurred in women and again this effect persisted over the 10 years (Figure 1).

Many centres published data throughout the 1980s and onwards, firmly dispelling the traditional perception that HSV-2 was responsible for the majority of anogenital herpes.[3–8,12–14]

One of the first reports of HSV-1 predominance was a small study of 35 Japanese women in a predominantly O&G outpatients setting, where 7/13 typed isolates were HSV-1 positive.[12] Since then, many centres have documented rising rates of HSV-1. For example, in the US state of Wisconsin, HSV-1 rose from 31% to 78% in newly diagnosed infections in female college students from 1993 to 2001.[6] And in Bangkok, Thailand separate studies suggested a rise in HSV-1 from 1.6% in 1989,[15] 18.7% in 1994-1996,[16] to 57% in 1985-2004 (note this study included 22% with dual HSV-1 and HSV-2 infections).[8]

Closer to home, a laboratory-based study from Sydney, Australia found the proportion of HSV-1 isolates increased dramatically between 1979 and 2003, from <10% in the early 80s to around 35% in the late 90s and early 2000s.[7] Whilst our data shows that rates in NZ have been relatively stable for the last decade and do not appear to be

trending upward, our rates do reflect the proportion found in Sydney in more recent years.

Interestingly, an inner London genitourinary medicine clinic is one of the few centres to report low rates of HSV-1, with only 6.5% of first infections, as recently as 2005.[9] This may highlight the importance of bias towards publishing only where the results refute a traditional belief, rather than support it.

Locations diverse in geography, ethnicity, and sociopolitical background have reported the importance of HSV-1 as a cause of anogenital herpes. In addition to those mentioned above, Tel Aviv (Israel),[3] Edinburgh (Scotland),[4] Newcastle (UK),[13] and Kentucky (US)[5] have all reported HSV-1 proportions ≥30%.

Several studies, in addition to ours, have demonstrated an inverse relationship between rates of HSV-1 and the age of patients.[3,7,14] Although not well studied, it is often suggested young adults becoming sexually active are at greater risk of anogenital HSV-1, because of a greater likelihood they will be seronegative for HSV-1, than in previous generations. However, the HSV-1 seropositivity in a subgroup of 12–19 year olds in a large population-based US survey, *increased* slightly between the 1976–80 and the 1988–1994 cohorts[17] (41 up to 45% seropositive, age-adjusted rates) and only moderately decreased in the 14–19 year olds in the 1999–2004 cohort (39%).[18] In a UK survey of 10–14 year olds, HSV-1 seroprevalence did decrease, from 34% to 24% over 1986–7 and 1994–5.[19]

Another often-suggested explanation is the popularity of orogenital sex among teenagers, who may favour this practice to avoid both pregnancy and sexually transmitted infections. Again, the association with orogenital sex has not been extensively studied, but one study found an Odds Ratio of 3.1 for a first episode of genital herpes being HSV-1 not HSV-2, with receptive oral sex in the 2 months before diagnosis.[20]

Worldwide, there is evidence that anogenital infections with both HSV-1 and HSV-2 are commoner in women. The evidence for HSV-2 can most reliably be found in population-based seroprevalence data and large studies from the USA, UK, Germany, and Australia demonstrate consistently higher rates of HSV-2 antibodies in women.[18,19,21,22]

For HSV-1, the seroprevalence data is harder to interpret, because of the impact of childhood orolabial herpes, nonetheless, HSV-1 data was also collected in the US, German, and Australian sero-surveys, and the rates of HSV-1 were again higher in females, by between 4–9%.[18,21,22]

Further evidence that HSV-1 is commoner in women, is seen in studies of symptomatic anogenital herpes, where the burden of anogenital HSV-1 does clearly fall on women;[7,14,23] but in this type of study, the numbers may be impacted by health-seeking behaviour.

Data available for NZ shows that women have higher HSV-2 seroprevalence rates than men, with 22.5% of women HSV-2 seropositive and only 14.6% of men seropositive by age 32 years in the Dunedin Cohort study.[24] Our study adds to this information by confirming higher rates of both HSV-1 and HSV-2 diagnosed in

Waikato women. We were not able to compare the Waikato rates to other areas of the country, as this is the first data of its kind for NZ.

Female susceptibility is possibly due to ease of acquisition by genital mucosa versus genital skin and by longer contact time with secretions. It is also possible HSV-1 rates in women are exaggerated because of differing health-seeking behaviour, especially in the case of HSV-1 when there may be only a single clinical episode and therefore a single opportunity for diagnosis.

Our data was de-duplicated, meaning that only a first diagnosis in WDHB was counted, with recurrences and multiple presentations excluded. All community and all hospital isolates for the whole DHB were studied. The study included nearly 4000 samples, so we feel confident that the HSV-1 proportion in the Waikato region is indeed significant at just over a third of all first diagnoses.

There are many limitations with a retrospective laboratory-based study; important correlates such as clinical presentation, sexual behaviour (e.g. orogenital sex in the incubation period), history of orolabial herpes, and other demographics (such as socioeconomic status and ethnicity) are not documented with the test results, and not available given the community-wide location of the requestors (all GPs, sexual health clinics, and hospitals in the area). Especially important confounding factors include the varying clinical features of HSV-1 vs HSV-2 that may affect the rate of presenting for healthcare.

For example, there is some (weak) evidence that primary infections with HSV-1 are more likely than HSV-2 to be symptomatic[25] and good evidence that HSV-1 leads to few, if any, clinically apparent reactivations[26] and that asymptomatic shedding is uncommon.[27] Therefore, if a patient acquires both infections, they may be more likely to receive a diagnosis of HSV-1 alone, if they present with their first symptoms, and more likely to be diagnosed with HSV-2 if they present later, during a recurrence.

Many infections remain asymptomatic and hence undiagnosed and would not be eligible for inclusion. A higher, and perhaps more representative, number of cases would be picked up in a seroprevalence study; however, as noted above, seroprevalence studies for HSV-1 also have limitations as they do not distinguish the site of infection.

This is the first NZ data that distinguishes rates of HSV-1 as a cause of anogenital herpes. This data will hopefully augment the currently available HSV-2 seroprevalence data by giving an indication, despite its clear limitations, of the proportion of confirmed anogenital herpes caused by HSV-1 in New Zealand.

Our results support the importance of typing viral isolates so as to provide optimal clinical care, as a substantial proportion of people will have HSV-1. In contrast to HSV-2, the natural history of HSV-1 is towards significantly fewer, if any, clinically apparent recurrences[26] and much less subclinical shedding.[27] It has been shown that prior HSV-1 infection does not alter the risk of acquisition of HSV-2, although it does attenuate the symptoms;[25,28] it is important for those diagnosed with HSV-1 anogenital herpes to understand that they remain at risk of HSV-2 infection.

To date, the only vaccine trial demonstrating efficacy against herpes was in a subgroup of women who were both HSV-1 and HSV-2 seronegative before

vaccination.[29] Our study demonstrates that such a vaccine would be suitable for Waikato preadolescents, as a high percentage are likely to be HSV-1 (and HSV-2) seronegative from childhood and hence at risk for both serotypes after first sexual exposure.

In a world where HSV-1 rates are strikingly dissimilar, our data finds a place for NZ, demonstrating that there is a significant proportion of anogenital herpes caused by HSV-1, but not a majority. Any HSV-2 seroprevalence data will significantly underestimate the true lifetime prevalence of anogenital herpes in New Zealand.

Routine typing of isolates enhances the clinician's ability to give prognostic information and optimal clinical care as it is no longer accurate to assume that anogenital herpes is due to HSV-2 infection.

Competing interests: None known.

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Walking to school: frequency and predictors among primary school children in Dunedin, New Zealand

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Abstract

Aim To estimate the frequency of walking to school among primary school children and examine associated factors.

Method In-class survey of Years 1–6 children attending Dunedin primary schools, November 2004, and a take home, written questionnaire for parents / caregivers.

Results On study day, 34.5% of children walked to school and 36.8% intended to walk home. Overall, 1157 completed caregiver questionnaires were returned (68%) indicating that 47.5% of children walked to or from school less than three times per week. The strongest predictor of walking was proximity to school (<1 km OR 29.3, 1–2 km OR 7.7, 2–3 km OR 3.0, >3 km OR 1.00). Other predictors were not having a car in the household (OR 10.9), attending a (low socioeconomic) decile 2 to 4 school (2.35), having three or more adults in the household (1.85), being in a higher school year (1.72), having non-New Zealand European ethnicity (>1.41), having a parent who had walked to school (1.35), and being male (1.33).

Conclusions This study established a baseline for the percentage of Dunedin primary school children walking to school. Key potentially modifiable predictors of walking were proximity to school and not having a car in the household. These findings have implications for health, transport and educational policies.

Childhood obesity is a global problem that has increased dramatically over the last 25 years.[1–6] The New Zealand Children's Nutrition Survey shows that 21% of New Zealand children are overweight and 10% are obese.[7]

Childhood obesity has numerous effects on both individuals and communities. Health consequences include diseases of the cardiovascular, gastrointestinal, respiratory, and endocrine systems. Neurological, orthopaedic, and psychological sequelae are also possible.[8–11] The direct cost of obesity had been estimated to be between 2 and 7% of a country's annual health budget.[12]

Although the increase in childhood obesity is multifactorial,[10,11] declining participation in physical activity is a major contributor. Attractive sedentary alternatives have drawn children away from discretionary physical activity, while the need for incidental physical activity has been reduced by energy saving devices such as remote controls.[13–16]

Safety concerns are also restricting children's freedom to play and exercise outside the home.[17] In New Zealand, more than one-third of children fail to achieve the recommended level of physical activity.[18] Similar trends have been observed in other developed countries.[19,20]

Walking to school provides children with a regular and convenient form of physical activity. This study seeks to estimate the prevalence of walking to and from school among Dunedin primary school children and identify major predictors of this activity.

Methods

Sample selection—Schools were identified from the Ministry of Education database.[21] All 46 primary schools (Years 1–6 [primer 1 to standard 4]; ages 5–11) within a 10 km radius of the Dunedin city centre were contacted by telephone and invited to participate. Special schools (those catering solely for children with physical or intellectual disabilities) were excluded. Thirty-nine schools (85%) agreed to participate in the study.

Two year-levels were randomly selected at each participating school: one from Years 1–3 (age 5–8) and one from Years 4–6 (age 8–11). All children from the selected year levels at each school were surveyed by members of the research group. 'Model' classes (those with a mix of students from all year levels in one classroom) were excluded.

Procedures—Children were asked to raise their hands if they had either walked or been driven to school, or got to school by any other means of transportation on Tuesday 30 November 2004. Children were then asked how they expected to get home after school that day. In each case, the numbers of children were recorded on a data sheet. In addition, every child received a questionnaire to take home for their caregivers to complete and return to school. Extra copies were left for any children who were absent on the sampling day. The questionnaires were followed by reminder letters 5–6 days later. Completed questionnaires were collected from the schools approximately 10 days after distribution.

Instrument—The questionnaire requested demographic data and information about the number of days the child walked to/from school in a typical week. Questions included sex, age, and ethnicity of the child; number of adults in household; number of other children in household; distance from school (less than 1 km, 1–2 km, 2–3 km, more than 3 km); hours of physical activity per week (less than 1 hour, 1–2 hours, 2–3 hours, 4 hours or more); and whether the parent had walked to school as a child. Parents were asked to specify the number of days (out of 5) that the child walked to school and from school in a typical week.

Analysis—Data were analysed using SPSS 12.0 and SAS 8 software. A logistic regression model was used to predict walking on a given day/time with a random effect for schools (taking into account the cluster effects of schools) and accounting for the correlated nature of the 10 possible walking periods for each child.

Ethical approval—Departmental ethical approval was obtained, in accordance with the University of Otago Human Ethics Committee guidelines.

Results

On the sampling day, 1524 children were present and all participated in the classroom survey. Overall, 1703 questionnaires were distributed to caregivers; 1157 were returned, thus giving a response rate of 67.9%.

Proportion of children walking to school—From the classroom survey, 34.5% of children had walked to school and 36.8% anticipated walking home on the study day. Parents' reports of how often their child walked to school in a typical week are presented in Table 1.

Table 1. Number of walking episodes per week and percentages of children walking to or from school in a typical week (caregivers' reports)

Walking episodes per week	Number of children (percentage)
8 or more	327 (28.9)
3 to 7	266 (23.5)
1 or 2	101 (8.9)
None	436 (38.6)

Predictors of walking behaviour—A logistic regression model was used to predict walking on a given day/time with a random effect for schools (taking into account cluster effects of schools) and accounting for the correlated nature of the 10 possible walking periods for each child. Of the 1157 caregivers' reports, 1006 provided full data and were included in the model. The model included sex, school year group, school decile group, distance to school group, ethnicity, number of adults in household, number of cars relative to adults (no cars, fewer cars than adults, same or more cars than adults), number of other children in household, physical activity group, whether the parent walked to school as a child, and presence of a walking school bus at school.

All variables were statistically significant ($p<0.05$) except for the number of other children in the household and the existence of a walking school bus. These results are summarised in Table 2.

- The proportion of children walking dropped off sharply as distance from school increased. Those living within 1 km of school were almost 30 times more likely to walk than children living more than 3 km from school.
- Families without a car were 10 times as likely to walk as those with as many or more cars than adults in the household (OR 10.9, 95% CI 7.72–15.4).
- Boys were one-third more likely to walk than girls (OR 1.34, 95% CI 1.21–1.48).
- Children in Years 4 to 6 were 72% more likely to walk than younger children (OR 1.72, 95% CI 1.55–1.90).
- Those attending (socioeconomically disadvantaged) decile 2 to 4 schools were twice as likely to walk as those at higher decile schools (OR 2.35, 95% CI 1.34–4.13).
- Māori children and Pacific Island children were more likely to walk to school than NZ European children (ORs 1.51 and 2.69, respectively).

There was a U-shaped relationship between physical activity and the odds of children walking to school. Children who exercised for 2 to 4 hours per week were less likely to walk to school than those who exercised for less than 2 hours or more than 4 hours. Children with a parent who had walked to school as a child were 35% more likely to also walk (OR 1.35, 95% CI 1.16–1.58).

Table 2. Summary of logistic regression model for predictors of walking to school

Variable	Odds Ratio	95% CI
Gender		
Female	1	–
Male	1.33	1.21–1.48
School year		
1 to 3	1	–
4 to 6	1.72	1.55–1.90
Distance to school		
< 1 km	29.3	24.10–35.70
1 to 2 km	7.70	6.33–9.38
2 to 3 km	3.04	2.38–3.88
> 3 km	1	–
School decile		
2 to 4 (disadvantaged)	2.35	1.34–4.13
5 to 7	1	–
8 to 10 (advantaged)	1.05	0.66–1.69
Ethnicity		
NZ European	1	–
NZ Māori	1.51	1.28–1.79
Pacific Island	2.69	1.90–3.77
Other	1.41	1.18–1.69
Cars in household		
No cars	10.9	7.72–15.4
Fewer cars than adults	1.47	1.30–1.66
Equal or more cars than adults	1	
Adults in household		
1	1	–
2	0.97	0.83–1.12
3 or more	1.85	1.46–2.35
Other children in household		
0	1.02	0.85–1.22
1 or 2	0.98	0.85–1.14
3 or more	1	–
Weekly physical activity		
Less than 1 hour	1.53	1.18–1.99
1 to 2 hours	1.64	1.36–1.99
2 to 3 hours	1	–
3 to 4 hours	0.93	0.78–1.11
More than 4 hours	1.32	1.15–1.52
Did parent walk to school?		
Yes	1.35	1.16–1.58
No	1	–
WSB* at school?		
Yes	1.47	0.94–2.33
No	1	–

*Walking school bus (WSB) consisting of adult volunteers walking a set route at a given time, collecting children along the way.

Discussion

This study indicates that 47.5% of Dunedin primary school children walked to/from school less than three times a week. Surveys from other cities in NZ and overseas have reported between 21% and 69% of children walk to school.[22–26] However, comparison between studies is limited by area-specific variations that may impact on walking patterns. These include: climate, season, availability of public transport, topography, population density, and socioeconomic status.

Moreover, methodologies also vary considerably; sampling may include one or several schools, response rates differ, and the questionnaires themselves are often not comparable.

As with other studies in the US,[27] UK,[28] and Australia,[29] the distance from home to school had the greatest impact on whether the child walked to school. This finding may have implications for school zoning policies. Education policy in New Zealand allows children to attend the school of their choice with few restrictions.[21]

Recent closures of small schools may also encourage children to use motorised transport as they no longer have the option of attending a school within reasonable walking distance of their homes.

Boys were more likely to walk than girls. This may reflect a gender difference in activity levels[30] or safety concerns related to a perception that girls are more vulnerable than boys.

Children in Years 1 to 3 were less likely to walk than older children; this finding is also likely to be related to safety issues. Caregivers may be concerned that younger children have not fully developed the perceptual, cognitive, and motor skills to successfully avoid hazards that may be encountered on the journey to school.[31] Indeed, the New Zealand Police recommend that children in Years 1 and 2 should not cross roads alone and (if possible) should walk to school accompanied by someone older.[31]

Attendance at a low-decile school and lesser car ownership were independently associated with a higher probability of walking. Indeed, other studies have also reported an association between high walking rates and low levels of a number of socioeconomic indicators.[24] This finding is likely to reflect the relatively high cost of motorised transport currently further exacerbated by high fuel prices.

Walking rates were lower among NZ European children when compared to other ethnicities. Higher walking rates have been reported in children from non-English speaking backgrounds.[24] This is of interest when placed in the context of childhood obesity within New Zealand; Pacific Island, and Māori have much higher rates of childhood obesity,[8] yet appear to be participating in greater levels of incidental physical activity via walking to school.

There are several potential explanations for this apparent contradiction. First, the higher rate of incidental physical activity in this group may not be enough to outweigh other contributors to obesity, such as nutritional factors (e.g. consuming relatively large amounts of snacks containing high levels of fat and sugar).

Secondly, body mass index data were not collected, so we do not know whether individual Māori and Pacific Island children in our study were more overweight than their peers.

Finally, the observed effect may be due to confounding by socioeconomic status. School decile and car access were used as proxy indicators of socioeconomic status in this study, but these measures may be too crude to adequately control for socioeconomic factors.

Cooper et al[30] suggested that children who walk to school are more active throughout the day. However, this study has shown that a higher frequency of walking is associated with both the highest and lower physical activity groups.

A walking school bus (WSB) consists of adult volunteers who walk a set route at a given time, collecting children along the way.[32] This study showed that WSBs had no effect on the proportion of children walking to school. This may be due to relatively few WSB routes in Dunedin and too few children taking part in each bus.

Identified constraints on the success of a WSB program include lack of parent volunteers, loss of key enthusiasts on the school staff, and loss of novelty value.[33]

Conclusions

The increasing burden of obesity and the decline in physical activity have been recognised as major public health issues.[12] Walking to school provides children with a convenient and regular means of increasing energy expenditure.

Living a short distance from school was the strongest positive, potentially modifiable predictor for walking to school. Male gender, higher school year, non-NZ European ethnicity, and attending a low decile school were all associated with a significantly increased rate of walking.

Results from this study could inform and guide the development of health, transport, and education policies directed at increasing the proportion of children walking to school.

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Rheumatic fever diagnosis, management, and secondary prevention: a New Zealand guideline

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Abstract

Aim The National Heart Foundation of New Zealand, and the Cardiac Society of Australia and New Zealand (CSANZ) recently launched an evidence-based review and guideline entitled *New Zealand Guideline for Rheumatic Fever Diagnosis, Management, and Secondary Prevention*. This paper is a brief summary.

Method This Guideline was developed by a writing group comprising experts in the area. Relevant literature was identified and reviewed, and the Australian guideline for rheumatic fever and rheumatic heart disease was reviewed and adapted for the New Zealand context. A peer review and stakeholder consultation process followed the development of the draft document.

Results The final draft of the New Zealand guideline was endorsed by Te Hotu Manawa Māori, Pacific Islands Heart Beat, The Paediatric Society of New Zealand, and the Rheumatic Fever Trust of New Zealand—plus approved by a number of organisations including the Royal Australasian College of Physicians, the Australasian Society for Infectious Disease, the Pasifika Medical Association, and Te Ohu Rata o Aotearoa. Two subsequent New Zealand guidelines for rheumatic fever: *Sore Throat Management* and *Primary Prevention* are also in production. The complete guideline, and associated summary algorithms, can be downloaded from www.nhf.org.nz

Conclusion A New Zealand Guideline for Rheumatic Fever Diagnosis, Management and Secondary Prevention of Acute Rheumatic Fever should result in improved consistency in the approach to this disease, and reduced mortality and morbidity from acute rheumatic fever and rheumatic heart disease.

Acute rheumatic fever (ARF) is an illness caused by a reaction to a bacterial pharyngeal infection with group A streptococcus (GAS) which can lead to lasting damage to heart valves—mainly mitral and aortic valvulitis. This is known as rheumatic heart disease (RHD) and is an important cause of premature mortality. Almost all cases of RHD and associated deaths are preventable.[3]

In most affluent populations, ARF is now rare. By contrast, the highest documented rates in the world have been found in Māori and Pacific people in New Zealand, Aboriginal Australians, and those in Pacific Island nations.[4–6] Almost all cases and deaths occur in developing countries and RHD is the most frequent form of heart disease in children worldwide.[7] RHD is a significant cause of premature death in New Zealand.[8–10]

In the 1920s, surveys of school records in New Zealand determined an approximate annual school population incidence of ARF of 65 per 100,000.[4] From 1956 to 1973, the Wairoa College Study determined that the decline in incidence of ARF seen in other developed countries was not evident in New Zealand and those pockets of the country which experienced isolation and socioeconomic deprivation had significantly higher rates of both ARF and RHD.[11] From 1995 to 2000, around 100 cases of ARF were notified annually in New Zealand, with an incidence of 13.8 per 100,000 population in 5 to 14 year olds.[10]

In New Zealand, the rates of ARF in Māori and Pacific people (mostly of Samoan, Tongan, Niuean, or Cook Islands origin) have always been reported as significantly greater than those seen in non-Māori (i.e. European, Asian). For example, from 1949 to 1953 the reported incidence of ARF in Māori children (rates of greater than 1000 per 100,000) was 11 times that of the non-Māori population.[4] The age-specific annual notification rates for ARF between 1990 to 1995 for children aged 10 to 14 years was 77.7 per 100,000 for Pacific children, 30.4 per 100,000 for Māori children, and 1 per 100,000 for European children.[10]

As well as higher rates of initial ARF incidence, Māori, and Pacific people also have the highest rates of ARF recurrence. From 1973 to 1982 (prior to the introduction of systematic penicillin prophylaxis delivery) recurrence rates in Māori were 40% compared to 22% in non-Māori.[12] Data from the Auckland Rheumatic Fever Register shows that although the recurrence rates dropped significantly from 22% in the 1980s to 5.5% by 1999, all recurrences were in Māori and Pacific people.[13] It is therefore not surprising that Māori and Pacific people have much higher rates of carditis, RHD, and consequent heart failure, as the risk of these complications increases with each attack of ARF.

There is no evidence to support Māori and Pacific people having an increased genetic susceptibility to rheumatic fever. It is more likely that their increased burden reflects social, political, and economic influences that result in overcrowded conditions, socioeconomic deprivation, an increased incidence of upper respiratory infections with GAS, and different options or opportunities for appropriate and effective health care.[6,14,15]

There are considerable personal, community, and national costs associated with this burden of ARF and RHD. These result from direct medical costs, time away from education and occupation, negative physical and psychological experience, disruption of the lives of patients and their families, loss of the ability for children and young adults to realise their full potential, and often from premature death.[8,16,17]

Diagnosis of acute rheumatic fever

It is important that an accurate diagnosis of ARF is made as:

- Over-diagnosis will result in the individual receiving benzathine penicillin G (BPG) injections unnecessarily every 4 weeks for a minimum of 10 years; and
- Under-diagnosis of ARF may lead to the individual suffering a further attack of ARF, probable cardiac damage, and possible premature death.

Jones criteria

The Jones criteria for the diagnosis of ARF were introduced in 1944.[18] The criteria divide the clinical features of ARF into major and minor manifestations, based on their prevalence and specificity. Major manifestations are those that make the diagnosis more likely, whereas minor manifestations are considered to be suggestive, but insufficient on their own, for a diagnosis of ARF.

The Jones criteria have been periodically modified and updated. The 1992 update is currently the most widely used and quoted version, and is intended only for the initial attack of ARF.[19] Each change of the Jones criteria is made to improve specificity at the expense of sensitivity, largely in response to the falling incidence of ARF in the USA. As a result, the criteria may not be sensitive enough to pick up disease in high incidence populations, such as Māori and Pacific people. In such populations, the consequences of under-diagnosis are likely to be greater than those of over-diagnosis.

Important circumstances where ARF can be diagnosed without fulfilling the Jones criteria are noted. These include:

- Chorea as the only manifestation of ARF; and
- Indolent carditis (carditis of insidious onset and slow progression) as the only manifestation of ARF.[19]

In both these situations, cases may have insufficient supporting historical, clinical, or laboratory findings to fulfil the Jones criteria.

New Zealand Criteria

Initial episode of ARF—The main modification made to the Jones 1992 criteria for the New Zealand situation is the acceptance of echocardiographic evidence of carditis as a major manifestation.[20] In addition, there is a greater emphasis that monoarthritis may be a presenting feature if there is a history of non-steroidal anti-inflammatory (NSAID) use that is likely to have aborted classical ARF migratory polyarthritis (as alluded to in the Jones Criteria of 1992).

Categories of definite, probable, and possible ARF can be determined by the application of the New Zealand criteria to each case (Table 1). See the full guideline text for definitions of the major and minor manifestations of ARF [1] and Algorithm 1: Guide for diagnosis of ARF.[1]

Recurrent ARF—Most episodes of recurrent ARF fulfil the Jones criteria for ARF. The New Zealand guideline adopts the World Health Organization (2004) recommendations: where there is established RHD or a reliable history of ARF a recurrent attack can be diagnosed by the presence of several minor manifestations plus evidence of a preceding GAS infection[21] (Table 1).

Table 1. New Zealand guidelines for the diagnosis of acute rheumatic fever

Variables	Diagnostic requirements	Category
Initial episode of ARF	2 major **or** 1 major and 2 minor manifestations **plus** evidence of a preceding GAS infection (see text)	Definite ARF
Initial episode of ARF	1 major and 2 minor with the inclusion of evidence of a preceding GAS infection as a minor manifestation (Jones 1956)[22]	Probable ARF
Initial episode of ARF	Strong clinical suspicion of ARF, but insufficient signs and symptoms to fulfil diagnosis of definite or probable ARF	Possible ARF
Recurrent attack of ARF in a case with known past ARF or RHD	2 major **or** 1 major and 2 minor **or** several minor **plus** evidence of a preceding GAS infection (Jones 1992)[19]	
Major manifestations modified from Jones 1992[19]	Carditis (including evidence of subclinical rheumatic valve disease on echocardiogram)* Polyarthritis** or aseptic monoarthritis with history of NSAID use Chorea (can be stand-alone for ARF diagnosis) Erythema marginatum Subcutaneous nodules	
Minor manifestations	Fever Raised ESR or CRP Polyarthralgia** Prolonged P-R interval on ECG	

*When carditis is present as a major manifestation (clinical and/or echocardiographic), prolonged P-R interval cannot be considered an additional minor manifestation in the same person; **Other causes of arthritis/arthralgia should be carefully excluded, particularly in the case of mono-arthritis. If polyarthritis is present as a major manifestation, polyarthralgia or aseptic mono-arthritis cannot be considered an additional minor manifestation in the same person. All categories assume that other more likely diagnoses have been excluded; CRP=C-reactive protein; ECG=electrocardiogram; ESR=erythrocyte sedimentation rate; GAS=group A streptococcus; RHD=rheumatic heart disease.

Patients who do not fulfil these criteria, but in whom the clinician remains suspicious that the diagnosis may be ARF, should be maintained on oral penicillin and reviewed in 2 to 4 weeks with a repeat echocardiogram to detect the appearance of new lesions.[23,24]

If there is evidence of rheumatic valve disease clinically or on echocardiogram, the diagnosis is confirmed and long-term secondary prophylaxis can be commenced. If there is no evidence of carditis in a case of migratory polyarthritis, and no alternative diagnosis has been found then ARF is the diagnosis of exclusion. The putative new syndromes of paediatric auto-immune neuropsychiatric disorder associated with streptococcal infection (PANDAS)[2,5,26] and post-streptococcal reactive arthritis[27,28] should be diagnosed with extreme caution in New Zealand, particularly in Māori and Pacific populations.

Special consideration should be given to high-risk population groups such as Māori and Pacific people, and those residing in poor socioeconomic circumstances who may not be readily available for review. In these cases, it may be important to err on the side of diagnosis and treatment, and ensure ongoing review.

Echocardiography—Prior to the introduction of echocardiography, the diagnosis of rheumatic carditis relied on clinical evidence of valvulitis or pericarditis, supported in some situations by radiographic evidence of cardiomegaly. Today, all patients with suspected or definite ARF should undergo echocardiography to identify evidence of carditis. The role of echocardiography in the New Zealand setting is critical to the diagnosis of ARF. The use of echocardiography as a major criterion for ARF diagnosis[20,23,24] requires expert interpretation and adhering to echocardiographic diagnostic standards. In New Zealand, ARF carditis is classified mild, moderate, or severe (Table 2); these categories are used to guide the duration of secondary prophylaxis.

Table 2. Severity of ARF carditis

Mild carditis*
Mild mitral or aortic regurgitation clinically and/or on echo, with no clinical evidence of heart failure and no evidence of cardiac chamber enlargement on CXR, ECG, or echo.
Moderate carditis
Any valve lesion of moderate severity clinically (e.g. mild or moderate cardiomegaly), **or** Any echocardiographic evidence of cardiac chamber enlargement **or** Any moderate severity valve lesion on echo**: Mitral regurgitation is considered moderate if there is a broad high-intensity proximal jet filling half the left atrium or a lesser volume high-intensity jet producing prominent blunting of pulmonary venous inflow. [24] Aortic regurgitation is considered moderate if the diameter of the regurgitant jet is 15% to 30% of the diameter of the left ventricular outflow tract with flow reversal in upper descending aorta.[24]
Severe carditis
Any impending or previous cardiac surgery for RHD, **or** Any severe valve lesion clinically (significant cardiomegaly expected, and/or heart failure), **or** Any severe valve lesion on echo: • Abnormal regurgitant colour and Doppler flow patterns in pulmonary veins are a prerequisite for severe mitral regurgitation. [24] • Reversal in lower descending aorta is required for severe aortic regurgitation. [24]

*Valvular regurgitation is usually relatively mild in the absence of pre-existing disease; in first episodes of ARF, severe mitral and aortic regurgitation occurred in less than 10% of patients in New Zealand[24]; **When there is both mitral and aortic regurgitation, one must be moderate by echo criteria for the carditis to be classified of moderate severity.

Tricuspid and pulmonary regurgitation graded mild or greater may be seen in people with normal hearts who have fever, volume overload, or pulmonary hypertension. For this reason, a diagnosis of carditis should not be based on right-side regurgitation alone.

Evidence of a preceding group A streptococcal (GAS) infection—Streptococcal antibody titres are crucial in confirming a diagnosis of ARF. In study conditions in the New Zealand sore throat school clinic study, 74% (14/19) of cases of RF in children enrolled in the programme had a documented sore throat.[29] The most commonly used tests are the plasma antistreptolysin O (ASO) and the antideoxyribonuclease B (anti-DNase B) titres. The reference range for these antibody titres varies with age and background rate of streptococcal infections.[30]

In New Zealand, an ASO titre of greater than or equal to 480 units and/or an anti DNase B titre of greater than or equal to 680 units is accepted as significant. A positive throat culture or rapid antigen test for GAS alone demotes a case to probable or possible ARF, as up to 50% of those with a positive throat culture will be carriers only.[19]

Management of ARF—With very few exceptions, all patients with definite or possible ARF should be admitted to hospital as soon as possible after onset of symptoms. This ensures that all investigations (particularly echocardiography) are performed and, if necessary, the patient observed for a period prior to commencing treatment to confirm the diagnosis.

Hospitalisation also provides an ideal opportunity to provide information to patients and families, to notify the disease to public health (and preferably also to a local rheumatic fever register), to organise a dental check and ongoing dental care, and to initiate secondary prevention. All cases should also receive regular review, and outpatient follow-up should be organised prior to discharge.

The frequency and duration of review is dependent on the individual clinical needs and local capacity, and should become more frequent in the event of symptom onset, symptomatic deterioration, or a change in clinical findings. Particular care should be taken when cases are transferred from paediatric to adult services. To ensure continuity of follow-up, a case can be made for maintaining less severe cases in the paediatric services until discharge at age 21 years.

Except in the case of heart failure management, none of the treatments offered to patients with ARF has been proven to alter the outcome of the acute episode or the amount of damage to heart valves.[31,32] Thus, there is no urgency to begin definitive treatment.

Salicylates or NSAID medications provide only symptomatic relief, and they should be withheld (with paracetamol used if required) until the diagnosis is confirmed to avoid masking the evolution of polyarthritis. Oral penicillin V (250 mg twice daily in children; 500 mg twice daily in adolescents and adults) should be commenced in all cases while the diagnosis is being established, and this should be continued until the first dose of intramuscular BPG for secondary prevention is delivered (also in hospital).

Many cases of chorea can be managed without medication. Where necessary, carbamazepine or valproic acid are recommended. Corticosteroids have not been proven to alter the likelihood of developing, or the severity of RHD.[32] Further details regarding the priorities in managing ARF and medication use are outlined in the Guideline.[1]

Secondary prevention—Secondary prevention of rheumatic fever is defined as the continuous administration of antibiotics to cases with a previous attack of ARF, or well-documented RHD. The purpose is to prevent colonisation or infection of the upper respiratory tract with GAS, and the development of recurrent rheumatic fever.[7,21] Secondary prophylaxis reduces the severity of RHD and is associated with regression of heart disease in approximately 50–70% of those with adequate adherence over a decade,[33–35] and reduces RHD mortality.[36]

Penicillin—The regular administration of antibiotics to prevent infection with GAS and recurrent ARF is recommended for all people with a history of ARF or RHD. A recent Cochrane meta-analysis concluded that the use of penicillin (compared to no therapy) is beneficial in the prevention of recurrent ARF, and that intramuscular benzathine penicillin G (BPG) is superior to oral penicillin in the reduction of both recurrent ARF (87–96% reduction) and streptococcal pharyngitis (71–91% reduction).[37]

Twice-daily oral regimens are also likely to result in poorer rates of adherence over long periods of time and less predictable serum penicillin concentrations, when compared to intramuscular BPG.[38,39] In addition, oral penicillin V incurs a cost to the patient, while BPG is free when provided through an ARF prevention programme.

The internationally accepted standard dose of BPG for the secondary prevention of ARF in adults is 1,200,000 U.[40] In New Zealand, it is recommended that 1,200,000 U of BPG should be used for secondary prophylaxis for all persons weighing 20 kg or more, and 600,000 U for those weighing less than 20 kg.[41] Four-weekly (28-day) BPG delivery is recommended for all cases as prospective data has showed that few, if any, recurrences occurred among people who were fully adherent to a 28-day BPG regimen.[13]

Three-weekly (21-day) BPG is recommended only for those who have confirmed recurrent ARF despite full adherence to 28-day BPG. Oral penicillin should be reserved for patients who refuse intramuscular BPG. If a patient is offered oral penicillin, the consequences of missed doses must be emphasised, and adherence carefully monitored.

The Auckland Acute Rheumatic Fever Register covers 60% of New Zealand ARF registrations. The overall programme failure rate is very low at 1.4 per 100 patient years and the penicillin failure rate 0.07 per 100 patient years.[13]

Duration of secondary prophylaxis

The appropriate duration of secondary prophylaxis depends on several factors. These include:

- Age (ARF recurrence is less common after the age of 25, and uncommon after the age of 30);[13,42]
- Clinical pattern (presence or absence of carditis or RHD, and severity of carditis or RHD); and
- Environment (particularly the likelihood of ongoing exposure to GAS), and time elapsed since last episode of ARF (the most vulnerable period for ARF recurrence are the years closest to the last episode).[12]

Based on these factors, the recommended duration of secondary prophylaxis is outlined in Table 3. See also the full guideline text[1] and *Algorithm 3: Guide for the duration of secondary prophylaxis*.[1]

Table 3. Duration of secondary prophylaxis

Category	Duration of prophylaxis
All persons with ARF with no or mild carditis	Minimum of 10 years after most recent episode ARF or until age 21 years (whichever is longer)
All persons with ARF with moderate carditis	Minimum of 10 years after most recent episode ARF or until age 30 years (whichever is longer).
All persons with ARF with severe carditis	Minimum of 10 years after most recent episode ARF or until age 30 years (whichever is longer), and then specialist review for consideration of the need for continuation of prophylaxis, probably lifelong.

Individuals working or living with children, or in a living situation where there is overcrowding or close proximity to others (such as boarding schools and hostels), have a higher risk of exposure to GAS and subsequent development of ARF. In these cases, consideration should be given to extending the duration of prophylaxis.

For those presenting with RHD for whom no initial episode of ARF can be identified, the decision to commence prophylaxis should be taken on an individual basis with regard to the age of the patient, severity of the disease, possible age of first attack, and risk of exposure to GAS.

Before stopping prophylaxis, recipients should be evaluated for symptomatic deterioration and the stability and severity of valve lesions. This should include echocardiographic assessment. Where limited echocardiography is available, priority should be given to patients with a history of moderate or greater carditis, a history of one or more ARF recurrences, or clinical evidence of carditis such as a murmur. The date of cessation may be reviewed if there is a change in clinical or echocardiographic severity, specialist recommendation, a change in environmental exposure to GAS, or a recurrence of ARF.

Improving adherence to secondary prophylaxis—Improved adherence to secondary prevention is seen with active follow-up of cases when BPG doses are missed, the identification of local dedicated staff members responsible for delivery of secondary prophylaxis, developing a personal rapport with each case, and coordinating routine care.

In New Zealand, it is particularly important to support and utilise the expertise, experience, community knowledge, culture, and language skills of Māori and Pacific health and community members to assist compliance to BPG. Three key methods for improved adherence to prophylaxis (which are covered in more detail in the full guideline text)[1] are: effective rheumatic fever registers; education of families and children affected; and attempting to reduce the pain of the BPG injection.

The efficacy of secondary prophylaxis achieved by the Auckland Register sets a benchmark for other registers in New Zealand and indeed, internationally.

Conclusion

Although ARF is rare in industrialised countries, it is a significant cause of disease among Māori and Pacific children in New Zealand. The prevalence of RHD is also high among these populations. There is often significant variability in the diagnosis and management of ARF cases and the persistence of recurrent ARF in New Zealand highlights that implementation of prevention strategies has been inconsistent.

The New Zealand Guideline for Rheumatic Fever: Diagnosis, Management and Secondary Prevention[1] summarised here increases the sensitivity for the diagnosis of ARF and provides opportunity for consistent management of ARF. The Guideline details effective and cost-effective secondary prevention strategies. Implementation of the Guideline should reduce ARF recurrences and reduce the burden of RHD in high risk populations in New Zealand.

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Rhabdomyolysis in a glue sniffer

Gonesh C Karmakar, Richard Roxburgh

Toluene is an aromatic organic hydrocarbon, widely used as a solvent. Toluene toxicity can occur from accidental or deliberate inhalation of 'glue'.

Chronic glue sniffing is known to be associated with cerebellar ataxia and wide range of biochemical abnormalities including hypokalaemia, hypocalcaemia, hypomagnesaemia, and hypophosphataemia.

Rhabdomyolysis as a complication of glue sniffing probably mediated by these electrolyte disturbances is less well described. We report a case of severe rhabdomyolysis associated with a very high creatine kinase (CK) concentration following incremental inhalation of glue in a chronic abuser.

Case report

We present the case of a 48-year-old unemployed man who was admitted to Auckland City Hospital in October 2006 complaining of 2-weeks progressive inability to walk, abdominal cramps, vomiting, and loose bowel motion 2 weeks after stopping glue sniffing.

He reported that typically he would consume one tube of glue *per week* but that 1 month prior to admission he increased his intake to one tube *per day*. The binge lasted approximately 3 weeks at which time he was unable to mobilise adequately to procure glue, and by the time of admission he was unable to walk.

He had had a history of regular glue sniffing over the preceding 10 years and had been noted to have moderately severe ataxia a year previously. He was also a chronic carrier of hepatitis B. Physical examination revealed poor oral hygiene, poor nutrition, and mild dysarthria. There was generalised muscular atrophy, normal tone, moderate proximal weakness but relatively preserved distal muscle power; knee and ankle jerks were absent and he was ataxic on finger-nose and heel-shin testing.

Laboratory tests showed severe hypokalaemia (K 1.9mmol/L, normal range [NR] 3.5–5.0), hypocalcaemia (corrected Ca^{++} 1.98 mmol/L, NR 2.10–2.55), and hypophosphataemia (PO_4 0.45mmol/L, NR 0.70–1.50)).

Liver function tests showed elevated AST (1030 u/L, NR <45) and ALT (383 u/L, NR <45), but normal albumin (41, NR 35–50) and prothrombin ratio (1.1, NR 0.8-1.2). Subsequent plasma CK was more than 24,300 units and urine was positive for myoglobin. Nerve conduction studies showed no evidence of neuropathy; nor did electromyography show myopathic features.

He received intravenous fluid with potassium replacement over 7days. The CK was >22,000 units for 4 days then dropped over 3 days to less than 200 units. Serum creatinine remained normal throughout. His AST and ALT came down in proportion to CK supporting our opinion that these originated from muscle. His muscle power improved and the abdominal cramps, vomiting, and diarrhoea settled. His ataxia

remained unchanged in keeping with his long-standing clinical history, but he was able to mobilise safely despite this.

Discussion

Ataxia is a well-recognised complication of chronic toluene abuse. We illustrate a lesser known (though well described[1]) complication of toluene abuse: rhabdomyolysis. In our patient this was overlooked because his inability to walk was initially attributed to ataxia and his elevated transaminases were attributed to abnormal "liver function" due to hepatitis B.

The onset of rhabdomyolysis occurred 3 to 4 weeks into a binge in which he increased his toluene intake several fold. In spite of his high CK and myoglobinuria, his renal function remained normal. This man's chronic ataxia was not affected by his acute illness nor did it improve with therapy for that illness.

The mechanism of toluene toxicity on muscles is poorly understood. Toluene is converted to hippuric acid, the excretion of which requires excretion of a cation such as ammonium, potassium, or sodium.[2] In our patient, this together with his diarrhoea caused significant hypokalaemia.

Renal phosphate wasting[3] and diarrhoea also probably contributed to hypophosphataemia. Mechanisms by which these electrolyte disturbances are thought to cause rhabdomyolysis include impaired glycogen synthesis,[1] failure to develop reactive hyperaemia,[4] and depletion of ATP[5] which is essential for membrane function—a mechanism supported in our patient by his high CK but normal EMG.

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Spontaneous passage of large ureteric stone in a child

Ranan DasGupta, Jonathan Glass, Imran Mustaq

Only 2–3% of all urolithiasis is observed in the paediatric population, though prompt and definitive management is still required to minimise morbidity for a child affected by a symptomatic stone.

Published guidelines for ureteric stones in adults state that those larger than 4 mm are unlikely to pass spontaneously. We report an unusual case of spontaneous passage of a large ureteric stone, questioning whether there may be a selective role for conservative management in children with ureteric calculi.

Case report

An 11-year-old boy presented with right-sided abdominal pain of 3 weeks' duration, which was attributed to a 18×6 mm right ureteric stone at the level of L3 (Figure 1). There were two small calculi in the right renal pelvis, and ultrasonography excluded any associated hydronephrosis. As his pain was controlled with oral analgesia, he was booked for semi-elective rigid ureteroscopy, fragmentation, and stone removal within 5 days.

The day prior to his planned admission, the child reported increasing discomfort during micturition, and radiography was repeated (Figure 2a); the repeat radiograph included a lower pelvic film (Figure 2b), which showed that the stone had reached the navicular fossa in the distal urethra.

By the time of his procedure, he had managed to pass the urethral stone, and therefore underwent a right flexible ureterorenoscopy and laser fragmentation of the two renal calculi. Postoperative imaging showed he was entirely stone free. His recovery was uneventful.

Discussion

Treatment options for ureteric stones include conservative measures, extracorporeal shockwave lithotripsy (ESWL), and ureteroscopic stone extraction. General anaesthesia is recommended for the latter two options. Since the chances of passing a stone greater than 4mm are reported as low[1] these latter two treatments should be considered for achieving stone clearance.

Ureteroscopy produces higher stone clearance rates than ESWL, particularly for stones greater than 10 mm.[2] The development of smaller calibre instruments avoids the need for ureteral dilatation in many cases, thereby reducing the risks of stricture, need for stenting and a second procedure.[3,4]

In the adult population, there has been recent interest in medical expulsive therapy, with a meta-analysis demonstrating a role for alpha-blockers on distal ureteric stones;[5] further research is investigating a role for calcium channel blockers in these patients.

The safety of these oral medications in the paediatric population is so far unproven. Our case demonstrates that a period of watchful waiting may occasionally result in spontaneous stone passage, albeit for an unusually large stone. This possibility should be given consideration when planning surgical treatment. Secondly, this case illustrates the importance of imaging the *entire* urinary tract to avoid overlooking a stone that is on the move.

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The changing epidemiology of food allergy—implications for New Zealand

Christine Crooks, Rohan Ameratunga, Greg Simmons, Penny Jorgensen, Clare Wall, Maia Brewerton, Jan Sinclair, Richard Steele, Shanthi Ameratunga

Abstract

Food allergy (FA) is recognised as an important public health problem in developed countries. Recent studies suggest a significant proportion of the general population has a definable FA. The methods used to study FA influence published estimates of incidence and prevalence. In particular, studies relying on self-assessment are likely to overestimate the condition compared to studies using a comprehensive approach including symptoms, allergy testing, rigorously conducted laboratory tests, and food challenges. Currently there are no reliable data on the prevalence of FA in New Zealand. This has had several adverse consequences including the lack of public hospital services for patients with severe allergies. In this article we summarise the epidemiological data on FA and discuss the implications for New Zealand.

Food allergy background

Food allergy (FA) is a common adverse reaction to food mediated by the immune system. The condition can result in a wide variety of clinical manifestations that involve IgE or non-IgE mediated mechanisms, or sometimes both (e.g. eosinophilic oesophagitis). In contrast, food intolerances can be considered adverse non-allergic responses to foods. This is best illustrated by lactase deficiency, where cow's milk can cause gut symptoms as a result of undigested lactose.[1]

The gastrointestinal tract is important not only in the absorption of nutrients but also in the protection against microbial invasion through appropriate immune responses. Oral tolerance develops when there is down-regulation of the immune response to non-harmful antigenic substances.[2]

Allergic reactions to food are largely thought to be due to genetic factors and can occur when oral tolerance is impaired.[2] The severity of FA can vary from trivial abdominal discomfort to death from anaphylaxis. FA accounts for a significant proportion of anaphylaxis and severe allergic reactions in children.[3–5] Much less is known about the pattern of FA in older adults.[6]

Most allergic reactions to food originate in childhood and few treatment options have the potential to alter their natural course.[7] Both genetic and environmental factors are likely to be involved in the pathogenesis of FA, but the interactions are complex with considerable gaps in knowledge.[7] Studies of identical twins indicate high concordance pointing to genetic predisposition, but inter-related environmental factors such as feeding practices are also likely to be important.[8]

Many children who develop allergy to food may outgrow their allergies.[9–12] For example, in a study of 118 children with cow's milk allergy (CMA), 42 (35.6%) with

non-IgE mediated CMA were found to be free of their allergy by the age of 5 years.[12] However 13 children (15% of the cohort) had persistent CMA at the age of 8.6 years. Thus, contrary to previous perceptions, CMA may persist into late childhood in a significant minority of patients.[12]

Accurate diagnosis and appropriate management of food allergy is critical. Failure to identify the offending food allergen(s) correctly may place the person at risk of recurrent anaphylaxis. On the other hand, inappropriate and unsupervised dietary elimination can increase the risk of nutritional deficiencies. This was recently illustrated by the case of a 14-month-old child with CMA who presented with rickets due to poorly managed dietary exclusions, resulting in deficiencies in Vitamin D, dietary calcium, and phosphate causing impaired bone mineralisation and growth.[13]

Therefore, FA sufferers are doubly challenged to find a diet that will not result in an adverse reaction, but which is nutritionally balanced, in order to maintain good health.[14]

In addition to the clinical symptoms, severe FA can pose a significant socioeconomic burden to families as diet and lifestyle may be adversely affected. Unfortunately, some of the commonest allergenic foods are also of high nutritional value (e.g. milk, eggs, and peanuts) relative to their cost. The financial burden of substituting alternative dietary options can be substantial.[13] FA can also limit social activities and school attendance in children, and young adults may have restricted career options.[15]

FA can generate considerable anxiety in affected families and the community, and some patients suffer post-traumatic stress disorder after a severe episode of anaphylaxis. Indeed, a recent study suggested that having a child with severe food allergy had the same adverse impact on the quality of life as having a child with Type 1 diabetes.[15]

Prevalence of food allergy: problems in ascertainment

FA is acknowledged to be a significant public health problem in developed countries, but there are major gaps in knowledge regarding the population burden of the condition as highlighted in a recent meta-analysis funded by the European Commission (part of the EuroPrevall research project).

Of the 934 papers that investigated the prevalence of allergy from January 1990 to December 2005,[16] only 54 met the authors' criteria for inclusion, and only 19 included the double-blind placebo-controlled food challenge (DBPCFC) method of diagnosis (considered to be the gold standard method[14,16–18]).

The articles that met the review criteria were classified by the diagnostic methods used; self-reported FA (SRFA), specific IgE (IgE cut-off levels varied between studies), skin prick test, a combination of SRFA and IgE or skin prick test, and SRFA and DBPCFC. The authors found widely varying estimates of prevalence depending on the method used. For example, the prevalence of CMA ranged from 1.2 to 17%, egg allergy from 0.2 to 7%, and peanut allergy from 0 to 2%.[16] Not surprisingly, studies based only on self-reported FA have tended to provide the highest estimates of prevalence.[16]

Inconsistent study design

The variability in reported results was subsequently highlighted by Keil, who critically reviewed published studies on the epidemiology of FA between October 2005 and January 2007.[19] Only six published studies were identified that met the review criteria for study design, recruitment process, assessment methods of FA outcomes, and interpretation.[19] These included two well-designed birth cohort studies from the Isle of Wight in the United Kingdom (UK).

The first of these studies found that the prevalence estimates of FA (based on clinical history, skin prick testing, open food challenges and DBPCFC) in the first year of life varied between 2.2% and 5.5%, which were considerably lower than the estimates based on parental reports (between 5.5% and 14.2%).[17]

In the second Isle of Wight study, FA prevalence was determined in a cohort of 6 year-olds. Adverse reactions to food were reported by 11.8% of the cohort. This is higher than the prevalence confirmed by clinical history, skin prick tests, and open food challenge (2.2%) and DBPCFC (1.6%).[20]

The propensity to over-estimate adverse reactions to food based on self-reported symptoms has also been observed among teenagers. For example, Pereira et al found that in contrast to the prevalence of self-reported symptoms among 11 year olds (11.6%) and 15 year olds (12.4%), only 2.2% had a diagnosis confirmed by food challenges.[21] Similar rates may also be perceived by adults; in a study of 1483 adult subjects in the Netherlands, 12.4% reported FA but only 0.8% were confirmed as FA by DBPCFC.[22]

Inconsistent study design has been identified as a problem in the Global Allergy and Asthma European Network (GA^2LEN) review of 18 on-going European birth cohort studies.[23] It is hoped that through the co-operation of participating research teams that some data may be pooled and common analyses used for endpoints such as the natural history of FA.[23]

Laboratory factors may influence the diagnosis of FA, including food-specific IgE cut-off values.[16] Limitations also exist due to a lack of standardisation of the skin test allergens used to assess sensitisation.[19,23] A survey of the participating research teams in the GA^2LEN study found that different standard panels of allergens are used across Europe.[23]

There may be variability in physician diagnosis which further complicates the perception of increasing FA prevalence.[9] Responses to a questionnaire sent to 7000 United States physicians indicated that non-allergist physicians diagnosed FA at a higher rate than allergy specialists. This is the first study to report differences in FA diagnosis between physicians. This survey suggests there may be a need for further training in this area.

The establishment of accurate data on FA prevalence is problematic because most estimates of prevalence are based on methods other than a comprehensive approach including symptoms, allergy testing, and the gold standard DBPCFC.

It is difficult to predict clinical reactivity to an allergen based purely on the measurement of IgE antibodies in serum and mast cell reactions by SPT.[24] Positive results from either method, especially with low levels of food-specific IgE, do not

mean that an allergic reaction is inevitable on consumption of the food.[24] The significance of other methods such as kinesiology, hair testing, and 'electroaccupuncture according to Voll' (EAV) are unknown.

One approach that could address the problems described is to recruit a large unselected birth cohort, where regular clinical assessments and allergy testing is undertaken. Children suspected of having FA then undergo DBPCFC. Such studies are, however, limited by expense and logistical complexities, and may not provide time-dependent-information (such as changing population demographics and changing dietary practices), and do not provide information about adults with FA. Furthermore, there may be ethical concerns with undertaking DBPCFC in children.

Alternatively, monitoring the change in patterns of food allergy (using the same research tools used to detect changes in prevalence of peanut allergy[18] and asthma[25]) would be a simpler and less expensive approach, and could provide relevant, useful information.

Is the incidence of food allergy increasing?

The extent to which the burden of FA has changed over time is contested. While some studies do not support evidence of increasing incidence of FA,[14,16,19] the well-designed Isle of Wight birth cohort studies showed an increase in prevalence of peanut allergy (from 0.5 to 1.0%) and peanut sensitisation (from 1.1 to 3.3%) from 1989–1994 to 1994–1996.[26]

By using the same research instrument (nationwide, cross-sectional, random telephone survey with a standardised questionnaire), Sicherer et al found self-reported peanut allergy doubled in children less than 5 years of age from 1997 to 2002 in the US.[18] Clinical reports of children with food-related anaphylaxis have also reportedly increased in Australia.[27,28] The explanations for these observed increases were not investigated in these studies.

Food allergy in non-European populations

FA reactions appear to occur at a higher rate in Asian children in Westernised countries.[29,30] A cohort study produced important results on self-reported wheeze in European children and south Asian children born in the UK.[29] Parental reports of food and drink triggered-wheeze were significantly higher in the south Asian children compared to the Europeans and doubled over a 5-year period.[29] While this was a clinically significant finding due to the impact on health services, the reasons for these differences were not reported.

Moreover, FA may be more common in Asian countries than previously suspected.[8] In Singaporean children (identifying with Chinese, Malay, Indian, and Eurasian ethnic groups) the most common foods causing allergies were peanuts, shellfish, and egg.[8]

In this study of FA, which was determined by SPT and questionnaire, peanut allergy was seen in a third of children. While Singaporean children develop shellfish allergy at an older age than peanut or egg allergies, it is the second most common allergen.[8]

This finding for Singaporean children was considered to be different to paediatric norms in the United States and Western Europe where the major allergenic foods are milk, egg, and peanuts.[8]

For Singaporean and Hong Kong adults, shellfish allergy is the major cause of anaphylaxis in patients reporting to emergency departments.[8] The high incidence of peanut allergy was also in contrast to previous results. It had been thought that the low incidence of peanut allergy in Chinese populations was due to different processing methods. FA appears to be of concern in Asian countries, and the authors recommended that large-scale epidemiological studies be carried out there.[8]

These studies highlight the changing epidemiology of food allergy and underscore the importance of environmental factors in food allergy.

Food allergy in New Zealand: what is known?

Recent publications have included limited data on FA prevalence in New Zealand.[16,31] In the first study, information was from the early 1990s and was part of the European Community Respiratory Health Study. Data were collected from 1148 New Zealanders by a brief questionnaire about respiratory health, which included four questions regarding dietary intake.[32]

From this study 11.4% of subjects reported illness from food, although it is unclear if the FA diagnosis was supported by allergy tests. In another paper the prevalence of CMA amongst New Zealanders was reported to be about 11% based on child and parental reported data from a cohort of 155 children aged 3 to 10 years in Dunedin.[31]

The burden and characteristics of FA in New Zealand are likely to have changed over time in view of the demographic changes (e.g. substantial increases in the proportions of Asian and other immigrants in the last two decades) and changes in the diversity and production of foods available to consumers.

Several species of shellfish are endemic to New Zealand.[33] Allergic reactions to shellfish and molluscs may be highly cross-reactive, which is thought to be due to the conserved nature of amino-acid sequences in the allergenic protein tropomyosin across species.[34]

The relationship between FA and New Zealand's unique species of shellfish may also require further investigation. Infant feeding patterns and genetic predisposition are also likely to vary in different ethnic communities. While equivalent data for FA are not available, Pacific children who migrate to New Zealand from countries such as Samoa and Tonga have been noted to experience an increased incidence of asthma compared to children in their home countries.[35]

The incidence of FA in Māori is unknown. In order to fully understand the aetiology of FA and initiate primary prevention and treatment strategies in New Zealand, it is important to identify any disparities between Māori and non-Māori.

Māori have on average the worst health status of any ethnicity in New Zealand.[36] The Māori population has a higher growth rate compared to Europeans, and between 2006 and 2021 the Māori population is expected to grow by 20% compared to only 5% in Europeans.[37] Therefore the paucity of information on FA in Māori limits our ability to

predict the future disease burden and plan the most appropriate delivery and access to health resources over the coming decades.

Overseas research has identified FA as a risk factor for life-threatening asthma attacks in children.[38] Māori experience greater morbidity associated with asthma and Māori are twice as likely to be hospitalised for their asthma as non-Māori.[36] In asthma and other atopic diseases, FA may have an important role in the health disparities we see between Māori and non-Māori.

Lower socioeconomic status is a well documented predictor of poor health outcomes and a barrier to care; it is well documented that Māori are more likely to live in more deprived areas than non-Māori.[36] Therefore, the collection and analysis of up-to-date, accurate epidemiological data and the development of specific health strategies must remain an ongoing priority in order to tackle these inequalities.

Consequences of the limited data on food allergy in New Zealand

Anecdotal evidence shows that the public hospital system in New Zealand experiences substantial constraints in responding to the needs of those affected with FA.

Children with the potential for outgrowing FA need to be reviewed regularly so that they are not confined to an unnecessarily restricted diet, while adults with FA also need to be reviewed particularly after experiencing a severe reaction.

With no national guidelines for the provision of allergy services there is an *ad hoc* approach by district health boards (DHBs) including the availability of specialists and purchase of laboratory tests.

Few New Zealand hospitals currently offer specialist allergy services for adults, and services for children are particularly limited from a national perspective. Indeed, many patients with complex allergic disorders have to fly long distances for diagnosis and treatment, often at their own expense. Similar shortages of allergy specialists and primary health care services are also seen in the UK[13,39] and in Australia.[27]

Access to laboratory tests in some parts of the country is also restricted. In Wellington, for example, patients of private specialists are required to pay for laboratory tests.

Better epidemiological data may assist health boards in prioritising the need for allergy/immunology services and ensure robust coordination and continuity of care across primary, secondary, and (where necessary) tertiary care services. Tertiary hospital-based multidisciplinary teams would ideally include specialist allergists, nurses, dietitians, and facilities for food challenges and immunotherapy.

A better understanding of the epidemiology of FA may also identify the gaps in established protocols and school services. The *ad hoc* approach to severe food allergy in New Zealand's schools may place some students at increased risk of reactions while attending school.

Currently, adrenaline auto-injector devices—the primary treatment for acute anaphylaxis in the community—are not publicly funded in New Zealand. Ensuring an up-to-date supply of this life-saving medication (e.g. EpiPen®—the commonly used

formulation—has a shelf-life of 12–16 months) can place an unacceptably high financial burden on families with severe FA. Better data on the prevalence of severe allergies may assist funding these devices.

Compliance with food-allergen labelling regulations is a significant issue for the food industry including manufacturers, the food service, and hospitality sectors. Mistakes can be costly for both the industry and consumers. Better data on the prevalence of food allergy would assist the food industry both in compliance and in producing food suitable for people with FA.

Studies of FA can provide useful information that is relevant to the national economy. New Zealand is heavily dependent on agricultural exports and the development of new foods. Recently, the kiwi fruit gold variety was identified as being as allergenic as the green variety in Europe.[40]

The inadvertent development of highly allergenic foods may inflict damage to New Zealand's international reputation as an exporter of high quality foods. On-going FA studies have the potential to identify these foods at an early stage, or ensure appropriate processing and labelling to mitigate the risk to vulnerable individuals.

While many advances have been made in the last two decades, much has yet to be learned about FA. More targeted research elucidating the burden, barriers to effective treatment, and related factors in New Zealand is necessary to ensure better services and support for all children and adults with food allergy, and improved safety for those at risk of severe reactions.

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Mental health services for children and adolescents in New Zealand, outcomes, and the Health of the Nation Outcome Scale for Children and Adolescents (HoNOSCA)

Matthew J F Eggleston, William G A Watkins

Abstract

Patients and their families as well as communities, service providers, and funders of services would be united in their desire that children and adolescents who require mental health services should receive those services. There would also be agreement that treatment delivered by these services should be safe, effective, and (given that resources for these mental health services are limited) delivered in a timely and cost-effective manner. Furthermore, there would be a consensus that outcomes of treatment are extremely important and that there is a need to evaluate these in a valid manner.

This article reviews current access to Child and Adolescent Mental Health Services (CAMHS) in New Zealand as well as issues relevant to the introduction of routine outcome measurement in these services; and critically appraises the psychometric properties and clinical utility of the first routine outcome measure introduced for CAMHS by the Ministry of Health (MOH)—the Health of the Nation Outcome Scale for Children and Adolescents (the HoNOSCA).[1] It is argued that the evidence base for the implementation of routine outcome measurement is poor, that systematic evaluation of its introduction should occur, and that already under-funded CAMHS should be adequately resourced to support the additional work involved.

Access to Child and Adolescent Mental Health Services (CAMHS)

Figures from the Mental Health Commission's *2006 Report on Progress*,[2] which examined progress towards the implementation of the *Blueprint for Mental Health Services in New Zealand*,[3] indicated that access to CAMHS for young people aged 0–19 years remains substantially below the equivalent figure for adults. Access rates to CAMHS for young people ranged between 0.9 to 1.6%, depending on region, which is substantially below the Blueprint's access target of 3%.

While the Blueprint recommended that 26% of Mental Health Service funding be applied to services for young people, the actual figure for 2004/05 was a disappointing 11%.[2]

Despite a real increase in funding for all Mental Health Services of 154% over the period 1993/94 to 2004/05 to NZ$866.6 million, commensurate increases in rates of access to services have not been realised.[2] While there has been no systematic investigation of this phenomenon, in recent years it is apparent that there has been a proliferation of clinical and non-clinical activities required of clinical staff, together with a burgeoning documentation requirement for each activity.

This increase in "compliance" requirements is a likely contributor to the finding that DHB provider expenditure per individual has increased by an average of 20% over three years for three of the four New Zealand regions.[2]

The outcome measures movement

Over the last decade, there have been strong international Governmental drives for mental health services to demonstrate the quality and cost-effectiveness of their services.[4–7] New Zealand is following this trend with formal national reporting of the HoNOSCA by CAMHS being required by the Ministry of Health (MOH) by June 2008.[8] In addition to hoping that this measure will demonstrate the quality and cost-effectiveness of services, other rationales for the introduction of routine outcome measures include embedding measures into the routine process of clinical assessment,[8] as well as supporting recovery by promoting and facilitating the development of an outcomes-focused culture in the mental health sector.[9]

The HoNOSCA is the first of a planned suite of outcome measures to be introduced as part of The New Zealand Health Standard Measures of Assessment and Recovery (MH-SMART) Outcomes Initiative.[9]

Strong advocates of routine outcome measurement for psychiatry acknowledge that there is little empirical evidence that their introduction is of benefit to patients.[10] Harnett et al considered that the effectiveness of the introduction of the HoNOSCA and other outcome measures in providing meaningful information for evaluating the impact of child and youth mental health services remains to be determined.[11] Bickman et al stated that, without identifiable clinical processes, outcome data alone are unlikely to enhance services.[12]

No evidence exists to suggest that the use of routine outcome measures, including the HoNOSCA, assists with demonstrating the quality of a CAMHS, its cost-effectiveness, or that it promotes an outcomes-focused culture. Hence, the proposed national introduction of the HoNOSCA is not evidence-based (as is correctly increasingly being demanded of treatments in the field) and should be considered experimental. It seems unfortunate that the manner of the introduction of the HoNOSCA by the MOH does not allow its usefulness in achieving its intended purposes to be evaluated.

Child and adolescent psychiatry has long had a tradition of embedding routine measures into clinical assessment as well as utilising measures for evaluating outcomes. Indeed, well-established standardised measures such as the Conners' rating scales constitute an integral part of both the assessment and monitoring of response to treatment for attention deficit hyperactivity disorder (ADHD).[13]

Our outpatient child psychiatry clinic has systematically embedded measures in the routine process of clinical assessment for some years by routinely utilising the Achenbach System of Empirically Based Assessment (the Child Behaviour Checklist, Teacher Report Form and Youth Self-Report).[14] These instruments gather systematic information from multiple informants; not only on a broad range of relevant emotional and behavioural problem areas, but also social and academic competencies. They are extensively utilised in clinical and research settings, have been normed on large samples of children and adolescents, and have sound psychometric properties.[14]

It is difficult to conceive how a single outcome measure would be considered appropriate for the entire field of paediatrics (from oncology to diabetic endocrinology), or indeed any other broad medical specialty area. Yet, this is exactly what is being implemented in child and adolescent as well as adult psychiatry. In fact, many issues make the understandable aims of demonstrating quality, cost effectiveness of services and evaluation of broad outcomes more difficult in child and adolescent psychiatry than in most other areas of medicine.

Child and adolescent psychiatric diagnostic entities are syndromally-based and are imperfect approximations. The natural course for most disorders is not well defined which adds to the difficulty of determining whether treatment may be making a difference.

The majority of children and adolescents who present to clinics with psychiatric disorder have comorbid conditions, making comparisons between outcomes for children and adolescents with similar primary psychiatric disorders very difficult. For many diagnostic entities, best-practice treatment guidelines are largely based on consensus expert opinion rather than solid empirical evidence.

McClellan and Werry, while strongly advocating for evidence-based treatment approaches, stated that "the notion of a well-defined standard of care for most childhood disorders remain elusive".[15]

There are also important aspects of CAMHS functioning which are simply not captured by a narrow focus on outcomes. A significant number of children and adolescents will be assessed by a CAMHS and, appropriately, not be offered treatment. A further group, including those with autistic spectrum disorders, may be assessed and offered appropriate treatment, but large improvement in the core symptoms of their condition would not be expected.

An important minority of children and adolescents presenting with conditions (including schizophrenia) prove to be treatment-resistant despite best-practice treatment approaches, yet denying them and their families ongoing psychiatric treatment and care on the basis that they do not show a positive change on outcome measures would be unethical due to the severity of their condition.

There has been insufficient consideration or exploration of other possible adverse effects of routine outcome measurement. It has been estimated that up to 10% of a clinician's time can be spent on a simple outcome measurement system.[16] Hence, the time-costs associated with the implementation of routine outcome measurement are not insubstantial and are likely to have a negative impact on access to already under-resourced and overburdened CAMHS.

Importantly, no additional funding for clinical or administration time has been allocated to assist with the demands of implementing and maintaining the collecting, processing, interpretation, and feeding back to consumers of routine outcome measurement data.

Because HoNOSCA scores are to be repeated within 2 weeks of every 3-month interval during a consumer's episode of care, a major increase in outpatient reviews will be needed solely for the purposes of scoring the HoNOSCA, rather than for indicated clinical reasons.

In addition to this further time-cost, consumers and their families are likely to object to attending reviews for the purpose of scoring the HoNOSCA, if this is considered by them to be clinically unnecessary. It is also conceivable that, rather than promoting an outcome-focused culture in CAMHS, embedding potentially suboptimal measures into the routine process of clinical assessment may result in clinicians having less time, commitment, and energy to devote to more relevant clinical measurement, audit, and research.

There are also, potentially, major clinical repercussions of formal national reporting of outcomes to the MOH. Some of these will depend on how the MOH intends to utilise the information and, to date, this has not been articulated. There are valid concerns that clinicians may become "outcome measure-focused" rather than "patient outcome-focused".

Given the requirement for reporting 3-monthly outcome measures, clinicians may preferentially choose short-term, narrow, and medication-oriented approaches rather than clinically indicated longer-term, systemic, and psychotherapeutic treatment approaches. Services may become reluctant to assess and treat individuals who are unlikely to show improvement on measures, but who nevertheless require significant care, stabilisation or support, such as those with autistic spectrum disorders. It is even possible that clinicians may become reluctant to retain in treatment those patients with complex or poor prognosis conditions whose outcome measures continue to deteriorate despite best-practice treatment.

Stakeholder consultation

The process of consultation with stakeholders regarding the choice of outcome measures for CAMHS deserves comment in its own right. The New Zealand Mental Health Research and Development Strategy commissioned a study to examine the use and acceptability of child and youth mental health outcome measures.[17] A very significant portion of the questionnaire for clinicians related to only two measures, including the HoNOSCA. In fact, the questionnaire appeared designed to favour these two measures in that no other measures were specifically mentioned.

Clinicians' understandable general support for the importance of outcome measures and for "routine use of outcome measures" (after all these should already be part of everyday practice, particularly for disorders such as ADHD) was then extrapolated to mean support for the introduction of a "routine outcome measurement *system*".

Clinicians' general endorsement of outcome measures and favouring of the HoNOSCA (despite most having limited experience with the instrument) as one of two clinician-rated outcome measures (the other measure was not specifically mentioned in the questionnaire, but needed to be spontaneously mentioned and endorsed by respondents) was extrapolated to the doubtful conclusion that clinicians considered the HoNOSCA "a worthwhile measure". The authors recommended the introduction of the HoNOSCA and the second measure as routine outcome measures.

Relevantly, 87.6% of respondents anticipated problems with the introduction of routine measures and, of those providing comment, 84.3% indicated that staff willingness or ability to take on extra work was anticipated to be the major complication.

The HoNOSCA: psychometric properties

In general, studies that have examined inter-rater reliability have found this to be within the moderate to good range for individual HoNOSCA items and for the total HoNOSCA score, although most of these estimates were based on ratings from clinical vignettes rather than actual clinical interviews.[1,11,18,19]

Harnett et al, however, found poor internal validity of the four main HoNOSCA subscales, "significantly limiting their use in any analysis of outcome".[11] Similarly, a New Zealand study found a weak degree of fit of the subscales with the data from the individual HoNOSCA items and concluded "not too much reliance should be placed on the HoNOSCA subscales".[20] Test-retest reliability was good over a period of 2 to 4 weeks in an inpatient adolescent sample.[11]

Several studies have examined the convergent validity between the total HoNOSCA score and a range of other clinician rated measures. Moderate correlations have been found between the HoNOSCA and a measure of clinical and environmental complexity, the Paddington Complexity Scale (r of 0.46 [1] and 0.640,[21] $p<0.001$).[21]

Similarly, moderate correlations have generally been found between the HoNOSCA and a very brief global measure of impairment, the Children's Global Assessment Scale (CGAS) (r significant and between -0.6 and -0.644,[11,20–22]).[23]

The Treatment for Adolescents with Depression Study (TADS), however, found only a weak correlation with the CGAS ($r= -0.14$, $p = 0.005$).[24] Even so, the moderate correlation with the CGAS found by some studies is relevant, given that the CGAS is very brief, has good inter-rater and test-retest reliability, considerable evidence to support convergent validity, is sensitive to change, and has been used extensively in both research and clinical settings.[25]

While neither the HoNOSCA or CGAS have formal normative data, the CGAS has been utilised in several large epidemiological studies that provide some standardised data[25]. It certainly could be argued that the CGAS may be a better researched and easier to use proxy measure for the significantly more time-consuming HoNOSCA.

Several studies have suggested that the HoNOSCA is able to detect change over time with total HoNOSCA scores correlating moderately with clinicians' global impressions of change.[1,11,18,19] In one such study "marked improvement" on parents' and referrers' global outcome measures were associated with HoNOSCA change scores of four to five points.[19] However, the order of change is variable. For example, in an adolescent inpatient sample, the mean HoNOSCA change scores were 1.51 and 1.92 points over 3 and 6 months of inpatient, presumably intensive, treatment respectively.[11]

One factor limiting the merits of clinicians' rated outcome measures is the finding that clinicians rate very few patients as deteriorating over time.[18] Harnett et al noted a tendency for HoNOSCA scores to fluctuate considerably for patients staying longer than 6 months on an inpatient adolescent unit, which was considered to limit the usefulness of the HoNOSCA as an outcome measure for individuals with psychotic symptoms or self-harming behaviour.[11]

In terms of the utility of the HoNOSCA in clinical settings, reports are highly variable. Bilenburg reported that 80% of clinicians found the HoNOSCA to have "substantial" clinical utility.[4] In contrast, Kisely reported that the HoNOSCA was not useful in providing relevant information.[5] Similarly, Patterson et al's survey found that the vast majority of respondents did not find the New South Wales-mandated routine outcome measures (including the HoNOSCA) clinically useful in their regular reviews of patients.[26]

Clinicians have also reported that the HoNOSCA is difficult to use with children under 6 years,[4] as well as being adolescent–oriented.[27] Three of the eight psychiatric symptom scales are of little clinical relevance to the vast majority of pre-adolescents; namely substance use, psychotic symptoms, and non-accidental injury. Two studies reported that a 3-month reporting interval may be too short a period of time for reviews.[4,26]

Several studies in clinical (rather than research) settings have found poor rates of data completion for the HoNOSCA, particularly at follow-up and discharge.[5,20,26] A trend for completion rates to diminish over time has also been noted, raising concerns that completing outcome measures is onerous or is seen by clinicians as not providing quality feedback.[20, 26] Of interest, in a head-to-head comparison in clinical practice, completion rates were higher for the CGAS than the HoNOSCA.[20]

Patients experiencing some very significant disorders, usually associated with significant disability, have had unexpectedly low scores on the HoNOSCA. Garralda and Yates found children with pervasive developmental disorders had a mean total HoNOSCA score of 11.5, while children with eating disorders had a mean total score of 7.6, compared with a mean total score for all disorders of 11.58.[19]

Children with no disorder had a mean score of 7.46, virtually the same as those with eating disorders. Similarly low scores for patients with eating disorders were found in a further study (mean score of 7.1),[20] but not in the Bilenburg et al study (mean score of 16.8).[4] Such variability in the few studies to date inevitably leads to doubts about the HoNOSCA's suitability as a universal measure.

Despite reasonably widespread introduction of the HoNOSCA to the United Kingdom and Australia, it is concerning that only a relatively small number of studies have been published involving the HoNOSCA. Additionally, if the HoNOSCA was a robust outcome measure, it is reasonable to expect it would be more widely utilised in randomised controlled trials.

Instead, many major randomised controlled trials utilise other clinician-rated global measures of change such as the Clinical Global Impression (CGI) scale.[28] This very brief, simple and easily completed scale also potentially allows direct comparison of clinician, parent and child or adolescent views on the benefits (or otherwise) of treatment.

Summary and conclusions

Child and adolescent mental health services in New Zealand remain significantly under-funded, and further progress toward improving this appears to have stalled. The *mandatory* requirement for universal reporting with routine outcome measures such as the HoNOSCA—despite no evidence that this is of benefit to patients or that it

improves the quality of services—is premature. The possible adverse effects of introducing routine outcome measurement to CAMHS have been inadequately evaluated. Furthermore, the requirement to utilise the HoNOSCA has not been costed and comes with no additional funding, thus it can be expected to further erode clinical time. This in turn is likely to adversely affect access to CAMHS for children and adolescents.

The HoNOSCA has some significant limitations, particularly for younger children and for some diagnostic groups such as pervasive developmental disorder and eating disorders. A number of other possible outcome measures remain worthy of reconsideration. For example, the CGAS, which compares well psychometrically and appears to have better clinical utility than the HoNOSCA.

Recommendations:

- The positive and negative effects of formal national reporting of routine outcome measures to the MOH need to be systematically evaluated.
- If further measures are to be introduced or become part of the MH Smart initiative, an experimental design, with services randomised to "demonstration" and "treatment as usual sites", would allow systematic investigation of the relative benefits and costs.
- Given the current poor state of funding of CAMHS, realistic additional resources should be provided to support the MH Smart initiative.
- Fostering and funding the process of good quality clinical audit and research in CAMHS may be a more effective method for improving the quality and cost-effectiveness of services.

Competing interests: None known.

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THE NEW ZEALAND MEDICAL JOURNAL
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Gangrenous gut

Case taken from a paper by Dr. Wilson published in NZMJ 1908;6(26):19–31.

I.G. a man of 32, was shovelling gravel at about 10 a.m. on August 25th., 1906, when he was gripped with sudden intense abdominal and inguinal pain.

Contrary to the ethics of the New Zealand labourer, be tried to work it off by shovelling more gravel, with the result that the " pain beat him," and he had to lie down. Dr. Peach was sent for and found a hernia, which he failed to reduce. He sent him into hospital at about 12.30. I saw him at about 2. He had the usual intense pain, had vomited several times non-feculent material, and had a large strangulated hernia in the right inguinal region. I operated at 4 o'clock, i.e., six hours after strangulation, and found gangrenous gut.

Assisted by Dr. Martin, I resected 5½ inches of intestine, did an end to end anastomosis over a Mayo-Robson bobbin, and did a radical cure by Bassini's method. Recovery was uneventful, and he was discharged from hospital on September 22nd.

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OxLDL-induced cell death is inhibited by the antioxidant 7,8-dihydroneopterin

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The presence of ©-interferon within atherosclerotic plaques demonstrates the chronic inflammatory nature of vascular disease. During inflammation, human macrophages are stimulated by ©-interferon to secrete 7,8-dihydroneopterin (7,8-NP) which is oxidised to neopterin. Plasma neopterin levels are known to increase with the severity of a patient's vascular disease. The reason for this cellular response is controversial but we have previously shown that 7,8-NP is a potent antioxidant that can protect macrophage cells from a range of oxidative stresses. Oxidised low density lipoprotein (OxLDL) induced cell death is considered to be an important process in the formation of necrotic lipid rich plaques and in atherosclerotic plaque destabilisation. We examined whether 7,8-NP can inhibit oxLDL mediated cell death in human monocyte derived macrophages.

Macrophages were isolated from whole blood and cultured in RPMI-1640 containing 10% heat inactivated human serum. Heavily oxLDL was prepared by incubation of human plasma LDL with copper ions.

Incubation of macrophages with oxLDL caused the rapid loss of cell viability, intracellular glutathione and the generation of intracellular reactive oxygen species within the first 6 hours. No caspase-3 activation was observed but cytochrome c release and phosphatidylserine exposure was observed reaching a maximum at 12 hours. The presence of micromolar concentration of 7,8-NP effectively inhibited the loss of cell viability as well as inhibiting phosphatidylserine exposure and the generation of intracellular reactive oxygen species in the mitochondria. Surprisingly, 7,8-NP did not appear to inhibit cytochrome c release.

Our data suggests that the presence of micromolar concentration of 7,8-NP in atherosclerotic plaques may have a significant effect on macrophage stability, cell death and advanced plaque development.

Impact of Oxidative Stress on DNA Methylation

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Maintenance DNA methyltransferase (DNMT) enzymes methylate cytosine residues during DNA synthesis to reproduce the methylation pattern of the original template strand. DNMT enzymes contain a critical active site cysteine residue. Therefore, we propose environmental factors that alter the redox homeostasis of cells have the potential to oxidize DNMT and inhibit DNA methylation. Cells are exposed to mild oxidative stress for short periods that do not affect cell viability or proliferative capacity, meaning that conventional techniques for measurement of global DNA methylation are likely to be too insensitive. We have developed a novel method to measure DNA methylation in newly synthesized DNA. In brief, cells are growth arrested in G1 phase with thymidine before being released from the block with isotopically labelled N^{15}-deoxycytidine, and the ratio of methyl-N^{15}-deoxycytidine to N^{15}-deoxycytidine determined by liquid chromatography mass spectrometry (LCMS). We will present data on the validation of the method and its advantages by using 5-aza-2'-deoxycytidine, and the effects on DNA methylation when cells are exposed to exogenous oxidants and chemicals that generate free radicals.

Pro-apoptotic isothiocyanates inactivate proteins involved in maintaining mitochondrial redox homeostasis

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The isothiocyanates are a class of phytochemical with recognized chemopreventive and chemotherapeutic potential. We have shown that aromatic isothiocyanates, in particular phenethyl isothiocyanate, possess the ability to induce apoptosis in cells that overexpress the anti-apoptotic protein Bcl-2. We have focused our study on identification of the cellular targets of the pro-apoptotic isothiocyanates that may play a role in mediating apoptosis. The predominant intracellular reaction of the isothiocyanates is with cysteine residues, which play a role in the activities of a number of proteins involved in the signaling pathways that regulate cell death. Using a proteomic method to fluorescently label oxidized cysteine residues, we have shown that a selective pool of thiol proteins are modified following exposure to phenethyl isothiocyanate. Our screen revealed that the mitochondrial thiol peroxidase, peroxiredoxin-3, is oxidized following a short exposure to pro-apoptotic isothiocyanates. Rapid oxidation of peroxiredoxin-3 was confirmed by western blotting for reduced (monomeric) and oxidized (dimeric) protein. Pure enzyme and cell studies have revealed that thioredoxin reductase, which helps maintain peroxiredoxin-3 in its reduced form, is irreversibly inhibited by pro-apoptotic isothiocyanates. Inhibition of thioredoxin reductase and oxidation of peroxiredoxin-3 occur upstream of induction of apoptosis, as determined by measurement of caspase activation. We propose that disruption of proteins involved in the maintenance of

mitochondrial redox homeostasis may provide the switch to trigger isothiocyanate-mediated apoptosis.

Outer Membrane Vesicles (OMVs) of *Helicobacter pylori* alter cellular signalling pathways in human gastric epithelial cells

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Helicobacter pylori infection is associated with chronic gastritis, ulceration and carcinogenesis. Bacteria colonise the gastric mucosa and are able to interact with epithelial cells. We previously showed that the small outer membrane vesicles (OMVs), which are constantly shed from *H. pylori,* may also have a direct and potentially carcinogenic effect on epithelial cells.

We studied the effect of OMVs isolated from *H. pylori* on micronuclei formation, as an indicator of DNA damage, glutathione (GSH) levels, morphology and proliferation of gastric epithelial cells. OMV isolated from *H. pylori* strain 60190 increased formation of micronuclei, but this was absent from a VacA- mutant suggesting a dependence on the presence of *H. pylori* vacuolating cytotoxin. The levels of intracellular GSH and proliferation of a human gastric adenocarcinoma (AGS) cell line were significantly decreased after incubation with OMVs from both strains indicating that these phenomena are not linked to micronuclei formation. We are currently exploring the mechanism of micronuclei formation, but our results clearly show that OMV could be an alternative mechanism for delivery of *H. pylori* virulence factors to the gastric epithelium.

Executive Function at Early School Age in Children Born Very Preterm

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While very preterm birth places children at risk of global intellectual delay and academic underachievement, less is known about the specific aspects of cognitive function that may be vulnerable in this population. The aim of this paper is to describe executive function performance in a cohort of 100 children born very preterm (<1500g/<34wks gestational age) and 100 children born full term (matched for gender and time of delivery). As part of a prospective, longitudinal study, children underwent a comprehensive neurodevelopmental assessment at six years of age. A battery of executive function measures including the Tower of Hanoi, Detour Reaching Box (a measure of set-shifting and inhibitory control), Corsi Blocks and a Digit Span test, were administered. These measures were supplemented by examiner ratings of behaviour during testing and parental ratings from the Behaviour Rating Inventory of Executive Function. Results revealed significant between-group differences in

childrens' performance on task measures of spatial working memory ($p<0.01$), inhibitory control ($p<0.05$), set-shifting ($p<0.05$), and planning/problem-solving ($p<0.05$), with children born very preterm showing impaired performance across these measures relative to their peers. Parents and examiners were also more likely to rate children born very preterm as showing executive impairments in the areas of initiation, planning, inhibitory control, flexibility, working memory and self-monitoring ($p<0.01$). These differences persisted when children with low general cognitive scores or moderate-severe cerebral palsy were excluded from analysis. Results suggest the presence of pervasive deficits across a range of domains of executive function in children born very preterm.

Oxidation of mitochondrial peroxiredoxin 3 during the initiation of receptor-mediated apoptosis

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It is hypothesized that activation of death receptors disrupts the redox homeostasis of cells and that this contributes to the process of apoptosis. We studied Jurkat T lymphoma cells undergoing Fas-mediated apoptosis and monitored oxidation of the peroxiredoxins, which are extremely sensitive to increases in H_2O_2 and disruption of the thioredoxin system. The only detectable change during the initiation of apoptosis was oxidation of mitochondrial peroxiredoxin 3. The protein was detected as a disulfide-linked intermolecular dimer, possibly resulting from inhibition of the NADPH/ thioredoxin reductase/ thioredoxin system responsible for maintaining peroxiredoxin 3 in its reduced form. Peroxiredoxin 3 oxidation was an early event, occurring within the same timeframe as caspase activation and cytochrome c release. It preceded other major apoptotic events including mitochondrial permeability transition, phosphatidylserine exposure and the oxidative stress associated with a decrease in cell viability. Peroxiredoxin 3 oxidation was also observed in U937 cells stimulated with TNF We propose that peroxiredoxin 3 oxidation is an early event in death receptor-mediated apoptosis, and that mitochondrial H_2O_2 levels rise following oxidation.

Spatial localisation of inflammatory and oxidative markers within an advanced atherosclerotic plaque

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Atherosclerosis is a chronic inflammatory disease characterised by the presence of a number of immune stimulants such as ©-interferon. ©-Interferon stimulation of macrophages causes the release of 7,8-dihydroneopterin, which can be oxidised to the highly fluorescent compound neopterin. A strong correlation in plasma neopterin and

the severity of vascular disease has been reported. 7,8-dihydroneopterin has also been shown to be a potent antioxidant which can inhibit lipoprotein oxidation, a key process in plaque development.

The aim of the study is to quantify and spatially localise oxidative and inflammatory markers within an advanced plaque to examine the antioxidant/oxidative balance.

Atherosclerotic plaques were surgically removed from the carotid artery bifurcation. The concentrations of neopterin, vitamin E, protein oxidation (carbonyls and protein-DOPA), cholesterol and lipid oxides (TBARS) was quantified. We present data from one of these plaques. This heavily calcified plaque was sectioned into 6 sections, with section one being proximal to the bifurcation.

Sections 2, 3 and 5 had higher levels of this calcification while neopterin and protein levels were significantly lower in these sections. The concentration of vitamin E, protein carbonyls and cholesterol showed no significant variation along the plaque. Sections 1, 3 and 5 have significantly higher concentrations of lipid oxides per mole of cholesterol.

The spatial localisation of the neopterin to the level of calcification suggests a relationship within this plaque. Surprisingly the level of cholesterol and vitamin E was not related to the level of lipid oxidation occurring. The level of neopterin measured suggests that 7,8-dihydroneopterin may have been functioning as an antioxidant.

Activation of paracetamol by horseradish peroxidase and cytochrome P450 1A2 in human endothelial cells and cancer cell lines

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Gene directed enzyme prodrug therapy (GDEPT) of cancer aims to improve the selectivity of chemotherapy by gene transfer, thus enabling target cells to convert non-toxic prodrugs to cytotoxic drugs. The peroxidase from horseradish (HRP) and the human cytochrome P450 isoform 1A2 (CYP1A2) are both able to convert the well known analgesic agent, paracetamol, to a potent cytotoxin (NABQ).

Thus far, gene therapy has targeted the malignant cells, but gene delivery has proven to be a considerable obstacle. To overcome this hurdle, we wish to target the tumour vasculature. In addition to improved delivery of genes and prodrugs, even minor damage to the vasculature can potentially amplify into tumour destruction. We hypothesise that the vascular endothelial cells may be particularly sensitive to NABQ, since damage to the endothelial cells *in vivo* is reported to be the first indication of paracetamol overdose.

Human tumour cells, normal human fibroblasts and human primary endothelial cells were exposed to purified HRP enzyme and paracetamol. Endothelial cells appeared to show increased sensitivity to HRP-activated paracetamol. Human cancer and

endothelial cells were also gene-modified to express either HRP or CYP1A2, and exposed to paracetamol. The drug response was monitored *in vitro* using cell viability assays.

The tumour vasculature is different from the normal vasculature, making it an ideal target for therapeutic interventions. Paracetamol has well defined clinical characteristics and is the most widely used analgesic in the world. The combination of tumour vascular targeted gene therapy with paracetamol has therefore potential for rapid translation into the clinic.

The effect of block co-polymer on hepatotoxicity following paracetamol overdose

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Liver sinusoidal endothelial cells (LSECs) form a 'sieve' between the sinusoidal blood and hepatocytes. Paracetamol in high doses damages LSECs and hepatocytes, with LSEC damage being the earliest stage in this process. Treatment with a block co-polymer coats the LSEC and causes temporary defenestration of the liver sieve. Therefore administration of block-copolymer could reduce paracetamol-induced liver toxicity through its protective effects on LSECs. To test this, block co-polymer was given to rats via intraperitoneal injection at various time periods in relation to paracetamol overdose (750 mg/kg intraperitoneal) and compared to saline-treated controls.

Liver function tests, light microscopy and scanning electron microscopy were analysed. Treatment with block co-polymer given either 18 hrs prior or at the time of paracetamol administration prevented paracetamol-induced increases in AST ($p<0.05$). Centrilobular necrosis was markedly reduced in block co-polymer and paracetamol treated animals compared to paracetamol-only animals ($p<0.001$). The results were inconsistent when block-copolymer was given 2 hrs after paracetamol. Scanning electron microscopy revealed that there was a decrease in LSEC injury as determined by large gap formation when block co-polymer was administered.

These preliminary results suggest that treatment with block co-polymer protects LSECs from paracetamol induced damage and prevents accompanying hepatoxicity as measured by centrilobular necrosis and AST levels.

Neighbourhood access to gambling opportunities and gambling behaviour in New Zealand

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Problem gambling is increasingly being recognised as a public health issue that affects the physical, mental and social well being of gamblers, as well as their close associates. There is limited research exploring potential neighbourhood effects upon individual gambling behaviour. The aim of this study is to investigate associations between neighbourhood accessibility to gambling outlets and individual gambling behaviour in New Zealand.

Distances from the population-weighted centroid of each neighbourhood to the closest gambling venue (Non-casino gaming machine venue, TAB, Casino) were calculated for 38,350 neighbourhoods using Geographical Information Systems. These neighbourhood measures of accessibility were appended to the 2002/03 New Zealand Health Survey of 12,529 adults. Two-level logistic regression models were fitted to examine the effects of neighbourhood access upon individual gambling behaviour, controlling for potential individual and neighbourhood level confounders.

People living in the quartile of neighbourhoods nearest to gambling venues were significantly more likely to have gambled at these types of venues in the last year (OR=1.51; 1.22-1.87), and to be problem gamblers (OR=2.05; 1.14-3.68), compared with those in the quartile of neighbourhoods furthest from gambling venues. When examined independently, neighbourhood access to specific types of gambling venues was similarly related to both gambling and problem gambling behaviour with non-casino gaming machine venues (gambling: OR=1.67, 1.28-2.18; problem gambling: OR=2.71, 1.45-5.07) and TABs (gambling: OR=1.67, 1.20-2.15; problem gambling: OR=2.70, 1.03-7.05).

Neighbourhood access to gambling opportunities is related to gambling and problem gambling behaviour and contributes substantially to neighbourhood inequalities in gambling over and above individual level characteristics.

Prognostic neurohormone levels in acute coronary syndromes patients of New Zealand European, Maori and Pacific ancestry

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Age, gender, body mass index (BMI) and ventricular function are known to modulate plasma natriuretic peptide levels in healthy and diseased subjects, but whether levels of these prognostic neurohormones differ between individuals of different ethnicities

is unknown. This research investigated natriuretic peptide concentrations in acute coronary syndromes (ACS) patients (n=1151) of New Zealand European, Maori and Pacific descent. Radioimmunoassay was used to analyse circulating levels of atrial natriuretic peptide (ANP), N-terminal pro-ANP (NT-proANP), brain natriuretic peptide (BNP), N-terminal pro-BNP (NT-proBNP), and C-type natriuretic peptide (CNP). Echocardiography was performed and cardiovascular risk factors recorded. In Maori/Pacific patients, levels of all neurohormones were lower ($p<0.05$), except NT-proBNP ($p=0.054$). Maori/Pacific patients had an earlier age of ACS onset (59 vs 68yrs) a higher BMI (30 vs 27kg/m^2), larger waists (99 vs 93cm), higher diastolic blood pressure (85 vs 73 mm Hg) lower HDL cholesterol (1.12 vs 1.36mmol/L) ($p<0.018$) and experienced myocardial infarction, heart failure, cerebrovascular accident, renal disease, type-II diabetes, and hypertension at an earlier age ($p<0.025$). There was no difference in left ventricular ejection fraction, left ventricular mass index or E/E1 (index of atrial filling), suggesting patients from both groups had equivalent severity of disease. Age of ACS onset, left ventricular mass, E/E1 but not ethnic group independently predicted all neurohormone levels except CNP ($p<0.05$). BMI and gender were independently prognostic of BNP and NT-proBNP levels ($p<0.01$). The lower natriuretic peptide levels observed in Maori/Pacific compared with European patients may be explained by their younger age of ACS onset and higher BMI.

Variation in New Zealand Public Hospital Outcomes, 2002 – 2005

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New Zealand public hospitals account for a substantial proportion of government expenditure on health. Evaluation of hospital performance is therefore an important concern of health services research. Issues of patient safety and health care quality are assuming increasingly high research and policy priority and these issues necessitate evaluation and comparison of health outcomes. Variations in hospital outcome rates suggest areas for quality improvement.

This study uses administrative data, drawn from the New Zealand National Minimum Dataset for public hospital discharges to compare patient outcomes across the New Zealand public hospital sector. We study variability in a series of mortality and patient safety indicators, developed by the Agency for Health Research and Quality. We use hierarchical Bayesian models to estimate the between-hospital distribution of comorbidity adjusted outcome rates. This approach accounts appropriately for the instability of outcome rates for smaller hospitals.

Preliminary results indicate, that after comorbidity adjustment and proper allowance for statistical uncertainty, there is close to two-fold variation between top and bottom of the hospital outcome distribution, for several outcomes. Despite some consistency in the degree of between-hospital variability for the outcomes studied, individual

hospitals often exhibit inconsistent relative performance with respect to different outcomes. This suggests that, rather than the existence of a generic "hospital effect" on patient outcomes, hospital effects may operate at a ward or service level.

A Mathematical Single Nephron Model

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Kidneys regulate water and electolyte outputs, excrete harmful metabolic wastes, and regulate blood pressure through controlling blood volume. The nephron is the main functional unit of the kidney, with approximately one million nephrons in each.

Current whole-organ models are based on mean field models which assume nephrons are homogeneous. In reality there are significant structural, biochemical and functional variations. A more accurate model of whole kidney function may be possible by incorporating stochastic variability between nephrons. Current single nephron models capture nephron dynamics quite accurately. However, they are too computationally expensive to run in a stochastic framework. This research aims to develop a computationally inexpensive single nephron model that captures these dynamics. This model will then be expanded to create a representation of whole-organ function.

The computationally expensive single nephron mathematical model of Holstein-Rathlou & Marsh (1990) has been simplified. This simplified model captures the tubuloglomerular feedback mechanism and tubular dynamics of the computationally expensive model. This model can now be implemented in a stochastic framework and a range of pathological and physiological states can be explored.

Vessel directed enzyme-prodrug therapy for cancer

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Gene therapy is a relatively novel cancer treatment which can potentially exploit differences between tumour and normal blood vessels for targeted therapy. Gene directed enzyme-prodrug therapy (GDEPT) consists of two components: delivery of an enzyme-encoding transgene to target cells, followed by systemic administration of a prodrug substrate. The prodrug is converted by the enzyme to a toxin by target cells. Vascular endothelial cells lining tumour blood vessels are an ideal anti-cancer target as they are systemically accessible and minor vascular damage can lead to macro-regional tumour destruction.

Gene transfer using non-viral transfection methods was optimised into human umbilical vein endothelial cells (HUVEC). Nine transfection reagents were evaluated using a green fluorescent reporter protein and FACS analysis. Transfection efficiency ranged from <1% to 50%, with optimised transfection efficiency using Lipofectamine LTX (Invitrogen) routinely reaching 30%.

Herpes simplex virus thymidine kinase/ganciclovir, E. coli nitroreductase/CB1954, and human cytochrome P450 reductase/tirapazamine are three enzyme-prodrug combinations shown to cause tumour regression *in vivo*. These enzyme-prodrug combinations, along with novel prodrugs, were tested *in vitro* for their ability to reduce viability of endothelial cells. HUVEC were transfected with the enzyme-encoding genes and treated with the corresponding prodrugs for three days. BrdU, MTT and Neutral Red assays were used to compare the effectiveness of each enzyme-prodrug combination.

These studies are intended to support future research comparing established versus novel enzyme-prodrug combinations on human endothelial cell survival to identify the optimal system to target tumour endothelium.

Failure of cell metabolism as mechanism of atherosclerosis

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The uptake of oxidized low-density lipoproteins (oxLDL) by macrophages is a key event in the initiation and development of atherosclerotic lesions. Our study is characterizing the effect of oxLDL on the cells' metabolic function by measuring the activity of glyceraldehyde-3-phosphate dehydrogenase (GAPDH), lactate dehydrogenase (LDH) and ATP synthesis. The human monocyte like cell line U937 was used in this study as a model of human macrophages. U937 cells at 0.5 x 10^6 cells/ml were incubated in RPMI-1640 with 0.5 mg/ml oxLDL before measuring various metabolic functions.

After 3 hours of incubation with oxLDL the cell viability dropped to 50% after three hours, with only 20% viability remaining after ten hours. During this time there was a marked decrease in cell metabolic activity in both lactate and ATP production. The activity of the glycolytic enzyme GAPDH rapidly decreased by 75% with total loss of activity by ten hours. LDH activity loss did not occur till after 6 hours when cell lysis became significant. The loss of GAPDH activity and overall lactate production was therefore due to enzyme inactivation. Glutathione, a key regulator of cellular reducing potential decreased by 30% after ten hours suggesting that oxidative stress triggered by the oxLDL, caused GAPDH inactivation and the failure of ATP generation within the cells. OxLDL inhibition of metabolism may be a key process occurring in the development of advanced atherosclerotic plaques which are characterised by large regions of necrotic cells.

Dynamic Myogenic Autoregulation in the Rat Kidney: A Whole Organ Model

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A transient 1D mathematical model of whole-organ renal autoregulation in the rat is presented, with the focus being the myogenic response on multiple levels of the renal vasculature. Morphological data derived from micro-CT imaging has been employed to divide the arterial structure via a Strahler ordering scheme, providing eleven distinct vascular levels which are represented as variable resistors in series. A previously published model of the myogenic response based on wall tension is expanded upon and adapted to fit the response of each level, corresponding to a distally dominant resistance distribution with the highest contributions localized to the afferent arterioles and interlobular arteries. The mathematical model is developed to include the effects of in-vivo viscosity variation and flow-induced dilation via endothelial nitric oxide production. Computer simulation results of the autoregulatory response to pressure perturbations are examined and validated with experimental data, showing good agreement in both isolated vessels and the intact system. Experiments in conscious, denervated, TGF-inhibited rats correspond most accurately to exclusively myogenic autoregulation, and the model predicts a 35.5 percent change in flow over the pressure range between 70 and 120 mmHg, while experimentally a 38.7 percent change is observed over the same range. The mathematical model supports the hypothesis that change in circumferential wall tension is the catalyst for the myogenic response, and provides a basis for examining the steady state and transient characteristics of the whole-organ renal myogenic response in the rat, as well as the modulatory influences of metabolic and hemodynamic factors.

Attitudes to, and Utilisation of Dental Care by Canterbury Adolescents

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Despite the widespread availability of free dental care to all teenagers, from the start of school year 9 until they turn 18 years, many adolescents are not accessing care. This study aimed to investigate potential barriers and enabling factors to accessing dental care, and to determine associations between age, ethnic group and access to dental care by teenagers. The data were collected using a self-completion questionnaire that was developed and tested with two adolescent focus groups. A survey was carried out in November, 2006 involving 944 Year10 and Year12 adolescents from 11 Canterbury High Schools. Data were analysed with SPSS v14 using chi square tests with significance levels set at $p<0.05$. Results: Analysis showed

that 12 percent of adolescents were unaware of the availability of free dental care. While 90 percent of adolescents had accessed care in either 2005 or 2006, only 51 percent had been to a dentist in both years. The utilization was lower for Maori and Pacific adolescents compared to NZ European and Others (2005-65%, 52% & 76% respectively p=0.001, 2006-59%, 50% & 72% respectively p<0.001). Self-perception of adolescents that routine dental visits were not important was the main reason for not accessing care. Nearly one in five adolescents had failed to attended their dental appointment, the main reason being forgetfulness. For those who had accessed care there was greater parental involvement in appointment making and dental appointment attendance. One in four adolescents had signs of being anxious about dental treatment and four percent had failed to attend their dental appointment because of fear or anxiety. Conclusions: The findings show that there are inequalities in accessing care and suggest that further interventions should be developed to address the barriers such as fear, anxiety, and the self-perception of adolescents. There is also a strong need to educate and encourage parents involvement in their teenagers dental care.

Asthma and Respiratory Conditions in a Sample of New Entrant Children in Christchurch

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The study's purpose was to examine relationships between asthma and respiratory conditions, academic achievement, and behaviour during the first year of school. A randomised list of primary schools within 3 socioeconomic bands was used to sample 8 schools with potential subjects. Inclusion criteria were age 5 at start of school, English, Maori or Pacific Island as first language and no identified special needs. Parents/carers of 318 children were contacted and 298 children were recruited (93.7%) by March 2007, with an even gender mix. Parents were interviewed using questions from the International Study of Asthma and Allergies in Children (ISAAC). Follow-up information about reported respiratory conditions was obtained from physicians. Parents reported family income and ethnicity using NZ Census categories. A clinical review of the information was conducted. Of 5 year old subjects, 40.9% were reported as ever having wheeze. The respiratory conditions of subjects were categorized as follows: 20.1% current asthma; 10.4 % current wheeze; 2.7% current dry cough; 1.7% current other; 7.4% past asthma; 4.7% past wheeze; 5.0% past other; 0.3% past dry cough and 47.7% control. Chi-square analyses showed no significant differences in the rate of current asthma by gender ($|^2$ =3.414, p =.065); NZ Census 2001 family income quintiles for Christchurch ($|^2$ =3.987, p =.564) or ethnic group ($|^2$ =1.405, p =.843). No significant differences with data collected for ISAAC Phase 1 on 6-7 year olds in 1992-1993 were shown. The academic achievement and behaviour of the subjects will be discussed in subsequent reports.

Age cohort effects of lifetime drug use in Te Rau Hinengaro: the New Zealand Mental Health Survey

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Use of four drug groups are compared by age and gender using data from the New Zealand Mental Health Survey, a cross-sectional community survey in 2003-2004 of people aged 16 years and over. They were asked if they had ever used drugs (alcohol, cannabis, cocaine and opioids) and the age of first use. Percentage who have ever used drugs are reported. The hazard ratios (HR) for age and gender are estimated using Cox's proportional hazards model. The cohort effect of age is examined further using life tables.

The response rate of 73.3% yielded 12,992 interviews. Alcohol was the most commonly used drug (94.6%) followed by cannabis (41.6%), cocaine (4.2%) and opioids (2.9%). Men are more at risk of ever using any drug group ($p<0.001$), more so for cocaine and opioids (both HRs = 2.3) than alcohol and cannabis (both HRs = 1.4). Younger participants are at risk of ever using any drug group ($p<0.001$). For participants under 65 years of age, the median age of first use in each age cohort was always lowest for alcohol, then cannabis, then opioids, then cocaine. The median age of first use for each drug was always lowest for the 16 to 24 year old cohort, then 25 to 44 year olds, then 45 to 64 year olds.

The analysis shows young New Zealanders are using drugs at an earlier age than their elders. Education and Public Health interventions to prevent or to delay drug use need to occur before and during adolescence.

Population Mixing and Type 1 Diabetes in Canterbury

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Over the past twenty years the incidence of type 1 diabetes (T1D) in children has risen in many developed countries. The most recent data available for the Canterbury region in New Zealand support this trend. However, the reasons for this increase and the precise aetiology of this disease remain unclear. Environmental factors are thought to be important and infections introduced through population mixing have recently been linked to T1D in children.

This research explored the geographical epidemiology of T1D and its possible association with population mixing in the Canterbury region of New Zealand for the

period 1980-2004. Children aged 0 to 14 years at the time of disease onset were included in this study from data provided by the Lipid & Diabetes Research Group at Christchurch Hospital.

T1D standardised incidence ratios were examined over time and space, between areas of differing socioeconomic status, and in urban and rural New Zealand. Cluster analysis was employed to test for spatial-temporal clustering of the disease and Poisson regression analyses were utilised to investigate the association between T1D and various population mixing measures at the area level. The incidence of T1D increased considerably over the study period from 5.27 cases per 100,000 population in 1980 to 32.45 in 2004, and preliminary results reveal higher rates in the more affluent areas of Canterbury and in areas classed as satellite urban communities. Spatial-temporal clustering of cases was noted and there was some evidence to support a connection with population mixing.

A Review of The Fertility Centre Semen Storage

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New Zealand is well known for having one of the world's highest vasectomy rates. The Fertility Centre has provided pre-vasectomy semen storage since in March 1993.

This study reviews the demographic data, the quality of the samples pre and post thaw and subsequent use for assisted reproductive treatment.

Between March 1993 and March 2007 we froze 785 samples for 498 clients. The average number of samples a person froze is 1.6. This service has evolved into freezing for several categories: pre-vasectomy, treatment back-up, vas-reversal, post surgical sperm, pre-medication, pre-surgery, personal, and medication.

Ages ranged from 18 years to 65 years. Of the samples used, 71% were used within 12 months. Other samples were discarded on average after 3.3 years. Overall, only a small percentage of clients have come back to use the samples they froze. From 498 clients, 43 have used their samples (8.6%). There have been 10 live births, 7 from treatment back-up, 2 from vas-reversals, and 1 from pre-vasectomy storage samples. Samples have been stored by 12% of the men for more than 10 years.

Considerable time and effort is involved in providing this service. These results indicate that semen storage provides definite treatment options for some couples.

Angiotensin-converting enzyme 2 gene variants and survival in an acute coronary syndromes cohort

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Polymorphisms of the angiotensin-converting enzyme 2 (ACE2) gene, which is located on the X-chromosome, have been associated with hypertension and left ventricular hypertrophy in previous studies. We tested the hypothesis that the rare alleles of two ACE2 gene variants are associated with risk factors for and adverse outcome following acute coronary syndromes (ACS) events. ACS patients were genotyped for the G8790A (n=1042) and A1075G SNPs (n=1038) of the ACE2 gene. These genetic markers were tested for association with baseline measurements, neurohormonal profiles, echocardiographic measurements and clinical outcome, over a median 2.2 years follow-up. As the ACE2 gene is X-linked, analyses were performed separately for males and females. The A1075 allele was significantly associated with covariate-adjusted poorer survival in male patients (unadjusted Hazard Ratio=1.14, p=0.558; adjusted HR=1.80, p=0.033; covariates included: age, centre, ®-blocker treatment, NTproBNP and peak creatine kinase levels, creatinine clearance, ethnicity and G8790A genotype) and also in female patients with AA compared to GG genotype (unadjusted HR=1.05, p=0.902; adjusted HR=7.09, p=0.043). The A8790 allele was associated at baseline with a higher incidence of renal disease (p=0.04) and abstinence from alcohol (p=0.008) in males. The G1075 (p<0.001) and A8790 (p=0.001) alleles were significantly more frequent in patients of Maori compared to European ancestry. Females heterozygous for either or both the ACE2 G8790A and A1075G SNPs (n=188) or homozygous for the diplotype G8790-G1075 (n=27) had a mean age at ACS admission 4 years older than the remaining (n=92) female patients (72 v. 68 years, p=0.038). The ACE2 A1075 allele may be useful for predicting survival following ACS.

Studying Attentional Ability in Children Born Very Preterm

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Studies on the neuropsychological outcome of children born very preterm indicate these children are more likely to suffer specific learning disabilities than full-term children. Common problems include poor attention and inhibitory control. Such impairments can lead to later learning and behavioural problems which may affect academic performance and other variables relevant to adult adjustment. It has been posited that attention and learning difficulties that often characterize preterm children

may relate to a central deficit in the ability to simultaneously process and integrate multiple pieces of information.

No studies have examined the impact of premature birth on the ability to suppress activated but goal-irrelevant mental representations. A means to study such control processes in selective attention and to identify potential vulnerabilities in preterm children is the negative priming (NP) procedure. NP refers to delayed responding to a stimulus recently ignored as a distractor. Recent developmental NP studies present strong evidence to suggest NP reflects inhibitory control processes. I outline a new NP study that is currently running with the Canterbury Child Development Research Group in Christchurch that aims to compare the abilities of preterm and full-term children to overcome attentional competition generated by concurrent stimulus inputs.

NP procedures offer an innovative approach to the study of attention, often used in studies of typical cognition to examine inhibitory control when variables such as working memory and perceptual load are manipulated. Such manipulations may help identify the conditions under which inhibitory control fails in preterm children, and whether this differs from full-term children.

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Functional asthma severity and impulsive behaviour in 6 and 7 year old children

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There is evidence that children with asthma exhibit more externalizing behaviour problems than other children. Impulsive behaviours can mark the onset and severity of externalizing behavior problems. The present paper reports an exploratory examination of relationships between functional asthma severity and impulsivity in 6 and 7 year old children with asthma (N = 16). Participants with varying functional asthma severity were recruited at age 5 from a larger community study (the Children's Learning Study). Parents completed items from three subscales of the Connors' Parent Rating Scale-Revised (CPRS-R), the *Hyperactive-Impulsive*, *Conners' Global Index: Restless-Impulsive* and the *DSM-IV Hyperactive-Impulsive* subscales. Children completed the Two Choice Impulsivity Paradigm (TCIP), a computer program measuring delay aversion type impulsive behaviours. Mean (SD) CPRS-R *Hyperactive-Impulsive*, *Conners' Global Index: Restless-Impulsive* and *DSM-IV Hyperactive-Impulsive* subscale scores were 59 (11), 56 (10) and 59 (10) respectively. There was no correlation between functional asthma severity and delay aversion or CPRS-R subscale scores and there was no evidence of a trend for such a relationship. Preliminary investigations conducted with a small sample of 6-7 year-old children with asthma thus suggest that functional asthma severity is not related to impulsivity.

Prx II Provides Protection against Neutrophil-Derived Oxidants in Human Erythrocytes

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The respiratory burst produced by neutrophils to combat infection results in the production of high amounts of HOCl, a highly reactive oxidant. HOCl reacts physiologically with amines to produce chloramines, which can react with biological targets and contribute to inflammatory tissue damage.

Our goal is to determine whether non-radical neutrophil-derived oxidants react with proteins in the peroxiredoxin/thioredoxin pathway as a mechanism of antioxidant defense in erythrocytes. We have compared the oxidation of peroxiredoxin II (Prx II, the third most abundant protein in the erythrocyte) to that of glutathione (GSH) in erythrocytes exposed to several oxidants: Tau-NHCl, Gly-NHCl, NH_2Cl and HOCl. Using spectrophotometric analyses, cellular consumption of oxidants was determined after 20-minute incubations. Following erythrocyte oxidant treatments, we investigated the oxidation of Prx II by Western Blotting, and the oxidation of GSH using an established HPLC protocol. Experiments were carried out both in phosphate-buffered saline (PBS) alone and in PBS containing glucose.

We have demonstrated that erythrocytes are impermeable to Tau-NHCl but that Gly-NHCl enters the cells slowly, while HOCl and NH_2Cl rapidly permeate the cells. Under conditions that include the presence of glucose, Prx II appears far more sensitive than GSH to oxidation by Gly-NHCl, HOCl and NH_2Cl in the erythrocyte. However, in the absence of glucose (preventing recycling of the antioxidants), GSH and Prx II are approximately equally sensitive to oxidation. Our results indicate that Prx II appears to play an important role in protecting the erythrocyte against oxidative damage from non-radical MPO-derived oxidants.

Examining the effectiveness of phonological awareness intervention for pre-school children with Down syndrome

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This presentation reports the findings of a multiple single-subject-design study investigating the effectiveness of an integrated phonological awareness intervention in facilitating phoneme awareness, letter knowledge and speech production. Participants were ten pre-school children with Down syndrome aged between 4; 4 (years; months) and 5; 5 (mean=4; 11*)* attending an early intervention centre. Children with known significant ongoing medical concerns or diagnosis of additional developmental disabilities were excluded. The experimental intervention involved a collaborative approach between the children's parents, teacher and speech-language therapist. The teacher and speech-language therapist each implemented the intervention for 20

minutes/week for 12 weeks (i.e. 8 hours total) and parents implemented the intervention four times/week throughout the intervention (approximately 12 hours total). All participants completed 48 trials at pre- and post-intervention with correct production of the target phoneme in the appropriate word position credited as correct. Participants scored between 0% and 18.7% correct at pre-intervention and between 20.8% and 70.8% at post-intervention demonstrating a gain of between 20.8% and 70.8%. Pre- and post-intervention speech sound data were analysed using paired t-tests and showed a significant treatment effect for all ten participants (each $p \leq .001$). Participants showed increased awareness of letter knowledge and initial phonemes in words but transfer to untrained phoneme awareness items failed to reach statistical significance. Individual profiles pre- and post-intervention showed there was considerable variation between the participants on all assessment measures. The techniques used in this intervention show promising results in facilitating phoneme awareness, letter knowledge and speech production in this population.

The effect of intracellular ascorbate on HIF-1 mediated gene expression and the phenotype of tumour cells

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The hypoxia-inducible factor (HIF)-1 controls the response of tumour cells to oxygen deprivation and changes in basic cell metabolism and it regulates the expression of many genes involved in glycolysis, iron transport, angiogenesis, cell survival and apoptosis. HIF-1 activity is tightly controlled by hydroxylation of the ⟨-subunit at proline and asparagine residues by members of the family of 2-oxoglutarate dioxygenases. These hydroxylation reactions do not occur in the absence of oxygen, 2-oxoglutarate or iron, or when the iron centre of the enzymes is poisoned by a non-redox cycling metal ion such as Co^{2+} or Ni^{2+}. The hydroxylases are known to require ascorbate for optimal activity, possibly to maintain the iron centre in a reduced state, but despite this, the effect of cellular ascorbate on HIF-1 activity has received little attention.

We have investigated the effect of intracellular ascorbate on HIF-1 protein levels and on HIF-1-mediated gene expression in human umbilical vein endothelial cells, in skin fibroblasts and in A431 tumour epithelial cells. We have found that basal levels of HIF-1 and its induction by hypoxia or by $CoCl_2$ were markedly inhibited when cells contained optimal levels of ascorbate. Gene expression was similarly affected, with VEGF and GLUT-1 up-regulation, and GFP expression in transiently transfected cells being inhibited by ascorbate. The results show that intracellular ascorbate is a major regulator of the hypoxic response and suggest that optimal levels of this vitamin will affect on HIF-1-regulated processes in the tumour microenvironment.

Asthma Severity and Internalising Behaviour Problems in Children with Asthma

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The study's aim was to examine relationships between asthma and internalising problems in 5-year old children (N = 66). Participants were selected from a larger community-based study. Three groups of children were identified, *never had asthma* (N = 22), *prior asthma* (N = 22), and *current asthma* (N = 22). Data were collected in face-to-face interviews with parents on child internalising behaviour, measured by the Behavior Problem Index (BPI) and on child asthma using International Study of Asthma and Allergies in Children (ISAAC) questions with physician follow-up. Parent responses to the ISAAC questions were used to calculate an asthma severity score. Children were additionally classified as having either low asthma severity (LAS) (N = 44), mild (N = 12) (MAS), or moderate/severe (N = 10) (MSAS). Mean (SD) BPI internalising behaviour scores for these groups were, LAS 1.89 (2.4), MAS 2.83 (2.3), and for the MSAS group 3.10 (2.03). A one-way ANOVA was performed to examine the relationship between children's internalising behaviours and their asthma severity across the three groups ($p = 0.22$). No statistically significant differences between any of the three asthma severity groups were identified. An independent samples *t*-test assessed whether children with higher internalising scores also have higher asthma severity scores. It was found that children with clinically significant internalising scores (n = 13) had higher asthma severity scores than other participants (n = 53) ($t(64) = -2.382$, $p = 0.02$). Findings indicated that internalising behaviour problems were associated with asthma severity in the participants.

Does recent cannabis use in New Zealand cluster geographically?

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To see if there are local influences on drug use, in addition to standard sociodemographic correlates, this paper investigates the extent of geographic clustering of cannabis use in the past year in New Zealand, at the census meshblock level (40-70 households) and within local authority areas. Te Rau Hinengaro, the New Zealand Mental Health Survey, was a national community face-to-face survey of people aged 16 years or above, selected within meshblocks. The response rate of

73.3% produced 12,992 interviews. Multi-level Alternating Logistic Regression was used to estimate the extent of clustering at each level. If there was no clustering at a particular level then the estimate would equal 1.0. Correlates were age, sex, ethnicity, education, marital status and income. Cannabis use was common: 42% had ever used cannabis and 13% had used in the previous year (31% in 16-24 year olds down to 0.3% in those aged 65 or over). There was clear clustering at the meshblock level (1.4; 95%CI 1.3, 1.5) and a small amount of clustering at the local authority level (1.09, 95%CI 1.03, 1.2). Estimates were only slightly reduced by the inclusion of individual level covariates: meshblock estimate of 1.3 (1.2, 1.4) and local authority estimate of 1.04 (1.00, 1.09). There is geographic clustering of recent cannabis use in New Zealand and this cannot be explained away by the socio-demographic characteristics of the people who live in each area. Possible explanations include the personal networks used to obtain supplies of cannabis and likely geographic clustering of suppliers.

A functional NADPH oxidase promotes neutrophil apoptosis by a caspase-independent mechanism

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Neutrophils play a prominent role in host defense. Upon phagocytosis of bacteria the NADPH oxidase generates large amounts of reactive oxygen species to facilitate bacterial killing. After the infection is cleared neutrophils undergo apoptosis. It is proposed that oxidants derived from the NADPH oxidase also have a fundamental role in mediating apoptosis of phagocytic neutrophils. In this study we used diphenyleneiodonium an inhibitor of the NADPH oxidase, and neutrophils isolated from an X-linked $gp91_{phox}$ knockout chronic granulomatous disease (CGD) mice to determine what role NADPH-derived oxidants have in the execution and resolution of apoptosis. We showed that the NADPH oxidase triggered exposure of phosphatidylserine and promoted uptake of phagocytic neutrophils by human macrophages. Phosphatidylserine exposure and macrophage uptake was not caspase-mediated, indeed, we showed that NADPH-derived oxidants actually prevented caspase activation in phagocytic neutrophils. Caspases are redox-sensitive thiol enzymes, and inactivation could be associated with oxidative stress within the neutrophil cytoplasm. Phagocytosis resulted in protein carbonyl formation in the cytosol. We proposed that the carbonyls were derived from NADPH oxidase-dependent lipid peroxidation generated reactive aldehydes such as 4-hydroxynonenal that diffuse into the cytoplasm to target thiol proteins including caspases. Consistent with the hypothesis, inhibitors of lipid peroxidation inhibited carbonyl formation and caspase inactivation, while exogenous 4-hydroxynonenal inactivated caspases and generated a similar pattern of protein carbonylation as phagocytosis. We conclude that neutrophil oxidants have a key role in driving the clearance of phagocytic neutrophils, via a novel mechanism because of excess lipid peroxidation in the phagosomal membrane producing an unfavourable environment for caspases.

Human macrophage necrotic cell death is triggered at threshold hypochlorite concentrations

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Hypochlorous acid (HOCl) within inflammatory sites is a significant source of oxidative damage to cells including immune cells. This research examines the mechanism of HOCl damage to human monocyte-derived macrophages with the aim of understanding the effect of various cellular protective mechanisms.

Macrophages were prepared from human blood monocytes by centrifugation and differentiated into macrophages over 14 days in RPMI-1640 supplemented with 10% heat inactivated human serum. Cell death was assessed by MTT reduction and trypan blue exclusion while caspase-3 activation was measured by immunoblot and enzymatic cleavage of Ac-DEVD-AMC. Intracellular calcium and mitochondrial membrane potential were measured by fluorescence microscopy using Fluo-3 calcium and TMRM binding probes, respectively.

Exposing human macrophages to increasing concentrations of HOCl showed little loss in cell viability until a specific threshold was reached upon which there was a rapid loss of cell viability, GSH levels, GAPDH activity, and mitochondrial membrane potential. Caspase-3 activation was not observed at any HOCl concentrations or any period of time after HOCl exposure.

The HOCl treatment caused an influx of calcium ions from media into cells, rather than from intracellular compartments. Calcium appears to have entered the cells via damaged plasma membranes, as tryptophan oxidation, a marker of protein oxidation, was also observed after HOCl treatment.

Our data suggests that HOCl causes necrotic cell death in macrophages due to the catastrophic failure of cellular mechanisms.

Is there a role for coenzyme Q10 supplementation in statin- induced myopathy?

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One postulated mechanism for statin-induced myopathic pain is mitochondrial dysfunction, secondary to depletion of coenzyme Q_{10} (CoQ), a key component of the mitochondrial electron transport chain. CoQ is increasing being used as a complementary medicine for statin-induced myopathies, but despite anecdotal reports

of improved statin tolerance with CoQ supplementation, this has not been evidence based. Recently two trials assessed the effect of CoQ supplementation on statin-induced myopathic pain. The first trial observed a significant reduction in myopathic pain in statin-treated patients following 30 days of CoQ (100 mg/day) supplementation in comparison to vitamin E (400IU/day).

In the second trial, we evaluated whether CoQ can reduce myalgia in patients with prior statin-induced myalgia in a placebo-controlled design. Forty-four patients were randomised to CoQ (200 mg/day) or matching lactose-filled placebo for 12 weeks in combination with upward dose titration of simvastatin from 10 mg/day to a maximum of 40 mg/day. Patients experiencing significant myalgia reduced their statin dose or discontinued treatment. The primary outcome was the change in myalgia scores that were assessed using a visual analogue scale to score pain severity. The study was powered (80%) to detect a 9mm difference in myalgia scores between treatment groups ($p<0.05$). This study was approved by the Upper South B Regional Ethics Committee.

Intention to treat analysis showed the change in myalgia scores did not differ between treatments (6.0 (2.1-8.8) mm vs 2.3 (0-12.8) mm, $p = 0.63$). Further, there was no difference in the number of patients who tolerated 40 mg/day simvastatin (CoQ 16/22 (73%) versus placebo 13/22 (59%), $p = 0.34$).

Our findings differ from the previous trial that demonstrated CoQ was associated with reduced statin-myopathic pain, but that study was limited by the lack of a placebo-control design and failure to standardise the dose and type of statin. Thus, larger, randomised, placebo-controlled trials are required to establish conclusively, whether CoQ is beneficial for treatment of statin-induced myopathy.

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Hydrocolpos in a child

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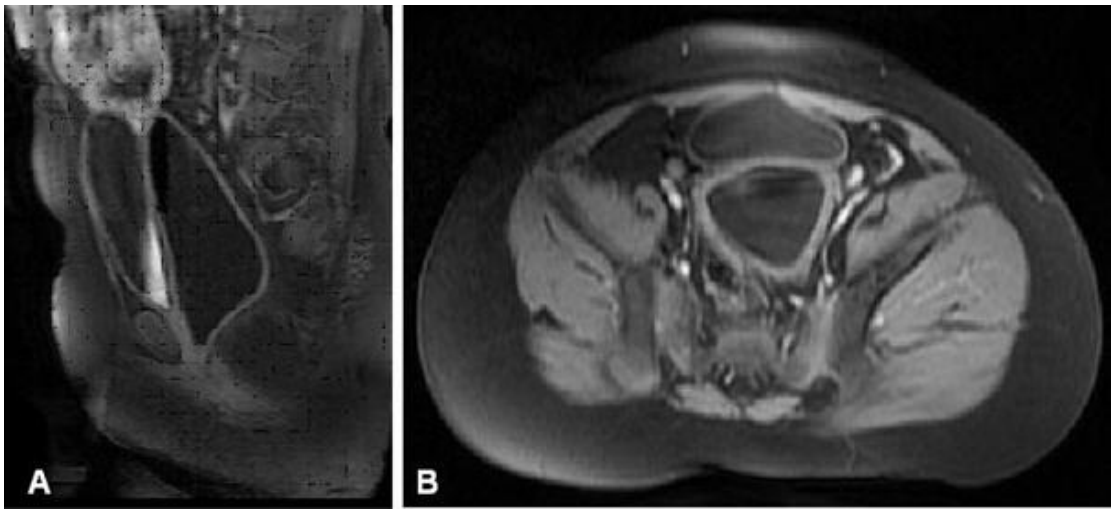
A 5-year-old girl was admitted our hospital complaining of intermittent abdominal pain. Ultrasonography revealed a large retrovesical hypoechogenic mass, however the sonographic findings were inadequate.

Magnetic resonance imaging (MRI) confirmed that the abdominal mass was fluid-filled vagina within the midline posterior to the bladder (Figure 1 A,B). These findings were consistent with a diagnosis of hydrocolpos.

Figure 1. Contrast-enhanced T1 weighted sagittal (A) and axial (B) MR images shows fluid-filled vagina with in the midline posterior to the bladder



Hydrocolpos was secondary to imperforated hymen. An elliptical excision of the membrane was performed and the obstructed material was evacuated. Postprocedural complication was not observed.

MRI is a useful complementary tool for assessing urogenital anomalies when ultrasonography is inadequate.[1]

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Reference:

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THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

A prepubescent mass

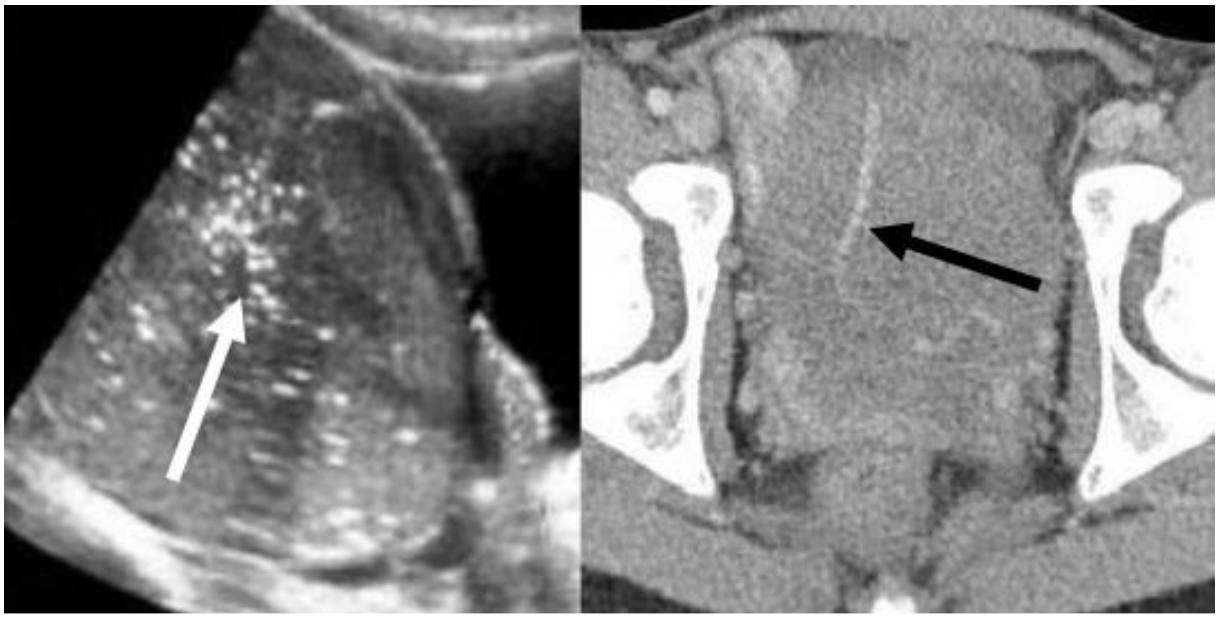
Helen C Addley, Lee A Grant, Robin Crawford, Evis Sala

Clinical

A 13-year-old girl presented with a 3-month history of abdominal fullness and discomfort. Raised tumour markers were present with a beta HCG of 75 μ/L (normal range 0–4 μ/L). There were no signs of puberty and a large abdominal mass extending from the xiphisternum to the pubic symphysis was clinically evident.

Transabdominal ultrasound examination (Figure 1A) and a CT scan were performed.

Figure 1A **Figure 1B**



What is the diagnosis?

Answer

The imaging demonstrated calcification (Figure 1A, white arrow) within a pelvic mass, with corresponding axial CT image post-intravenous contrast medium displaying vascular enhancement within the mass (Figure 1B, black arrow). The diagnosis was *Swyer syndrome with a bilateral dysgerminoma*.

Discussion

Swyer syndrome is a disorder of sex development in which the karyotype is 46XY and there is defective formation of the gonads (gonadal dysgenesis). In gonadal dysgenesis there is a high risk (approximately 30%) for development of germ cell tumours including dysgerminoma.

Germ cell tumours are the most common type of ovarian tumours after epithelial tumours and represent 15%–20% of ovarian tumours but less than 5% of malignant ovarian tumours. Of the malignant germ cell tumours, dysgerminomas are the most common and can be defined as pure dysgerminomas or mixed with other cell types of the germ cell tumours.

Gonadoblastomas are benign tumours and are regarded as an *in situ* tumour from which invasive germ cell tumours can develop. Transformation into a dysgerminoma, which has the potential to metastasise, has been noted to occur in approximately 30% of gonadoblastomas.

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THE NEW ZEALAND MEDICAL JOURNAL
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Regulation of costly pharmaceuticals in Australia, England and New Zealand

Drug appraisal is done through the National Institute for Health and Clinical Excellence (NICE) in the UK, the Pharmaceutical Benefits Advisory Committee (PBAC) in Australia, and the Pharmaceutical Management Agency of New Zealand (PHARMAC) in New Zealand. Each use different criteria for public funding of pharmaceuticals, but all include estimates of clinical effectiveness and cost-effectiveness.

Interestingly of the 10 drugs deemed least cost-effective by NICE between 1996 and 2005, all were approved for funding in the UK, 6 were approved in Australia, and 5 were approved in New Zealand. It should be noted, however, that the drug companies involved did not seek fundingin New Zealand for 3 of the drugs rejected in Australia.

So the PHARMAC scoresheet is 5 approvals and 2 rejections.

Med J Aust 2008;188:26–8.

Ovarian cancer and oral contraceptives (OCs)

Combination OCs with low-dose oestrogen are hailed as the very best form of contraception. It has been suggested that they may also low the risk of subsequent ovarian cancer.

This paper from the Oxford Epidemiology Unit seems to confirm this. They collected data for 23,257 women with ovarian cancer (cases) and 87,303 without ovarian cancer (controls) from 45 epidemiological studies in 21 countries. The relative risk of ovarian cancer in relation to oral contraceptive use was estimated, stratifying by study, age, parity, and hysterectomy. Their conclusion was that use of oral contraceptives confers long-term protection against ovarian cancer. These findings suggest that oral contraceptives have already prevented some 200,000 ovarian cancers and 100,000 deaths from the disease around the world.

Very good news as ovarian cancer is common and usually diagnosed when well advanced and incurable.

Lancet 2008;371:303–14.

Oral or topical non-steroidal anti-inflammatory drugs (NSAIDs) for chronic knee pain?

Two papers in a recent *BMJ* compare the value of a topical NSAID (ibuprofen gel) with oral use of the same drug for osteoarthritis of the knee. And it seems that topical NSAIDs may be a useful alternative to oral NSAIDs as the pain relief was about equal for both treatments except for those with the most severe symptoms.

It would be expected that adverse effects would be less with topical treatment but in these trials significant adverse effects were not seen with either treatment.

An editorial commentary was favourable and pointed out that a systematic review of a different topical NSAID found similar results and also established that topical treatments are cost-effective.

On the other hand it also offered the opinion that placebo effects explain most of the value of topical agents in osteoarthritis.

BMJ 2008;336:105–6 & 138–42 & 142–5.

Something else topical—lignocaine for pain relief in acute otitis media in children

Acute otitis media (AOM) is a very common problem in childhood with the majority of children having had the condition at least once by their third birthday.

The routine administration of antibiotics for AOM has recently come under scrutiny. The 2004 Cochrane review of antibiotics for AOM suggests minimal benefit from early use, with no reduction in pain for 24 hours and only a 30% reduction at 2–7 days. This report concerns a placebo-controlled trial comparing the topical use of 2% lignocaine or saline eardrops.

The results—rapid pain relief in the lignocaine group at 10 and 30 minutes. Sounds good, but would probably require repeated treatments and should be avoided in those with perforated eardrums.

Arch Dis Child 2008;93:40–4.

Ethics guidelines for clinical trials to be revised

The World Medical Association is proposing to again update its cornerstone statement of ethical principles regarding human experimentation.

The main issue prompting this are the ethical problems surrounding drug safety trials. One trial in particular resonated around the world. It was a trial involving HIV-positive women in the developing world. It gave one-half of all participants the drug azidothymidine to determine if a shorter-course treatment would be as, or almost as, effective as the proven longer-course treatment, and the other half a placebo, even though an existing treatment was available.

The fault is obvious but some ethicists defended the trial by arguing that the women would likely have received no treatment had the trial not been conducted.

No easy solution here.

CMAJ 2008;178:138.

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Increased advertising of medicines on New Zealand television since 2001

The advertising of medicines is controversial. In New Zealand most debates have been about direct to consumer advertising of prescription medicines.[1] However consumers are regularly exposed to advertisements for a wide range of both prescription and non-prescription medicines that attempt to convince them that a particular medicine is the solution to their problems.

Advertisers might argue that advertisements for medicines improve health by ensuring people are provided with information about how to treat their problems[2] but there is also the potential to increase irrational use of medicines, over-prescribing, inappropriate self-medication, and to contribute to excessive medicalisation of everyday life.[3,4] Thus it is important to monitor the extent of medicines advertising that consumers are exposed to.

In 2001–2, we examined the frequency of advertisements for medicines on New Zealand television and found an average of 1 medicines advertisement per 102 minutes, and considerable variation by channel and time of day.[5] In 2006, we set out to further describe the extent of medicines advertising and compare these with the 2001 data.

We identified the peak advertising hours from the previous study (4–8pm), and concentrated this study on those peak hours. We video-recorded 168 hours of television from the six free-to-air national New Zealand television channels between 1 May 2006 and 23 June 2006. A random sample stratified by channel and day of the week was recorded. Videotapes were viewed by two independent observers and all advertisements for medicines were identified, recorded and classified.

In 2001, the rate of medicines advertisements in the 4–8pm period was 0.72 advertisements per hour. By 2006, this had increased to 1.14 medicine advertisements per hour, a 58% increase.

Medicines advertisements per hour had significantly increased on TV1, TV2, and Prime, and significantly decreased on TV4, in comparison to 2001. Māori Television started between the two observation periods, and carried few advertisements for medicines (and comparatively fewer other advertisements compared to other channels). The majority of medicines advertisements screened (67.7%) were for general sale medicines.

Advertisements for prescription-only medicines declined as a proportion of medicines advertisements from 28% in 2001 to 17% in 2006. Medicines advertisements constituted 5.3% of all advertisements on television during our study period.

Medicines advertising is pervasive, and television watchers are exposed to a considerable number of medicines advertisements. Further research is needed into these advertisements (such as analysis of their health related claims, and compliance with regulations) and their impact on consumers.

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SSRI antidepressants for adolescents: Professor Werry replies to Medsafe

I shall summarise the main issues raised by Medsafe's official reply[1] to my earlier letter.[2]

- It took Medsafe 3 months to reply to my complaint to the Minister and the reply was forthcoming only after I made a second complaint.
- While it may be true that there was no written communication with Medsafe, Dr Jessamine was made aware of the unhappiness of NZ Child and Adolescent Psychiatrists in a discussion with me on Nine to Noon on National Radio. Further, Dr Jessamine in that discussion not only refused to accept our concerns but failed to give reasonable answers to the concerns that I raised.
- The main points I made in that discussion were that there was now good evidence from meta-analyses of studies that fluoxetine could have a beneficial effect on adolescent depression, that while suicidal thinking may increase there have been no reported actual suicides, that during the period that there has been a huge increase in prescribing of SSRIs there has been a major decline in youth suicide. It was therefore scientifically unacceptable to state that "the risk of SSRIs outweighed the benefits."
- Despite this, Medsafe continued and continues to make media statements that the risks outweigh the benefits.
- Medsafe has not made any reference to the latest research I quoted saying that in the US and Europe there has been a rise in the youth suicide rate pari passu with the scaremongering about SSRIs.
- Though I cannot be sure of this, Medsafe has indicated publically that cognitive behaviour therapy is the preferred option whereas the balance of evidence suggests it is better than placebo but inferior to fluoxetine.
- Medsafe cites in defence that both UK and US agencies have similar positions, but fails to note that, as in NZ, the child and adolescent psychiatric bodies have vigorously contested those positions as not only unsupportable but dangerous. Also, there is far too much tugging at the forelock to erstwhile overseas masters and not enough attention to local experts.
- The position that Medsafe states about waiting for a move from the drug companies on downward extension of SSRIs, suggests that it and PHARMAC are like some coy maiden waiting to be courted by rich and virile suitors (drug companies) before it will approve of a drug.

  As drug companies find both markets small and carrying out trials in children and adolescents difficult, they simply do not do them and then forbid the use below the age of 18! I pointed out to both Dr Jessamine and PHARMAC that such a position seriously disadvantages children and youth. I cited the

examples of adolescent schizophrenia where psychiatrists in this country have had, until very recently, to carry the responsibility for off-label use of antipsychotics because no antipsychotic was approved in this age group.

- I also cited as an example of a serious problem arising from this passivity in the unsatisfactory funded preparations for methylphenidate affecting 6000 children in NZ and the burden that this imposes upon schools and the resultant serious problem of poor compliance. There are in wide use overseas much better alternatives but no official body will take any initiative to register and fund these alternatives in NZ. There is currently a long-acting version (Ritalin LA) which would increase costs of prescribing only lightly.
- I proposed that instead of passivity that official bodies should ascertain health needs in minors and be pro active as they were for example over the meningococcal vaccination. I suggested that no drug should be registered in NZ for a condition which occurs not only in adulthood but also in children and adolescents unless the drug company could provide data as to the safety in young persons. Dr Jessamine dismissed this idea out of hand.
- When the interviewer on Nine to Noon asked me what I was going to do, I said that like most doctors in NZ, I would ignore Medsafe's advice. This has proven correct, as the prescribing of SSRI's to adolescents in NZ continues to increase.

The most serious issue that all this raised is of course that Medsafe has all the authority but none of the responsibility. What should happen as here, when its position is contrary to that held by experts in NZ? (we have experts in not only in paediatric psychopharmacology but also in adolescent depression). What should happen when doctors thumb their noses at advice from Medsafe?

In short, there is a crisis of confidence between profession and officialdom. Can we really afford to have an organisation in Government ostensibly there to protect the public which is ridiculously risk-aversive and stubbornly clings to opinion when wrong thus incurring the contempt of the profession and which appears, like Busby, to be a man of war without guns?

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Bacteraemia at Gisborne Hospital

An awareness of microbial infection and susceptibility patterns on a local scale is valuable information in guiding rational antibiotic treatment, especially with respect to initial therapy. On a wider scale, this information is helpful nationally to add to established knowledge of susceptibility patterns and sources of infection.

The aims of our study were to describe, within the Gisborne Hospital population, anatomical sources and places of acquisition of bacteraemia, the implicated organisms, antibiotic sensitivities, and the associated mortality. This was a retrospective audit of patients with positive blood cultures in the year starting 1 April 2006. Gisborne Hospital is a 91-bed secondary referral centre and serves a population of 44,828, 46.4% of which identify as Māori.[1]

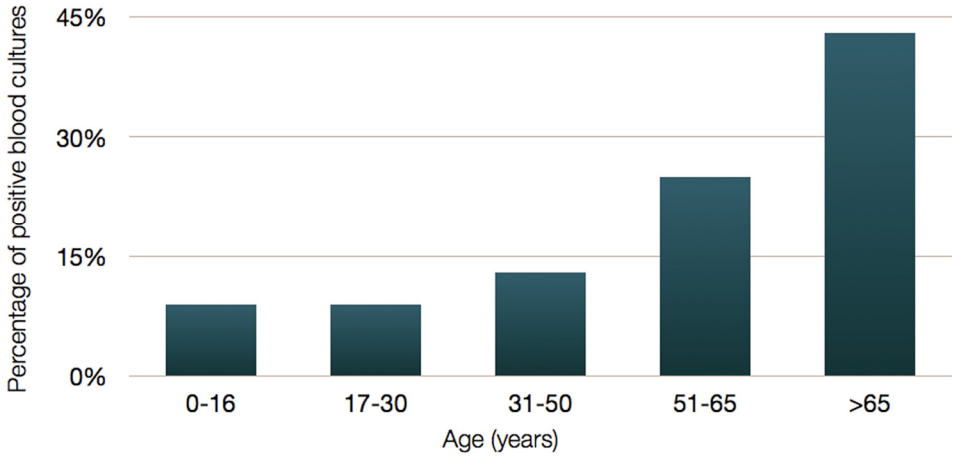
The Gisborne Hospital Microbiology Laboratory provided us with a list of all positive blood culture results for blood cultures (BacTech system) taken between 1 April 2006 and 31 March 2007. This included patient NHI numbers, blood culture acquisition dates, and the organism(s) grown. From this list—using a combination of electronic lab entries on the Gisborne Hospital lab result repository, iSOFT, and patient clinical notes—we gathered basic demographic information, microbial antibiotic sensitivities, previous hospital encounters, and mortality data.

Judgements could then be made as to the anatomical source of the bacteraemia, the place of acquisition (community-acquired bacteraemia [CAB] versus health-care-associated bacteraemia [HAB]) and whether or not the positive culture result represented a contaminant organism or a true bacteraemia. The definitions of contamination and of health-care-associated acquisition were those previously used by Raymond et al,[2] and included both inpatient and outpatient HAB.

Our results showed that over this 1-year period, the laboratory incubated 2279 individual blood culture bottles. This was determined to represent roughly 1200 blood culture sets, taking into account single paediatric bottles and occasional single bottles being sent for adult patients.

From these 1200 sets, there were 72 reported positive cultures. Of these 72, 19 were deemed contaminants, leaving 53 results from 52 patients deemed true positives, and therefore to represent true bacteraemias. This gave respective contamination and positivity rates of 1.6% and 4.4%. Twenty-seven (51%) of the bacteraemias occurred in patients identifying as Māori, 17 (32%) as NZ European, and 9 (17%) were not stated/other; 24 of the bacteraemias (45%) occurred in females, 29 (55%) in males. There were more bacteraemias with increasing age, 43% occurring in the over 65 age group, see Figure 1.

Figure 1. Age distribution of those patients returning positive blood cultures



There were 45 (85%) CAB, and 8 (15%) HAB. One of the 8 HAB was an outpatient, the remaining 7 were considered hospital-acquired. Thirty-three (62%) occurred in patients admitted under general medicine, 12 (23%) general surgery, 4 (8%) paediatrics, and the remainder shared between orthopaedics, gynaecology, and the emergency department.

Cellulitis/soft tissue infections (36%) were the most frequent source of CAB, see Table 1. Of the 8 HAB, 6 were from artificial material (2 PICC lines, 1 peripheral IV cannula, 1 portacath, 1 peritoneal catheter, and 1 JJ stent), 1 from an intra-abdominal source, and 1 unknown.

Table 1. Anatomical sources of community-acquired bacteraemias

Source	*n (%)*
Cellulitis/soft tissue	16 (36)
Lower respiratory	6 (13)
Intra-abdominal	6 (13)
Urinary tract	5 (11)
Bone/joint	5 (11)
Other	7 (11)

From the 53 positive blood cultures there were 4 polymicrobial results; the remainder represented single species growths. Streptococci were the most common cause of CAB, with 13% *S. pneumoniae* and 33% other streptococci, including 13% *S. pyogenes*, and one to two isolates of several other types.

Of the 8 HAB, there were 3 enterobacteriaceae, 1 *S. aureus*, 1 *E. coli*, 1 non-pneumococcal streptococci, 1 other gram-negative aerobe, 1 other gram-positive aerobe, and 1 due to anaerobes.

Table 2. Causative organisms of community-acquired bacteraemias

Organism	*n (%)*
E. coli	7 (16)
S. aureus	6 (13)
S. pneumoniae	6 (13)
Other streptococci	15 (33)
- *S. pyogenes*	6 (13)
Anaerobes	3 (7)
Enterobacteriaceae	2 (4)
N. meningitidis	2 (4)
C. albicans	1 (2)
Other gram-positive aerobes	6 (13)
–coagulase-negative staphylococci	4 (9)
Other gram-negative aerobes	1 (2)

The organisms cultured from CAB showed expected antimicrobial sensitivity, see Table 3. Seven out of nine (78%) community-acquired organisms plated against cefuroxime were susceptible, 8/8 (100%) were susceptible to ceftriaxone. There were no multiresistant organisms (e.g. MRSA, ESBL-producing organisms) grown in blood cultures during the audited time period.

Table 3. Number of growths resistant to plated antibiotics

Common organisms (*n*):	*E. coli* (7)	*S. aureus* (6)	*CNS** (4)	*S. pneumoniae* (6)
penicillin		3	4	1 **
oxacillin (flucloxacillin)		0	0	
amoxicillin	7			
augmentin	1			
tetracycline		0	0	0
erythromycin		1		0
vancomycin		0	0	0
gentamicin	0			
cefuroxime	0			
ceftriaxone	0			

**CNS: coagulase negative staphylococcus*

***the MIC for this growth of pneumococcus was 2.0 mg/L, classifying it as highly resistant*

Mortality following return of a positive blood culture was 7/53 (13%) at 1 week, and 10/53 (19%) at 1 month. The mean age for mortality was 74 (range 57–97) years. In the >65 years age group, 7/23 (30%) died within 1 month.

The majority of bacteraemias were due to community-acquired infection. The low rate of health-care-associated bacteraemia in comparison to a recent NZ report[2] likely reflects Gisborne Hospital being a secondary rather than tertiary centre, and therefore having fewer patients vulnerable to nosocomial infection, particularly ICU/CICU patients, haematology-oncology patients, and neonates.

Cellulitis/soft tissue infections represented by far the largest single cause for CAB. This differed from previous Wellington results,[2] however is consistent with local paediatric infections,[4] and supports the hypothesis that cellulitis rates within the Gisborne/East Coast region are disproportionately high in comparison to other New Zealand populations; even after taking into account possible inequalities due to frequency of blood culture taking for cellulitis in other centres.[5]

This result is mirrored by high rates of streptococcal infections, particularly *S. pyogenes*. The majority of HAB arose from artificial material, emphasising this as an important source of bacteraemia.[6,7]

Most CAB were susceptible to commonly used antimicrobials, supporting the notion that community-acquired infections generally remain susceptible to narrow-spectrum antibiotics. The small numbers of HAB makes it impossible to comment on their sensitivity patterns.

These data affirm the high mortality associated with infection causing bacteraemia.[6] They also show that advancing age is not only a risk factor for bacteraemic illness, but also carries a significantly higher mortality associated with bacteraemia.

In conclusion, this audit of positive blood cultures in a small district hospital in New Zealand shows that CAB largely involves common organisms, which retain sensitivity to a narrow antimicrobial spectrum.

Acknowledgements: We would like to thank Helen Geard and Iain Slack of the Gisborne Hospital Microbiology Lab for providing the necessary data for our audit. We also thank Chris Morell for tracking down many sets of notes for us.

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Do doctors in the First World have an obligation to help their counterparts in the developing World?

Infectious diseases kill more than 14 million people each year, mostly in Third World countries. For many of these diseases the drugs are too expensive, ineffective, or non-existent. This is because medicines are big business: the World relies almost exclusively on the increasingly consolidated and highly competitive multinational drug industry to generate new medicines. The result is that a large part of the World's population is ignored. The sad fact is that most of these diseases can be cured or prevented.

The dilemma that is facing medical professionals in developing nations is that they can not to anything about it. Having diagnosed an individual with a curable infectious disease the doctor knows that the patient cannot afford treatment, or in some cases like Zimbabwe, the treatment is not available. In most cases the failure of the health system is due to the political failure of the country. The failure of a politician to provide for his/her people has a direct correlation to the downward spiralling healthcare.

However, does this mean that health-care professionals in the developing World should ignore this? Being in a country like New Zealand, where the health system in comparison to most developed countries (let alone developing countries) is very good, there needs to be some way of giving something to communities that really need it.

Aid agencies and organisations like Médecins Sans Frontières, do provide some assistance but is this enough? Should we not take it upon ourselves as a country to help health professionals in the developing World? Simple things like Pando are a rare yet valuable necessity, which is not available.

Instead of working with NGOs and Aid Agencies, is it not possible for health professionals in New Zealand to tie up with a community centre in a chosen country and work with someone there to help?

According to the Pharmaceutical Research and Manufacturers of America (PhRMA) the following are the essential areas that are the main problems in the inability to provide a sustainable health care system in the developing world (2008):

- Countries need functioning public health systems to get health-care services and medicines to the sick.
- There are fewer than 3 doctors per 10,000 people in many countries in sub-Saharan Africa.
- Storage and distribution systems are nonexistent or are poorly managed, resulting in significant losses of medicines. World Bank estimates that for every $100 spent by African governments on drugs, only $12 worth of medicines reaches patients.

- Countries need adequate infrastructure—roads, transportation, electricity, clean water supply—to operate an effective health-care system.
- Health facilities are often located in urban areas, far away from rural populations most in need, or are not accessible to large numbers of the population via public transportation.

Of the five listed problems that have been raised, at least three of them can be addressed by forming a partnership with healthcare professionals in developing World. A case in point would be Zimbabwe, a country that is undergoing a huge economic struggle. This struggle has lead to individuals being unable to access basic healthcare, it has also left the health professionals in a catch-22 situation, as they are not able to see patients because they cannot afford the consultation costs, but the patient needs treatment. Even those that can afford treatment face a second hurdle in that there are resources to conduct basic blood tests, or obtain medicine for treatment.

In Zimbabwe, a country where HIV/AIDS affects 1 in 3, this plays a huge burden on an already crippled economy. By helping one community doctor, the effects will be felt by a large number of people. It all starts with doctors in countries like New Zealand stepping up and taking responsibility for what they can do.

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Professional Misconduct – Inappropriate Prescribing (Med06/44P)

Charge

Dr Rhys Michael Cullen, general practitioner of Auckland, was found guilty of professional misconduct following a charge by a Professional Conduct Committee. The charge alleged that the doctor:

1. In the period from on or about January 2003 until December 2004, wrote a substantial number of prescriptions for sudomyl (a Pseudoephedrine based drug) including but not exclusively at least 790 prescriptions (in excess of 46,000 tablets) for sudomyl dispensed by the Cleveland Road Pharmacy, when there was no medical/clinical justification for much of that prescribing;
2. In the period from on or about January 2003 until December 2004, wrote out prescriptions for sudomyl in the names of persons in the knowledge that those persons were unaware of that prescribing and/or that they would not be the persons receiving the prescribed medication and/or in breach of the Medicines Regulations 1984.

Background

The main issues raised by the charge related to the various means by which it was alleged the doctor acquired substantial quantities of sudomyl in 2003 and 2004; the explanations the doctor gave for doing so; and whether or not those explanations were truthful.

Sudomyl is the generic name for pseudoephedrine tablets. Pseudoephedrine is the main precursor used in the manufacture of the Class A drug methamphetamine, for which there is a very lucrative black market.

An unusual "bulk prescribing" arrangement was entered into with the Cleveland Road Pharmacy. Under this arrangement the doctor would frequently present several prescriptions for 60 sudomyl tablets in the names of patients. Often those patients were unaware of the existence or presentation of the prescriptions. He would pay the full price for the medication and take it away.

The majority of these pills did not find their way into the hands of his patients; but he gave small quantities of the pills directly to patients who needed them, and, he said, he destroyed the rest.

When comparing the numbers of sudomyl prescriptions filled at Cleveland Road Pharmacy by GPs other than the doctor it was apparent that the doctor's prescribing of sudomyl was substantial. In the period for which the doctor prescribed 46,364 pills of sudomyl (1 January 2003 to 31 December 2004), 10 other GPs in the same period prescribed 14 (lowest) to 1,339 (highest). In the period 1 January 2005 to 31 March

2007 the doctor prescribed 110,440 pills of sudomyl, and the 10 other GPs prescribed 10 (lowest) to 417 (highest).

In early 2004, the doctor presented a number of prescriptions at pharmacies around Auckland on which he had written in the names of associates, most of whom lived at the same Remuera address as he did.

The doctor acknowledged that he was a heavy prescriber of sudomyl. He advanced multiple explanations for the amassing of sudomyl:

- that it was for a mystery shopping survey;
- that it was to discover the sources of precursors for the manufacture of methamphetamine;
- that it was to advance a theory of savings; and
- that it was to research what influences demand for discretionary goods.

Reasons for Finding

The Health Practitioners Disciplinary Tribunal found that it was clear beyond doubt that in the period from January 2003 to December 2004, the doctor wrote a substantial number of prescriptions for sudomyl and that numerous prescriptions were written in the names of the patients without their knowledge and often without them even receiving any sudomyl tablets.

The Tribunal was satisfied that there was no intention of obtaining much of the sudomyl for administration to patients, and consequently there could be no clinical or medical justification for obtaining it. The Tribunal was satisfied that there were multiple breaches of the requirements of Medicines Regulations 39 and 40 during the period of the charge.

The Tribunal found that the facts of Particulars 1 and 2 were established.

The Tribunal went on to consider the various explanations which were given by the doctor for his prescribing practice and concluded that the explanations given were not believable, not credible, and not rational. The Tribunal was not satisfied that the doctor was doing any research and was satisfied, to the very high degree of persuasion required, that the acquisition of the sudomyl was for illegal purposes.

The Tribunal would have been prepared to find that discipline was undoubtedly warranted, even without the conclusion that substantial volumes of pseudoephedrine were being acquired for an unlawful purpose. On that more limited basis, there was a reckless disregard for professional standards, and it was quite irresponsible for patients' identities to be used on the scale which occurred here. There was a very significant breach of professional boundaries which would have supported the proposition that a disciplinary sanction was required for the purposes of protecting the public, for maintaining professional standards, and for punishing the practitioner.

When the further finding that the medication was obtained for illegal purposes was also considered then the justification for discipline was even more apparent. The Tribunal considered his conduct to be outrageous and reprehensible. The Tribunal was

satisfied that professional discipline was unquestionably warranted and the charge of professional misconduct was upheld.

Penalty

The Tribunal imposed the following penalty orders:

- The doctor's registration was cancelled.
- A fine of $15,000.00 was imposed.
- An order for costs of $25,000.00 was made.
- The doctor was ordered to deliver to Counties-Manukau District Health Board forthwith copies of all patient records currently held by him and not already transferred to another practitioner.
- The Tribunal directed that details of this decision were to be published in the New Zealand Medical Journal and on the Tribunal's website.

The full decisions relating to the case can be found on the Tribunal's web site at www.hpdt.org.nz
Reference No: Med06/44P.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

John William Corboy

08/12/1969—20/12/2007

John Corboy was a general surgical advanced trainee at Auckland City Hospital. Sadly he lost his battle with leukaemia just before Christmas 2007.

John began his life in Otorohanga, growing up as the eldest of five brothers on a dairy farm in Te Awamutu. He was schooled as a boarder at Sacred Heart College in Auckland as was the tradition in his family. After choosing between his childhood dreams of priest, pilot, or doctor, John completed Medical School at Otago University in 1994 where he initially stayed in Carrington Hall before flatting with friends.

John managed to fit an extraordinary life into the next 13 years while remaining quietly unassuming. He was a modest man of great internal strength, able to continue to give to others while he himself was fighting for his life. Writing an obituary is difficult as John did not let many know of his achievements and it is only on his passing that many of us found out the complex and courageous man he was.

John perhaps showed signs of things to come by organising his elective at NASA, Houston, studying space medicine and becoming a 3rd Dan in Seido Karate! Initially spending 2 years as a house officer in Wellington, he chose surgery (in his own words, his “calling”) and worked as a surgical registrar from 1997 to 1999.

In 1998 life turned upside down for John with the diagnosis of leukaemia. Determinately, John had ongoing treatment as he continued to work as a GP, obtaining his Primex and becoming a Fellow of the Royal New Zealand College of GPs.

He also gained a Diploma in Aviation Medicine and sat exams in Appearance Medicine which allowed him to become instrumental in laser tattoo removal in South Auckland, writing what was to become a national reference on the subject.

John continued to work towards a surgical career passing his Part I exam after a number of attempts, while unwell, but adamant he was to be treated just like anyone else. He then became an advanced general surgical trainee and worked at Auckland City Hospital 2006 to mid 2007. Never one to back down from the political, John was elected to be our (the NZ General Surgical Advanced Trainees) representative in 2006 and after one meeting in Australia had impressed everyone so much with his integrity and diligence that they promptly elected him to be the Chair of the RACSTA.

On 27 December 2006, John and his wife of 2 years, Susan, had a baby boy named William. Again beating the odds after all of his chemotherapy!

However, in a devastating blow he developed myelodysplasia and required a bone marrow transplant. With plans to return to work within the year John battled graft vs host disease but sadly in the company of his loved ones he passed away peacefully at home just before Christmas.

Dr Susan Gallop wrote this obituary. It originally appeared in *Nova*, the official staff newsletter for the Auckland District Health Board. We thank editor Sneha Paul for allowing us to republish it.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

GRANTS AWARDED MARCH 2008

At the March 2008 meeting of the Scientific Advisory Group of the National Heart Foundation, a total of 12 limited budget grants were awarded. The awards included 3 Small Project Grants and 9 Travel Grants.

SMALL PROJECT GRANTS

Ms Joanna Young

Lipid & Diabetes Research Group, Canterbury District Health Board

CoQ_{10}: Potential to reduce cardiovascular risk – extension study

$7,500 for 19 months.

Dr Nigel Lever

Department of Medicine, University of Auckland

Characteristics of induced and spontaneous Ventricular Fibrillation during myocardial ischaemia

$10,776 for 12 months.

Dr Adrienne Edwards

Department of Respiratory Medicine, Christchurch Hospital

Markers of pulmonary artery hypertension

$14,760 for 12 months.

TRAVEL GRANTS

Dr Hannah Badland

Centre for Physical Activity and Nutrition Research, Auckland University of Technology

10th International Congress of Behavioural Medicine, Tokyo, Japan.

Dr Kirsten Coppell

Edgar National Centre for Diabetes Research, University of Otago, Dunedin

5th World Congress on Prevention of Diabetes and its Complications, Helsinki, Finland.

Dr Jade Hollis-Moffatt

Department of Biochemistry, University of Otago, Dunedin

British Society for Rheumatology Annual Meeting & the British Health Professionals in Rheumatology Spring Meeting, Liverpool, UK.

Dr Barry Palmer

Department of Medicine, University of Otago, Christchurch

13th International Congress of Endocrinology, Rio de Janeiro, Brazil.

Dr Jeremy Shearman

School of Applied Science, Christchurch Polytechnic Institute of Technology

2008 American College of Sports Medicine 55th Annual Meeting, Indiana, USA.

Ms Alwyn Todd

Department of Medicine, University of Otago

Hypertension 2008, Berlin, Germany.

Dr Cliona Ni Mhurchu

Clinical Trials Research Unit, University of Auckland

2008 Annual Meeting of the International Society for Behaviour Nutrition and Physical Activity, Banff, Canada.

Ms Nicola Scott

Department of Medicine, University of Otago, Christchurch

13th International Congress of Endocrinology, Rio de Janeiro, Brazil.

Dr Rachel Webb

Department of Paediatric and Congenital Cardiac Services, Starship Children's Hospital

XVII Lancefield International Symposium, Porto Heli, Greece.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Computerworld Excellence Awards 2008: Calling for Technology Entries from the Health Sector

Computerworld Excellence Awards 2008

The Computerworld Excellence Awards is calling for entries from ICT teams who have implemented great projects within the health sector. The Excellence in the Use of ICT in Health category is specifically focused on the benefits technology is bringing within this highly-specialised field.

The 2007 winner was the National Paediatric Oncology Steering Group for LEAP IT (Late Effects Assessment Programme IT). This is an online clinical tool to manage children and young people who have completed cancer therapy. The system allows illnesses associated with the long-term effects of cancer treatments to be tracked throughout the life of the patient, leading to better care and understanding of the patient's medical history and at the same time providing a research database to see which kinds of treatments and therapies produce the best results.

The Computerworld Excellence Awards is the premier awards programme within the ICT industry honouring the users of technology. A win is a highly sought after accolade amongst ICT professionals and is an opportunity to celebrate the country's ICT talent. Other categories for the 2008 programme include individual awards for Young ICT Talent, ICT Educator of the Year and CIO of the Year, plus team awards in areas such as education, small business and government and best sustainable ICT project.

Each category is judged by an independent panel of three judges with some of the country's most prominent ICT and business leaders involved, adding credibility to the achievement. Entry and nomination is open now, with submissions due by Friday 11 April 2008.

For a full category listing, eligibility details and entry process information, please go to **computerworld.co.nz/awards** or contact Claire Baker on 09 375 6050 or email Claire_Baker@fairfaxbm.co.nz

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Erratum

Disgraceful Conduct (05/127C). N Z Med J. 2008 March 14;121(1270).
http://www.nzma.org.nz/journal/121-1270/2966 (full text) and
http://www.nzma.org.nz/journal/121-1270/2966/content.pdf (PDF)

The medical practitioner referred to in the medicolegal notice issued by the Medical Practitioners Disciplinary Tribunal (MPDT) is Dr Dhammika Pradeepa Dassanayake, of Christchurch and **not Dr Chan Dassanayake** of Wellington, General Practitioner.

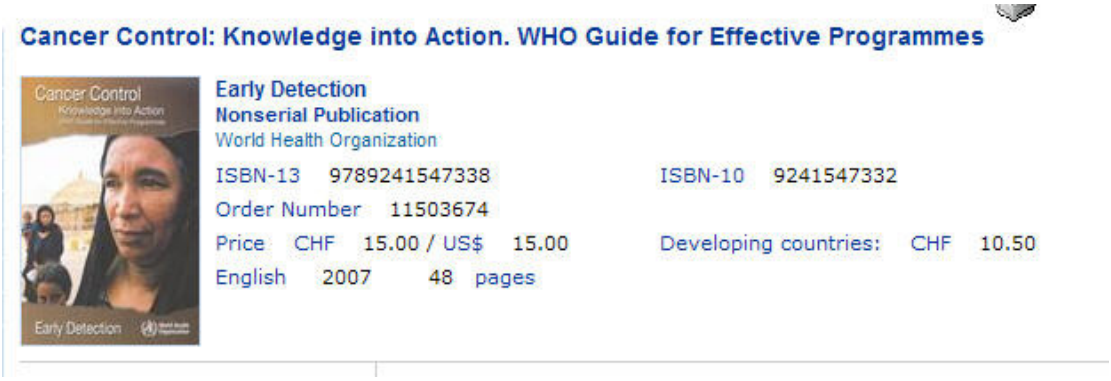
Previously only the surname Dassanayake was listed hence the above clarification with addition of first names and residence to accompany the surname.

Please refer to the above links to view the new copy of the notice, and replace any hard copies of the PDF held.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Cancer Control: Knowledge into Action. Early Detection (Module 3)

Written and published by the World Health Organization (WHO), 2007.
ISBN 9789241547338. Contains 48 pages. Price CHF15.00 / US$15.00



This is part of a series of six WHO Guides about setting up effective cancer control programmes. Written by seven core contributors, it reports the collaborative work of a 29-person technical group and additional participants and observers from a World Health Association Resolution on Cancer, adopted in May 2005.

It is simply presented, and authoritative. A brief introduction is followed by pre-planning, and then three steps common to all the modules: Where are we now? Where are we going? How do we get there? Early detection can be by early diagnosis, with major gains in some low resource countries, or by more complex screening programmes in better resourced countries. Simple tables and some examples are used, together with references.

The monograph would be of most value to those interested in cancer control itself, and has most value to lower resource countries where little screening exists. However the principles in this monograph should not be lost sight of by the more fortunate countries.

The publication is supported by the WHO website (under programmes and projects) which has already made two other Guides in the series available as PDF files, and this one should appear there in time.

Bridget Robinson
Associate Professor
Department of Medicine
University of Otago, Christchurch