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Maternal alcohol use before and during pregnancy among women in Taranaki, New Zealand
Reena Ho, Raimond Jacquemard

Our Ministry of Health guidelines of total abstinence in alcohol consumption during pregnancy are not well followed, as more than a quarter of NZ women continue consuming alcohol during pregnancy, with a significant minority drinking heavily. NZ's culture of binge drinking coupled with unplanned pregnancies puts our babies at risk of developing birth defects with life-long consequences (Fetal Alcohol Spectrum Disorders). We need to remember that Fetal Alcohol Spectrum Disorders are completely preventable and their prevention needs to be of urgent public health importance.

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Many obstetric caregivers express concern about the possible course of pregnancy in women who have previously undergone gastric bypass surgery for severe obesity. Such concerns include the possible problems which severely obese women may encounter in pregnancy and the possibility of nutritional problems arising because of the very small food intake which follows gastric bypass surgery. This study reviews the course of 24 pregnancies in 17 women who had previously undergone gastric bypass surgery. The results suggest that the course and outcome of pregnancy in this context is not dissimilar to that of more normal weight mothers, and was much more straightforward than would have been expected had they not undergone gastric bypass. There was no suggestion from the study that the baby's growth was affected by the small food intake of the mother.

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The availability of cancer services, in this case, radiotherapy treatment machines, should match the needs of patients with cancer. For all patients with lung cancer in the Auckland/Northland region in 2004, 43% received radiotherapy. When we looked carefully at the medical records of those patients who did not receive radiotherapy, we found that some declined treatment and others were not referred for treatment although the medical record suggests that they may have benefited from treatment. In total, radiotherapy facilities should have been provided for approximately 50% of all lung cancer patients. This is significantly lower than the estimate of 76% derived from

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Dorothy Begg, John Langley, Rebecca Brookland, Anna McDowell, Shanthi Ameratunga, John Broughton

The findings from a study of 3992 newly licensed car drivers in New Zealand showed that 49% supported 16 years or older as the minimum age to start licensing but this varied significantly by the age, gender and residential location of the learner driver. The most frequently reported reasons for getting a licence were independence and freedom. This applied equally to males and females, rural and urban drivers, and across all ages, although to drive to work was also a very important reason for learner drivers aged 18+ years. Contrary to what many may believe to be the case, the evidence presented here showed that there was not universal opposition by young people to raising the driver licensing age.

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In utero brain damage from alcohol: a preventable tragedy

Doug Sellman, Jennie Connor

Alcohol (ethanol) has been New Zealand's favourite recreational substance since British colonisation gathered pace 170 years ago. Regular consumption of large quantities of this drug is a common occurrence involving 25% of New Zealand drinkers.[1] This amounts to about 700,000 people, the total population of Wellington and Christchurch combined. But how many of these heavy drinkers are aware that alcohol has toxic as well as intoxicating effects? Moreover, how can we best protect mothers-to-be from inadvertently putting their babies at risk?

Neurotoxic effects of alcoholism have been well-known medically for many years, the main syndromes being alcoholic cortical dementia, Wernicke-Korsakoff's syndrome, cerebellar degeneration, and peripheral neuropathy.[2] However, signs of brain damage and associated cognitive dysfunction have recently been identified in social drinkers,[3] although low social drinking (less than 12 standard drinks per week) has been demonstrated to not be associated with any loss of brain volume.[4]

Undoubtedly the most tragic neurotoxic scenario involving alcohol is fetal alcohol spectrum disorder (FASD) where babies are born burdened with a preventable form of brain damage. Ho and Jacquemard[5] provide chilling data reminding us all once again about the continuing high prevalence of consumption of this neurotoxic drug by many mothers-to-be in one of the best New Zealand studies of the area to date. Ninety-one percent of mothers-to-be reduced their alcohol use because of pregnancy. However, more than half did this *after* pregnancy had commenced and an astounding 28% continued drinking during their pregnancy. This tallies closely with 2007–2008 Ministry of Health data,[6] which found 29% of women who had been pregnant in the previous 3 years had consumed alcohol during the pregnancy. Of additional concern was the finding that only 68% of women in this report who had been pregnant in this period reported having received advice not to drink at all during pregnancy, despite this being the unequivocal recommendation from the Ministry of Health since 2006.

The incidence of FASD has previously been estimated to be at least 1% of all births in the US.[7] If this conservative estimate is applied to New Zealand, where there are about 60,000 births per annum, there would be at least 600 children borne with FASD each year. However, Ho and Jacquemard produce evidence that drinking during pregnancy in New Zealand is markedly higher than in the US. Further, an updated US estimate[8] has put the prevalence of FASD in populations of younger school children as high as 2–5%. The true rate of FASD in New Zealand therefore could be greater than 5%.

Why are so many New Zealand children being exposed to the risk of brain damage in utero?

One reason is that a large proportion of pregnancies, including wanted ones, are unplanned and fetuses are exposed to alcohol and its metabolites before the pregnancy

is recognised. This is a function of the prevalent drinking culture, and therefore must be addressed by population-based measures.

The per capita consumption of alcohol has increased 9% over the past 10 years[9] despite an aging population. Part of this increase is due to drinking starting at earlier ages,[10] part because of greater volumes being drunk on each occasion by young people,[11] but there has also been an increase in consumption by women across all ages that has been most marked in young women.[11]

This increase in women's drinking has not been an accident of history. It has been driven by a highly successful marketing campaign of the alcohol industry which spends in the region of $200,000 a day[12] targeting sub-populations where there is potential for growth in consumption, including women. The industry's tactic of linking alcohol with the "good life" by associating drug use with having sex, being successful, being accepted by peers, and being grown-up and independent has been known for some time.[13] For women, there is also the added leverage of "keeping up with the boys" and emancipation through imitation of male behaviour.

How much of the industry's enormous marketing effort has been allocated to informing their women customers that there is a risk of producing a brain damaged baby if they become pregnant while drinking alcohol or if they continue to drink alcohol while being pregnant? The answer is zero.

In fact, the alcohol industry goes out of its way to resist such measures being undertaken. While the effectiveness of warning labels about FASD on alcoholic drinks has not been fully evaluated scientifically, it is surely the responsibility of the industry that produces and markets this potential neurotoxin to make sure drinkers know about the risk before they get pregnant for the first time.

As well as exposure to alcohol before a pregnancy is identified, more than one in four pregnant women in New Zealand continue to drink. This is a national tragedy. And whether the 68% quoted above is the proportion of pregnant women who are never offered advice, or the proportion that never hear it, the pregnancy abstinence message is not being received clearly. This needs urgent rectification.

The current Law Commission review gives New Zealand a once in a generation opportunity to reduce the levels of hazardous drinking in the population that contribute to the incidence of FASD. The relentless promotion of alcohol by the sophisticated marketing machine that the industry uses to keep its favourite customers drinking heavily needs to be dismantled, and health warnings applied to alcohol beverage containers. This will provide a new supportive environment for the dissemination of the Ministry of Health's pregnancy abstinence message by all health and maternity providers and by informed members of the community at large.

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Author information: Doug Sellman, Professor of Psychiatry and Addiction Medicine and Director, National Addiction Centre, University of Otago, Christchurch; Jennie Connor, Senior Lecturer, Epidemiology, Department of Preventive and Social Medicine, University of Otago, Christchurch

Correspondence: Doug Sellman, Professor of Psychiatry and Addiction Medicine, Department of Psychological Medicine, University of Otago, Christchurch, PO Box 4345, Christchurch 8140, New Zealand. Email: doug.sellman@otago.ac.nz

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Hazardous drinking: not just an issue for the minority of drinkers

Simon J Adamson, J Elisabeth Wells

The Law Commission is currently undertaking a first principles review of this country's alcohol policy.[1] A feature of the public discourse on alcohol use in New Zealand is the recurrent theme that hazardous drinking is a minority issue, in that only a minority of people drink too much.

An extension of this view is that legislative change that impinges on the drinking habits of most New Zealanders represents a blunt and unjust response to the problem, and instead we should be implementing legal and treatment interventions targeted at the small group of problem drinkers.

This perspective was well encapsulated by the Associate Minister of Health, the Hon Peter Dunne, when he opened Cutting Edge, a national alcohol and drug treatment conference, in Wellington in September of this year. The minister stated: "For the majority of people, alcohol will never be an issue in their lives, so our focus must be on enabling the majority to continue to enjoy alcohol responsibly while at the same time mitigating the adverse impacts on the minority and our communities generally."[2]

We believe this widely expressed view to be incorrect, underestimating the prevalence of hazardous drinking in New Zealand society. In particular, it mis-identifies cross-sectional prevalence as being indicative of lifetime prevalence.

Te Rau Hinengaro: The New Zealand Mental Health Survey reveals that 25% of those who drank alcohol in the past year were drinking in a hazardous fashion over that period, with 49% doing so among those aged 16–24.[3] A finer age breakdown shows a peak of 56% among those drinkers aged 18–21: males 64%, females 49% (Wells, personal communication, 2009). Similar results have been found in another recent large national household survey.[4]

Hazardous drinking has therefore been found to characterise the drinking pattern for the *majority* of drinkers during the peak years 18–21. Do these results represent the likely past drinking profile of older drinkers and the likely future drinking profile of younger New Zealanders, or might they pertain only to the narrow cohort aged 18–21 at the time of *Te Rau Hinengaro* (2003/04)?

We do not believe there is justification to dismiss this high prevalence of hazardous drinking as merely a cohort effect, as there is evidence that heavy and problematic drinking has characterised youth drinking patterns for a sustained period in New Zealand. For example, in the Christchurch Psychiatric Epidemiology Study, carried out in 1986, around 70% of young males and 25% of young females reported having experienced at least one DSM-III symptom of alcohol disorder by 25 years of age.[5]

We have identified a peak hazardous drinking rate of 56% for ages 18–21. Does this represent the maximum rate of lifetime hazardous drinking? This would be the case if *none* of the 44% of 18–21 year old drinkers who are not identified as drinking

hazardously were drinking hazardously prior to the 12 months captured by *Te Rau Hinengaro* or would go on to do so subsequently. We believe this scenario is highly improbable, although the magnitude of increase in hazardous drinking across the adult lifespan is difficult to estimate.

There is good evidence, however, from a New Zealand longitudinal cohort study showing that drinkers move in and out of problem drinking, even over a relatively narrow period of time[6] and that lifetime rates of alcohol problems are substantially higher than 12-month prevalence rates.[7]

Hazardous drinking by most youth means that most New Zealand drinkers will have a problem with their drinking at some time in their life. Nonetheless it is important not to focus only on youth; a substantial proportion of older drinkers currently drink hazardously. This proportion sits between 30% and 33% for men aged in their 40s and 50s—and even at 70–74 years, 22% of males are drinking hazardously as revealed by *Te Rau Hinengaro* data. The proportions are smaller for women but still not trivial: 19% for 40–45 year olds, 8% for 55–59 year olds and 4% for 70–74 year olds (Wells, personal communication, 2009).

Hazardous drinking as identified in *Te Rau Hinengaro* does not simply refer to one or a small number of occasions of drinking more than might be healthy, although this in itself would not be a trivial point. Rather hazardous drinking is defined using the international standard of an AUDIT score greater than eight,[8] representing a sustained pattern of drinking in a heavy or problematic way during the year preceding interview.

Problems arising from alcohol are not limited to a small minority drinking most heavily. The risk of alcohol-related problems rises steadily as alcohol consumption increases. Physical health complications, negative impact on mental health, work absenteeism or underperformance, interpersonal conflict, assault and motor vehicle accidents all become more likely with increasing consumption.[9]

There is increased risk for most health problems even at a low level of alcohol consumption and certainly at the levels associated with AUDIT-defined hazardous drinking.

Concerted actions are needed to reduce the harms caused by alcohol consumption. Targeting of strategies is important but it needs to be acknowledged that broader strategies that affect most drinkers, such as taxation, are also an important element, both because they disproportionately affect priority populations, heavy drinkers and youth, and because alcohol-related harms are so widely distributed across the population of New Zealand drinkers that population levels of reduction are a legitimate public health aim. Such measures have been clearly identified in *Alcohol: No Ordinary Commodity*[10], a distillation of the international evidence base.

The Law Commission, in its preliminary consultation document, has outlined many policy options compatible with the recommendations contained within *Alcohol: No Ordinary Commodity*. We call upon the Law Commission to be bold in its recommendations to government and urge health professionals and the public to support these measures, given the high impact of alcohol-related problems and the fact that hazardous drinking is not just an issue for the minority of drinkers in New Zealand.

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Author information: Simon Adamson, Acting Director, National Addiction Centre, and Senior Lecturer, Department of Psychological Medicine, University of Otago, Christchurch; Elisabeth Wells, Research Associate Professor, Department of Public Health and General Practice, University of Otago, Christchurch

Correspondence: Simon Adamson, National Addiction Centre, Department of Psychological Medicine, University of Otago, PO Box 4345, Christchurch, New Zealand. Fax" +64 (0)3 3641225, email: simon.adamson@otago.ac.nz

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Obesity and gestational diabetes mellitus: breaking the cycle

Sarah Bristow, Janet Rowan, Elaine Rush

In New Zealand and around the world, the prevalence of obesity is increasing.[1] A growing body of evidence suggests that the environment an individual is exposed to early in life can alter their long-term health and risk of disease. Throughout the lifecycle, glucose homeostasis affects development and future health, and is of key importance during pregnancy, as the long-term health of the baby may be influenced by maternal glucose concentrations. Of particular concern for future generations is the increase in rates of gestational diabetes mellitus (GDM), where maternal glucose intolerance develops or is first recognised during pregnancy.

The prevalence of known GDM has increased by 10% to 100% in certain ethnic groups over the past 20 years, reflecting the increasing prevalence of obesity and type 2 diabetes mellitus (T2DM) in young women.[2] It is, however, unclear as to whether this represents a true rise in GDM prevalence or merely reflects an increase in GDM screening. The definition of GDM as a glucose intolerance that develops during pregnancy makes it difficult to distinguish between pre-existing undiagnosed T2DM and true GDM, particularly in women of childbearing age, who are not usually screened for diabetes.[2]

In New Zealand, several factors will contribute to increases in the prevalence of diagnosed GDM over the next few years. Firstly, with the adoption of universal screening recommendations, more women with GDM will be recognised.[2] Secondly, international criteria for diagnosing GDM are being developed and, as data show that glucose levels below those currently used to diagnose GDM are associated with perinatal complications, the recommended glucose thresholds for diagnosis are likely to be lowered.[3,4] Thirdly, with our changing population, rates will rise as ethnicity is an important factor determining the risk of GDM.

Data from women delivering at National Women's Hospital, Auckland, during 2008 show GDM was diagnosed in over 16% of Indian, almost 10% of other Asian, and over 6% of Pacific Island and Māori women (groups known to have high rates of T2DM)[5] compared with 3% of NZ European women.[6]

The association of GDM with pregnancy complications and later risks of T2DM in the mother is well-recognised,[7] however, the long-term implications for the offspring deserves better recognition. There are accumulating data demonstrating increased risk of obesity and early T2DM in the offspring.[8–11] Society needs to consider how we should address this issue to improve the health of the next generation.

Developmental programming of obesity

Obesity is a disease of altered energy balance, where energy intake exceeds expenditure over a period of time. The pathogenesis of obesity, while complex and poorly understood, is thought to result from interactions between an individual's genes and environment.[12] In a recent review, Bouchard proposes common forms of

childhood obesity result from a genetic predisposition towards obesogenic behaviours in an obesogenic environment.[13] Thus, within any population, individuals will exhibit variable risk towards becoming obese as their genetic background determines how they respond to environmental factors such as the food supply and opportunities for physical activity. This relationship between genes and environment is further complicated by the fact that at critical times, environmental influences, such as fetal nutrition, can lead to a heritable change in gene expression without changing gene structure. This is termed an epigenetic phenomenon, and may be of key importance when considering how the intrauterine environment influences later health.

Later consequences of environmental influences, the 'fetal origins of disease' was first proposed by Barker and colleagues, who demonstrated a relationship between low birth weight (a marker of poor fetal nutrition *in utero*) and adult hypertension, dyslipidaemia and insulin resistance.[14] They suggested the fetus was not "programmed" to cope with a postnatal environment of plentiful nutrition, thus it has a reduced ability to develop an increased muscle mass, a reduced subcutaneous fat storage capacity and has to store excess nutrients as ectopic/visceral fat, which leads to later health consequences. This hypothesis formed the basis of a broader concept of 'developmental programming', which refers to any situation where an insult at an important period in development has a lasting effect on health or function.[15] A comparable situation may arise for offspring of women with diabetes whose placental function is impaired in association with hypertensive complications of pregnancy.

At the other end of the spectrum, exposure to an unbalanced excess nutrient supply secondary to maternal diabetes is also associated with a greater risk of obesity and T2DM in offspring. A long-term prospective evaluation of offspring exposed to maternal diabetes was carried out by the Northwestern University Diabetes in Pregnancy Center.[10] Children were compared with controls whose mothers did not have diabetes. At birth, 50% of the offspring of mothers with diabetes were above the 90th percentile of weight for gestational age. Although weight normalised by 12 months of age, after 5 years weight increased dramatically in the offspring of mothers with diabetes and by 8 years 50% had weights above the 90th percentile.

During adolescence, mean body mass index (BMI) in the affected offspring was 24.6 ± 5.8 kg/m² compared with 20.9 ± 3.4 kg/m² in control subjects (p=0.001).[8] In addition to obesity, 36% of the offspring of mothers with diabetes had evidence of impaired glucose tolerance by the age of 17 years compared with only 3% of the control population.

Nuclear family studies, where children have similar genetic risks for obesity and T2DM, have further examined the effect of the intrauterine environment.[11] Children born before their mother developed diabetes were compared with a sibling born after their mother developed diabetes, thus differing in only in their intrauterine environment. The offspring exposed to maternal diabetes had a mean BMI 2.6kg/m² higher and were more likely to develop T2DM (odds ratio 3.7, p = 0.02) than their sibling born before their mother developed diabetes. These results suggest exposure to GDM transmits greater risk to offspring for T2DM than that from inherited genetic predisposition alone.

The intergenerational transmission of increased risk is supported by a report from McLean et al,[16] who screened 5850 pregnancies for hyperglycaemia and asked

women to report their family history of T2DM (a mother, father, both parents or no parents with T2DM). As GDM during pregnancy is often the first sign of a genetic predisposition to T2DM, the authors postulated that subjects who had a mother with diabetes may have been exposed to GDM. As expected, among the pregnant study population, GDM/T2DM was more common in those women who had a mother with diabetes than a father with diabetes, while having two parents with diabetes conferred no additional risk than for mother with diabetes alone. The authors interpreted their results as not supporting a predominantly genetic transmission of T2DM, where risk would have been transmitted equally by both parents.

The relative contribution of genetic versus intrauterine transmission of risk for T2DM was examined more recently in an elegant study in an European popluation.[17] Four groups of participants were compared in this study: offspring with a genetic predisposition to T2DM (family history of diabetes) whose mothers had GDM (O-GDM), offspring with a similar genetic predisposition whose mothers did not have GDM (O-NoGDM), offspring with a low genetic predisposition to T2DM whose mothers had type 1 diabetes (O-Type1) and offspring with a low genetic predisposition whose mother did not have diabetes during pregnancy—the background population (O-BP).

At a mean age of 22 years, the prevalence of T2DM was 21% in O-GDM, 12% in O-NoGDM, 11% in O-Type1 and 4% in BP. This study demonstrates the increased risk of T2DM in offspring with higher genetic risk and in offspring exposed to maternal diabetes *in utero*, with the highest risk in offspring exposed to both factors.

Developmental programming may occur in the absence of full-blown maternal diabetes, with exposure to milder levels of maternal hyperglycaemia linked with an increased risk of obesity in offspring. This was demonstrated in a recent prospective study, where a positive trend for increasing childhood overweight and obesity between the ages of 5 to 7 was found across a range of increasing maternal glucose screen values—even after adjustment for maternal weight, age, parity and birth weight.[18]

Importantly, it was shown that the risk of obesity was attenuated in the offspring of mothers with diagnosed, treated GDM, compared with the risk in offspring whose mothers had untreated milder hyperglycaemia. It is possible these mechanisms are also relevant for offspring of obese women during pregnancy, who have higher postprandial glucose levels than lean women.[19]

Dysregulation of the adipoinsular axis

What could be the mechanism behind maternal hyperglycaemia leading to obesity and T2DM in the offspring? In a pregnancy complicated by GDM, maternal hyperglycaemia causes increased amounts of glucose to cross the placenta, leading to increased fetal insulin release. Insulin is an anabolic hormone which is very important in fetal growth. In the presence of excess glucose, raised insulin in the fetus during the third trimester is thought to lead to increased fat synthesis and deposition.[20] Indeed it has been shown that even in a sample of average-for-gestational-age newborns, those exposed to GDM *in utero* have greater fat mass, body fat percentage and skin fold thickness when compared with those born to glucose tolerant mothers.[21]

For an individual in energy balance who can choose when to eat, an increase in fat stores results in increased plasma leptin concentrations, which signals satiety, reduces food intake and inhibits insulin production and adipogenesis. This endocrine feedback system, termed the adipoinsular axis, is associated with maintaining adipose homeostasis.

In the intrauterine environment, a fetus exposed to excess nutrition will develop an increased fat mass, but despite increased leptin levels, there is continued excess nutrient supply from the mother. Over time this may lead to dysregulation of the adipoinsular axis and development of leptin resistance. A New Zealand study demonstrated hyperinsulinaemia and hyperleptinaemia in newborns of women with GDM in Māori, Pacific Island, Indian, and European populations, suggesting leptin resistance may be present at birth.[22] Additionally, there is evidence from an animal study hyperinsulinaemia and hyperleptinaemia play a role in the development of postnatal hyperphagia in offspring, which could make postnatal interventions to improve long-term health more difficult to achieve.[23]

Though the molecular mechanisms that underlie the programming of obesity and T2DM are beyond the scope of this article, it important to understand that fetal programming is thought occur via epigenetics rather than changes to actual DNA base sequence.[24] Epigenetics refers to the mechanisms that lead to long-term changes in the expression of a gene, such as gene silencing by methylation in the promoter region—a process involved in the differentiation of cells for different tissues.

Vitamin B12 and folate are important methyl donors, and are essential for normal cell growth and division. Research now suggests these micronutrients may play a role in fetal programming. Genetically obese Agouti mice fed a methylating cocktail of vitamin B12, folic acid, betaine and choline during pregnancy had offspring who were less obese and had a different coat colour—despite inheriting the Agouti mutation.[25]

A recent study in India demonstrated women with low B12 status had increased adiposity and a higher prevalence of insulin resistance and GDM compared to those with adequate B12,[26] while the longitudinal Pune Maternal Nutritional Study has shown that 6-year-old offspring of women with high folate and low B12 concentrations during pregnancy had greater adiposity and insulin resistance than offspring whose mothers had normal B12 status.[27] B12 deficiency is common among Indian women due to vegetarian dietary practices,[28] and could potentially contribute to the high prevalence of GDM and T2DM seen in Indian women in New Zealand and overseas.

The postnatal environment

What is not yet known is the relative importance of the prenatal and postnatal environments in determining an individual's risk of disease during their life course. Evidence from animal models suggests the early postnatal environment may modify the effect of the prenatal environment.[29] For example, intrauterine growth restricted rats exposed to a nutritionally limited prenatal environment develop obesity when exposed to a non-restricted postnatal diet. When these rats are exposed to a postnatal high fat diet, obesity is amplified,[30] and when they exposed to moderate daily exercise, the development of obesity is prevented.[31]

Whether postnatal exposures such as breastfeeding, as well as later diet and physical activity can alter the risk of obesity in the offspring of mothers with diabetes are areas that require further research. In the general population there is evidence breastfeeding is protective against obesity,[32] suggesting the lactation period could be important in the programming of disease risk. The beneficial effects of breast milk are thought to be due to its macronutrient composition, which may alter the hormonal responses that regulate body fatness and growth. In NZ only 55.8% of infants are estimated to be breastfed exclusively at 3 months of age, this number drops to 7.6% by 6 months.[5]

There are limited data available on breastfeeding after GDM. Two studies have demonstrated an inverse relationship between breastfeeding and overweight in GDM offspring. The Nurses' Health Study found risk of overweight at ages 9 to 14 years in the offspring of mothers with diabetes was inversely associated with having been breastfed during the first 6 months of life.[33] Similarly a German study reported that offspring of mothers with diabetes who were breastfed for >3 months had a 45% decrease in rates of overweight (BMI $\geq 90^{th}$ percentile) at the ages of 2-8 years compared with those who were formula fed.[34]

Implications for New Zealand

Childhood obesity has reached epidemic proportions in developed countries, with New Zealand no exception. The 2006/2007 New Zealand Health Survey found that one in every five children was now overweight, and one in 12 obese.[5] Based on BMI definitions, the prevalence of obesity was higher in Pacific Island and Māori children (23.3% and 11.8% respectively) compared with NZ European and Asian (5.5% and 5.9%, respectively). These data will underestimate rates of obesity in Asian populations, as they have a greater degree of adiposity than Europeans at a specific BMI.[35] This has been studied extensively in Indians and is known as the 'thin-fat' phenotype, and places this ethnic group at risk for obesity related diseases at relatively low BMI's. In contrast, Pacific populations have less adipose mass at a given BMI compared with European populations.[36]

Childhood obesity is associated with a number of co-morbidities including dyslipidaemia, hypertension and abnormal glucose tolerance,[37] and increases the risk of children developing chronic diseases such as T2DM and cardiovascular disease later in life.[38] Though previously rare, the diagnosis of T2DM in youth is becoming increasingly common and strongly associated with obesity.[39] Obesity tends to track from childhood into adulthood,[40] and is notoriously difficult to treat. For these reasons, early life interventions that prevent the onset of overweight and obesity are urgently needed.

Conclusion

Infants born to mothers with GDM are at an increased risk of obesity and T2DM in childhood and adolescence. This could impact on the rates of these diseases, particularly in Indian, Pacific Island, and Māori populations, who tend to have higher rates of GDM. In effect a cyclical relationship could develop; where obese and diabetic mothers give birth to infants who become obese and develop diabetes before their childbearing years, only to pass this on to their offspring.

Breaking this cycle requires education and focused efforts to optimise the environment and health of young women before and during pregnancy, recognition and treatment of women with GDM, continued promotion of breastfeeding and follow up of women who have had GDM, plus their children. These are important public health issues that are likely to have far-reaching effects on future generations.

Competing interests: None known.

Author information: Sarah Bristow, MPhil candidate, Centre for Physical Activity and Nutrition Research**,** AUT University**,** Auckland; Elaine Rush, Professor of Nutrition, Centre for Physical Activity and Nutrition Research, AUT University, Auckland; Janet Rowan, Obstetric Physician, National Women's Health, Auckland District Health Board, Auckland

Correspondence: Sarah Bristow, Centre for Physical Activity and Nutrition Research**,** AUT University, Private Bag 92006, Auckland 1142, New Zealand. Fax +64 (0)9 9219960; email: sarah.bristow@aut.ac.nz

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Maternal alcohol use before and during pregnancy among women in Taranaki, New Zealand

Reena Ho, Raimond Jacquemard

Abstract

Aims The study researched alcohol consumption and drinking patterns before and during pregnancy.

Method This was a 1 month self report survey of postnatal women from 21 May–22 June 2006. A multiple choice questionnaire was handed out to them on the first or second postnatal day.

Results There were a total of 117 deliveries. The questionnaire was completed by 100 of the 104 women who received it. Before pregnancy, 80% of women reported drinking alcohol; 66% binge drinking. Twenty-eight percent continued consuming alcohol throughout pregnancy. The majority did reduce or stop their alcohol consumption, 7% however did not. Ten percent were drinking more than 2 units per typical day and more than 7 units per week during pregnancy. Four percent was drinking a lot more than this. Nine percent of the total cohort reported binge drinking during pregnancy.

Conclusion Just over a quarter of women drink alcohol throughout pregnancy. A significant minority of women drink relatively heavily (more than 4 units per occasion and multiple times per week) during pregnancy. Many women do reduce their alcohol use because of the pregnancy, but often only after they become aware of it. In New Zealand there is a real risk of fetal alcohol spectrum disorders.

Fetal Alcohol Syndrome (FAS) is more likely to occur following continuous or heavy intake of alcohol during pregnancy. Effects have also been observed after intermittent or binge drinking.[1,4,5,11,22] It is also now recognised that there is a wide set of effects from lower-level alcohol use. Other factors such as timing of exposure, maternal or fetal genetic factors affecting metabolism or individual susceptibility coupled with other harmful behaviours could all contribute in determining the outcome of an alcohol exposed pregnancy. The lower limit of alcohol intake at which no adverse effect will occur for any developing foetus has not yet been determined, and may not exist.[1,3,12,30]

Measurable behavioural changes have been observed in children exposed to intrauterine alcohol consumption of as little as one standard drink per week,[5,12] and in cognitive skills with one or two drinks per day.[1,24,25,27] There is therefore no definitive information that can be conveyed to women regarding a safe quantity of alcohol use during pregnancy.[3,4,11]

The New Zealand Ministry of Health advises women who are pregnant, planning pregnancy, or breastfeeding to avoid any alcohol.[1,9] There is evidence that such advice

is not universally applied. A survey of New Zealand health professionals showed that only 46% recommended total abstinence.[10,26]

Reports indicate consistently that about 80–85% of all New Zealand women drink some alcohol.[1,29] Existing New Zealand data indicate that about a quarter of the New Zealand adult population binge drink, and this binge drinking behaviour is supported by a general tolerance for drunkenness.[8] New Zealand surveys on pregnant women and midwives reported our pregnant women drinking two to three times more on average than the Americans, and our teenagers consuming alcohol in pregnancy at eight times the average rate in the United States.[13,28,29]

There have been studies which looked at alcohol consumption in New Zealand women during pregnancy and factors that influence it.[6,20,21] These studies do not clearly relate this to alcohol consumption before pregnancy, nor provide much data on the timing of alcohol reduction during pregnancy.

Clearly, the crucial issue is whether women who drink alcohol continue to do so during pregnancy, and to what extent. Does the pregnancy lead to a decrease in their alcohol consumption? If it does, at what stage of the pregnancy does that take place? That is important as the risks to the foetus are recognised to be the greatest during early pregnancy.[3,4,11,30]

The answers to these questions reflect the rate of alcohol-exposed pregnancies and could well mirror the incidence of FAS and Fetal Alcohol Spectrum Disorders (FASD) in New Zealand.

Therefore the main purpose of the study was to evaluate the following:

- The amount of alcohol women drink before pregnancy
- The proportion of women who continue to drink during pregnancy
- Whether the women change their drinking pattern, and if they do, at what stage of the pregnancy that takes place
- The amount of alcohol consumed when pregnant

Methods

The survey was conducted at Taranaki Base Hospital, New Zealand. The hospital serves the city of New Plymouth and the surrounding semi-rural areas. The total population of the region is 104,000 people. About 16% of the population identifies as Māori, 2% as Asian, 1.5% Pacific peoples, 80% as European or from other descent. The hospital has 1200–1300 deliveries per year. All the women who had delivered between 21 May to 22 June 2006 were approached on the day of delivery or the next day i.e. Day 0 or Day 1 postnatal, and given the multiple choice survey questionnaire (see Appendix 1). There were no exclusion criteria.

A visit to the labour ward, neonatal unit and postnatal ward was done at least once a day. The authors then handed out the questionnaires to every woman in person, with an explanation about them. The questionnaires contained no identifying data. Self-addressed envelopes were provided and the questionnaires were to be returned sealed. The sealed envelopes could either be handed in to a member of the nursing staff or the ward clerk to be then handed to the authors, or dropped off in an allocated collecting box.

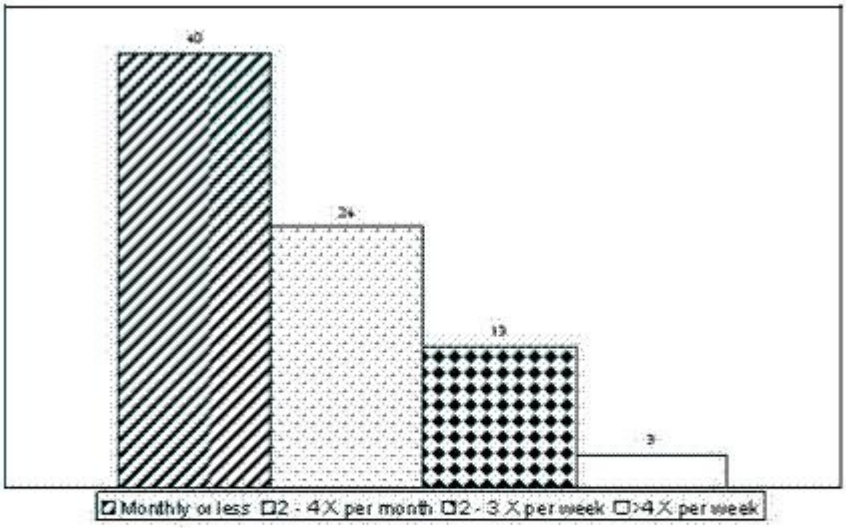
Results

There were a total of 117 deliveries over the 1-month period. One hundred and four questionnaires were handed out. Thirteen women were missed because they had gone home before they could be contacted in the ward.

Of the 104 questionnaires handed out, 102 were successfully retrieved (98%). Two of the 102 questionnaires were excluded as multiple questions were incompletely filled out. A total of 100 women were included in the study.

Drinking before pregnancy—80% of the women consumed alcohol before they were pregnant with the remaining 20% being non-drinkers. Among these who drank alcohol, 50% (40 out of 80) said they drank monthly or less, 30% (24 out of 80) reported drinking 2 to 4 times a month, just over 16% (13/80) drank 2 to 3 times a week, and 4% (3/80) drank four or more times a week (Figure 1).

Figure 1. Before pregnancy – frequency of alcohol use



On a day when they were drinking alcohol before they were pregnant (i.e. "a typical day" NB: some may drink more than one occasion in a day; therefore this could be the total number of drinks from a few occasions on a typical drinking day), 59% (47/80) drank 1 to 2 drinks, 24% (19/80) 3 to 4 drinks, 10% (8/80) 5 to 6 drinks, and 7.5% (6/80) would have 7 or more drinks (Figure 2). (By definition, one standard drink contains approximately 10g of pure alcohol. All alcohol containers now have 'Standard Drinks' content on the label).[36]

On the question of having more than 4 drinks on one occasion (the definition of binge drinking for women)[4,37], only 16% (13/80) said they had never done that, and as many as 66% of the total cohort (66 out of 79, instead of 80, as one person did not answer the question) reported drinking more than 4 drinks on one occasion. Forty percent of the total cohort said they did that less than monthly, 20% said monthly, 6% said weekly and none reported drinking that amount daily (Figure 3).

Figure 2. Before pregnancy – amount per typical day (when drinking)

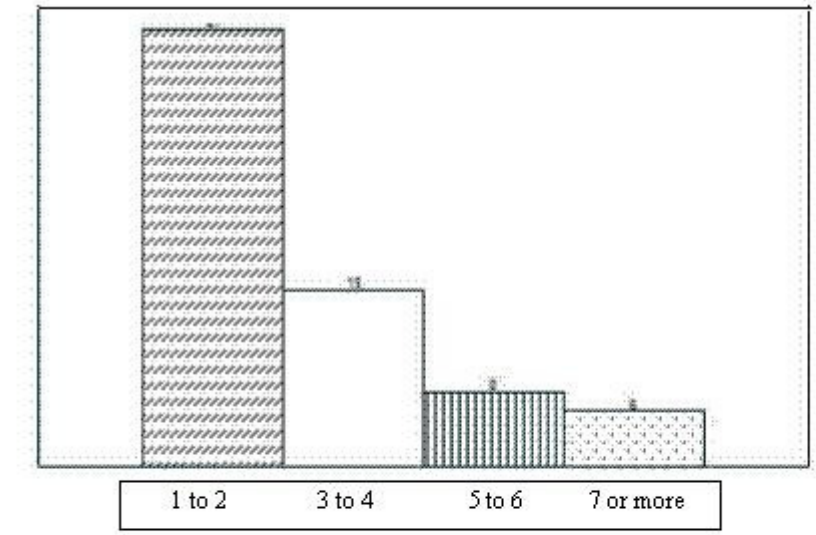
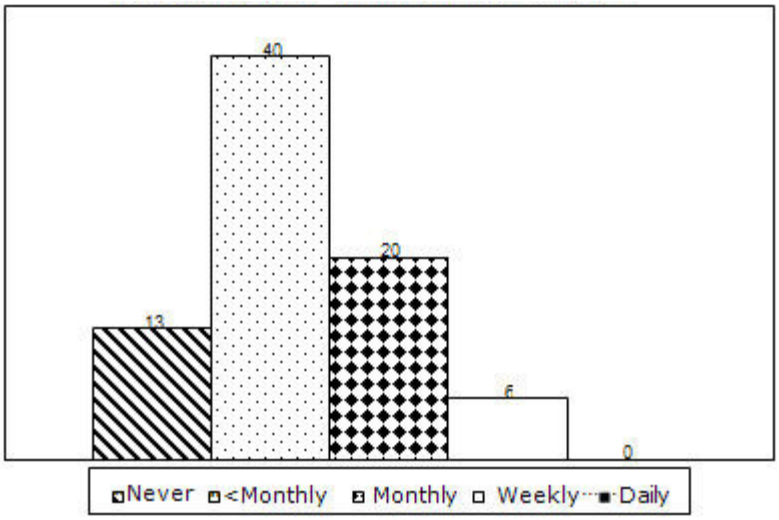


Figure 3. Before pregnancy – more than 4 on 1 occasion (binge drinking)



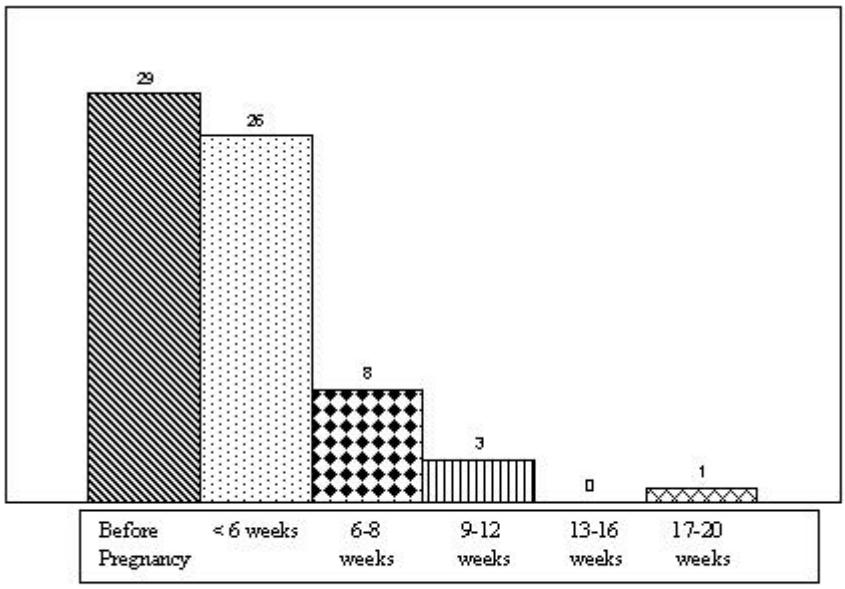
Drinking during pregnancy—Of the total cohort of 100 women, 28 continued consuming alcohol during pregnancy.

Seventy-three (91%) of the 80 women who consumed alcohol before pregnancy said they changed the amount they were drinking because of their pregnancy. Fifty-two (65%) had stopped drinking altogether, and 21 (26%) women continued drinking during pregnancy, albeit less. Seven women (8.7%) out of the 80 said they did not change their drinking habit, i.e. continued drinking the same amount during pregnancy.

Of the 73 who reported drinking less alcohol during pregnancy, only 67 women completed the question on when they started to decrease their consumption. Twenty-nine of these 67 women (43%) said they decreased their drinking before pregnancy. Twenty-six women (39%) said that took place when they were less than 6 weeks pregnant, eight women (12%) said 6 to 8 weeks, three women (4.5%) at 9 to 12 weeks, and one (1.5%) woman at 17 to 20 weeks (Figure 4).

Of the 28 women who continued alcohol consumption when pregnant, 20 (71%) drank monthly or less, seven (25%) drank 2 to 4 times a month, and one (3.5%) woman drank 2 to 3 times a week (Figure 5).

Figure 4. Timing of reduction in alcohol use



Twenty three out of 28 (82%) of these pregnant mothers had 1 to 2 drinks on a typical day. Three (11%) women had 3 to 4 drinks; one woman reported drinking 5 to 6 drinks, and another woman 7 or more drinks on a typical day (Figure 6).

Overall, 19 out of 28 women (68%) who drank during pregnancy said they did not drink more than 4 drinks on each occasion (binge drinking). A total of nine women reported binge drinking while pregnant. Seven said they did that less than monthly, one woman monthly, and another weekly (Figure 7).

Figure 5. Pregnancy – frequency of alcohol use.

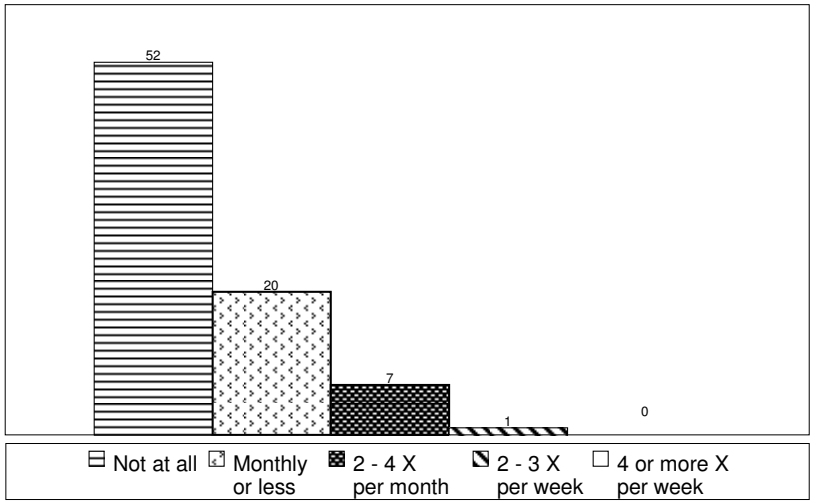
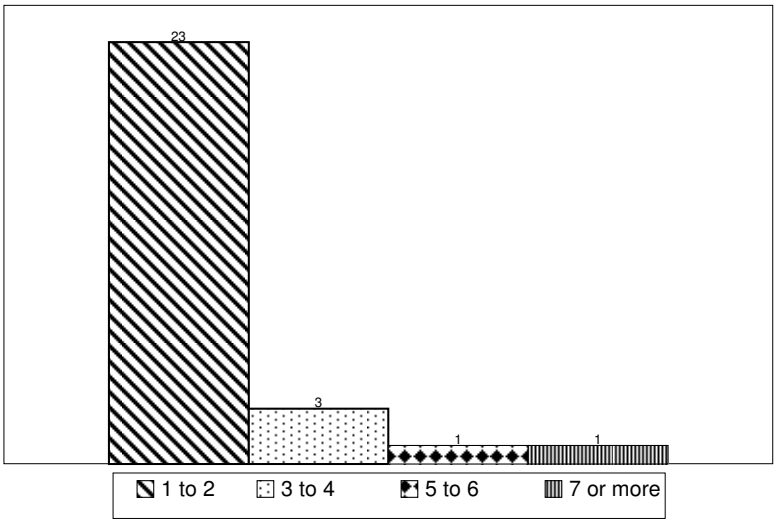


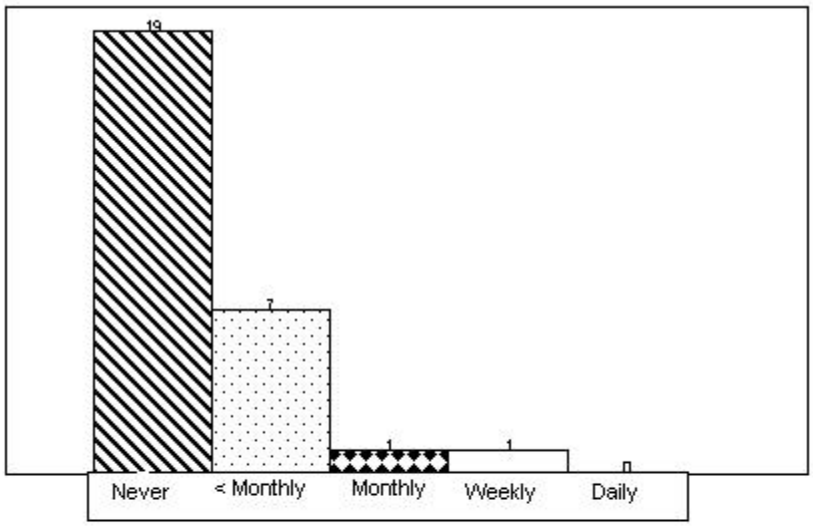
Figure 6. Pregnancy – amount per typical day (when drinking).



Of the 28 women who continued drinking throughout their pregnancy, 18 were noted to be drinking about 1 to 2 per typical day, no more than 7 per week, and did not report binge drinking at any point during their pregnancy. The other 10 women however were found to be drinking above these levels.

Four women from the total cohort in particular stood out because of their risky drinking patterns: The first woman was drinking 5 to 6 drinks on a typical day, about 18 a week, binge drinking weekly before she was pregnant, and only stopped drinking at 6 to 8 weeks gestation.

Figure 7. Pregnancy – more than 4 on 1 occasion (binge drinking).



The second woman was drinking more than 7 drinks per typical day, about 21 drinks a week, binge drinking weekly before she was pregnant, and only stopped drinking at 9 to 12 weeks.

The third woman was drinking 5 to 6 drinks on a typical day, 2 to 4 times a month, binge drinking weekly before her pregnancy, and continued her drinking pattern during pregnancy.

The fourth woman was drinking more than 7 drinks per typical day, 2 to 4 times a month, binge drinking monthly, and continued the same drinking pattern during her pregnancy, except that she reported binge drinking less (down to less than monthly), from 17 to 20 weeks onwards.

Discussion

This study relied on recall and therefore could have been subject to bias. Our data on the number of women that drink alcohol before and during pregnancy are very similar to those in other New Zealand reports and in this sense our results seem reliable.[1,6,20,21,29] We also think that it is fair to assume that bias would tend in the direction of under-reporting of alcohol use. If anything, we think that the situation could be worse than that found in the study. The exceptionally high response rate, with 98% of the questionnaires retrieved is a strength of this study.

The results show that 80% of women in our population drink some alcohol. This is consistent with previous findings in New Zealand.[1,29] Up to 8% are high risk drinkers according to the New Zealand low risk drinking guidelines outlined by the Alcohol Advisory Council. [2,8,9] Another significant finding was that as many as 66% of non pregnant women (66 women out of the total cohort) reported binge drinking.

Our finding that 28% (of the total cohort) of women continued drinking alcohol throughout their pregnancy was consistent with previous surveys that were done in New Zealand.[6,10,20,21] A 1999 nutrition report on 500 pregnant women in New Zealand showed that 29% continued to drink alcohol after their pregnancy was confirmed.[10,20] Another study done in Wellington in 2002 also showed that about 25% of women drink alcohol in pregnancy. [6]

Guidelines are in agreement that levels of alcohol use of more than 2 drinks per occasion, 7 drinks per week and/or binge drinking pose a definite risk for the developing foetus.[3,4,7,22–25,30,31] We found that more than half of the women who drank alcohol during pregnancy were drinking below these levels (64%—18 out of 28 women). Although these women could possibly be considered as drinking at a lower risk level, their babies may still be at risk of developing Alcohol Related Neurodevelopmental Disorder (ARND) as some evidence suggest.[5,12,27,32,33]

There were 10 women who reported drinking above these levels. These women are at a much higher risk of their pregnancy having alcohol induced adverse effects.

The percentage of women who reported binge drinking during their pregnancy in this study (9%) is again consistent with the "Nutrition during Pregnancy" report to the Ministry of Health in 1999 and a survey of pregnant women, which also reported that 10% of pregnant women in New Zealand drank to intoxicating levels.[20] Heavy episodic drinking can result in increased teratogenic effects because of the higher peak blood alcohol levels achieved.[3,4,22,30]

Ninety-one percent of the women reduced their drinking because of their pregnancy, but more than half of these only reduced it after they knew they were pregnant. The majority did this before 6 weeks of pregnancy, but a significant minority after 6 weeks (12% of the total cohort after 6 weeks or more). Studies looking at binge exposure prior to pregnancy recognition found that the children at 7 years of age were having learning problems, low academic achievement and hyperkinetic and impulsive behaviour problems.[3,22]

There are no precise data on the prevalence of FASD in New Zealand. In the United States (US), the prevalence of FAS is estimated to be 0.5 to 2 per 1000 live births, the prevalence of FAS and ARND combined at least 10 per 1000.[34] In the US, 12.5% of women drink alcohol during pregnancy, 3% at least 7 days per week and 3.4% of women binge drink.[35] Our results show alcohol use during pregnancy that is markedly higher than this. Therefore we could expect the prevalence of FASD in New Zealand to be well above US estimates.

In summary our results show that the Ministry of Health guidelines concerning alcohol consumption in pregnancy i.e. total abstinence, are not well followed. More than a quarter of women continue consuming alcohol throughout pregnancy. Most of these women drink at lower levels relatively, but their babies could still be at risk of neurobehavioural effects secondary to intrauterine alcohol exposure.

About 1 in 10 pregnant women drinks alcohol at levels that definitely puts their babies at risk for fetal alcohol spectrum disorder. The majority of women reduce or stop alcohol intake because of pregnancy, more than half only do this once they know they are pregnant. This can put their babies at risk in early pregnancy. Our culture of binge drinking further exacerbates this risk.

Every effort needs to be made to reduce these numbers. Women and health care providers must be made more acutely aware of the adverse effects of alcohol on the fetus. Alcohol consumption should be stopped before pregnancy. Fetal alcohol spectrum disorders are completely preventable, and their prevention needs to be of urgent public health importance.

Competing interests: None known.

Author information: Reena Ho, Paediatric Registrar, previously Taranaki Base Hospital, New Plymouth, currently Waitakere Hospital, West Auckland; Raimond Jacquemard, Paediatrician, Taranaki Base Hospital, New Plymouth

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Correspondence: Raimond Jacquemard, Paediatric Department, Taranaki Base Hospital, Private Bag 2016 New Plymouth. Tel: (06) 7536139; Fax: +64 (06) 7537791; email: raimond.jacquemard@tdhb.org.nz ; Reena Ho, Paediatric Registrar, Waitakere Hospital, Private Bag 93-115, Lincoln Rd, Henderson, Waitakere 0650. Tel: (09) 8381838; FAX: (09) 8381819; email: reena.ho@waitematadhb.govt.nz

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Appendix 1

1. Did you consume alcohol-containing drinks before you were pregnant or before you planned your pregnancy?

□ Yes □ No

If you answered 'No', you are finished with this questionnaire. Please return questionnaire to the ward clerk or nurse in the envelope provided, sealed. Thank you.

If you answered 'Yes', please continue. Items 2a, 2b and 2c concern the situation *before* you were pregnant (or *before* you planned your pregnancy).

2a. How often would you drink alcohol containing drinks?

□ Monthly or less

□ 2-4 times a month

□ 2-3 times a week

□ 4 or more times a week

2b. How many alcohol containing drinks did you have on a typical day when you were drinking?

□ 1-2

□ 3-4

□ 5-6

□ 7 or more

2c. How often did you have 4 or more drinks on one occasion?

□ Never

□ Less than monthly

□ Monthly

□ Weekly

□ Daily or almost daily

3. If you consumed alcohol before you were pregnant or before you planned your pregnancy, did you change the amount you were drinking (because of the pregnancy)?

□ Yes □ No

If you answered 'No', you are finished with this questionnaire. Please return questionnaire to the ward clerk or nurse in the envelope provided, sealed. Thank you.

If you answered 'Yes', please proceed to 4a, 4b, 4c & 4d. Items 4a, 4b, 4c & 4d concern the situation when you were *pregnant*:

4a. If you were drinking less alcohol containing drinks during pregnancy, did you decrease this:

□ before pregnancy

□ at less than 6 weeks pregnancy

□ at 6 to 8 weeks pregnancy

□ at 9 to 12 weeks pregnancy

□ at 13 to 16 weeks pregnancy

□ at 17 to 20 weeks pregnancy

4b. How often did you drink when you were pregnant?

□ Not at all (You are finished with the questionnaire. Please return questionnaire to the ward clerk or nurse in the envelope provided, sealed. Thank you.)

□ Monthly or less

□ 2-4 times a month

□ 2-3 times a week

□ 4 or more times a week

4c. How many alcohol containing drinks did you have on a typical day when you were pregnant?

□ 1-2

□ 3-4

□ 5-6

□ 7 or more

4d. How often did you have 4 or more drinks on one occasion when you were pregnant?

□ Never

□ Less than monthly

□ Monthly

□ Weekly

□ Daily or almost daily

Thank you for taking the time to fill in this questionnaire. Please return questionnaire to the ward clerk or nurse in the envelope provided, sealed.

THE NEW ZEALAND MEDICAL JOURNAL
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Pregnancy following gastric bypass surgery: what is the expected course and outcome?

Nikhil Sapre, Karen Munting, Archana Pandita, Richard Stubbs

Abstract

Aim To examine the course of pregnancy, labour, and the neonatal period in a group of women who have become pregnant following gastric bypass surgery for severe obesity.

Methods Women who had experienced pregnancy following gastric bypass surgery were identified by an initial questionnaire. A second questionnaire was sent to those identified by the first questionnaire, who were willing to provide details concerning such pregnancies.

Results Seventeen women experienced a total of 24 pregnancies and 25 live births. Five had experienced difficulties with conception or pregnancy prior to surgery. The average maternal weight gain was 6.13 kg. No major problems with fetal growth were observed. Babies were delivered at a mean gestational age of 37.5 weeks and with a mean birth weight of 3038 g. Six women reported a complication during pregnancy (25%) and five a complication in labour (20%). Two babies born to the same mother had congenital abnormalities attributable to a rare genetic disorder.

Conclusion The course of pregnancy and labour appears normalised for severely obese women following gastric bypass surgery. The weight loss and marked reduction in food intake following gastric bypass surgery does not lead to growth or development problems for offspring. Careful monitoring of expectant mothers who have undergone gastric bypass surgery is nevertheless to be recommended.

The prevalence of obesity in New Zealand is increasing with 1 in every 5 adults being obese.[1] It is well documented that pregnancy in obese women is associated with an increased risk of many adverse events and outcomes. These include such things as pre-eclampsia, the need for induction of labour, caesarian section, post-partum haemorrhage, and large for gestational age (LGA) deliveries.[2] In addition, severely obese women are at increased risk of gestational diabetes and their babies of congenital birth defects, neonatal hypoglycaemia, jaundice, and the need for admission to neonatal intensive care.[3]

Severely obese mothers are at increased risk of thromboembolism, anaesthetic complications, and wound infections following caesarean section.[4] Similar adverse neonatal and perinatal outcomes have been reported for overweight adolescent women.[5]

Bariatric surgery has emerged in the last 10–15 years as an effective and reliable solution to severe obesity[6] and more and more severely obese individuals are choosing this option for managing their problem. Although a number of previous

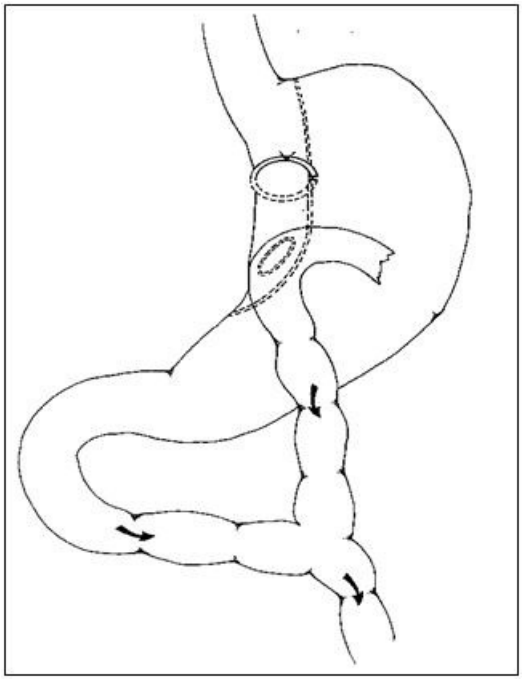
reports have addressed the course and outcome of pregnancy in women who have undergone bariatric surgery[7–12] such information remains poorly disseminated.

At a time when most health professionals have little personal knowledge or experience of bariatric surgery, many remain uncertain about the expected course of pregnancy and delivery for mothers who have had bariatric surgery. This report documents the outcomes of 24 pregnancies in 17 women following gastric bypass surgery for severe obesity.

Methods

This study was undertaken on women who had undergone gastric bypass surgery at Wakefield Hospital, Wellington. All surgeries were performed by the senior author (RSS) who has performed over 1100 gastric bypass operations since 1986. See Figure 1.

Figure 1. A schematic representation of the gastric bypass operation



A full description of the operation and its outcomes is available elsewhere.[13] In brief, the stomach is divided into two component parts. A small 5-10 ml gastric pouch extending from the oesophago-gastric junction down the lesser curve of the stomach and a large distal component of the stomach, which together with the duodenum and initial 50–70 cm of jejunum is excluded from the food stream.

A 70 cm Roux loop of jejunum is created and joined to the small gastric pouch, which means ingested food enters directly into the jejunum. A silastic rubber ring of 6.5 cm circumference (approximately 19mm diameter) is placed around the small gastric pouch above the anastomosis in such a way as to ensure the size of the outlet of the pouch remains fixed throughout life.

Food intake is severely restricted with average daily caloric intakes being reduced to less than 1000kcal for life. Weight loss of around 65–75% of excess weight is to be expected over a 12-18 month period with much of this occurring in the first 6 months.[14]

Vitamin intake is reduced commensurate with food intake, and absorption of fat soluble vitamins may be particularly affected by low fat intake. Absorption of vitamin B12 is affected by the bypass of the body of the stomach and folic acid and iron absorption by the bypass of the duodenum. All those who

have undergone gastric bypass are encouraged to take a multivitamin tablet daily for life, and those who develop deficiencies of vitamin B12, folic acid or iron, are placed on appropriate supplements for life in the case of the former two, and as required in the case of iron.

A variety of blood tests are performed at intervals throughout the first 2 years following gastric bypass to detect these deficiencies, and are then recommended at annual intervals throughout life.

The subjects of this study were identified by sending an initial questionnaire to all women of childbearing age (20–40 years) who appeared on the prospective Wakefield Clinic obesity surgery database (1100 patients), who had undergone surgery since 1991, seeking those who had become pregnant at any time following their gastric bypass operation. Those who had experienced a pregnancy, whether successful or not, were asked to participate in the present study, by completing a further questionnaire seeking details of the course of the pregnancy, labour and subsequent outcome.

Additional information was obtained from some participants by phone call, where responses were either incomplete or unclear. As this was simply an audit of pregnancy in a single doctor's practice, Ethics Committee approval was not considered necessary.

Obstetric care—The obstetric care was provided in various facilities throughout New Zealand. The Wakefield Clinic was not actively involved in the management of any pregnancy, although in some cases was contacted by attending doctors/midwives for information and/or advice regarding vitamin and mineral supplementation during the pregnancy.

Results

The initial questionnaire was mailed to 165 women who had undergone gastric bypass between May 1991 and December 2005. From this group 19 were identified as having become pregnant at a time subsequent to the gastric bypass surgery. Seventeen agreed to participate in the study, but it is known there were no major problems encountered by the other two, who preferred not to provide details concerning their pregnancy.

Of the 17 women, 9 had experienced one pregnancy and delivery each, 7 had each experienced two pregnancies and deliveries since their surgery (14 pregnancies) and 1 had twins which were counted as one pregnancy and two deliveries. The average age at the time of delivery was 30.9 years (range 27–42). Twelve of the women had experienced 16 pregnancies prior to having gastric bypass, of which 13 resulted in live births. Two women had miscarried prior to their gastric bypass on one and two occasions respectively.

The issues related to the pregnancy, labour, and delivery are summarised in Tables 1–3 below.

Table 1. Issues related to pregnancy itself

Changes in eating pattern	
increase	8
no change	9
decrease	6 (3 morning sickness)
Weight gain	mean 6.13 kg (range, loss 25 kg – gain 26 kg)
Miscarriages	0 (1 threatened pre-term labour at 22/40)
Pre-eclampsia	1
Gestational diabetes	0
Premature rupture of membranes	1 (30 weeks)

Table 2. Issues related to labour and delivery

Gestational age at birth	mean 37.5 weeks (range 30–40 weeks)
Duration of labour	mean 7.2 hr (range 35 min–26 hr)
Caesarian section	
elective	2
emergency	6
Vaginal delivery	
normal	14
assisted	2

Table 3. Issues related to the babies and the outcome of pregnancy

Live births	25 (1 set of twins)
males	12
females	13
Gestational age at birth	mean 37.5 weeks (range 30–40 weeks)
Birth weight	mean 3038 g (range 1720–4240 g)
Mean APGAR scores	
0 min	7.75
5 min	8.54
10 min	9.38
Breast fed	18 (median time 4.5 mo, range 3 d–12 mo)

Three of the 17 women had a history of infertility prior to surgery, two having polycystic ovary disease (PCOD) alone and one having PCOD and endometriosis. All three had previously received medication to assist fertility without a positive outcome. Each of the two women, who gave a history of miscarriage and no viable pregnancy prior to surgery, conceived within 12 months following surgery and achieved live births.

All 17 women received nutritional supplements in the course of their pregnancies in the form of intramuscular vitamin B12 (54%), daily folic acid tablets (92%) or iron supplements (92%). In 21 pregnancies, supplements were given for a documented deficiency, detected prior to or during the pregnancy, but simply as a precaution against deficiency in the remaining three pregnancies.

Delivery occurred at a mean time of 42.5 months following gastric bypass (range 12-147 months). Four women conceived within 12 months of the bypass, at a time when weight loss was still occurring. Only two of these reported a slowing of the rate of weight loss. Six mothers reported a complication in pregnancy, four of whom also had a complication in labour.

One, who reported low blood pressure and low blood sugars in pregnancy, was induced at 38.5 weeks which resulted in an emergency caesarean section being performed for fetal distress. She delivered a boy weighing 4240g, with APGAR scores of 2, 8, and 10 at birth, 5 min and 10 min respectively. Although the baby needed bag masking for 2 minutes following delivery there were no subsequent concerns with growth and development. The second mother, who suffered chronic pelvic pain throughout pregnancy, developed signs of fetal distress after a labour of 12 hours at 35 weeks and went on to have an assisted vaginal delivery. Although the

baby's weight and APGAR scores were satisfactory he spent 8 days in the neonatal unit as per that hospital's policy for premature babies. There were no subsequent growth or developmental concerns.

A third mother experienced high blood pressure during pregnancy and after a prolonged 24 hr labour at 38 weeks she failed to dilate and developed signs of fetal distress. She underwent an emergency caesarean section and delivered a healthy 2400 g baby girl. Another mother had threatened preterm labour at 22 weeks and spent five nights in hospital. As a consequence of disrupting her symphysis pubis she was in a wheelchair from 28–36 weeks. She went on to have a normal delivery at 38 weeks and produced a healthy 3210 g baby boy. Two other women experienced complications during pregnancy, (one low blood pressure, one incisional hernia) but delivered normal, healthy babies following normal labour.

In addition to the already mentioned four complications in labour, a further five women (total nine) experienced complications during labour. One woman had premature rupture of membranes at 30 weeks and, following steroid therapy, underwent caesarean section. She delivered a 1600 g son, who spent 5 weeks in the neonatal unit requiring ventilatory and feeding support. He exhibited catch up by the end of his second year and there have been no subsequent concerns. Another, whose baby was thought to have intrauterine growth retardation, was induced at 39 weeks and had an assisted vaginal delivery. A healthy baby of normal weight (3035 g) was the result.

The third mother, who gave birth to twins, had an antenatal haemorrhage at 36 weeks which led to an emergency caesarian section. Although one twin spent 3 weeks in the neonatal unit as a result of poor feeding, both babies ultimately had excellent outcomes. In two other instances, babies developed fetal distress necessitating emergency caesarean section in one. Both pregnancies resulted in the delivery of normal, healthy babies.

Congenital abnormalities occurred in two babies born to the same mother, in two consecutive pregnancies. In the first, there was mild spina bifida, caudal regression, heterotaxy, polysplenia, and she now has a colostomy. The second had caudal regression with bladder and bowel dysfunction recognised after 20 weeks of life. It is now known that both have an extremely rare autosomal recessive genetic disorder—*caudal regression with polysplenia and heterotaxy syndrome*. There was no evidence of folic acid deficiency at any stage in either pregnancy, and the mother was receiving folic acid supplements throughout.

In both instances the pregnancy and labour proceeded normally. The birth weights of the daughters were 1720 an 2795 respectively and both had normal APGAR scores at birth. The first baby spent five weeks in the neonatal unit requiring assisted ventilation. Both have displayed growth retardation with delayed milestones as a result of their genetic disorder.

Discussion

There has been growing acceptance over the last 10 years, that bariatric surgery is not only a highly effective tool for managing severe obesity, but is currently the only reliably effective tool for doing so. In spite of the financial and resource implications

for health services, the numbers of operations performed has increased exponentially in the US, Australia, and throughout Europe. New Zealand has been slower than many other countries in recognising the need for and desirability of offering such surgery to severely obese individuals, but that is changing, as the Public Hospital sector begins to face the bourgeoning problems and cost of severe obesity.

There are currently estimated to be in excess of 800 cases being performed per year in New Zealand, a number which is likely to grow steadily over the next few years. Around eighty percent of procedures are currently being undertaken in women, and at least half of these, in women of child bearing age.[14] Pregnancy in those who have undergone surgery will therefore be encountered with increasing frequency. It is appropriate that there be some dissemination of knowledge in respect of the course and outcome of pregnancy after bariatric surgery.

It is known that severe obesity is a complicating feature for pregnancy with an increased incidence of problems related to conception, course of pregnancy, labour and in the neonatal period.[2-4] While gastric bypass surgery is very reliable at bringing about major weight loss in the severely obese,[6,13,14] it does so in part by bringing about a major reduction in food intake, and carries with it a prospect of micronutrient and vitamin deficiency. It is therefore not surprising that concerns are raised from time to time, in the minds of those managing pregnancy in women following gastric bypass surgery, in respect of the course and outcome of pregnancy in such women.

It is known that women with high BMI put on less weight during pregnancy than women of lower BMI. The mean weight gain of 6.13 kg seen in our patients is a little less than the average weight gain for an obstetric population. The Institute of Medicine, Washington DC, recommends upwards of 6.8 kg weight gain for women of BMI>29.[15] In a study of pregnancy outcomes following laparoscopic adjustable gastric banding (LAGB) Ducarme et al reported an average weight gain of 5.5 kg in those who had undergone LAGB compared to 7.1 kg in a control group of obese women who had not undergone surgery.

In spite of this lower weight gain, they reported a significantly lower incidence of pre-eclampsia, gestational diabetes, low birth weight, fetal macrosomia and caesarian sections in the LAGB group compared to the controls and neonatal outcomes were not different.[7]

Our study also seems to indicate that bariatric surgery may reduce the risk of the adverse neonatal outcomes that are usually associated with obese populations such as macrosomia, hypoglycaemia, jaundice and birth defects – an average birth weight of 3038 grams and APGAR scores at 0, 5, and 10 minutes of 7.75, 8.54, and 9.38 certainly reflect excellent outcomes compared to those usually associated with pregnancies in unoperated severely obese women. There was only one baby in our group with a birth weight over 4000 g and the only instance of low birth weight was associated with premature birth at 30 weeks.

The findings of this and other reports are reassuring for those who wonder if a degree of intrauterine growth retardation or restriction may also occur in this context. However, it should be noted that in making this statement we have relied on birth weights rather than a formal monitoring of Gestation Related Optimal Weights as suggested by the GROW programme.

Apart from issues for the two babies born to the same mother, with congenital abnormalities related to a very rare, autosomal recessive syndrome, there are no ongoing concerns regarding growth and development in any of the babies in our series.

The incidence of hypertension in pregnancy in the normal non-obese population is reported to be 12–25% and that of pre-eclampsia 5–7%.[16] In severely obese women, the incidence figures are reported in various case series to be three to seven times higher.[2–4] The incidence of gestational diabetes in pregnancies in normal weight women is 1–2%[17] but in an Australian study of pregnancy in obese women the incidence was seven times higher.[3]

Given that only one mother in our series developed hypertension and none developed gestational diabetes, it seems reasonable to conclude that these complications are less likely to occur than in pregnancies of obese women who have not undergone bariatric surgery. This statement is supported by other reports of pregnancy outcomes following bariatric surgery.[7–12]

It is perhaps important to note that oral glucose tolerance testing will not usually be possible following gastric bypass because of intolerance to 75 g of glucose by mouth. Such an intake will generally lead to severe dumping and vomiting, which will affect the test result. Detection of gestational diabetes should therefore rely on the measurement of fasting glucose and perhaps 2-hour post-prandial glucose.

Six deliveries (24%) were by emergency caesarian sections. This compares with reported figures of 10–15% in pregnancies in the non-obese population[16] and reported rates 2.5–3 times higher in an obese obstetric population.[2,3] In two of the six instances caesarian section was done for ante-partum haemorrhage in a known twin pregnancy. It is well known that this complication is significantly more common in twin pregnancies. Another was for a premature delivery at 30 weeks and the remaining three (12%) for fetal distress at term. Perhaps our caesarian section rate is then best considered similar to or less than that for obese women who have not undergone bariatric surgery.

The caesarean section rate is also affected by parity, and previous caesarean section, when severe obesity existed, and also by practice norms. Thus the rates reported for bariatric surgery patients in North America are not surprisingly rather higher.[9,11]

It is now known that gastric bypass surgery improves fertility by reducing insulin resistance. Our study supports this finding—3 women with diagnosed infertility who had been unable to conceive despite medical treatment prior to surgery, did conceive following surgery. Two others, each of whom had experienced one or more miscarriages and no viable pregnancies prior to surgery, conceived within one year of surgery, suggesting a degree of subfertility prior to gastric bypass surgery.

It has often been thought and advised that following bariatric surgery, women should not conceive within the first year when rapid weight loss is taking place. In our study group, four women did conceive within this timeframe but in no instance was there any basis for concern in relation to the baby's growth or development. Indeed, in two of these women, a slowing of weight loss was noted during the pregnancy.

Another report of pregnancy in 26 women following laparoscopic gastric bypass similarly found no greater complication rate in four pregnancies where conception occurred within the first 12 months following surgery when compared with those that occurred beyond the first 12 months.[9]

Small bowel obstruction requiring surgery was not seen in this series of patients who all underwent *open* gastric bypass surgery, but has been reported particularly in pregnancies following *laparoscopic* gastric bypass.[18,19] Open surgery is generally required to address the problem, with a significant prospect of need for bowel resection and even maternal and infant mortality.[18] The frequency of this complication remains uncertain but given the high numbers of procedures in women of child bearing age that may be expected to be carried out in the future, this is of some concern. Knowledge of the possibility and its early recognition is key to avoiding significant risk to both mother and baby.

Diagnosis is difficult and problematic because of the reluctance to undertake abdominal X-rays during pregnancy and because of confusion with abdominal signs and symptoms in the pregnant state. The complication is caused by internal herniation of small bowel, through mesenteric defects created at the time of the bypass, and may occur even when these have been closed. This complication is less often seen after open gastric bypass because mesenteric defects are more easily and reliably closed at open surgery and because adhesion formation may also reduce the likelihood of internal hernia occurrence.

This study, although involving relatively small numbers, when considered alongside other published reports indicates that pregnancy following bariatric surgery, far from being of particular concern, is likely to proceed more normally that pregnancy in severely obese women. It seems that bariatric surgery reduces the rate of comorbidities conferred by obesity in pregnancy such as the gestational diabetes, hypertension, spontaneous abortion, pre-eclampsia and macrosomia.

Even when mothers conceive within the first year following surgery, there do not appear to be any significant nutritional issues for their babies. Given the known reduced intake and absorption of a number of vitamins that occurs particularly after gastric bypass surgery, it is very important that mothers be taking folic acid and iron supplements throughout pregnancy and that their vitamin B12 levels are watched and supported if necessary.

It is also sensible that a regular multivitamin tablet be taken. The principal and only reported downside for pregnancy following gastric bypass, particularly *laparoscopic* gastric bypass, is the possibility of small bowel obstruction occurring in pregnancy from an internal hernia. Such an occurrence will usually require laparotomy, and is associated with serious risk to both mother and baby. Prompt and careful fluid and surgical management, is necessary for good outcomes.

Despite the reassuring findings of this study it remains desirable that expectant mothers who have undergone surgery for severe obesity should be kept under careful and regular supervision throughout pregnancy.

Competing interests: None known.

Author information: Nikhil Sapre, House Surgeon[2,3]; Karen Munting, Practice Nurse[1]; Archana Pandita, House Surgeon[2]; Richard Stubbs, Surgeon and Professor[1,3]

1. The Wakefield Clinic, Wellington
2. Waikato Hospital, Hamilton
3. Wellington School of Medicine and Health Sciences, University of Otago, Wellington

Correspondence: Professor Richard Stubbs, Wakefield Gastroenterology Centre, Private Bag 7909, Wellington, New Zealand. Email: rsstubbs@wakefieldclinic.co.nz

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Radiotherapy utilisation in lung cancer in New Zealand: disparities with optimal rates explained

Graham Stevens, Wendy Stevens, Sudha Purchuri, John Kolbe, Brian Cox

Abstract

Background and purpose The intervention rate (IR) of radiotherapy (RT) is important for health service planning. As actual IRs are commonly lower than those predicted by models, we sought to determine the reasons for this discrepancy, using lung cancer in a mixed urban-rural region of New Zealand (NZ).

Methods The appropriate utilisation of RT was calculated as the sum of the actual utilisation 3 years post diagnosis (88% of cases deceased), the estimated utilisation of the 12% remaining alive, and the percentage of cases that may have benefited from RT but did not receive it.

Results The actual utilisation was estimated as 43% (range 40–48%). A further 8% of deceased cases may have benefitted from RT (but were not referred), giving an appropriate utilisation of 51%. An additional 3.5% that may have benefitted from RT declined management. The difference from modelled IRs was due to a combination of early mortality, refusal of treatment and assumed higher RT treatment rates for many clinical scenarios.

Conclusion The appropriate utilisation of RT was substantially lower than IRs derived from models. The assumptions from which these models were derived may result in over-estimates for resource planning purposes.

The intervention rate (IR) for radiotherapy (RT) is the proportion of cases that receive RT at any stage during the course of the disease. The IR is important to clinicians, health planners and funders, as it is directly linked to the requirement for high capital cost irradiating equipment and staff.

Using evidence-based decision tree models, the "optimal" or "ideal" intervention for lung cancer has been estimated to be 76% in Australia[1]; 61% in Canada;[2] 63% in Scotland[3] and 66% in England,[4,5] but has not been modelled for New Zealand (NZ). Actual IRs also vary widely internationally, often with marked deviation from the modelled rates. The IR for RT in the treatment of lung cancer is unknown in NZ.

Lung cancer is the commonest cause of cancer death in NZ.[6,7] It accounts for 9% of all new cancer registrations[6] and about 8% of all cancer patients that receive RT.[8] An audit of the management of lung cancer patients in the Auckland-Northland region of NZ[9-12] provided the opportunity to assess the actual IR for lung cancer and to compare the results with estimates derived from published models.

The Auckland-Northland region which has a population of 1.5 million (37% of the NZ population), is serviced by a single regional oncology service. This region is divided into four District Health Boards (DHBs). Those within the greater Auckland urban area are Auckland DHB (ADHB), Counties Manukau DHB (CMDHB) and

Waitemata DHB (WDHB). Northland DHB (NDHB) is within a predominantly rural area 160–320 km north of Auckland.

During this study, RT for the entire region was available only at Auckland City Hospital (within ADHB), although radiation oncology clinics were held regularly in all DHBs. Maori and Pacific peoples in the region are overly represented in areas of socio-economic deprivation.

We report the RT utilisation in lung cancer patients, compare actual utilisation with international estimates of optimal RT utilisation and account for the difference between the actual and optimal IRs.

Method

A retrospective review of the clinical records of patients diagnosed with lung cancer in 2004 in the Auckland-Northland region was undertaken to document patterns of initial secondary care management and RT utilisation throughout the course of the disease.[9–12] The study was approved by the Northern Ethics Committee.

Eligibility criteria included a diagnosis of primary lung cancer (ICD-10 33-34), date of diagnosis during the calendar year 2004, and receipt of some component of initial secondary care management in the Auckland-Northland region. Cases managed entirely within primary care (<13 cases) and those with a post-mortem diagnosis of lung cancer (19 cases) were excluded. Cases were identified from regional hospital and oncology databases and were checked against a listing obtained from the NZ Cancer Registry (NZCR) to ensure completeness of the cohort.[13] A total of 565 eligible cases were identified. Data relating to the demographics, to the cancers and to the initial management of all cases were collected from the clinical records. For some analyses, non small cell lung cancer (NSCLC) stages I and II were combined. When NSCLC and small cell lung cancer (SCLC) cases were combined for analysis, limited stage SCLC was combined with stage III NSCLC and extensive stage SCLC was combined with stage IV NSCLC.

Ten cases were excluded from the assessment of RT utilisation, being 7 cases that left the region prior to any decision regarding treatment and 3 cases that did not have primary lung cancer on subsequent histological review. A total of 555 cases were eligible for assessment of RT utilisation.

For cases that died without receiving RT, but survived for a minimum of one week following diagnosis, the clinical history from presentation until death was summarised from all available documentation in secondary care medical records. This summary consisted of a narrative component and two sets of tick boxes; one being recognised indications for RT (haemoptysis, bronchial obstruction, mass effect, bone pain, brain metastases) and the other being relative contraindications for RT (pleural effusion, marked frailty or weight loss, liver or adrenal metastases).

The summary contained the patient's age and comorbidities, but did not contain the patient's identity, ethnicity, social circumstances or place of domicile. These summaries were assessed independently by two radiation oncologists (GS and SP) to review whether RT may have been beneficial. The assessment related to whether RT should have been recommended, rather than whether there was a specific indication for RT.

This assessment attempted to mimic the real clinical assessment process, incorporating the complexities of clinical decision-making with consideration of general fitness and comorbidity. However the assessment specifically disregarded a decision by the patient or carers to decline further investigation and treatment.

The assessment was made using the following scoring system: 1 = RT definitely not beneficial; 2 = RT probably not beneficial; 3 = insufficient data or uncertainty in benefit of RT; 4 = RT probably beneficial and 5 = RT definitely beneficial. For cases with non-matching scores, the higher score was used, to provide the most generous estimate of the need for RT. Dates of death to May 2008 were obtained from the NZ Cancer Registry (NZCR).

The "actual IR" was calculated as the sum of RT utilisation at May 2008 (3-4 years post diagnosis with 88% of cases deceased) plus the estimated future use of RT for cases that were alive without previous

RT. The term “appropriate IR” was defined as the IR that would provide adequate RT capacity for all cases that might derive a net benefit from RT and that would accept RT.

This was the sum of the actual IR plus the deceased cases that did not receive RT but for which RT was assessed as potentially beneficial (scores 3-5) and who did not refuse treatment. “Optimal IR” was the proportion of cases identified as suitable for RT in published models.

Analysis was performed using SPSS version 14 (SPPS Inc, Illinois, USA, 2005). The Chi-squared test was used to assess differences in receipt of RT in different population sub-groups. Univariate and multivariate logistic regression assessed associations between factors and receipt of RT. Consistency or agreement of the scores assigned by the 2 ROs was measured by the intra-class correlation coefficient (ICC)[14,15] and the inter-rater reliability (Kappa).[16] Mean and median survivals were calculated; survival was displayed by Kaplan-Meier curves.

Results

The patient and tumour characteristics of the cohort and their patterns of initial secondary care management have been reported elsewhere.[9–12] Most cases (87%) had NSCLC; 13% SCLC. At diagnosis, 25% had localised disease (stages I and II NSCLC), 27% had locally advanced disease (stage III NSCLC or limited stage SCLC), and 44% had metastatic disease (stage IV NSCLC or extensive stage SCLC), with unknown stage in 4%.

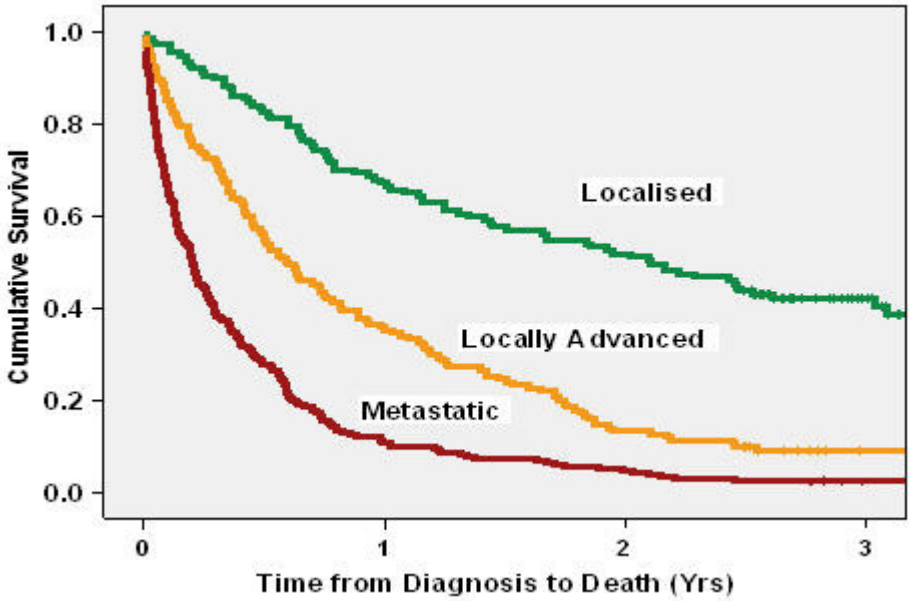
Overall, 50% of cases received some form of initial anticancer treatment; 20% with curative intent and 30% with palliative intent. The remaining 50% of cases received supportive care.

Survival—Of the 555 cases, 486 (88%) had died by May 2008. Of the 69 cases (12%) for which no date of death was registered with the NZCR, 45 had localised disease, 13 had locally advanced disease, 8 had metastatic disease and 3 had unknown stage; 49 were treated with curatively and 20 were treated palliatively. Although all 69 were assumed alive, it was suspected that some had died overseas, with no record of death in NZ.

The median survival from diagnosis for the 555 cases was 178 days (95%CI: 147–209). Of the 486 deceased cases, 33 died within one week of diagnosis, 28 within the second week and 24 within the third week. The observed survival of cases is shown for each extent of disease category in Figure 1. Survival curves had largely flattened by 3 years post-diagnosis.

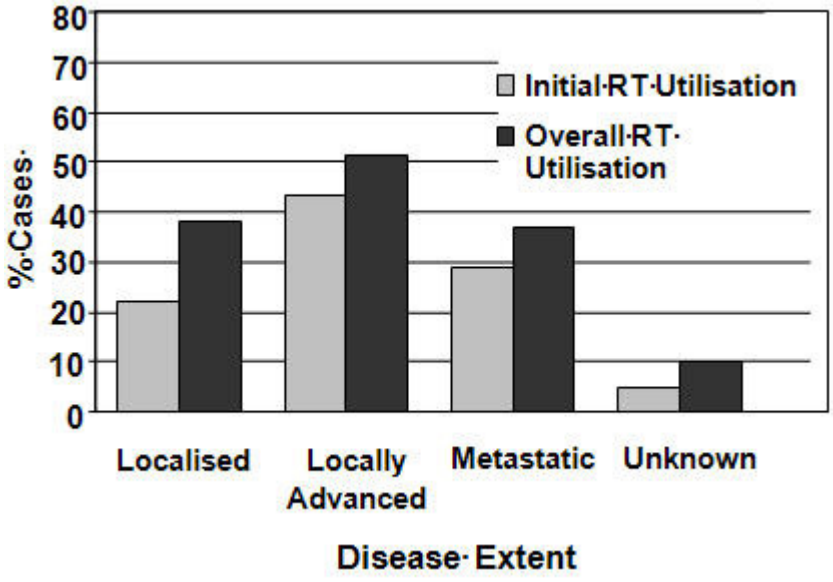
Radiotherapy and the factors associated with its use—Overall, 40% (222) of cases received RT; 30% (166) as a component of initial treatment and 10% (56) subsequently. Figure 2 shows the initial and overall use of RT by stage of disease. In locally advanced and metastatic disease, the majority of RT utilisation occurred as initial treatment. For localised disease, approximately half of RT usage occurred initially, the remainder subsequently.

Figure 1. Kaplan-Meier survival curves according to extent of disease



Note: The curves for each extent of disease tended to plateau by 3 years, allowing an estimate of long-term survival. This information was used to estimate the maximum future utilisation of RT for cases remaining alive at 3 years.

Figure 2. Initial and overall utilisation rates of RT. At all stages of disease, greatest utilisation of RT was at presentation



The characteristics of cases that did and did not receive RT, and the univariate odds of receiving RT, are shown in Table 1. In univariate analysis, utilisation of RT decreased significantly with increasing age ($p<0.0005$) and for cases of Pacific ethnicity

(p=0.03), and was significantly increased for locally advanced disease (p=0.03). RT utilisation did not vary significantly across DHBs in univariate analysis.

Table 1. Characteristics of cases that received RT and the odds of receiving RT

	Total (555)		RT (222)		No RT (333)		Unadjusted		P value†
	N	Col%	N	Row %	N	Row %	OR	(95% CI)†	
Age *(Mean yrs; SD)*	69	(11.6)							
<60yrs	118	21	68	58	50	42			
60-69yrs	161	29	75	47	86	53	0.6	0.4 1.03	0.07
70-79yrs	173	31	58	34	115	66	0.4	0.2 0.6	<0.0005
≥80yrs	103	19	21	20	82	80	0.2	0.1 0.3	<0.0005
Gender									
Male	304	55	124	41	180	59			
Female	251	45	98	39	153	61	0.9	0.7 1.3	0.68
Ethnicity									
European	377	68	154	41	223	59			0.23 ₸
Mäori	92	17	44	48	48	52	1.3	0.8 2.1	0.03
Pacific peoples	53	10	13	25	40	75	0.5	0.2 0.9	0.94
Asian	20	4	8	40	12	60	1.0	0.4 2.4	0.26
Other	6	1	1	17	5	83	0.3	0.1 2.5	0.52
Unstated	7	1	2	29	5	71	0.6	0.1 3.0	
NZDep Deciles									
1-2 (low deprivation)	72	13	33	46	39	54			
3-4	90	16	37	41	53	59	0.8	0.4 1.5	0.55
5-6	89	16	31	35	58	65	0.6	0.3 1.2	0.16
7-8	127	23	54	43	73	57	0.9	0.5 1.6	0.65
9-10 (high deprivation)	175	32	67	38	108	62	0.7	0.4 1.3	0.27
Unknown	2	<1	-	-	2	100	-	-	-
Charlson Comorbidity Index									
0	152	27	64	42	88	58	1.2	0.8 1.8	0.40
1	193	35	90	47	103	53	0.7	0.4 1.03	0.07
≥2	208	37	68	33	140	67	-	-	-
Unknown	2	<1	-	-	2	100			
Tumour Type									
NSCLC	483	87	189	39	294	61			
SCLC	72	13	33	46	39	54	1.3	0.8 2.2	0.28
Tumour Stage #									
NSCLC&SCLC									
Localised	139	25	53	38	86	62			
Locally Advanced	150	27	77	51	73	49	1.7	1.1 2.7	0.03
Metastatic	245	44	90	37	155	63	0.9	0.6 1.4	0.79
Unknown	21	4	2	10	19	90	0.2	0.0 0.8	0.02
NSCLC									
I/II	139	29	53	38	86	62			
III	126	26	58	46	68	54	1.4	0.8 2.3	0.19
IV	197	41	76	39	121	61	1.0	0.7 1.6	0.93
SCLC									
Limited	24	33	19	79	5	21	0.1		
Extensive	48	67	14	29	34	71		0.0 0.3	<0.0005
DHB									
ADHB	126	23	55	44	71	56	0.7		0.12
CMDHB	186	34	65	35	121	65	1.2	0.4 1.1	0.53
WDHB	156	28	74	47	82	53	0.6	0.7 1.9	0.09
NDHB	87	16	28	32	59	68		0.3 1.1	
Initial Management									
Curative Treatment	108	20	53	49	55	51			

Palliative Treatment	176	32	157	89	19	11	8.6	4.7 15.7	<0.0005
Supportive Care Alone	271	49	12	4	259	96	0.05	0.0 0.1	<0.0005
No registered Date of Death	69	12	23	33	46	67			
Dead (by 5/08)	486	88	199	41	287	59	1.4	0.8 2.4	0.23

† P values derived from univariate logistic regression OR: odds ratios indicating the odds of receiving any RT throughout the course of the disease (initial and subsequent RT); 95%CI: 95% confidence interval of the odds ratio; Ŧ Maori cases were significantly more likely to receive initial RT (OR 1.9; 95%CI 1.2–3.1; p=0.006) but they were not significantly more likely to receive RT overall (i.e. throughout the course of the disease); # Tumour stage (clinical stage) at diagnosis.

In the multivariate logistic regression model including age, gender, ethnicity, New Zealand Deprivation Index (NZDep),[9] Charlston Comorbidity Index (CCI),[9] tumour type and stage, cases aged ≥70 years were 70-80% less likely to receive RT than younger cases (70-79yrs: OR 0.3; 95%CI (0.2, 0.6); p<0.0005; ≥80yrs: OR 0.2; 95%CI (0.1, 0.3); p<0.0005). Cases with Pacific ethnicity were 60% less likely to receive RT than Europeans (OR 0.4; 95%CI (0.2, 0.8); p=0.01).

In multivariate analysis, RT utilisation also varied across DHBs, with relatively fewer cases at CMDHB and NDHB receiving RT than those at ADHB or WDHB. After adjusting for age, gender, ethnicity, NZDep, CCI, tumour type and stage, cases at CMDHB had 40% reduced odds of receiving RT (OR 0.6; 95%CI (0.3, 0.9); p=0.03) and those at NDHB had 60% reduced odds of receiving RT (OR 0.4; 95%CI 0.23, 0.7; p=0.004) than cases at ADHB. There was no significant difference in utilisation of RT between ADHB and WDHB.

Estimation of the Actual IR—RT utilisation was 40%, with 88% of cases deceased. Of the 69 cases (12%) with no death registration, 23 had received RT. As any of the remaining 46 un-irradiated cases without a death registration (8% of the total cohort) could contribute to the IR in the future, the actual IR, being the IR when all cases are deceased, had a range of 40-48%. However this maximum value of 48% assumed that all cases alive would require RT (that is, no cases in the entire cohort will be cured).

Using the reported NZ lung cancer 5 year observed survival of 8.2%[17] and Figure 1 to assess the chance of cure in cases that had survived 3 years following diagnosis, we estimated that an additional 3% of the total cohort were realistically at risk of receiving RT in the future for this cancer. On this basis the actual IR for this cohort was estimated as 43% (range 40-48%).

Estimation of the appropriate IR—Of the 333 cases that did not receive RT, 287 (86%) had died. We considered that the 33 cases that died within a week of diagnosis would not have benefited from RT. We also considered that RT would not have been beneficial to a further 29 cases that deteriorated rapidly and died during referral or assessment for RT, either following diagnosis (22) or later in the course of the disease (7).

For the remaining 225 cases, the case files were accessed and summarised, and the potential benefit of RT was assessed using the scoring system as described above. The results are shown in Table 2 for the entire cohort and individual DHBs. There was close concordance in the independent assessments by the 2 ROs. Inter-rater reliability Kappa was 0.83 (95%CI: 0.75; 0.90) and the intra-class correlation coefficient (ICC) was 0.92 (95%CI: 0.89; 0.94; p<0.0005) for single measures (data not shown).

Table 2. Additional cases assessed to potentially benefit from RT

Variables	Cases assessed for appropriateness of lack of RT (225 cases)									
	Overall		ADHB		WDHB		CMDHB		NDHB	
Total Cases	N	Col %	N	Col%	N	Col%	N	Col %	N	Col %
	555	100	126	100	156	100	186	100	87	100
Cases that had not received RT and had died (by 5/08)	225	41	43	34	56	36	85	46	41	47
Scores 1-2 (RT unlikely to have been beneficial)	163	30	33	26	40	26	62	34	28	32
Scores 3-5 (May have benefitted from RT)	62	11	10	8	16	10	23	12	13	15

Col% = Column %.

Of the 225 cases assessed, RT was not considered beneficial (scores 1-2) for 163. Of the remaining 62 cases that may have benefitted from RT (score 3: 8 cases; score 4-5: 54 cases), 20 cases declined to accept recommended management. There remained 42 cases (8% of the total cohort) that had not declined recommended management and had died without receiving RT that may have been beneficial.

Only 2 of these 42 cases had been referred to radiation oncology. Reasons for the failure to refer the other 40 cases to radiation oncology were unknown, although 5 died within 3 weeks of diagnosis and 30 were aged over 70yrs.

The appropriate IR in this study was therefore estimated as 51% (range 48-56%), being the sum of the actual IR (43%) plus the percentage of cases that died without receiving RT but may have benefited from RT at any stage and would have accepted RT (42 cases; 8%).

The proportion of deceased cases that failed to receive RT varied across DHBs (p=0.03) as shown in Table 2. At CMDHB and NDHB, 46% and 47% of cases respectively failed to receive RT compared with 34% at ADHB and 36% at WDHB. Similarly, a higher proportion of cases were assessed as being likely to benefit from RT at CMDHB and NDHB than cases at ADHB and WDHB (12-15% vs 8-10%, non significant difference).

Of note, all cases at ADHB, WDHB and CMDHB with score 5 had declined recommended management. By contrast, only 43% of such cases at NDHB declined management; the remaining 57% were not referred to radiation oncology.

Timeliness of radiation treatment—The median time from receipt of RO referral until the first appointment with RO was 12 days (IQR: 5, 21); with 36% of cases being seen within the 7-day recommendation of the British Thoracic Society.[18] The median time from diagnosis to initiation of RT was 61 days (IQR: 54, 79) for curative treatment and 31 days (IQR: 16, 50) for palliative treatment. The median time from

first appointment with RO until initiation of RT was 30 days (IQR: 28, 38) for curative RT and 14 days (IQR 7, 21) for palliative RT. Cases with emergent conditions (neurological, obstruction of main bronchus or trachea, major haemoptysis) were treated within 1-3 days of referral.

Discussion

The audit of 565 cases, comprising essentially all cases of lung cancer in the Auckland/Northland region in 2004, was the first study to document the management and patterns of care of lung cancer in NZ.[9-13] The audit provided the opportunity to assess RT utilisation in this cohort and to compare utilisation with actual and optimal values internationally. The actual IR was estimated as 43% (range 40-48%), comprising 40% utilisation up to 3 years post diagnosis plus 3% utilisation (range 0-8%) for cases alive without RT at the last follow-up. This value was lower than other reported actual IRs (Australia – 56%;[19] Sweden – 71%[20]) and theoretical IRs derived from decision tree models (61-76%[1,2,3,5]).

We sought to determine whether the lower IR in our study was due to a deficiency in service provision (such as lack of referral), or was appropriate for the clinical context. Therefore each case that had died without receiving RT was reviewed by two experienced ROs. We found high early mortality, with 6% of cases dying within one week of diagnosis, and a further 5% dying during assessment for RT. Although the transit times in this study were longer than recommended, it is unlikely that the impact on the IR was large. Whilst disease progression during a delay may have changed the intent of treatment from curative to palliative,[21] most of these cases would have received palliative RT.

The likelihood of receiving RT varied across the DHBs. Cases at CMDHB and NDHB were less likely to receive RT. All cases at CMDHB assessed to have definitely required RT (score 5) had declined recommended management. However at NDHB, less than half of such cases had declined management; the remaining cases had not been referred to radiation oncology. Geographical isolation could have presented a barrier to access to RT, as reported previously[22] despite regular oncology clinics.

The appropriate IR from this study was 51% and additional 3% declined recommended management. The IR would have been lowered further by inclusion of cases solely managed in primary care (<13) or diagnosed at post-mortem (19), as these cases would have been included in a population cancer registry but were excluded in this study of secondary care management. The difference of 10% between our appropriate IR (51%) and the optimal IRs of 61% in the Canadian model may be explained by early mortality among study cases, with 6% dying within a week of diagnosis and 5% during RT referral or assessment. This is consistent with the poorer survival in NZ compared with Canada.[23,24]

To explain the additional 15% difference between our 51% IR and the Australian optimal IR of 76%, we tabulated the actual and theoretical IRs according to tumour stage (Table 3). Unlike the Canadian model, The Australian model does not adjust for patients declining recommended management and assumes higher treatment rates for cases with advanced disease (stage III and stage IV NSCLC and extensive stage SCLC).

Table 3. Comparison of NZ IRs with international actual and optimal IRs

Variables	Initial IR			Overall IR				
	NZ Actual %	Canadian Optimal[1] %	Australian Optimal[20] %	NZ† Actual %	Australian Actual [18] %	Canadian Optimal[1] %	Australian Optimal[20] %	English Optimal[4] ₸ %
NSCLC								
All	30	46	68	39	60	64	75	
Stage I	24	27	31 (I/II)	35	35	41	50 (I/II)	48 (I/II)
Stage II	27	37		54	57	55		
Stage III	38	76	81	46	81	84	92	63
Stage IV	32	35	83	39	65	66	83	76
Unknown stage	5	-	-	10	-	-	-	-
SCLC								
All	31	45	68	46	55	54	79	
Limited	67	81	94	79	74	82	94	67
Extensive	13	20	49	29	37	33	68	54
Lung Cancer	30	45	68	40†	56	61	76	66

† These figures are likely to be up to 3% higher for the total IR as not all cases had died: lung cancer overall estimate 43%; possible range 40-48%; ₸ English optimal IRs are based on the Scottish estimate but adjusted for English practice. The Scottish optimal estimates were based on the Australian model using some Scottish data[3]. The Scottish estimate for lung cancer overall is 63%[3].

The Australian model is based on clinical indications for which there is evidence for a benefit of RT, and assumes that all cases with this indication should receive RT (with a discount for cases with poor performance status). Therefore the model provides a value for the maximum IR, and does not take into account some of the realities of the clinical situation, such as early mortality, comorbidity, and patient preferences (particularly in a palliative setting), including the option to decline treatment.

Based on our study and assessment, provision of radiotherapy resources to treat 76% of lung cancer cases would have over-estimated the requirement significantly in the Auckland/Northland region. Whilst theoretical models may provide a useful starting point for consideration of resource requirements, it would seem prudent for individual health areas to determine local circumstances when establishing resource requirements. The current NZ study provides the first detailed explanation of the disparity between actual and model-derived IRs for lung cancer. Clearly, individual patient data are needed from other centres to validate the accuracy and utility of theoretical models.

Conclusion

The actual (43%) and appropriate (51%) IRs for lung cancer in this NZ study were considerably less than optimal IRs derived from models. By reviewing the individual files of cases that did not receive RT, we established that the shortfall from the Canadian model was explained by high early mortality.

The additional shortfall from the Australian model was only partially explained by cases failing to accept management and may be related to deviation of modelling assumptions from patient-focused clinical decision making. Use of model-derived optimal IRs for resource planning should be used with caution.

Competing interests: None known.

Author information: Graham Stevens, Department of Oncology, Auckland Hospital, Discipline of Oncology, University of Auckland; Wendy Stevens, Department of Oncology, Auckland Hospital Discipline of Oncology, FMHS, University of Auckland; Sudha Purchuri, Department of Oncology, Auckland Hospital, Auckland; John Kolbe, Respiratory Services, Auckland Hospital Department of Medicine, FMHS, University of Auckland; Brian Cox, Hugh Adam Cancer Epidemiology Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin

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Correspondence: A/Prof Graham Stevens, Department of Oncology, Auckland Hospital, Park Rd, Grafton, Auckland, New Zealand. Fax: +64 (0)9 3691606; email: gstevens@adhb.govt.nz

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Prevalence of Raynaud's phenomenon in the adult New Zealand population

Gordon Purdie, Andrew Harrison, Dianne Purdie

Abstract

Aims To estimate the prevalence of Raynaud's phenomenon (RP) in the New Zealand adult population.

Methods 350 adults 18 years and over, random selected from the electoral roll, were sent a postal survey based on the UK Scleroderma Study Group questionnaire. Participants were classified as having RP if they had biphasic colour changes.

Results There was a 67% response rate. The prevalence of RP was estimated to be 18.8% (95% Confidence Interval (CI) 13.0%–27.1%) in females and 4.9% (95%CI 1.9%–13.0%) in males. The prevalence decreased with age. There was a higher prevalence in the warmer north of the country. People of Māori descent and in more manual occupations had more serve symptoms. Among those reporting symptoms 11% (95%CI 7%–17%) had consulted a doctor.

Conclusion New Zealand has high rates of RP. Few people with RP consult medical practitioners about their symptoms.

Raynaud's phenomenon (RP) has been defined as peripheral vasoconstriction in response to cold, characterised by colour changes, pain, and tautness/fullness in the digits.[1] The prevalence of RP is generally found to be less than 10% with some studies finding rates of about 20%. De Angelis et al[2] presented a summary table of studies of RP prevalence. Prevalence ranged from less than 1% to 16% in men and from 3% to 22% in women. Other studies have published prevalence estimates within these ranges.[3–6]

Differences in climate,[7] questionnaires and definitions account for some of the differences in prevalence. The UK Scleroderma Study Group[1] proposed criteria for definite RP of repetitive episodes of biphasic colour (at least two of pallor, cyanosis, erythema), in either cold or normal environments.

Occupational factors increase the risk of RP.[8] Hand transmitted vibration increases the risk of RP.[9] RP may be classified as primary (unrelated to other diseases) or secondary, most commonly in association with scleroderma. Solvent exposure increases the risk of the connective tissue disease scleroderma.[10]

A meta-analysis of follow up studies of people with primary RP (not secondary to a diagnosable disease) found, after an average follow up of four years, that a related disease was diagnosed in 13%, with 65% of these being scleroderma.[11] Higher rates and more severe RP have been observed in people exposed to solvents.[5]

There appears to be no published estimates of RP prevalence in New Zealand. With a geography that ranges from subtropical to sub-Antarctic, New Zealand provides an opportunity to explore effects of latitude. Estimates of the general population

prevalence of RP will allow comparison with exposed workers. This paper presents estimates of the prevalence of Raynaud's phenomenon in the New Zealand adult population.

Method

A random sample of 350 people from the New Zealand electoral roll were sent a postal questionnaire. People with overseas addresses were excluded. The New Zealand Electoral Roll contains 92% of the estimated midyear 2006 resident population 18 years of age and over. The survey was sent in September 2006. Non-responders were sent two reminder letters.

The sample size was chosen so that with an expected 210 responses the 95% confidence interval width for the proportion of people with RP would be within ± 7%.

Raynaud's phenomenon was assessed using the UK Scleroderma Study Group[1] questionnaire with the following questions:

(a) Are your fingers sensitive to cold?

(b) Do your fingers show unusual colour changes? If Yes, do they become white, blue, red or purple?

(c) Have your fingers become numb or had pins and needles in response to cold?

(d) If applicable have the colour changes or numbness described occurred in the absence of cold exposure—i.e. at normal temperature?

Participants were classified as having possible RP if they answered yes to only one colour in question (b) or answered yes to question (c) and definite if they answered yes to at least two colours in question (b). If in addition to two colours they answered yes to (c) and (d) then they were classified as severe.

People were also asked their gender, if they had ever consulted their general practitioner or a specialist concerning their symptoms and if so what was the diagnosis. People's age when the survey was sent was calculated from the period of birth provided by the electoral roll. People's names on the roll were used for gender response rates. Meshblocks on the roll were used for an area-based index of socio-economic deprivation NZDep2006[12] with decile 10 being the most deprived.

District Health Boards, provided by the electoral roll, were used to assign people to Regional Health Authority (RHA) regions. Occupations recorded on the roll were used to assign socioeconomic stratification (SES) using NZSEI-96.[13]

The study was approved by the Multi-region Ethics Committee.

Questionnaire data was double entered. Non-responses to yes or no questions were treated as negative responses. Prevalences were post-stratified to the estimated midyear resident 18 years and over 2006 population estimates from Statistics New Zealand. Confidence intervals for stratified estimates were calculated using log transformations.[14] Proportions were compared with chi-squared tests, chi-squared tests for trends, Spearman rank correlation and Kruskal-Wallis Test.

Results

Response rate—There were two further exclusions due to the persons being overseas. 234 completed questionnaires were returned, a 67% response rate. Eight envelopes were returned as no longer at that address.

Prevalence—The prevalence of RP (definite and severe) was 11.5% (95%CI 7.7%–16.3%) (Table 1). The prevalence of RP with post-stratification by age group and sex was 12.1% (95%CI 8.5%–17.1%).

Table 1. Prevalence of Raynaud's phenomenon (RP)

Variables	Not RP	Possible	Definite	Severe	P-value RP	P-value severity trend
Female						
18 – 24	27% (3)	45% (5)	27% (3)	0% (0)	0.082*	0.18†
25 – 44	40% (17)	35% (15)	21% (9)	5% (2)		
45 – 64	44% (22)	46% (23)	8% (4)	2% (1)		
65 and over	45% (13)	41% (12)	7% (2)	7% (2)		
Female total	41% (55)	41% (55)	14% (18)	4% (5)	0.002‡§	0.036*§
Male						
18 – 24	10% (1)	80% (8)	10% (1)	0% (0)	0.10*	0.040†
25 – 44	44% (12)	48% (13)	4% (1)	4% (1)		
45 – 64	51% (23)	47% (21)	2% (1)	0% (0)		
65 and over	53% (9)	47% (8)	0% (0)	0% (0)		
Male total	46% (46)	50% (51)	3% (3)	1% (1)		*
Māori descent	25% (6)	54% (13)	8% (2)	13% (3)	0.14‡	0.009
Māori descent	45% (19)	44% (94)	9% (19)	1% (3)		
not indicated						
NZDep2006 (decile)						
1 – 3	48% (42)	43% (38)	8% (7)	1% (1)	0.11*	0.15†
4 – 7	41% (39)	48% (46)	7% (7)	3% (3)		
8 – 10	39% (19)	43% (21)	14% (7)	4% (2)		
SES						
1 & 2	54% (13)	33% (8)	13% (3)	0% (0)	0.55*	0.025†
3 & 4	42% (25)	49% (29)	7% (4)	2% (1)		
5 & 6	24% (9)	59% (22)	16% (6)	0% (0)		
RHA						
Southern	40% (21)	51% (27)	6% (3)	4% (2)	0.011‡	0.50‖
Central	41% (25)	52% (32)	7% (4)	0% (0)		
Midland	50% (18)	47% (17)	3% (1)	0% (0)		
Northern	44% (36)	35% (29)	16% (13)	5% (4)		
Total	43% (101)	45% (106)	9% (21)	3% (6)		

* Chi-squared test for trend; † Spearman rank correlation; ‡ Chi-squared test; § Gender comparison; ‖ Kruskal-Wallis Test

The prevalence of RP (definite and severe) was significantly higher in females 17.3% (95%CI 11.3%–24.8%) than males 4.0% (95%CI 1.1%–9.8%) (p=0.002). The prevalence was significantly higher in younger people (p=0.031 chi-squared test for trend). The prevalence of RP with post-stratification by age group was 18.8% (95%CI 13.0%–27.1%) in females and 4.9% (95%CI 1.9%–13.0%) in males.

The prevalence was higher, but not statistically significantly so, in people of Māori descent 20.8% (95%CI 7.1%–42.2%) than others 10.6% (95%CI 6.7%–15.6%) (p=0.14) with a significant trend in severity (0.009). There was a non-statistically significantly trend to higher prevalence in more deprived areas (p=0.11). There was no significant trend in RP with SES (p=0.57) with a significant increase in severity with increasing SES (p=0.025). The prevalence was significantly different between

the RHA areas (χ^2=11.1; df=3; p=0.011), higher in the Northern RHA area, with no significant difference in severity (p=0.50).

Fifty-two percent (95%CI 45%–58%) (121/234) reported their fingers were sensitive to cold. Forty one percent (95%CI 35%–48%) (97/234) reported colour changes, among those, white was the most common 65%, followed by red 36%, purple 24%, and blue 16%. Forty four percent (95%CI 38%–51%) (103/234) reported their fingers become numb or had pins and needles in response to cold.

Fifteen percent (95%CI 11%–21%) reported their colour changes or numbness occurred in the absence of cold exposure. Sixty nine percent (25/36) of these were classified as possible RP. There was a significantly increasing tend with SES, with those in SES 5 & 6 reporting occurrences in the absence of cold exposure 7.8 (95%CI 1.1–56.1) times more often than SES 1 & 2. Symptoms in the absence of cold exposure were reported 2.1 (95%CI 1.0–4.3) times more often by people of Māori descent.

Of those reporting any symptom 11% (95%CI 7%–17%) (17/153) had consulted their general practitioner or a specialist concerning their symptoms. Among those reporting any symptom, there was no significant difference (p=0.50) in the consultation rate of those with RP (definite and severe), 7.4% (2/27), and those without, 11.9% (15/126). The consultation rates were 1.4 (95%CI 0.5–3.7) times higher in people who reported colour changes, 3.6 (95%CI 0.9–15.3) times higher for those whose fingers become numb or had pins and needles in response to cold and 2.3 (95%CI 0.9–5.5) times higher for those with colour changes or numbness occurring in the absence of cold exposure.

Females had a significantly higher consultation rate (15%) than males (5%) (p=0.047). There was a significantly increasing trend in consultation rates with age (p=0.046). Diagnoses reported were nerve damage (four), arthritis (one), rheumatoid arthritis (one), osteoarthritis (one), chill blains (two), poor circulation (three), lack of oxygen (one). Two reported that no diagnosis was made, one was not sure of the diagnosis and one was being investigated. The two people responding to the diagnosis question who were classified as having RP reported lack of oxygen (definite RP) and not sure (severe RP).

Rates for alternative definitions of RP used in other surveys are shown in Table 2.

Table 2. Alternative definitions of Raynaud's phenomenon (RP)

RP definition	Female N=133 % (95%CI) (n)	Male N=101 % (95%CI) (n)	Total N=234 % (95%CI) (n)
Sensitive*, white† occurring at normal temperatures	5% (2–11) (7)	4% (1–10) (4)	5% (2–8) (11)
Sensitive*, at least a biphasic† and numb‡	11% (6–18) (15)	2% (0–7) (2)	7% (4– 11) (17)
Sensitive*, white† and numb‡	21% (14–29) (28)	11% (6–19) (11)	17% (12– 22) (39)
Sensitive*, white†	25% (18–33) (33)	21% (13–30) (21)	23% (18– 29) (54)
Sensitive*, white or blue†	29% (22–38) (39)	22% (14– 31) (22)	26% (21– 32) (61)
Sensitive*	58% (49– 66) (77)	44% (34– 54) (44)	52% (45– 58) (121)

* Sensitive to cold; † colour change; ‡ Numb or had pins and needles in response to cold

Discussion

Adult women had a higher prevalence of RP than men. The prevalence decreased with age. High rates of symptoms were reported by people of Māori descent and SES 5 & 6 (more manual occupations), particularly possible RP or colour changes or numbness occurring in the absence of cold exposure.

Response bias could account for some of the differences found with many of the groups with higher prevalence having lower response rates.

The UK Scleroderma Study Group classification criteria[1] was chosen as a brief questionnaire that would allow international comparisons. Their classification criteria that relied on answers to most questions failed to classify 13% of responses, which were classifiable with modified criteria. The criteria, which require at least two colour changes for definite RP, may be failing to identify vibrating white finger syndrome as RP. Higher rates of possible RP were found in groups likely to have higher rates of exposure to vibration in their occupations. Failure of the classification system to classify RP from vibration damage is supported by three of the four people reporting diagnoses of nerve damage being classified as possible RP (all with white colour change and numbness or pins and needles in response to cold).

The UK Scleroderma Study Group found that their questionnaire criteria classified as possible RP 29% of those classified as definite and severe RP by majority clinician assessment.[1] In that study all those classified as RP (definite or severe) by the questionnaire criteria were classified as either definite or severe by majority clinician assessment, suggesting that the prevalence of RP is being underestimated by this study.

The prevalence in New Zealand, 12%, is in the upper range of those found in other countries. Other surveys using similar questions and the same definition have found a lower prevalence of RP. The prevalence found in Massachusetts[4] was 8%, in a Greece population[15] 5% and in a Turkey population[6] 6%. Other surveys with different definitions of RP have similar rates for comparable questions in Demark,[16] Great Britain[17,18] and Estonia.[19] Lower rates were found in South Carolina,[20-22] Sweden,[23] Massachusetts,[24] Netherlands,[25] Spain,[26,27] Great Britain[9] and Italy.[2] This study was within the range of rates found in France.[7] A relationship between RP and climate was found in France.[7]

The cooler climate in New Zealand may have influenced higher prevalence found. No trend with climate in New Zealand was found in this study. The higher prevalence in the Northern RHA may result from migration of people with RP to warmer areas. A five region prevalence study[7] found that the majority of people with RP in the warmest regions had previously lived in a colder climate.

This study found a higher prevalence of RP for women than men, as has been found in most[2,4-7,9,15,17,18,22,24,25,28] but not all[3,7,19,20,26,27,29,30] other studies.

Although there was a trend of decreasing RP with increasing age this is not consistent with some other studies reporting increasing trends.[4,19] The decreasing prevalence

with age in this study is consistent with serial examinations of the Framingham Heart Study offspring cohort.[31] People were assessed at baseline and approximately seven years later, there was a significant decrease in prevalence from 9.4% to 5.2%.

Variation in occupational vibration exposures may account for some of the differences between studies. In a survey in Japan[29] 49% of males with RP were exposed to vibration, with vibration contributing to the increase prevalence with age for men and the small gender difference. In the Framingham study[31] 27% of males with RP were exposed to vibration.

There was no significant relationship between RP classification and whether a person had consulted a doctor about their symptoms. Experiencing fingers becoming numb or having pins and needles in response to cold or having colour changes or numbness occurring in the absence of cold exposure may influence medical consultation. Females were more like to consult, possibly partly reflecting high consultation rates for female generally.[32] Older people may have had symptoms for a longer during which might have influenced the increasing consultation rates with age. Low consultation rates for RP have also been found in other studies.[6,9,15,17,18,27]

In this study 15% (95%CI 11%–21%) reported colour changes or numbness that occurred in the absence of cold exposure, with 3% (95%CI 1%–5%) of people classified as severe. Few other studies have reported similarly classifications. A study in Sweden[23] found 20% of women reported cold and white fingers and for 21% of a sample of these it occurred in summer, giving a prevalence of 4% close to a similar estimate in this study (table 2). A study in Greece,[15] with a lower prevalence of RP, found 27% (7/26) of participants with RP reported colour changes in emotional stress, similar to the proportion in this study with RP reporting symptoms at normal temperature, 22% (6/27).

New Zealand has high rates of RP. There are disparities, with females, Māori and those in more manual SES having higher rates of symptoms. Occupational exposures are likely to contribute to the later differences.

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Author information: Gordon Purdie, Senior Research Fellow, Department of Public Health, University of Otago, Wellington, Andrew Harrison, Senior Lecturer, Department of Medicine, University of Otago, Wellington, Dianne Purdie, Medical Cytologist, Wellington

Correspondence: Gordon Purdie, Department of Public Health, University of Otago, Wellington, PO Box 7343, Wellington South, New Zealand. Fax +64 (0)4 3895319; email: gordon.purdie@otago.ac.nz

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The opinions of newly licensed drivers in New Zealand on the minimum car driver licensing age and reasons for getting a licence

Dorothy Begg, John Langley, Rebecca Brookland, Anna McDowell, Shanthi Ameratunga, John Broughton

Abstract

Aim To investigate the opinions of newly licensed drivers towards the minimum age of car driver licensing, and reasons for getting a licence.

Method The New Zealand Drivers Study (NZDS) is a prospective cohort study of 3992 newly licensed car drivers in New Zealand, recruited between 1 February 2006 and 31 January 2008 from driver licensing agencies and licensing courses throughout New Zealand. The cohort comprises 51% females and 49% males, 49% were aged 15 years and 28% 16–17 years, 55% self-identified as New Zealand European, 21% Māori, 13% Pacific, 11% Asian, and 15% as "other". After passing the learner licence theory test all participants completed a questionnaire that included a range of questions on driver licensing topics, including minimum driver licence age and reasons for getting a car driver's licence.

Results Overall, 51% of newly licensed drivers supported 15 years as the minimum age to start licensing but this varied significantly by the age, gender, and residential location of the learner driver. The most frequently reported reason for getting a licence related to independence and freedom. This applied equally to males and females, rural and urban drivers, and across all ages, although for learner drivers aged 18^{+} years, to drive to work was also a very important reason for having a licence.

Conclusion Contrary to what many may believe to be the case, the evidence presented here showed that there was not universal opposition by young people to raising the driver licensing age. Also those in rural and urban areas had much in common with respect to the reasons for obtaining a licence. With respect to the latter it is worth noting that travel for work was of most relevance to the learner drivers aged 18 years or older. Overall, these findings suggest that increasing the minimum age for licensing would have relatively little impact on essential travel among young people in New Zealand.

In 1985, New Zealand had the second highest population-based traffic crash fatality rate for young people aged 15–24 years, in the OECD.[1] In response to this unacceptable situation, the Ministry of Transport proposed that New Zealand consider introducing a graduated driver licensing system (GDLS).[2] The GDLS was to replace a licensing system which allowed a young person to gain a full privilege car driver's licence on their 15^{th} birthday, and this could be achieved by answering a few questions on the road code and passing a relatively undemanding driving test.

An alternative strategy to the GDLS, that was also considered by the Ministry, was to raise the minimum driver licensing age. It was decided, however, that a GDLS was a

more constructive alternative because it addressed the combination of youthfulness and lack of driving experience, which are the two key factors associated with high crash risk among young drivers.[2]

The GDLS was introduced in New Zealand on 1 August 1987, and there is good evidence that it has contributed to a significant reduction in serious traffic related injury among young people.[3] Nevertheless, the adolescent/young adult age groups continue to be over-represented in the traffic crash statistics.

In 2007, young drivers (aged 15–24 years) were involved in 127 fatal crashes, and were considered to be at fault in 106 of these.[4] In the same year, the total social cost of the crashes (fatal and injury crashes) where a young driver was considered at-fault was $1.1 billion, which is almost a third of all the social costs associated with all injury crashes.[4] In addition, internationally New Zealand still has an unenviable population based crash record for young people, with only one out of 28 OECD countries having a higher crash rate among the 15–17 age group.[5]

Overseas evidence [6] indicates that allowing young people to commence the licensing process at age 15 years, and to drive unsupervised on a public road at 15½, is likely to be an important factor contributing to this situation.

In recent years the New Zealand Government has acknowledged the problem of high crash rates among young drivers and late 2007 a bill to raise the minimum driver licensing age from 15 to 16 years, and to extend the length of the learner licensing period from 6 months to 12 months, was introduced into the House of Parliament (the House).[7] After its first reading the bill was referred to Select Committee for detailed consideration. As of May 2009 this bill was still in "business", but submissions had not been called.

Prior to the bill that is currently before the House there was one other relatively recent attempt to raise the minimum driver licence age in New Zealand. From 1995–1999 the Land Transport Safety Authority (LTSA) (the government agency responsible for driver licensing at that time) undertook a major review of all licensing policy. As part of this extensive review, which involved wide consultation with experts and the community, a Land Transport bill was introduced into the House in 1998.

This bill included a clause that sought to raise the minimum driver licensing age from 15 to 16 years. The LTSA and the New Zealand Police were sufficiently confident this change would occur that raising the licensing age was published as one of the key new road safety measures in the Safety (Administration) Programme for that year.[8] However, this clause of the bill was rejected by the House and the minimum licensing age remained at 15 years. [9]

In the debate associated with the earlier attempt to raise the licensing age, very little evidence was produced to support or negate the arguments for or against raising the age. To ensure the discussion for the current bill is better informed we (Begg and Langley) undertook a critical examination of the evidence for and against the typical arguments that are promoted against raising the licensing age.[10]

The results of this examination showed that age, independent of driving experience, is a major determinant of risk. The period of highest risk is when solo driving commences, therefore increasing the minimum age when licensing can commence,

and extending the supervised learner licence stage for another 6 months, should produce substantial safety benefits.

While there is little doubt that the rural sector would be somewhat disadvantaged, because of the distances they have to travel and the lack of alternative transport, if the licence age was raised, contrary to commonly expressed views, the evidence showed that relatively few 15 or 16 year olds, both urban and rural, were employed and of those that were relatively few drove themselves to work.[10]

Furthermore, both main political parties have publicly stated that they support increasing the school leaving age to 17 or 18 years. If this should eventuate, then driving to or from work should no longer be an important reason for not raising the licensing age.

Although the Begg, Langley study[10] provided evidence for some key issues related to driver licence age, it also highlighted the paucity of evidence in other areas. For example, it is often assumed that all young people believe that they should be allowed to get their car driver's licence as soon as they turn 15, but we found no evidence to support, or refute, this view. Also, it is often claimed that young people need their licence for essential travel such as to work, school, or sport, and this is especially important for young people in rural areas where there is no public transport.

Apart from the census data on travel to work, we found very little evidence on the reason why young people need a driver's licence. The lack of New Zealand based scientific evidence to inform driver licensing policy was one of the main reasons why the New Zealand Drivers Study (NZDS) was developed. As part of the NZDS, a large cohort of newly learner licensed car drivers from throughout New Zealand were asked questions relating to driver licensing policy, and it is these questions which are the subject of this paper.

Our aim was to investigate the opinions of newly licensed drivers on what they considered should be the minimum age to start car driver licensing, and their reasons for getting a licence. These topics were examined by factors that, based on commonly held beliefs, could be expected to influence their opinions, namely: gender, age, and their place of residence (rural versus urban). There is a popular belief that males are much more enthusiastic about cars than females (e.g. boy racers). Therefore, it was predicted that males would support a younger licensing age than females. For age, it was predicted that the current minimum licence age of 15 years would receive more support from the 15 year old learner drivers, than those aged 16 years or older. Finally, there is a widespread belief that because of distance and lack of public transport, young people in rural communities need a car driver's licence more than their urban counterparts. Therefore, we predicted greater support for a 15 year old licence age among the rural drivers than the urban drivers.

Method

The New Zealand Drivers Study (NZDS), is a prospective cohort study of 3992 newly licensed car drivers in New Zealand. The protocol for the study has been previously published[11]. The NZDS cohort was recruited between 1 February 2006 and 31 January 2008 from driver licensing agencies and licensing courses throughout New Zealand.

The main locations where face-to-face recruitment took place were Auckland, Gisborne, Ruatoria, Wairoa, Napier, Hastings, Christchurch, and Dunedin. In addition, participants from rural and less densely populated areas were recruited by responding to a postcard placed at licensing centres. All participants were recruited soon after they passed their car learner licence theory test (Class 1L Licence) at which time a consent form was signed and a self-administered baseline questionnaire completed. Data from the baseline questionnaire was used in the present study.

Demographic characteristics of NZDS cohort—Each member of the NZDS cohort provided data on the following demographic characteristics: gender, age (at time of obtaining a learner licence), and ethnicity. The ethnicity question was the same as used in the New Zealand Census,[12] which allowed multiple ethnicities to be recorded.

The Statistics New Zealand "urban/rural profile" was used to allocate the residential address of each study participant to a urban or rural area.[13] Under this classification the main urban areas were classified urban, and the rest were classified rural. The main activity currently engaged in was recorded as: secondary school student, university student, other student (e.g. polytechnic), full-time employed, part-time employed, homemaker, unemployed, other. Those in paid employment were asked to report their main occupation.

Socioeconomic variables—The *NZDep2006* score is a measure of socioeconomic deprivation created by combining nine variables, which reflect eight dimensions of deprivation, from the 2006 Census.[14] The residential address of each study participant was used to assign a deprivation score based on the meshblock in which they lived.

Licensing issues—One question on licence age was examined in this study—*What do you think should be the minimum age that you can start getting your driver's licence?* Several questions on reasons for getting a licence were examined:

How important are each of these reasons to **you** for getting your car drivers licence?:

- To use for identification (ID);
- Freedom to go where I wish;
- To drive to school;
- To drive to or from a job;
- To go to sporting activities
- Your parents want you to;
- To help parents;
- To go out with mates;
- Your friends have licences.

The response options were: Not at all important; Important; Very important.

To provide participants an opportunity to record the main reasons why they wanted to get a licence, two further semi-structured questions, which allowed a free text response, were also included. *"Overall how important is it for you to have your car drivers licence. And why?*"; *"Why are you getting your Learner Licence now?"* Free text responses were examined and classified by theme.

Statistical analysis—The analysis was undertaken using SAS v9.1 software. The Chi-squared statistical test was used to examine difference between categorical variables. A multivariate logistic regression analysis was conducted to examine the independent effect of gender, residential location, and age on a minimum licence age of 15 years or older.

Results

Of the 3992 study participants, 2685 (67%) were recruited at a licensing centre, 916 (23%) at a learner licence course, and 391 (10%) by way of the postcards. For those recruited at the licensing centre, of all the eligible newly licensed drivers invited to take part in the study the recruitment rate recorded across all research assistants was approximately 75%.

For those recruited at a learner licence course the overall recruitment rate was approximately 90%. A recruitment rate for postcard recruitment could not be calculated because no denominator can be ascertained.

The demographic characteristics of the 3992 members of the NZDS cohort are summarised in Table 1. This shows that gender was evenly distributed with 51% females and 49% male, nearly half (49%) were aged 15 years, 55% identified as New Zealand European, 21% as Māori, 13% as Pacific (includes Samoan, Cook Island Māori, Tongan, Niuean), 6% Chinese, 5% Indian, and 15% as "other". The majority were students: 69% secondary school, 10% university or "other".

Table 1. Demographic characteristics of the New Zealand Drivers Study cohort

Gender	**N**	**%***
Females	2049	51
Males	1943	49
Total	3992	100
Age (at learner licence) in years		
15 years	1975	49
16 years	712	18
17 years	413	10
18 years	212	5
19 years	125	3
20^{+}years	555	14
Total	3992	99
****Ethnicity**		
NZ European	2191	55
Māori	824	21
Pacific	516	13
Asian	444	11
Other	613	15
Current main activity		
Secondary school student	2747	69
University or other student	405	10
Full-time, part-time employed	460	12
Homemaker	84	2
Unemployed	134	3
Other (includes missing)	162	4
Total	3992	100
Number in paid employment (by age)		
15 years	386	19***
16 years	203	29
17 years	133	32
18^{+} years	475	53
NZDEP2006 score		
1 Least deprived	488	12
2	414	10
3	383	10
4	374	9
5	323	8
6	340	9
7	318	8
8	317	8
9	400	10
10 Most deprived	635	16
Total	3992	100

Place of residence		
Urban areas	3557	89
Independent & satellite urban areas	122	3
Rural areas with high or moderate urban influence	152	4
Rural areas with low urban influence	91	2
Highly rural/remote areas	70	2
Total	3992	100

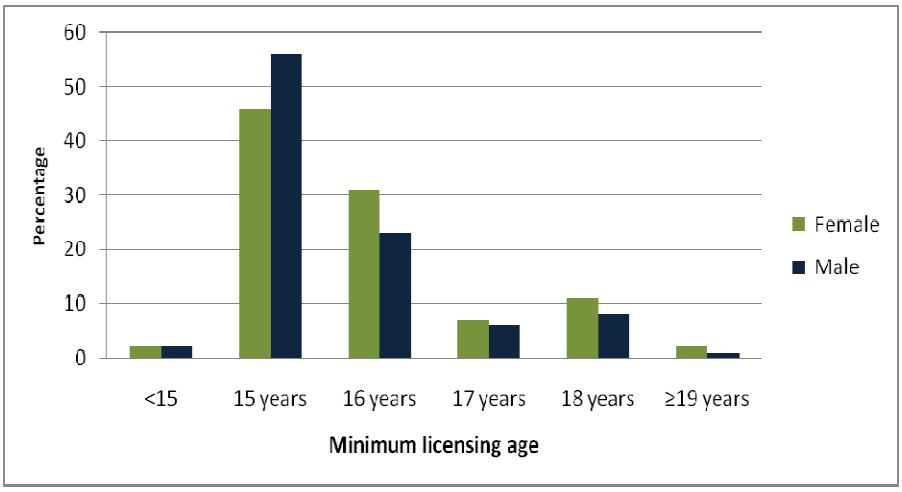
*% Some totals many not equal 100 due to rounding; **More than one ethnicity could be recorded therefore total exceeds 3992; ***The % of each age group employed.

The number in paid employment varied by age, with 19% of 15 years olds, 29% of the 16 year olds, 32% of the 17 year olds and 53% of those aged 18^{+} years in paid employment. The distribution of NZDep2006 scores showed that all levels of social deprivation are represented in the NZDS cohort, with an especially high representation (16%) from the most deprived areas (score=10).

The distribution by residential location (rural/urban) showed that 89% were from a main urban area and 11% rural (which compares with 70:30 ratio for the total New Zealand population).[13]

Minimum driver licensing age—The responses from the learner drivers to the question about what they considered should be the minimum age to start getting a car driver's licence are presented, by gender, in Figure 1. The main difference by gender was a higher proportion of males (56%) than females (46%) supported 15 years but 16 years was supported by a higher proportion of females (31%) than males (23%).

Figure 1. Newly licensed car drivers opinion on minimum age to start getting a driver's licence, by gender of the driver

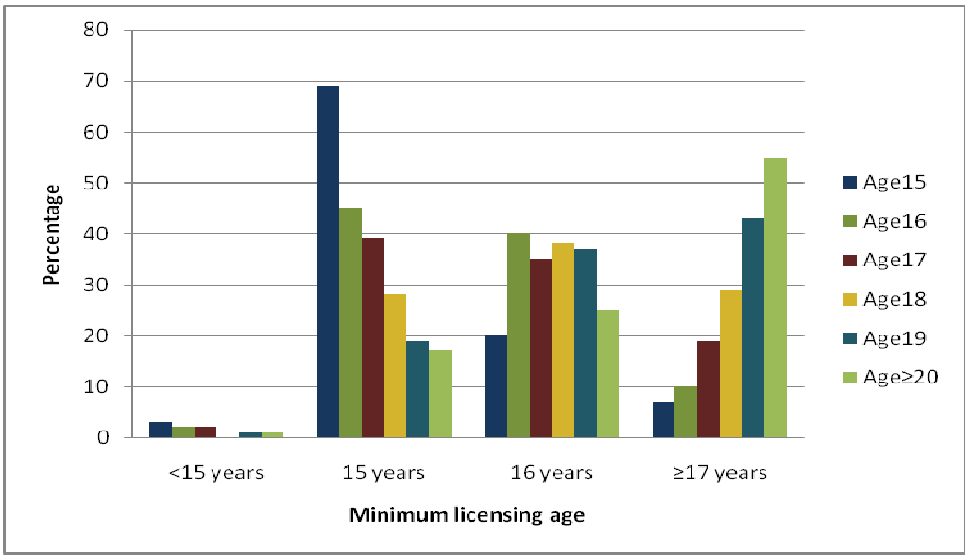


Note: Missing data not included in figure therefore totals may not equal 100%.

When examined by the age of the newly licensed driver (Figure 2), a minimum licence age of 15 years was supported by 69% of the 15-year-old learner drivers but less than 20% of those aged 19 or older. Support for 16 years as the minimum starting

age was reasonably evenly distributed (20%–40%) across all ages and 17+ years was most strongly supported by the older drivers (55% of the 20+ age group) and least supported by those aged 15 or 16 years (7% and 10%, respectively).

Figure 2. Newly licensed car drivers opinion on minimum age to start getting a car driver's licence, by age of the driver

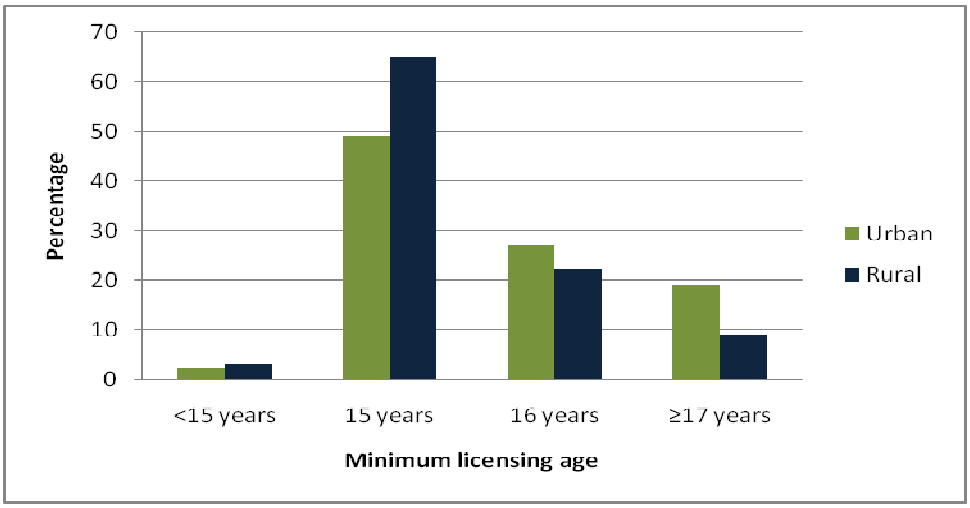


Note: Missing data not included in figure therefore totals may not equal 100%.

Figure 3 shows that a minimum licensing age of 15 years received support from 65% of the rural and 49% of the urban learner drivers, 16 years from over 20% of both urban and rural, and 17 years by nearly 20% of the urban drivers. When examined by gender, 15 years was supported by 70% of rural males, 60% of rural females, 54% of urban males, and 44% of urban females.

To determine the independent effect of gender, residential location (rural versus urban) and age of the learner licence driver (15, 16, 17, or 18+ years) (i.e. after controlling for each of the other variables) on whether the minimum licensing age should be 15 years or older, a multivariate logistic regression analysis was conducted. The adjusted results are presented in Table 2.

Figure 3. Newly licensed car drivers opinion on minimum age to start getting a car driver's licence, by place of residence



Note: Missing data not included in figure therefore totals may not equal 100%.

Table 2. The effect[1] of gender, residential location, and age at learner licence on a minimum licence age of 15 years or older.

Variables	Odds ratio	95% confidence interval		P value
Females	1			
Males	1.3	1.1	1.5	<0.001
Urban	1			
Rural	1.6	1.2	2.0	<0.001
Age 15 years	1			
Age 16	0.36	0.30	0.43	<0.001
Age 17	0.29	0.23	0.36	<0.001
Age 18 +	0.11	0.09	0.13	<0.001

[1] Each variable adjusted for other variables in the table.

Reasons for getting a car driver's licence—The level of importance assigned to a given list of reasons for getting a licence by gender, age of learner driver, and by residential location (urban or rural) are presented in Tables 3-5

Gender: Table 3 shows that, except for "to drive to school", the level of importance for each of the reasons differed significantly by gender. "Freedom to go where you wish" and "To drive to or from a job" were the most important reasons reported by the highest proportion of both males and females, but were *very important* for a slightly higher proportion of females (53% and 52%, respectively) than males (46% and 49% respectively). "To go to sporting activities" was *very important* for a higher proportion of males than females (40% compared with 32%). Getting a licence because "Your friends have a licence" was *not at all important* for 53% of females and 44% of males.

Age of learner driver: Table 4 shows that the level of importance for each of the reasons for getting a licence, by the age of the learner driver. To simplify the presentation of these results the responses from drivers aged 18^{+} years were combined. For all of the reasons, the level of importance differed significantly by the age of the learner driver. "To drive to or from a job" was *very important* for a larger proportion of the 18^{+} year old learner drivers (66%) compared with the 15 year old learners (42%). "Freedom to go where you wish" was *very important* for nearly half of the 15-17 year olds, and 54% of the 18 years or older. The importance of a having a car driver's licence "To use for identification (ID)" increased with age, with only 17% of 15 year olds considering it *very important* compared with 50% of those aged 18 years or older. "To drive to school" was *very important* for around 28-32% of all ages, and "to go to sporting activities" was *very important* for 39% of 15 year olds and 32% of the other age groups.

Table 3. Importance of reasons for getting car driver's licence, by gender of the learner driver (%*)

Variables	Not at all important	Important	Very important	Chi squared p value (df=2)
To use for identification (ID)				
Females	28	41	30	11.2
Males	26	47	27	.004
Freedom to go where you wish				
Females	10	37	53	19.4
Males	13	41	46	<.001
To drive to school				
Females	33	37	30	3.4
Males	32	39	28	.182
To drive to or from a job				
Females	13	35	52	8.0
Males	16	35	49	.019
To go to sporting activities				
Females	31	37	32	75.0
Males	19	42	40	<.001
Your parents want you to				
Females	39	40	21	11.8
Males	34	43	23	.003
To help parents				
Females	18	44	39	6.3
Males	17	49	35	.043
To go out with mates				
Females	21	46	30	9.3
Males	17	48	29	.010
Your friends have licences				
Females	53	31	15	30.5
Males	44	37	19	<.001

*% totals may not equal 100 due to rounding.

Residential location (urban vs. rural): Table 5 shows the results when the importance of the reasons for getting a car driver's licence was examined by the place of

residence (urban or rural) of the learner driver. There were no significant differences for any of the reasons, except "To go to sporting activities" which was *very important* for 39% of the rural drivers compared with 35% of the urban drivers, and *not at all important* for 26% of urban versus 19% of rural drivers.

Table 4. Importance of reasons for getting car driver's licence, by age of learner driver (%*)

Reasons	Not at all important %	Important %	Very important %	Chi-squared p value df=6
To use for identification(ID)				
15 years	34	49	17	367.2
16 years	29	43	28	<.001
17 years	18	40	41	
18^+ years	15	35	50	
Freedom to go where you wish				
15 years	10	41	49	
16 years	14	39	48	18.5
17 years	14	37	49	.005
18^+ years	11	35	54	
To drive to school				
15 years	26	44	30	
16 years	32	36	32	112.9
17 years	37	35	28	<.001
18^+ years	45	27	28	
To drive to or from a job				
15 years	19	38	42	
16 years	13	36	51	163.7
17 years	12	32	56	<.001
18^+ years	6	28	66	
To go to sporting activities				
15 years	21	39	39	38.1
16 years	27	41	32	<.001
17 years	29	40	32	
18^+ years	30	38	32	
Your parents want you to				
15 years	37	45	17	
16 years	28	43	29	100.4
17 years	31	39	30	<.001
18^+ years	45	32	23	
To help parents				
15 years	18	50	33	43.6
16 years	16	45	38	<.001
17 years	14	44	41	
18^+ years	18	38	44	
To go out with mates				
15 years	17	51	32	79.7
16 years	17	51	32	<.001
17 years	19	47	34	
18^+ years	31	45	24	
Friends have licences				
15 years	51	36	13	81.2

16 years	41	38	21	<.001
17 years	42	35	23	
18^{+} years	55	25	20	

*% totals may not equal 100 due to rounding.

Table 5. Importance of reasons for getting car driver's licence, by residential location, (urban or rural) of the learner driver*

Variables	Not at all important %	Important %	Very important %	Chi-squared p-value df=2
To use for identification(ID)				
Urban (n=3557)	27	44	29	0.7
Rural (n=435)	26	46	29	.703
Freedom to go where you wish				
Urban	11	39	50	0.06
Rural	11	39	50	.972
To drive to school				
Urban	33	38	30	0.3
Rural	32	39	29	.880
To drive to or from a job				
Urban	15	35	50	0.4
Rural	15	34	52	.818
To go to sporting activities				
Urban	26	39	35	8.0
Rural	19	41	39	.019
Your parents want you to				
Urban	37	41	22	1.7
Rural	35	41	25	.436
To help parents				
Urban	17	45	37	1.8
Rural	16	49	36	.406
To go out with mates				
Urban	20	50	31	4.3
Rural	24	45	31	.118
Your friends have licences				
Urban	49	34	17	1.6
Rural	52	33	15	.448

*% totals may not equal 100 due to rounding.

The free text responses to the first of the semi-structured questions showed that, of the 65% of females and 61% males who reported that getting their licence was *very important* to them, the main reason was classified as related to "Independence, freedom, mobility" (64% females, 60% males).

No other single reason was reported by more than 10%. When examined by age of the learner driver, "Independence, freedom, mobility" was reported by a higher proportion of younger drivers (69% of 15 year olds, 61% of 16 year olds, 49% of 17 year olds, and 45% of 18^{+} year olds) and also by a higher proportion of rural (65%) than urban (59%) drivers. No other single reason was reported by more than 10%.

The most common responses to the second semi-structured question, "Why are you getting your licence now?"were "Independence, freedom, mobility" (42% females and 42% males), and "ready to/old enough/because I can" (20% females, 21% males).

When examined by age, a higher proportion of younger drivers gave "Independence, freedom, mobility" as their main reason for getting their licence now (43% of 15-17 year olds, 35% of the 18^{+} year olds) followed by "ready to/old enough/because I can" (23% of 15-17 year olds, 11% of 18^{+} age group). There was little difference between the reasons given by the urban and rural drivers with "Independence, freedom, mobility" the most common reason (43% rural, 41% urban) followed by "ready to/old enough/because I can" (24% rural, 20% urban).

Discussion

The newly licensed drivers included in this study were recruited face-to-face at driver licensing centres or at learner licence courses, or responded to a postcard at a licensing centre. They are not, therefore, a random sample of the newly licensed driving population, or the youth population, of New Zealand. However, the description of the socio-demographic characteristics of the 3992 participants (see Table 1) shows that they represent a wide cross section of New Zealanders with drivers of all ages (15–69 years) but predominantly young (15–17 year olds), from a wide range of ethnicities, (including a very good representation of Māori and Pacific people), from all levels of the socioeconomic spectrum, and a reasonable representation from rural and urban residential locations.

When compared with all newly licensed drivers in New Zealand over the 2 years of recruitment, the gender distribution of the NZDS cohort was almost identical to that in the total population, but the NZDS cohort has a higher proportion of 15 year olds (49% compared with 41%) and 16 year olds (18% compared with 14%) and fewer aged 20+ years (13% compared with 24%) than the total newly licensed population. The results presented here, therefore, should represent the views of a substantial proportion of the newly licensed drivers in New Zealand, although the views of younger drivers will be over-represented.

This study focused on two key car driver licensing issues: the minimum age to commence car driver licensing, and the reasons for getting a licence. These two issues were chosen because of their relevance to the current discussion on driver licensing policy in New Zealand, where there is a bill before the House to raise the minimum driver licensing age from 15 to 16 years.

In the past, raising the minimum licensing age has been a contentious subject, often eliciting very polarised views and opinions. As reported previously[10] many of these views and opinions have been supported by little or no scientific evidence, so often the "loudest voice" has predominated. The study presented here was designed to help fill this gap so that future driver licensing policy in New Zealand can be based on sound scientific evidence.

The results from this study showed that just over half of the cohort of newly licensed drivers favoured 15 years as the minimum age to start getting a licence, which is not

surprising given that nearly half of the cohort were 15 years old when they themselves got their learner licence.

As predicted, however, the older the learner driver the more likely they were to support an older starting age. Also, as predicted, a younger licence age was more likely to be supported by males than the females, and by those who resided in a rural versus urban location. When considered together in a multivariate analysis, these three key factors (age, gender, and residential location) were all independently significantly associated with a minimum licensing age of 15 years or older, in the predicted direction.

Perhaps the most surprising results was the level of support for a minimum licence age older than 15 years, from especially the urban drivers, but also the rural drivers (around 25%). This suggests that, as in the past, the strongest opposition to raising the minimum licence age is likely to be from the rural sector, but, overall the level of opposition from young people may not be as high as commonly believed.

The arguments against raising the licensing age are often related to the need for essential travel for young people such as to be able to drive to work, school, sport or recreational activities. To determine if this was the case for the newly licensed drivers in this study we included a range of questions that gave the participants the opportunity to convey as many reasons as they wished, as to why they were getting a licence.

Across all of the questions, and especially those with open-ended response options, the most commonly reported reason for getting a licence was for the independence and freedom to go where they wanted, when they wanted. This applied to both males and females, rural and urban, and for the younger (15 year old) learner drivers. For the older learner drivers (16 years or older), independence and freedom was also an important reason but an even more important reason, and especially for those aged 18 or older, was being able to drive to or from work.

Given that 53% of those aged 18 or older were in paid employment this is not at all surprising. What was perhaps surprising was the number of 15 year olds who reported being able to drive to work was important or very important for them, as only 19% were in paid employment at the time of obtaining their learner licence. Perhaps they were anticipating a future need, when they did have a paid job. To drive to sporting activities, school, or to help parents, was considered very important by a relatively small proportion of drivers, both rural and urban Also, in response to the open-ended questions about why it was important to be getting their licence, and why they were getting their licence now, these reasons were seldom given. The findings presented here, therefore, do not provide strong support for this argument that is often put forward in opposition to raising the minimum licence age.

Conclusion

Contrary to what many may believe, the evidence presented here shows there was not universal opposition by young people to raising the driver licensing age. Furthermore, those in rural areas have much in common with their urban counterparts in respect to their reasons for obtaining a licence. It is also worth noting that travel for work was of most relevance to those aged 18 years or older. Overall, these findings suggest that

increasing the minimum age for licensing may have a higher impact on non-essential rather than essential travel among young people in New Zealand.

Competing interests: None known.

Author information: Dorothy Begg, Senior Research Fellow, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin; Rebecca Brookland, Assistant Research Fellow, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin; Anna McDowell, Assistant Research Fellow, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin; John Langley, Professor, Injury Prevention Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin; Shanthi Ameratunga, Associate Professor, Section of Epidemiology & Biostatistics, School of Population Health, University of Auckland; John Broughton, Associate Professor and Director, Ngai Tahu Māori Health Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin

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Correspondence: Dr Dorothy Begg, Senior Research Fellow, Injury Prevention Research Unit, Dunedin School of Medicine, University of Otago, PO Box 913, Dunedin, New Zealand. Fax: +64 (0)3 4798337; email: Dorothy.Begg@IPRU.otago.ac.nz

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"Pigs might fly"—a case of *Erysipelothrix* endocarditis

Michael Addidle, Kate Grimwade, Stuart Tie, Hina Rahman, Richard Sorenson

Erysipelothrix rhusiopathiae is a well recognised zoonotic pathogen affecting particular at risk groups.

We present a case of *Erysipelothrix* endocarditis in a middle-aged female who lived on a farm in New Zealand and was receiving treatment for chronic hepatitis C infection. *Erysipelothrix* infection should always be considered in the differential diagnosis of patients that have had close contact with pigs.

Case report

A 42-year-old female presented to Outpatients with a several-month history of weight loss, anorexia, intermittent fevers, migratory joint pains, and nausea. At the time of presentation she was on pegylated interferon and ribavirin for chronic hepatitis C infection. She had been on this treatment for the previous 48 weeks. The only other medical history of note was of hypothyroidism. She lived on a farm which had pigs, deer, and horses on site.

On examination she was mildly cachectic and was tattooed. She was afebrile. A soft systolic murmur was heard at the apex which had not been noticed before. She had mild tenderness in the left upper quadrant. Skin abrasions were present on her hands. Examination was otherwise unremarkable and in particular there was no skin rash to suggest erysipeloid and no peripheral stigmata of chronic liver disease or endocarditis.

Her initial blood tests revealed an elevated ESR of 67. There was also a decreased neutrophil count of 1.6×10^6/ml. Looking back over the patient's blood tests whilst on therapy for hepatitis C, her neutrophil counts were consistently in the range 0.9–1.1×10^6/ml.

A CT scan performed to investigate the LUQ tenderness revealed moderate splenomegaly with multiple areas of low attenuation suggestive of infarcts.

Despite the cessation of pegylated interferon therapy her clinical condition worsened with increased fatigue and persistent fevers and she was admitted to hospital. Two sets of blood cultures taken on admission were positive after 48 hours incubation, with *Erysipelothrix rhusiopathiae* isolated from all four bottles. The penicillin MIC was 0.032mg/L.

A trans-thoracic echocardiogram revealed mild mitral valve regurgitation and no vegetations. However in view of the positive blood cultures, the patient had a transoesophageal echocardiogram which demonstrated non-mobile vegetations on both the anterior and posterior mitral valve leaflets. The aortic valve was normal.

She was empirically treated with IV ceftriaxone for 48 hours which was then changed to IV benzylpenicillin in light of the blood culture and susceptibility results. She was discharged after 7 days and was commenced on a continuous intravenous penicillin infusion through the Hospital in the Home service. This was continued to complete 8

weeks of intravenous therapy, before she was switched to oral amoxycillin for a further 3 months.

Repeat echocardiogram three months after initial presentation showed the vegetations had resolved and there was no worsening of the mild mitral regurgitation. The patient remained well at one year after the initial infection with no evidence of compromised cardiac function.

Discussion

Erysipelothrix bacteria comprise two species, *Erysipelothrix rhusiopathiae* and *Erysipelothrix tonsillarum*. The latter is thought to be non-pathogenic. *E. rhusiopathiae* is widespread in nature—and mammals, birds, and fish can all act as reservoirs. Disease is most frequently associated with pigs. It is a well recognised zoonotic pathogen most often seen in farmers, vets, butchers, abbatoir workers, and fishermen,[1] as well as more unusual occupations such as parrot breeding.[2]

The bacterium gains entry to the body via skin abrasions or through ingestion. *Erysipelothrix* disease has also been reported in patients without obvious contact with animals.[3]

Erysipelothrix rhusiopathiae is a facultatively anaerobic Gram-positive rod. Grey, alpha haemolytic colonies are produced on blood agar after 48–72 hrs incubation. A characteristic feature is its ability to produce H_2S on TSI agar[4].

Fig 1. Gram stain showing the pleomorphic Gram variable, slightly curved rods of *E. rhusiopathiae*

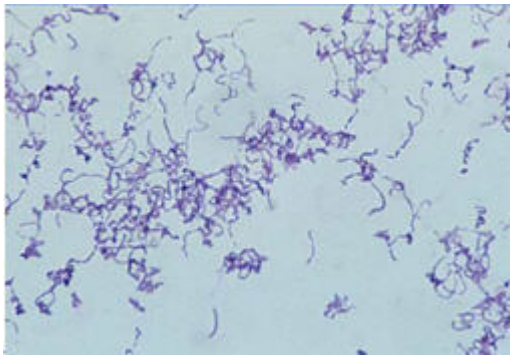
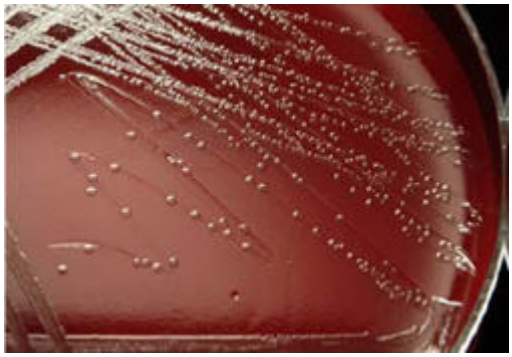


Fig 2. Culture of *E. rhusiopathiae* showing weakly alpha-haemolytic colonies on blood agar



Photographs reproduced courtesy of the RCPA Quality Assessment Programme.

In humans *E. rhusiopathiae* essentially causes two distinct clinical entities:

- Erysipeloid, a localised cellulitis around the inoculation site. Diagnosis may be confirmed by skin biopsy.
- Disseminated disease, usually in immunocompromised patients and often resulting in endocarditis. This is less common than localised disease. Blood cultures readily support the growth of *Erysipelothrix* organisms.

As far as we are aware there have been no previous cases of *Erysipelothrix* endocarditis occurring in New Zealand that have been documented in the medical literature. Most cases involve native valves, and may affect previously normal heart valves in up to two-thirds of cases.[5]

The aortic valve is the most commonly affected by *Erysipelothrix*,[5] however in this particular case the mitral valve was affected. *Erysipelothrix* endocarditis requires valve replacement in approximately a third of patients[5] and the overall mortality is up to 38%.[6] However these figures may be subject to publication bias.

We believe this is the first case report documenting the successful use of a continuous penicillin infusion for the treatment of *Erysipelothrix* endocarditis. Intravenous penicillin is the treatment of choice for systemic *Erysipelothrix* infection.[7] It is worthwhile noting that *E. rhusiopathiae* is intrinsically resistant to vancomycin, which is unusual amongst Gram-positive organisms.

Treatment is usually continued for 6 weeks. However in our patient it was decided a longer treatment course was necessary. This was primarily because her inflammatory markers were slow to resolve but also because of the patient's remote residential location and consequent difficulty with close clinical monitoring.

The pigs on the patient's farm may have been the source of the bacterium. However on further questioning around exposure after the diagnosis was made it was found that the patient regularly hunts wild pigs with her husband, which they then butcher themselves. The patient had been on a hunting expedition approximately a month before the onset of symptoms. This offers a second and possibly more likely route of exposure than the pigs on the patient's farm.

The patient was on pegylated interferon and ribavirin and had mild neutropenia at the time of presentation with her initial symptoms. There is evidence both supporting[8] and opposing[9,10] the statement that combination therapy with pegylated interferon and ribavirin can predispose to bacterial infection.

This case report adds supporting evidence to the postulate that there is some sort of a link between combination treatment for hepatitis C and bacterial infection. However to prove such causality would be very difficult. We recommend however that bacterial infection should always be considered in the differential diagnosis of symptomatic patients that are receiving combination treatment for hepatitis C.

Author information: Michael Addidle, Clinical Microbiologist, Pathlab Bay of Plenty, Tauranga; Kate Grimwade, Infectious Diseases Physician, Tauranga Hospital, Tauranga; Stuart Tie, Cardiologist, Tauranga Hospital, Tauranga; Hina Rahman, Long Island Jewish Medical Center, New York, USA; Richard Sorenson, General Physician, Wind River Internal Medicine, Riverton, Wyoming, USA

Correspondence: Michael Addidle, Clinical Microbiologist, Pathlab Bay of Plenty, PO Box 130, Tauranga, New Zealand. Email: Michaela@pathlab.co.nz

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Get a handle on HaNDL (headache, neurological deficit, and lymphocytosis)

Harriet Cheng, Judy Huang, Joey Yeoh

Headache is a common medical presentation. However, severe headache and transient focal neurological deficit in the presence of normal imaging represents an important diagnostic challenge definable only upon cerebrospinal fluid (CSF) analysis.

We describe a case of headache, transient focal neurological deficit, and cerebrospinal fluid (CSF) lymphocytosis consistent with the headache, neurological deficit and lymphocytosis (HaNDL) syndrome.

Case report

A 20-year-old Caucasian male presented with a 3-hour history of expressive dysphasia and right hemiparesis. This was associated with a severe frontal headache of sharp quality, nausea, and vomiting. There were no symptoms consistent with meningoencephalitis. No significant medical or family history was noted. He was on no regular medication and had no pertinent social history.

Initial physical examination revealed right upper limb weakness of grade 3/5 with significant expressive dysphasia. No other focal neurological signs were elicited. The neurological signs resolved within 20 minutes. Other organ system examination was unremarkable.

Laboratory results (including inflammatory markers) were unremarkable. His chest radiograph was normal. A non-contrast computed tomography (CT) excluded intracranial bleeding and raised intracranial pressure while gadolinium-enhanced magnetic resonance imaging (MRI) excluded parenchymal inflammation, oedema, infarct, and mass lesion. A lumbar puncture was subsequently performed.

The CSF showed 970 red blood cells (number reducing from tubes 1 to 3) and 150 white blood cells (99% lymphocytes and 1% monocytes). Protein was 0.57g/L (0.15–0.45) and glucose was 3.8 mmol/L (2.8–4.4). Gram stain and culture were negative. Polymerase chain reaction (PCR) for *Herpes simplex virus*, *Varicella zoster* virus, and *Enterovirus* were negative.

There has been no recurrence of neurological symptoms or headache 12 weeks after the initial presentation. A diagnosis of HaNDL syndrome was made based on the presenting symptoms and signs, CSF lymphocytosis, and normal brain imaging.

Discussion

A number of case reports and series have described HaNDL syndrome as a headache syndrome associated with temporary neurological deficit (lasting from hours up to 2–3 days). Associated features of HaNDL include fever, elevated CSF protein, and an abnormal, non-epileptiform electroencephalography (EEG).

Brain imaging is usually normal, although some case reports have shown transient focal decreased radionuclide uptake in single photon emission computed tomography (SPECT).[1] HaNDL effects young to middle-aged adults with a slight male predominance.[2–4]

HaNDL is generally a transient syndrome although recurrence may occur in up to 75% of cases. Recurrence has a close temporal relationship to the original event and may present with varying neurological deficits. Recurrence is unlikely to occur after 12 weeks.[5] The aetiology of HaNDL is unknown but it is not thought to be related to any personal or family history of migraine.[6]

The most widely accepted theory is of an inflammatory or infectious origin such as a viral infection. This is supported by the recognition of concurrent viral illness in many patients with HaNDL and the monophasic nature of the disease (events are clustered within a 10–12 week period and then resolve).

HaNDL syndrome is frequently underdiagnosed and attributed to migraine. This case report highlights the importance of CSF examination in young patients presenting with headache and focal neurological deficit. Diagnosis of HaNDL enables the clinician to reassure the patient that the disease is self limiting and that full functional recovery will occur without the need for any specific therapy.

Author Information: Harriet Cheng, Registrar; Judy Huang, Registrar; Joey Yeoh, Registrar; Department of Medicine, Middlemore Hospital, Auckland

Correspondence: Dr Judy Huang, Department of Medicine, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland 1640, New Zealand. Email: ju2c@hotmail.com

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Calcified facial haematoma

Patrick Mehanna, Anthony Bertram, Ian Wilson

A 45-year-old male presented to Accident and Emergency in August 2008 after being kicked in the face by a horse. The initial CT showed no bony abnormality but the presence of a large left masseteric haematoma. He was discharged without follow-up.

Due to trismus of 18 mm and ongoing pain he was referred to the Maxillofacial Surgery Department in November 2008. A repeat CT demonstrated a large calcified mass in continuity with the left mandible (Figure 1 and Figure 2).

The trismus has been improving with conservative management with mouth opening now approximately 30 mm. Although large calcified haematomas are rare in the maxillofacial region, the case stresses the importance of followup in significant soft tissue trauma.

Figure 1. Axial CT Scan showing calcified mass arising from ramus of left mandible

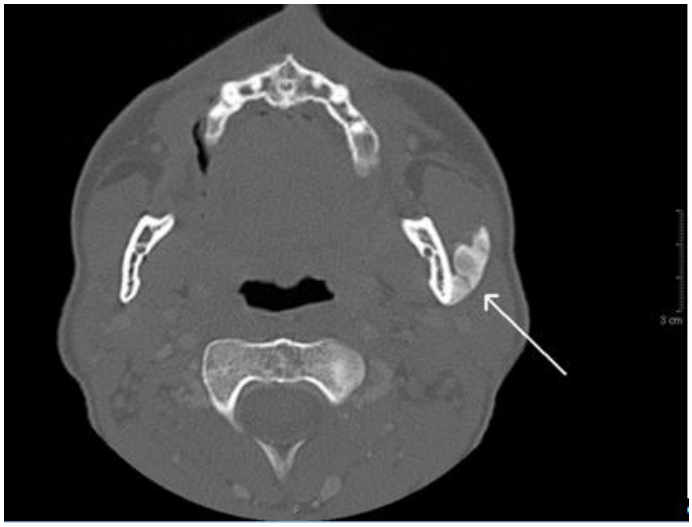
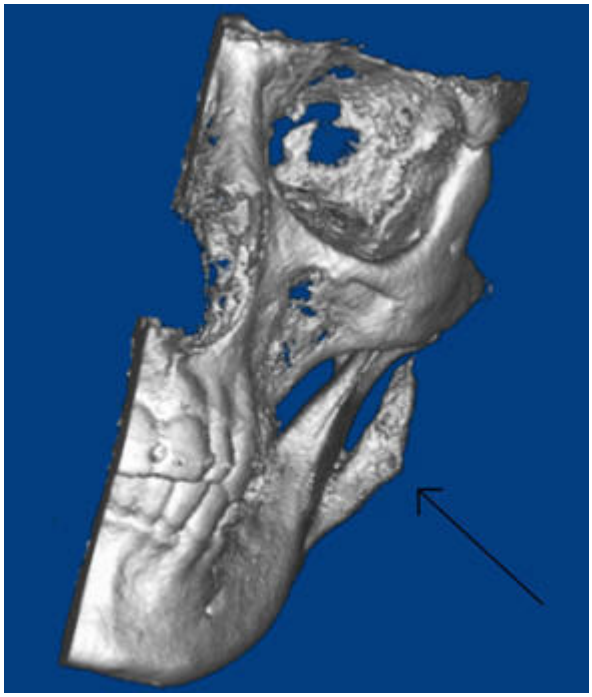


Figure 2. 3D CT reconstruction demonstrating calcified mass arising from ramus of left mandible.



Author information: Patrick Mehanna, Fellow in Maxillofacial Surgery; Anthony Bertram, Registrar in Oral and Maxillofacial Surgery; Ian Wilson, Consultant in Oral and Maxillofacial Surgery; John Hunter Hospital, Newcastle, NSW, Australia

Correspondence: Dr Patrick Mehanna, Fellow in Maxillofacial Surgery, John Hunter Hospital, Lookout Road, New Lambton, Newcastle, NSW 2305, Australia. Email: drpmehanna@hotmail.com

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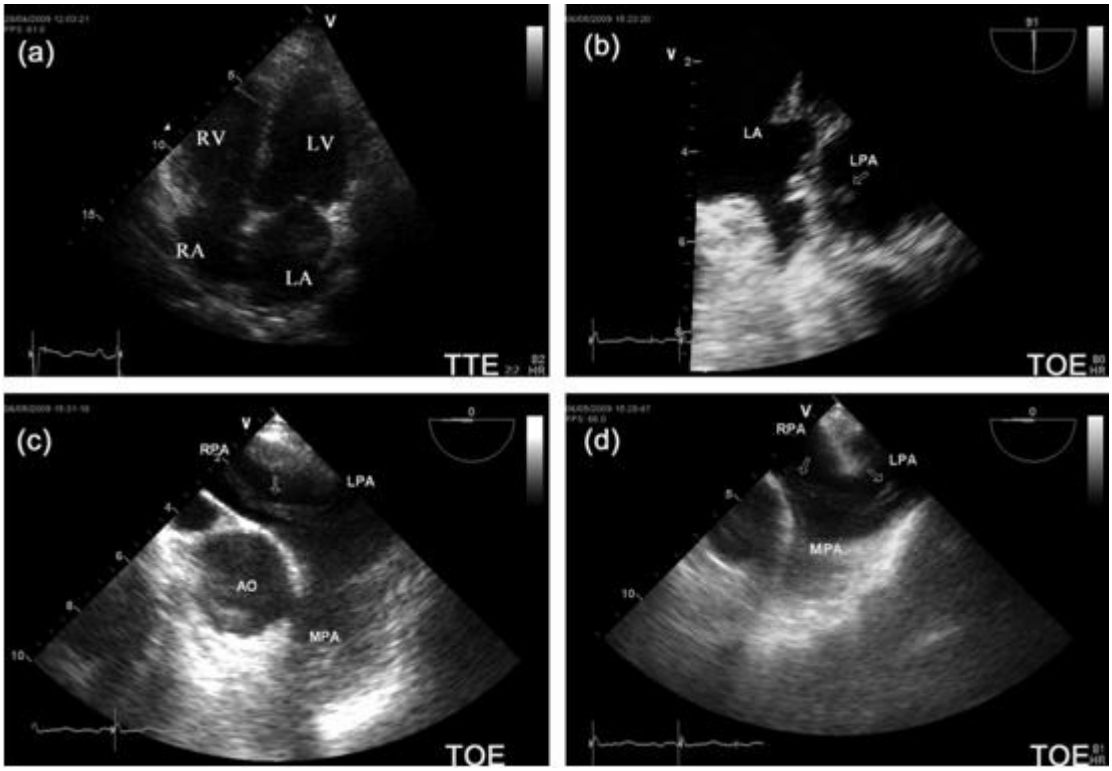
An unexpected embolus revealed in transoesophageal echocardiogram

Michael Liang, Sumi Munthree, Raewyn Fisher, Gerard Devlin

Clinical

A 59-year-old lady with 1-week history of dyspnoea presented with acute onset of dysarthria and dense right-sided hemiparesis. Diagnosis of left middle cerebral artery infarction was confirmed by brain magnetic resonance imaging. No cardiac source of emboli was seen on transthoracic echocardiogram (TTE). However, mildly dilated right ventricle was noted (Figure 1a) and, unexpectedly, transoesophageal echocardiogram (TOE) revealed thrombus in the main, right and left pulmonary artery (Figure 1b, c , d).

Figure 1(a). TTE of this patient showing mildly dilated right ventricle; (b) TOE of this patient focusing at left atrial appendage showed a hyperechogenic lesion in left pulmonary artery (arrow); (c) & (d) The main, right and left pulmonary arteries were examined by TOE and a large pulmonary embolus (arrow) was clearly demonstrated



RV = right ventricle, LV = left ventricle, RA = right atrium, LA = left atrium, Ao = ascending aorta, MPA = main pulmonary artery, RPA = right pulmonary artery, LPA = left pulmonary artery.

Subsequent lower limb ultrasound showed a likely source of emboli from a partially occlusive thrombus in the right popliteal vein. This patient was found to have antiphospholipid syndrome and required anti-coagulation therapy.

Discussion

Acute pulmonary embolism (PE) is a common and potentially fatal disease. The recommended approach involves suspicious history or signs of PE, confirmed by helical computed tomographic scanning with intravenous contrast.[1] TTE is not routinely recommended as the imaging modality of choice because abnormalities are seen only when the PE is large.[2,3] Although TOE is also not the investigation of choice, it may allow direct visualisation of a thrombus in pulmonary artery and thus assess the extent and surgical accessibility of the thromboembolism.

The procedure can be easily performed in a unconscious patient, while conscious patients almost always require sedation. In most cases, the main pulmonary artery can be visualised first and then the right pulmonary artery. The left pulmonary artery can be examined by rotating the transducer from the main pulmonary artery, however it could be limited due to interposition of the air in the left main bronchus. Nevertheless, large pulmonary saddle emboli are easily seen in most cases.

Author information: Michael Liang, Senior Registrar in Cardiology, Department of Cardiology; Sumi Munthree, Senior Registrar in General Medicine, Department of General Medicine; Raewyn Fisher, Cardiologist, Department of Cardiology; Gerard Devlin, Interventional Cardiologist, Clinical Director of Cardiology; Waikato Hospital, Hamilton

Correspondence: Dr Michael Liang, Department of Cardiology, Waikato Hospital. Pembroke & Selwyn Sts, Private Bag 3200, Hamilton 3240, New Zealand. Email: LiangM@waikatodhb.govt.nz

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Medical student selection in New Zealand: looking to the future

Phillippa J Poole, Helen J Moriarty, Andy M Wearn, Tim J Wilkinson, Jennifer M Weller

Abstract

Aims To review whether current New Zealand (NZ) medical student selection policies are likely to result in specialists prepared to work in areas of greatest health need in the future.

Method This paper describes approaches used to select medical students, with some details about NZ medical student cohorts. It then discusses the evidence linking selection and career choice.

Results and Conclusions Selection processes have to serve multiple purposes and no tools are ideal. The NZ medical student population is more diverse than previously with more females than males, and higher proportions of students who are Māori, Pacific, rural, Asian or born overseas. Tracking projects are already underway to obtain data to better understand the effect of student factors on career choice. The Māori and Pacific Admission Scheme and Rural Origin Medical Preferential Entry affirmative action pathways have been successful, but to increase the number of doctors who identify as Māori or Pacific will require a larger pool of students with strong educational backgrounds from which to select. The strongest evidence between selection and future practice exists for students from rural backgrounds – they are more likely to practice in rural areas and to enter general practice. Therefore, increasing the numbers or rural students, or broadening the definition of 'rural', should be considered.

New Zealand (NZ) medical students starting their programme in 2010 will enter the specialist workforce from 2022 onwards. Healthcare delivery will be very different then if NZ is to meet the challenge of providing quality healthcare to an aging population in the face of static financial and human resources.[1]

Having a strong primary care sector, and an increased proportion of 'generalist' specialists who work over traditional primary, secondary and tertiary boundaries may provide the best strategy to maintain quality and keep costs down.[2,3] For example, a recent Ministry of Health review of how best to configure agencies in the mental health and addictions sector concluded there is a need for more doctors working in both psychiatry and general practice.[4]

After years of piecemeal growth, medical student numbers in NZ are about to increase significantly.[5] A fundamental step in shaping the future workforce is the selection of medical students, making this an opportune time to review how future doctors are chosen.

This paper describes approaches used to select medical students, with some details about the current NZ medical student cohorts. It then outlines evidence between selection and career choice for the types of doctors NZ will need in the future.

Selection tools and their rationale

The ideal medical student selection tool would predict future performance in medical school and beyond, so that those selected progress through a rigorous programme and graduate as effective medical practitioners. Additionally, this tool would address issues of equity and diversity, 'screen in' those with desirable qualities that can't be taught, and 'screen out' those with unfavourable traits for medicine.

Finally, an ideal selection tool would select those attributes that are desirable in a doctor but which cannot be learnt in medical school. As there is no such tool, medical schools use a variety of approaches. These range from nihilism (relying on lottery alone), to measuring the easily measurable (such as grades or aptitude scores), to best guesses (where selection is based on what seems to have high face validity).

Evaluating the effect of these different approaches is problematic, due mainly to restriction of range where those with the lowest scores are not admitted and are thereby excluded from further analysis.

Although academic grades are the single best predictor of academic success in the early years of medical school,[6] the correlation is only moderate except in the case of very high grades and very low grades. Academic grades are less predictive of assessment measures after graduation.[7]

A reliance solely on grades for selection to medical school preferentially rewards those with prior educational advantage and ignores issues of social equity, fitness for the task, community expectations and the desirability of a diverse medical workforce.[8,9]

To address some of the aforementioned areas, tests of non-cognitive attributes are used in medical student selection.[10] However, personal references and statements have been shown to be of no predictive value.[11,12] Traditional interviews have low reliability characterised by low agreement between raters, interview bias based on non-relevant candidate attributes and potential for candidates to adopt socially desirable stances in response to questions.

More structured interviews show higher reliability,[13] and seeking examples of past behaviour ("tell me a time when...") may produce more honest responses than asking candidates what they would do if faced with a particular scenario.[14] A recent development is the Multiple Mini Interview (MMI)[15] during which candidates pass through a range of stations, each testing for specific attributes considered desirable in doctors. Stations may include interviews, standardised patient stations or video clips. The principal advantage of an MMI is the ability to sample across more areas of interest (increasing validity) and to aggregate the scores of a number of assessors (increasing reliability).

Other advantages include fewer problems with security violations,[16] being less expensive to run than traditional interviews,[17] and less subject to influence by coaching.[18] Disadvantages include the time and expense to develop the MMI process.

The attributes to be tested will depend on local circumstances and require stakeholder consultation. There is emerging evidence supporting reliability, validity and potential to predict future performance of the MMI process,[10, 19, 20] but as the MMI is a process and not a test in itself, the validity and reliability are dependent on how the MMI is constructed in each individual location.

Aptitude tests such as the Undergraduate Medical Admissions Test or UMAT (©ACER, Melbourne) are increasingly used in Australasia and beyond. The UMAT requires no previous scientific knowledge and is designed to measure logical reasoning and problem solving, interaction skills and non-verbal reasoning. There is some evidence supporting the ability of these tests to predict future performance in medical school, but as only those with high scores gain entry, it is difficult to investigate the predictive validity of these instruments.[18]

Commercial coaching courses for UMAT purport to influence scores but the only evidence is of a very small effect on non-verbal reasoning tasks.[18] The personal qualities assessment procedure (PQA) is a portfolio of psychometric tests that may offer an advance in predicting performance in medical school and professional progress.[21]

The use of the scores on these tests varies among schools. Some schools use all of them in ranking; others set thresholds for certain tests, with final selection decisions based on other data. Lotteries are appealing to some, and may be weighted towards region of origin or demographic characteristics. They may, however, perpetuate cognitive selection bias as the initial cut is still based on examination results.[22]

Another problem is that students selected using a lottery system are more likely to drop out of their medical programme than those selected using a combination of academic and non-cognitive tools.[22]

Faced with the paucity of robust data and the 'high stakes' consequences of declining a candidate a place in medical school, it is little wonder this area is controversial. Selection tools are usually combined by schools based on their own assessments of the fairest way to select the most appropriate population of medical students.

Student numbers and selection practices

Medical students in NZ are selected after one year at university, or following completion of a degree. Those who are not graduates must undertake a health sciences first year at the same university to which they intend to apply for medicine; this is then credited to their six year programme. In recent years, there were 190 domestic medical student places available in Year 2 at the University of Otago and 135 at the University of Auckland, with another 20 domestic places allocated to each school for 2009.

There will be an additional 60 medical student places available nationally in 2010 with more likely over the next few years.[23]

At Auckland, all applicants are ranked using a combination of Grade Point Average (GPA), and scores from the UMAT. To be invited to the next stage which is a semi-structured interview, the GPA across the eight courses undertaken must be a B+ or higher. With the exception of a small number of students included or excluded

directly as a result of interview performance, the GPA, interview and UMAT are weighted 60:25:15 respectively towards the final ranking for selection.

For entry to Otago, ranking decisions are based upon GPA and UMAT in a 66:34 weighting, provided the designated academic and UMAT thresholds are met. In contrast to Auckland, no interview is undertaken, and graduate applicants do not need to sit UMAT. In both NZ schools there are over three eligible applicants for every one place offered. As such, decisions may be based on very small differences in scores, and many who would otherwise be fine doctors are declined entry.

The attrition rates within both schools are low, suggesting that either method is satisfactory in screening out people with low potential for completion of the programme. However, the relative merits of the two methods in predicting students who may require additional assistance during the course is less clear.

Diversification of the medical student population through affirmative action

Māori and Pacific Admission Scheme (MAPAS)—Until about 20 years ago, the predominant medical student characteristics were being white, male, coming from a higher socio-economic group, and having university-educated parents, including one in eight with a parent in medicine.[24,25,26]

Until recently, medical schools have tended to focus on preparing students to function as PGY1 interns and for any branch of speciality training. Internationally there have been calls for medical schools to provide more evidence of their impact on the public good[27]. One aspect is the expectation that the population of doctors reflects the social and ethnic diversity of the community it serves.[28] This expectation is underpinned by two main principles. The first is based on social justice and equity of access for minority groups; the second, because a diversified student population may be more disposed towards addressing priority areas of need.[29,30]

A study of the practice registers of black and Hispanic doctors in California, for example, found that doctors from these minority groups were more likely to take care of patients from their own ethnic groups as well as uninsured and Medicaid patients.[31]

For many years each school has had a MAPAS affirmative action pathway to provide equitable access for students who are Māori or Pacific with potential to undertake medical training. The majority of Māori and Pacific medical students enter via this pathway and to graduate, they must meet the same educational standards as other students.

It should be noted that a MAPAS pathway is far more than a selection process – there are specific recruiting and student support initiatives. The MMI is being piloted for MAPAS admissions at Auckland with one driver being to better understand individual academic and pastoral needs at entry.

Of the 155 domestic places available at Auckland, up to 30 may be offered to MAPAS students, however Otago does not have a fixed quota. Currently 3% of doctors identify as Māori, and 1.8% as Pacific[32] compared with the population percentages of 15% and 7% respectively.[33] While there are encouraging increases in

the numbers of Māori and Pacific medical students (see Table 1), the levels are nowhere near high enough to redress the shortages of Māori and Pacific doctors.

Table 1. Self-identified ethnicity MBChB domestic students, compared with NZ population at 2006 census

Ethnicity	Auckland 2008 All MBChB, n = 703	Otago 2009 All MBChB, n = 1391	New Zealand population overall	NZ ages 15-39 years
Māori	9.8%	5.0%	15%	17%
Pacific Islands	7.1%	2.7%	7%	13%
Asian (including Indian)	34.6%	33.3%	10%	8%
European and other	48.5%	59%	77%	71%

Rurality—Following a government initiative, the Rural Origin Medical Preferential Entry (ROMPE) pathway was established in 2004. Since then each school has admitted 20 students per year who meet the following criteria:

- Undertaken a significant proportion of their pre-secondary education while living in a New Zealand rural area; or
- Spent at least three years at a secondary school in a New Zealand rural area; or
- Have equivalent New Zealand rural experience

Note: a rural area includes those towns in New Zealand with a population or 20,000 or fewer.

It has been shown repeatedly that a rural background is associated with increased likelihood of practice in a rural setting, although most rural doctors have not grown up in rural areas.[34,35] A systematic review based mainly on US data found that characteristics at admission were more likely than curriculum experiences to result in doctors working in rural settings. The number needed to 'teach' in a rural immersion curriculum to result in one extra rural practitioner was 17, compared with six as the number needed to be 'admitted' under revised admission criteria.[35]

A recent study of the intentions of 4112 Australian medical students at entry to medical school confirmed rural practice intentions were more likely in students from rural areas.[36] Other predictors were plans for a generalist career, and being bonded, or in receipt of a scholarship.

A census of all NZ medical students in 2001 found under-representation of those from smaller towns and rural settings compared with the general population.[37] Four years after the introduction the ROMPE pathway, a quarter of all students entering MBChB at Otago fulfilled ROMPE rural entry criteria, and 20% of Auckland Year 2 students reported coming from a provincial centre with 8% from smaller towns. Data from the 2006 and 2007 Auckland graduating classes showed 58% intended to work in a city and 15% in a regional/rural area, with the latter significantly more likely to be Māori and less likely to be Asian.[38]

Nothing is known about which students are more likely to work in outer metropolitan and regional centres, although based on hospital sizes and comparative populations,

some regional centres in NZ would be regarded as 'rural' in Australian and US literature.

Other trends

Feminisation—For about 15 years the proportion of women in medical programmes in NZ, Australia and the UK has been between 50 and 65%.[39] A detailed review of the effect of gender on specialty choice in Britain found women graduates consistently more likely than men to choose general practice, obstetrics and gynaecology, paediatrics and pathology.[40]

Feminisation has implications for the medical workforce given that women work on average around seven fewer hours per week than men.[32] Concerns have also been raised about the status of the profession once the majority of specialists are women.[41] On the other hand, women have already demonstrated a readiness to provide care to underserved populations and to work in teams with a focus on the broader aspects of health.[42]

Graduate students—Up to a quarter of the medical students in the two NZ undergraduate programmes have a prior tertiary degree. Older entrants including graduates are more certain of their career choice and use more desirable learning styles[43]; they also have more diverse backgrounds than school leavers.

Most of the new programmes in Australia are four year graduate programmes and a separate graduate pathway within a medical programme in NZ has been considered.[44] This would shorten the medical school portion of training for graduates to around four years, but does not take into account the time taken for the first degree, or that the medical academic year would need to be lengthened.

Despite the appeal of graduate programmes, increasing the proportion of medical students who are graduates may not result in higher numbers of doctors working in areas of need than the current system for the following reasons:

- Graduates perform at a similar level in medical school and internship to their school-leaver colleagues;[45]
- Even though they are more professionally and altruistically motivated on entry to medical school, graduates as a group enter the same medical careers as other medical students;[46]
- Having to complete a degree prior to selection for medicine may create a significant barrier to Māori and Pacific students, and those from lower socioeconomic groups.

On the other hand:

- Graduates may be more likely to be in a permanent relationship and may therefore be more likely to be committed to work in New Zealand;
- Having undertaken a previous degree may 'even out' inequities related to secondary schooling, thereby advantaging students from less advantaged schools;

- Graduates may bring useful attributes resulting from skills acquired in obtaining their prior degree.

Migration—Around 40% of Auckland's current domestic medical students were born overseas, most commonly in South Africa or Asia (including India and Sri Lanka). While most have NZ citizenship, a significant proportion (11%) has permanent resident status.[39] This contrasts with the situation in Otago where only 9% of the domestic students were born outside NZ.

Why overseas-born students do so well in the multi-facetted selection process at Auckland has not been studied in depth. As the majority of Auckland students come from the greater Auckland area, reasons for the difference may include the rapidly-changing demography of Auckland and the emphasis placed on educational achievement by the immigrant parents of these students.

Priority specialty areas—In 2009 the NZ government offered $30,000 scholarships for medical graduates prepared to work for 2 years in health board areas with shortages, then enter training in one of five discipline areas – general practice, general medicine, general surgery, pathology or psychiatry.[47]

Data from the Auckland 2008 graduating class showed varying levels of interest in these careers (see Table 2). Over 50% of the class had 'some' or 'strong' interest in general practice, general medicine and general surgery. While far fewer were interested in psychiatry and pathology, levels of interest still exceeded the proportion of these specialists in the workforce in that year.

Table 2. Level of Auckland graduate interest in the priority discipline areas compared with current proportions on MCNZ register

Variables	General Practice	General Medicine	General Surgery	Psychiatry	Pathology
Strong Interest	31.6%	27.5%	19.7%	10.7%	2.3%
Some Interest	45.1%	48.9%	34.1%	22.9%	7.6%
No Interest	23.3%	23.7%	46.2%	66.4%	90.1%
Current MCNZ proportion (house officers excluded)[32]	35%	14%*	4%	8%	3%

*Whole of internal medicine, not just general medicine.

General practice—Student factors associated with an increased likelihood of entering general practice include a desire for varied scope of practice,[48] being female and older,[49] and having a rural home town address.[49,50] There is a strong association between wanting to work in general practice and to work in a regional-rural setting.[38] In NZ, the proportion of women entering general practice training (53%)[32] is about the same as the proportion of women medical students.

General medicine—In NZ, general physicians are consultants usually in secondary care. This is in contrast to the situation in the USA where general internists may be involved in primary or secondary care. Data from one USA study found that desiring

an intellectual challenge and having an affinity for the continuity of patient care were determinants of a general medicine career choice.[51]

There was a slight preponderance of males in that study. In NZ, there is no gender difference in preference, and those interested in general medicine did not have any greater interest in general practice. Instead, they were more likely to express interest in a medical subspecialty.[52]

General surgery—Traditionally, surgery has been a male-dominated specialty although this is changing as more women move into surgical careers.[53] Those women who chose surgery, though, are less likely than other women to value flexibility in regard to training and work.[54] As a balanced lifestyle is increasingly important for most doctors regardless of background,[55] for more trainees to choose general surgery will likely require attention to surgical work patterns and incentives, rather than a change in medical student selection per se.

Psychiatry—When it was introduced, a secondary intention of the ROMPE pathway was to increase the numbers of psychiatrists in regional and rural NZ. Evidence is scarce, however, as to what selection methods are most useful in this regard. One older study found that doctors entering psychiatry were less likely to have studied science in their first degree, had wider intellectual interests and better developed social skills than their fellow students.[56]

Since 2004, several students with a background in mental health have been offered preferential entry to Otago, but it is too early to say whether or not they will enter a career in psychiatry. The reason for the relatively high number of Auckland students interested in psychiatry is unknown. Auckland has made no specific changes to select students for a career in psychiatry, however the interview process might allow evaluation of some relevant attributes.

Pathology—Medical students interested in pathology are reportedly not concerned they will have limited patient contact, and may view it as a scholarly and isolated specialty.[57] This specialty has the highest proportion of female trainees in NZ (63%).[32] As there are relatively small numbers of doctors in this specialty and career choices are often made in the early post graduate years, it is difficult to see how selection policy will solve workforce issues in this specialty.

Conclusions

Selecting medical students is a complex and 'high stakes' endeavour, yet the tools available are limited in number and predictive ability. The current selection tools, processes and pathways have been arrived over time through iterative review including input from a range of stakeholders. The only major change to the selection tools in recent years has been the introduction of UMAT which is not known to differentiate among students with different demographic characteristics or career intentions.

A collaborative predictive validity study is now being undertaken by the two schools to study its value. The current selection processes are generally accepted and feasible, although interviews will be more problematic with increasing numbers. On the other hand, having more places on offer will hopefully reduce competition and the need to use tools that are able to separate candidates with similar attributes.

Despite the shortcomings, the selection processes and pathways used in NZ to date have generated a more diverse and representative range of medical students than previously. Women now slightly outnumber men and there are increasing proportions of students who identify as Māori or Pacific, or come from rural backgrounds.

Predictions for the population profile of NZ in 2026 show that 17% of the population will be Māori, 10% Pacific and 16% of Asian descent.[33] As yet the numbers of Māori and Pacific students are insufficient to ensure the medical workforce mirrors current, let alone future population demographics. As there are relatively small numbers of Māori and Pacific peoples with sufficiently strong science education backgrounds for medicine, greater efforts must be directed towards increasing the level of educational attainment at high school and promoting health as a career.

An important area of future study is to look at career patterns and support needs of doctors who entered via MAPAS pathways[58] in order to maximise participation of these valuable practitioners in the health system. Specific pathways for students from lower socioeconomic groups have been introduced in Britain, however there is little evidence yet that this approach will provide better servicing of patients with the greatest health need.[59]

The current over-representation of students of who identify as Asian is multifactorial. As this sector of the population is growing, this overrepresentation will not be as marked in the future and another research priority is to understand better career patterns of Asian students.

While there are slight differences in how data have been collected, it is notable that Otago admits a higher proportion of students from rural backgrounds (25%) while Auckland admits significant numbers of domestic students born overseas (40%). The only major difference in the selection process is that Otago does not have an interview, whereas Auckland does.

These findings support the notion of a contrast in the nature of the applicants between the two schools that may be due to catchment population differences, or differential student preferences for medical school location. In terms of developing a medical workforce that mirrors the community it serves, these differences are positive and offer opportunities for evaluation and research.

NZ data suggest that the correlations among a rural background, likelihood of practice in rural area and a career in general practice, apply in NZ. This would support the case to increase the numbers of ROMPE students significantly in order to increase the rural and regional workforce[20,21] and the number of NZ-educated GPs.[25,26]

As there is not a large pool of applicants who meet the existing definition of ‘rural’, a review might consider whether rural criteria need to be broadened to include smaller regional centres where workforce shortages are also marked. It is encouraging that the majority of medical students at entry are interested in general practice.

As with other medical careers, translating this interest into practice likely requires conducive learning experiences and a relative valuing of general practice compared with other specialties; a discussion that is outside the scope of this paper. It highlights the more general research need to understand better the multiple and interacting factors that affect career choice.

Both schools are mindful of the dilemma created by the long lead time in medical training: there is a need for careful evaluation of any changes in selection policy, yet prompt implementation of changes in response to anticipated workforce needs. Further efforts to quantify the complex interplay among the effects of student characteristics, undergraduate curriculum and early post graduate experience on career choice are already underway.

Since 2006, entering and graduating University of Auckland medical students have been enrolled in a longitudinal investigation of the characteristics, study and career patterns of undergraduate medical, nursing, pharmacy and health science students.

Over the past two years, both medical schools have joined the Australasia-wide Medical Student Outcome Database project that has similar aims.[60] Already the project has been valuable in providing data about students and their intentions, however, the most useful data will come from 2011 onwards once entry and exit data from the same cohort can be analysed, and the original exit cohorts are differentiating by speciality and location of practice.

The main conclusion that may be drawn about medical student selection and the future workforce in NZ is that an immediate increase in the numbers of medical students entering through the ROMPE and MAPAS pathways would have a positive effect on the future workforce. Other links between student characteristics at entry and career remain speculative, and not robust enough to justify major changes in selection approaches.

As new evidence comes to light through the tracking projects, medical schools might be joined by the broader health community in the debate as to how the future medical workforce should be chosen.

Practice points

- Health care delivery in 2020 and beyond will be very different owing to health needs of the ageing population and constrained financial and human resources.
- Medical student selection practice has to predict future performance, rank among many with the capability to succeed, and address issues of equity and diversity. It should select those attributes that are desirable in a doctor but which cannot be learnt in medical school. There is a very long lag time to evaluate the effects of any change in selection policy on workforce.
- The NZ medical student population now has more females than males, and higher proportions of students who are Māori, Pacific, rural, Asian or born overseas.
- The MAPAS and ROMPE affirmative action pathways have been successful, but to increase the doctors who identify as Māori or Pacific will require a larger pool of students with strong educational backgrounds from which to select.
- The strongest evidence between selection and future practice exists for students from rural backgrounds – they are more likely to practice in rural areas and to enter general practice. Thus, increasing the ROMPE numbers or broadening the definition of ‘rural’ should be considered.

Disclaimer: The opinions expressed in this article are those of the authors and not necessarily those of the University of Auckland or University of Otago.

Author information: Phillippa Poole, Head, Medical Education Division, School of Medicine, University of Auckland, Auckland; Helen Moriarty, Senior Lecturer, Dept of Primary Health Care and General Practice, University of Otago, Wellington; Andy Wearn, Director of Clinical Skills Resource Centre, University of Auckland, Auckland; Tim Wilkinson, Associate Dean (Medical Education), University of Otago, Christchurch; Jennifer Weller, Director, Centre for Medical & Health Sciences Education, University of Auckland, Auckland

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Correspondence: Assoc Prof Phillippa Poole, Head, Medical Education Division, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand. Fax: +64 (0)9 3737555; email: p.poole@auckland.ac.nz

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THE NEW ZEALAND MEDICAL JOURNAL
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President's Address (part 3)

Excerpts of a speech delivered at the Annual Meeting of the British Medical Association in New Zealand on February 21st, 1910, by James Purdy M.B.C.M., Hutt. Published in NZMJ 1910;8(33):1–14.

We have in New Zealand excellent provision for the care of the sick in our public hospitals. Here as far as the public concerned (and I admit they are chiefly to be consider if our aim is to be the greatest good for the greatest number), the arrangements are excellent. But what about the medical profession?

On every side they have allowed themselves to be exploited. We even find hospitals which emp1oy a medical man at a good salary with quarters allowing him to compete in private practice with his brother practitioner. Not only does be get a big pull by being the hospital doctor, but he starts in the race without the handicap of having to pay rent and other expenses, which are more than covered by his salary.

Fortunately the Government has recognized this competition between subsidised medical men and general practitioners, this in connection with the recent retrenchments, and we find that now at some of our health resorts, instead of paying the whole salary of the rnedical officer, they now allow him the use of the consulting rooms at the sanatorium and he collects his own fees. We also find that no provision has been made for payment of medical practitioners summoned to attend cases by registered midwives. This question has been settled satisfactorily in the old country. Now quite recently another duty has been put upon medical men, a11 must admit rightly. The wonder is the old system was tolerated so long. No longer can the medical practitioner, in giving a death certificate, use the phrase "as I am informed.

In all cases be must have attended the patient before be can give a certificate of death. It would only be fair to allow a fee for such certificate; the certificate of death ought to be a privileged and confidential document between the doctor and the registrar.

We all know at present for instance what offence would be given to relations by according say alcoholism on a death certificate. Here one might suggest that the registration of births and deaths and even marriages should be a function of the Health Department. Of what use to New Zealand has been for instance the Early Notification of Births bill? In England and other countries it was introduced in order to give an opportunity to health officials to protect infant life.

Health visitors or nurses visited the homes of the people more especially in the congested areas, and since early notification in Great Britain much good has been done. In our own country of what benefit is it for a local authority to learn that someone died from, say, consumption in a house in their area a month previous? If such deaths were notified to the Health Department direct, then the houses could be disinfectant immediately after the removal of the corpse.

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

Proceedings of the Waikato Clinical School Research Seminar, Thursday, 17 September 2009

General practice funding to improve provision of adolescent primary sexual health care in the Waikato: results from an observational intervention

J. Morgan [1], J. Haar [1]

[1]Waikato Hospital, Hamilton, New Zealand

Objective

To determine if introducing free General Practice (GP) sexual health visits for registered adolescents improved provision of primary sexual health care using testing and detection of Chlamydia *trachomatis* as an outcome measure.

Methods

In 2003-4, additional funding enabled 20 practices in Waikato, New Zealand to offer free sexual health consultations for registered under-25 year olds. Practice selection was non-random and biased towards lower socio-economic, Māori and rural populations. Enrolled practice population data was linked to laboratory testing for Chlamydia *trachomatis* from January 2003 to December 2005. Twenty-nine practices not selected for additional funding served as controls.

Results

Chlamydia testing amongst under-25 year olds at the 20 intervention practices increased over time, in contrast to non-intervention practices, with coverage of females aged 18-24 years within the intervention increasing from 13.9% in 2003, to 15.5% during the roll-out phase and to 16.8% in 2005. In addition, there is no increase in testing or detection amongst those aged 25 years and older at intervention practices. Chlamydia test positivity increased at intervention practices from 7.7% in 2003 to 10% in 2005, relating mainly to increases in positive tests amongst females aged under-25 years.

Conclusions

Introducing free GP visits for under-25 year olds living in rural and lower socio-economic areas of the Waikato district was associated with a significant increase in testing and detection for Chlamydia *trachomatis* in the target age group. This observational intervention supports the ongoing provision of free adolescent primary sexual health care.

Inter-observer variability of teledermoscopy

E. Tan [1], M. Rademaker [1], A. Oakley [1]

[1]Department of Dermatology Waikato Hospital, Hamilton, New Zealand

Teledermoscopy is a rapidly developing field of dermatology with numerous studies demonstrating excellent agreement to face-to-face diagnosis. However, there are few studies evaluating the reproducibility of diagnosing skin lesions between teledermoscopists. This evaluation is important not only to establish diagnostic consistency but also to improve comparability among different epidemiologic studies on skin cancers.

Inter-observer variability in diagnosis was assessed amongst 4 independent experienced teledermoscopists in New Zealand, Australia and America for 979 lesions in 206 patients. This cohort consisted primarily of an elderly, Caucasian population with non-melanocytic lesions.

There was excellent agreement between teledermoscopists for pathologically confirmed melanoma with no melanomas missed. The agreement for melanocytic lesions was better than that for non-melanocytic lesions with more variability in benign, non-melanocytic lesions (mean kappa values of agreement for benign naevus was K=0.90 and atypical naevus K=0.80 whereas seborrhoeic keratosis K = 0.70, SCC-in-situ K=0.41, Solar keratosis K= 0.49, BCC K= 0.67 and Inv SCC K=0.58)

There was good ability to distinguish malignant from benign lesions with 3 out of the 4 teledermoscopists showing moderate to substantial agreement for basal cell carcinoma and invasive squamous cell carcinoma.

These variations may be explained by differences in training and by familiarity with the clinical characteristics of the study population, different medico-legal work environments or otherwise.

Abbreviations: BCC – basal cell carcinoma, SCC – squamous cell carcinoma, Inv SCC – Invasive squamous cell carcinoma

Fractional flow reserve and long term outcomes in the real world

A. Puri [1], S. Perera [1], K. Abercrombie [1], G. Devlin [1]

[1]Department of Cardiology, Waikato Hospital, Hamilton, New Zealand

Background: Coronary pressure derived fractional flow reserve (FFR) is an invasive physiological index of the functional severity of coronary artery stenosis. The FAME study clearly identifies FFR $\geq$0.80 as being associated with a good clinical outcome at 12 months in patients continuing with conservative management strategies. Our aim was to assess longer term outcomes of patients with angiographic coronary artery disease (CAD) in whom FFR was $\geq$0.80 and did not undergo percutaneous coronary intervention (PCI).

Method: Patients having symptomatic CAD who underwent coronary angiography between January 2006 and January 2008, and having an FFR $\geq$0.80 which was not intervened with PCI were identified. Clinical Outcomes were assessed, which

included any presence of anginal chest pain, revascularization and death during the follow up period till April 2009.

Results: 61 Consecutive lesions in 51 patients were included in the group. The mean age was 64.4 years (72% males). The group included 18% diabetics, 67% dyslipidemics, 71% hypertensives and 37% smokers. Lesions assessed were in the Left Main 13%, LAD 57%, Circumflex 15% and RCA 15%.Under conditions of maximal hyperaemia the mean FFR for the cohort was 0.87 (range 0.80-0.98). Symptoms were minimal during a mean follow-up of 23 $\pm$ 7.5 months with , 15% patients reporting recurrent anginal chest pain, 5% had non anginal chest pain. and only 6% required PCI None of the patients within this group died.

Conclusion: An FFR of greater than 0.80 even in patients presenting with symptomatic CAD predicts an excellent longer term outcome at 2 years with conservative management and is a valuable aid to prevent unnecessary interventions in equivocal lesions.

Associated anomalies may not prolong the length of hospital stay in patients with abdominal wall defects

R. Pradhan [1] , V. Srivastava [1] , U. Samarakkody [1]*

[1] Waikato Hospital, Hamilton, New Zealand

*Corresponding author. Tel.: +64 7 8398765; fax: +64 7 8398716.

E-mail address: samaraku@waikatodhb.govt.nz (U. Samarakkody).

Objective: To study the abdominal wall defects treated at a regional tertiary centre over 25 years with emphasis on associated anomalies and the impact they had on the length of stay in the hospital.

Methods: From 1984 to 2008, 114 newborns with abdominal wall defects were treated at our hospital. 42 of these had omphalocele and 72 had gastroschisis. The relationship between the associated anomalies and the average length of hospital stay was analysed using ANOVA.

Results: Out of 72 patients with gastroschisis, 18(25%) had GI anomalies, 6(8.3%) had non-GI anomalies. 43(59.7%) did not have any anomalies. Patients with and without anomalies had a hospital stay of 32.7 days and 23.1 days respectively. 5(6.9%) patients had both GI and non-GI anomalies, their hospital stay being 22 days.

Out of 42 patients with Omphalocele, 6(14.3%) had GI anomalies, 21(50%) had non-GI anomalies. 10(23.8%) patients did not have any anomalies. Patients with and without anomalies stayed 20.7 days and 16.7 days respectively. 5(11.9%) had both GI and non-GI anomalies with 7.0 days stay.

In the entire study group we had only one death. Our survival rate was 98.6% for gastroschisis and 100% for omphalocele.

Conclusion: This study shows that the associated anomalies in both gastroschisis and omphalocoele may not prolong the hospital stay in a statistically significant fashion.

Diagnosis and treatment of heart failure in Māori and New Zealand Europeans at the Waikato Hospital

Dr Richard Wall [1], Dr Anita Bell [1], Dr Ross Lawrenson [2], Dr Gerard Devlin [1]

[1] Waikato Hospital, Hamilton, New Zealand, [2] Waikato Clinical School, Waikato Hospital, Hamilton, New Zealand

Background: Heart failure is associated with high rates of morbidity and mortality. In New Zealand, the burden of disease and its outcomes falls unequally on Māori. National and International heart failure guidelines provide evidence-based recommendations for diagnosis and management, and have been shown to improve survival and reduce hospitalisation. Differences in adherence to these recommendations at secondary care level may be a factor contributing to the inequalities in heart failure seen between Māori and non-Māori.

Aim: To determine the rate of use of key components of heart failure guidelines, and to make comparisons of their use between Māori and New Zealand Europeans.

Method: An audit of medical files was performed on a randomly selected sample of equal numbers of Māori and New Zealand European patients, with a first admission for heart failure to the Waikato Hospital between 01/01/2007 and 31/08/2008. Demographic and clinical data were collected, including the use of an echocardiogram and a BNP test for diagnosis, and the prescribing of key medications at hospital discharge. The diagnosis and management of cases was compared against the recommendations for care in the most up to date heart failure guidelines.

Results: A total of 71 Māori and 69 New Zealand European files were examined. Māori heart failure patients were more likely to be younger, male, have a diagnosis of diabetes and live in an area with a lower NZ deprivation score than New Zealand Europeans. An echocardiogram was performed in 57% and a BNP test in 54% of all cases during the admission, with no difference observed between Māori and NZ Europeans. ACE inhibitors were prescribed to 96% and beta-blockers to 82% of patients with systolic dysfunction and no contraindications at hospital discharge. Diuretics were prescribed to 84%, aldosterone antagonists to 17% and angiotensin receptor blockers to 4% of patients at hospital discharge, with no difference observed in prescribing rates between Māori and New Zealand Europeans.

Conclusion: There were no differences observed in the use of diagnostic tests or medication prescribing rates for heart failure between Māori and New Zealand Europeans during a hospital admission for heart failure at the Waikato Hospital. However data suggests that improvements could be made in the adherence to heart failure guidelines for diagnostic tests and several of the key medications.

Advanced gastric carcinoma and outcomes with palliative chemotherapy— does diffuse-type histology fare any worse?

R. Tota [1], M. Kuper [1], M. Jameson [1]

[1]Regional Cancer Centre, Waikato Hospital, Hamilton, New Zealand

Background: Advanced gastric cancer (AGC) is sensitive to numerous chemotherapy agents and combination chemotherapy regimens improve patient outcomes, including quality of life, though survival benefits are modest at best. It was our clinical impression that, of the various histological subtypes, diffuse gastric cancer seemed to have particularly low response rates and survival. We therefore reviewed our experience with palliative chemotherapy for AGC patients in the Midland region and in particular the influence of diffuse-type histology.

Methods: Patients with AGC assessed by Medical Oncologists in this institution between 2003 and 2008 were included in the analysis. Data was gathered on patient characteristics in those who received palliative chemotherapy, duration of treatment, response and survival. Actuarial survival was calculated using the Kaplan-Meier method and compared to outcomes from published trials.

Results: Over 6 years 68 patients were seen with a diagnosis of advanced (stage IV) gastric cancer, of whom 42 received chemotherapy. This treated cohort included 16 females and 26 males, median age 60 years (range 31–86). Histological subtypes, where defined, included diffuse (n=20) and intestinal (n=5) adenocarcinoma. Patients with diffuse-type histology comprised 10 males and 10 females, median age 57 years (range 42–76).

The most common chemotherapy regimen administered was epirubicin, cisplatin and 5-fluorouracil (ECF) in 32 patients, or ECX (where capecitabine was substituted for 5FU) in 6 patients. Treatment was administered for a median duration of 10 weeks (range 1–40). In the 30 patients whose tumour response to chemotherapy was documented in the clinical record, 13 (43%) achieved a partial response or better. Median survival was 6.7 months from start of treatment, with 24, 11 and 6 % of patients surviving at 12 months, 24 months and 36 months respectively. Survival in the subgroup of patients with diffuse histology was not significantly different (median 6.2 months, logrank p = 0.91).

Conclusions: Outcomes in this relatively small cohort of patients with AGC are similar to those documented in the literature with the use of palliative chemotherapy. For patients with diffuse-type histology, the sub-type most commonly in hereditary gastric cancer, the outcome was no worse. Only 5 patients had a family history of gastric cancer.

Intraoperative factors influencing pain in the immediate postoperative period

J. MacColl [1], C. Law [1], J. Sleigh [1] , L. Voss [1], J. Barnard[1].

[1] Waikato Hospital, Hamilton, New Zealand

Whether or not a “deep” or a “light” anaesthetic is better for the smoothness of patient recovery is unknown. In this exploratory study, we gathered a range of intraoperative data to see what might be used to predict the degree of post-operative pain. This data was collected from 103 gynaecological, orthopaedic, general and urological surgical patients. Post-operative pain was assessed using a simple ordinal 0-3 scale (0=no pain. 1=mild pain, 2=moderate pain, 3=severe pain). For the purposes of using

multiple logistic regression, this scale was subsequently condensed into two groups: 0&1 (Group A) and 2&3 (Group B). The variables used to predict which group a patient would fall into included: demographic data, degree of surgical trauma, the depth of anaesthesia (MAC, SE, ETaa), other EEG signs of arousal (spindles, delta waves) and intra-op morphine dose. Univariate analysis (t-test or chi-squared test) revealed that patients in group B (who had moderate or severe postoperative pain) had: (i) more severe surgery (p=0.02), (ii) more EEG spindles near the end of surgery (last 30min) (0.48(0.17) vs 0.40(0.16), p=0.01), (iii) a lower SE (37(8) vs 43(15), p=0.04), and (iv) a trend towards more delta waves (9.3(0.7) vs 8.9(1.1), p=0.06).

Multivariate analysis showed that surgical code, EEG spindles and SE significantly contributed to predicting post-operative pain. A linear discriminant function using these variables revealed that 32 of the 43 patients in group A and 41 of the 58 patients in group B were correctly classified, giving an overall 72.3% correct prediction of moderate or severe immediate post-operative pain.

Outcome of abdominal aortic aneurysms in the Māori population in New Zealand

N. Chiang[1], T. Vasudevan[1], C. Holdaway, D. Ferrar.

[1]***Department of Vascular Surgery, Waikato Hospital, Hamilton, New Zealand***

Background: Anecdotally, the indigenous Māori population presents earlier with abdominal aortic aneurysms (AAA) and have worse outcome following repair compared to Caucasians. The Māori population in Waikato comprises 20% of total regional population.

Purpose: Demographics and outcome of AAA repairs were evaluated with respect to ethnicity.

Methods: All AAA repairs over a 12-year period in Waikato Hospital were retrospectively reviewed. In particular, age of presentation, length of stay (LOS) and 1-, 3-, 6-, 12-, 36-, 60-, 120-month survival rates were studied.

Findings: 867 patients underwent AAA repair from 1996 to 2008. 627 were open and 240 were EVARs. Our EVAR program began in 1997. 324 were acute and 661 were males (M:F = 3.1:1). Average age was 73.1. Eighty-one (9.3%) patients were of Māori origin. Overall 30-day mortality rate was 12.6% with 27.2% in the acute group; 10-year survival rate was 33.8% with 29.3% in the acute group.

Mean age for the Māori patients was 67.6 compared to 73.7 in Caucasians (p<0.001). Mean age in the acute group for Māori was 66.0 compared to 73.6 (p<0.001). 10.6% of Māori patients presented acutely under the age of 50 compared to 0.8% of Caucasians. The M:F in the Māori and Caucasian population were 1.7:1 and 3.5:1 respectively.

Average LOS between the two groups was similar (Māori 9.9 days, Caucasian 9.1 days, p=0.24). Average LOS for EVARs was 5.4 days and 10.8 days for open repairs. 30-day mortality in Māori was 17.3% compared to 11.8% (p>0.05). The Māori

population appeared have lower long-term survival. 10-year survival rates in the Māori and Caucasian patients were 22.2% and 35.8% ($p<0.001$) respectively.

Conclusions: This study suggests that the Māori population present younger and have worse early and late outcome. Our results support the need for further population based studies.

Prognostic molecular markers in head and neck squamous cell carcinomas

I. Gunawardena[1], M. Arendse[2], T. Rogers[2] T. Lemaua[2] M. Jameson[3], G. Dutu[4], L. Plank[4], R. Lawrenson[4], R.T. Gregor[1]

[1]Department of Otolaryngology- Head and Neck Surgery, Waikato hospital, Hamilton, New Zealand

[2]Department of Pathology, Waikato hospital, Hamilton, New Zealand

[3]Department of Medical Oncology, Waikato hospital, Hamilton, New Zealand

[4]Clinical School, University of Auckland, New Zealand

Purpose: The survival rate for head and neck squamous cell carcinoma is among the lowest of the major cancers and has not improved significantly in the past two decades. Pathological features that correlate with an aggressive clinical course and poor prognosis include poorly differentiated tumours, tumour bulk, nodal involvement and perineural, stromal and vascular invasion. Despite such assessment, many tumours with similar histological features have widely differing clinical outcomes. Therefore improving prediction of prognosis would be an extremely valuable tool in determining appropriate clinical management strategies for each patient.

The aim of this study is to establish the prognostic significance of 6 molecular markers [Matrix metalloproteinase 2 and 9 (MMP2, MMP9), Tissue Inhibitors of MMP-1 (TIMP-1), Sialyl Lewis antigens a and x (sLe^a and sLe^x) and Alpha B Crystalline] in 145 New Zealand patients.

Methodology: Six sections were cut from each patient's paraffin-embedded tumour block, immunostained and scored by a consultant pathologist blinded to clinical features.

The Kaplan-Meier survival and Cox regression model were used to evaluate the influence of each biomarker on overall survival.

Results: Cox Regression model showed MMP-2 ($p=0.019$, $HR=1.82$) to be statistically significant for overall survival. Alpha B Crystalline positivity shows a trend towards worse survival ($p=0.093$).

Conclusion: MMP-2 is a significant prognostic marker for overall survival in head and neck squamous cell carcinoma in a multivariate analysis.

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Intermittent blood glucose measurements may miss episodes of hypoglycaemia in at risk newborn babies

D.L. Harris [1,2], P.J. Weston [1], J.E. Harding [2]

[1] Newborn Intensive Care Unit, Waikato District Health Board, Hamilton, New Zealand

[2] Liggins Institute, University of Auckland, Auckland, New Zealand

Email: harrisde@waikatodhb.govt.nz

Background: Neonatal hypoglycaemia can cause brain damage and death. However, the best approach to detection and management remains unclear. Blood glucose concentrations are measured intermittently by heel prick blood samples, and thus episodes of hypoglycaemia may go undetected. Continuous interstitial glucose monitoring is useful in diabetic patients, but remains untested in newborn babies at risk of hypoglycaemia.

Aim: To determine whether continuous glucose monitoring is useful in newborn babies at risk of neonatal hypoglycaemia.

Method: Babies at risk of hypoglycaemia and admitted to the Newborn Intensive Care Unit received routine management and intermittent blood glucose measurement using the glucose oxidase method. The interstitial glucose sensor was placed subcutaneously in the babies' thigh and recorded an interstitial glucose concentration every five minutes. Low glucose concentration was defined as blood glucose or interstitial concentration < 2.6mmol/l.

Results: One hundred and two babies were enrolled with a median (range) birth weight 2327g

(1032 - 4960 g), and gestation 35 wk (31 - 42 wk). Blood glucose concentrations were measured on 1750 occasions and were < 2.6mmol/l in 98 (6 %). Interstitial glucose concentrations were measured on 97119 occasions, and were < 2.6mmol/l in 2337 (3 %). There was good agreement between blood and interstitial glucose concentrations (mean difference 0.0mmol/l, 95% confidence intervals -1.1 to 1.1 mmol/l, correlation coefficient 0.91). Low glucose concentrations were detected in 32 (31%) babies by blood sampling and in 45 (44%) babies by continuous interstitial monitoring. Low interstitial glucose concentrations lasted from 5 to 475 minutes, with 34 babies (33%) experiencing at least one episode of >30 minutes. There were 107 episodes of low interstitial glucose concentrations lasting at least 30 minutes and 78 (73%) of these episodes were not detected by blood glucose measurement.

Conclusion: Continuous glucose monitoring may be useful in newborn babies at risk of hypoglycaemia. Many episodes of hypoglycaemia may not be detected by

intermittent blood glucose measurement. The physiological significance of undetected hypoglycaemic episodes is unclear.

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COMMD2 interacts with the epithelial sodium channel ENaC.

L Glass, F McDonald. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

COMMD2 is a member of the newly discovered Copper Metabolism Murr1 Domain (COMMD) containing family of proteins. The first member, COMMD1, is known to negatively regulate the epithelial sodium channel (ENaC). ENaC is an important channel in the body, regulating sodium homeostasis and blood pressure. This study investigated the effects of COMMD2 on ENaC.

Interactions between COMMD2 and ENaC were examined using co-immunoprecipitation in COS-7 cells transiently transfected with ENaC and COMMD2. The functional effect of COMMD2 on ENaC was examined by comparing the amiloride-sensitive sodium current generated by ENaC-transfected FRT (Fisher rat thyroid) epithelial cells in the absence and presence of COMMD2. The effect of COMMD2 on formation of fully functional FRT epithelia was tested by transfecting FRT cells with ENaC, with and without COMMD2. Then cell proliferation was examined by cell counts using a haemocytometer, and tight junction integrity was tested with fluorescein isothiocyanate dye that can only move through the epithelium via the paracellular pathway.

This study showed that COMMD2 interacted with αβγ ENaC (n = 3) and δβγ ENaC (n = 3), and also the ENaC regulator serum and glucocorticoid-regulated kinase (SGK) (n = 3). There were no significant effects of COMMD2 at any of the tested doses on the amiloride-sensitive current of either αβγ ENaC (n = 3 – 7 per dose) or δβγ ENaC (n = 1 – 2) transfected FRT cells. Samples transfected with δβγ ENaC showed decreased resistance in functional experiments suggesting loss of epithelial polarity, however cell counts (n = 3) and permeability assays (n = 2) showed that COMMD2 had no significant effect on FRT cell proliferation or the integrity of tight junctions formed between cells.

These results show that COMMD2 interacts with ENaC, however the effect of this interaction is still unclear. It is possible that COMMD2 also regulates ENaC providing another mechanism by which body sodium homeostasis is maintained.

Suppressor of cytokine signalling-3 is partially responsible for infertility in mice fed a high fat diet. H McEwen, J Quennell, G Anderson. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Fertility in the western world has been decreasing, and at the same time the average age of puberty has been decreasing. Both of these changes have been correlated with an increased consumption of high caloric foods leading to a rise in the incidence of obesity. A critical neuroendocrine signal for puberty onset and fertility is the hormone leptin, produced by adipose tissue. Obese individuals have elevated leptin levels due to increased adipose deposits. In the long term this induces leptin resistance by mechanisms such as elevated levels of suppressor of cytokine signalling-3 (SOCS-3), a negative regulator of leptin signalling in the hypothalamus. This investigation aimed to test whether upregulation of SOCS-3 plays a role in fertility suppression in animals fed a high fat diet (HFD).

Using CRE/LoxP-transgenics, SOCS-3 was selectively knocked-out from all forebrain neurons in both male and female DBA/2J mice. Neuronal SOCS-3 knockout mice (KO) and control littermates were fed either a HFD (24% fat) or standard chow diet (4.6% fat) from weaning and subjected to a comprehensive fertility analysis (n = 5 – 7). Onset of puberty, measured as first estrus in females or the first successful mating in males, was advanced by 5.3 ± 1.7 days in male ($P < 0.01$; two-way ANOVA) but not female mice fed a HFD when compared to chow-fed littermates. The bodyweight at puberty onset of KO mice fed a HFD was on average 2.2 g less than that of controls ($P < 0.05$ for both sexes) implying enhanced hypothalamic leptin signalling. Male mice showed no effects of diet or genotype on adult fertility, however female KO mice on a HFD were partially saved from infertility compared to control mice on the same diet (interval between litters 44.8 ± 6.5 and 63.6 ± 5.0 days, respectively; $P < 0.01$) and both genotypes on chow diet (27.6 ± 1.1 days; $P < 0.05$).

These results show that upregulation of hypothalamic SOCS-3 is responsible in part for HFD-induced infertility, and supports the idea that resistance to leptin is a cause of infertility in obese individuals.

The blood supply of the major duodenal papilla: a microdissection study. A Mirjalili, M Stringer. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Arterial bleeding from the major duodenal papilla is a serious complication after endoscopic intervention in the biliary tract, occurring in up to 6% of procedures. Only two previous studies of the blood supply of this region have been reported, both based on gross dissection. The aim of our study was to investigate the blood supply of the major duodenal papilla by microdissection.

The distribution pattern of papillary arteries supplying the major duodenal papilla was investigated by dissecting specimens from 10 embalmed cadavers (6 males, 4 females, median age 80 y). The number, origin, and distribution of arteries seen under a dissecting microscope were recorded and their diameter measured using digital calipers. Additionally, an arterial cast was produced in two fresh cadaver specimens to delineate these arteries.

A total of 57 papillary arteries were identified in 10 specimens (range 3 – 9 per specimen); with an external diameter of 0.73 ± 0.19 mm (mean ± SD). Of these arteries, 42 (74%) were from the communicating artery, 14 (25%) were from the posterior pancreaticoduodenal arcade, and only 1 from the anterior arcade. Importantly, their distribution around the circumference of the papilla was not uniform: most arteries were located in the posteroinferior and anterosuperior quadrants. Only 9 (16%) and 3 (5%) of the arteries were located in the posterosuperior and anteroinferior quadrants, respectively. Two vascular corrosion casts helped to clarify pancreaticoduodenal arterial supply but papillary branches could not be distinguished with confidence.

Prior to this study, only one or two arteries were reported as supplying the major duodenal papilla. This novel microdissection study shows that there are between 3 and 9 arteries originating from two main sources. Knowledge of the circumferential distribution pattern of these branches might help to reduce the risk of bleeding associated with interventional endoscopic procedures in this region.

The orf virus chemokine binding protein is a novel broad-spectrum chemokine inhibitor. M Corbett, N Real, S Fleming, A Mercer, L Wise. Department of Microbiology and Immunology, Otago School of Medical Sciences, University of Otago, Dunedin.

Orf virus (ORFV) is a large DNA virus that causes skin lesions in sheep and zoonotic infections in humans. Previous studies on the chemokine binding protein (CBP) encoded by ORFV showed that it binds the CC- and XC- chemokine classes and inhibits LPS-induced recruitment of leukocytes. In this study the aim was to further examine the spectrum of chemokine binding by the ORFV CBP and determine its preference for the different classes of chemokine.

To determine which chemokines the ORFV CBP binds, chemokines were mixed with increasing amounts of the ORFV CBP and then unbound chemokine was detected using an ELISA. The ORFV CBP inhibited detection of the CC-chemokines RANTES, MIP-1α, MIP-3β and MCP-1 with IC_{50} of 0.2, 1, 1.2 and 3.5 nM, respectively. The ORFV CBP inhibited detection of the CXC-chemokines MIP-2 and PF4 with IC_{50} of 1 and 2.8 nM, respectively, but did not bind IL-8, IP-10 or SDF-1. The ORFV CBP inhibited detection of the XC-chemokine lymphotactin with an IC_{50} of 10 nM.

A competition ELISA using a primary chemokine incubated with increasing concentrations of a secondary chemokine before the addition of the ORFV CBP was used to determine the CBPs class preference. The ORFV CBP displayed a preference for lymphotactin, which was able to prevent both MCP-1 and MIP-2 binding, while

MIP-2 was unable to inhibit binding to lymphotactin or MCP-1. In a displacement ELISA with pre-bound chemokine, lymphotactin was displaced by MCP-1 and MIP-2, however MIP-2 was recalcitrant to displacement by lymphotactin or MCP-1.

These results demonstrate that ORFV CBP has a broader spectrum than previously described, binding representatives of all three classes of chemokine with high affinity. While the chemokines appear to compete for the same binding site, the ORFV CBP did exhibit differences in its ability to bind and retain the different classes of chemokine.

Differential effects of agmatine treatment on spatial working and reference memory. D Bergin, P Liu. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Agmatine is a metabolite of L-arginine. Recent research suggests that endogenous agmatine is a novel neurotransmitter and exogenous agmatine can modulate behavioural function, including learning and memory. The present study investigated the effects of intracerebroventricular agmatine microinfusion on spatial learning and memory.

Adult male Sprague-Dawley rats were anaesthetised and implanted with a cannula into the lateral ventricle. Rats received a single daily infusion of low (10 µg/5 µl, n = 8) or high (100 µg/5 µl, n = 8) concentration agmatine, or saline (5 µl, n = 8) for 13 consecutive days from six days post-surgery. The water maze experiment began 9 days post-surgery (after 4 infusions) and each daily test commenced 10 min after each infusion. In the reference memory version of the task, rats were trained to find a visible platform (cued navigation, day 1), and a hidden platform in a fixed location (place navigation, days 2 – 6). The animal's memory of the platform location was tested by conducting a probe trial with no platform presented on day 6. Rats with agmatine treatment at both concentrations performed similarly to the saline controls in these tests. In the working memory version of the task (days 7 – 10), rats were trained to find a new platform location either 30 s or 180 s following a sample trial. The two agmatine groups generated markedly shorter path length and took significantly less time to reach the platform at the 180 s, but not 30 s, delay as compared to the saline group (all $P < 0.05$; two-way repeated measures ANOVA with Newman-Keuls *post-hoc* tests).

These results demonstrate that agmatine treatment significantly facilitates spatial working memory under a longer delay, but not reference memory, suggesting its differential influence on the two types of spatial learning and memory. The underlying mechanisms will be explored in the future.

Cellular mechanisms of prolactin regulation of oxytocin neurons in reproduction. Y Alyousif[1,2] , V Scott[1] , I Kokay[2] , D Grattan[2] , C Brown[1]. Centre for Neuroendocrinology and [1]Department of Physiology, [2]Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

The hormone oxytocin is secreted from the posterior pituitary gland to regulate parturition and lactation. Oxytocin secretion is determined by the action potential discharge of magnocellular oxytocin neurons of the hypothalamic supraoptic nucleus (SON) and paraventricular nucleus. Oxytocin neurons undergo plasticity to prepare for the increased demands of parturition and lactation, and express receptors for another reproductive hormone, prolactin. Prolactin, produced from the anterior pituitary gland, decreases the firing rate of oxytocin neurons in virgin (diestrous) rats, but stimulates oxytocin mRNA expression in lactating rats. Therefore, we hypothesised that prolactin inhibition of oxytocin neuron firing rate will be reduced in early lactation.

To test this hypothesis, extracellular single-unit activity was recorded from identified oxytocin neurons in the SON of urethane-anaesthetised diestrous and lactating (day 6-12 post-partum) female Sprague-Dawley rats. Using intracerebroventricular (icv) dose-ranging experiments in diestrous rats, 1.0 µg (in 1 µl) of prolactin was determined to be the minimum effective dose, and this dose was used in all subsequent experiments.

Following baseline recording, the effect of icv prolactin on the firing rate of oxytocin neurons was examined. In diestrous rats (n = 6), 1.0 µg (in 1 µl) prolactin reduced oxytocin neuron firing rate from 4.3 ± 0.7 to 2.1 ± 0.4 spikes s^{-1} (mean ± SEM, $P < 0.05$, two-way-repeated-measures ANOVA, followed by Bonferroni's *post-hoc* test), averaged over 5 min before and after prolactin injection. By contrast, 1.0 µg prolactin did not alter the firing rate of oxytocin neurons in lactating rats (n = 6; 4.3 ± 1.1 and 4.5 ± 0.9 spikes s^{-1}, before and after prolactin, respectively; $P > 0.05$).

Hence, prolactin inhibition of oxytocin neuron activity is reduced in early lactation. This might contribute to the increased activity of oxytocin neurons required for delivery of milk to the offspring during lactation.

Preclinical development of a novel curcumin analogue for the treatment of oestrogen receptor negative breast cancer. K Allen[1], L Larsen[2], S Taurin[1], R Rosengren[1]. [1]Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin; [2]Plant and Food Research Limited, Dunedin.

New treatments are required for breast cancers lacking the oestrogen receptor (ER-) as these are more aggressive and have a worse prognosis than ER+ breast cancer. Curcumin exhibits anti-cancer effects *in vitro* and *in vivo* but lacks suitable oral bioavailability for cancer treatment. This has lead our laboratory to synthesise many novel curcumin analogues. After early screening, the analogue RL-92 showed a high

cytotoxic potency towards ER- breast cancer cells and was detected in the plasma of mice following a single oral dose. The aim of this project was to examine the safety and efficacy of RL-92 in a xenograft model of ER- breast cancer.

MDA-MB-231 breast cancer cells were implanted subcutaneously in female athymic nude mice. Mice (n = 10/group) were dosed daily for 10 weeks with vehicle (water), or RL-92 (0.85 or 8.5 mg/kg). Tumour volume was measured weekly and animals were weighed daily. The results showed that mean tumour growth was reduced 2.9-fold after RL-92 (0.85 mg/kg) administration compared to vehicles (from 584 +/- 496 to 243 +/- 200 mm^3, mean +/- SEM, $P<0.01$, two-way ANOVA with Bonferroni *post-hoc* test). Plasma alanine aminotransferase levels and organ weights were normal, demonstrating that RL-92 is non-toxic.

To determine if RL-92 reduced tumour growth by inhibiting angiogenesis, *in vitro* assays were performed. In the scratch assay, MDA-MB-231 cells were treated with DMSO or RL-92 (2, 4, or 6 µM) for 24 hours and images compared at 0 and 24 hours. Cell migration was reduced with maximal reduction occurring at 6 µM RL-92. RL-92 also inhibited endothelial tube formation (HUVEC cells) and transwell migration (1 and 0.5 µM, respectively).

The xenograft tumour model data will be repeated. Immunohistochemistry on tumour tissue will assess reduction in CD105 levels (angiogenesis marker) to compliment the *in vitro* data.

Effect of 17β-estradiol on ERK phosphorylation in a neurodegenerative mouse model. M Turnbull, I Ábrahám. Centre for Neuroendocrinology and Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

Cholinergic neurons of the basal forebrain, which includes the substantia innominata (SI), provide a major source of cholinergic projections to the cortex and are involved in various cognitive functions. Deterioration of this system is responsible for cognitive deficits seen in ageing and neurodegenerative diseases. 17β-estradiol represents the major estrogen and is an essential contributor of the cholinergic response to harmful stimuli and neurodegenerative processes. 17β-estradiol action is mediated through two pathways; the classical pathway which directly alters gene transcription, and the non-classical pathway which activates second messenger signalling systems and is known to be protective against injury. Previous studies have shown 17β-estradiol to restore cholinergic cortical innervation after injury. Therefore we aimed to investigate non-classical signalling, represented by phosphorylated extracellular signal-regulated kinase (pERK), in promoting cholinergic fibre growth.

N-methyl-D-aspartic acid (NMDA) was unilaterally injected into the SI of ovariectomised mice to induce cholinergic cell death. Fifteen minutes later, the mice were given either 17β-estradiol (1 µg/30 g body weight) or vehicle (ethyl oleate; 1 µg/30 g body weight) subcutaneously. Presence of pERK in cholinergic neurons was determined using fluorescence immunohistochemistry (all results: mean ± SEM, n = 5). NMDA injection resulted in pERK upregulation in cholinergic neurons of the SI (31.4 ± 4.2%) compared to the contralateral SI (17.3 ± 2.5%, $P < 0.001$, two-way

ANOVA with Bonferroni *post-hoc* test) in vehicle-treated animals. However in 17β-estradiol-treated animals, NMDA did not affect pERK expression in the SI (21.9 ± 2.6%) and levels remained similar to the contralateral SI (19.1 ± 2.0%).

Hence, 17β-estradiol does not upregulate pERK expression but instead reduces NMDA-induced pERK upregulation in cholinergic neurons of the SI. It has been reported that pERK inhibitors reduce 17β-estradiol ameliorative action, thus we suggest that pERK is upregulated in other cells of the SI and 17β-estradiol neuroprotection is mediated indirectly.

Investigation of the stabilising effect of COMMD1 on SGK1. H-J Liu, Y Ke, F McDonald. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

Serum- and glucocorticoid-inducible kinase 1 (SGK1) contributes to the regulation of sodium homeostasis, cell proliferation and apoptosis. COMMD1 (copper metabolism Murr1 domain containing protein 1) is known to stabilise SGK1. SGK1 protein level is controlled by ubiquitination and degradation. We hypothesised that COMMD1 interferes with SGK1 ubiquitination and degradation.

Protein interaction and stability assays were used in this study to further investigate the mechanism by which COMMD1 stabilises SGK1. All western blot results were quantified by densitometry, normalised to the appropriate controls, and analysed by one-way ANOVA and *post-hoc* Dunnett's test. Using co-immunoprecipitation, COMMD1 was found to interact with wild-type SGK1, a form of SGK1 that is unable to interact with an ubiquitin ligase ($SGK1_{Y298A}$) and a stable form of SGK1, lacking the N-terminal 60 amino acids ($SGK1_{\Delta N60}$). Steady-state expression level studies revealed that COMMD1 exerted the largest stabilising effect on wild-type SGK1 (260 ± 22%, mean ± SEM, n = 3, $P < 0.001$).The stabilising effect of COMMD1 on $SGK1_{Y298A}$ was reduced (204 ± 22%, n = 3, $P < 0.01$). COMMD1 did not affect the stability of $SGK1_{\Delta N60}$ (105 ± 5%, n = 3, $P > 0.05$). Conversely, knockdown of endogenously expressed COMMD1 by small interfering RNA de-stabilised wild-type SGK1 (40 ± 7.7%, n = 4, $P < 0.001$), whereas for $SGK1_{Y298A}$ the de-stabilising effect was reduced (15 ± 6%, n = 3, $P < 0.001$). Knockdown of COMMD1 had no effect on the stability of $SGK1_{\Delta N60}$ (3 ± 1.2%, n = 3, $P > 0.05$).

These results suggest that COMMD1 only stabilises wild-type SGK1 that undergoes ubiquitination and degradation. An ubiquitination assay demonstrated that knockdown of COMMD1 increased ubiquitination of wild-type SGK1 (239 ± 18%, n = 3, $P < 0.001$). A cycloheximide-based protein chase assay revealed that over-expression of COMMD1 increased the half-life of wild-type SGK1 (from 25 to 46 min, n = 3).

In conclusion, COMMD1 enhances the stability of SGK1 by attenuating its ubiquitination and degradation. The stabilising effect of COMMD1 on SGK1 may potentially have implications in maintaining sodium homeostasis under chronic salt intake conditions and protecting against cell apoptosis.

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Incidental findings on brain magnetic resonance imaging (MRI)

MRI has proven to be a very powerful tool in evaluating brain pathology. However, there is a down side—the incidental findings. How frequently they are found and their impact is reviewed in this meta-analysis. Sixteen studies involving 19,559 subjects are evaluated and the crude prevalence of incidental findings on brain MRI is 2.7% or 1 for every 37 neurologically asymptomatic people scanned. The asymptomatic people included volunteers, research controls, and some were clinical or occupational screening subjects. 135 of 19,559 people had neoplastic incidental brain findings (prevalence 0.7%). A further 375 had non-neoplastic incidental findings—cysts, structural vascular abnormalities, inflammatory lesions and other non-specific abnormalities. The analysis excluded white matter hyperintensities, silent brain infarcts or lacunae, and brain microbleeds because of their known increasing prevalence with age. And the concluding advice is that "these findings deserve to be mentioned when obtaining informed consent for brain MRI in research and clinical practice but are not sufficient to justify screening healthy asymptomatic people".

BMJ 2009;339:b3016.

Advanced dementia—under-recognised as a terminal illness

In this report from Boston, USA, the health of 323 nursing home residents with advanced dementia has been documented over an 18-month period. Over this period 54.8% died. The probability of pneumonia was 41.1%; a febrile episode, 52.6%; and an eating problem 85.5%. The 6-month mortality rate for residents who had pneumonia was 46.7%; a febrile episode, 44.5%; and an eating problem, 38.6%. Furthermore, in the last 3 months of life, 40.7% of residents underwent at least one burdensome intervention (hospitalization, emergency room visit, parenteral therapy, or tube feeding). The opinion is offered that such burdensome interventions are inappropriate in terminally ill patients. They could be avoided if the relatives or carers were better informed of the expected clinical complications and the associated poor prognosis in such patients.

N Engl J Med 2009;361:1529–38.

Carpal Tunnel Syndrome and diabetes mellitus

Carpal tunnel syndrome is the most common entrapment neuropathy, with a reported prevalence in the general population of 2% to 4%. But its prevalence may be as high as 15% in diabetics and up to 30% in those with polyneuropathy. Some believe that surgical decompression is less effective in the diabetic subject. The authors of this paper from Sweden dispute this. They have prospectively matched 35 diabetic and 31 non-diabetic patients who underwent surgical treatment for their carpal tunnel syndromes. And the outcome of the study showed that patients with diabetes have the

same beneficial outcome after carpal tunnel release as non-diabetic patients. Only cold intolerance demonstrated a lesser extent of relief for diabetic patients.

J Hand Surg 2009;34A:1177–87.

Induction of labour for gestational hypertension or mild pre-eclampsia

Up to 8% of pregnancies are complicated by hypertensive problems which may cause maternal problems and foetal morbidity and mortality. In this report from the Netherlands an hypothesis is tested—does induction of labour after 36 weeks gestation produce better maternal and foetal outcomes when compared to medical management? 756 patients were allocated to receive induction of labour (n=377 patients) or expectant monitoring (n=379). 31% of the induced patients developed a poor maternal outcome compared with 44% of those allocated to expectant monitoring—a very significant difference ($p<0.0001$) favouring induction. The maternal morbidities reduced included less severe hypertension and need for hypotensive drugs, less need for anticonvulsant treatment, less postpartum blood loss and less need for intensive care. No maternal or foetal deaths occurred. Their conclusion is that "induction of labour is associated with improved maternal outcome and should be advised for women with mild hypertensive disease beyond 37 weeks' gestation". We note that "after 36 weeks" has been superseded by "beyond 37 weeks".

Lancet 2009;374:979–88.

Chronic atrial fibrillation (AF)—which drugs for rate control management?

The National Institute for Health and Clinical Excellence (NICE) and the American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) have recently recommended the use of β-blockers or rate limiting calcium antagonists as first line treatment, thus relegating digoxin which previously held pride of place. The authors of this systematic review dispute the recommendations. 46 studies comparing oral digoxin, β-blockers and calcium antagonists, alone or in combination, for rate control in chronic atrial fibrillation have been evaluated. They found little evidence that monotherapy with β-blockers or calcium antagonists improves symptoms or exercise capacity in patients with chronic atrial fibrillation. Instead it is associated with dose related side effects. They recommend a combination of digoxin and β-blocker or calcium antagonist as the synergistic effect would achieve rate control with smaller dosage of drugs and less dose related adverse effects.

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Obesity and mental disorders in the Māori population of New Zealand: the difficulties conducting culturally specific research

Research has identified a high prevalence of mental disorders, especially substance use disorders, in the Māori population compared to other ethnic groups in New Zealand.[1] In addition, 27.5% of Māori adults are obese compared with 14.7% of New Zealand European adults[2] and extreme obesity affects 5.0% of Māori children compared to 1.0% of New Zealand European/Other children.[3] This apparent co-predilection for obesity and mental disorders in Māori may be due to the disproportionate representation of Māori in lower socioeconomic strata[2]; disadvantage in terms of educational opportunities; cultural norms for body image and dietary habits; and limited perceived and actual access to healthcare, compared to non-Māori.[1,4]

The problem of access to healthcare is thought to be due to concerns of miscommunication[5], feelings of alienation, and not being entitled to high-quality health services[5] and an under representation of Māori as health professionals.[6] To overcome these ethnic disparities in healthcare, culturally specific services such as the Māori mental health service have developed in New Zealand.

As a final year medical student from the United Kingdom I planned to conduct research within the Māori mental health service to investigate the coexistence of obesity and mental disorders. This work aimed to assist in the prevention and treatment of both of these conditions and to improve total (physical and mental) Māori health.

Despite thorough planning, a number of barriers restricted this work. These included the instability of the service itself at the time of the planned research, sample size limitations, and time constraints restricting the ability to adhere to Māori practices necessary to develop the level of rapport and trust needed to obtain consent for research with Māori patients. This included the importance of sharing ones whakapapa/identity.[7] In addition the apparent inappropriateness of the nature of the project for a non-Māori international student to conduct meant this work had to be adapted. Instead a reflective, observational study was conducted, including interviews with three medical students who had experience of this culturally specific service.

The analysis of the three interviews concluded that, consistent with the literature, the co-prevalence of mental health problems and obesity in the Māori population appeared to be considerable. There was consensus among those interviewed that the Māori mental health team provided a necessary and effective service to an already ‘at risk’ group. In addition, contrary to initial concerns, the service appeared to have minimal negative societal impact, for example promoting cultural discrimination and segregation, which may be due, at least in part, to the ‘opt in’ inclusive nature of the service for non-Māori patients. The holistic approach to health based on the whare tapa wha model[8] was the main difference identified in this service compared to other

health services experienced by those interviewed. This was noticeable through the focus on developing rapport with the patient's' family and a Māori - specific view of the concept of confidentiality. There was agreement among those interviewed that obesity may be associated with mental health problems among Māori patients; however confounding factors complicate pursuit of evidence for this association.

As a result, this review promotes the extension of this culturally tailored approach to the co-management of obesity and mental disorders in the Māori population. This notion of targeted care is supported by Metcalf et al. who highlighted the importance of providing ethnic specific obesity management programmes by addressing food and health in the context of culture and socioeconomic status of the group.[9]

Despite restrictions to the initial research planned, this review has raised my awareness of the culture and health problems of this population group; formulated interesting conclusions from observations and interviews to assist in future co-management of obesity and mental health among the Māori population; and finally, rather importantly, identified key barriers to research in this area which will inform future work in this area.

Amber Appleton
Final year medical student
King's College, London Medical School
London, UK
(amber.appleton@gmail.com)

Note: Research conducted in the Māori mental health team, Hutt Hospital, Hutt Valley District Health Board, New Zealand.

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Smoker misperceptions around tobacco: national survey data of particular relevance to protecting Māori health

The Māori Affairs Select Committee is undertaking an inquiry into "the tobacco industry in Aotearoa and the consequences of tobacco use for Maori".[1] Whilst tobacco impacts on all New Zealand society, this inquiry is very appropriate from a public health perspective given tobacco's contribution to poorer health for Māori and to health inequalities in New Zealand.[2,3] Tobacco use also has a highly adverse impact on Māori social, cultural and economic development (e.g. expenditure on cigarettes by Māori in 2000 was estimated at $266 million per year[4]). The timeliness of this issue is further demonstrated by recent calls by Māori political leaders for advancing the tobacco endgame.[5]

A possible contributor to the continued high prevalence of tobacco smoking among Māori is underestimation of the extent of the risks to health posed by tobacco smoking and secondhand smoke exposure, and misperceptions that "light", menthol and roll-your-own (RYO) cigarettes are safer forms of smoked tobacco. Other possible misperceptions include that second-hand smoke is not a hazard to health and that smokeless tobacco products are as, or almost as, harmful as smoked tobacco products. To better understand issues around smoker knowledge and misperceptions, we analysed relevant data from a cohort study of smokers.

The data came from a national survey of 1376 New Zealand adult (18+ years) smokers surveyed between March 2007 and February 2008. This process involved some booster sampling of Māori (n=607 respondents). This study was the New Zealand arm of the International Tobacco Control Policy Evaluation Survey (ITC Project).[6] Specific questions relevant to possible misinformation included: perceptions related to light/mild cigarettes/tobacco, to menthols, and to RYO tobacco etc. Many of the same questions (and some additional ones) were repeated in the second wave of the survey, over the subsequent year (n=923 respondents). Further detail on the survey methods are available in an online *Methods Report*[7] and in other journal article publications from this project.[8,9]

The results in Table 1 indicate that sizeable minorities of both Māori and European/Other smokers have various misperceptions. Regarding light and mild cigarettes, nearly half (48%) of Māori smokers have at least one of three misperceptions which suggest (erroneously) that these cigarettes have health benefits compared to "regular" cigarettes. We have reported elsewhere[8] on the international literature on these misperceptions, which indicates that they are linked to branding and marketing by the tobacco industry. This marketing behaviour continues in the New Zealand setting, in the form of certain words on packs (e.g. "subtle" and "mellow") and in the colour coding of packs.[10] In some New Zealand settings the words "light" and "mild" continue to be used (e.g. Woolworths online shopping as of November 2009), which is counter to the ruling by the Commerce Commission in 2008.[11]

The addition of menthol to tobacco helps to disguise the harsh taste of tobacco smoke and may ameliorate smoker concerns that tobacco smoking is intrinsically dangerous. Indeed, some New Zealand smokers have misperceptions about mentholated cigarettes ("menthols") being less harmful relative to "non-mentholated" cigarettes. This misperception was significantly more common (13% vs 7%) among Māori smokers (Table 1). The available international data are generally consistent with the notion that menthols are at least as dangerous as their non-mentholated counterparts[12] and may even pose a greater health risk than regular cigarettes.[13]

Table 1. Selected results of ITC Project surveys relevant to smoker (mis)perceptions (with all results weighted and adjusted for the complex sample design to represent the national population of smokers in New Zealand)

Question asked (all Wave 1 data unless otherwise indicated)	Māori	European/ Other (excluding Pacific and Asian smokers)	Crude odds ratios (Māori vs European/ Other) (95% confidence intervals)	Comments
Light and mild cigarettes				
Agree or strongly agree that "lights" make it easier to quit smoking.	26.5%	19.3%	1.50 (1.06 – 2.13)	Māori smokers were significantly more likely to have this misperception.
Agree or strongly agree that "lights" are less harmful than regular cigarettes.	27.8%	26.8%	1.05 (0.76 – 1.46)	Further details on the use and beliefs around light cigarettes among New Zealand smokers is published elsewhere.[8]
Agree or strongly agree that smokers of "lights" take in less tar than smokers of regular cigarettes.	38.1%	36.4%	1.08 (0.80 – 1.46)	
Holding at least one of the above 3 beliefs that "lights" confer health benefits.	48.3%	46.4%	1.08 (0.81 – 1.44)	
Menthols				
Agree or strongly agree with the statement "menthol cigarettes are less harmful than regular cigarettes". (Wave 2 data)	13.3%	6.7%	2.14 (1.03 – 4.42)	Māori smokers were significantly more likely to have this misperception.
Roll-you-own (RYO) tobacco				
Give the reason "not as bad for your health" for smoking RYOs among RYO smokers.*	21.8%	21.0%	1.05 (0.67 – 1.64)	–
Harm from secondhand smoke				
Disagree or strongly disagree that "cigarette smoke is dangerous to non-smokers".	6.3%	8.6%	0.72 (0.44 – 1.17)	–
Say "no" when asked if secondhand smoke can cause "lung cancer in non-smokers".	9.9%	14.7%	0.64 (0.43 – 0.96)	Māori smokers were significantly *less* likely to have this misperception.
Say "no" when asked if secondhand smoke can cause "asthma in children".	8.5%	10.8%	0.77 (0.50 – 1.20)	–
Lower-harm products				
Say "no" when asked if any smokeless tobacco products are less harmful than ordinary cigarettes?	48.1%	50.8%	0.90 (0.68 – 1.19)	Further details on the knowledge and beliefs around smokeless tobacco products among
State that they have "never heard of	18.3%	15.3%	1.23	

Question asked (all Wave 1 data unless otherwise indicated)	Māori	European/ Other (excluding Pacific and Asian smokers)	Crude odds ratios (Māori vs European/ Other) (95% confidence intervals)	Comments
smokeless tobacco products".			(0.86 – 1.78)	New Zealand smokers is published elsewhere.[9]
For those saying that smokeless tobacco products are less harmful – saying that they are only "a little" less harmful.	60.7%	48.2%	1.66 (0.76 – 3.63)	
Other				
Agree or strongly agree that "tobacco companies have done everything they can to reduce the harm caused by smoking".	23.8%	18.1%	1.42 (1.02 – 1.97)	Māori smokers were significantly more likely to have this view (arguably a misperception given the evidence[21]).

* Includes exclusive RYO smokers and mixed (RYO + factory-made cigarette) smokers. For further details on why RYO smoking is at least as hazardous and a comparison with the misperceptions of Australian smokers, see elsewhere.[22]

Research has also shown that many smokers believe that "the harsher the smoke feels in your throat, the more dangerous the smoke is".[14] The addition of menthol to tobacco by the tobacco industry may therefore contribute to such misperceptions, because smokers frequently agree that menthols are "more soothing on the throat".[15] Indeed, over half (56%) of smokers in our study agreed with the statement that "menthol cigarettes are smoother on your throat and chest than regular cigarettes". Māori were also significantly more likely to agree with this statement compared to Europeans (additional data not shown).

A minority (up to 10%) of Māori smokers also have specific misperceptions about the adverse health effects of second-hand smoke (as detailed in Table 1). It is possible that these misperceptions also reflect tobacco industry misinformation on this hazard which was promulgated during the build up to the last major smokefree environments law in New Zealand.[16]

Around a fifth of Māori and European/other smokers gave health reasons for smoking RYO cigarettes (Table 1). Yet the tobacco industry has done nothing to warn smokers about the misperception that RYO tobacco is less hazardous than smoking factory-made cigarettes. For example, while British American Tobacco (NZ) has a section on RYOs on its website, there is no mention of its health risks (as of November 2009). Furthermore, some descriptors on RYO packaging in New Zealand (e.g., the word "original") may reinforce some smokers' misperceptions that RYO is more "natural" and therefore less hazardous (with this "natural" belief being discussed for RYO smokers in other countries,[17] but not yet specifically investigated in New Zealand).

Smokers also have high levels of knowledge deficits and misperceptions around smokeless tobacco products (Table 1), with around half of Māori and European/other smokers disagreeing that smokeless tobacco products are less harmful than ordinary cigarettes. Yet smokeless products are substantially less hazardous to health than smoked tobacco, and could be used as part of a well-regulated phase-out strategy to end all tobacco sales.[9]

Finally, a substantial group of smokers agree or strongly agree that "tobacco companies have done everything they can to reduce the harm caused by smoking", and Māori smokers were significantly more likely to have this view (24% compared to 18% of European/other, Table 1). This view is echoed in Year-10 students surveyed in 2008.[18] Over half of these students disagreed, or "did not know", when asked if they "*support government laws that control what tobacco companies do*", and only 38% of Māori students agreed.[18] Forty three percent of the students agreed that they "*would trust what tobacco companies say about the harmful/health effects of smoking*". Such attitudes by adult smokers and by students may reflect the lack of adequately funded and effective media campaigns that inform youth and smokers about the industry. Such campaigns elsewhere have very cost-effectively lowered the risk of youth starting to smoke.[19,20]

In conclusion, some of these data on smoker misperceptions are likely to be associated with tobacco industry messages on packaging and elsewhere. These data may therefore help the Māori Affairs Select Committee in informing their recommendations on ending the tobacco problem in this country. While we plan further analyses of these data, we think that these early findings are of immediate relevance to policymakers, especially in the context of other evidence that the tobacco industry has consistently acted to oppose all major steps to reduce the harm from smoking in New Zealand.[21]

Nick Wilson,* George Thomson, Deepa Weerasekera, Tony Blakely, Richard Edwards, Jo Peace
Department of Public Health, University of Otago, Wellington
*nick.wilson@otago.ac.nz

David Young
Cancer Council Victoria, Carlton, Victoria, Australia

Heather Gifford
Whakauae Research Services, Whanganui

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Open access: science and undeveloped countries, a reality of discrimination

The number of published articles in diverse scientific journals increases daily, with an additional increase in the number of journals with "free open access",[1] allowing a free diffusion of articles in electronic format. The mean number of citations for articles online is 157% more than the mean number of citations for offline articles,[2] probably due to availability.[2,3] In addition, online open access has had the greatest effect in developing countries rather than wealthier nations.[4]

Currently, a large number of journals require an account to access publications generated by other scientists around the world and in most cases, these accounts are restricted to those with public funds and those that supported the initial investigation.

Additionally, the increased number of online journals decreased the overall cost compared to those with solely print versions. The recent economic recession has caused some publishers to force universities and research centers in developed countries to purchase access to additional journals to gain access to journals of constant use, online access to scientific articles could overcome this and select specific journals or articles, instead of a package of needless magazines.[5]

On the other hand, it is difficult to be up to date on current research without free access to all information, and it is important to remember that some publishers have free open access specifically for developing countries, allowing researchers in developing countries to access information in a quick and timely manner for their work.

To address these problems, new ways of transferring information have been created, for example, on some servers or internet groups, when you request a paper, other investigators send you the paper in electronic format. This allows individuals at both public and private institutions, in developed and developing countries, to share articles in electronic format and enable them to conduct their research without having to pay for subscriptions, since investing in articles or magazine subscriptions decreases the overall amount of money spent on the research itself.

I believe that having to pay for access to scientific articles will not occur in the near future, creating equality at a scientific level between developed countries and those under development. In conclusion, since scientists in the poorest countries have less money to do their research and are less likely to access scientific information that would require additional fees, more articles need to be available to those in less developed countries to avoid a delay in the completion and publication of their results.

Walter Cardona-Maya
Reproduction Group, University of Antioquia
Medellín, Colombia
(wdcmaya@medicina.udea.edu.co)

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Heterophile antibodies and troponin results: implications in rural setting

Elevated troponins are the most sensitive and specific biochemical markers of myocardial damage and are the current preferred marker to detect myocardial cell injury.[1] In patients with clear-cut unstable angina, cardiac troponin measurements provide superior prognostic information over creatinine kinase (CK).[2]

It is vital to remember that raised troponins indicate the presence but not the mechanism of myocardial cell injury and besides acute ischemia there are a number of non-cardiac causes responsible for this "troponitis" syndrome.[3,4] Of importance is that a raised troponin result has been linked to poor outcomes and also seen to be an independent prognostic factor in a number of non-cardiac syndromes, in particular pulmonary embolism (PE).[5]

Interference by human anti-animal immunoglobulins in immunologic assays is referred as Heterophile Antibodies (HAb)[6–8]. It is an important consideration for medical testing laboratories as their interference causing false positive assays has been well documented.[6–14]. The prevalence of HAb in the general population has a wide range from 0.17 to 40%[10, 13-16] with various sources being proposed for formation of HAb. These include rheumatoid arthritis[11,13], exposure to microbial antigens [12,18] & animal proteins[14] and the use of mouse monoclonal antibodies in diagnostic imaging and cancer therapy.[17]

In Greek, *Hetero* means different and *Phile* means affinity. Typically, these antibodies are IgG class with an affinity for the Fc portion of the foreign antigen although there are reports of location of the epitope in the Fab region.[19,20] To further complicate matters, the binding of these antibodies is not always species-specific. This further raises the possibility that antigens from two different species can be cross-linked. It is not clinically significant but the presence of high titers of these antibodies can lead to analytical errors in commonly used "sandwich" immunoassays by cross-linking the capture and label antibodies in the absence of specific analyte.[10] Such cross-linking by HAb has been shown to lead to falsely increased CK-MB and CA125.[19–21]

HAb is a fairly general term that can be applied to two particular groups:

- **True Heterophile antibodies** are defined as antibodies produced against poorly defined antigens. These are generally weak antibodies with multispecific activities. This is important, as multispecific antibodies are weak antibodies.
- **Human anti-animal antibodies (HAAAb)** develop as a result of treatments with animal immunoglobulins are antibodies with strong avidities, produced against well-defined antigens. In addition there are situations where heterophile antibodies will coexist with specific HAAAs.[9]

Both of these antibodies can interfere with all immunological assays by similar mechanisms namely a 'bridging effect' between capture and detection antibody. This means that the false positive results are seen with both cTnI and cTnT.

Heterophile antibodies arise from the natural processes of antibody diversity that produce weak, early, multispecific antibodies against diverse antigens. This process gives rise to more than 5×10^7 different antibodies with different antigen combining sites. Although they can affect various assay formats, their main effect is on 2-site immunometric assays.[9]

These assays use at least two antibodies directed against different epitopes of an antigen; one antibody is bound (or becomes bound) to a solid-phase, while the other is in solution and tagged with a signal moiety such as I-125, enzyme, fluorophore, CLIA label, etc. Normally, antigen present in the sample "bridges" the two antibodies so that the amount of labelled antibody, which becomes bound to the solid-phase, is proportional to the antigen concentration in the sample.[9]

As a rural referral centre, which caters to a large farming population, we thought that it is important to standardise our laboratory testing. After trialling a variety of immunoassays, we found that we had the best results with the Beckman Coulter (Sydney, Australia) Access AccuTnI assay, which is a two-site immunoenzymatic "sandwich" assay. In those cases where there is a high suspicion of HAb, we use the Scantibodies Heterophilic Blocking Reagent (HBR).[22] HBR is a novel reagent, which has been specifically designed to combat the problems of heterophilic antibody interference in immunoassays. It is a unique formulation of immunoglobulins targeted specifically against heterophilic antibodies to neutralize their interference. HBR is a defined reagent with a purity >95%.

Unlike most of the non-specific "passive blockers" which are available from other suppliers (which need to be added in vast excess to ensure that heterophilic antibodies will bind to them in preference to assay-specific components), Scantibodies' HBR is an "active blocker". This formulation of immunoglobulins is targeted specifically against heterophilic antibodies and is therefore able to neutralize their interference.[23]

In the Hunter New England Health Network, Pathology New England is the only Laboratories who use the HBR assay. It would be useful to study what assays and reagents other rural centres in Australia and New Zealand use as it has strong implications in clinical decision making in those patients with positive troponins secondary to HAb.

Seshasayee Narasimhan (docsesh@gmail.com)
Department of Cardiovascular Medicine

Don Clausen
Pathology New England

Tamworth Rural Referral Hospital, Tamworth, NSW, Australia

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THE NEW ZEALAND MEDICAL JOURNAL
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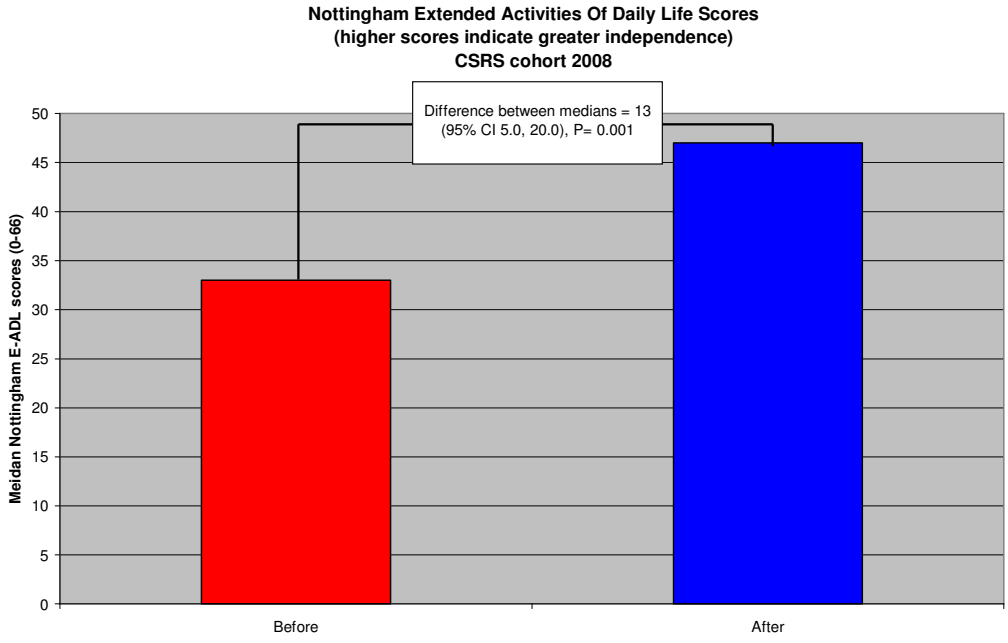
Changes to health service delivery—successes and pitfalls

The Minister of Health has spoken recently of improving health services using the expertise of front line clinical staff.[1,2] He stated that changes in health service delivery are inevitable to cope with both an increasing demand and an ageing population. He wants services that "better, sooner, more convenient and closer to home".[2] These are laudable aims, but there are caveats that the Minister should be aware of. I wish to illustrate this reporting on one such clinician led project.

Figure 1



Prior to 2001, stroke services for older people in Christchurch were not organised. However with the implementation of initially a dedicated Stroke Rehabilitation Unit (SRU)[3] in 2001 and then an Acute Stroke Unit (ASU) in 2004,[4] patients benefited. However a further important component of an overall organised stroke service is a stroke specific community based stroke rehabilitation service (CSRS).[5] The CSRS was finally established in June 2008, after a long gestation period -the first formal proposal was made in 1998.

Rehabilitation is important to maximise a person's independence. Home based (HB) rehabilitation has many features necessary for effective rehabilitation—familiar

context, relevant tasks to practise as part of their daily routines, both patient and family are readily involved, autonomy is promoted and participation in their own social networks is naturally encouraged.[6] Despite these advantages, a Day Hospital (DH) model provided 'post- hospital' stroke rehabilitation in Christchurch. There is evidence that HB rehabilitation is at least as effective as a DH model,[7] hence we wanted to change to a HB rehabilitation model.

The first six months of our CSRS are reported here. An interdisciplinary team (IDT) consisting of 10.3 FTE staff (physio [2.6], occupational [2.6], speech and language therapists [1.3], generic assistant [1.0] and an administration assistant [0.5] was developed with geriatrician oversight. The team uses a goal directed, pragmatic IDT rehabilitation approach, in the patient's own home. Patients are seen within one week of referral and family are encouraged to be involved wherever appropriate. Each applicable discipline aims to visit once weekly with relevant functional tasks practised as homework between sessions. This is crucial as intensity of practice is a cornerstone of effective rehabilitation.[8]

159 patients (mean age 77.6 years, 51% female) were seen in the first six months. Referrals came from SRU (44%), ASU (30%) and the remainder from other hospital wards and the community. Median Functional Independence Measure (FIM)[9] was 102 (IQ range 85-112) and most (83%) were residing in their own home. Time from referral until first visit was significantly shorter than a comparable DH cohort (median (days) 7 versus 18, difference between medians = 10 days (95% CI 7.0, 15.0) $P < 0.0001$). However the duration of intervention (50 versus 48 days) and the number of visits per patient (8 versus 8) were similar in both cohorts. Independence in extended activities of daily living tasks (Nottingham E-ADL[10]) improved significantly in the CSRS cohort (Figure 1).

These changes in service delivery fulfil Mr Ryall's criteria; they are clinician led, deliver better health outcomes, sooner and are certainly closer to home. I believe his drive for improved services is appropriate, but I wish to draw your readers' attention to some potential difficulties.

Firstly the time line was long - much longer than one parliamentary term! This project was first proposed 10 years before its introduction. Changing health services has been likened to using a rubber dinghy to change the course of the *Titanic* on the open sea; institutional systems and funding mechanisms have considerable inertia to overcome.

The project was thwarted several times during that period which resulted in the clinical champions feeling battle fatigue and ready to desert. It was only through the constant encouragement of colleagues and community groups which spurred us on. So my second point is that clinical champions need considerable support, both collegial and administrative.

Thirdly clinical leaders need some of their clinical time freed up so that they can devote their energies to developing the model and engaging the many stakeholders. It takes time and energy to get all key stakeholders and staff "on board".

Fourthly, seed funding is often needed to start new initiatives as well as to monitor outcomes. As with the introduction of any new medication, it is vital that changes in services are monitored to ensure that outcomes are better or comparable to existing services. Without such monitoring, we may unwittingly cause harm.

Furthermore, funding models need to change at the same time; otherwise incentives for maintaining the status quo persist.

I agree that changes in health service delivery are necessary but there is resistance at many levels. The Minister needs to be aware that they take a lot of thought, a lot of time, a lot of energy and a lot of dialogue. As the cheese makers say "Good things take time".

H Carl Hanger
Geriatrician, Older Persons Health Specialist Services,
The Princess Margaret Hospital, Canterbury District Health Board,
Christchurch
Carl.hanger@cdhb.govt.nz

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Auckland Community Laboratory contract: the most monumental disaster in health management in the past 60 years?

Recently, on television, the chairman of the Auckland District Health Board expressed surprise that things turned out as they have with the recent provider change. This seems quite naïve. It is not as if the DHBs had not been abundantly warned. There must have been many hundreds of letters, as well as the petition signed by 120,000 people. This is not to retrace the issues, which have been exhaustively covered already. Apart from one.

That is that there is a basic fault in the present system: a wide split between management and governance on the one hand, and the work face (patients and health professionals) on the other. Some split there has always been, but it has been far worse since the Rogernomics era from 1984, aggravated by what has been called the 'cult of the so-called Expert Manager'[1]: the said 'Expert' being one who lacks 'domain knowledge', i.e. a proper knowledge of what he or she is supposed to be managing.

The laboratory contract issue is only the most recent and spectacular of the problems that have occurred as a result. The health scene in this country has seen many troubles, but this must be the most monumental management disaster of the past 60 years. Can anyone suggest a worse one?

Des Gorman and John Scott have argued that medical professionals should have more part in management.[2] I agree with them, but would add that possession of a medical degree is no guarantee of expertise in managing, nor is it enough even when doctors have this expertise.

Among many others, I had a part in developing newborn services in this country especially in the period 1975 to 1985. We made little progress until the community became involved. This it did, strongly, in a crisis we had in 1980. Its part was crucial. In a different field, cervical cancer had its crisis with the Cartwright Inquiry in 1987–8, since which time women 'consumers' have felt more ownership of the services. Is it too much to suggest that one way out of the present laboratory crisis, and a way to avoid future troubles, may be to give everyone concerned more of a feeling that they have been properly involved?

Doubters might argue that it is no use dwelling on the past: nothing will change. To adapt a saying attributed to the German philosopher Hegel, the only thing to be learnt from history is that people do not learn from history. But the present crisis could serve a good purpose *if* managers learn enough from the experience to avoid problems of the kind in the future. Is this too much to hope?

Ross N Howie
Retired Paediatrician, Auckland
h.r.howie@xtra.co.nz

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Ian Graham Robinson

MB ChB, Dip Obst, FRNZCGP, DAvMed (3 November 1946 to 15 October 2009)

General practitioner Graham Robinson lost his life in mid-October, when he was tragically killed while cycling north of Auckland.

In his 20s, already a qualified and experienced pharmacist, Graham moved with his wife, Lin, and family of two small girls, to embark on medical training in Otago in 1973. By working as a pharmacist's locum during university holidays, the Robinsons made ends meet.

The family continued to grow, with the arrival of 3 more children, including twins, by the time Graham's MB ChB was finished in 1978. Graham graduated with distinction in medicine and won the Marjorie McCallum Medal. As Wanganui Hospital offered accommodation which was sufficiently big to cater for 2 adults and 5 children, Graham took up his house surgeon position there.

There were no registrars, which meant house surgeons carried heavier responsibilities. While at Wanganui, Graham gained his Diploma of Obstetrics.

His house surgeon years completed, Graham moved to Taihape to a vacant practice—one of only two practices in the town. The community action committee set up surgery for him in a house donated by a local motel. The rugby club provided couches for the waiting room. Boards were put over the bath to make an examination bed. Two years later, a Keith Hay home was trucked in, providing a new surgery.

Graham enjoyed Taihape, where he delivered around 100 babies each year and carried out much of the emergency work in the area, as local hospitals had limited facilities. In 1984 he gained his MRNZCGP.

The family moved to the North Shore as Graham joined the Mairangi Medical Centre, where he became an FRNZCGP. Described as an extraordinary family doctor, Graham will be missed by colleagues and patients. During his 23 years at the medical centre, Graham was on the Boards of Procare and Shorecare and was the Chairman of PreMec.

Around 600 mourners gathered at Graham's funeral to honour the life of a man who won friends easily through his wonderful sense of humour, kindness, and his wise and gentle manner. Testimonies from patients spoke of a man who knew how and when to reassure, and how and when to take action.

He was a fit and athletic man. As a Mount Albert Grammar School student he was the Auckland Schools' Cross Country Champion and the Intersecondary Schools Champion in the 880 yards. His best mile time was 4 minutes 04 seconds. Throughout

his life, he jogged for pleasure and fitness, becoming a cyclist in recent years when a persistent Achilles tendon problem made running difficult. A popular Rangi Rocket, he rode long distances and lapped up the companionship and the coffee which followed each ride.

In his 50s, Graham took up flying and gained his private pilot's licence, further developing a long-held interest in aviation. He was one of the country's earliest doctors to gain a Diploma of Aviation Medicine from the University of Otago. In 1995 he became an Aviation Medicine Assessor, AMA – 1, which allowed him to issue medical certificates to pilots. He was medical advisor to the Gliding Association. He was a member of the Aviation Medicine Society of New Zealand for over 20 years, many of these years on the committee and the last few of these as Treasurer.

Graham was an outstanding example of how to live life. Recent highlights included seeing the sun rise on Macchu Picchu and doing a tandem parachute jump on a day out with his family.

Above all, Graham was a family man. He was loved by his best friend Lin, his wife of 40 years, and his 5 adult children, who speak of the way that Graham was always there for them. His 7 grandchildren adored him. Deepest sympathy is extended to Lin, Nicki, Tracey, Brenda, Bridget, and Brett and their families.

Written by Diana Wood, Public Relations Consultant, Hamilton (Diana@morethanwords.co.nz).

This obituary was written not in the line of my work, but as a tribute to my good friend, Graham. Significant help was provided by Lin Robinson, Graham's widow, and Dr John Faris, also a close friend of Graham.

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Heart Foundation GRANTS AWARDED NOVEMBER 2009

At the November 2009 meeting of the Scientific Advisory Group of the National Heart Foundation, a total of 16 limited budget grants were awarded. The awards included 10 Small Project Grants, 3 Grants-in-Aid and 3 Travel Grants.

SMALL PROJECT GRANTS

Drs Alexandra Chisholm & Rachel Brown

Department of Human Nutrition, University of Otago, Dunedin

Nuts for LIFE (Lipids, InFlammatory, Endothelial).

$14,750 for 15 months

Ms Louise Foley

Clinical Trials Research Unit, University of Auckland

Exploring sedentary behaviour in New Zealand children and young people.

$12,652 for 3 months

Dr Andrew Kerr

Cardiology Department, Middlemore Hospital, Auckland

Personalised electronic early warning system to reduce post ACS treatment gaps: a feasibility study.

$14,930 for 1 year

Dr Jun Lu

Faculty of Health and Environmental Sciences, AUT University

Detecting heart failure in obesity.

$15,000 for 1 year

Ms Enid Dorey

Clinical Trials Research Unit, University of Auckland

Patient perceptions around alternative options for the delivery of cardiac rehabilitation.

$14,659 for 10 months

Dr Simon Green

Department of Physiology, University of Otago, Dunedin

Exercise and muscle blood flow in peripheral arterial disease.

$12,540 for 1 year

Professor Diana Lennon

Department of Paediatrics: Child & Youth Health, University of Auckland

ARF/RHD Burden of Disease in Samoa to Aid Control.

$14,700 for 3 months

Dr Ralph Maddison

Clinical Trials Research Unit, University of Auckland

Fit2Quit: Exercise to enhance smoking cessation outcomes.

$15,000 for 2 years

Dr Ria Schroder

National Addiction Centre, University of Otago, Dunedin

Abstinence vs moderation in obesity treatment: client perceptions of what works.

$14,520 for 1 year

Ms Maria Turley

Clinical Trials Research Unit, University of Auckland

Do trends in food prices support healthy eating?

$14,422 for 6 months

GRANTS-IN-AID

Dr Samuel Lucas

Department of Physiology, University of Otago, Dunedin

Travel and study costs for work at The Peninsula Sleep Laboratory, Sydney, Australia.

$10,000

Dr Lauretta Muir

Centre for Postgraduate Nursing Studies, University of Otago, Christchurch

Reducing the burden of familial hyper-cholesterolaemia: developing a New Zealand model of care.

$14,070

Associate Professor Sally McCormick

Department of Biochemistry, University of Otago, Dunedin

Purchase of a High Performance Liquid Chromatography system for shared use.

$15,000

TRAVEL GRANTS

Dr Alexandra Chisholm

Department of Human Nutrition, University of Otago, Dunedin

78th European Atherosclerosis Society Congress, Germany; 28th International Symposium on Diabetes & Nutrition. Norway; Heart UK 24th Annual Conference, Scotland.

Dr Nicola Scott

Department of Medicine, University of Otago, Christchurch

14th International Congress of Endocrinology 2010, Kyoto, Japan.

Dr Natalie Walker

Clinical Trials Research Unit, University of Auckland

Society for Research into Nicotine and Tobacco Annual General Meeting 2010, Baltimore, USA.

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University of Otago Faculty of Medicine

Postgraduate Scholarship in Obstetrics and Gynaecology

The above Scholarship is open to medical graduates who will normally be Registrars undertaking the Royal Australian and New Zealand College of Obstetrics and Gynaecology (RANZCOG) Integrated Training Programme, or are Members or Fellows of the College who intend to enrol for a research degree, e.g. Master of Medical Science (MMedSc) or PhD.

The Scholarship is $34,000 per annum for one year commencing 1 February 2010.

Further details are available from:

Debbie Moore
Department of Women's and Children's Health
Dunedin School of Medicine
P O Box 913
Dunedin 9054

Email: debbie.moore@otago.ac.nz

Applications close on 11 December 2009

THE NEW ZEALAND MEDICAL JOURNAL
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Erratum

Evan Lee, Rhiannon Braund, June Tordoff. Examining the first year of Medicines Use Review services provided by pharmacists in New Zealand: 2008. N Z Med J. 24-Apr-2009 - Vol 122 No 1293. (http://www.nzma.org.nz/journal/122-1293/3560 and http://www.nzma.org.nz/journal/122-1293/3560/content.pdf)

Pharmaceutical Review Services (PRS) were introduced in 1998 *(not 1990 as stated when published)*. Thus they were the second generation of pharmacist-led medication reviews in New Zealand following Comprehensive Pharmaceutical Care (CPC) which was introduced in 1996.

Please refer to the above URLs to view the corrected copy of the article.

THE NEW ZEALAND MEDICAL JOURNAL
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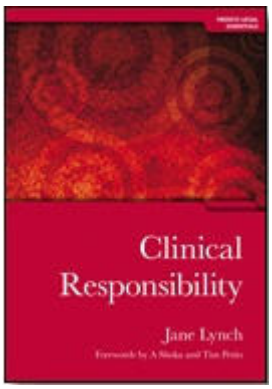
Clinical Responsibility

Jane Lynch. Published by Radcliffe Publishing, 2009. ISBN 9781846192234. Contains 228 pages. Price £22.99

Reading the title to this book may make your heart sink. However, I read it over a series of night shifts, and was pleasantly surprised. This is an easy read, and informative. Not a common combination. And the chapter on Harold Shipman was horrifyingly gripping!



This book is written for all health professionals, not just doctors. It is written in plain English and seeks to demystify complex and sometimes frightening legal processes. I believe it achieves this goal.

It is very readable, and makes a potentially dry subject interesting. The case histories and workplace examples bring this subject within the realm of everyday experience.

The glossary also acts as an aide memoir for the Latin-based legal terminology.

Clinical Responsibility is written predominantly for a UK audience. It is based on UK law and the National Health Service (NHS). As a UK trainee I found it explained UK law clearly, in easy to understand terms. I suspect many of the broader issues regarding accountability, responsibility, negligence and clinical governance apply equally in New Zealand. However, the legal fine print is likely to differ.

In some ways this book confirms our worst nightmares regarding legal proceedings. In some cases you are indeed “damned if you do and damned if you don’t”! This particularly applies to “protocols, policies, procedures, and guidelines”. However, there is a comfort in knowing that the law recognises that a bad outcome for a patient is not necessarily due to negligence, and therefore not open to litigation.

This is an interesting read for all health professionals. I would particularly urge anyone who is currently working or planning to work in the UK healthcare system to read this book, and check their personal indemnity!

Rhian S Finn (MBChB, MRCP)
Respiratory Medicine
Christchurch Hospital

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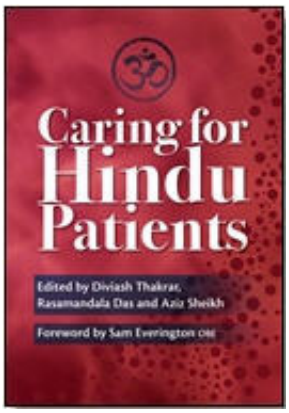
Caring for Hindu Patients

D Thakrar, R Das, & A Sheikh, eds. Published by Radcliffe Publishing, 2008.
ISBN 9781857755985. Contains $142 pages. Price £24.95

This beautifully presented book provides valuable information for health professionals who want an in-depth understanding of the Hindu culture and traditions and its impact on health. Whilst Hinduism is a diverse and complex religion, the authors have presented information in an easy to comprehend format.



The book is divided into two parts. The first part deals with the origins of Hindu tradition and spiritual practice. It then goes on to explore the social and demographic characteristics of the British Hindu population. The second part deals directly with issues which relate to health care. This part focuses the health care needs at each stage of life from conception through to death and bereavement. It also deals with important issues related to contraception, infertility, abortion, adoption, and organ donation. The appendix provides a wealth of information on important festivals, dietary advice, and Hindu resources on the World Wide Web.

This comprehensive and easy to read book will provide the reader with an insight into the complexities of dealing with Hindu patients. It also provides advice that helps clinicians to develop a better cultural awareness of the needs of Hindu patients.

This book is highly recommended to all health professionals who work in a diverse cultural environment which involves the care of Hindu patients.

Sunil Kumar
Consultant Physician and Rheumatologist
Middlemore Hospital, South Auckland