## CONTENTS

### This Issue in the Journal

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

### Editorials

### Original Articles

## Medicolegal

## Obituary

## Notices

## Book Reviews

## This Issue in the Journal

### Insulin resistance in a rural Maori community

D Tipene-Leach, H Pahau, N Joseph, K Coppell, K McAuley, C Booker, S Williams, J Mann

Diabetes is known to be common among New Zealand Maori. This study shows that nearly half of the adult population surveyed on the East Coast of the North Island have insulin resistance. Insulin resistance is believed to be the earliest identifiable phase of type 2 diabetes and a major risk factor for heart disease. The fact that insulin resistance is particularly prevalent in young people endorses the need for early lifestyle intervention.

### Risk factors for type 2 diabetes in postmenopausal New Zealand women: a cross-sectional study

S Rose, B Lawton, A Dowell, A Fenton

This paper investigates risk factors associated with type 2 diabetes in 3377 postmenopausal New Zealand (NZ) women. A diabetes risk analysis tool revealed that over half were at 'high risk' for developing diabetes in the future. Over half of this predominantly NZ European sample had modifiable risk factors including overweight or obesity, and physical inactivity. These data highlight the urgent need to identify those with modifiable risk factors before they develop type 2 diabetes in order to appropriately target lifestyle intervention programs.

### Body size, body composition, and fat distribution: a comparison of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities

E Rush, L Plank, V Chandu, M Laulu, D Simmons, B Swinburn, C Yajnik

The World Health Organization (WHO) threshold for classification of obesity is a BMI (weight in kilograms divided by square of height in metres) of 30. This study of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities showed that, for the same BMI, Pacific Island men had markedly less fat and more muscle while Asian Indian had more fat and less muscle than European men. A BMI of 30 in the European men was equivalent to a BMI of 33 in Pacific Island and 25 in Asian Indian men, respectively, who had relatively more abdominal fat. Universal BMI thresholds are not appropriate for comparison of obesity prevalence between these ethnic groups.

### Obesity and health-related quality of life: results from a weight loss trial
C Ni Mhurchu, D Bennett, R Lin, M Hackett, A Jull, A Rodgers

The New Zealand population is becoming increasingly overweight and it is well known that this is an important cause of disease and death. This study measured health-related quality of life among 250 participants in a weight loss trial and compared results with the general population. Findings showed that overweight and obese adults experience significantly impaired quality of life compared to population norms, particularly in the physical function domains. Small reductions in body weight did not improve quality of life in this substantially overweight population. The study confirms that obesity has a significant negative impact on the health-related quality of life of New Zealand adults.

### Gastric bypass surgery for severe obesity: what can be achieved?
M He, R Stubbs

The results of gastric bypass surgery performed at the Wakefield Clinic (Wellington, New Zealand) in over 300 severely obese individuals are presented. They show a mean percentage of excess weight loss of around 70% within 2 years of surgery with good maintenance of weight loss out to 5 years. In the majority of those with serious comorbidities including type 2 diabetes, hypertension, dyslipidaemia, asthma, and obstructive sleep apnoea, these were either resolved or substantially improved following surgery.

### Cost-effectiveness of physical activity counselling in general practice
R Elley, N Kerse, B Arroll, B Swinburn, T Ashton, E Robinson

Physical activity counselling in primary healthcare using the Green Prescription is not only effective but also cost-effective. A randomised controlled trial in the Waikato showed that brief advice (to become more active) from a general practitioner or practice nurse, accompanied by a Green Prescription, increased exercise levels over 12 months. And with increases in exercise levels, savings in healthcare are likely in the long term—because active people have lower rates of heart disease, diabetes, stroke, osteoporosis, and other diseases.

### Diabetes in children and young adults in Waikato Province, New Zealand: outcomes of care
A Scott, S Whitcombe, D Bouchier, P Dunn

Diabetes remains an important cause of morbidity and mortality among young people. Despite improvements in technology, maintenance of good glycaemic control is hard to achieve particularly during the teenage years. Young people with diabetes in Waikato have poor glycaemic control (HbA1c 9.2%) and after 10 years over a quarter have evidence of early kidney and eye complications. These results are similar to published European studies but little is known about the outcomes of care in the rest of New Zealand.

**Under-reporting of diabetes on death certificates among a population with diabetes in Otago Province, New Zealand**

K Coppell, K McBride, S Williams

In New Zealand, little information is known about the under-reporting of diabetes on death certificates—a recognised problem worldwide. This study (using data from the Otago Diabetes Register) found that diabetes was not mentioned on the death certificates of 45% of 508 patients known to have diabetes. If the impact of the diabetes epidemic on mortality is to be monitored appropriately in New Zealand, attention needs to be given to improving the completion of death certificates, including always recording diabetes when it is present, irrespective of whether it is considered to be the underlying or a contributing cause of death.

**Indigenous disparities in disease-specific mortality, a cross-country comparison: New Zealand, Australia, Canada, and the United States**

D Bramley, P Hebert, R Jackson, M Chassin

Indigenous peoples suffer from poor health and lower life expectancy. This study compares indigenous mortality with non-indigenous mortality in New Zealand, Australia, Canada, and the United States. Findings show that New Zealand Maori and Australian Aboriginals and Torres Strait Islander suffer from the highest levels of mortality and also the largest levels of inequity when compared to their non-indigenous population. Diabetes mortality is especially high for all indigenous peoples included in this study. Action is required to address indigenous health disparities and to improve the quality of indigenous mortality data.

## Obesity and diabetes: questions remain but action should not be delayed

Jim Mann, Kirsten McAuley, Rachael Taylor

That obesity and its comorbidities, especially type 2 diabetes (T2DM), have reached epidemic proportions in New Zealand and globally, has been established beyond doubt. This issue of the *Journal* includes papers that highlight some of the many outstanding questions that must be answered if there is to be any serious hope of stemming the tide of these epidemic diseases of the 21st Century.

Rush and colleagues[1] have carried out studies of body composition in young New Zealand European, Pacific Island, and Asian Indian men and have confirmed both that the relationship between percentage body fat and body mass index (BMI) is different among the three groups (possibly due to differences in muscularity), and that Asian Indians have more abdominal fat that the other ethnic groups. The latter observation might explain the high rates of cardiovascular disease amongst people of Indian descent.

The Rush study confirms earlier observations that people of Pacific Island descent have a lower percentage body fat than Europeans for any given BMI and that the reverse applies for Indians. Hence, the suggestion that different BMI cut-offs for the definitions of overweight and obesity from those applied to European populations, should apply to Pacific Island and Indian populations.

While this may be appropriate if one wishes to compare body composition in different population groups, it is important to remember the original, and still clinically the most important, reason for the introduction of BMI cut-offs to define the categories of overweight and obesity—the identification of individuals who are at increased risk of the comorbidities associated with excess adiposity.

The cut-offs were principally determined from prospective epidemiological studies in predominantly European populations.[2] Given that for the relatively common comorbidities, there is a gradient of risk with increasing adiposity, the levels used to define overweight and obesity are inevitably somewhat arbitrary. Were it true that a given level of adiposity was associated with a comparable level of risk in all populations, it would be appropriate to adjust the categories according to degree of adiposity. However as there may well be inherent differences in risk in different racial groups it is potentially misleading, at least from a clinical point of view, to suggest the redistribution of BMI cut-offs on the basis of percentage or amount of body fat as measured by dual-energy X-ray absorptiometry.

Indeed, some evidence suggests that susceptibility amongst different racial groups to adverse health effects varies—even if the degree of adiposity is similar. A pilot study from Dunedin has suggested that for comparable levels of adiposity Maori have a greater degree of insulin resistance than New Zealanders of European descent.[3] If confirmed (a large study is currently underway), this suggests that it would be inappropriate to recommend higher BMI cut-offs for the definitions of overweight and

obesity in Maori, despite the fact that for any given BMI Maori may have greater lean body mass and less fat mass than Europeans.

While it might be inappropriate to use higher BMI cut-offs for Maori, it may well be appropriate to suggest lower cut-offs for Asian Indians given their well recognised high cardiovascular risk at lower levels of BMI. In general, Asian Indians have greater levels of total and central fat than Europeans for a similar BMI.[2] Ideally, this issue should be resolved by prospective observations on non-European populations for whom body composition data are available. However as such data are unlikely to become available in the near future, further body composition studies of Asian and Pacific Island populations should be undertaken in conjunction with measurements of associated comorbidities, especially those related to abnormal carbohydrate metabolism, which are particularly relevant in New Zealand.

Thus, it seems appropriate to retain current cut-offs at present, while acknowledging that Asian Indians may be at increased risk of cardiovascular disease even if their BMIs are within the currently defined normal range.

Another issue relating to measurement is highlighted in the paper by Hohepa and colleagues.[4] There is no doubt that physical inactivity is a major contributing factor to the obesity epidemic, and that increasing physical activity facilitates weight loss and weight maintenance and increases insulin sensitivity.[5] Indeed, promotion of physical activity is a pivotal component of public health messages as well as management of overweight and obese individuals.

The New Zealand Government intends to expend substantial sums of money on a national programme aimed at increasing physical activity in schools. However, without appropriate instruments for measuring activity, it will not be possible to accurately relate trends in obesity with trends in physical activity, and evaluation of such interventions will be impossible. Thus, the call made by Hohepa and colleagues for accurate measurement tools is strongly endorsed. There is also a need to establish with greater certainty the level and type of physical activity, which is most likely to facilitate reduction of overweight and obesity and their comorbidities.

Current advice in New Zealand centres around the recommendation to have at least 30 minutes of physical activity most days of the week without any clear indication of the level of activity. There has also been the suggestion that this amount of activity can be achieved by having several shorter periods of activity ('snacktivity') which may be added together to achieve the required amount.

While this may be a useful approach to initiate physical activity in previously inactive individuals, and while any level of activity is undoubtedly better than none, there is evidence that longer and more intensive levels of activity than currently recommended are required to improve insulin sensitivity in insulin-resistant individuals.[6] Furthermore, a combination of endurance and resistance training may be preferable to larger amounts of only one type of activity.[7]

Until further evidence is available regarding optimal type and amount of exercise, it may be appropriate to indicate (in both public health messages as well as in advice to individual patients) that current recommendations represent minimal requirements and that whenever possible the level of activity should be sufficient to raise the pulse rate.

Ni Mhurchu and colleagues draw attention to a somewhat neglected set of comorbidities associated with obesity.[8] They report on the impaired health-related quality of life (HRQOL) experienced by overweight and obese individuals. Of particular importance is the fact that HRQOL measures did not improve appreciably with weight loss. However only 23 of the 250 participants lost more than 5% of their baseline weight. Thus the overall finding is perhaps not surprising. It seems likely, therefore, that a greater than 5% weight loss is necessary both to reduce the risk of progression from impaired glucose tolerance (IGT) to T2DM, and probably also other comorbidities of obesity, as well as improve quality of life.

Much attention has been focussed on the need to prevent and treat obesity in childhood, and indeed this must be one of the greatest health priorities in New Zealand. However the paper by Rose and colleagues reminds us that adults too require attention.[9] They found that more than half of over 3,000 women screened are at 'high risk' of developing T2DM. Such statistics along with observations such as those by Tipene-Leach and colleagues[10] showing that a comparable proportion of adult Maori living in a rural environment have T2DM, impaired fasting glucose, IGT, or insulin resistance endorse the New Zealand guidelines for the detection and management of cardiovascular risk published in 2003.[11] All adult New Zealanders should be screened for T2DM and pre-diabetic states by age 45 (male) and 55 (female), with high-risk individuals screened earlier.

With current health care costs relating to T2DM approaching NZ$400 million and expected to rise to more than NZ$1,000 million by 2021, and evidence showing that appreciable risk reduction by lifestyle modification is achievable, urgent implementation of lifestyle modification programmes is essential.

The questions posed by the papers presented here need not delay the implementation of existing knowledge. Perhaps the greatest issue remaining to be solved is how to persuade at-risk individuals and populations to make the necessary changes.

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## Preventing diabetes—time is running out

Robert Scragg

These days we are continually bombarded in the popular press about the current obesity epidemic and the resulting tidal wave of diabetes expected soon to cover Aotearoa (New Zealand). Indeed, despite predictions of an 80% increase in the number of people with diabetes during the 15 year period from 1996 to 2011,[1] a degree of apathy and indifference appears to prevail among influential circles in the Ministry of Health and District Health Boards, creating the impression that these organs of Government believe there is plenty of time left to put diabetes preventive strategies in place and that urgent measures are not required.

The article by Tipene-Leach and colleagues,[2] in this issue of the *Journal*, comes as a wake up call—to everyone—that time is running out. The very high prevalences of insulin resistance among Maori from the east coast north of Gisborne, particularly in young adults below 40 years of age, indicate that the main pathophysiological precursor of type 2 diabetes is already well-established in our midst.

The study is the first within New Zealand to document the prevalence of insulin resistance in a community sample, using the authors own method based on fasting insulin and triglycerides, which has higher sensitivity and specificity compared with other population measures of insulin resistance.[3] Half (51%) of participants aged 25–39 years in this survey had insulin resistance (including diabetes).

As a check, the authors calculated the prevalence of the metabolic syndrome in their sample using the United States' (US) Adult Treatment Panel III definition;[4] which was 34% in the 25–39 year age group, much higher than 12–14% reported for 30–39 year old participants in the 3rd National Health and Nutrition Examination Survey from the US.[5]

Can the results from this East Coast study be applied to Maori across the whole of New Zealand? The relatively small sample size (n=247) means that confidence intervals (and the degree of uncertainty) around prevalence estimates are quite wide. Furthermore, the low response rate may have led to selection bias, but given that responders are usually healthier than non-responders, the survey may have actually under-estimated the prevalence of insulin resistance.

Indeed, the prevalence of obesity in this study (67% with body mass index (BMI)≥30) is higher than that reported for Maori in the 2002-2003 National Health Survey (about 30% with BMI≥32).[6]

If obesity is the only driver of insulin resistance, then it is quite likely that the prevalence of insulin resistance is lower among the wider Maori community than reported in this paper. A south Auckland cross-sectional survey has previously reported higher insulin levels in Maori (and Pacific) people, compared with European, which are entirely due to ethnic differences in BMI.[7] In contrast, the Workforce Survey showed that the diabetes prevalence in Maori remained elevated after adjusting for BMI.[8] So it is possible the very high insulin resistance prevalences

reported in the study by Tipene-Leach and colleagues may not be due exclusively to the very high obesity levels in their study sample.

Other possible lifestyle factors contributing to the high prevalence of insulin resistance among Maori in this survey include: a diet high in animal fats, a risk factor for diabetes[9] which is increased in Maori (and Pacific people) compared with Europeans ;[10] and decreased physical activity—although nationally, Maori are more likely to do regular physical activity than other ethnic groups.[6]

As the study authors rightly conclude, a national survey of diabetes and insulin resistance is urgently required to confirm whether the very high prevalences of insulin resistance reported in their study occur more widely across New Zealand in Maori and in others who are also at high risk of diabetes such as the Pacific and Asian communities.

If very high prevalences of insulin resistance do occur in these ethnic groups, then the Ministry of Health and District Health Boards have a huge problem on their hands, with much bigger consequences than those they are trying to prevent via the multi-million dollar meningococcal vaccination campaign currently underway in the North Island.

The Ministry of Health is to be lauded for driving through its 'Get Checked' programme to improve the clinical management of diabetes by providing free annual medical checks to diabetes patients. It now needs to show the same conviction (and funding) towards implementing programmes for preventing diabetes.

The recent PricewaterhouseCoopers report shows that the health cost savings from a diabetes prevention programme are likely to be greater than programme itself.[11] While there has been some investment by the Ministry of Health and District Health Boards into the development of diabetes prevention programmes, it is miniscule compared to funding of prevention programmes for other diseases, such as meningococcal meningitis and cancer screening.

The high levels of insulin resistance among young adults reported in the article by Tipene-Leach and colleagues indicates there is little time left to implement preventive strategies on a national basis. Continuing failure by the Ministry of Health to commit the funds required now for diabetes prevention will result in much greater health costs downstream, than currently, and many more people will suffer with diabetes.

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## Bariatric surgery: folly or the future?

Iain Martin

Surgery for morbid obesity (bariatric surgery) has had a long and painful birth and childhood; has it finally come of age? It is more than 50 years since DeWind and Payne introduced the jejuno-ileal bypass.[1] Whilst the procedure produced reasonable weight loss for the majority of patients, severe and often life-threatening complications coloured the views of many regarding surgery for morbid obesity for the next 4 decades.

With the rapidly increasing burden of obesity, improved surgical techniques, and the introduction of laparoscopic surgery, surgery for morbid obesity has become a 'growth industry'; in 2004, it is estimated that more than 140,000 patients will undergo such surgery in the United States (US).

Surgery for weight loss involves two physical mechanisms and probably several endocrine mechanisms. The two physical mechanisms are either gastric restriction or malabsorption, or (as in the case of gastric bypass) a combination of the two. The endocrine mechanisms are more poorly understood but there is good evidence that the mechanism of actions of such surgery are far more than just a pure mechanical restriction of calorie intake or absorption. The effect of gastric bypass on the hunger-inducing hormone ghrelin has been well described,[2] and it is likely that other gut-brain hormonal mechanisms are altered by bariatric surgery.

The data published in this issue of the *Journal* [He M, Stubbs RS. Gastric bypass surgery for severe obesity: what can be achieved?. N Z Med J. 2004;117(1207). URL: http://www.nzma.org.nz/journal/117-1207/1207] demonstrate that in a carefully selected series of patients treated by an experienced team the results of open Roux-y gastric bypass can be excellent. The results of this series are very similar to those contained within a recent meta-analysis of published data.[3]

There is now little doubt that bariatric surgery can result in improvements in quality of life and startling improvements in obesity related comorbidities, especially type 2 diabetes; the vast majority of patients with insulin resistance or type 2 diabetes will be rendered normoglycaemic by such surgery. For the patient and the surgical team, there is no more satisfying outcome that for a patient on huge quantities of insulin being rendered euglycaemic. The effects on comorbidities seem generally to be robust and sustained although there is some evidence that the effect upon hypertension may decrease with time.[4]

Even in the best series there is still morbidity (some of it significant) and occasional mortality associated with bariatric surgery. Most specialist bariatric surgeons would quote a risk of perioperative death of less than 0.5%; most of the deaths arising from cardiac or thromboembolic causes. A recent large overview series from the US would suggest that the 30-day mortality could be as high as 1.9%.[5]

In this series of patients, 81% of the deaths occurred in patients operated upon in the surgeon's first 19 cases, emphasising that this is technically demanding surgery requiring a skilled and experienced surgeon and team for optimal outcomes.

Bariatric surgery requires commitment from both the surgical team and the patient. Only a minority of patients who are morbidly obese would consider such an approach; and of these, not all would be suitable. Thus, there is little doubt that good outcomes require a team approach and very careful patient selection.

Whilst there is ample case series data to quantify some of the effects of bariatric surgery, there are few examples of well-controlled trials looking at the full range of outcome data including quality of life and health economic data. It is important, therefore, that such data is generated so that the relative merit of this intervention can be appropriately and fairly judged against the multitude of other demands upon the healthcare dollar.

Should such procedures be available to patients in New Zealand through the public system? The answer is a qualified 'yes'. It must be recognised that surgery is not going to solve the problem of the rapid rise in the levels of severe and morbid obesity, and indeed conventional healthcare approaches alone cannot deal with the problem. Society as a whole must adopt and implement strategies that deal with the fundamental issues behind the growth in obesity. These must occur at all levels including education, legislation, and perhaps fiscal measures.

Whatever these measures are, there are currently significant numbers of patients who could potentially benefit from such surgery. We have a treatment, albeit not perfect, which can (in carefully selected and informed patients) make significant improvement in their health status. Although the evidence supporting the use of bariatric surgery is substantial, much of it is poor in quality and there is a dearth of well-conducted trials.

Perhaps New Zealand should grasp this opportunity and instead of just introducing such surgery, do what should be done with all new treatments and carry out a well-conducted nationwide study to gather a full range of outcome data. The outcomes from such a study would not only benefit New Zealand but the wider international community, and if successful, it would set a fantastic precedent for the introduction of other procedures into the publicly funded healthcare system.

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# Insulin resistance in a rural Maori community

David Tipene-Leach, Helen Pahau, Nathan Joseph, Kirsten Coppell, Kirsten McAuley, Chris Booker, Sheila Williams, Jim Mann

## Abstract

**Aim** To determine the prevalence of insulin resistance, impaired fasting glycaemia, impaired glucose tolerance, and diabetes mellitus in a rural Maori community, and to compare different methods for identifying individuals with insulin resistance.

**Methods** 589 randomly selected individuals from the Ngati Porou Hauora Register aged 25 years and over and resident on New Zealand's East Coast north of Gisborne were invited to participate in the study. A questionnaire was administered, anthropometric measures made, and blood samples taken for an oral glucose tolerance test and biochemical analysis. Impaired fasting glycaemia, impaired glucose tolerance, and diabetes mellitus were defined according to World Health Organization (WHO) diagnostic criteria, and among those persons with normal glucose tolerance, insulin resistance was calculated according to the McAuley formula and three other recognised methods for calculating insulin sensitivity.

**Results** The overall age-standardised prevalence of diabetes (both known and newly diagnosed) was 10.6% and the age-standardised prevalence of insulin resistance was 37.0%. Age-specific diabetes rates were high among the older age groups, peaking at 34.1% for 60–69 year olds, whereas age-specific insulin resistance rates were high among the young age groups with the highest rate (44.3%) occurring among 30–39 year olds. Persons identifying as insulin-resistant reported higher rates of gout and family history of diabetes—and were found to have a higher waist circumference, blood pressure, and lower high-density lipoprotein (HDL) cholesterol than those without a glucose metabolism disorder.

**Conclusion** Diabetes is a common disorder among this population, but insulin resistance is even more prevalent, especially among young age groups. This is considerable cause for concern given that insulin resistance is believed to be the underlying cause of most cases of type 2 diabetes mellitus, and is confirmed by these data to be associated with a high degree of cardiovascular risk.

The prevalence of diabetes is increasing worldwide.[1] In New Zealand, only limited prevalence data are available, but evidence suggests that this increase is also occuring.[2–9] The most recent New Zealand Health Survey (NZHS) found the prevalence of self-reported diabetes for people aged over 45 years to be 8.1% for females and 10.0% for males.[6] Prevalence surveys have consistently shown diabetes (both known and newly diagnosed) to be more common among Maori compared with New Zealanders of European descent. Most recently, in the NZHS self-reported diabetes prevalence among Maori aged over 45 years was 21.4% and 13.0% for males and females, respectively—compared with 8.6% and 7.5% for non-Maori males and females, respectively.[6]

Previously, Simmons et al[8] found the prevalence of known diabetes mellitus in South Auckland was 6.9% among Maori compared with 2.8% among Europeans—and in the New Zealand Multiracial Workforce Survey, the prevalence of known diabetes mellitus was 5.3% among Maori compared with 1.1% among Europeans.[7]

In New Zealand, information about the prevalence of impaired glucose tolerance (IGT) and impaired fasting glycaemia (IFG) is limited[2,3,7,9]. Even less is known for any population regarding the prevalence of insulin resistance, a condition generally present prior to the development of IGT and IFG, and a major risk factor for cardiovascular disease, but it has been estimated that approximately 25% of people of European descent have insulin resistance.[10]

While there is no information on prevalence of insulin resistance in New Zealand, Simmons et al[9] found that, compared with Europeans, Maori and Pacific people have poorer insulin sensitivity—when applying the Homeostasis Model Assessment (HOMA) of fasting glucose and insulin as a proxy measure of insulin resistance. This finding was attributed to the high rates of obesity among Maori and Pacific people, rather than of inherent insulin resistance among people of Polynesian descent.

Trials have demonstrated that progression from IGT to type 2 diabetes can be halted through lifestyle changes,[11–13] but approximately 40% of those people still develop type 2 diabetes despite lifestyle intervention. This may be due to lack of compliance, but also likely to be due to the considerable beta-cell dysfunction already present in those with IGT. Thus, lifestyle intervention among those with insulin resistance, but normal glucose tolerance, may be a more effective approach to preventing or delaying the onset of type 2 diabetes, as well as reducing cardiovascular risk.

However, people with poor insulin sensitivity need to be easily identified, and this study examines different methods to achieve this goal in the clinical setting. A Maori Primary Health Organisation (PHO), Ngati Porou Hauora, has initiated a 2-year community lifestyle intervention programme (the Ngati and Healthy Programme) aimed at reducing the prevalence of insulin resistance among a predominantly Maori community living along the rural East Coast area, north of Gisborne, in the North Island of New Zealand. The outcome of the intervention will be assessed by pre- and post-intervention prevalence surveys of insulin resistance, as well as IFG, IGT, and diabetes. This paper presents results of the pre-intervention prevalence survey.

## Methods

This study was based on the sparsely populated East Coast of New Zealand, among communities from Tolaga Bay (50 km north of Gisborne) to Potaka (near Te Araroa). (See the map of the East Coast region; Figure 1.)

The East Coast region has a population of approximately 6000 people. The main types of employment are forestry and farming. Ngati Porou Hauora has over 13,500 enrolled patients in Gisborne and the East Coast, and provides comprehensive services to the East Coast communities through six community clinics and is the only primary care provider in this rural region.

The Ngati Porou Hauora East Coast Enrolled Patient Register was used to obtain a random sample stratified by sex, age group, and ethnicity. Ethical approval was obtained from the Tairawhiti Ethics Committee in March 2003 and the study took place throughout May to December 2003.

The Project Co-ordinator, and Ngati Porou Hauora rural health nurses and kaiawhina, (community health workers) invited selected individuals to participate in the survey by letter, and if necessary by phone and home visit (up to three visits in some cases).

**Figure 1: The survey was based on the East Coast region of New Zealand's North Island—from Tolaga Bay (50 km north of Gisborne) to Potaka (near Te Araroa)**



THE EAST COAST

- Potaka
- Te Puia
- Tolaga Bay
- Gisborne

Of the 741 individuals selected to participate in the study, two individuals were excluded because they had a terminal illness or died, and 150 were unable to be contacted because they had moved away from the East Coast study area. Thus, 589 individuals received an invitation to participate in the study.

At clinics undertaken at four different sites in the East Coast region, a questionnaire including demographic information, relevant medical history, and exercise and dietary history was administered. Height, weight, and waist circumference (midpoint between the anterior superior iliac crest and the lowest rib) were measured, body mass index (BMI) calculated, and blood pressure recorded (after 10 minutes of rest using random zero sphygmomanometers).

Duplicate measures were taken for each of the anthropometric measures, and the average of the two measures used in the analysis. A 75 g oral glucose tolerance test (OGTT) was performed with glucose and insulin measured at 0 and 120 minutes post-glucose load. Participants with documented diabetes did not have an OGTT. Blood was also taken for fasting lipids. All samples were spun and separated after collection. The plasma insulin samples were frozen and transported to Gisborne Laboratory in a mobile freezer (approximately -15°C) either the same day of collection or the following day.

Blood samples were packaged with Bio-freeze Blue Ice bottles to keep the samples at approximately -15°C, and sent immediately to Canterbury Health Laboratory (Christchurch, New Zealand), where they were processed. All other samples were stored in polystyrene boxes for transportation the same or the following day, and were processed on arrival at Gisborne Hospital's Laboratory (New Zealand).

Plasma glucose, total cholesterol, and triglycerides were measured using an enzymatic colorimetric method (Ortho-Clinical Diagnostic reagents). HDL cholesterol was measured using the direct magnetic method. In accordance with the Royal College of Pathologists of Australasia Quality Assurance Programme, coefficients of variation were 2.2% for glucose, 3.7% for total cholesterol, 6% for HDL, and 3.7% for triglycerides.

Canterbury Health Laboratory, using a Roche Elecsys 2010 automated analyzer with polyethylene glycol to remove antibodies, measured plasma insulin after extraction. The assay detection limit is 0.4 mIU/L. The intra-assay coefficient of variation was 6%.

IFG, IGT, and diabetes were defined according to WHO diagnostic criteria.[14] Insulin resistance was predicted using the McAuley formula based on fasting insulin, triglycerides and BMI,[15]—where predicted insulin sensitivity was expressed as exponent (3.29-0.25ln[fasting insulin]-0.22ln[body mass index]-0.28 ln[fasting triglycerides]). Normoglycaemic individuals with calculated values = 6.3 M• mU-1• l-1 were defined as insulin resistant. As there is no internationally agreed simple method for predicting insulin sensitivity, insulin resistance was also estimated using three other methods: the Homeostasis Model Assessment, HOMA Calculator computer model (version 2.1, 2004),[16] based on fasting insulin and fasting glucose, the National Education Program (NCEP) Adult Treatment Panel (ATP III) definition,[17] which uses a set of clinical criteria (based on blood pressure, waist circumference, triglycerides, HDL, and fasting glucose) and an insulin sensitivity index (based on the average of a fasting and 2-hour glucose, and the average of fasting and 2-hour insulin.)[18]

The ATP III criteria were applied to all study participants, while HOMA 2.1 and ISI0,120 calculations excluded known diabetics taking oral hypoglycaemic medications or insulin.

Data were entered into a Microsoft Access-based software program. Regression analysis was used to estimate differences between groups after adjustment for sex.

## Results

289 people agreed to participate in the study, giving an overall response rate of 48.7%. Males aged 25–29 years had the lowest response rate (24.0%)—whereas males aged 60 years and over, and females aged 30 years and over, had response rates higher than 50%, the highest being 76% for females aged 50–54 years. The female:male ratio was 1.5, and 249 (86%) respondents self-identified as Maori. The following results are for Maori participants only.

Table 1 shows the demographic and clinical characteristics of Maori respondents. The mean BMI was 33.4 kg/m², and more than 90% of both females and males were either overweight or obese, defined as a BMI of 25 kg/m² or more.

Table 2 shows the age-standardised prevalence of insulin resistance, IFG or IGT, and diabetes (estimated using our equation based on fasting insulin, triglycerides, and BMI). Overall, the age-standardised prevalence of diabetes, both known and newly diagnosed, was 10.6%. The age-standardised prevalence of known diabetes was about twice that for newly diagnosed diabetes. IGT or IFG was relatively uncommon, whereas the age-standardised prevalence of insulin resistance was 40.3% for females and 36.0% for males.

Figure 2 shows the overall age-specific prevalence rates for diabetes (both known and newly diagnosed) and insulin resistance with normal glucose tolerance. Insulin resistance was more common among the young age groups—with the 30–39 year age group having the highest age-specific rate (44.3%), whereas the prevalence of diabetes increased with age, peaking in the 60–69 year age group at 34.1%.

The characteristics of the group identified as being insulin resistant were compared with the group that did not have any disorders of glucose metabolism (Table 3). The mean age of these groups was similar, as was the proportion who smoked. A history

of gout and a family history of diabetes were more common among the insulin-resistant group. Also, individuals in this insulin-resistant group were more likely to be overweight or obese and have an elevated blood pressure, and an elevated triglyceride level. Total cholesterol and LDL levels were similar.

**Table 1. The demographic and clinical characteristics of study participants by sex**

| Variable | Female (n=153) | Male (n=94) | Total (n=247) |
|---|---|---|---|
| Age (years) | 47.8 (±14.1) | 51.8 (±14.2) | 49.3 (±14.2) |
| Current smoker (%) | 44.4 | 31.9 | 39.7 |
| Family history of diabetes (%) | 44.4 | 40.4 | 42.9 |
| Weight (kg) | 85.9 (± 22.4) | 92.4 (± 16.1) | 88.4 (± 20.4) |
| BMI (kg/m$^2$) | 33.9 (± 8.2) | 32.7 (± 5.2) | 33.4 (± 7.2) |
| Waist (cm) | 98.9 (± 17.1) | 101.8 (± 11.7) | 100.0 (± 15.3) |
| Systolic BP (mmHg) | 124.4 (± 15.1) | 125.0 (± 11.8) | 124.7 (± 13.9) |
| Diastolic BP (mmHg) | 82.5 (± 13.3) | 81.9 (± 10.9) | 82.2 (± 12.4) |
| Total cholesterol (mmol/L) | 5.34 (± 0.98) | 5.53 (± 1.10) | 5.41 (± 1.03) |
| Triglycerides (mmol/L) | 1.70 (± 1.08) | 2.01 (± 1.73) | 1.82 (± 1.37) |
| HDL (mmol/L) | 1.31 (± 0.35) | 1.23 (± 0.31) | 1.28 (± 0.34) |
| Fasting insulin (mIU/L) | 15.2 (± 11.5) | 13.9 (± 15.9) | 14.7 (± 13.3) |
| Overweight (25≤BMI<30) (%) | 26.1 | 21.3 | 24.3 |
| Obese (BMI≥30) (%) | 64.7 | 71.3 | 67.2 |

Mean values and standard deviations are presented unless otherwise stated; BP=blood pressure; HDL=high-density lipoprotein; BMI=body mass index.

**Table 2. Age-standardised prevalence of insulin resistance, IFG or IGT, and diabetes in adults aged 25 years and over**

| Variable | Female (n=153) | | Male (n=94) | | Total (n=247) | |
|---|---|---|---|---|---|---|
| | % | (95% CI) | % | (95% CI) | % | (95% CI) |
| Known diabetes | 8.2 | (3.5–12.9) | 6.2 | (1.2–11.2) | 7.1 | (4.0–10.2) |
| New diabetes | 3.4 | (0.6–6.3) | 3.5 | (0.0–7.6) | 3.6 | (1.4–5.3) |
| Total diabetes | 11.6 | (6.1–17.1) | 9.7 | (3.5–15.8) | 10.6 | (6.8–14.4) |
| IFG or IGT | 2.5 | (0.2–4.7) | 5.9 | (0.9–10.9) | 4.1 | (1.6–6.6) |
| Insulin resistance* | 40.3 | (29.6–50.9) | 36.0 | (19.1–52.9) | 37.0 | (28.6–45.5) |

Age-standardised to the WHO world population; IFG=impaired fasting glycaemia; IGT= impaired glucose tolerance;*Insulin resistance calculated using the McAuley formula among those with normal glucose tolerance.

Table 4A shows different estimates of age-specific prevalence of glucose metabolism disorders in three age categories using our prediction equation and the ATP III criteria. Comparable age trends are evident with the two approaches. As no cut-offs to define insulin resistance have been applied to the HOMA2.1 and the $ISI_{0,120}$ method, means and standard deviations are presented in Table 4B rather than prevalence rates. Those persons taking diabetes medications were excluded for the calculation of the HOMA2.1 as fasting insulin levels are less meaningful in this setting, and as this group did not have an OGTT, they could not be included in the $ISI_{0,120}$ calculation.

**Figure 2. Age-specific prevalence of diabetes (known and newly diagnosed) and insulin resistance with normal glucose tolerance**
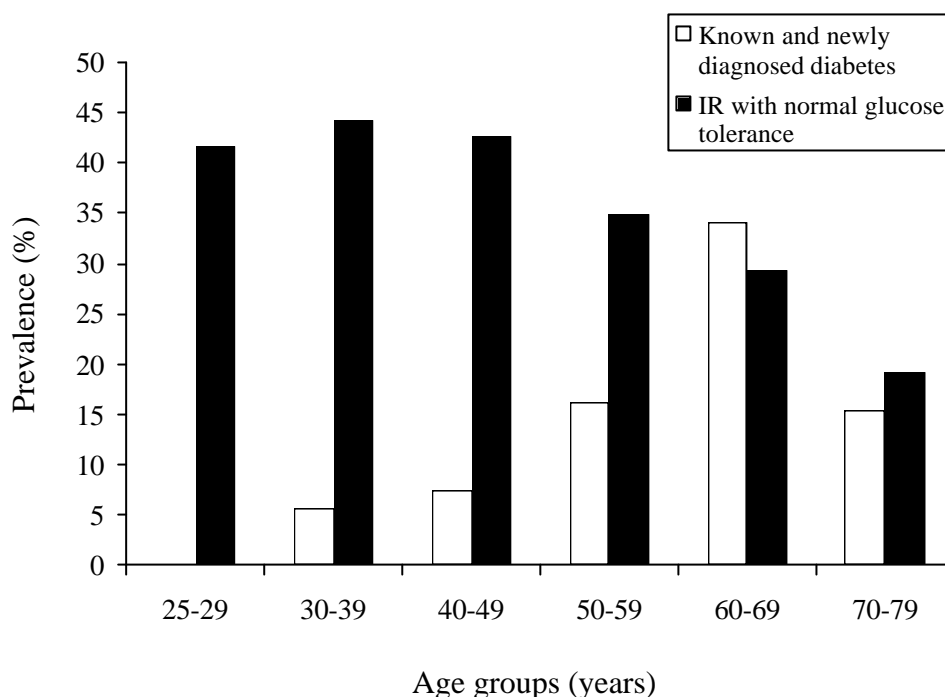


**Table 3. Characteristics of the insulin resistance but normal glucose tolerance group and the 'healthy' group**

| Variable | Insulin resistance (n=91) | | 'Healthy' (n=112) | | Difference (95%CI) |
|---|---|---|---|---|---|
| Sex (male) | 31.9 % | | 40.2 % | | |
| Age (yrs) | 46.2 | (12.8) | 48.7 | (14.8) | -2.5 (-6.4, 1.3) |
| Current smoker | 42.9 % | | 44.6 % | | 0.9 (0.5, 1.6)* |
| History of gout | 20.9 % | | 7.1 % | | 4.2 (1.8, 10.6)* |
| History of hypertension | 12.1 % | | 16.1 % | | 0.7 (-0.3, 1.6)* |
| History of IHD | 8.8 % | | 5.4 % | | 1.9 (0.6, 5.7)* |
| Family history of diabetes | 51.6 % | | 31.3 % | | 2.3 (1.3, 4.2)* |
| Family history of IHD | 33.0 % | | 27.7 % | | 1.2 (0.7, 1.2)* |
| Weight (kg) | 98.9 | (19.5) | 77.5 | (15.3) | 22.1 (17.4, 26.8) |
| BMI (kg/m²) | 37.0 | (6.9) | 29.5 | (5.3) | 7.4 (5.7, 9.1) |
| Waist (cm) | 107.5 | (13.0) | 90.4 | (11.4) | 17.5 (14.1, 20.8) |
| Systolic BP (mmHg) | 125.8 | (15.7) | 120.9 | (10.3) | 4. 9 (1.3, 8.6) |
| Diastolic BP (mmHg) | 84.0 | (12.5) | 79.0 | (11.7) | 5.0 (1.6, 8.3) |
| Total cholesterol (mmol/L) | 5.54 | (1.00) | 5.19 | (1.02) | 0.37 (0.09, 0.65) |
| HDL cholesterol (mmol/L) | 1.16 | (0.28) | 1.44 | (0.35) | -0.29 (-0.38, -0.20) |
| Triglycerides (mmol/L) | 2.35 | (1.24) | 1.04 | (0.38) | 1.3 (1.1, 1.6) |
| Fasting insulin (mU/L)[†] | 17.81 | (15.91, 19.95) | 6.49 | (5.84, 7.21) | 2.72 (2.33, 3.18) |

Data presented are mean values and standard deviations unless otherwise stated; Differences or odds ratios are adjusted for sex; IHD=ischaemic heart disease; BP=blood pressure; HDL=high-density lipoprotein; BMI=body mass index; *Odds ratio (95% CI); †Geometric means (95% CI) and their ratio (95% CI) based on a log transformation.

**Table 4A. Comparison of age-specific prevalence rates of insulin resistance using our formula and the ATP III criteria**

| Variable | Age groups (years) | | | | | |
|---|---|---|---|---|---|---|
| | 25–39 (n=82) | | 40–59 (n=97) | | 60–79 (n=67) | |
| | Number | (%) | Number | (%) | Number | (%) |
| **McAuley formula**[*] | | | | | | |
| Diabetes | 4 | | 11 | | 18 | |
| IFG or IGT | 2 | | 5 | | 4 | |
| Insulin resistance | 36 | | 38 | | 17 | |
| Total insulin resistance | 42 | (51.2) | 54 | (55.7) | 39 | (58.2) |
| **ATP III criteria** | | | | | | |
| Total insulin resistance | 28 | (34.1) | 35 | (36.1) | 28 | (41.8) |

[*]McAuley formula = $\exp[3.29 - 0.25\ln(\text{insulin}) - 0.22\ln(\text{BMI}) - 0.28\ln(\text{TAG})]$. (The formula was developed to predict insulin sensitivity, values = 6.3 M• mU-1• l-1 define those who are insulin resistant.); IFG=impaired fasting glycaemia; IGT=impaired glucose tolerance.

**Table 4B Comparison of means and standard deviations by age groups for HOMA 2.1 and ISI0,120**

| Calculation | Age groups (years) | | | | | |
|---|---|---|---|---|---|---|
| | 25–39 (n=79) | | 40–59 (n=90) | | 60–79 (n=52) | |
| | Mean | (SD) | Mean | (SD) | Mean | (SD) |
| HOMA 2.1 - %S[*][†] | 87.4 | (119.0) | 101.7 | (84.7) | 104.8 | (83.4) |
| $\text{ISI}_{0,120}$[*][†] | 145.1 | (76.5) | 149.4 | (103.5) | 110.7 | (51.4) |

[*] %S is derived from the HOMA 2.1 computer model, and is a measure of insulin sensitivity with 100% defined as normal, and higher numbers signifying greater sensitivity; $\text{ISI}_{0,120}$ is a calculated measure of sensitivity with higher numbers signifying greater sensitivity; [†] HOMA 2.1 and $\text{ISI}_{0,120}$ calculations exclude those on diabetes medications.

## Discussion

Slightly less than half the eligible participants (49%) completed all components of the survey. While a higher response rate would have been desirable, this rate was comparable with that of similar surveys such as the recent AUSDIAB Study.[19] The low overall response rate in our study can to a considerable extent be explained by the poor response among the younger age group, especially males. The forestry industry employs a high proportion of the young men, and because their working days begin early, many young men were unable to obtain time away from work.

The population of the East Coast is spread over a large geographic area, and the distances required to travel to the survey centres was a major disincentive to participation in all age groups. However, the coordinated efforts of the study coordinator, kaiawhina, and rural health nurses to provide frequent reminders and to arrange transport to the survey centres resulted in a much higher response rate among middle aged and older individuals. Among women aged 40 years and over, the response rate was 62%, which compares favourably with the 68% response rate among 40–79 year old Maori women who were invited to complete a questionnaire

and have anthropometric measures and a random blood glucose in a 1995/96 South Auckland survey.[9] Comparison of the response rate for males is less favourable (43% vs 63%). The high response rate (93% of households) in an earlier South Auckland survey involved only the completion of a questionnaire and suggests that the oral glucose tolerance test (OGTT) and physical examination may be disincentives.[8] However, an OGTT is an essential component of any study which aims to assess the prevalence of disturbances of carbohydrate metabolism.

The limited number of prevalence studies among Maori populations in New Zealand are not directly comparable, and it was not possible to assess changes in diabetes prevalence over time. However, the comparable prevalence of known diabetes among women aged over 45 years in the East Coast population studied in the present survey (14%) and of self-reported diabetes in the 2002/03 NZHS (13%)[6] provides strong confirmation of the overall high prevalence. Of interest, are the appreciably higher rates of self reported diabetes among males aged over 45 years in the NZHS (21%) than in the present study (10%), where the diagnosis was confirmed. The difference may be partly explained by the low response rate among East Coast men, but we cannot explain this difference with certainty.

Interestingly, in our data, newly diagnosed diabetes rates were only half that of known diabetes whereas most other surveys have reported comparable rates of known and newly diagnosed cases.[3,7,9] This may well be due to the high level of awareness of diabetes in the study area. This in turn has resulted in more frequent screening of high-risk individuals.

A key purpose of the present study was to assess the prevalence of insulin resistance. Individuals with insulin resistance in the general population urgently need to be targeted for diabetes prevention and cardiovascular risk reduction strategies, but no universally accepted method for predicting insulin sensitivity exists. Euglycaemic clamps and intravenous glucose tolerance tests (IVGTTs) are limited to research settings. Various surrogate methods for predicting insulin sensitivity have been used in studies and a number of newer approaches have been suggested.[20]

The most widely published method for predicting insulin sensitivity is the Homeostasis Model Assessment based on fasting glucose and insulin,[21] which should ideally be based on three separate blood measurements taken 5 minutes apart and calculated using the model programme, but in most cases is based on a single measure and is estimated using a simplified formula.[16] Only the HOMA-model has been shown to correlate well with the euglycaemic clamp.[16,18]

Our study and others have found the HOMA formula to be no better than a fasting insulin in this regard.[15,18] The HOMA formula is generally applied to those with normal glucose tolerance (NGT), IFG, IGT, and diabetes with several caveats for use in those on sulponylureas and exogenous insulin.[16]

No cut-off has been proposed to identify a group with poor insulin action. Furthermore, there has been criticism of choosing surrogates to predict insulin sensitivity that correlate well with a euglycaemic clamp, a dynamic test under non physiological conditions. Thus, we have selected a further method for predicting insulin resistance, developed by Gutt et al, based on the average of the fasting and 2-hour glucose and insulin levels. This has been shown in prospective studies to be the best method for predicting the development of type 2 diabetes.[20]

The failure to show a deterioration in insulin sensitivity with age (Table 4B) with both these measures reflects the fact that those with diabetes on medication have been excluded because of the difficulty in interpreting their insulin sensitivity data using this approach. It is clear that HOMA 2.1 and $ISI_{0,120}$ formulae are currently inappropriate to determine prevalence of insulin resistance. However they are likely to be of value in assessing response to intervention programmes aimed at improving insulin sensitivity, and will be used for this purpose in the Ngati and Healthy programme.

To date, the most frequently used approach for determining frequency of insulin resistance or identifying insulin resistant individuals has been to use a set of surrogate clinical and laboratory criteria. We have compared our equation[15] which has been independently validated[22] with the ATP III criteria[17] for the definition of those with the metabolic syndrome. Our equation combines fasting insulin, triglycerides and BMI as continuous variables, so that those who would have fallen just outside a particular cut off can still be included depending on the other variables.

An arbitrary cut off (of less than or equal to 6.3 M•mU-1•l-1) is applied to select those with poor insulin sensitivity, based on the lowest quartile for a lean population. Inevitably, the cut-off point is somewhat arbitrary but a similar difficulty applies to the ATP III criteria which might be expected to miss an even greater number of insulin resistant individuals since arbitrary cut offs are applied to several clinical and metabolic variables.

Table 4A shows that using our approach, more than half the population have insulin resistance, and this increases with increasing age, when those with IFG, IGT, and type 2 diabetes are included. Not surprisingly, the ATP III criteria gives rates substantially lower than this, but the same pattern of increasing rates across age groups is observed.

It has been estimated that as many as 25% of adults of European descent may be insulin resistant.[10] The appreciably higher rates observed here (40% among women, 36% among men) represent considerable cause for concern given that this condition is believed to be the underlying cause of most cases of type 2 diabetes mellitus as well as being an important contributor to cardiovascular risk. Of especially great concern are the high rates among young individuals (Figure 2). This suggests that the future burden of diabetes and other diseases associated with insulin resistance and the metabolic syndrome is likely to escalate in the near future unless effective intervention programmes are in place.

The Ngati Porou Hauora Ngati & Healthy Programme is one such pioneering programme, which will be formally evaluated using well established methods.[23] A national diabetes prevalence survey, which would include estimates of IFG, IGT, and insulin resistance as well as associated clinical, anthropometric, and metabolic variables and assessment of nutritional status, is imperative since no such national data exist. Such information is essential for health care planning for what is arguably the most important epidemic disease in New Zealand and for assessing the effects of national strategies aimed at reducing obesity and diabetes rates.

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# Risk factors for type 2 diabetes in postmenopausal New Zealand women: a cross-sectional study

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## Abstract

**Aim** To describe the diabetes risk profile of 3377 postmenopausal New Zealand women.

**Methods** Participants took part in a face-to-face interview with a research nurse. Medical history, lifestyle information, height, weight, and random capillary blood glucose were recorded. Risk scores for type 2 diabetes were later calculated for each participant using a modified diabetes 'risk analysis' tool developed by the United States Diabetes Prevention Program (DPP).

**Results** 157 women had diagnosed diabetes (4.4%) and were not therefore included in the risk analysis. Over half of the women (1843 of 3377) were at 'high risk' for the development of type 2 diabetes when assessed using the DPP diabetes risk analysis tool; 38.6% of participants were overweight, 25.6% were obese, 32.5% were physically inactive, 16.9% had a family history of diabetes, and 6.5% had random capillary blood glucose recordings of 7.5 mmol/L or greater.

**Conclusion** Risk factors for type 2 diabetes are prevalent in this group of postmenopausal New Zealand women. To appropriately target lifestyle modification programmes, these findings (in conjunction with recent evidence that lifestyle modification can reduce diabetes onset) highlight an urgent need to identify those women with risk factors before they develop type 2 diabetes.

Diabetes has risen to alarming levels in New Zealand and internationally, and is associated with significant morbidity and mortality[1] as well as personal and economic costs.[2] Healthcare costs relating to type 2 diabetes in New Zealand are estimated to be NZ$398 million in 2006/07, increasing to NZ$1066 million by the year 2021.[2]

A combination of genetic and behavioural or 'lifestyle' factors appear to contribute to the aetiology of type 2 diabetes.[3] Non-modifiable risk factors for diabetes include family history of diabetes, ethnicity, and age.[4] In New Zealand, diabetes is more prevalent in Maori and Pacific people than in European, and is often first diagnosed in middle age.[5] Lifestyle risk factors such as obesity and physical inactivity are believed to be the primary modifiable determinants of the disease,[3] and are independently associated with an increased risk for diabetes.[6–8] A diet high in saturated fats and low in fibre has also been recognised as a risk factor for type 2 diabetes,[9] and smoking appears to confer a small increased risk.[6]

Type 2 diabetes is preceded by a period of impaired glucose tolerance (IGT) and/or impaired fasting glucose (IFG).[10] These 'pre-diabetes' conditions (IGT and IFG) increase in prevalence with age, with estimated rates of 15.7% and 4.5% respectively in Australian women aged 55–64 years.[11] Large randomised controlled trials involving intensive lifestyle intervention programs in Finland, China, and the United

States have demonstrated that increasing physical activity and diet modification can effectively reduce the onset of diabetes by up to 58% in individuals with IGT.[3,12,13] Research in New Zealand has also been carried out to develop culturally appropriate lifestyle-intervention strategies for groups at high-risk for type 2 diabetes.[14–17]

Historically, there has been a strong focus on the identification of individuals with cardiovascular risk factors in primary care. Although diabetes will often be detected during a cardiovascular risk assessment, the recent trials confirming the ability to reduce diabetes by reducing risk factors give impetus to focussing on diabetes risk independently of cardiovascular risk. Given the strong evidence in favour of diabetes risk reduction through lifestyle modification, it is important to determine the best way to identify those individuals who would most benefit from lifestyle changes, and to determine the best way to quantify the degree of risk in those individuals.

Given that up to 50% of diabetes cases may go undiagnosed,[11] the estimated 4.3% of 45–64 year old New Zealand women diagnosed with diabetes in 1996–1997[18] is likely to be an underestimate of the true prevalence of the disease. The prevalence of risk factors for diabetes has not been well documented for women in this age group, nor has a diabetes risk profile been presented to date.

Several assessment tools have been developed to ascertain 'diabetes risk scores' for patients by taking into account various demographic, lifestyle, and clinical variables.[19,20] A web-based tool was developed by the US based Diabetes Prevention Program (DPP) that allows individuals to self-assess diabetes risk based on age, weight, ethnicity, gestational diabetes, family history of diabetes, blood glucose level and physical inactivity.[20] The present study will describe the prevalence of risk factors and calculate diabetes risk scores using data collected from postmenopausal New Zealand women during the recruitment process for an international clinical trial and a national observational study.

## Methods

**Study population**—Between January 2000 and November 2002, 7035 women were invited from 24 general practices in four regions of New Zealand to attend an interview with a research nurse.

**Recruitment method**—Participants were sent a letter from their general practitioner that invited them to an appointment with a research nurse to discuss their health, menopause, and a long-term trial involving hormone replacement therapy. Eligibility criteria for invitation included: female, age between 49 and 69 years, current patient at participating practices, and no menstrual period in the previous 26 weeks.

**Data collection and analysis** Data were collected by research nurses in face-to-face interviews that took place at the patient's general practice or at a nearby specialist medical centre. The interviews were conducted as part of the joint recruitment process for the 'Women's International Study of long Duration Oestrogen after Menopause' (WISDOM)[21,22] and recruitment to 'The observational study of mid-life New Zealand women'.

Interviews took approximately 1 hour and informed consent was obtained for all participants. Data collected included sociodemographic information, medical history, lifestyle information—and clinical recordings that included height, weight, and random capillary blood glucose. Interview data were entered directly into a laptop-based computer program and with the exception of clinical recordings, all data collected were based on patient self-report. Data were imported into Microsoft Access97 tables and then into the statistical package Epi-Info 6.04b for calculation of frequencies.

**Risk factor definitions** Height was measured in centimetres (cm) using wall-mounted Tollot S/M standardised measure, with the patient standing, bare-foot with their head in the horizontal 'Frankfurt plane position'. Weight was measured in kilograms (kg) to the nearest gram using Salter bathroom scales with the patient lightly clothed and bare-foot.

Individuals with a BMI of 25.0–29.9 kg/m$^2$ were classed as overweight, and those with a BMI of >30kg/m$^2$ were classed as obese.[23] Participants were classified as 'physically inactive' if they reported having exercised 'rarely/never'; 'less than once a week'; 'once a week'; or '2-3 times per week' when interviewed by the research nurse,. Individuals who reported having a mother, father, or sibling with a history of diabetes were regarded as 'having a family history of diabetes'. Non-fasting capillary blood glucose levels were measured by the research nurse using an Accu-chek® Advantage blood glucose meter and an Accu-chek® softclix pro pen. Ethnicity was self-defined using the 1996 New Zealand census question for interviews carried out up until March 1 2002 after which date the 2001 census question was used.

The DPP risk analysis tool calculates scores by totalling the number of points accumulated for each risk factor that the individual has (Table 2). The DPP website advises individuals with a score of 4 or less that they are probably at low risk for having diabetes, but not to forget about it—especially if they are Hispanic, African American, Native American, Asian American, or a Pacific Islander.

Individuals with a score of 6 or more are advised that only a doctor can determine if they have diabetes, and are given information on free screening by the DPP at the University of Washington, or are advised to see a doctor to find out for sure.[20] Minor modifications were made to the DPP risk analysis tool used in the present study as patients were not asked at recruitment if they had been told they have a high blood sugar level (awarded 6 points). Elevated random capillary blood glucose recordings were used in place of this measure; individuals with a reading equal to 7.5mmol/L or above were awarded 6 points. Patients were asked about any children weighing greater than 9 pounds at birth but not if they had diabetes during pregnancy. In addition to the ethnic groups regarded as 'high risk' in the DPP analysis, individuals identifying themselves as Maori in the present study were also allocated 3 points based on the higher rate of diabetes for New Zealand Maori.

## Results

Of the 7035 women invited to participate in the study, 569 (8.1%) were not eligible because they were still having periods, 515 (7.3%) did not reply to the invitation, and 2417 (34.4%) declined participation for 'other' reasons. A total of 3534 women who met initial eligibility criteria (54.7%) attended an interview. At the time of recruitment, 157 participants (4.4%) had diagnosed diabetes so were excluded from this diabetes risk analyses. Participants had a mean age of 59.7 years, 39.3% had a tertiary qualification, and the majority (82.8%) of the respondents identified themselves as New Zealand European. Table 1 displays the characteristics of the study population.

Diabetes and risk factor prevalence **Using the modified DPP risk analysis tool, 54.6% of the women in this sample were regarded as being at 'high risk' for the development of type 2 diabetes (Table 2). Age and overweight were the most common risk factors in this group of women. Only 19.2% of the sample had 'age' as their sole risk factor (with a DPP risk score of 1 or 3). Risk scores and frequencies are presented in Table 3. Of those interviewed, 38.6% were overweight and 25.6% obese, 32.5% were physically inactive, 16.9% had a family history of diabetes, and 6.5% had capillary blood glucose levels of 7.5 mmol/L or greater so were referred to their general practitioner for follow-up.**

## Table 1. Characteristics of 3377 postmenopausal New Zealand women

| Characteristics | Total sample | |
|---|---|---|
| | **n** | **%** |
| **Age band (years)** | | |
| 49–54 | 701 | 20.8 |
| 55–59 | 974 | 28.8 |
| 60–64 | 862 | 25.5 |
| 65–69 | 840 | 24.9 |
| **Education** | | |
| Tertiary | 1328 | 39.3 |
| Non-tertiary | 1677 | 49.7 |
| Other/not known | 372 | 11.0 |
| **Ethnicity** | | |
| NZ European | 2796 | 82.8 |
| Maori | 72 | 2.1 |
| Pacific | 27 | 0.8 |
| Asian | 65 | 1.9 |
| Other European | 172 | 5.1 |
| Other* | 181 | 5.4 |
| Not known | 64 | 1.9 |
| **NZDep scores** | | |
| 1 to 4 | 2299 | 68.1 |
| 5 to 6 | 511 | 15.1 |
| 7 to 10 | 430 | 12.7 |
| No score available | 137 | 4.1 |
| **Parity** | | |
| >1 term pregnancy | 3015 | 89.3 |
| **Body mass index (BMI)** | | |
| BMI average kg/$m^2$ | 27.4 | – |
| BMI 25–29.9 kg/$m^2$ | 1304 | 38.6 |
| BMI 30+ kg/$m^2$ | 863 | 25.6 |
| BMI Not known | 61 | 1.8 |
| **Smoking status** | | |
| Never smoked | 1998 | 59.2 |
| Past smoker | 1025 | 30.4 |
| Current smoker | 352 | 10.4 |
| Not known | 2 | 0.1 |
| **Physically inactive** | | |
| Exercise rarely or never | 148 | 4.4 |
| Exercise less than once a week | 104 | 3.1 |
| Exercise once a week | 178 | 5.3 |
| Exercise 2–3 times a week | 667 | 19.8 |

*Includes 1 African American and 4 Hispanic women.

**Table 2. Diabetes risk factors and risk scores for 3377 postmenopausal New Zealand women**

| Risk factor | Points allocated in DPP risk analysis | Total sample | |
|---|---|---|---|
| | | **n** | **%** |
| 45 to 64 years old | 1 | 2537 | 75.1 |
| 65 years or older | 3 | 840 | 24.9 |
| Under 65 years old and physically inactive | 3 | 828 | 24.5 |
| Overweight (BMI≥25) | 3 | 2167 | 64.2 |
| Random blood glucose >7.5 mmol/L* | 6 | 219 | 6.5 |
| Baby ≥9 pounds at birth | 6 | 625 | 18.5 |
| Family history of diabetes† | 3 | 570 | 16.9 |
| Ethnicity ‡ | 3 | 104 | 3.1 |
| **Diabetes Risk Score** | | | |
| Low risk at present | 1 to 5 | 1534 | 45.4 |
| High risk at present | 6 or more | 1843 | 54.6 |

*Used as surrogate measure in place of the statement 'I have been told I have a high blood sugar level' used in the DPP risk analysis; †Parent(s), sister, or brother with diabetes; ‡Includes Hispanic, African American, American Indian, Asian American, Pacific people, and Maori.

**Table 3. Diabetes risk score frequencies calculated for 3377 postmenopausal New Zealand women**

| DPP risk score | Frequency of score | % | DPP risk classification |
|---|---|---|---|
| 1 | 439 | 13.0 | |
| 3 | 210 | 6.2 | **Low risk** |
| 4 | 885 | 26.2 | |
| 6 | 342 | 10.1 | |
| 7 | 642 | 19.0 | **High risk** |
| 9 | 120 | 3.6 | |
| 10 | 324 | 9.6 | |
| 12 | 117 | 3.5 | |
| 13 | 192 | 5.7 | |
| 15 | 34 | 1.0 | |
| 16 | 38 | 1.1 | |
| 18 | 15 | 0.4 | |
| 19 | 17 | 0.5 | |
| 21 | 2 | 0.1 | |

## Discussion

Risk factors for type 2 diabetes are prevalent in this group of postmenopausal New Zealand women, with more than half of the participants at 'high risk' for developing type 2 diabetes when scored using the DPP risk analysis tool. There was a high proportion of modifiable risk factors for type 2 diabetes in this group, with nearly two-thirds of participants classed as 'overweight' or 'obese', and one-third as 'physically inactive'. There has been emphasis on the high rates of diabetes in Maori and Pacific people in New Zealand, but the current findings highlight the high prevalence of risks for diabetes in this large group of predominantly New Zealand European women.

Although participants in the present study were not drawn from a random sample, they represent approximately half of all 49–69 year old women enrolled at the participating practices. This study population has similar rates of physical inactivity, overweight and obesity to those reported in New Zealand population data,[24,25] but a higher proportion of women have a tertiary education than the national average for this age group.[26]

The percentage of women with diagnosed diabetes in this study was similar to national rates observed in 1996/97. The fact that up to half of all diabetes cases go undiagnosed means that women with undetected diabetes are likely to have been included in the risk analysis presented here. A limitation of the present study is the use of random capillary blood glucose tests as a surrogate measure of raised blood sugar. Finger-prick tests are often used in clinical practice as an opportunistic screening tool to detect raised blood sugars, but day-to day variability in recordings means they are not used for differential diagnosis of diabetes in New Zealand.[27] The percentage of women with raised capillary blood sugars reported here might therefore be an over- or an underestimate of underlying abnormalities.

Diabetes itself is irreversible, but recent evidence has shown that lifestyle or behavioural risk factors predisposing an individual to diabetes are not.[3,12,13] The challenge for health professionals now is not only to identify and effectively manage current diabetics, but also to identify those individuals at risk for developing disease, and providing appropriate advice regarding lifestyle modification. Targeting diabetes risk separately from cardiovascular disease is also important as a means of addressing the current alarming rise in diabetes and pre-diabetic syndromes.

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# Body size, body composition, and fat distribution: a comparison of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities

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## Abstract

**Aims** To investigate body size and body fat relationships and fat distribution in young healthy men drawn from New Zealand European, Pacific Island, and Asian Indian populations.

**Method** A total of 114 healthy men (64 European, 31 Pacific Island, 19 Asian Indian) aged 17–30 years underwent measurements of height, weight, and body composition by total body dual-energy X-ray absorptiometry (DXA). Body mass index (BMI) was then calculated. Percent body fat (%BF), fat-free mass, bone mineral content, bone mineral density, abdominal fat, thigh fat, and appendicular skeletal muscle mass (ASMM) were obtained from the DXA scans.

**Results** For the same BMI, %BF for Pacific Island men was 4% points lower and for Asian Indian men was 7–8% points higher compared to Europeans. Compared to European men for the same %BF, BMI was 2–3 units higher for Pacific Island, and 3–6 units lower for Asian Indian. The ratio of abdominal fat to thigh fat, adjusted for height, weight, and %BF, was significantly higher for Asian Indian men than European (p=0.022) and Pacific Island (p=0.002) men. ASMM, adjusted for height and weight, was highest in Pacific Island and lowest in Asian Indian men.

**Conclusions** The relationship between %BF and BMI is different for European, Pacific Island, and Asian Indian men which may, at least in part, be due to differences in muscularity. Asian Indians have more abdominal fat deposition than their European and Pacific Island counterparts. Use of universal BMI cut-off points are not appropriate for comparison of obesity prevalence between these ethnic groups.

It is widely recognised that obesity (defined as an excess of body fat) and obesity-related diseases are an increasing global problem now reaching epidemic proportions.[1] Because of its general use and ease of measurement, body mass index (BMI) is commonly used as a surrogate measure for obesity. The World Health Organization (WHO) cut-off point for classification of obesity as a body mass index (BMI) above 30 kg/m$^2$ is intended as an internationally useful threshold for reflecting risk for Type 2 diabetes and cardiovascular diseases.

Based on this cut-off, Asian immigrants from the Indian subcontinent have low rates of obesity yet, relative to Europeans, they have a higher prevalence of coronary heart disease and Type 2 diabetes.[2–5]

Increased BMI levels explain only about half of the increased prevalence of diabetes[6] and hypertension[7] among Pacific Island peoples compared with New Zealand Europeans. In New Zealand (specifically, in inner urban South Auckland), the

prevalence of Type 2 diabetes in Asian Indian peoples is more than four-fold and in Pacific Island peoples more than two-fold higher than in Europeans.[8] These observations are of particular concern because of the increasing size of the resident Asian Indian and Pacific Island populations in this country. The 2001 census indicated that there are now more people of Asian than Pacific Island ethnicity resident in NZ. The Asian Indian subgroup comprises approximately 26% of the New Zealand Asian population.[9]

Ethnic differences in body build, fat patterning, and muscularisation may all contribute to differences in the relationship between BMI and body fat between ethnic groups. Asian Indians have a more central distribution of body fat than Europeans,[2] which is associated with increased risk of diabetes Type 2 and ischaemic heart disease.[10,11] Polynesians have higher bone mass and muscle mass than Europeans.[12–14]

The WHO has recognised the deficiencies of a universal cut-off for overweight and obesity[15,16] and in a recently published report[17] suggested that further body composition studies of Asian and Pacific Island populations are needed to determine equivalent fatness levels and the relation of BMI to body size.

Dual-energy X-ray absorptiometry (DXA) is widely accepted as a valuable technique for the assessment of body composition and, in particular, fat distribution, muscle mass and bone mass. While not without drawbacks as a reference method,[18] it has clear advantages over the traditional anthropometric approaches such as skinfold thicknesses, girths, and BMI. A significant drawback is that individuals with very high BMI are not easily accommodated.

On the other hand, the technology provides a more direct assessment of total body fat than anthropometric methods and offers regional composition analysis. We are not aware of any comparative analysis of the body composition of European, Asian Indian, and Pacific Island subjects using this technique.

We sought in the current study of a group of young healthy males who underwent DXA to identify ethnic differences in:

(1) The relationships between body fatness and body size,

(2) Fat distribution,

(3) Muscularity, and

(4) Bone mineral density and mass.

## Methods

Data from healthy male volunteers aged 17–30 years, who participated in cross-sectional studies of body composition conducted in the Department of Surgery, University of Auckland, were examined. All studies were approved by the local ethics committees and all participants provided written informed consent. Recruitment for these studies, principally from the urban Auckland area, was by personal contact, advertisement, or through existing networks of the recruiters.

Exclusion criteria were: total joint replacement, lifting weights more than once per week, major medical conditions (such as diabetes or cancer), and medication which could possibly affect body composition (such as oral steroids). Only one member of a family was measured. In addition, one subject was subsequently excluded from analysis because of a large difference (>3 kg) between recorded scale weight and DXA weight (sum of fat mass, fat-free soft tissue and bone mineral content). Of 114 volunteers, 64 self-identified as European, 31 as Pacific Island, and 19 as Asian Indian.

Height and weight were measured with participants wearing light clothing or standard hospital gown and no shoes. An estimated clothing weight was subtracted. Body composition (fat, fat-free soft tissue, and bone mineral content) and whole-body bone mineral density measurements were made using a single DXA machine (model DPX+ with software version 3.6y, Lunar Radiation Corp., Madison, WI). Fat-free mass (FFM) was calculated as the sum of the values for fat-free soft tissue and bone mineral content. Percent body fat was calculated as 100 x fat mass/(fat mass+FFM).

For assessing regional fat distribution, the whole-body DXA scans were analysed. Abdominal and thigh regions of interest were defined by the criteria of Ley et al.[19] Abdominal fat was obtained from analysis of a region of interest positioned with the lower horizontal border on top of the iliac crest and the upper border approximately parallel with the junction of the T12 and L1 vertebrae. The sides of this region were adjusted to include the maximum amount of abdominal tissue. A region of interest of identical height placed over the thighs (with the upper horizontal border positioned immediately below the ischial tuberosities) was used to obtain fat content of the thighs. The lateral margins were adjusted to follow the shape of the thighs.

Appendicular skeletal muscle mass (ASMM) was derived from the DXA scans as total limb mass minus the sum of limb fat mass and wet bone mass, estimated as bone mineral content divided by 0.55.[20] In this model, mass of the skin and associated dermal tissues is assumed to be negligible relative to the skeletal muscle component.

Results are presented as means ± SD. Between-group differences in subject characteristics were tested using one-way ANOVA followed by pairwise comparisons if a significant F test was obtained. Analysis of covariance was used to adjust body composition results for comparison across ethnic groups. Before carrying out analysis of covariance, similarity of regression slopes among the ethnic groups was verified by examining the significance of the interaction between the covariate(s) and the group variable. Data were analysed using SAS software, version 6.12 (SAS Institute Inc., Cary, NC). Results with p values <0.05 were considered significant.

## Results

The subject characteristics are summarised in Table 1. Pacific Island men in this study were heavier with higher BMI than Europeans and Asian Indians. Asian Indians were shorter than Pacific Islanders and significantly fatter than Europeans and Pacific Islanders.

As a proportion of total body fat, abdominal fat was significantly higher for Asian Indians than Europeans (p<0.0001) or Pacific Islanders (p<0.0001), while thigh fat was significantly lower than Europeans (p=0.037) or Pacific Islanders (p=0.015) (Table 1). After adjustment for weight, height, and %BF, the ratio of abdominal to thigh fat was significantly higher for Asian Indians than Europeans (p=0.022) and Pacific Islanders (p=0.002).

ASMM for Pacific Islanders was significantly higher than Europeans (p<0.0001); and for Europeans, ASMM was significantly higher than Asian Indians (p=0.0021) (Table 1). This pattern remained after adjustment of ASMM for height and weight, with Pacific Islanders having significantly higher ASMM than Europeans (p<0.0001), and Europeans having significantly higher ASMM than Asian Indians (p=0.0012).

After adjustment for height and weight, bone mineral density was significantly higher in Pacific Islanders than European (p=0.0009) and Asian Indian (p=0.0014). Adjusted bone mineral density for European was similar to that for Asian Indian (p=0.46). When adjusted for height and weight bone mineral content was significantly higher in Pacific Islanders than European (p=0.0021) and higher in European than Asian Indian (p=0.0008).

**Table 1. Characteristics of 64 New Zealand European, 31 Pacific Island, and 19 Asian Indian men aged 17–30 years**

| Variable | European Mean (SD) | Pacific Island Mean (SD) | Asian Indian Mean (SD) | p value* |
|---|---|---|---|---|
| Age (y) | 23.8 (3.7) | 22.7 (2.6) | 24.2 (3.4) | 0.24 |
| Height (cm) | 177.5 (6.2) | 179.1 (7.3) | 174.0 (6.1)[P] | 0.030 |
| Weight (kg) | 79.1 (10.7)[P] | 94.7 (17.5) | 79.6 (14.9)[P] | <0.0001 |
| BMI (kg/m$^2$) | 25.2 (3.4)[P] | 29.6 (5.3) | 26.3 (4.8)[P] | <0.0001 |
| Fat-free mass (kg) | 63.2 (5.9)[P] | 72.0 (8.9) | 55.9 (7.2)[PE] | <0.0001 |
| Total body fat (kg) | 15.9 (8.7) | 22.7 (12.6) | 23.7 (10.2)[PE] | 0.001 |
| Total body fat (%) | 19.4 (8.2)[P] | 22.7 (9.5) | 28.8 (8.0)[E] | 0.002 |
| Abdominal fat (kg) | 1.23 (0.86)[P] | 1.73 (1.09) | 2.11 (0.99)[E] | 0.001 |
| Abdominal fat (% of total fat) | 7.3 (1.2) | 7.3 (1.3) | 8.8 (1.2)[PE] | <0.0001 |
| Thigh fat (kg) | 1.47 (0.70)[P] | 2.14 (1.11) | 2.07 (0.86)[E] | 0.0005 |
| Thigh fat (% of total fat) | 9.5 (1.1) | 9.7 (1.1) | 8.9 (1.3)[PE] | 0.045 |
| Abdominal-to-thigh fat ratio | 0.79 (0.20) | 0.77 (0.19) | 1.02 (0.24)[PE] | <0.0001 |
| ASMM (kg) | 26.9 (2.8)[P] | 31.7 (2.4) | 24.1 (3.4)[PE] | <0.0001 |
| ASMM (%) | 34.3 (4.0) | 34.0 (4.3) | 30.7 (4.0)[PE] | 0.004 |
| Bone mineral content (kg) | 3.33 (0.42)[P] | 3.81 (0.45) | 2.92 (0.44)[PE] | <0.0001 |
| Bone mineral density (g/cm$^2$) | 1.24 (0.08)[P] | 1.34 (0.09) | 1.22 (0.10)[P] | <0.0001 |

ASMM=appendicular skeletal muscle mass; *analysis of variance; [E]p<0.05 vs European, [P]p<0.05 vs Pacific Island

Curvilinear relationships between %BF and BMI for each ethnic group were linearised by logarithmically transforming BMI (Figure 1). No significant difference was found between the slopes of the regressions of %BF on the logarithm of BMI for the three ethnic groups, but covariance analysis showed their elevations to be significantly different (p<0.0001).

The common slope regression equation for predicting %BF from BMI for the three ethnic groups was:

- %BF = 105.79 $\log_{10}$(BMI) – 128.42 – 3.77 group1 + 7.60 group2

- (SEE (standard error of estimate) = 4.89%, $R^2$ = 0.72)

where group1 is coded as 0 for European, 1 for Pacific Islanders, 0 for Asian Indians—and group2 is coded as 0, 0, 1 for these respective ethnic groups. Hence, for fixed BMI, compared with Europeans, Pacific Islanders had lower %BF by 3.8% (95% confidence interval: 1.4%–6.1%) and Asian Indians had higher %BF by 7.6% (5.0%–10.2%). At a BMI of 30 for Europeans the predicted %BF (28%) equates to a BMI of 33 for the Pacific Islanders and 25 for the Asian Indians (Table 2).

**Table 2. Comparison of European body mass index (BMI) and corresponding percent body fat with estimated BMI equivalents for Pacific and Asian Indians derived from equations relating BMI to percent body fat**

| European | | Pacific Island | Asian Indian |
|---|---|---|---|
| BMI (kg/m$^2$) | Body fat (%) | Approximate BMI equivalent (kg/m$^2$) | Approximate BMI equivalent (kg/m$^2$) |
| 20 | 9 | 22 | 17 |
| 25 | 20 | 27 | 21 |
| 30 | 28 | 33 | 25 |
| 35 | 35 | 38 | 30 |
| 40 | 41 | 43 | 34 |

**Figure 1. Relation between percentage body fat (%BF) and BMI for 64 European (closed circles), 31 Pacific Island (open circles), and 19 Asian Indian (triangles) men. The common slope linear regressions are given by %BF = 105.8 log$_{10}$(BMI) – intercept, where intercept=128.4 for the European (solid line), 132.2 for the Pacific Island (dashed line), and 120.8 for the Asian Indian men (dotted line)**
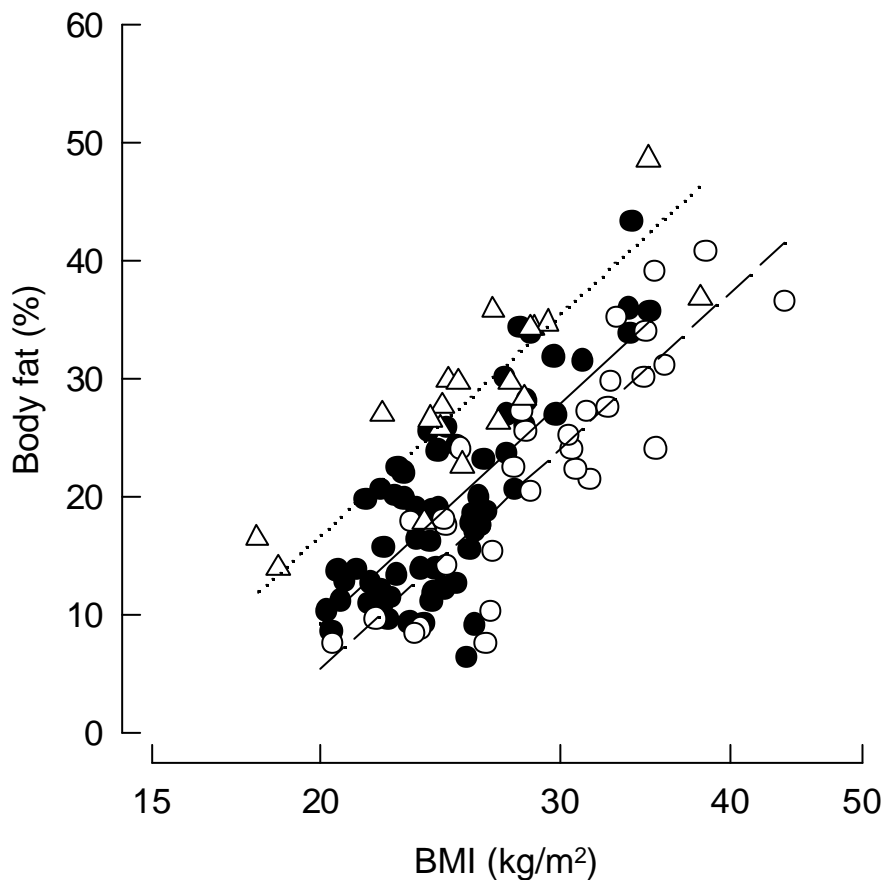
## Discussion

In a group of young European, Pacific Island and Asian Indian young men, we have shown that the relationship between percent body fat and BMI is ethnicity specific.

The most commonly used measure of obesity is BMI, and we found that, for a fixed BMI, Pacific Island men had significantly less body fat while Asian Indian men had significantly more body fat than their European counterparts. At a fixed %BF, BMI in Pacific Island men was 2–3 units higher and in Asian Indian men was 3–6 units lower than in European men. The BMI differences are smallest at low %BF and diverge with increasing %BF. This effect is seen in Table 2 and also is evident in Figure 1 when allowance is made for the logarithmic transformation of BMI. The results for Pacific Island men confirm our previous observations.[21]

Ethnic differences in the BMI-body fat relationships may be explained, at least in part, by differences in body build, particularly muscularity. We have shown that, compared with European men of similar weight and height, Asian Indian men have significantly less skeletal muscle in the limbs while Pacific Island men have significantly more. (Appendicular skeletal muscle is approximately 75% of total body skeletal muscle mass.)[22]

We have also shown, by examination of the distribution of fat in our subjects, that Asian Indian men have a more central fat deposition pattern than European or Pacific Island men. The propensity for abdominal adiposity found in Asian Indians had been inferred from measurements of waist-to-hip girth ratios in a number of studies.[2,23] Central obesity is closely associated with risk for cardiovascular disease and Type 2 diabetes.[3]

The greater bone mineral mass and bone density that we observed in Pacific Island men (relative to Europeans) may also contribute to differences in the body fat-BMI relationship for these ethnic groups. While bone mineral mass was lower in Asian Indians than Europeans, their bone mineral density was similar after adjustment for body size. Others have shown that both bone mineral density and bone mineral content in Asian men (predominantly Chinese) were similar to European men after controlling for weight, height, and age.[24] Age was not a significant covariate for our restricted-age range data.

A limitation of the present study is the comparatively small Asian Indian group and our results need to be confirmed with a larger sample from this ethnic group. In addition, our study does not address the other Asian subgroups which make up the majority of Asians in New Zealand.

The WHO BMI classifications of overweight ($\geq$25 kg/m$^2$) and obesity ($\geq$30 kg/m$^2$), although intended for international use, are based on the relationship between BMI and cardiovascular morbidity in Western populations.[1] Based on percent body fat levels a BMI of 26 kg/m$^2$ has been suggested as an obesity cut-off point in Asian Indians equivalent to that for Europeans,[25] and revised cut-off values to define overweight (23 kg/m$^2$) and obesity (25 kg/m$^2$) in Asian Indians have been proposed by the WHO.[15]

Current New Zealand Ministry of Health cut-offs for 'overweight' and 'obesity' are 26 and 32 kg/m$^2$, respectively in both Maori and Pacific Island adults. Studies are required to define the BMI range that may be considered 'healthy' in Asian Indian

and Pacific Island people on the basis of risk for obesity-related diseases. A consistent finding among migrant Asian Indian populations is hyperinsulinaemia and insulin resistance,[26] characteristics which may be important in the development of type 2 diabetes and cardiovascular disease.

Simmons et al[27] have reported that young Asian Indians are relatively hyperinsulinaemic compared to their European counterparts with the same BMI. Vikram et al[28] have shown that Asian Indians with 'normal' BMI ($<25$ kg/m$^2$) have high cardiovascular disease risk. Pacific Islanders in New Zealand, by contrast, are not hyperinsulinaemic relative to Europeans of the same BMI [29] and whilst they have a high prevalence of type 2 diabetes, they are believed to have a lower rate of cardiovascular disease.[30]

Our results demonstrate the marked differences in body build, body composition, and fat distribution that characterise male New Zealanders of European, Asian Indian and Pacific Island ancestry. We speculate that these may be related to differences in risk for cardiovascular disease and different pathways to Type 2 diabetes among these ethnic groups. The results emphasise the inadequacy of universal BMI cut-off points for determination of percentage body fat and obesity and the need to consider ethnic-specific weight targets.

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# Obesity and health-related quality of life: results from a weight loss trial

Cliona Ni Mhurchu, Derrick Bennett, Ray Lin, Maree Hackett, Andrew Jull, Anthony Rodgers

## Abstract

**Aims** To measure health-related quality of life (HRQoL) in overweight and obese New Zealand adults taking part in a weight loss trial, and to compare findings with the New Zealand population.

**Methods** Individuals (aged over 18 years with a BMI of 28–50 kg/m$^2$) participated in a randomised controlled weight loss trial. HRQoL was measured using the SF-36 questionnaire.

**Results** The 250 study participants had a mean (SD) age of 48 (12) years and a mean BMI of 35.4 (5.3) kg/m$^2$. Mean physical component (PCS) and mental component summary scores (MCS) were 47.2 (9.0) and 46.9 (11.1) respectively. Participants in the highest BMI tertile (>37 kg/m$^2$) reported significantly lower PCS scores compared with those in the middle and lowest tertiles (p=0.01), but no significant differences were seen in MCS scores (p=0.65). Comparison with population norms revealed significantly lower mean scores in all 8 SF-36 domains except mental health. No significant effect of modest weight loss on HRQoL was seen.

**Conclusions** These overweight and obese New Zealand adults experienced significantly impaired HRQoL compared to the New Zealand population. Small reductions in weight had no significant impact on HRQoL in this substantially overweight population.

Overweight and obesity are increasingly prevalent in developed and developing countries[1–3] and are important contributors to cardiovascular disease,[4–6] type 2 diabetes mellitus,[7,8] and several common cancers.[9,10] Body mass index (BMI) is the anthropometric measure that provides the most useful population-level indicator of excess body weight, although because it is a generalised measure that does not distinguish between weight associated with lean body mass and fat it is possible that measures of central body fat such as waist circumference and waist-hip ratio may be better predictors of certain diseases including diabetes.

The World Health Organisation (WHO) guidelines define a BMI of 18.50 to 24.99 kg/m$^2$ as normal and >25 kg/m$^2$ as overweight.[11] Estimations of the burden of disease attributable to excess weight indicate that high BMI is a leading cause of loss of healthy life worldwide;[12] and across developed regions, high BMI has been estimated to account for approximately 7% of all disability-adjusted life years (an integrated measure of population health incorporating both fatal and non-fatal outcomes),[12] which places high BMI close behind tobacco (12%), high blood pressure (11%), alcohol (9%), and high cholesterol (8%) as a leading cause of loss of healthy life in these regions.

While the physical effects of excess body weight are well recognised, less is known about the social and psychological effects. Evidence suggests that overweight and obese individuals are subject to stigmatisation and discrimination in various areas of life, including employment, education, and healthcare;[13,14] and they have an increased incidence of depression.[15]

Health-related quality of life (HRQoL) refers to the 'physical, psychological, and social domains of health, seen as distinct areas that are influenced by a person's experiences, beliefs, expectations and perceptions'.[16] Assessment of HRQoL can be made using a variety of measures, the most widely used and evaluated of which is the Short Form 36-question Health Survey (SF-36),[17] a generic measure based on ratings made by individuals themselves.[18] There is evidence that people who are overweight or obese experience significant impairment in quality of life,[19 20] but no New Zealand-specific data exist.

The objective of these analyses was to measure HRQoL in 250 overweight and obese New Zealand adults and compare findings with the New Zealand population. In addition, the effects of weight loss on HRQoL were evaluated.

## Methods

**Study participants**—Individuals were participants in a randomised controlled trial of the effect of the dietary supplement, chitosan, on body weight.[21] Study participants were recruited using newspaper advertisements and all participants provided written informed consent. Men and women aged over 18 years who wished to lose weight and had a BMI of between 28 and 50 kg/m$^2$ were included. Exclusion criteria were current treatment with chitosan containing supplements; current or recent treatment with weight loss medications; current or recent attendance at a commercial weight loss clinic/programme; allergy to seafood; pregnancy or lactation; active gastrointestinal disease or obesity surgery; involvement in another clinical trial; and individuals judged to be unlikely to comply with study treatment and follow-up procedures.

The study was conducted at the University of Auckland, New Zealand, between November 2001 and December 2002. The study protocol and related documents were approved by the Auckland Ethics Committee.

**HRQoL**—HRQoL was measured using the Australasian standard version (version 1) of the SF-36 questionnaire,[18] a generic measure of HRQoL that assesses eight domains of perceived health over the previous 4 weeks. These domains are: physical functioning (PF), role limitations related to physical problems (RP), bodily pain (BP), general health (GH), vitality (VT), social functioning (SF), role limitations related to emotional problems (RE), and mental health (MH). These dimensions are ordered from first to last according to the extent to which they measure physical or mental functioning.

Scores range from 0 (worst health state) to 100 (best health state) for all domains. For example, a score of 100 indicates the individual can perform all activities without limitations related to health. In three domains (GH, VT, MH), scores of 50 indicate an absence of problems. To obtain scores in excess of 50 in these three domains, health must be evaluated positively. Two summary component scores can be calculated from the SF-36: the physical component summary (PCS) score and the mental component summary (MCS) score. Both scores have a mean of 50 and a standard deviation of 10 and are standardised using means, standard deviations, and factor score coefficients from the general New Zealand population scores. Scores below 50 represent scores below the population mean.

HRQoL was measured at baseline and 6 months post-randomisation in this study. All questionnaires were checked for errors or missing data prior to data entry and standard guidelines for handling missing data were applied.[18]

**Statistical analysis**—Crude PCS and MCS scores were calculated using the New Zealand-specific factor weights.[22] Participants were stratified by baseline BMI tertile (28-32 [n= 83], 32.1-37 [n=85], >37 kg/m$^2$ [n=80]) to ensure equal numbers of participants across all groups, and scores were compared across these groups using analysis of variance (ANOVA).

Potential confounding factors such as age, gender, ethnicity (European/Non-European), socioeconomic status (SES) (using the New Zealand Socio-economic Index),[23] comorbidities (sum of doctor-diagnosed conditions including diabetes, hypertension, hyperlipidaemia, coronary heart disease, stroke, hyperthyroidism, hypothyroidism, gallbladder disease, osteoarthritis, back pain, sleep apnoea, shortness of breath, asthma, cancer, depression, other), and any other confounding factors discovered during stratified analyses, were controlled for by including these factors as covariates in an analysis of covariance (ANCOVA) model. Due to the small size of our study sample, comorbidities were not weighted, and a crude classification was used for adjustment as has been used in other similar studies.[24,25]

We assessed whether percentage weight change from baseline to 6 months was associated with changes in summary scores using multiple regression analysis after adjustment for age and other confounders (see above). Analyses were based on an intention-to-treat (ITT) approach with the last recorded observation carried forward (LOCF) for any missing data. In the case of missing SF-36 domain scores, scores were assumed to have remained the same as baseline scores—i.e. no change. In the case of missing weight data, the last recorded weight was used, which may have been recorded at baseline or at any of the subsequent 6 follow-up visits. A 'completers only' analysis (limited to participants with complete data) was also conducted as a sensitivity analysis. All statistical analyses were conducted using SAS for Windows (version 8.0) or Microsoft Excel (version 9.0).

## Results

**Participant characteristics**—The 250 study participants had a mean (SD) age of 48 (12) years, 206 (82%) were female, and their mean BMI was 35.4 (5.3) kg/m$^2$ (Table 1).

**Table 1. Characteristics of the study participants**

| Variable | Mean | SD |
|---|---|---|
| Age, years | 47.6 | (11.7) |
| Females, n (%) | 206 | (82.4) |
| Current smoker, n (%) | 23 | (9.2) |
| Current alcohol drinker, n (%) | 122 | (48.8) |
| Ethnic group, n (%) | | |
| - New Zealand European | 186 | (74.4) |
| - Maori | 29 | (11.6) |
| - Pacific Islands | 7 | (2.8) |
| - Other | 28 | (11.2) |
| Body Mass Index, kg/m$^2$ | 35.4 | (5.3) |
| Systolic Blood Pressure, mmHg | 123.2 | (18.3) |
| Diastolic Blood Pressure, mmHg | 69.7 | (9.5) |
| Total Cholesterol, mmol/L | 5.5 | (1.0) |
| Blood glucose, mmol/L | 5.3 | (1.4) |
| Diagnosed comorbidities, n (%) | | |
| - 0 | 76 | (30.4) |
| - 1 | 78 | (31.2) |
| - 2 | 41 | (16.4) |
| - 3 | 30 | (12.0) |
| - >3 | 25 | (10.0) |
| SF-36 Physical Component Summary Score, 0–100 | 47.2 | (9.0) |
| SF-36 Mental Component Summary Score, 0–100 | 46.9 | (11.1) |

Seventy-four percent of participants classified themselves as New Zealand European, and the remainder were Maori (12%), Pacific peoples (3%), or other ethnicities (11%). Seventy percent had one or more comorbidities. Younger age (p=0.01), female

sex (p=0.04), low high-density lipoprotein (HDL) cholesterol levels (p=0.003), and high systolic blood pressure (p=0.002) were significantly associated with higher BMI tertile, but no significant associations were seen with other potential confounders examined including SES, ethnicity, smoking status, and comorbidities (although they were included as covariates in the analyses).

**HRQoL**—Complete SF-36 questionnaires were available for 248 (99%) participants at baseline and 156 (62%) at follow-up. The mean (SD) PCS score for participants was 47.2 (9.0) and mean MCS score was 46.9 (11.1). Participants in the highest BMI tertile reported significantly lower mean [SD] PCS scores (44.1 [10.3]) compared with those in the middle (48.0 [7.5]) and lowest BMI tertiles (49.2 [8.3]) (p=0.01), but no significant differences were seen in MCS scores across tertiles (p=0.65) (Table 2).

**Table 2. Effect of baseline body mass index on baseline health related quality of life**

| Variable | BMI Tertile | | | |
|---|---|---|---|---|
| Domain, mean (SD) | 28–32 kg/m$^2$ (n=83) | 32.1–37 kg/m$^2$ (n=85) | >37 kg/m$^2$ (n=80) | p value |
| Physical functioning | 82.6 (17.2) | 81.2 (15.2) | 69.6 (22.8) | 0.004 |
| Role physical | 78.7 (30.9) | 76.5 (31.0) | 71.3 (36.0) | 0.50 |
| Bodily pain | 72.0 (24.2) | 71.7 (21.2) | 66.2 (23.1) | 0.45 |
| General health | 70.7 (19.6) | 71.3 (17.8) | 60.6 (21.4) | 0.09 |
| Vitality | 56.6 (21.6) | 57.6 (19.1) | 48.5 (20.0) | 0.26 |
| Social functioning | 84.6 (21.12) | 85.9 (19.6) | 77.8 (24.2) | 0.50 |
| Role emotional | 73.9 (36.8) | 78.0 (35.5) | 79.2 (32.4) | 0.55 |
| Mental health | 75.0 (15.6) | 78.0 (14.2) | 74.2 (16.8) | 0.46 |
| Physical component summary score | 49.2 (8.3) | 48.0 (7.5) | 44.1 (10.3) | 0.01 |
| Mental component summary score | 46.4 (11.8) | 48.3 (10.3) | 45.8 (11.0) | 0.65 |

Analyses adjusted for age, gender, ethnicity (European/Non-European), socioeconomic status (using the New Zealand Socio-economic Index)[23], comorbidities, baseline systolic blood pressure, and baseline HDL-cholesterol level.

Significantly lower PF scores (p = 0.004) were also reported by those in the highest BMI tertile (69.6 [22.8]) compared with those in the middle (81.2 [15.2]) and lowest (82.6 [17.2]) tertiles. There was a general trend for people in the highest tertile to have lower scores for most domains but differences were not statistically significant for domains other than PCS score and PF domain (Table 2).

Comparison of standardised (for age and sex) mean SF-36 domain scores reported by study participants with the New Zealand population norms revealed significantly lower mean scores in all domains except in the case of MH (Figure 1). The largest differences were seen in the VT (16.2), BP (13.5), and RP (12.3) domains, but substantial differences were also seen in the GH (10.9), PF (9.7) and RE (9.1) domains, with more modest differences seen in the SF (4.8) domain.

**Figure 1. Standardised comparison of health related quality of life reported by overweight and obese study participants with New Zealand population norms**
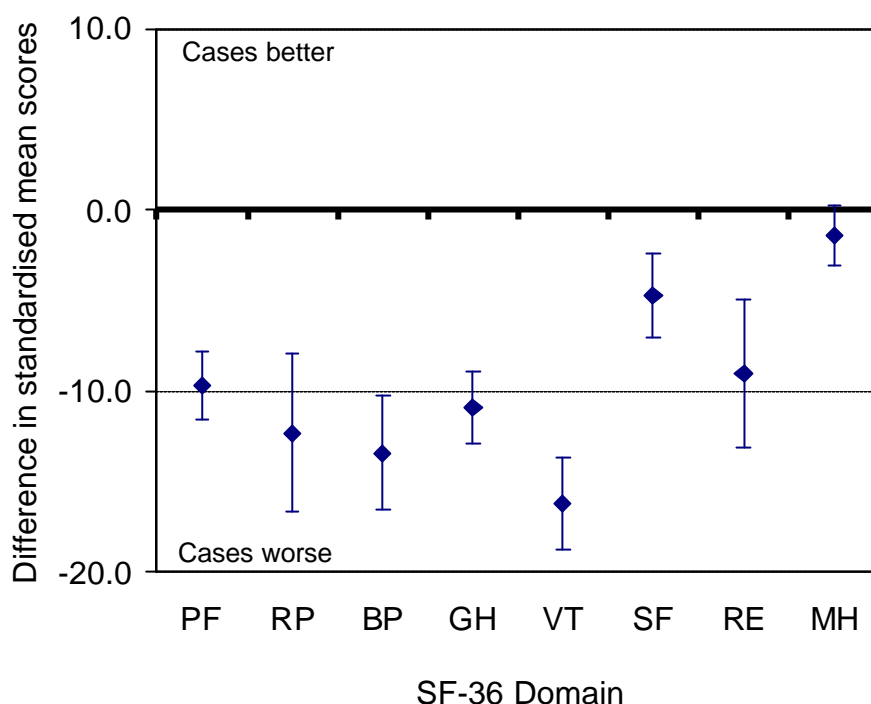


PF=physical function; RP=role limitations related to physical problems; BP=bodily pain; GH=general health; VT=vitality; SF=social functioning; RE=role limitations related to emotional problems; MH=mental health; Point estimates are standardised mean differences and bars are 95% confidence intervals around the differences.

The effect of weight change from baseline to 6 months on the PCS score was evaluated in an ITT analysis. Twenty-three participants lost more than 5% of their baseline weight (mean=8.2%) over the 6-month study period, 95 lost less than 5% (mean=1.7%), while 123 did not change or gained weight (mean=+1.7%).

After controlling for age, gender, ethnicity, SES, comorbidities, baseline SF-36 scores, baseline SBP, and baseline HDL-cholesterol, the effect of weight change on PCS was evaluated but no significant effect across the three categories was seen (p=0.16). A sensitivity analysis limited to participants with complete data (n=150) was also undertaken, but no significant effect of weight change was seen in this analysis either (p=0.43).

## Discussion

These results demonstrate that overweight and obese New Zealand adults experience significantly impaired HRQoL compared to the population norms, particularly in the vitality, bodily pain, and role physical domains. Small reductions in body weight did not significantly improve HRQoL in this substantially overweight population.

The strengths of this study include the large, well-defined study population and the use of the SF-36 questionnaire to measure HRQoL. The SF-36 is widely used,[17] and norms (by age and sex) have been produced for New Zealand and other populations allowing international comparisons.

However, our study population may differ from overweight and obese New Zealand adults generally because study participants were relatively healthy and ambulant. Their HRQoL scores might be expected to reflect this and perhaps be higher than those of overweight and obese New Zealanders in general. However, it is equally possible that these trial participants may have been particularly keen to lose weight, thus biasing the sample towards dissatisfaction and lower HRQoL. In addition, the SF-36 (a generic measure) may have failed to evaluate the impact that excess weight would have on obesity-specific aspects of HRQoL. This might explain why no effect of BMI was detected on MCS despite it being recognised that people who are overweight or obese are more likely to suffer from discrimination[13] and depression.[15]

An additional point to note is that a crude classification (sum of comorbidities) similar to that used in other studies[24,25] was used to adjust for the effect of comorbidities, but it is possible that there could be a differential impact of comorbidities associated with pain (e.g. osteoarthritis) and those that are asymptomatic (e.g. hypertension), although unfortunately it was beyond the scope of this small study sample to weight individual comorbidities accordingly.

Finally, the New Zealand population norms for HRQoL are based on data collected in 1996/7 and the indications are that the population prevalence of overweight and obesity has increased since then.[26] This shifting baseline may have influenced the comparison of HRQoL data collected in our weight loss study (2001/2) with the population data (1996/7).

Mean PCS and MCS scores in this group of overweight and obese adults were 47.2 (9.0) and 46.9 (11.1) respectively. Because the standardised means of the summary scores are set at 50, these scores indicate some impairment in both physical and mental domains. This places obesity in the same category as chronic conditions such as visual impairments, cerebrovascular and/or neurological conditions, cancer, and respiratory conditions, which have also been found to have a negative impact on both summary scores.[27]

Participants with BMI levels exceeding 32 kg/m$^2$ also had significantly lower PCS scores of approximately 5 points, when compared with those who had a BMI in the lowest tertile (28–32 kg/m$^2$). It is suggested that a difference of 5 points in summary scores is clinically and socially significant (John Ware, personal communication, 2001).

Age and sex standardised analyses demonstrated significant differences in HRQoL domain scores between this group of overweight adults and New Zealand population norms of up to 16 points, with differences being most pronounced with respect to the vitality, bodily pain, and role physical domains. It is uncertain what difference in domain scores is clinically significant, but differences of 9–16 points (compared with population norms across most domains) suggests impairment in HRQoL that is socially as well as statistically significant.[18]

We found no significant association between reduction in body weight and HRQoL. This finding is contrary to previous studies,[28,29] that found a linear relationship between HRQoL and weight loss. There are two reasons why this discrepancy may have occurred. Firstly, previous study participants on average lost 10%[29] to 17%[20] of their weight over a 1-year period, whereas only a minority (23) of our study participants lost more than 5% body weight over the 6-month period.

Several studies have confirmed that there is a dose-response effect of weight loss on HRQoL[30,31] so it seems likely that people who are very overweight and obese may need to lose in excess of 10% of their body weight in order to experience a positive impact on HRQoL. Secondly, previous studies[28,29] used obesity-specific measures of HRQoL, whereas we used a generic measure that may be less sensitive to obesity specific issues.

It seems likely that differences in proportional weight loss are most likely to account for discordance because Fontaine et al used the SF-36 questionnaire and found that weight loss was significantly associated with higher scores relative to baseline on the PF, RP, GH, VT, and MH domains.[32] The participants in the study by Fontaine et al also lost on average 10% of their body weight over the 13-week treatment programme.

The prevalence of overweight and obesity is increasing rapidly in New Zealand[33] and the results of our study confirm that obesity has a significant negative impact on HRQoL in addition to the known increase in risk of disease and death, suggesting that the psychosocial consequences of obesity should also be considered in the management of obesity. However, our results also imply that small reductions in weight have little impact on HRQoL in people who are substantially overweight, supporting the urgent need for more effective interventions to prevent and treat overweight and obesity in New Zealand.

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## Gastric bypass surgery for severe obesity: what can be achieved?

Mike He, Richard Stubbs

### Abstract

**Background** As severe obesity becomes an increasing problem, it is important that the role of surgery in the management of the condition becomes better understood and recognised. This study reports the outcome of a large series of gastric bypass operations performed for severe obesity by a single surgeon.

**Methods and Results** Between 1990-2002, 310 patients underwent gastric bypass surgery with all data being collected prospectively and recorded on a computerised database. Mean preoperative weight and percentage excess weight were 132 kg and 119%, respectively. There was no 30-day mortality and only one life threatening complication. Re-operations were required in 43 patients, most of which were related to ring removal or staple line disruption seen in the earlier form of gastric bypass. These problems are largely overcome with the currently performed technique.

Percentage excess weight loss at 1, 2, 3, 4 and 5 years was 73%, 73%, 66%, 64%, and 59% respectively. Forty-nine of the 52 patients with type 2 diabetes were cured following surgery and 87% of those with hypertension resolved or improved their condition. Dyslipidaemia resolved or improved in 87% and asthma was cured or improved in 89% of those affected. 61 patients had symptoms of obstructive sleep apnoea prior to surgery and in 51 of these symptoms resolved following surgery.

**Conclusion** This study confirms that gastric bypass is a very effective treatment for severe obesity and can be performed with a high level of safety. Gastric transection is an important component of the operation. The majority of important comorbidities seen before surgery either resolve or improve following surgery. Bariatric surgery is establishing itself as a very important and satisfying branch of upper gastrointestinal (GI) surgery.

Severe obesity is becoming a major problem in New Zealand, as well as other developed countries. It is not only associated with social and psychological conflicts, but is also associated with an increased risk of a number of serious diseases including diabetes, hypertension, dyslipidaemia, heart disease, and obstructive sleep apnoea. Direct health care costs in New Zealand attributable to obesity were estimated some years ago to be NZ$135 million, which represented about 2.5% of total healthcare costs.[1]

More recently, the overall costs of obesity in the United States (US) were estimated to be almost 17% of total health care costs.[2] Non-surgical approaches to the treatment of obesity have consistently been found to be ineffective in achieving significant, long-term weight loss. Weight loss in excess of 10% is seldom achieved, and weight is often regained within 1 year, and almost always within 5 years.[3] While medications such as subitramine (Reductil) and orlistat (Xenical) achieve an average weight loss

of 8–10% over a 6-month period, weight regain almost always occurs with cessation of medication.

Therefore, bariatric surgery has been pursued and developed over the last five decades and has emerged as a reliable means of major, long-term weight loss. In 1991, the National Institutes of Health in the US proposed that bariatric surgery should be considered for those with a body-mass index (BMI) over 40, or over 35 if there were coexisting comorbidities.[4] This recommendation was adopted by the National Institute of Clinical Excellence (NICE) in the UK in 2002[5] and is gaining steadily in acceptance throughout the World.

Gastric bypass was first proposed in 1967[6] and has become one of the two most commonly performed bariatric operations today. Indeed, it is widely considered to be the gold standard of bariatric surgery. More reports are appearing in the World literature to indicate bariatric surgery not only achieves substantial and sustained weight loss but also improves and often resolves associated comorbidities.[7–12] There does, however, remain a need for this information to be more widely promulgated and accepted.

This report focuses on the senior author's (RS) extensive experience with gastric bypass surgery, in terms of weight loss and improvement in comorbidities for his patients.

## Methods

310 consecutive patients, who underwent gastric bypass Roux-en-Y surgery at Wakefield Hospital (Wellington, New Zealand) between June 1990 and August 2002, are the subjects of this study. Two similar types of gastric bypass surgery, initially described by Fobi (from Los Angeles, US),[13,14] were performed during this time. The first type of surgery, termed a silastic ring gastric bypass (SRGB), is shown in Figure 1(a), and has been described in detail in a previous publication from our group.[15]

The later modification, known as a Fobi pouch, is shown in Figure 1(b), and mainly differs from SRGB in that gastric transection is performed along the staple line. The Fobi pouch was performed as follows. Surgery was performed under general anaesthesia with epidural analgesia through an upper midline incision. A window was produced adjacent to the lesser curve of the stomach, 9 cm from the angle of His, and a passage was created from this point, behind the stomach, to the angle of His.

A TCT 10® stapler (Johnson & Johnson) was placed between these two points, and its position adjusted prior to firing so as to produce a blind lesser curve stomach pouch approximately 9 cm long and 1.5–2 cm in diameter. Firing of the stapler achieves gastric transection with 2 rows of staples on either side. The staple line on the bypassed stomach was oversewn with a continuous 2/0 Ethibond suture.

A 70 cm Roux loop of jejunum was fashioned with the entero-entero anastomosis being performed with two layers of 2/0 chromic catgut at a convenient point 40–60 cm from the ligament of Treitz. The Roux loop was passed in a retrocolic, retrogastric fashion to lie alongside the newly created lesser curve pouch separating this from the oversewn distal stomach.

The Roux loop is sutured with 2/0 prolene to the lesser curve pouch in two layers, in such a way as to create a serosal patch over a buried staple-line. A 6.0 cm (for age under 50 years) or 6.5 cm (for age over 50 years) length of 8F silastic rubber tubing was passed circumferentially around the lesser curve pouch 5 cm from the angle of His—thereby defining the size of the pouch above the ring (approximately 10–15 ml).

This ring was fixed in place with an internal 2/0 prolene suture. The prolene sutures creating the serosal patch above the ring were continued to a point around 1 cm beyond the silastic ring, at which point a 2 layer end-to-side gastro-jejunal anastomosis was created over 1–1.5 cm, after removal of a portion of the staple line. The inner layer was fashioned with all-coats 2/0 chromic catgut, and the outer seromuscular layer completed with 2/0 prolene.

Mesenteric defects were closed and cholecystectomy was performed if gallstones were present. The abdominal cavity was lavaged with normal saline, and the abdomen was closed with a mass 1 nylon suture. To dislodge all loose fat, the subcutaneous fat layer was vigorously lavaged with saline, and the skin was closed with subcuticular Vicryl® suture (Johnson and Johnson) and steri-strips.

All patients received a single intra-operative dose of a prophylactic antibiotic (usually Cefotetan 2 g), and were commenced on preoperative Clexane® (Aventis) 20 mg subcutaneously for DVT / PE prophylaxis. The latter was continued daily after surgery, till discharge. Epidural analgesia was continued postoperatively for 4 days.

Urinary catheters were not routinely employed and patients were initially mobilised off the side of the bed 4 hours postoperatively. All the operations were carried out by the senior author (RS). Obesity was assessed by body mass index (BMI), calculated using the formula: BMI=body weight (kg)/height $(m)^2$ and percentage excess body weight (%EW), was calculated from New York Metropolitan Life Insurance Company tables (1959).

A prospective, computerised database has been maintained since 1990 recording all relevant preoperative details (including weight, height, BMI, %EW, and existence of comorbidities), operative details, and follow-up data. Patients were seen postoperatively at 3-monthly intervals for the first year, at 6-monthly intervals for the second year, and annually thereafter. Detailed preoperative blood tests included fasting lipid profiles, HbA1c, fasting glucose in known diabetics and glucose tolerance test in all other patients.

Data collected during the follow-up included weight, BMI, %EW, and changes in comorbidities. Blood tests were taken prior to each follow-up visit to monitor, among other things, fasting lipids, HbA1c, fasting glucose, iron, folate, and vitamin B12 levels. All patients were commenced on a multivitamin tablet at the first postoperative visit and recommended to remain on this for life. Supplementation of Vitamin B12, folic acid, and iron was given as indicated by blood tests. In the case of vitamin B12 and folic acid, supplements were maintained life-long once a deficiency appeared. Where regular follow-up had been lost or information was lacking, this was gathered by a phone call or mailed questionnaire.

The comorbidities of particular interest in this study were: hypertension, diabetes, dyslipidaemia, asthma, and obstructive sleep apnoea (OSA). Hypertension was deemed to be present if patients were taking antihypertensive medication or had a diastolic pressure in excess of 90 mmHg at hospital admission. Asthma was defined by prior history and the taking of bronchodilator medication. Diabetes was determined by history or preoperative oral glucose tolerance test. OSA was deemed present if patients had been formally diagnosed prior to consultation or they admitted to symptoms suggestive of the diagnosis—such as snoring, disturbed, or restless sleep with apnoeic episodes accompanied by daytime somnolence and chronic fatigue.

Dyslipidaemia was judged present if one or more of the following abnormalities existed on fasting lipid profile. Total cholesterol >5.2 mmol/L, triglycerides >2.0 mmol/L, or total cholesterol/HDL cholesterol >5.0 mmol/L.

Changes in comorbidities were classified as follows:

- Resolution—normalisation of the comorbidities without requirement for medication.
- Improvement—better control of the comorbidities with the same or reduced medication requirement.
- No change—no evidence of resolution or improvement.

Staple line disruption was suspected when there was ether substantial weight regain or a marked change in eating capability. It was frequently associated with pain related to the presence of stomal ulceration. The diagnosis was confirmed in all cases by either barium meal or gastroscopy.

All numerical data were expressed as mean ± standard error with a range. Changes in lipid profiles were analysed using a paired student t test. A p value of <0.05 was taken to indicate statistical significance. Weight loss is demonstrated by reduction in weight, BMI, and the percentage of excess weight lost (%EWL).

**Figure 1. Diagrammatic representation of the gastric bypass operations; (a) Silastic ring gastric bypass (SRGB), (b) Fobi pouch**



(a)   (b)
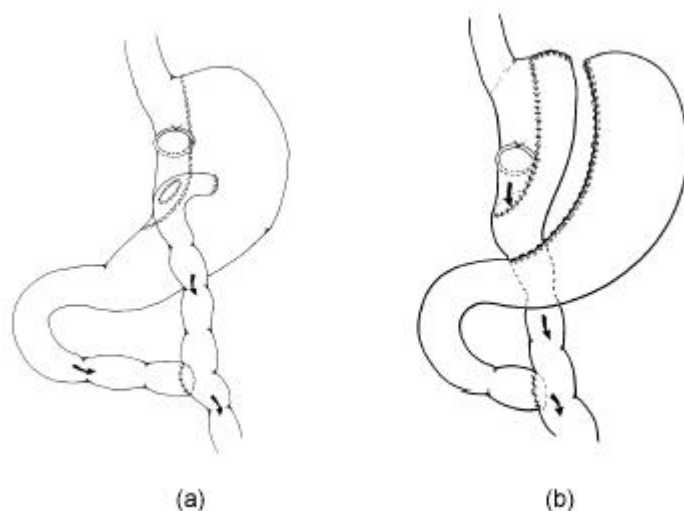
## Results

There were 71 males and 239 females aged between 15 and 66 years (mean 41.9±0.6). Of the 310 patients, 175 had an SRGB and 135 had a Fobi pouch procedure. The latter were all performed after August 1997. Mean operating time (excluding anaesthetic time) was 143±2 min (60–420), and blood loss was 279±20 ml (100–4000).

Fifty-three patients had cholecystectomy and 1 had splenectomy (for bleeding) during the surgery. There was no 30-day mortality. The median hospital stay was 7±0.1 days (5–14). A pulmonary embolus in one patient was the only life threatening postoperative complication. Other perioperative complications are shown in Table 1.

**Table 1. Postoperative complications in 310 patients**

| Complication | n |
|---|---|
| Pulmonary embolus | 1 |
| Wound infection | 30 |
| Atelectasis | 10 |
| Chest infection | 7 |
| Congestive heart failure | 2 |
| Urinary tract infection | 1 |
| Deep vein thrombosis | 1 |

The mean follow-up period is 3.5 years (1–12). Only two patients have had no follow-up. 221 patients (87%) have been followed for over 2 years and 87 patients (27%) for at least 5 years. 181 patients (58%) developed vitamin B12 deficiency during the period of follow-up (usually within 2 years) requiring institution of regular vitamin B12 injections. 134 (43%) patients developed folate deficiency requiring supplementation, and 175 (56%) required occasional iron supplementation.

A total of 49 re-operations for related issues were required in 43 (14%) patients during the period of follow-up as outlined in Table 2. The majority of these were in patients who underwent SRGB, with only 7 being in those who had a Fobi pouch. A total of 24 patients (14%) who had SRGB are known to have had partial staple line disruption of whom 19 have had revision surgery with gastric transection (i.e. conversion to Fobi pouch).

Two episodes of ring erosion occurred, both in association with staple line disruption and ulceration. Removal of the silastic ring was undertaken to improve quality of eating and was most often required for those with the smaller diameter ring as shown in the Table 2. A total of 20 patients (6.5%) required cholecystectomy for symptomatic gallstones during the follow-up period. This was undertaken and accomplished laparoscopically in most instances.

**Table 2. Related re-operations required during follow-up period (according to operation type)**

| Re-operation type | SRGB (n=175) | Fobi pouch (n=135) |
|---|---|---|
| Revision | 19 | 0 |
| Repair of incisional hernia | 7 | 0 |
| Ring removal: | | |
| 5.5 cm (n=66) | 10 | N/A |
| 6.0 cm (n=165) | 4 | 5 |
| 6.5 cm (n=77) | 1 | 1 |
| Laparotomy for bowel obstruction | 1 | 1 |
| **Total** | **42 (24%)** | **7 (5%)** |

SRGB= Silastic ring gastric bypass.

Mean ± SE preoperative weight, BMI, and % excess weight were 132±2 kg (72–360), 46.3±0.5 (28–99), and 119±2.4 % (33–377), respectively. The gastric bypass operation produced excellent weight loss, with a maximum loss being achieved after 18 months (as shown in Figure 2) and good maintenance of weight loss thereafter.

Percentage excess weight loss at 18 months ranged between 36% and 122% with only 18/244 (7.3%) obtaining less than 50% excess weight loss at this time point. Mean±SE percentage excess weight loss at 1, 2, 3, 4, and 5 years after surgery was 72.7±1.2, 73.1±1.1, 66.1±1.6, 63.9±2.2, and 58.9±2.4. Not surprisingly, better weight loss was achieved and maintained in those patients who did not experience staple line disruption, as indicated in Figure 3.

Prior to surgery, 52 patients had type 2 diabetes; 25 were taking oral hypoglycaemic agents, 16 were taking insulin, 3 were 'diet controlled,' and 8 were only diagnosed by routine preoperative testing. Following surgery all but three were completely cured of their diabetes, as evidenced by normal fasting blood glucose and HbA1c levels. Of the three in whom diabetes did not resolve, two required insulin and one required oral hypoglycaemic medication, but in all instances at much reduced doses.

**Figure 2. Indices of weight loss after gastric bypass; (a) mean weight loss (kg) after gastric bypass in 310 patients; (b) mean BMI after gastric bypass in 310 patients, (c) mean % excess weight loss after gastric bypass in 310 patients**

**(a)**
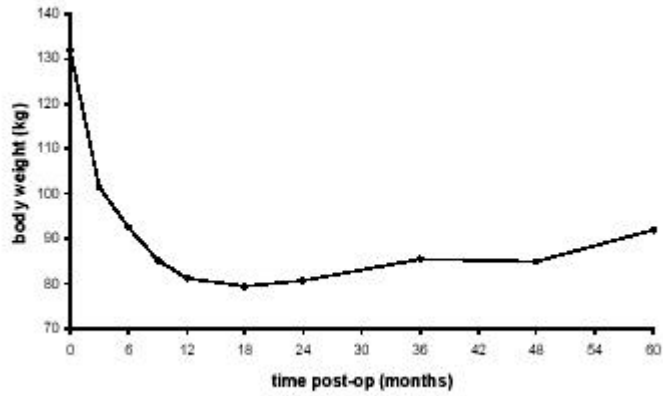


**(b)**
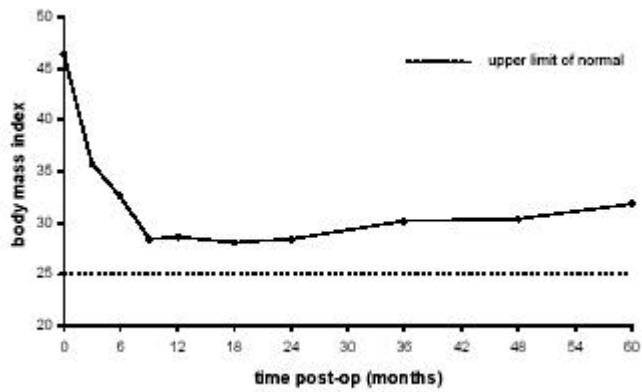


**(c)**
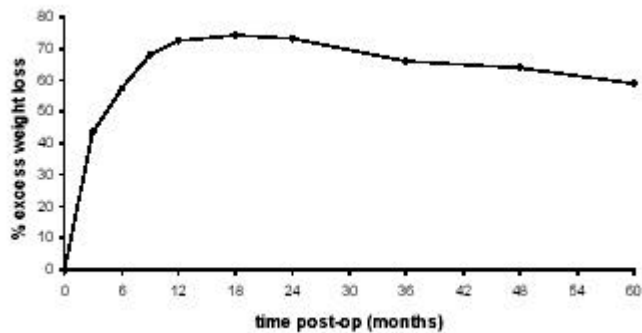
**Figure 3. The effect on weight loss after gastric bypass of staple line disruption; (a) mean weight (kg), (b) mean BMI, (c) mean % excess weight loss**

**(a)**
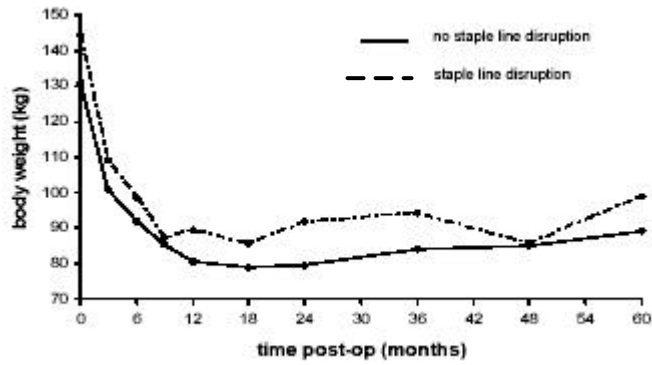


**(b)**
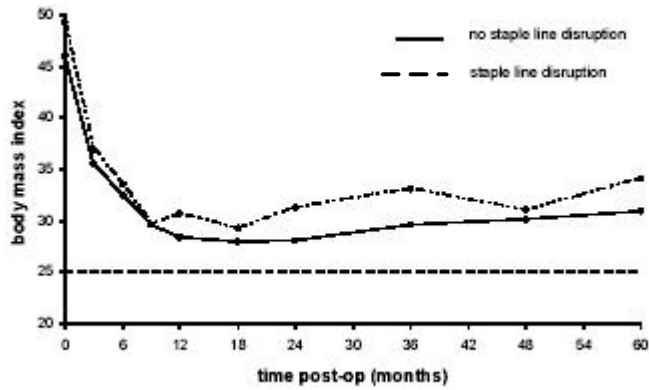


**(c)**
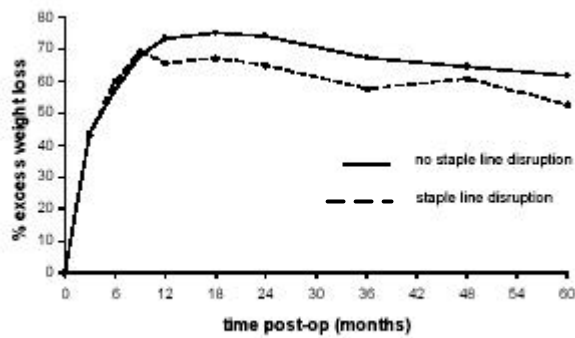
One patient who had diet controlled diabetes prior to surgery has been lost to follow-up (ie has never attended follow-up or been contactable). Fifty-six patients (18%) had abnormal glucose tolerance tests prior to surgery and none of them have developed type 2 diabetes during the period of follow-up. 73.8% (214/290) had dyslipidaemia before surgery. Postoperatively, 48.3% (85/176) were completely cured, 38.6% (68/176) improved, 12.5% (22/176) got worse, and 0.5% (1/176) remained unchanged. Changes in the mean fasting lipids over the first 12 months of follow-up are shown in Table 3. One hundred and fourteen patients were hypertensive before surgery of whom follow-up is available in 111. Following surgery, hypertension resolved in 69 (62%), improved in 27 (24%), remained unchanged in 14 (13%) and became worse in one (1%).

**Table 3. Mean ± SE fasting serum lipids before surgery and 12 months later.**

| Serum lipids | n | Preoperative | 12 months postop | |
|---|---|---|---|---|
| Total cholesterol (mmol/L) | 161 | 6.31±0.07 | 5.39±0.06 | p<0.001 |
| Triglyceride (mmol/L) | 73 | 3.56±0.40 | 1.46±0.08 | p<0.001 |
| Total cholesterol / HDL (mmol/L) | 96 | 6.81±0.16 | 4.01±0.11 | p<0.001 |

HDL=high-density lipoproteins.

Eighty-two patients had asthma prior to the surgery of whom we have follow-up information for 76. Following surgery, 47 (62)% have been cured, 21 (28%) have improved, and 8 (10%) remained unchanged. Sixty-one patients had obstructive sleep apnoea (OSA) prior to surgery, and we have follow-up information on 60 of them. Fifty-one patients (85%) had resolution of all symptoms following surgery, 8 (13%) were much improved, and 1 (2%) was unchanged. These changes in comorbidity are illustrated in Figure 4.

**Figure 4. Changes in major comorbidities after gastric bypass surgery**
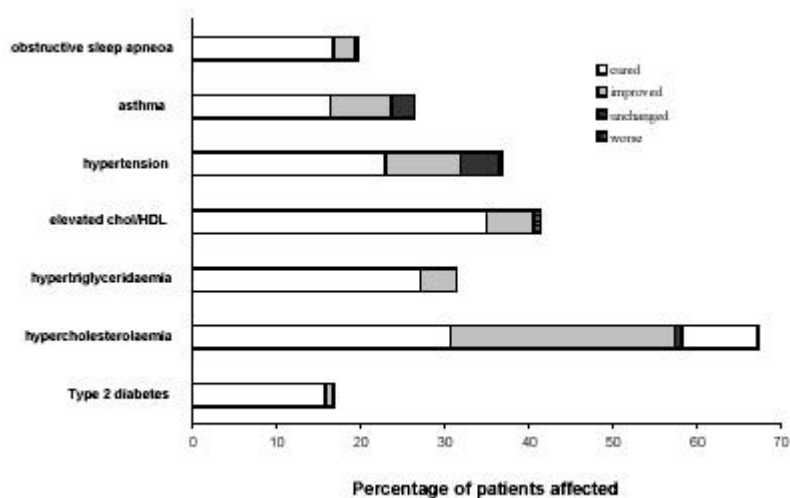


## Discussion

Bariatric surgery has not enjoyed a good reputation with surgeons over the years, and has been practiced by only a small minority of surgeons. Severe obesity has been regarded as a self-inflicted state, which should be addressed by the affected individual—and surgery has been thought to be inappropriate, hazardous, and to yield disappointing results. However, with the passage of time, all of these beliefs are changing. We are beginning to better understand that obesity is a disease state with devastating effects on quality of life and life expectancy, and that at the present time surgery is the only means of providing reasonably reliable and lasting weight loss. This report provides further evidence to rebuff the misconceptions held by many people regarding gastric bypass surgery, and provides further compelling evidence of the benefits afforded by the surgery.

Gastric bypass is one of two main types of bariatric surgery commonly performed today, and is considered by many to be the 'gold standard'. The other type is represented by the laparoscopic placement of an adjustable gastric band around the stomach just below the oesophagogastric junction to restrict eating capacity.

Gastric bypass surgery was first reported in 1967 by Mason,[6] but has since been through many modifications that have made it a safer and more reliable procedure. The most important of these modifications were being mooted in the late 1980s and the early 1990s and entailed creation of a vertically disposed lesser curve pouch, which distends much less readily than the previously used horizontal pouch based on the greater curve,[13] and transection of the stomach along the staple line[14]—to overcome the propensity for staple line disruption seen in 10–48% of cases[16,17] where transection is not undertaken.

Yet another modification (also advanced by Fobi) in the late 1980s, and adopted by us at the Wakefield Gastroenterology Centre, entails the placement of a silastic ring around the lesser curve pouch to permanently fix the size of the outlet of the pouch.[13] Most surgeons have been loathe to use a ring in this way, because of their previous

bad experience with the rings and bands applied in vertical banded gastroplasty (VBG) operations. In those operations, the ring/band size was in the range 4.5–5.0cm circumference and proved too limiting for many individuals, leading to frequent regurgitation or vomiting, and erosion into the lumen in others.

Fobi initially proposed a 5.5cm circumference ring for the SRGB operation, but we have previously reported that a 6.0 cm ring was preferable because it permitted a better quality of eating.[18] The present report indicates a need for removal of the 5.5 cm ring in 15% of patients compared with 4.5% of those with a 6.0 cm ring.

We currently employ a 6.0 cm ring for those patients aged under 50 years, and a 6.5 cm ring in those aged over 50 years, in the belief that gastric motility may be less effective in the older age group. Certainly, we now see very few problems attributable to the ring, and believe its presence will help promote long-term weight control, by fixing the size of the gastric outlet. Band erosion in the present series was seen in only 2 patients (0.64%) in association with staple line disruption and ulceration. Fobi has reported a band erosion rate of 1.63% in almost 3000 patients who underwent SRGB or Fobi pouch procedures.[19]

There is now abundant published evidence to indicate the effectiveness and safety of gastric bypass surgery. Mortality rates are generally less than 1% and median percentage excess weight loss after 2 years is in the range 65 to 75%.[7,16,20–22] There is similar abundant published evidence to indicate the effectiveness and safety of the laparoscopic banding procedure.[23–25] For this latter procedure, mortality is similar (generally less than 1%), and median percentage excess weight loss after 3-4 years is between 50 and 60%.

There is uniform agreement in the literature that gastric bypass achieves a quicker and greater weight loss than laparoscopic banding. Experience indicates that a significant but variable proportion of patients require band removal over time[26] and that the range of weight loss achieved by gastric banding is wider than that following gastric bypass—with as many as 10% losing very little weight, making it a rather less reliable procedure than gastric bypass.

The present report demonstrates clearly the benefit of gastric transection and of using a larger ring, in terms of reducing the need for later re-operation or revision.

There is growing interest, particularly in the United States, in the performance of gastric bypass laparoscopically. There is a rather long learning curve for this complex procedure during which time potentially serious complications are encountered with a frequency several times that for competent open surgery.[27–32] However, it is being performed safely and in large numbers in many units, and the reported experience is of favourable outcomes.[29,33–35]

Wound and abdominal wall complications are diminished by laparoscopic performance of the surgery, but other complications such as bowel obstruction, gastrointestinal tract haemorrhage, and stomal stenosis are greater[36] and the long-term results have yet to be fully evaluated. The results of the present series indicate that open surgery can be accomplished with very few early complications, and (if a large ring is used and gastric transection is performed) late complications and re-operations can also be very few. Thus, based on these findings, we believe open gastric bypass should for the present at least be retained as the gold standard bariatric operation.

There is an acknowledged heightened risk of gallstone formation during the rapid weight loss after bariatric surgery[37] and some researchers have suggested the gallbladder should be routinely removed.[38] Our own experience is that it is worth removing the gallbladder if stones are encountered (12% of patients) and that adoption of this practice leads to fewer than 10% of patients requiring cholecystectomy following gastric bypass.

The weight loss that follows bariatric surgery is life-changing for most patients. Self esteem and confidence grow immeasurably, with accompanying improvement in most psychosocial parameters and quality of life.[39–41]

In addition, there is also a growing awareness and acknowledgment of the almost universal improvement in serious comorbidities seen after bariatric surgery. This is clearly shown in the present study, and is particularly seen in the cluster of disease states associated with insulin resistance—including hypertension, diabetes, dyslipidaemia, heart disease (known collectively as the metabolic syndrome or Syndrome X), polycystic ovary disease, and gout.

While the mechanism for this improvement in insulin resistance and these comorbidities remains unproven, there has been speculation that changes in gut hormone profiles after gastric bypass may be important.[8,42–45] In addition, marked improvements occur in respiratory status including resolution or improvement in asthma in the majority of affected individuals[7,12] and the resolution of obstructive sleep apnoea.[46,47]

Mobility is often threatened in the severely obese by problems with the back and the weight bearing joints, particularly the knees and hips. These problems are also seen to improve dramatically after major weight loss, such as is achieved by surgery.

Our study, like many others, has shown that gastric bypass surgery, and particularly the more recent modifications of this procedure, is a safe and highly effective means of achieving substantial and reliable long-term weight loss. Furthermore, important and previously unattainable benefits in terms of life-impairing and life-shortening comorbidities are frequently seen following the surgery.

Bariatric surgery is proving to be a cost-effective alternative to non-surgical treatment, providing substantial life-time benefits[48] and should be available to and offered to those people suffering from severe obesity. The number of bariatric surgery programs, and the demand for bariatric surgery and surgeons trained to perform it, is increasing worldwide.[49] There is good reason for those practitioners interested in upper GI surgery to embrace this important and growing field of surgery.

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## Cost-effectiveness of physical activity counselling in general practice

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### Abstract

**Aim** To assess the cost-effectiveness of the 'Green Prescription' physical activity counselling programme in general practice.

**Method** Prospective cost-effectiveness study undertaken as part of a cluster randomised controlled trial with 12-month follow-up of 878 'less-active' patients aged 40-79 years in 42 general practices in the Waikato. The intervention was verbal advice and a written exercise prescription given by general practitioners, with telephone exercise specialist follow-up compared with usual care. Main outcome measures included cost per total and leisure-time physical activity gain from health-funders' and societal perspectives.

**Results** Significant increases in physical activity were found in the randomised controlled trial. Programme-cost per patient was NZ$170 from a funder's perspective. The monthly cost-effectiveness ratio for total energy expenditure achieved was $11 per kcal/kg/day. The incremental cost of converting one additional 'sedentary' adult to an 'active' state over a twelve-month period was NZ$1,756 in programme costs.

**Conclusion** Verbal and written physical activity advice given in general practice with telephone follow-up is an inexpensive way of increasing activity for sedentary people, and has the potential to have significant economic impact through reduction in cardiovascular and other morbidity and mortality.

There is now substantial epidemiological evidence to implicate a sedentary lifestyle as a risk factor for obesity, diabetes, cardiovascular disease, depression, bowel and breast cancer, and various other disease states.[1–4] Existing evidence suggests that at least 30 minutes of moderate activity on most days of the week is associated with significant health gains and has led to major position statements such as the 1996 US Surgeon General's report on physical activity and health.[4]

In New Zealand, one-third of adults do not undertake the recommended 2½ hours of moderate-intensity physical activity per week.[5] As a result, the Hillary Commission developed the Green Prescription physical activity counselling programme for New Zealand primary healthcare. A randomised controlled trial to assess the effectiveness of the programme in the Waikato region found that the programme was effective in increasing physical activity and improving quality of life over a 12-month period.[6] However, the cost-effectiveness of the intervention was not known.

The aim of this study was to calculate the incremental cost-effectiveness of the Green Prescription programme in increasing physical activity compared with 'usual care' in general practice, and to compare this with other community-based physical activity interventions reported in the literature.

## Methods

### Background

The cost-effectiveness analysis of the Green Prescription programme was incorporated prospectively into a cluster randomised controlled trial undertaken from mid-2000 to mid-2002.[6] General practices in the Waikato region of New Zealand were randomised to give the Green Prescription or 'usual care' to patients enrolled in the study. Baseline and 12-month follow-up measurements were taken at each practice by research staff. The cost-effectiveness analysis was undertaken from health funders' and societal perspectives. The Waikato Ethics Committee approved the study in 1999.

### Participants

Consecutive 40 to 79 year-old patients were screened at the reception area of 42 rural and urban general practices over a 5-day period. Those not achieving the recommended 2½ hours of at least moderate activity per week were invited to participate in a study involving a lifestyle intervention.

### Intervention

Study participants from intervention practices prompted the general practitioner or nurse to give verbal advice to increase physical activity with activity goals written on a Green Prescription. Patients from control practices received usual care. The Green Prescription was then faxed to exercise specialists in Sports Foundations who provided telephone support on three occasions over the following three months to each intervention patient and sent written material including newsletters.

### Measures

Primary outcome measures in the clinical trial were change in leisure-time physical activity, total energy expenditure, quality of life (using the SF36 scales), 4-year coronary heart disease risk, and systolic and diastolic blood pressure. A post-hoc analysis comparing the proportion of participants that achieved 2.5 hours of leisure activity was carried out to allow comparison with previous studies carried out in primary care.[7]

Primary outcomes measured for the cost-effectiveness study were the incremental cost of change in self-reported physical activity over 12 months. These outcomes included the cost per total energy expenditure gained, the cost per leisure moderate- and vigorous-intensity energy expenditure gained, and the incremental cost of moving one additional 'sedentary' person into the 'active' category (achieving 2½ hours of at least moderate-intensity leisure activity per week).

**Costs**—Green Prescription programme development costs incurred in previous years were obtained from the developers of the programme, the Hillary Commission, and were adjusted for inflation using the December consumer price index from each corresponding year compared with that of December 2001.[8] A discount rate of 5% was used to calculate present equivalent values of programme costs from 1996 to 2001.[9]

Programme delivery costs included general practitioner and practice nurse time, Sports Foundation exercise specialists, and Green Prescription resources. Delivery costs within the general practice were estimated using usual consultation charges for participating practices, national award rates for practice nurses, and the time, estimated as 7 minutes by general practitioners and 13 minutes by practice nurses, for programme delivery.[6] Charges for each general practice in the region were obtained at baseline and average charges calculated for each consultation type.

Actual regional Sports Foundation personnel and overhead costs associated with the programme were obtained from the Sports Foundation's accounting department for the year 2001/2002. Average wage costs rather than marginal costs were used as the exercise specialists were permanent staff of the Sports Foundation.

**Offset cost**—Self-reported costs to the individual associated with exercise were identified by study participants in a 12-month follow-up questionnaire and included exercise equipment purchased, sports club or exercise group subscriptions, travel expenses to and from exercise, and any other costs associated with exercise over the 12 months of the study.

Costs associated with primary and secondary healthcare utilisation and costs of time off work were also recorded. Primary healthcare offset costs were calculated for each participant for the 12 months prior to study enrolment and compared with the 12 months after study enrolment. Actual number and type of

general practice consultations were obtained from practice records. Actual government subsidies for each type of consultation were used and were adjusted for inflation.

Patient charges and subsidies vary. Average patient part-charges of participating practices were used for consultations of non-subsidised patients (A3) (NZ$35) and low-income (A1) or high-user patients (AZ) (NZ$20), and for accident-related consultations NZ($10). Government subsidies for each consultation were NZ$15 for A1 and AZ visits, and NZ$26 for all accident-related visits. Numbers of accident-related visits to physiotherapists, chiropractors, and osteopaths were obtained from patient questionnaires. These visits were subsidised at a rate of NZ$19 per visit, with an average patient surcharge of NZ$10.

Secondary care costs were established using each participant's national health index, a unique identifier in primary and secondary healthcare allowing tracking of individual's health care utilisation. Actual hospital inpatient, outpatient, and investigation costs for each patient from all public regional and base hospitals were obtained from the local district health board for the year prior to and the year following each patient's enrolment in the study. Costs for private hospital-use could not be obtained. However, self-reported private hospital admission-rates were recorded.

To calculate the cost of loss of productivity due to illness and accident for the year prior to baseline compared with the year after baseline, the change in the number of days of illness- and accident-related leave taken were obtained by self-report. The average wage for the June quarter from wages, salary, and self-employment for those in paid employment was NZ$121.80/day for 2000 and NZ$128.20/day for 2001.[10]

All costs were adjusted for inflation using the 2001/2000 consumer price index ratio to calculate the incremental change. All costs are reported as New Zealand dollars. Where comparisons with programmes from the United States or the United Kingdom were carried out, values were converted to the New Zealand dollar according to the exchange rate of December 2001.[11]

## Analysis

Total setup and programme administration costs were obtained to calculate programme cost per patient. Actual offset costs of primary and secondary healthcare utilisation, personal expenditure, and productivity changes were collected wherever possible. The differences in change in offset costs to the patient and health funder for intervention patients compared with control patients, with 95% confidence intervals, were calculated using a random effects generalised least squares regression model, where the general practice was entered as the clustering variable in STATA version 7.0.

Cost-effectiveness ratios were obtained by calculating programme costs per activity gain from a programme-funder perspective. These ratios were compared with those from other physical activity interventions reported in the literature. Sensitivity analyses were conducted using the confidence intervals for calculated physical activity gains as the relevant range.[7,12]

All analyses were carried out using an intention-to-treat approach, where no change from baseline was assumed in those who did not attend follow-up, except personal costs associated with exercise, where costs were assumed to be the mean of those in the equivalent group.

## Results

Table 1 shows the characteristics of the 878 study-participants from 42 practices.[6] Results from the randomised controlled trial, which achieved 85% follow-up at 12 months, showed a mean total energy expenditure increase of 9.4 kcal/kg/week (p=0.001) and leisure exercise increase of 2.7 kcal/kg/week (p=0.02), or 34 minutes/week more in the intervention group than in the control group (p=0.04).[6]

SF-36 scores of self rated 'general health', 'role physical', 'vitality', and 'bodily pain' improved significantly more in the intervention group (5.95, 10.53, 5.36, and 6.51, respectively) compared with the control group (1.60, 4.16, 3.06, and 2.50, respectively) (p<0.05).[6]

**Table 1. Baseline characteristics of less-active 40–79 year-old patients in general practice, by intervention and control group[6]**

| Patient Characteristic | Intervention Group Mean (SD) or N (%) [n=451] | Control Group Mean (SD) or N (%) [n=427] |
|---|---|---|
| Age, years | 57.2 (10.8) | 58.6 (11.5) |
| Systolic BP (mmHg) | 135.1 (19.6) | 135.4 (17.9) |
| Diastolic BP (mmHg) | 82.4 (12.2) | 81.8 (12.1) |
| Body mass index, kg/m$^2$ | 30.0 (6.7) | 29.9 (6.4) |
| CHD 4-year risk*, % risk | 5.7 (6.2) | 5.5 (5.8) |
| Total energy expenditure, kcal.kg$^{-1}$.day$^{-1}$ | 33.9 (6.0) | 33.7 (6.5) |
| Leisure physical activity[a], kcal.kg$^{-1}$.day$^{-1}$ | 0.9 (1.7) | 0.9 (1.6) |
| Leisure exercise[#], mins.day$^{-1}$ | 11.3 (21.7) | 12.0 (20.5) |
| Female participation: N (%) | 301 (67) | 281 (66) |
| Lower economic status:** N (%) | 205 (45) | 211 (49) |
| European: N (%) | 354 (78) | 324 (76) |
| Smokers: N (%) | 78 (17) | 76 (18) |
| Diabetes: N (%) | 46 (10) | 46 (11) |
| Hypertensive[$]: N (%) | 240 (53) | 220 (52) |
| Previous cardiovascular disease: N(%) | 93 (21) | 74 (17) |

This table is reproduced from: Elley CR, Kerse N, Arroll B, Robinson E. Effectiveness of counselling patients on physical activity in general practice: cluster randomised controlled trial. *BMJ* 2003;**326**:793-6; *Risk of 4-year coronary heart disease risk [13, 14] was carried out on a randomly selected sub-sample to contain costs (n=787) and a further 51 participants declined to have cholesterol testing done. [$]Hypertensive refers to a previous diagnosis of hypertension and taking antihypertensive medication or a mean blood pressure (BP) of greater than 150 mmHg systolic or 90 mmHg diastolic; [a]Leisure physical activity refers to the energy expenditure of all leisure-time physical activity considered moderate or vigorous by the respondent; [#] Leisure exercise refers to time spent in moderate (3.0-4.9 MET) and vigorous (≥5.0 MET) leisure-time activities undertaken at least once per 2 weeks. [15] **Economic status was measured at baseline by qualification for a low-income health subsidy card. (Forty-three percent of adults over 45 years of age in New Zealand qualify for this card.)

**Table 2. Offset costs per patient for the intervention group compared with the control group (intention-to-treat analysis)**

| Cost Variable (NZ$) | Intervention # Change [Yr2-Yr1] (95%CI) | Control Group # Change [Yr2-Yr1] (95% CI) | Between–Group * Difference (95% CI) |
|---|---|---|---|
| *Health-funder costs* | | | |
| Accident-related referrals** | $1.21 (-8.08–10.50) | $1.56 (-9.13–12.20) | -$0.36 (-14.43–13.72) |
| Non-accident related GP visits | -$4.01 (-7.98–-0.04) | -$0.05 (-7.15–7.05) | -$4.39 (-15.41–6.62) |
| Accident-related GP visits | $0.34 (-5.05–5.73) | $0.78 (-6.07–7.63) | -$0.45 (-9.09–8.20) |
| Hospital costs | $320.85 (-69–711) | $495.03 (108–882) | -$174.19 (-722.75–374.38) |
| *Patient costs* | | | |
| Accident-related referrals | $0.84 (-4.01–5.69) | $1.04 (-4.48–6.56) | -$0.20 (-7.50–7.10) |
| Non-accident related GP visits | -$7.24 (-16.80–2.37) | $0.89 (-11.00–12.80) | -$8.21 (-27.75–11.32) |
| Accident-related GP visits | $0.24 (-1.82–2.30) | $0.44 (-2.18– 3.06) | -$0.20 (-3.51–3.11) |
| Costs of exercise | $236.29 (192–281) | $209.37 (152–267) | $26.95 (-45.08–98.98) |
| *Productivity costs* | | | |
| Sick-days off work (accident and non-accident-related) | $42.19 (-166–251) | $37.47 (-78.20–153) | $1.21 (-522.06–524.49) |

*Adjusted for clustering; # Not adjusted for clustering; **Accident-related referrals to physiotherapy, osteopathy or chiropractor.

Ninety-five percent of intervention patients and 2.5% of control patients attending follow-up recalled receiving a Green Prescription in the previous 12 months, indicating a low level of 'contamination' of intervention.

The total discounted and annuitised national set-up and coordinating cost for the Green Prescription programme from mid 1996 to mid 2002 was NZ$2,861,016 (see Appendix 1). Approximately 34,708 patients received Green Prescriptions during that period. The programme set-up and coordinating cost per patient (excluding exercise specialist referral costs) was NZ$82.43 per Green Prescription recipient. The general practice-based delivery cost of the intervention and follow-up over the following 12 months was NZ$19.20 per patient (see Appendix 2). Of the 451 in the study, 410 (91%) were referred to the Sports Foundation exercise specialists. The total exercise specialist direct and overhead costs attributable to study patients was NZ$31,032.65 (see Appendix 3) or NZ$68.81 per intervention patient.

Table 2 shows the decreased healthcare costs per individual in the intervention compared with the control group, particularly in hospital costs, but with wide confidence intervals due to large individual variations in actual costs of hospitalisation. There was no significant difference in change in number or cost of days off work due to illness or accident between the groups for the year before and the year after the intervention. (Changes in rates of health care utilisation and days off work are presented in Appendices 4-6.) Personal exercise-related costs were NZ$26.96 per patient per year more in the intervention group (see Appendix 7).

Table 3 shows the cost from the programme-funders' perspective was NZ$170.43/patient/year. Table 4 shows the cost effectiveness ratios for the Green Prescription with sensitivity analyses compared with those of the 'Lifestyle' and 'Structured' Project Active exercise programmes.[12]

The proportion of participants in the intervention who achieved 2.5 hours of at least moderate activity per week increased by 14.6% (66/451) compared with 4.9% (21/427) in the control group (p=0.003).[6] Therefore, the incremental cost of converting one additional adult in the Green Prescription programme from sedentary to active over 12 months, compared with the control group, was NZ$1,756 in programme costs.

**Table 3. Incremental cost per patient of the Green Prescription programme, including programme and offset costs and savings (intention-to-treat analysis)**

| Description of costs | Incremental costs / patient in NZ$ (95% CI) |
|---|---|
| Green Prescription set-up and coordinating costs | $82.43 |
| Regional Sports Foundation support costs | $68.81 |
| General practice delivery of intervention costs | $14.59 |
| General practice follow-up support costs | $4.60 |
| **Total programme costs** | **$170.43** |
| Total patient offset costs | $18.62 (-55.63–92.88) |
| Total health funder offset costs | -$178.94 (-728.58–370.70) |
| Productivity offset costs (accident- and non-accident-related) | $1.21 (-522.06–524.49) |

* It was inappropriate to calculate total cost difference estimates taking offset costs into account because of the large confidence intervals and imprecision around the offset costs.

**Table 4. Cost-effectiveness ratios for the green prescription compared with project active 'lifestyle' and 'structured' physical activity promotion programmes**

| Monthly Incremental Cost Categories | Green Prescription Programme | | | Project Active | |
| --- | --- | --- | --- | --- | --- |
| | Programme funder's perspective[#] | Sensitivity analysis[1] | Sensitivity analysis[2] | 'Lifestyle' Program at 24 months* | 'Structured' Program at 24 months* |
| Cost of programme per participant per month | $14.20 | – | – | $41.26 | $118.62 |
| Cost of change in energy expenditure per kcal/kg/day | $10.59 | $6.57 | $24.91 | $48.11 | $170.80 |
| Cost of change in at least moderate intensity activity per kcal/kg/day | $37.37 | $20.46 | $205.80 | $43.31 | $358.43 |

[#]Offset costs are excluded from this analysis due to the large confidence intervals around the offset costs estimations. [1]Using upper 95% confidence interval estimate of physical activity gain.[6] [2]Using lower 95% confidence interval estimate of physical activity gain'; *Comparisons with the Project Active 6-month results were not used, as these values were even less cost-effective than at 24 months [12]; 95% confidence intervals were not available for Project Active estimates. All costs were converted to New Zealand dollars using the December 2001 exchange rate, $NZ1=$US0.4157 or $US1=$NZ2.4056.

## Discussion

This study represents one of the most comprehensive cost-effectiveness analyses of a physical activity programme in primary healthcare to date. The Green Prescription programme cost per patient was NZ$170.45 from a programme funders' perspective. Cost-effectiveness ratios were favourable compared with other physical activity interventions reported in the literature. Cost-effectiveness could not be calculated from a societal perspective because of large confidence intervals around offset costs.

### Limitations

Thirteen percent of patients attending their general practitioner during the recruitment phase were too ill to be screened, missed or refused screening for eligibility. In addition, one-third of those eligible declined to participate. There are few details available about those that chose not to participate, which may limit generalisability of results.

'Usual care' may have included some verbal advice about physical activity, 2.5% of control patients received a Green Prescription during the study year, and the control group also increased physical activity participation possibly due to participation in a trial about exercise. This may have diluted the effect of the intervention.

Private hospital cost data was not available. However, of the 337 participants that reported inpatient or outpatient attendance, only 41 used private hospitals (21 intervention and 20 control). When average daily public hospital costs were applied to self-reported days in private hospital for the year following the intervention, the total private hospital costs in the control group were substantially more than those in the intervention group (Appendix

5 footnote). Therefore hospital-related savings in the intervention group may have been greater than reported in this paper.

There are large 95% confidence intervals and imprecision around changes in major offset costs, particularly healthcare utilisation costs to the patient (NZ$18.62 [95% CI: -55.63–92.88]) and to the health funder (-$178.94 [95% CI: -728.58–370.70]), as well as productivity costs ($1.21 [95%CI: -522.06–524.49]). As a result, overall cost-effectiveness from a societal perspective could not be calculated.

Given this degree of variability in actual healthcare utilisation costs, it would take a very large study to have sufficient power to achieve confidence intervals that did not cross zero. Nevertheless, there was no evidence of increased costs in health care utilisation or loss of productivity as a result of the intervention.

## Strengths

This cost-effectiveness study was conducted prospectively, costing data collected was comprehensive, follow-up rates were high, and in almost all cases, actual costs, rather than estimated costs, were used. Accordingly, few assumptions were made. This is in contrast to many of the previous cost-effectiveness studies conducted of lifestyle interventions, which estimated costs retrospectively.[12,16]

## Implications

The Green Prescription appears to be cost-effective when compared with other physical activity interventions reported in the literature, such as Project Active in the United States.[12] Furthermore, the incremental cost of converting one additional person to an active state was NZ$1,756 using the Green Prescription. Using the United Kingdom the 'Prescription for Exercise' programme in primary care the incremental cost of converting one additional person to an active state was $NZ8,663 (UK£2,500).[7]

Although the costing structures and components may be quite different in these countries, the cost-effectiveness ratios of the Green Prescription appear favourable, as presented in Table 4. However, to allow comparisons with other types of interventions, a cost utility analysis is needed.

Ten percent more intervention patients than control patients went from 'sedentary' to 'active' and maintained this at 12 months. This has potential economic implications. For example, an estimated NZ$55 million could be saved in direct and indirect costs associated with ischaemic heart disease and hypertension if 10% of the population in New Zealand changed from 'sedentary' to 'active'.[17,5] The most recent New Zealand physical activity survey estimates that 878,000 adults over 18 years of age in New Zealand are not achieving 2½ hours of leisure-time activity per week.[18]

If all less-active adults were to receive a Green Prescription, the total programme cost (without offset costs), would be NZ$150 million to save at least NZ$55 million per year in costs associated with cardiovascular disease, alone. If changes detected after 1 year were permanent, then the programme may be cost-saving in approximately 5 years, assuming a 2-year delay[19] before cardiovascular benefits were evident.

The potential savings would be even greater if quality of life benefits (demonstrated in SF-36 score changes), and other potential health benefits associated with increased

physical activity, were considered. In addition, interventions become more cost-effective over time as the proportion of set-up costs declines.[12]

This study represents a cost-effectiveness analysis, using cost per physical activity unit gained as its primary outcome to allow comparison with previous community-based physical activity interventions. Modelling of the potential savings from health outcomes related to the increased proportion of active adults, and a cost-utility analysis are the next step and are underway.

The research will allow future comparison of cost-effectiveness of physical activity counselling in primary care with other lifestyle and pharmacological interventions.[20]

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# Appendices

## Appendix 1. Costs of set-up and coordination of the Green Prescription programme nationally

| Year | Cost# (NZ$) | Adjusted Cost* (NZ$) | Discounted Cost** (NZ$) |
|------|-------------|----------------------|-------------------------|
| 1996/97 | 400,000 | 430,800 | 549,822 |
| 1997/98 | 180,000 | 192,240 | 233,669 |
| 1998/99 | 390,000 | 414,960 | 480,368 |
| 1999/00 | 450,000 | 476,550 | 525,396 |
| 2000/01 | 490,000 | 498,820 | 523,761 |
| 2001/02 | 548,000 | 548,000 | 548,000 |
| **Total** | **$2,458,000** | | **$2,861,016** |

# Costs associated with intervention and resource development, grants to regional sports foundations, general practitioner training, coordination and resource production and distribution. Figures supplied by the Hillary Commission; * Adjusted for inflation, using the consumer price index (CPI) ratio (December 2001 CPI: annual December CPI ratio).[8]; ** Discounted at a 5% rate.[9]

## Appendix 2. Costs of delivery and follow-up of the Green Prescription programme by general practitioners and practice nurses

| Green Prescription Delivery | No. of patients | **Mean delivery time (mins) | Mean number of sessions | Hourly cost of personnel | Total cost | Cost per patient |
|-----------------------------|-----------------|------------------------------|-------------------------|---------------------------|------------|------------------|
| General Practitioner | 385 | 7 | 1 | $140.00 | $6,306.30 | $16.38 |
| Practice Nurse | 66 | 13 | 1 | $19.12 | $273.90 | $4.15 |
| Sub-total | 451 | | | | $6,580.20 | $14.59 |
| Practice Follow-up Advice | | | | | | |
| General Practitioner | 64 | 2.3 | 2.9 | $140.00 | $995.84 | $15.56 |
| Practice Nurse | 14 | 7.8 | 6.1 | $19.12 | $212.27 | $15.16 |
| Not Stated * | 9 | 5.8 | 4.8 | $140.00 | $584.64 | $64.96 |
| No Practice Follow-up | 302 | | | | | |
| Sub-total | 389# | | | | $1792.75 | $4.61 |
| **Overall Total** | | | | | **$8,372.95** | **$19.20** |

*Assumed to be a general practitioner (more conservative assumption than assuming the nurse saw them); ** As estimated by GP, nurse and patient participants. # Total cost/389 patients who had received intervention and had returned for follow-up. It was assumed that non-attendees had the same rate of follow-up advice from health professionals.

**Appendix 3. Costs of Sports Foundation attributable to the Green Prescription study participants (n=451) for exercise specialist follow-up over 12 months (2001/2002)**

| Cost Category | Details | Cost per item | Total Cost |
|---|---|---|---|
| Staff Wage Rates: | | | |
| Staff 1 | $21.27 per hr x 7.2 hrs/ week | $7,965 | |
| Staff 2 | $20.19 per hr x 7.2 hrs/ week | $7,560 | |
| Staff 3 | $14.00 per hr x 11.25 hrs/ week | $8,190 | |
| Staff training | | $900 | |
| Course fees | | $900 | |
| **Total staff costs** | | | **$25,515** |
| Office Space | Rental/ Cleaning | $1,512 | $1,512 |
| Admin. Support | Reception/Admin | $1,350 | $1,350 |
| Tolls | Staff 1 | $108 | $1,350 |
| | Staff 2 | $216 | |
| | Staff 3 | $1,026 | |
| Mail-outs | Newsletter | $200 | $1,305.65 |
| | Postage | $491.40 | |
| | Envelopes | $122.85 | |
| | Photocopying | $491.40 | |
| **Total overheads** | | | **$5,517.65** |
| **Total cost of intervention support** | | | **$31,032.65 ($68.81 per patient)** |

(Figures supplied by the regional Sports Foundation accounting department)

**Appendix 4. Change in primary healthcare use by study participants for the year-before and the year-after baseline**

| | | Intervention | | | Control | | | Incremental Change |
|---|---|---|---|---|---|---|---|---|
| Description | Unit cost to: patient (health funder) | Yr 1 (n*) [av. no. visits/ patient] | Yr 2 (n) [av. no. visits/ patient] | Yr2-Yr1 av. no. visits/ patient | Yr 1 (n) [av. no. visits/ patient] | Yr 2 (n) [av. no. visits/ patient] | Yr2-Yr1 av. no. visits/ patient | Average no. visits/ patient |
| 'A1'** non-accident visits to general practitioner | $20 ($15) | 1227 (n=145) [8.46] | 1139 (n=146) [7.80] | -0.66 | 1294 (n=157) [8.24] | 1296 (n=157) [8.25] | 0.01 | -0.67 |
| 'A3'** non-accident visits to general practitioner | $35 ($0) | 846 (n=170) [4.98] | 808 (n=171) [4.73] | -1.46 | 858 (n=172) [4.99] | 851 (n=170) [5.01] | 0.02 | -1.48 |
| 'AZ'** non-accident visits to general practitioner | $20 ($15) | 191 (n=15) [12.73] | 189 (n=15) [12.60] | -0.13 | 233 (n=15) [15.53] | 257 (n=15) [17.13] | 1.6 | -1.73 |
| Total non-accident visits to general practitioner | | 2264 (n=330) [6.86] | 2136 (n=332) [6.43] | -0.43 | 2385 (n=346) [6.93] | 2404 (n=344) [7.03] | 0.10 | -0.53 |
| Total accident-related visits to general practitioner | $10 ($26) | 286 (n=330) [0.87] | 299 (n=331) [0.90] | 0.03 | 341 (n=346) [0.99] | 360 (n=344) [1.05] | 0.06 | -0.03 |
| Physio, chiropractor or osteopath[#] visits | $10 ($19) | 518 (n=366) [1.42] | 602 (n=367) [1.64] | 0.22 | 521 (n=337) [1.55] | 577 (n=333) [1.73] | 0.18 | 0.04 |

* 'n' refers to the number of patients in each category. ** 'A1' refers to 'low income' patients and 'AZ" refers to 'high user' patients, both of whom receive government subsidies for visits to general practice, 'A3' refers to higher income patients who do not qualify for government subsidies for general practice visits. [#] Accident-related visits to a physiotherapist, chiropractor, or osteopath. NB: Data about number of general practice visits were collected on 677 study participants (74% intervention participants [n = 332] and 81% control participants [n = 346]). Eight practices (5 intervention and 3 control) were not able to, or chose not to provide the information on general practice visits. The number of physiotherapy, osteopathy and chiropractic accident-related patient visits for the year prior to baseline and the year following baseline, was available on 750 of the 878 participants because this data were collected at follow-up by self-report. Intention-to-treat analyses were conducted that assumed those for whom data were not available had no change in their rate of visits over the two years.

**Appendix 5. Change in costs of public hospital admissions and outpatient use for the year-before and the year-after baseline (costs adjusted for inflation)**

| | Intervention[#] (N = 451) | | | Control[#] (N = 427) | | | Incremental Change * (N=878) |
|---|---|---|---|---|---|---|---|
| **Cost description** | **Year 1** | **Year 2** | **Change: Yr2-Yr1** | **Year 1** | **Year 2** | **Change: Yr2-Yr1** | **Intervention-Control** |
| Total secondary care costs in $NZ (n**) | $370,189 (113) | $514,889 (117) | $144,700 (144) | $267,119 (107) | $478,497 (108) | $211,378 (138) | -$66,678 |
| Mean cost per patient who used secondary care (95%CI) | $3,276 ($2,276–$4,276) | $4,400 ($2,987–$5,813)) | $1,005 (-$219–$2,229) | $2,496 ($1,886–$3,106) | $4,431 ($3,009–$5,853) | $1,531 ($341–$2,721) | -$527 (-$2,221–$1,167) |
| Mean cost including all study patients (95%CI) | $828 ($546–$1110) | $1,142 ($738–$1,546) | $321 (-$69–$711) | $626 ($443–$809) | $1,121 ($721–$1,521) | $495 ($108–$882) | -$174 (-$723–$374) |

[#] Calculations are not adjusted for clustering. * Calculations adjusted for clustering using STATA 7.0; ** Number of patients admitted or attended outpatients during each time period and in each group.

**Note:** Year 1 and Year 2 refer to the year before and the year after baseline (costs adjusted for inflation). Approximately 34% (299/878) of study patients reported being admitted to a public hospital or attending a hospital outpatients clinic in the year prior to baseline or the year between baseline and follow-up. Public hospital costing-data were obtained for 282/299 (94%) of these patients (144 intervention and 138 control patients). These costs were not available on 17 participants (10 intervention patients and 7 control patients). In addition, 37 patients said they had attended a private hospital in the year between enrolment and 12-month follow-up plus four had attended both public and private hospitals (Total: 21 intervention and 20 control patients). Actual costs for private hospital admission could not be obtained. However, number of days of admission had been collected by self-report. When the average daily cost (with associated outpatient costs) from the public hospital figures ($1,605) was applied to private hospital admissions, the total year-2 cost of private admissions in the control group was $97,102 (average cost/patient $4,855, SD$5,464). In comparison, the total year-2 cost of private admissions in the intervention group was $72,225 (average cost/patient $3,439, SD $3,704). Although these calculations assume similar daily costs in public and private, which is unlikely to be the case, they do suggest that the incremental savings in hospital savings within the intervention compared with the control group are likely to be greater than presented in the analysis of public hospital costs only.

**Appendix 6. Change in the number of days off work due to sickness or accident during the year-before compared with the year-after study enrolment for intervention and control study participants**

| Variable | | Intervention (n = 204) | | | Control (n = 178) | | | Incremental Change [95%CI] |
|---|---|---|---|---|---|---|---|---|
| | | Yr1 | Yr 2 | Yr2-Yr1 | Yr1 | Yr2 | Yr2-Yr1 | Intervention-Control |
| Unit cost per day | NZ$ | 121.8 | 128.2 | | 121.8 | 128.2 | – | – |
| Illness-related days leave | Total | 842.5 | 887.5 | | 721.5 | 749.5 | – | – |
| | Mean (SD) | 4.15* (20.5) | 4.25 (16.6) | 0.17* (26.2) | 4.05 (14.9) | 4.16 (15.8) | 0.08 (9.36) | 0.09 [-3.98–4.16] |
| Accident-related days leave | Total | 69.5 | 153 | | 187 | 267 | | – |
| | Mean (SD) | 0.34 (1.94) | 0.74 (4.39) | 0.41 (4.83) | 1.05 (7.26) | 1.50 (10.7) | 0.45 (11.4) | -0.04 [-1.76–1.68] |

**Note:** Of the 393 (52%) participants in paid employment 382 (97%) gave data on the loss of productivity; * Data were missing on 2/204 participants for illness-related days off in year 1. Therefore, change in illness-related days was calculated from 202 in the intervention group.

**Appendix 7. Patient costs associated with exercising for the year between baseline and follow-up**

| Description | Intervention* (n=389) Total Cost | Average Cost (SD) | Control* (n = 361) Total Cost | Average Cost (SD) | Incremental Difference** Average cost/patient [95%CI] | |
|---|---|---|---|---|---|---|
| Exercise/ sports shoes | $16,823 | $43.25 ($65.08) | $12,380 | $34.29 ($70.69) | $9.80 | [-1.50, 21.11] |
| Exercise group, sports club or gym membership | $23,004 | $59.14 ($166.00) | $17,986 | $49.82 ($142.22) | $9.31 | [-12.89, 31.52] |
| Exercise or physical activity equipment | $15,871 | $40.80 ($221.86) | $9,673 | $26.80 ($182.38) | $14.00 | [-15.19, 43.20] |
| Other costs associated with exercise | $3,991 | $10.26 ($70.64) | $3,688 | $10.22 ($63.10) | $0.11 | [-9.83, 10.05] |
| Travel cost # | $32,228 | $82.85 ($336.33) | $31,843 | $88.21 (402.89) | -$5.36 | [-58.34, 47.62] |
| **Total** | **$91,917** | **$236.30 ($515.14)** | **$75,570** | **$209.34 ($659.41)** | **$26.96** | **[-45.08, 98.98]** |

*Calculations not adjusted for clustering; **Calculations adjusted for clustering by practice; # Travel cost equals total km per week x 16.6 cents/km x 52 week=cost per year.

# Diabetes in children and young adults in Waikato Province, New Zealand: outcomes of care

Adrian Scott, Susie Whitcombe, David Bouchier, Peter Dunn

## Abstract

**Background** Diabetes is an important cause of morbidity and mortality among young people. Despite improvements in technology, maintenance of good glycaemic control is hard to achieve particularly during the teenage years.

**Aims** To assess the outcomes of care in young people aged under age 26 years with diabetes living in New Zealand's Waikato District Health Board (DHB) area.

**Methods** Audit of health records

**Results** Two hundred and fifty-one patients who had attended an outpatient consultation at least once in the last 3 years were identified. The mean HbA1c was 9.2% (CI 8.8–9.8). There were no gender differences. The prevalence of retinopathy was 13%, and nephropathy up to 19%. Eleven patients were taking ACE inhibitors and one person had end-stage renal failure treated with continuous ambulatory peritoneal dialysis (CAPD). Forty percent of those patients over 10 years of age had a low density lipoprotein (LDL) cholesterol level of >2.6mmol/L. All of those patients with type 2 diabetes mellitus (T2DM) were overweight, compared to 28% of those patients with type 1 diabetes mellitus (T1DM).

**Conclusions** These results confirm the difficulty of achieving good glycaemic control in children and young adults. Microvascular complications are common, particularly in those of long duration. Risk factors for macrovascular disease are present from an early age, especially in those with T2DM. It is likely that these outcomes of care are typical for children and young adults with diabetes in New Zealand.

Children diagnosed with type 1 diabetes (T1DM) have a poor prognosis compared to their non-diabetic counterparts.[1–4] It is 10 years since the Diabetes Control and Complications Trial (DCCT) reported the beneficial effects of tight glycaemic control in adults and adolescents.[5]

Unfortunately, the care of young people with diabetes is challenging and for many centres a mean HbA1c over 9% for this group is quite common (despite intensive efforts) leading some clinicians to believe that poor control is inevitable. Indeed, recent studies from Europe and Japan have illustrated the difficulties of achieving and maintaining good glycaemic control; the high prevalence of complications and the wide range of glycaemic control between centres being unrelated to patient selection or choice of insulin regimen.[6–11]

Nevertheless, some centres consistently have mean HbA1cs as good if not better than the Intensive arm of DCCT, without the increased risk of hypoglycaemia.[12–14] Type 2 diabetes was once unknown in children but is increasingly recognised with the rise of obesity. We have reviewed our own experience of managing these young people.

## Setting

Waikato District Health Board (DHB) serves approximately 9% (312,918 in the 1996 Census) of the total New Zealand population, in a largely rural setting in the North Island. Approximately 20% of the Waikato population identify themselves as Maori. An estimated 10,000 (New Zealand Ministry of Health data) of these 312,918 people have diabetes (predominantly type 2 diabetes mellitus [T2DM]), which is more common among Maori and Pacific Islanders.

Children and young people with diabetes attend the Diabetes Unit or Paediatric service in Hamilton City, or one of the outlying clinics, and are looked after by an adult endocrinologist (PD) or paediatrician (DB) and nurse educator (SW). Dietetic input is more limited and most advice comes from the nurse educator.

Children up to age 16 were always admitted at diagnosis; above that age, some patients living near the hospital may be treated as an outpatient provided they were not in ketoacidosis. Since Waikato Hospital serves a wide geographical area, most young people were admitted for at least 48 hours for initiation of treatment and initial education.

Twice daily regimens were common but now most patients aged over 7 years move swiftly to a basal bolus regimen with twice-daily Isophane and mealtime short-acting insulin, predominantly Lispro or Aspart, although some patients remain on soluble insulin. Insulin glargine was not available in New Zealand at the time of the audit.

Under that age (7 years), twice daily regimens are more common because of the difficulty of giving insulin during school hours. For about 4 years we have taught carbohydrate-counting (to facilitate insulin adjustment) and daily blood glucose testing before meals, and bedtime is encouraged. Patients going on to pumps attend a 1-week education course with a specially trained nurse specialist and dietitian.

Retinal screening is undertaken by retinal photography every 2 years from the age of 16 years. Photographs are taken with pupils dilated and graded by a consultant ophthalmologist who undertakes slit lamp examination if significant retinopathy is identified.

Foot examination is performed using either a biothesiometer or a 10 gram monofilament. Data on severe hypoglycaemia is not consistently recorded so has not been included. This audit of the outcomes of care of children and young adults with diabetes was carried out in July 2003

## Methods

A Microsoft Access database holds data on all patients who are current attenders or have attended the diabetes unit in Hamilton, Waikato. Records on 315 people with diabetes born after 1st January 1978 were analysed. Type 1 diabetes was diagnosed in the presence of diagnostic blood sugars in combination with ketonuria or ketoacidosis and/or positive anti IA2 and anti-GAD antibodies. Type 2 diabetes was diagnosed in those individuals who had negative antibodies or had no history of ketoacidosis. One patient had ketonaemia at diagnosis but was not acidotic. When subsequently found to have negative antibodies his insulin was successfully withdrawn and he was treated with Metformin for over 1 year with HbA1c of <7%. 251 (237 with T1DM, 13 with Type 2, 1 with secondary diabetes) had attended at least once in the last 3 years. The remainder had either moved away or been lost to follow-up and were excluded.

Where data was missing, the paper records were examined (n=105). For missing blood and urine values, the local laboratories (Waikato Hospital, Medlab, Pathlab) were contacted.

Admission and outpatient attendance data was obtained by a query to the HOSPRO information system.

Where appropriate, data is expressed as mean (95% Confidence Intervals).

Microalbuminuria defined as an Albumin / Creatinine Ratio (ACR) >2.5 in males; >3.5 in females, on more than one occasion. Two or more abnormal results are required to confirm persistent microalbuminuria. Proteinuria was defined as ACR >30. Body mass index (BMI) was calculated from weight (kg) / [height (m)]2

For patients under 20 years of age, BMI-for-age percentiles were calculated using the National Center for Chronic Disease Prevention Growth Charts.

For this audit, we used the American Diabetes Association recommendations[15] for treatment of hyperlipidaemia over the age of 10, if the LDL cholesterol is ≥4.1 mmol/L and to consider therapy if LDL is 3.4-4 mmol/L if there are other cardiovascular risk factors present. Target LDL is < 2.6 mmol/L.

## Results

### All patients

There were 251 young people with diabetes (237 Type 1; 13 Type 2; 1 chronic pancreatitis); 135 were female; and 212 described themselves as of European origin, 27 as Maori, 5 as Pacific Islander, 4 as Asian (Chinese), and 3 as Indian.

Four patients (1.6%) were hypothyroid and 1 (0.4%) had previously been thyrotoxic and was now euthyroid; 6 (2.4%) had coeliac disease. Three patients had symptomatic peripheral neuropathy (1%) and 6 (2.4%) were recorded as having depression.

Over 5 years there have been 504 in-patient episodes (2 per patient) amounting to 1994 days in hospital (7.9 days per patient). There were a total of 7063 outpatient attendances (5.6 per patient per year).

### Type 1 diabetes

Demographic details are indicated in Table 1. Glycaemic control was generally poor but worst in the 15-19 year age group (Table 2). There was no relationship between insulin regimen and glycaemic control.

In the patients who were 15 years and younger with T1DM, urine had been measured for protein in 48 of 99 (48%). In those patients aged 16 year and over, urine was measured for protein in 119 of 138 (86.2%). Proteinuria was present in 4 patients, microalbuminuria in 25 patients (7 with intermittent microalbuminuria but in 4 patients, only 1 specimen had been tested) giving a prevalence of nephropathy of 18-29/119 (15-22%).

Of 220 children aged over 10 years, 166 (75%) had lipids measured. One patient was taking a statin. There were 89 (54%) with an LDL >2.6mmol/L, 14 (8.4%) with LDL≥4.1mmol/L, and 3 (1.8%) with LDL 3.4–4.0 mmol/L and one other risk factor (smoking, hypertension, or microalbuminuria). All patients had smoking status recorded; 18 are current smokers , 5 are ex-smokers.

**Table 1. Type 1 diabetes by age—glycaemic control and insulin regimens**

| Variable | All ages | 16–25 years | Under 16 years |
|---|---|---|---|
| N | 237 | 138 | 99 |
| Male (%) | 109 (46) | 63 (45.6) | 46 (46) |
| Age (CI) | 16.7 (16–17.4) | 20.9 (20.4–21.3) | 10.9 (7.3–14.5) |
| Duration (yr) | 7.22 (6.5–7.93) | 9.5 (8.6–10.5) | 4.0 (0.7–7.3) |
| Latest HbA1c | 9.3 (9.0–9.7) | 9.3 (8.6–9.9) | 9.4 (8.0–10.8 |
| Mean HbA1c (last 3) | 9.17 (9.0–9.4) | 9.4 (9.1–9.7) | 8.9 (7.8–10.0) |
| A1c measured in 1st year after diagnosis (%) | 66 (28) | 31 (22.5) | 35 (35) |
| % achieving A1c < 6.2% during first year | 8 (12) | 3 (10) | 5 (14) |
| Insulin injections: | | | |
| 5 or more | 114 | 67 | 47 |
| 4 | 41 | 35 | 6 |
| 3 or less | 53 | 12 | 41 |
| Pumps | 22 | 15 | 7 |
| Insulin dose (u/kg) | 0.96 | 0.87 | 1.1 |

In the patients aged 16 and over, blood pressure (BP) was measured in 135 (97.8%) of them; 11 (8.1%) patients had a systolic BP ≥130 mmHg, and 10 (7.4%) patients had a diastolic BP ≥80 mmHg.

BP was measured in 89% of the patients aged 15 years and younger; 2 (2.2%) patients had systolic BP >120 mmHg (1 with coarctation), and 3 (3.4%) patients had a diastolic BP >70 mmHg.

Of those patients with T1DM, 26.9% of males and 29% of females were overweight or obese. Of those patients aged under 20 years, 16 females and 9 males had a BMI-for-age between the 85-95th percentile; 5 females and 3 males under 20 year had a BMI-for-age ≥95th centile. In the over 20 years age group, 16 males and 4 females were overweight, 4 males and 12 females were obese.

Of those patients with T1DM, 67 had a duration of diabetes over 10 years. Of these, 25% had evidence of nephropathy (12 had microalbuminuria and 5 proteinuria), one of whom is on CAPD. Six (9%) patients had no documented eye screening—but of the remainder, 25% had retinopathy (10 background, 5 sight threatening); 41 (61%) had LDL cholesterol >2.6 mmol/L, and 9 (13.4%) would qualify for statins using the ADA criteria. Nine (13.4%) patients are current smokers, 2 (3%) peripheral neuropathy, and 3 (4.5%) are listed as suffering from depression.

## Type 2 diabetes:

There were 13 patients (including 7 males) with T2DM. The 13 patients included 7 Maori, 1 Pacific Islander, 4 European, and 1 Asian Indian. One teenager had Prader Willi syndrome. The mean age was 19.6 years (range 14–23 years) and duration of diabetes was 1.7 years.

All patients were obese with a BMI 39 (males 37.3; females 40.3). Mean HbA1c 8.8%. Six were treated with diet alone, 4 with Metformin alone, 1 with insulin only, 2 with insulin plus Metformin, and 1 with insulin plus Acarbose. Five (38%) patients had a systolic BP >130 mmHg or diastolic BP >80 mmHg. Two (15%) had microalbuminuria, one of whom was on an ACE inhibitor. Six (46%) patients had attended retinal screening—none had retinopathy.

Nine patients had their fasting lipids measured; their mean total cholesterol level was 5.6 mmol/L, HDL 1.1 mmol/L, triglycerides 6.4 mmol/L, and mean LDL 3.1 mmol/L. Three (33%) patients had LDL-C >3.4 mmol/L and 1 (11%) patient had severe hypertriglyceridaemia (28 mmol/L).

**Table 2. Glycaemic control by age in 251 children and young adults with diabetes**

| Age (years) | Numbers | Mean HbA1c (CI) |
|---|---|---|
| 20–25 | 94 | 9.2 (CI 8.8–9.6) |
| 15–19 | 67 | 9.9 (CI 9.5–10.3) |
| 10–14 | 61 | 8.7 (CI 8.4–9.0) |
| <10 | 29 | 9.4 (CI 8.9–9.9 ) |

## Discussion

This audit of the process and outcomes of care of children and young adults with diabetes, in a semi-rural district in North Island New Zealand, has demonstrated a disappointing picture of poor glycaemic control and moderately high rates of microvascular complications, as seen in other studies. Despite the use of multiple injection therapy and carbohydrate-counting, few people achieved satisfactory control (only 25% had a recent HbA1c ≤8%).

A limitation of our study is that the exact number of people aged under 26 years with diabetes in Waikato is unknown. The published literature on defaulters from follow-up, however, suggests they have worse control and greater risk of complications.[16]

Although glycaemic control is poor, it is similar to other published studies in Europe of unselected young people with diabetes. Few data on young people with diabetes in New Zealand are available. In Christchurch, the mean HbA1c for people aged between 13 and 20 years was 10.2% and 9.5%, respectively, for females and males.[17]

The type of insulin regimen (including use of pumps) does not appear to have much impact on glycaemic control—although of 23 patients with a duration of diabetes >2 years and an HbA1c ≤7.5%, 6 were on 2 injections, 1 on 3 injections, 1 on 4 injections, and 13 on 5 injections (per day).

Twenty-two patients were using pumps. The pump data may be misleading, however, as we have chosen to offer pump therapy to young people having difficulty achieving satisfactory control with multiple injections, and in this group there has been an overall reduction in HbA1c and admissions with DKA (data not shown).

Age, sex, insulin regimen, BMI, season, social circumstances, and family history were all associated with glycaemic control in the Scottish study but not with deprivation score based on post code. Neither the Hvidore nor the Scottish study[6,10] were able to identify definitive reasons for centre differences in mean HbA1c.

Dabaghdao[18] suggested that poor control in childhood led to poor control in adolescence and beyond. Other studies have suggested that poor early control is associated with a four-fold increase in the subsequent prevalence of nephropathy[19]. An intriguing observation of the DCCT collaborators was that the tight control

(initiated 1 year after diagnosis) was associated with preservation of islet cell function for a greater period than the group randomised to conventional (poor) control.[20]

There have been a few small studies, looking at the impact on beta cell function and intensive normalisation of glycaemic control from diagnosis with conflicting results, but no long-term randomised studies.[21,22]

Interestingly, there are huge differences in the number of children with a normal HbA1c during the first year after diagnosis. Some centres achieve this result in as many as 74% of their patients.[23] Irrespective of the long-term benefits, this implies significant differences in both expectation and training of the person with diabetes.

Even when centres are benchmarked against others, it is difficult to achieve a change in the overall mean HbA1c.[24] Nevertheless, Berger and colleagues demonstrated that (in adults) a 5-day training course resulted in prolonged overall improvements in HbA1c without a corresponding increase in severe hypoglycaemia.[25,26] Similar improvements were seen in the DAFNE study in the UK, although adolescents were not included.[27]

The prevalence of retinopathy among screened patients is similar to published series,[7,8,28,29] but there were a worrying number of patients who appeared to have avoided retinal screening for prolonged periods of time. Similarly, screening for microalbuminuria appears to have been done in the majority of young people but positive results were not always followed up.

Use of ACE inhibitors is reasonable in those with confirmed nephropathy, although this partly depends on the criteria for diagnosis (of 13 young people with 3 or more abnormal results, 11 were on ACE inhibitors. None of those patients with just 2 abnormal results were treated with ACE-inhibitors). A significant proportion appear to have intermittent proteinuria and a recent publication found that up to 60% of people with T1DM have spontaneous resolution unrelated to ACE inhibitor use.[30]

Suboptimal lipid profiles were very common, and only one patient was receiving any treatment. Cardiovascular risk charts will underestimate risk and are inappropriate for this age group.[31] Only the American Diabetes Association has published specific guidelines for young people with T1DM, and with the knowledge that most will die prematurely from a vascular accident, earlier use of statins may be appropriate. As with use of ACE inhibitors, however, consideration has to be given to the risk to the developing foetus in the event of conception occurring whilst taking them.

Nearly 30% of those with T1DM, and 100% with T2DM, are overweight. This may reflect the rising incidence of obesity in children and adolescents. In New Zealand, in 1997, approximately 25% of 15 to 18 year olds and one in three 19 to 24 year olds were found to be overweight or obese.[32] Weight gain is common during adolescence (especially in girls); and with intensive insulin therapy, the weight gain can sometimes be spectacular[33] and likely to be a disincentive to better glycaemic control.

Perhaps the most worrying finding in our study was that of those patients with T2DM. All 13 were diagnosed in the last 4 years (mean duration 1.7 years) at an average age of 19.6 years. This rising incidence of T2DM is predictable and parallels the increasing prevalence of obesity in childhood.

Lastly, as we do not know how representative these data are of New Zealand in general, we are undertaking a similar national audit in this age group.

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# Under-reporting of diabetes on death certificates among a population with diabetes in Otago Province, New Zealand

Kirsten Coppell, Karen McBride, Sheila Williams

## Abstract

**Aims** To estimate the degree of under-reporting of diabetes on death certificates, and to describe the population of patients enrolled on the Otago Diabetes Register known to have died during the 6-year period to 31 December 2003.

**Methods** The Otago Diabetes Register was established in 1998, as part of the Otago Diabetes Project, to monitor and evaluate diabetes care in the Otago region, New Zealand. Demographic and clinical data, including vital status, type of diabetes and year of diagnosis, diabetes complications, diabetes medication and clinical examination, and biochemistry test results were collected annually from general practice medical records. Copies of death certificate information were obtained from the national Births, Deaths and Marriages office, Department of Internal Affairs for 508 enrolled diabetic patients known to have died before 31 December 2003. Causes of death were coded using ICD-10. Date, place, and causes of death were added to the Otago Diabetes Register.

**Results** The mean age at death was 78 years (SD=9.7) and the average duration of diagnosed diabetes was 12.1 (SD=8.6) years. Diabetes was mentioned on the death certificates of just over half (55.1%) of the 508 diabetic patients. More of those using insulin only, or oral hypoglycaemic and insulin therapies combined, before death had diabetes mentioned on their death certificate, 67.8% and 81.0%, respectively, compared with those taking oral hypoglycaemics only (55.7%) or diet only treatment (38.0%). Almost 50% of deaths were due to a circulatory system disorder, either cardiovascular or cerebrovascular disease. Five men with type 1 diabetes, all aged less than 50 years, died from diabetic ketoacidosis or hypoglycaemia.

**Conclusion** Diabetes is under-reported on death certificates in New Zealand. Improvements in the completion of death certificates are necessary, if the impact of the diabetes epidemic on mortality is to be monitored appropriately.

Diabetes prevalence is increasing worldwide, and studies have consistently shown excess death rates amongst diabetic populations compared with the general population.[1,2,3] However, the impact of increasing diabetes prevalence on mortality rates is difficult to determine. Death certificates are the only routine source of mortality information with which to monitor the national burden of diabetes-related deaths, but it has been repeatedly shown that diabetes is under-reported on death certificates worldwide.[1,4,5] Thus, the contribution of diabetes to cause of death is usually underestimated.[6]

The degree of under-reporting of diabetes on death certificates varies between countries,[1,5,7] as does death certificate coding practices, although the same World Health Organization (WHO) rules and guidelines are usually used.[8,9] This partly stems from differences in opinion about the causal role of diabetes when it is associated with

other conditions such as cardiovascular disease.[8,10] Also, coding practices have changed over time resulting in inconsistent time trends. For example, in Australia, higher death rates attributable to diabetes were reported in the 1940s compared with the 1950s and 1980s[11]—and in the Oxford region, England, diabetes mortality rates based on underlying cause decreased stepwise between the 1984-92 and 1993-99 study time periods.[12] Thus, the validity of comparisons of diabetes mortality rates between countries and over time is questionable.

In New Zealand, there is only one published report estimating the degree of under-reporting of diabetes on death certificates.[5] For this recent Christchurch-based study, it was necessary to link several health information sources to identify deceased diabetic patients. The Otago Diabetes Register, established in 1998, enables diabetes-related data for enrolled individuals to be directly linked with death certification information.

The purpose of this study was to use data from the Otago Diabetes Register to estimate the degree of under-reporting of diabetes on death certificates, and to describe the population of enrolled patients known to have died during the 6-year period to 31 December 2003.

## Methods

The Otago Diabetes Register was established as part of the Otago Diabetes Project to monitor and evaluate diabetes care in the Otago region of New Zealand. The project also involved establishing or updating general practice diabetes registers, organising education sessions for general practitioners (GPs) and practice nurses and developing and implementing guidelines for the management of core aspects of diabetes care. Details of how general practice registers and the regional diabetes register were established have been previously described.[13]

Briefly, a project nurse established or updated general practice diabetes registers for participating GPs (about 95% of all GPs in the region). Identified diabetic patients were sent an invitation from their GP to participate in the project, along with an explanatory pamphlet, a consent form and a stamped addressed envelope for return of the form. Consent was also obtained opportunistically, when patients attended their general practice or the local retinal screening programme.

Data (including demographic details, type of diabetes and year of diagnosis, dates and results of retinal and foot examinations, diabetes complications, diabetes medication, and dates and results of biochemistry tests) were collected annually from general practice medical records. For patients who had attended Dunedin Hospital's Outpatient Diabetes Clinic or Eye Department, checks were made for missing data. If patients' diabetes type was uncertain, this was checked with the local specialist diabetes clinic.

Those patients who had died during the previous 12 months were noted as such on the regional register. The vital status of the 279 patients who had moved from the Otago region or had changed to a non-participating GP during the 6-year study period was not known. Between 1998 and 2003, the annual number of enrolled alive diabetic patients living in the Otago region increased from 1693 to 3387, which was about 71% of the estimated 4800 people with diagnosed diabetes in the province.

Copies of death certificate information were obtained from the national Births, Deaths and Marriages office, Department of Internal Affairs for patients known to have died. ICD-10 was used to code all diseases recorded on the death certificate.[14] The date of death, place of death, and causes of death were added to the Otago Diabetes Register—a Microsoft Access-based program developed by the project.

Means and standard deviations, or frequencies and percentages based on the patients' last review, were calculated for variables of interest. Student's t -tests, Chi-squared tests, or Fisher's exact test (as appropriate) were used to examine differences between men and women.

Ethical approval was obtained from the Southern Regional Health Authority Ethics Committee.

## Results

At 31 December 2003, 4320 diabetic patients had ever been enrolled on the Otago Diabetes Register, of whom 509 (11.8%) were known to have died. Death certificate information was matched for 508 enrolees who had died, of whom 253 were females and 255 were males. Most people (96.1%) were of European descent, with 3.0% self-identifying as Maori. Of the 508 with matched death certificate information, 17 had type 1 diabetes, 482 had type 2 diabetes, and 9 had diabetes secondary to another condition or steroid medication. More than one-half (57.1%) of the patients died in a hospital. The proportion of males who died at their own home (22.4%) was higher than for women (13.0%). Other places of death were rest homes (17.9%), hospices (6.3%), and the community (1.0%).

For those with type 1 diabetes, 4 were females and 13 were males. Five people, all males, were aged less than 50 years at death and died in their own home. The immediate cause of death for three of these five males was diabetic ketoacidosis and the other two died from a hypoglycaemic event.

**Table 1. Characteristics of patients with type 2 diabetes or diabetes secondary to another condition or steroid medication**

| Variable | Females (n=249) | Males (n=242) | Total (n=491) | Difference between males and females |
|---|---|---|---|---|
| | Mean (SD) or % | Mean (SD) or % | Mean (SD) or % | P value |
| Age at death (yrs) | 78.6 (9.6) | 77.5 (9.8) | 78.1 (9.7) | 0.21 |
| Age at diagnosis (yrs)* | 66.2 (12.4) | 65.3 (11.6) | 65.8 (12.0) | 0.41 |
| Duration diagnosed diabetes (yrs)* | 12.4 (9.0) | 11.8 (8.1) | 12.1 (8.6) | 0.44 |
| Current smoker (%) | 9.2 | 9.5 | 9.4 | 0.92 |
| Ex-smoker (%) | 36.9 | 71.5 | 54.0 | 0.00 |
| *Treatments* | | | | |
| Diet only (%) | 25.7 | 26.9 | 26.3 | 0.77 |
| OHs only (%) | 50.2 | 46.3 | 48.3 | 0.39 |
| Insulin only (%) | 19.3 | 23.1 | 21.2 | 0.30 |
| OHs and insulin (%) | 4.8 | 3.7 | 4.3 | 0.55 |
| ACE inhibitor (%) | 55.0 | 52.9 | 54.0 | 0.64 |
| Lipid lowering (%) | 14.5 | 17.4 | 15.9 | 0.38 |
| Other antihypertensives (%) | 27.7 | 16.1 | 22.0 | 0.002 |

*Year of diagnosis values were missing for 16 females and 22 males; SD=standard deviation.

The characteristics of the 491 patients with type 2- or secondary-diabetes are shown in Table 1. Overall, the mean duration of diagnosed diabetes was about 12 years. Females were slightly older at both the time of diagnosis of diabetes and time of death compared with males. About one-quarter used insulin treatment prior to death, and a further one-quarter were being treated with diet only. Over one-half were prescribed an ACE inhibitor during the review year prior to death. A further 48 people (9.8%) had previously been prescribed an ACE inhibitor, but were no longer taking it. (Reasons for stopping their ACE inhibitor therapy were not recorded on the Otago Diabetes Register.) A statistically significant higher proportion of females were

prescribed antihypertensive treatments (excluding ACE inhibitors) compared with males (p=0.0002).

Diabetes was mentioned on the death certificates of 55.1% of all 508 diabetic patients. For seven people, diabetes was listed as the immediate cause of death. Diabetes was not recorded anywhere on the death certificate of one person with type 1 diabetes. People who died in hospital or their own home were more likely to have diabetes mentioned on their death certificate, 59% and 62% respectively, compared with those who died at other places: rest home (50%), hospice (31%), and community (40%).

Table 2 shows the clinical characteristics of those who had diabetes mentioned on their death certificate compared with those who did not have diabetes mentioned on their death certificate. Overall, the group with diabetes mentioned on their death certificate were younger at the time of diagnosis and duration of diabetes was about 5 years longer than those where diabetes was not mentioned. A significantly higher proportion of the group who did not have diabetes mentioned on their death certificate were using diet only treatment for glycaemic control compared with the group who had diabetes mentioned (p=0.0). The reverse was observed for insulin treatment (p=0.001). Similar proportions of each group used oral hypoglycaemic treatments.

Table 3 shows the immediate causes of death by major disease category as recorded on the death certificate. Almost 50% of deaths were due to a circulatory system disorder, either cardiovascular or cerebrovascular disease. Respiratory diseases and neoplasms were also frequent causes of death. A neoplasm was the cause of death for a significantly higher proportion of the group with no diabetes mentioned on the death certificate compared with the group with diabetes on the death certificate.

**Table 2. Clinical characteristics of the group with diabetes mentioned on their death certificate and the group without diabetes mentioned on their death certificate**

| Variable | Diabetes on death certificate (n=280) | | Diabetes *not* on death certificate (n=228) | | Difference |
|---|---|---|---|---|---|
| | **Number** | **Mean (SD) or number (%)** | **Number** | **Mean (SD) or number (%)** | **P value** |
| Age at death (yrs) | 280 | 77.6 (9.7) | 228 | 77.4 (11.2) | 0.82 |
| Age at diagnosis (yrs)* | 263 | 62.3 (14.8) | 207 | 66.8 (12.8) | 0.001 |
| Diabetes duration (yrs)* | 263 | 15.0 (11.5) | 207 | 10.3 (7.5) | 0.00 |
| Diet only (%) | | 49 (17.5) | | 80 (35.1) | 0.00 |
| OH only (%) | | 132 (47.1) | | 105 (46.1) | 0.93 |
| Insulin only (%) | | 82 (29.3) | | 39 (17.1) | 0.001 |
| OH and insulin (%) | | 17 (6.1) | | 4 (1.8) | 0.015 |
| Diastolic BP (mmHg)† | 274 | 74.3 (11.3) | 222 | 75.5 (10.8) | 0.23 |
| Systolic BP (mmHg)† | 274 | 134.5 (21.2) | 222 | 137.3 (21.2) | 0.15 |
| HbA1c (%)† | 264 | 7.4 (1.5) | 216 | 7.0 (1.5) | 0.004 |
| Total cholesterol (mmol/L)† | 160 | 5.3 (1.2) | 136 | 5.5 (1.3) | 0.20 |
| HDL-cholesterol (mmol/L)† | 158 | 1.06 (0.31) | 134 | 1.2 (0.52) | 0.002 |
| Triglycerides (mmol/L)† | 159 | 2.3 (1.6) | 134 | 2.0 (1.2) | 0.03 |

*Age at diagnosis was not recorded for 38 patients; †For some patients clinical measures or blood tests were not completed during the 12 months prior to death. OH=oral hypoglycaemics; BP=blood pressure; HDL=high-density lipoprotein; SD=standard deviation.

**Table 3. Causes of death by major category for those with and without diabetes mentioned on the death certificate**

| Cause of death | Diabetes on death certificate (n=280) | | Diabetes *not* on death certificate (n=228) | | Total (n=508) | | Difference |
|---|---|---|---|---|---|---|---|
| | N | (%) | N | (%) | N | (%) | P value |
| Infectious and parasitic | 14 | (5.0) | 9 | (3.9) | 23 | (4.5) | 0.57 |
| Neoplasms | 22 | (7.9) | 45 | (19.7) | 67 | (13.2) | 0.00 |
| Endocrine | 7 | (2.5) | 0 | (0.0) | 7 | (1.4) | 0.02 |
| Cardiovascular disease | 111 | (39.6) | 90 | (39.5) | 201 | (39.6) | 0.97 |
| Cerebrovascular | 26 | (9.3) | 16 | (7.0) | 42 | (8.3) | 0.36 |
| Respiratory | 46 | (16.4) | 25 | (11.0) | 71 | (14.0) | 0.08 |
| Gastrointestinal | 10 | (3.6) | 13 | (5.7) | 23 | (4.5) | 0.25 |
| Genitourinary | 13 | (4.6) | 3 | (1.3) | 16 | (3.1) | 0.03 |
| Other | 35 | (12.5) | 23 | (10.1) | 58 | (11.4) | 0.40 |

## Discussion

Under-reporting of diabetes on death certificates was recognised worldwide some time ago,[4] but this practice continues.[7] This study found that 45% of people with documented diabetes had no mention of diabetes on their death certificate. While this is consistent with a recent Christchurch study,[5] some studies have found that as many as 73% of diabetic patients have diabetes mentioned at any level on the death certificate, but this was amongst an insulin treated diabetic population,[1] whereas other studies have found as few as 36% of death certificates amongst a diabetic population mention diabetes.[7]

The proper completion and accuracy of death certificates has been questioned in several countries, including New Zealand.[15,16,17] Clinical diagnoses may be erroneous, but often little can be done about incorrect or unknown diagnoses. For known diseases, the sequence of events leading to death may be entered incorrectly on the certificate or important events omitted.[9] Guidelines and calls to ensure that doctors complete certificates correctly have been made in many countries.[16,17] In New Zealand, directions for completing death certificates are included on the front cover of each book of certificates, and detailed in a booklet published by NZHIS.[18] As diabetes is not always the immediate, underlying, or contributing cause of death it will correctly not be recorded on the death certificate, but this is unlikely to be the explanation for the absence of diabetes on all 45% of the 508 death certificates that we examined.

The range of diseases known or thought to be associated with diabetes, including some cancers such as pancreatic, liver and more recently bowel cancer,[19,20] may not always be recognised, and this may contribute to the under reporting of diabetes on death certificates. There may be other explanations. Nevertheless, because of the complex nature of diabetes, it has been suggested that diabetes should always be recorded on the death certificate of all those with this condition, regardless of whether it is considered to be the underlying or contributing cause of death.[4]

The most recent published mortality data for New Zealand shows that for the year 2000, 1455 people died in the Otago DHB region.[21] From this study, it is known that

in 2000, at least 115 people with diabetes died (8% of the total number of deaths in the Otago region). This proportion could be an underestimate, as not all people with diabetes are enrolled on the Otago Diabetes Register and hence were not included in this study, and generally up to 50% of people with diabetes have not had the condition diagnosed.

The median age at death for this diabetic study population (79.6 years for females and 78.2 years for males) compares favourably with the median age at death for the general New Zealand population. For the 2000-2002 period, half of female deaths occurred at ages 81 years and over, and half of male deaths occurred at ages 75 years and over.[22] A similar observation was noted using data from the Skaraborg Diabetes Registry, Sweden, where diabetic patients aged over 80 years had a survival similar to that of the background population,[23] as did a group of Scottish men who were aged over 65 years at the time type 2 diabetes was diagnosed.[24] This supports the suggestion that the onset of diabetes at an older age may not decrease life expectancy.[25] However, quality of life may be reduced, particularly if diabetes related complications are present. At the other end of the age spectrum, the deaths of 5 males with type 1 diabetes aged less than 50 years from diabetic ketoacidosis or hypoglycaemia was surprising and concerning.

The higher proportion of females prescribed antihypertensive medication (compared with males) was an unexpected finding, but it is consistent with results of the most recent New Zealand Health Survey which found that the prevalence of self reported hypertension was higher amongst females than males in the 65–74 and 75+ age groups.[26] Also, among patients aged over 60 years registered at Swedish primary health centres males had generally better blood pressure control (defined as less than 140 mmHg systolic and/or 85 mmHg diastolic) than females.[27]

Cardiovascular disease was not unexpectedly the most common cause of death. While this study did not compare cardiovascular mortality rates between diabetic and general or non-diabetic populations, many studies have found that the death rate for cardiovascular disease (particularly ischaemic heart disease) is higher among diabetic populations compared with the general population.[1,3,28] There is no reason why this would not be the case for the Otago region.

Diabetes prevalence is increasing worldwide, yet the impact on mortality cannot be accurately monitored. Under-reporting of diabetes on death certificates was recognised more than two decades ago, and this study found that this practice continues. If the impact of the diabetes epidemic on mortality is to be monitored appropriately in New Zealand, attention needs to be given to improving the completion of death certificates, including always recording diabetes when it is present, irrespective of whether it is considered to be the underlying or a contributing cause of death.

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## Indigenous disparities in disease-specific mortality, a cross-country comparison: New Zealand, Australia, Canada, and the United States

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### Abstract

**Aims** To compare the disease-specific mortality rates of the indigenous populations of New Zealand, Australia, Canada, and the United States with the non-indigenous populations in each country.

**Methods** For New Zealand, Australia, Canada, and the United States, we compiled and calculated (from crude data) ethnic-specific mortality rates by primary cause of death in 1999 for the indigenous and non-indigenous populations in each country. We calculated age-adjusted mortality rates, using direct standardisation and weights based on the World Health Organization world population.

**Results** Australia experienced the largest relative and absolute disparities in life expectancy between indigenous and non-indigenous populations. For specific causes of death, New Zealand Maori, and Australian Aboriginals and Torres Strait Islanders experienced the highest levels of disparities when compared to their respective non-indigenous population group. Large disparities exist for indigenous peoples in all four countries for diabetes mortality.

**Conclusion** The indigenous peoples of New Zealand and Australia suffer from high disease-specific mortality rates. The relative size of indigenous/non-indigenous mortality disparities are highest in New Zealand and Australia. There appears to be a number of common issues that adversely affect the quality of the mortality data that is available in the four countries. Action is required to address indigenous health disparities and to improve the quality of indigenous mortality data.

## Background

Disparities in health status for the indigenous peoples of New Zealand, Australia, Canada, and the United States (US) have been well documented.[1–6] In each of these 'rich' countries, the indigenous peoples invariably suffer from poorer health, with an excess of early mortality and lower life expectancy when compared to the non-indigenous population.

Numerically, the indigenous populations of each country represent a small proportion of the total population. Maori represent approximately 15% of the New Zealand population, Aboriginals and Torres Strait Islanders represent 2–3% of the Australian population, American Indians and Alaskan Natives represent 1–1.5% of the US population, and Aboriginal Canadians represent 4% of the Canadian population.[7–10]

Although gains in health status have been made for all the indigenous peoples of these four countries, large disparities remain. In New Zealand, a recent report has highlighted that although life expectancy has improved dramatically for non-Maori

non-Pacific people, Maori life expectancy has remained largely static, leading to a relative increase in the life expectancy disparity experienced by Maori.[11] In particular, Maori disease specific mortality disparities have increased for cardiovascular disease and cancer when compared to non-Maori.[11]

The aim of the research is to compare the disease specific mortality rates of the indigenous population of New Zealand, Australia, Canada, and the US with the non-indigenous population in each country. The New Zealand population is the reference population for which all comparisons are made. The size of the relative disparities (indigenous/non-indigenous) in disease-specific mortality rates, within and between countries are compared.

## Method

Life expectancy at birth data for New Zealand (2000-2002) was obtained from Statistics New Zealand.[12] Life expectancy data for the US (2001) was obtained from the Centers for Disease Control and Prevention (CDC).[13] Life expectancy data for Australia (2000) was obtained from the Australian Institute of Health and Welfare.[14] Life expectancy data for Canada (2000) was obtained from Health Canada.[4] Mortality risk ratios for comparison are those accounting for the leading causes of death in New Zealand and the US in 1999.[15,16] These included malignant neoplasms of the lung, bowel, cervix, female breast, and prostate; ischaemic heart disease; cerebrovascular disease; chronic obstructive pulmonary disease (COPD); intentional self harm; diabetes; human immunodeficiency virus (HIV); assault; pneumonia and influenza.

Mortality data for New Zealand are compiled by the New Zealand Health Information Service (NZHIS).[15] Cause of death in 1999 was defined by International Disease Classification–9th edition (ICD–9) codes (Appendix 1). Crude mortality data (1999) for this study was obtained from the NZHIS. The national mortality dataset in New Zealand contains ethnicity information. Since 1996, the ethnicity question recorded on death registration certificates has been the same as that asked in the 1996 national census of population and dwellings.[15] Mortality data for the US are compiled by the National Center for Health Statistics of the US CDC.[17] Cause of death in 1999 was defined by ICD-10 codes (Appendix 1). Crude mortality data for this study was obtained from the CDC. The national mortality dataset in the US contains race information.[18]

Mortality data for Australia is complied by the Australian Bureau of Statistics.[19] Currently, there is incomplete coverage of indigenous deaths in some state and territory registration systems in Australia. Therefore, the mortality data used for this study was from those jurisdictions assessed by the Australian Bureau of Statistics as having a sufficient level of coverage to enable statistics on Aboriginal and Torres Strait Islanders to be produced. These states and jurisdictions include Queensland, South Australia, Western Australia, and the Northern Territory.[8] The Australian Institute of Health and Welfare supplied crude mortality data (1999) for this study.[20] Cause of death in 1999 was defined by ICD–10 codes. The Australian population denominator values used in this study were derived from the 2001 census.[20]

Statistics Canada supplied mortality data for Canada, for the population group 'all Canadians'. The national mortality dataset held by Statistics Canada does not contain ethnicity data. At present there is no mortality data available for off-reserve indigenous Canadians. Indigenous mortality data (1999) was only available for First Nation on-reserve indigenous Canadians.[21] Crude mortality data for First Nation on-reserve indigenous Canadians was obtained from Health Canada (First Nations and Inuit Health Branch).[22] Cause of death in 1999 was defined by ICD–9 codes. For New Zealand, Australia, Canada, and the US, we complied and calculated from crude data ethnic specific mortality rates by primary cause of death in 1999 for the indigenous and non-indigenous populations. We calculated age-adjusted mortality rates, using direct standardization[23] and weights based on the WHO world standard population.[24] We also used New Zealand, Australian, Canadian, and US-based weights; and Segi standard population-based weights[15]—and found results similar to those presented here.

## Results

Life expectancy overall for males (76.6 years) and females (82.1 years) was highest in Australia (see Table 1). Male indigenous life expectancy was highest in New Zealand (69.0 years) and female indigenous life expectancy was highest in Canada (76.6 years). The lowest life expectancy for indigenous peoples for both males (56 years) and females (63 years) was in Australia. Australian Aboriginals and Torres Strait Islanders, therefore, experienced the greatest disparity in life expectancy, when compared to the non-indigenous population.

Maori had the highest mortality rates among all population groups (see Table 2), for ischaemic heart disease, COPD, total malignant neoplasms and malignant neoplasm of the lung, female breast, prostate, and cervix. Non-Maori New Zealanders had the highest mortality rate for malignant neoplasm of the bowel among all population groups. The only three disease-specific mortality rates measured where Maori mortality was lower than non-Maori mortality occurred in malignant neoplasm of the bowel, pneumonia and influenza, and intentional self-harm.

Australian Aboriginals and Torres Strait Islanders had the highest mortality rates among all population groups for cerebrovascular disease and diabetes. When indigenous mortality rates were compared with non-indigenous mortality rates in Australia, Aboriginal and Torres Strait Islander mortality rates were higher for every disease-specific mortality rate measured, except for malignant neoplasm of the bowel.

Canadian First Nation peoples had the highest mortality rate among all population groups for intentional self-harm and, pneumonia and influenza. Indigenous mortality rates were lower than non-indigenous mortality rates in Canada for total malignant neoplasms, malignant neoplasm of the lung and female breast, ischaemic heart disease, cerebrovascular disease, and COPD.

American Indians and Alaskan Natives had the highest mortality rate among all the population groups for assault. Indigenous mortality rates were lower than non-indigenous mortality rates in the US for total malignant neoplasms and each of the individual neoplasms reported (lung, bowel, female breast, cervix, and prostate), ischaemic heart disease, cerebrovascular disease, HIV and COPD.

In terms of the size of the relative disparities that exist between population groups within a country, New Zealand Maori and Australian Aboriginals and Torres Strait Islanders experienced the highest levels of disparities when compared to their respective non-indigenous population groups (Figures 1–3).

The size of mortality risk ratio for indigenous/non-indigenous populations groups (Table 2) across all four countries was highest in New Zealand for total malignant neoplasms (risk ratio [RR] 1.6) of the lung (RR 2.9), breast (RR 1.5), cervix (4.5), and prostate (RR1.5); HIV (2.0) and ischaemic heart disease (RR of 1.9, which was the same as the Australian indigenous/non-indigenous RR). In Australia, the size of mortality risk ratio for indigenous/non-indigenous populations groups was the highest for all four countries for: cerebrovascular disease (RR 2.1), COPD (RR 2.5), pneumonia and influenza (RR 2.1), diabetes (RR 9.8), ischaemic heart disease (RR 1.9), and assault (RR 5.6).

**Table 1. Life expectancy at birth (years)**

| Sex | New Zealand | | | Australia | | | Canada | | | United States | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Relative Difference | Maori | All | Relative Difference | Aboriginal | All | Relative Difference | First Nation | All | Relative Difference | AIAN | All |
| Males | 0.9 | 69.0 | 76.3 | 0.73 | 56 | 76.6 | 0.9 | 68.9 | 76.3 | 0.91 | 67.4 | 74.1 |
| Females | 0.9 | 73.2 | 81.1 | 0.77 | 63 | 82.1 | 0.94 | 76.6 | 81.8 | 0.93 | 74.2 | 79.5 |

AIAN=American Indian and Alaskan Native.

**Table 2. Age standardised mortality rates (per 100,000 population)**

| Disease | New Zealand | | | Australia | | | Canada | | | United States | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **I** | **NI** | **RR** | **I** | **NI** | **RR** | **I** | **NI** | **RR** | **I** | **NI** | **RR** |
| Total malignant neoplasms | 228.2 | 146.1 | 1.6 | 149.9 | 124.3 | 1.2 | 98.9 | 143.0 | 0.7 | 91.3 | 142.0 | 0.6 |
| Malignant neoplasm of trachea, bronchus, and lung | 74.6 | 25.6 | 2.9 | 45.4 | 25.3 | 1.8 | 29.3 | 39.6 | 0.7 | 25.7 | 40.3 | 0.6 |
| Malignant neoplasm of breast (female) | 18.9 | 12.9 | 1.5 | 12.0 | 9.0 | 1.3 | 9.6 | 11.3 | 0.8 | 6.5 | 11.1 | 0.6 |
| Malignant neoplasm of the prostate | 14.8 | 9.6 | 1.5 | NA | NA | NA | 8.8 | 7.4 | 1.2 | 2.9 | 7.0 | 0.4 |
| Malignant neoplasm of the cervix uteri | 5.4 | 1.2 | 4.5 | NA | NA | NA | 2.1 | 1.1 | 1.9 | 0.9 | 1.2 | 0.8 |
| Malignant neoplasm of colon, rectum, and anus | 16.2 | 22.6 | 0.7 | 7.6 | 16.2 | 0.5 | 13.1 | 11.4 | 1.1 | 8.2 | 14.1 | 0.6 |
| Ischaemic heart diseases | 206.1 | 110.2 | 1.9 | 162.6 | 87.1 | 1.9 | 83.8 | 89.6 | 0.9 | 83.1 | 118.9 | 0.7 |
| Cerebrovascular diseases | 55.2 | 47.2 | 1.2 | 73.8 | 35.5 | 2.1 | 28.1 | 30.9 | 0.9 | 25.3 | 35.6 | 0.7 |
| Other chronic obstructive pulmonary disease | 34.0 | 19.6 | 1.7 | 33.7 | 13.7 | 2.5 | 10.8 | 16.1 | 0.7 | 15.8 | 22.7 | 0.7 |
| Intentional self-harm | 12.9 | 13.1 | 1.0 | 19.4 | 11.9 | 1.6 | 27.8 | 12.2 | 2.3 | 12.0 | 9.8 | 1.2 |
| Pneumonia and influenza | 9.9 | 10.3 | 1.0 | 13.2 | 6.2 | 2.1 | 24.4 | 17.7 | 1.4 | 14.0 | 13.2 | 1.1 |
| Diabetes mellitus | 62.5 | 11.0 | 5.7 | 85.4 | 8.7 | 9.8 | 19.5 | 13.3 | 1.5 | 36.2 | 16.7 | 2.2 |
| Human immunodeficiency virus | 1.0 | 0.5 | 2.0 | NA | NA | NA | NA | 1.2 | NA | 2.9 | 5.0 | 0.6 |
| Assault | 3.9 | 1.0 | 3.9 | 7.8 | 1.4 | 5.6 | 8.1 | 1.5 | 5.4 | 10.6 | 6.4 | 1.7 |

I=indigenous; NI=non-indigenous; RR= risk ratio.

**Figure 1. Maori/non-Maori mortality risk ratio versus Australian Indigenous/non-Indigenous risk ratio in New Zealand and Australia respectively**
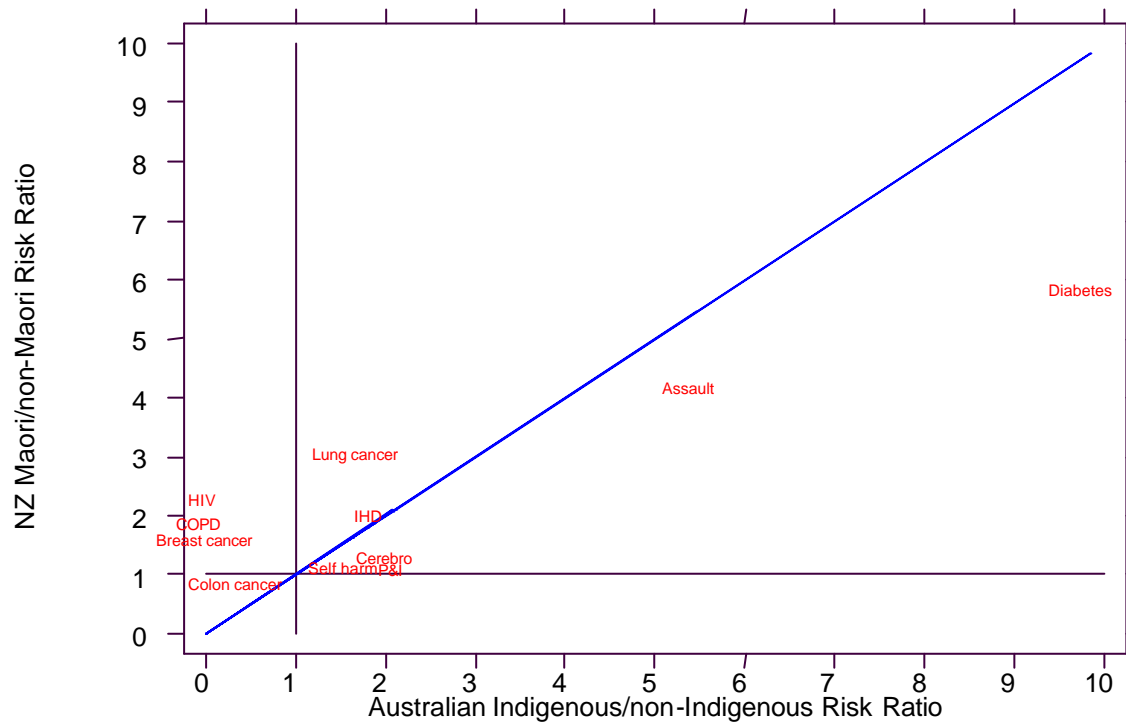
**Figure 2. Maori/non-Maori mortality risk ratio versus Canadian Indigenous/non-Indigenous risk ratio in New Zealand and Canada respectively**
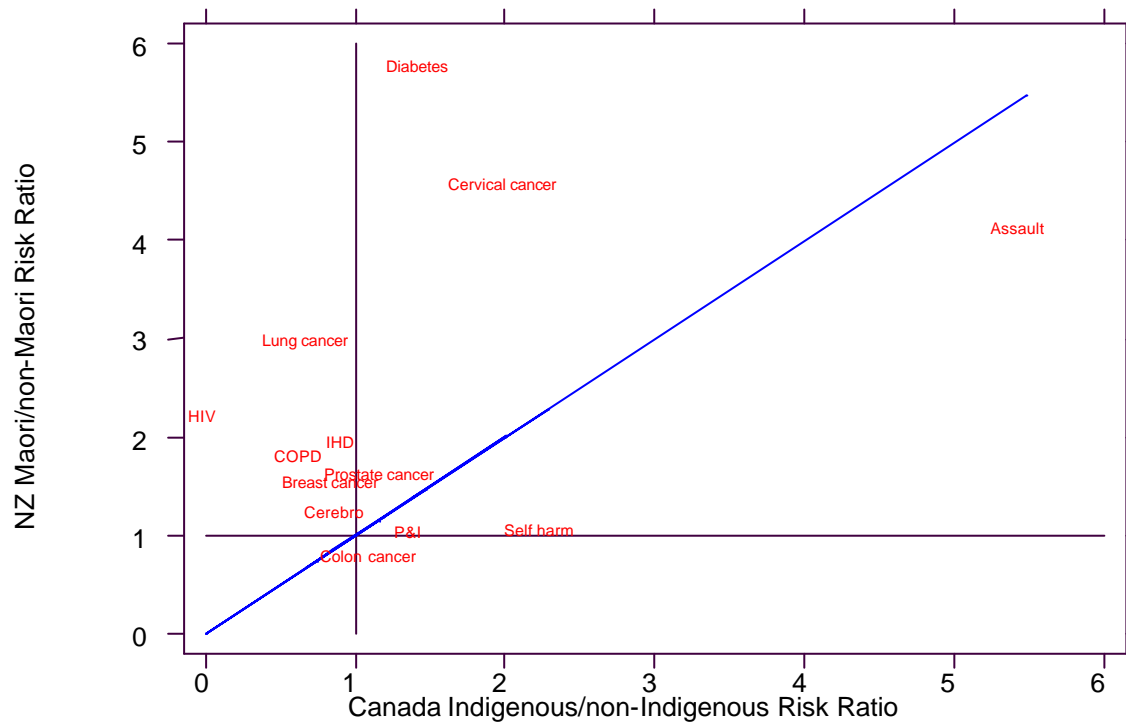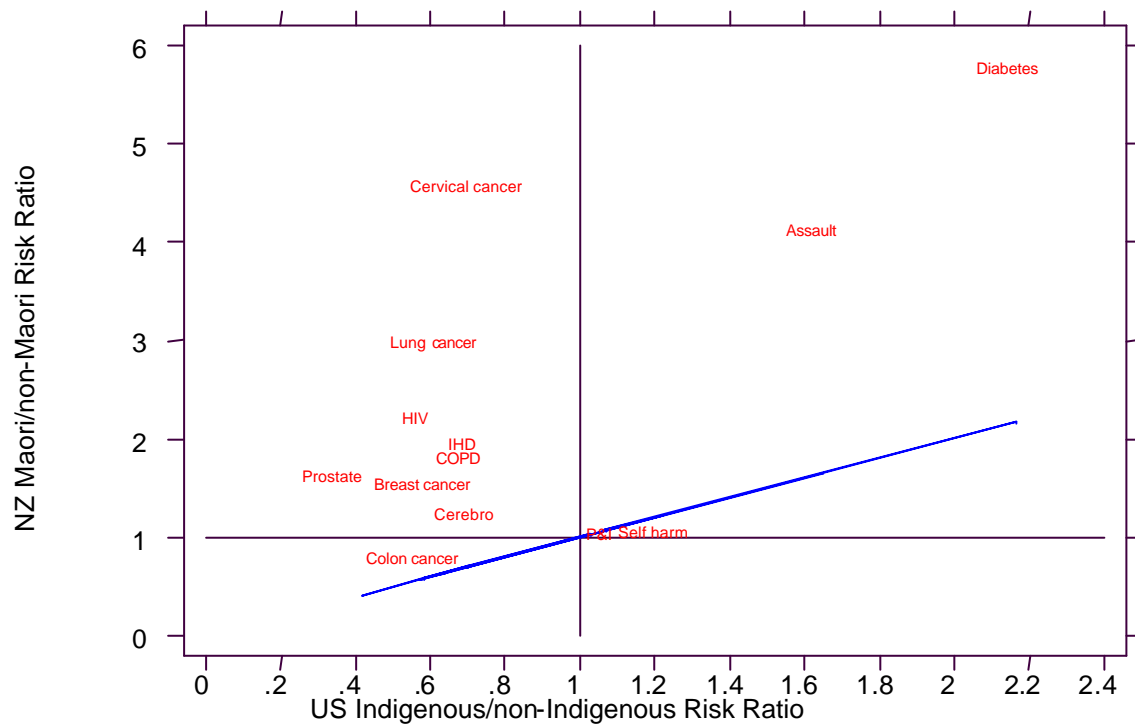
**Figure 3. Maori/non-Maori mortality risk ratio versus US Indigenous/non-Indigenous risk ratio in New Zealand and the US respectively**

**Appendix 1. International Classification of Disease (ICD) codes used for defining major causes of death**

| ICD-10 DESCRIPTION | ICD-9 CODES | ICD-10 CODES |
|---|---|---|
| Malignant neoplasms | 140-208 | C00-C97 |
| Malignant neoplasm of trachea, bronchus, and lung | 162 | C33-C34 |
| Malignant neoplasm of the female breast | 174 | C50 |
| Malignant neoplasm of prostate | 185 | C61 |
| Malignant neoplasm of cervix uteri | 180 | C53 |
| Malignant neoplasm of colon, rectum, and anus | 153-154 | C18-C21 |
| Ischaemic heart diseases | 410-414 | I20-I25 |
| Cerebrovascular diseases | 430-434, 436-438 | I60-I69 |
| Other chronic obstructive pulmonary disease | 496 | J44 |
| Intentional self-harm | E950-E959 | X60-X84, Y87.0 |
| Diabetes mellitus | 250 | E10-E14 |
| Human immunodeficiency virus disease | 042-044 | B20-B24 |
| Assault | E960-E969 | X85-Y09, Y87.1 |

Across all four countries (New Zealand, Australia, Canada, and the US) the indigenous peoples had higher mortality rates for diabetes and assault when compared to their non-indigenous populations.

## Discussion

This paper compares indigenous and non-indigenous disease-specific mortality rates and risk ratios (in New Zealand, Australia, Canada, and the US) for the leading causes of death in New Zealand. There have been a limited number of academic papers published comparing indigenous disparities in mortality among rich countries.[25–28] In 1992, Hogg attempted to place Australian Aboriginal mortality within the broader context of other countries. Hogg found that Australian Aboriginals had higher age and cause-specific death rates, and a strikingly different mortality profile overall compared to indigenous peoples in New Zealand, the US, and Canada.[25]

The main findings of this research are that:

Life expectancy in all four countries was lower for the indigenous peoples—with Australian Aboriginals and Torres Strait Islanders having the lowest life expectancy of all population groups and the greatest relative disparity when compared to the non-indigenous population.

The highest disease-specific mortality rates for ischaemic heart disease and malignant neoplasms are found in New Zealand Maori (except for malignant neoplasm of the bowel where New Zealand non-Maori have the highest rate). Canadian First Nation peoples have the highest mortality rates of all population groups for intentional self-harm and pneumonia/influenza. American Indians and Alaskan Natives have the highest mortality rates of all population groups for assault. Non-indigenous Americans have the highest mortality rate for HIV.

In terms of the size of the relative disparities that exist for disease-specific mortality, New Zealand Maori, Australian Aboriginals and Torres Strait Islanders have the highest levels of disparities when compared to their non-indigenous population groups.

Diabetes is a powerful determinant of health outcome and for indigenous peoples across the four countries, diabetes related mortality is high. Australian Aboriginals and Torres Strait Islanders, in particular, have very high mortality rates associated with diabetes—and the indigenous/non-indigenous risk ratio of 9.8 was the highest reported. The prevalence of diagnosed diabetes has in recent years been increasing in all four indigenous populations.[29–32] Also, the prevalence of obesity is increasing in some indigenous populations,[30,32] this will result in a rise in diabetes related mortality in the near future.

The current high levels of indigenous mortality and disparities that exist in New Zealand and Australia are not acceptable. In comparison, indigenous mortality in Canada and the US is lower in many of the disease-specific areas reported in this study when compared to their non-indigenous counterparts.

There are several cross-country and country-specific lessons that should be explored following these analyses. For example, cancer deaths in indigenous Americans (and to a lesser extent, cancer deaths in indigenous Canadians) are very low—these findings are consistent with other published reports. Low indigenous mortality rates for lung

cancer in the US may be partially explained by the rarity of habitual cigarette smoking among Southwest tribes but reasons for the low rates of other cancers are not so evident.[33] Cobb, in a recent report on indigenous cancer deaths in the US, stated that further research is required to elucidate why American Indians have low cancer mortality. This research may have significant implications for cancer prevention in other ethnic groups.[33]

Although disparities are large in New Zealand for death from assault, the absolute rates are lower than in the US. For example, the Maori age-standardised mortality rate from assault is 3.9 per 100,000 (RR of 3.9 compared to non-Maori) compared to the non-indigenous rate of 6.4 per 100,000 in the US. Further research should be undertaken to explore how the national response to violence differs between countries. A review of factors that have been successful in keeping death related to assault comparatively low in New Zealand may have implications for policy development in the US.

New Zealand has a low annual incidence of new HIV infections and subsequent low mortality rates as reflected in the study findings (although new infections have been increasing in recent years). The New Zealand response to the HIV epidemic has been viewed as a public health success story. The New Zealand response was characterised by law change (the Homosexual Law Reform Act was passed), national coordination of a policy response (the National Council on AIDS and a medical advisory committee were formed), and empowerment of affected communities (groups such as the AIDS Foundations, Injecting Drug User Community Groups, and the New Zealand Prostitutes Collective were formed).[34] Such a public health approach could be undertaken to protect the health of indigenous and non-indigenous populations in other countries.

The publication of comparative data such as this should stimulate increased cross-country learning, research, and policy development.

## The quality of indigenous mortality data

There are several common issues that adversely affect the quality of indigenous mortality data. These include the lack of an accurate denominator value for the indigenous population concerned (mainly due to undercounting) and the lack of agreement over which population denominator values to use if they do exist (e.g., whether to use single ethnic response groups as the denominator value vs the multiple ethnic response groups).

Denominator values for the indigenous population in all four countries are usually derived from census data. However, in Australia, estimating the size of the Aboriginal and Torres Strait Islander population has proved difficult due to uncertainties attached to interpreting indigenous population counts from the 5-yearly census.[8]

Between 1996 and 2001, the Australian indigenous population increased 16 %, however the expected increase based on natural increase (births minus deaths) was 12%.[8] This variance is in part due to the increased propensity of indigenous people to self-identify as indigenous on the census forms. As it is not possible to estimate how these factors may change over time, it is therefore problematic to estimate the inter-census population denominator counts that are needed to calculate annual death rates.

There is a lack of agreement as to how official agencies define indigenous status and the way in which ethnic specific mortality data is recorded. In New Zealand (as in other countries), there has been frequent modification of the ethnicity question recorded in the censuses and it was not until 1991 census that the biological concept of ethnic origin was replaced with that of self-identified ethnicity.[35] These frequent changes in the census ethnicity question has led to difficulty comparing mortality trends over time and have also produced difficulties in estimating inter-census population denominator counts.

Perhaps the most important issue in regards to the quality of indigenous mortality data is the undercounting of deaths (the numerator for mortality data). In each of the four countries, the undercounting of indigenous deaths is likely to lead to an underestimation of the relative size of disparities that exist between indigenous and non-indigenous populations. In Australia, for example, the Australian Bureau of Statistics (which administers the national mortality database), recommends that the coverage of indigenous mortality data is of sufficient quality to be used for research purposes only from the jurisdictions of Queensland, South Australia, Western Australia, and the Northern Territory.[8] This is primarily due to the fact that indigenous ethnicity status on death certificates is not always recorded, or recorded incorrectly, leading to an undercounting of the number of indigenous deaths.[8]

In New Zealand, research has been undertaken that attempts to adjust for this undercounting by a process of probabilistic record linkage of death registration data with census data. This research has produced estimates of the considerable extent of the undercounting of Maori deaths.[36,37] Unfortunately, this data could not be used for this study as there was no similar 'corrected' mortality data available from the US, Australia, or Canada.

An issue that is unique to Canada is that the national mortality database administered by Statistics Canada does not contain ethnicity data. The regional offices of Health Canada collect mortality data for the indigenous, on-reserve, First Nations population. Via a series of partnerships with each provincial vital statistics registrar, First Nations specific death certificate information is sent to the regional First Nations and Inuit Health Branch regional office. However, in a number of areas no such relationships exist (for example the Atlantic, Ontario, and Quebec regions), and therefore data is obtained directly from the local communities, or not at all.[21] The availability of indigenous mortality data in Canada is further limited by the lack of information that is available for off-reserve, or non-status, indigenous peoples.

## Methodological considerations

The varying degrees of completeness and accuracy of the indigenous mortality databases that exist within the four countries are likely to affect these findings. For example research by Ajwani (2003), has reported that during 1996-1999, 7% more decedents identified Maori as one of their ethnic groups on the 1996 census compared with mortality data.[38] Therefore the accuracy of the Maori deaths rates used in this study is relatively high. This level of accuracy is unlikely to be present in the three other countries.

In the US, some estimates of the under-reporting of American Indian deaths have ranged from 11% to 25%.[39,40] In Canada, it is difficult to determine an accurate

overview of indigenous mortality for the reasons reported already, and due to the fact that only limited information is available regarding indigenous people that reside in urban areas.

The implication of these findings may therefore be that when New Zealand indigenous/non-indigenous mortality risk ratios are compared with indigenous/non-indigenous mortality risk ratios from these countries (Figures 1–3), the results may be somewhat improved to that described.

Although it is impossible to quantify the exact amount of the measurement bias that may exist in our calculations, the data presented here is the most reliable currently available. Differences in the calculation of life expectancy and in ICD coding practices between countries could bias the findings, but this is likely to have a minimal effect on the relative differences in mortality between indigenous and non-indigenous populations within a country, which is the main focus of this paper. It should also be noted that grouping of data for indigenous peoples may obscure important differences that may exist between large tribal grouping, an issue that may be particularly important in North America where mortality and other health status indicators vary widely between tribal and geographical indigenous populations. Further, this analysis was for a 1-year period, if a longer period were available for analysis, this could increase the consistency of the rates reported.

## Conclusion

The indigenous peoples of New Zealand and Australia suffer from high disease-specific mortality rates. The relative size of indigenous/non-indigenous mortality disparities are highest in New Zealand and Australia. There appears to be a number of common issues that adversely affect the quality of the mortality data that is available in the four countries. Action is required to address indigenous health disparities and to improve the quality of indigenous mortality data

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# Development of reversible diabetes mellitus after cessation of interferon-alpha therapy for chronic hepatitis C infection

Tsung-Neng Tasi, Chang-Hsun Hsieh

Interferon-alpha (INF-alpha) is widely used in a number of neoplastic and viral diseases, including chronic hepatitis C virus (HCV) infection. The reported cumulative incidence of diabetes mellitus (DM) associated with INF-alpha therapy varies from 0.08 to 0.7%.[1,2] The onset of DM can occur frequently during interferon treatment,[3] but rarely following the cessation the INF-alpha therapy.[4,5] We describe such a case.

## Case report

A 39-year-old Chinese male with a chronic active hepatitis C infection (without evidence of cirrhosis—as confirmed by liver biopsy) came to our hospital for treatment with Peginterferon alfa-2b (Pegintron, Schering-Plough, Keniworth, NJ) and Ribavirin (Schering-Plough).

The patient had no overt signs of diabetes and there was no familial history of diabetes. There was abnormal liver function as evidenced by an alanine aminotransferase (ALT) level of 312 IU/L and an aspartate aminotransferase (AST) level of 375 IU/L. Serum anti-HCV showed a positive result with a viral load of 489,000 copies/ml as determined by real time PCR.

The patient was started on a therapeutic regimen of INF-alpha-2b (100 MCG weekly) and Ribavirin (1000 mg/day). The treatment lasted 9 months. Serum conversion was detected after combination therapy (HCV RNA virus load < 100 copies/ml) and normalisation of the liver function test. Normal fasting plasma glucose (FPG) had been detected during treatment (86–95 mg/dl).

Three months following the end of the INF-alpha therapy, the patient suffered from polyuria and developed an excessive thirst. Laboratory analysis of blood detected elevated levels of randomised plasma glucose (766 mg/dl), FPG (288 mg/dl), and haemoglobin glycated A1c (HbA1c) (12.7%). However, elevated ALT and AST levels of 193 IU/L and 138 IU/L combined with positive serum anti-HCV result suggested recurrence of HCV infection considered. Ketoacidosis was absent. Poor plasma glucose control resulted using oral hypoglycaemia agents, but the patient later displayed an acceptable response to the administration of insulin, which produced a glycaemic level of 150–180 mg/dl.

Insulin was continuously administered. Glycaemic control was achieved during outpatient follow-up. The use of insulin was discontinued after 8 weeks. Normal FPG level was measured at 70 mg/dl and HBA1c was 5.5% during an outpatient visit. No further insulin or OHA therapy was required.

# Discussion

Little data has been presented in the literature pertaining to the frequency of serious adverse reactions during INF-alpha treatment, particularly concerning glucose intolerance. It has been reported that INF-alpha impairs glucose tolerance,[6] consistent with a role as an independent risk factor for the development of glucose intolerance.[7] Accordingly, it is reasonable to propose that DM may develop with the autoimmune disruption of pancreatic beta cells. Such a role for INF-alpha therapy is consistent with previous studies in the past.[4,8–10]

The frequency of INF-alpha-induced autoimmunity of diabetes varies from 0.08–0.7%,[1,2] occurring for the most part during the period of treatment.[3] The majority of these patients displayed an autoimmune reaction to glutamaic acid decarboxylase (GAD Abs), tyrosine phosphate Abs (IA2), insulin Abs (IAA), or islet cell Abs (ICA).[3] Unfortunately, current technical limitations prevented a similar detection of an autoimmune marker. Nonetheless, our other observations are supportive of the role of the interferon treatment as a trigger for the development of DM.

It has been observed that impaired peripheral insulin resistance and defective insulin secretion were seen in hepatitis C infection,[11] regardless of stage, and in cases with liver cirrhosis. However, our initial biopsy did not show evidence of liver cirrhosis. Besides, relapse of hepatitis C infection was seen 3 months after cessation of INF-alpha therapy, which may disclose chronic hepatitis C per sc may not play a direct role in the development of diabetes mellitus in our case. INF-alpha may at least partly be response for the development of diabetes mellitus.

Development of autoimmune form of diabetes is usually irreversible. However, there have been reports of transient insulin dependency associated with INF-alpha therapy.[4,5,9,10] Only a few cases have occurred after cessation of INF-alpha therapy.[4,5] Autoimmune attack may be partially reversible—with the interruption of INF-alpha or INF-alpha-related insulin resistance, or due to the defective secretion of insulin.

The rare development of DM following interferon therapy is a challenge to explain. Presently, the INF-alpha-triggered autoimmune destruction of beta-cells is one obvious cause. Glucose intolerance induced by INF-alpha is another possible explanation. Another avenue of exploration involves the time necessary for the induction of DM upon INF-alpha treatment. The literature is not substantive, with estimates ranging from 10 days to 4 years.[3] Finally, the even rarer development of DM following the termination of therapy is poorly understood.

In conclusion, INF-alpha can induce diabetes both during the period of active therapy and following its removal. Monitoring for the appearance of pancreatic autoantibodies may thus be a prudent precaution in patients undergoing interferon therapy, even in those with no predisposing diabetic factors.

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# Thyroid papillary carcinoma in subhyoid ectopic thyroid tissue

Hsin-Yu Lee, Mei-Hsiu Chen, Chih-Yuan Wang

## Abstract

Ectopic thyroid tissue is a rare entity in thyroidology; however, the occurrence of thyroid carcinoma in such aberrant thyroid tissue has been reported. Carcinoma arising in subhyoid thyroid is especially unusual, with even fewer reports published. Usually, surgical excision is considered to be therapeutic strategy for managing possible malignancy in ectopic thyroid. Thyroid ultrasonography, radioactive iodine scanning, and fine-needle aspiration cytology are rapid, safe, and minimally invasive diagnostic procedures. We report one case of papillary carcinoma in subhyoid ectopic thyroid to emphasise the importance to evaluate all ectopic thyroid tissues.

Thyroid tissue may develop in unusually anatomical location, i.e. ectopic thyroid. An ectopic thyroid can occur anywhere along the course followed by thyroglossal duct during its embryonic descent from the tongue—resulting in a lingual, suprahyoid, subhyoid, or even an intratracheal thyroid.[1] A thyroglossal duct cyst is commonly associated with the clinical ectopic thyroid. Since ectopic thyroid tissue always raises the possibility of metastatic thyroid cancer, it is pivotal to identify the possible malignancy when an ectopic thyroid or thyroglossal duct cyst is noted.[2] However, the absence of a normal thyroid gland may occur in the patients with clinically evident ectopic thyroid.

Removal of such ectopic tissue can lead to permanent hypothyroidism.[2] Therefore, delicate investigations for ectopic thyroid should be carried out to decide therapeutic intervention before operation. Here, we report a case of subhyoid ectopic thyroid to emphasise the importance for evaluating such patients.

## Case report

A 50-year-old Chinese woman presented to her local practitioner with a 5-year history of a painless and elastic mass in the midline of neck, moving up and down with swallowing in the region of hyoid bone. Under the impression of thyroglossal duct cyst, she was referred by the local practitioner to our hospital for surgical intervention.

A thyroid function test revealed a euthyroid state, and anti-thyroglobulin or anti-microsomal antibody could not be detected. A thyroid I-[131] scan revealed heterogenous and multinodular goitre, with ectopic I-[131] uptake in the subhyoid area. Neck ultrasonography showed a bilateral fine multinodular goitre, and a 1.66 x 1.64 x 1.58 cm solid, hypoechoic, heterogenous tissue just below the hyoid bone.

Perinodular vascularisation was noted via colour Doppler ultrasound (Figure 1). Fine-needle aspiration cytology of this mass showed clusters of follicular cells with larger cellular nuclei by rapid staining.[3] Ectopic thyroid was favoured with potential

neoplastic change, and an operation was suggested. Pathological examination showed papillary carcinoma arranged in papillae with ground-glass nuclei in ectopic thyroid; and adenomatous goitre without primary carcinoma was found in thyroid parenchyma. No lymph nodes metastasis was recognised. Postoperative I-$^{131}$ ablation (30 mCi) was carried out, and cancer work-up revealed residual thyroid tissue without metastasis.

**Figure 1. Thyroid ultrasonography of ectopic thyroid showed a hypoechoic, heterogenous nodule with perinodular vascularisation (arrowed)**
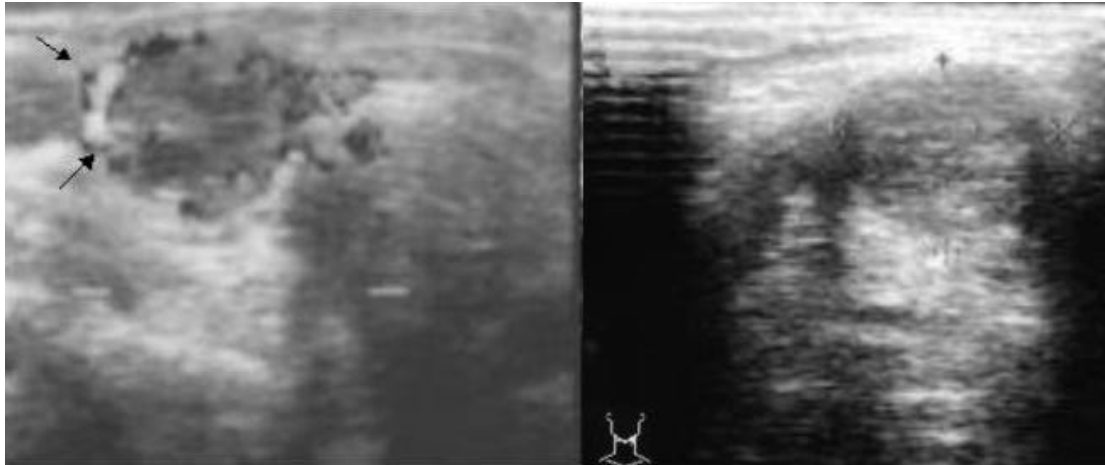


## Discussion

The thyroid gland is formed during embryonic stage as epithelial proliferation of the primitive alimentary tract. The median anlage of thyroid parenchyma migrates from the pharyngeal floor in the foramen caecum of the tongue (during the third to fourth week of development) and forms a shield in front of thyroid cartilage and trachea. Ectopic thyroid formation usually, represents an arrest of thyroid anlage in the descent along the normal pathway. These anatomically correctly positioned thyroid tissues are subject to nodular hyperplasia, and rarely neoplastic formation.

Several decades ago, lateral aberrant thyroid tissue was usually concluded to be metastatic cancer.[4] Surgical excision of ectopic thyroid seemed to be the best policy to identify possible malignancy in the past experience.[5] However, the old dictum was modified, because absence of the normal thyroid gland may occur in 70% of patients with ectopic thyroid.[2,6] Indeed, if the ectopic thyroid is benign and is the only thyroid tissue present, surgical excision of ectopic thyroid will result in permanent hypothyroidism.[2,7,8]

Therefore, to decide the correct therapeutic strategy between benign and malignant lesions, delicate investigations are indicated for such ectopic thyroid tissue. Thyroid ultrasonography, radioactive iodine scanning, and fine-needle aspiration cytology are safe, rapid, and minimally invasive methods for preliminary diagnosis in ectopic thyroid.

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## Thyrotoxic periodic paralysis in a Maori patient

Edward Wild

### Abstract

A case of thyrotoxic periodic paralysis (TPP) in a patient of Maori heritage is described. The epidemiology, aetiology and pathogenesis of TPP are discussed. The case demonstrates that neurological examination and biochemical findings may be normal between episodes of paralysis. Given that there is much racial variation in the prevalence of TPP, and the suggestion that non-thyrotoxic periodic paralysis may be more prevalent in Maori, the case highlights the need for more research into the prevalence and pathogenesis of TPP in Maori patients.

Thyrotoxic periodic paralysis is a rare complication of thyrotoxicosis in New Zealand. In the acute setting, it causes transient flaccid paralysis with hypokalaemia. Between attacks, examination and biochemistry may be normal. It is more common in Asian than Western populations, but the incidence in Maori is unknown.

## Case report

A 25-year-old man of New Zealand Maori ethnicity was referred to the neurology clinic following two episodes of transient limb paralysis.

The episodes had occurred within 1 month. On both occasions, he had awoken early in the morning, unable to move his upper or lower limbs. After 3 hours, there was complete recovery of strength with no residual deficit. The paralysis was painless and there was no disturbance of consciousness. No unusual precipitating activity was reported prior to the attacks. As the patient had remained at home during the episodes, no examination or blood tests could be performed during attacks.

In the preceding 3 months the patient had experienced declining strength, and lost 5 kilograms in weight. There was no significant past medical history or family history, and he took no medicines.

Physical examination and routine biochemistry and haematology values were unremarkable. Serum thyroid stimulating hormone (TSH) was undetectably low; free T3 was 20.7 pmol/L (2.2–5.5 pmol/L) and free T4 48.9 pmol/L (2.2–5.5 pmol/L). Thyroid microsomal antibodies were detected at 1:1600 dilution. A radioisotope scan of the thyroid gland demonstrated diffuse, homogeneous, avid uptake consistent with Graves' disease.

A diagnosis was made of periodic paralysis as the presenting feature of thyrotoxicosis due to Graves' disease. The patient was commenced on a reducing dose of carbimazole. After 4 months, thyroid function returned to normal. The patient's strength returned and there were no further episodes of paralysis.

## Discussion

Thyrotoxic periodic paralysis (TPP) is characterised by episodic muscle weakness occurring in a thyrotoxic patient, without a familial periodic paralysis syndrome. While a rare complication of thyrotoxicosis in Western populations (occurring in 0.2% of American thyrotoxic patients[1]), racial variations exist in its incidence in Oriental populations where it occurs in around 2% of thyrotoxics. There is a male-to-female ratio of 11:1 and it usually occurs between 20 and 40 years of age.[2]

The typical presentation is of rapid onset, bilateral, symmetrical, flaccid paralysis. It often occurs after exercise, after a large meal or on waking. The weakness resolves spontaneously between 1 and 36 hours.[3] Bulbar, ocular, and respiratory paralysis are rare.[1] It is usually painless but can be heralded by muscle stiffness or pain.[2]

Treatment consists of potassium supplementation and beta-blockade to terminate the acute episode, followed by correction of hyperthyroidism, which prevents further attacks. Definitive treatment of the underlying thyrotoxicosis with surgery or radioiodine is recommended.

The pathogenesis of TPP is incompletely understood. An abnormality of potassium metabolism is thought likely: most patients are hypokalaemic during paralytic episodes. The $Na^+/K^+$/ATPase pump may be overactive in hyperthyroid tissue, and more so in patients with TPP. Catecholamine mediation of $Na^+/K^+$/ATPase activity may explain the ability of beta-blockers to terminate attacks. Activation of $Na^+/K^+$/ATPase by insulin may explain the triggering of paralysis by carbohydrate meals.[1] Nonetheless, normokalaemia during or between episodes does not exclude the diagnosis. The combination of flaccid paralysis and hyperthyroidism is adequate to secure the diagnosis clinically.[4]

To my knowledge, this is the second reported case of TPP occurring in a patient of Maori ancestry. The first case in a patient of Polynesian heritage was reported in 1994.[3] Fink and colleagues suggested that hypokalaemic periodic paralysis may be more common in Maori than New Zealanders of European heritage, although they excluded thyrotoxic patients from their analysis.[5]

Indeed, racial variation is significant in TPP: it has been suggested that a different ion transport defect is responsible for TPP in Asians and Caucasians.[6] Hence, further study of the pathogenesis of TPP in Maori is needed to investigate whether it more closely resembles a Caucasian or Asian pattern.

This case demonstrates that TPP may present not during an acute paralytic episode, but in the outpatient setting, where the examination and biochemistry (notably serum potassium) may be entirely normal. This affirms the diagnosis of TPP as a clinical one to considered in the assessment of intermittent paralysis.

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## Adolescent obesity and physical inactivity

Maea Hohepa, Grant Schofield, Gregory Kolt

**Abstract**

Globally, obesity and physical inactivity are two health issues affecting young people. In New Zealand, the most current statistics indicate that 33.6% of 11 to 14 year olds, and 27% of 15 to 18 year olds, are considered overweight or obese.[1,2] Despite these high prevalence levels, only 38% of young people aged 13 to 17 years in New Zealand are considered physically inactive.[3]

Future effort needs to be directed towards enhancing the existing national surveys to ensure a comprehensive and valid surveillance system of adolescent obesity and inactivity is conducted on a regular basis. This would involve the development of age, sex, and ethnic specific body mass index cut-off thresholds to define overweight and obesity, validation of an adolescent questionnaire that examines physical activity from a broad perspective, and development of physical activity recommendations for youth based on international best practice.

Although the main focus of this paper is on obesity and physical inactivity, diet is also a key determinant of obesity. Therefore, to provide an accurate assessment of factors associated with youth obesity in New Zealand, surveillance of diet must occur concurrently with that of obesity and physical activity.

The development of accurate measurement tools is critical for (1) determining obesity and inactivity trends, (2) identifying at-risk groups, (3) tracking progress toward national health priorities, and (4) evaluating the efficacy of interventions targeting obesity and physical inactivity. Furthermore, attention needs to be directed towards identifying correlates of inactivity and obesity to help inform the development of comprehensive multisectorial, multisetting, prevention, and management initiatives.

The worldwide prevalence of overweight and obesity among the adult population is reaching epidemic proportions, and evidence indicates children and adolescents are following in this trend. Obesity and physical inactivity are two interrelated health issues with a cyclic relationship. That is, physical inactivity plays a key role in the development and management of obesity, while obesity often impacts negatively on an individual's level of physical activity.

Obesity is a complex phenomenon that is influenced by genetic, behavioural, and environmental factors. A positive balance between energy expenditure and energy intake is a possible underlying cause of obesity. The increase in obesegenic living environments (i.e. environments that encourage sedentary pursuits, energy saving activities, and excessive consumption of high density, high fat food) has likely supported this positive energy balance. Current evidence supports an association between sedentary activities such as television viewing and obesity during childhood and adolescence.[4]

The financial burden of obesity and physical inactivity is substantial. It is estimated that obesity health care expenditure in New Zealand is NZ$303 million per annum.[5] Further estimates indicate a saving of NZ$25 million per year could result from a 5% increase in physical activity levels and that $160 million each year could be saved if all New Zealanders were to become physically active to levels that afford health benefits.[6,7]

As well as economic burden, obesity and physical inactivity have a significant impact on an individual's health and quality of life. Obesity and physical inactivity are two risk factors for a number of lifestyle related conditions including type 2 diabetes, coronary heart disease, hypertension, and some types of cancers.[5, 8]

By general consensus, participating in a minimum of 30 minutes of moderate intensity physical activity on most days of the week reduces the likelihood of developing these morbidities, and additional activity produces further health benefits. These findings are well summarised in the 1996 US Surgeon General's Report on Physical Activity.[8] Despite the benefits conferred by regular activity, physical inactivity in New Zealand ranks behind smoking as the second highest modifiable risk factor for poor health, and is associated with 8% of total deaths.[9]

Targeting child and adolescent obesity is a health priority for several reasons. First, severe childhood obesity is associated with a diverse range of morbidities including orthopaedic problems, sleep disorders, menstrual abnormalities, insulin resistance, and psychological issues of early discrimination and victimisation.[10] As well, persistence of obesity can lead to further long-term complications (including cardiovascular disease), and all cause mortality.[10] Second, children and adolescents with severe obesity are at greater risk of obesity persisting into adulthood. Specifically, compared to childhood obesity status, adolescent obesity is a stronger predictor of adulthood obesity.[11]

In addition to preventing obesity, the promotion of physical activity at an early age is beneficial for a number of reasons. Physical activity has been found to track over time,[12] and there is increasing evidence that regular physical activity among young people is associated with improvements in various health outcomes including blood lipid profile, blood pressure, body composition, glucose metabolism, bone strength, psychological health[13] and the maintenance of normal growth and development.[14]

Recently developed New Zealand health policy documents highlight the need to address obesity and physical inactivity. For example, the Healthy Action – Healthy Eating Report,[5] and the New Zealand Health Strategy[15] state that obesity and physical inactivity are two of top four health priorities for New Zealand. Despite well-developed national policy documents, the national prevalence of obesity and physical inactivity for New Zealanders (especially New Zealand youth) is not clear.

The purpose of this paper is two-fold. First, the question 'Are New Zealand youth obese and physically inactive?' will be examined and methodological flaws of current research highlighted. Second, future research directions based on identified gaps within New Zealand youth obesity and physical activity research will be presented. The focus of this paper is on adolescents (aged 13–17years old). Thus, the epidemiological evidence presented has been extracted from available data to ensure the focus is predominantly on people of this age range.

## New Zealand obesity trends

Before examining the obesity trends, it is important to understand the measures of adiposity utilised in population level epidemiological research. Field measures of adiposity including body mass index (BMI), skinfolds, and girth measures are commonly used as screening tools because of their practicality, ease of implementation, cost effectiveness, and low participant and researcher burden compared to laboratory based measures.[16]

Although the International Obesity Task Force recommend BMI as an appropriate measure to use in epidemiological studies,[17] several limitations of this method need to be considered. The most significant issue is that currently no agreed BMI cut-off thresholds exist to classify a child or adolescent as obese or overweight.

To date, different studies have used different cut-off thresholds based on different growth reference charts. This lack of consensus makes between-country comparisons difficult. Recently, however, the International Obesity task Force (ITOF) proposed international age-and sex-specific BMI cut-off thresholds based on pooled BMI data from six countries.[17] Such definitions help develop international applicability and therefore ensure comparability of obesity rates between countries, however, they are still arbitrary and do not account for ethnic groups not considered in the population sample.

Ethnicity is an important factor when considering BMI definitions of overweight and obesity because the same BMI value does not correspond to the same percent body fat (%BF) across different population groups.[18, 19] Differences in the %BF–BMI relationship may exist due to differences in body build variables[20] such as slenderness, muscularity, and trunk-to-leg-length ratio.[19]

Recent New Zealand research indicates at the same BMI value, female children (aged 5–14 years) of Pacific Island and Maori decent have a lower percent fat mass compared to their New Zealand European peers.[21] In another study using a larger sample size, however, no clinically significant difference in the relationship between BMI and body composition was found between young children (5–10.9 years) of Maori, Pacific Island, or European descent.[22] Thus, further research clarifying the BMI - %BF relationship according to ethnicity among the New Zealand youth population is warranted.

Because of New Zealand's ethnically diverse population some New Zealand researchers advocate the development ethnic specific BMI cut-off thresholds. There are, however, several difficulties that arise when applying ethnic specific cut-off points, especially in a country like New Zealand where ethnic intermarriage is increasing, and with each generation reporting concurrent increases in proportion of children with a mixed ethnic background.[23]

First, ethnicity is based on self-identity and not necessarily a genetic link. Second, in population level research identifying an individuals' ethnicity by means other than self-identity is difficult and not necessarily practical. As body build may account for a large proportion of the variation in the BMI–%BF relationship,[19] adjusting BMI cut-offs according to frame size (rather than ethnicity) may provide a more accurate criterion to base BMI cut-offs upon.[24] Applying frame size based cut-off points, however, may be practically applied within a clinical setting, but not necessarily

within population level research due to the measures required to ascertain frame size (i.e. ankle and wrist girths).

Three national surveys have examined New Zealand youth obesity levels. The 1989 Life in New Zealand Survey,[25] and the 1997 National Nutrition Survey[2] examined obesity prevalence levels in youth aged 15 to 18 years old. Recently, the 2002 Child Nutrition Survey was conducted and provided a snapshot of the nutritional status (including BMI data) of children aged between 5 and 14 years old. No single survey has examined overweight and obesity among all adolescents.[1]

Based on New Zealand ethnic-specific BMI cut off points, the National Nutrition Survey found 27% of 15 to 18 year olds were considered overweight or obese (see Table 1). Between 1989 and 1997 obesity levels rose from 3% to 12.6% in males, and from 2% to 5.3% in females. Therefore, over an 8-year period, obesity levels increased by 300% for males, and a 160% increase for females.[2, 25] This comparison may under represent the true increase in overweight and obesity because the 1989 LINZ Survey used a lower BMI cut-off value ($30 \text{ kg/m}^2$) to define obesity among individuals of Maori and Pacific Island descent, compared to the 1997 NNS ($32 \text{ kg/m}^2$). Additionally, the New Zealand Child Nutrition Survey found 23% of children aged between 11 and 14 years old were overweight and a further 10.6% were obese.[1]

Both the National Nutrition Survey and the Child Nutrition Survey found that a disproportionate number of Maori and Pacific peoples were considered overweight and obese compared to New Zealand European and Other children.[1, 2] This may in part be due to the universal BMI definition used to define an individual as overweight or obese. This universal definition does not take into account body composition differences noticed among different ethnic groups within New Zealand. For example, Maori and Pacific Island females, on average, have a higher proportion of lean muscle mass at a similar BMI than New Zealand European females.[26]

The prevalence of youth overweight and obesity in New Zealand is similar to other countries. Results from countries including Australia,[27] the United States,[28] Great Britain,[29] United Kingdom,[30] and Brazil,[31] indicate that between 11 and 44% of youth are considered overweight or obese. In contrast, lower prevalence levels (around 7%) have been documented in Russia,[31] China,[31] and Finland.[32]

In addition to the high levels noticed in many countries, studies indicate the prevalence levels are on the increase,[29,33–36] and that the greatest changes have occurred at the higher BMI values.[37–39] Also, similar to Maori and Pacific Island populations in New Zealand, specific ethnic minority groups including American Indians,[40] Mexicans,[41] and Hispanic and African Americans[42] are at an increased risk of being overweight or obese.

The global picture of youth obesity is clear. Obesity is a growing issue and is a health threat to the adolescent population and the future adult population. New Zealand is no exception, with high prevalence levels compared to some westernised countries.

## Table 1. Adolescent obesity levels

| Place | Survey | Age (years) / no. of children | Date of survey | % OW + OB |
|---|---|---|---|---|
| Brazil [*† 31,43] | Nationally representative surveys | 10-18 / not stated | 1975 | 3.7 |
| | National Research of Health and Nutrition | 10-19 / 13,715 | 1989 | 7.7 |
| | Nationally representative surveys | 10-18 / not stated | 1997 | 12.6 |
| China [* 31] | China Health and Nutrition Surveys | 10-18 / not stated | 1991 | 4.5 |
| | | 10-18 / not stated | 1997 | 6.2 |
| East Germany [* 36] | Not stated | 11-14 / 798 | 1992/93 | 8.92 |
| | | 11-14 / 957 | 1995/96 | 12.82 |
| | | 11-14 / 950 | 1998/99 | 15.85 |
| Finland [* 32] | Adolescent Health and Lifestyle Survey | 12,14,16,18 / 2832 | 1977 | 3.17 |
| | | 12,14,16,18 / 8219 | 1999 | 7.64 |
| Great Britain [‡ 29] | British Standards Institute Survey | 11-16 / 3784 | 1977/87 | 6.8 |
| | National Diet and Nutrition Survey | 11-16 / 776 | 1997 | 18.95 |
| New Zealand [25] | Life In New Zealand Survey | 15-18 / 676 | 1989 | 5[††] |
| New Zealand [§ 2] | National Nutrition Survey | 15-18 / not stated | 1997 | 27 |
| New Zealand [* 1] | Child Nutrition Survey | 11-14 /1119 | 2003 | 33.6 |
| Russia [* 31] | Russian Longitudinal Monitoring Survey | 10-18 / not stated | 1992 | 11.5 |
| | | 10-18 / not stated | 1998 | 8.5 |
| UK [‖ 30] | The Health Survey for England | 13-15 / 756 | 1996 | 44.3 |
| United States [¶33] | NHES | 12-17 / 6710 | 1963-1970 | 15.15 |
| | NHANES I | 12-17 / 1911 | 1971-1974 | 17.3 |
| | NHANES II | 12-17 / 1970 | 1976-1980 | 15.9 |
| | NHANES III | 12-17 / 1103 | 1988-1994 | 21.45 |
| United States [** 42,44] | Youth Risk Behaviour Survey | 13-16 / 13,601 | 2001 | 24 |
| | | 13-16 / 15,214 | 2003 | 28.9 |

OW=overweight; OB=Obese;*Defined overweight and obesity as a BMI $\geq$ 85th centile for BMI by age and sex based on reference data from international reference data [17]; †The OW+O was defined from a body mass index (BMI) equal or superior to the 85th percentile of the reference population of the NCHS; ‡Defined overweight as a BMI value 91st percentile and obesity as a BMI value 98th percentile on the 1990 BMI index reference curves for UK. §Defined overweight and obesity as a BMI$\geq$25kg/m$^2$ for New Zealand Europeans, and a BMI$\geq$26 for Maori and Pacific Island peoples; ‖Defined at risk of overweight and overweight as a BMI$\geq$85th percentile for BMI based on UK reference data; ¶Defined overweight and obesity as a BMI$\geq$85th percentile for BMI by age and sex based on reference data from National Health Examination Survey (NHES) II & III; **Defined at risk of overweight and overweight as a BMI$\geq$85th percentile for BMI by age and sex based on reference data from CDC growth charts; ††Obesity data only.

## Physical activity

Physical activity is an important lifestyle behaviour that impacts positively on both the prevention and management of obesity. Before physical activity prevalence data are examined, it is important to understand the measures and definitions used in large-population physical activity research. A number of objective, and subjective methods exist to measure physical activity. Population research has relied heavily on subjective measures of physical activity such as questionnaires, (e.g. self report, proxy reports, interviews). Although questionnaires are cost effective, easy to implement, and have a low researcher and participant burden, issues of recall bias, social desirability, and deliberate misrepresentation may make interpretation difficult. Also, proxy reports which are often used, provide limited validity when measuring subjective matters such as physical activity.[45]

A limitation of adolescent physical activity research is that currently no standardised physical activity recommendations exist for the adolescent population. The lack of such a recommendation impacts on the ability to define an individual as active versus inactive. Although physical activity guidelines have been proposed,[46] several studies have examined different durations (e.g. 30 minutes per day, 60 minutes per day, 150 minutes per week), intensities (e.g. moderate, vigorous), frequencies (e.g. five days per week, every day), and type (e.g. incidental, transportation, school related, sport) of physical activity. This increases the difficulty in making between-country comparisons.

In New Zealand, physical activity data relating to age, sex, and ethnicity of adolescents have been collected through several surveys, including the New Zealand Health Survey,[47] the Youth 2000 Survey,[48] National Children's Nutrition Survey,[1] and the main physical activity monitoring system, namely, the Sport and Physical Activity Surveys.[3]

Sport and Recreation New Zealand (SPARC) has been a world leader in examining New Zealand physical activity and inactivity trends by implementing the Sport and Physical Activity Survey (SPAS), a comprehensive physical activity surveillance system. To date, SPARC has carried out the SPAS at three time points, 1997/98, 1998/99, and 2000/01.[3] The combined results of the three SPAS's indicate that 37% of New Zealand adolescents aged 13 to 17 years are physically inactive (i.e. they did not participate in a minimum of 150 minutes of physical activity per week). Also, compared to Maori and Europeans, Pacific peoples, and people from other ethnic groups were considered least active.[3]

By comparison, young people in New Zealand appear relatively active to their peers in countries such as Canada,[49] and England.[50] In contrast, only 31% of adolescents (Grades 9 through 12) in the United States were classified as 'insufficiently active' in 2001[51] which is similar to levels noticed in New Zealand. In Table 2, adolescent physical activity statistics from various countries are presented.

Comparing physical activity data sets from different countries, however, is often difficult because of differences in measures and criterions for defining an individual as 'sufficiently' active. Adolescent data sets are no different. The New Zealand definition of being physically active for youth is less strict compared to definitions in other countries. For example, in Canada, young people are required to achieve energy expenditure =8kcal/kg body-weight per day (KKD). This equates to 1 hour of

moderate physical activity and 30 minutes of vigorous activity per day. Based on this definition, 65% of 13–17 year old Canadians are considered inactive.[49]

When countries have applied similar definitions of 'physically active' utilised by New Zealand, in fact, rates of inactivity are comparable. One such example in highlighted in the Health Survey for England. When the criterion employed changed from 60 mins of physical activity per day to 30 minutes per day, the level of inactivity decreased from 62.7% to 42.3%[50] which is similar to the level noticed among New Zealand youth.[3]

**Table 2. Adolescent physical inactivity levels**

| Place | Survey title | Age | Date of survey | Percent | Definition |
|---|---|---|---|---|---|
| Canada [49,52,53] | Physical Activity Monitor | 13-17 | 1995 | 64 | Did not achieved Energy expenditure =8kcal/kg body weight per day (KKD) |
| | | 13-17 | 1998 | 66 | |
| | | 13-17 | 1999 | 59 | |
| | | 13-17 | 2000 | 65 | |
| England [50,54] | Health Survey for England | 13-15 | 1997 | 62.7 | Did not participated in 60 minutes or more on at least 5 days |
| | | | 1997 | 42.3 | Did not participated in 30 minutes per day over the past 7days |
| | Health Survey for England | 2-15 | 2002 | 25.5 | Did not participated in 30 minutes per day over the past 7days |
| New Zealand [3] | Sport and Physical Activity Survey | 13-17 | 1997-2001 | 38 | Took part in < 2.5hr of physical activity in the previous 7 days |
| United States [44,51] | Youth Risk Behavior Survey | 13-16 | 2001 | 35 | Did not participate in sufficient vigorous physical activity |
| | | | 2003 | 37.4 | |
| | | | 2001 | 74 | Did not participate in sufficient moderate physical activity |
| | | | 2003 | 75.3 | |
| | | | 2001 | 31.2 | Did not participate in vigorous activity AND did had not participate in moderate activity |
| | | | 2003 | 33.4 | |

Despite the difficulty in making direct comparisons between countries, several trends have emerged from the SPAS that are noticed worldwide. Three trends that emerged from the SPAS are (1) physical activity levels decrease from childhood to adolescence, (2) physical activity levels decrease during adolescence, and (3) females are more physically inactive than males (45% and 33% respectively).

Similar trends have been found for youth in England,[50] Australia,[55] and Canada.[49,52,56] Data pertaining to the United States shows that participation in either moderate or vigorous activity decreases with increasing age,[51] physical activity patterns generally decline most from ages 15 through 18, and then continue to decline from 18 to 29 years of age.[57] As well, males compared to females, and Whites compared to their Hispanic and Black counterparts, participate in significantly higher levels of moderate and vigorous physical activity.[51] For example, in a recent Canadian survey, physical activity levels were higher among children aged 5 to 12 years compared to youth aged 13 to17 years. [49]

The main advantage of the current SPARC physical activity surveillance system is that regular monitoring has taken place. Four limitations, however, exist which impact on the efficacy of this survey. First, the existing surveillance system has been based largely on parental proxy report. Proxy reports require an adult (aged 18 years or older; living in the same house as the child) to report on the child's habitual activity levels. Proxy reports have little correlation with more direct measures of physical activity.[45] Second, the Sport and Physical Activity survey has not been validated for the population under study. As well, the previous surveys carried out by SPARC have focused predominantly on sport and exercise rather than physical activity *per se*. Finally, the quantity of physical activity required to define a youth as physically active in the New Zealand survey is substantially lower compared to other countries and is not in line with proposed physical activity guidelines for children and adolescents.[46]

Hence, the positive picture of New Zealand youth physical activity levels may largely be an artefact of the existing surveillance system, and, therefore helps explain the considerably lower prevalence levels of inactivity in New Zealand youth compared to youth in other countries. By building upon and overcoming these methodological flaws of the existing surveys, a more accurate and reliable picture of the health-related physical activity patterns of New Zealand youth will emerge.

Although the main focus of this paper is on obesity and physical inactivity, diet is also a key determinant of obesity. Despite epidemiological evidence indicating a stability,[58] and in same instances a decrease in energy intake,[59] such research also provides insight into the changes in eating patterns that have occurred simultaneously with the increasing rates of youth obesity. Specifically, there has been a shift towards consuming more energy intake from restaurants and fast food places compared to home sources of food,[60] an increase in energy dense foods including pizza, cheeseburgers, and salty snacks,[60] as well as a greater proportion of energy consumed from sugar added beverages.[59]

When examining the link between dietary behaviours and obesity, recent research suggests particular eating patterns and sources of dietary intake are important factors associated with youth obesity. United States data indicate overweight youth consume a greater proportion of total energy intake from soft drink consumption compared to

their non-overweight peers.[58] Skipping breakfast has also been associated with overweight cross sectionally[61], and with an increase in BMI among normal weight youth over time. [61] Furthermore, a greater frequency of eating food purchased away from home was positively associated with change in BMI z-score during adolescence. [62]

In terms of New Zealand specific data, the New Zealand Child Nutrition Survey provided an extensive examination of nutrition intake and dietary behaviours among New Zealand children aged 5 to 14 years old. Analysis of such data in relation to BMI are not yet published.

A difficulty in assessing the importance of lifestyle behaviours in the aetiology of obesity is that obesity occurs over time while many measures of lifestyle habits occur once the obese state has been reached. For instance, a recent study found that (over a 1-year period) skipping breakfast was associated with a decrease in BMI among overweight children, while normal weight peers who skipped breakfast gained weight over the same time period.[61]

Thus to provide an accurate assessment of factors associated with youth obesity in New Zealand, surveillance of diet must occur concurrently with that of obesity and physical activity.

## Where to now for New Zealand?

Youth obesity and inactivity are two growing health problems worldwide. Because obesity and physical inactivity have been identified in the top-four health priorities for New Zealand,[15] as a country we need to monitor and characterise these risk factors in terms of prevalence, distribution, and secular trends. For this to occur, an enhancement of existing national surveys is required. Such surveillance is essential to identify at-risk groups, inform public health policy, develop appropriate and effective prevention and management initiatives, and track progress toward national health priorities.

In terms of youth obesity, the recent Child Nutrition Survey[1] has overcome the paucity of data pertaining to obesity levels of young people aged 14 years and younger. Future research needs to ensure (1) regular monitoring of obesity levels among adolescents and adults, and (2) development of age, sex, and ethnic specific BMI cut-off thresholds to define overweight or obese.

In relation to physical activity, improving the current surveillance system would involve the validation of a youth physical activity questionnaire, establishing physical activity recommendations for youth based on international best practice, and incorporating a more holistic view of physical activity that includes health-related activity accumulated in activities such as active transportation and part-time work. Overcoming these issues will result in an enhanced physical activity monitoring system capable of providing an accurate and holistic assessment of youth physical activity levels.

Finally, attention needs to be directed towards identifying correlates of obesity and inactivity among different cultures and socioeconomic groups. Literature indicates numerous biological, behavioural, and environmental factors are linked to the development and maintenance of obesity. A systematic review [63] found that risk factors for childhood obesity included parental fatness, social factors, birth weight,

timing of maturation, physical activity, dietary factors, and other behavioural and psychological factors. In terms of behavioural factors, time spent watching television,[4, 64–66] and physical activity (inverse),[67–69] eating lunch regularly[70] breakfast skipping,[71] and quick service food purchases[62] have all been associated with obesity status.

In New Zealand, however, no published research has examined such factors among New Zealand youth according to gender and ethnicity. Future research examining the factors associated with obesity and physical activity among New Zealand youth will provide a foundation of knowledge to inform the development of multisectoral, multisetting, and sustainable health promotion initiatives aimed at reducing the prevalence of obesity and inactivity among New Zealand youth.

But first we must have a real picture of the status of our adolescents in terms of both overweight/obesity and physical inactivity—only then will we have a basis on which to build effective interventions, and monitor the success of such interventions.

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## Appendicostomy

*This is an excerpt from an editorial that was published in the New Zealand Medical Journal 1905, Volume 4 (16), p222–227.*

In a recent issue we made a note of the operation of flushing the colon through a catheter passed into the incised appendix. That proceeding we attributed to a well-known surgeon, but subsequent inquiries point to the fact that the original suggestion was made by Mr. C. B. Keetley, who first proposed appendicostomy as a substitute for caecal colotomy in a debate at the Medical Society of London, 12th November, 1894. It was soon afterwards performed in the United States by Weir.

Appendicostomy has been performed by Mr. Keetley for severe and intractable constipation. He passed a pint of warm water with some saline aperient through the appendix into the colon, a painless and simple procedure that insures a free motion. Nothing escapes through the incision, as the muscular coat of the appendix probably renders that structure a more or less perfect valve. The same surgeon has also performed appendicostomy after reducing an ileocaecal intussusception by operation. In that case there were three objects in view—namely, in the first place, to anchor the caecum and thus to prevent recurrence; secondly, to favour the recovery of a much bruised and inflamed colon by means of lavage through the appendix; and, thirdly, to prevent shock by injecting suitable remedies.

The ingenuity and success of this excellent piece of conservative abdominal surgery reflects a good deal of credit on British surgery.—*The Medical Press*.

## Proceedings of the Waikato Clinical School Research Seminar, Thursday 16 September 2004

**Effect of cool temperature dialysate on the quality and patients' perception of haemodialysis. A Ayoub, Waikato Hospital, Hamilton.**

**Background:** The effects of cool dialysate on the urea reduction ratio (URR) in high efficiency haemodialysis have not been completely studied. After reviewing the literature, it appeared that patients' perceptions of cool dialysis have not been studied. Since patients' perception have an impact on patient satisfaction, this motivated the authors to research this area of practice.

**Methods:** This study was designed to determine whether a high URR and haemodynamic stability could be achieved by using cool dialysate in two groups of patients. The first group of five patients were known to have hypotension episodes during dialysis, and the second group of five patients were documented as having stable blood pressure (BP) during and after dialysis, after excluding vascular access recirculation and any other problems. Each patient was dialysed for three sessions using cool dialysate (35°C) followed by another three sessions using a standard dialysate temperature (36.5°C). All other dialysis session parameters were maintained.

**Results:** The results show that the dialysate cooling resulted in an increased ultrafiltration in the low BP group (P=0.05). Cool dialysis had neither an adverse nor a beneficial effect on urea removal in the two groups (P=NS). The mean arterial pressure post- and intra-dialysis was significantly higher in dialysis with cool dialysate in the low BP group (P<0.01 and P<0.007, respectively). The mean arterial pressure in the stable BP group remained unchanged when cool dialysate was used (P=NS). The intra-dialytic pulse rates in the low and stable BP groups were similar. A total of seven episodes of symptomatic hypotension were observed in the low BP group, but none in the stable BP group (P<0.0001). Patients' perceptions about cool dialysate were measured by a questionnaire which showed that 80% of them felt more energetic after dialysis and requested to be always dialysed with cool dialysate.

**Conclusion:** Cool dialysate improves tolerance for dialysis in hypotensive patients and helps increase ultrafiltration while maintaining haemodynamic stability during and after dialysis. Patients' perceptions were positive, as most of the selected sample felt more energetic and generally well during and after dialysis, and this had a positive impact on their activities of daily living.

**Melanoma incidence in greater Tauranga, North Island New Zealand: Preliminary findings of an epidemiologic study. W Chan, Waikato Hospital, Hamilton.**

**Introduction:** A marked increase in the number of patients presenting with primary cutaneous melanoma to a private Dermatology practice in Tauranga, New Zealand resulted in the undertaking of this study. The primary objective was to ascertain the

incidence of melanoma in this area and to compare the findings with those of other regions, both within New Zealand and other countries.

**Method:** Data were obtained from retrospective review of histology reports from a pathology laboratory servicing the public and private health systems in greater Tauranga (Tauranga and Western Bay of Plenty Districts). Primary cutaneous melanoma (including melanoma in situ) reported from January 1st to December 31st 2002 were included. The denominator population consisted of non Maori in this area identified from the 2001 New Zealand Census.

**Results:** Preliminary data show the age standardised incidence of melanoma in the greater Tauranga area was approximately 79/100 000. When in situ melanoma was included, the total incidence increased to 157/100 000.

**Discussion:** Tauranga, New Zealand has a high incidence of cutaneous melanoma. Clinical, demographic and histological details were also analysed in this study and will be discussed. Factors that may account for this high incidence of melanoma will also be considered.

**Examining the Role of Stigma-Tolerance, Self-Concept and Pathology in Adolescent Help-Seeking and Service Utilisation. J Clarkson, J Fitzgerald, G Dolan, M Hsieh, I Evans, M Connelly. The Psychology Centre, Hamilton.**

Factors that may impact on the decisions of young New Zealand people to access specialist mental health services were examined. A community sample of approximately 200 non-referred adolescents and current service users undertook semi-structured interviews and/or completed measures focusing on current psychological functioning, service utilization, attitudes and perceptions guiding decisions related to help-seeking and barriers to help-seeking. The findings from our preliminary analysis of the data are presented, along with a consideration of the implications for service planning and development.

**Staff feelings and the process surrounding smokefree policy implementation in a major health organization: A descriptive qualitative study. J Lash[1], K Evison[2], J Henry[2], L Adams[2]. [1]AUT, Auckland; [2]Waikato Hospital, Hamilton.**

In 2003, the Waikato District Health Board (WDHB) launched their 'Go Smokefree' campaign throughout all WDHB campuses in the region. The campaign played a major part in the implementation of the smokefree policy. This led to all campuses going smokefree on January 1st, 2004.

The purpose of this study was to evaluate the feelings of staff regarding the implementation of a smokefree policy in a large organization and was part of a larger evaluation plan of the effectiveness of policy implementation. Using a combination of snowballing and purposive sampling, four focus groups were conducted to explore staff feelings. The groups consisted of Maori non-smokers (8), Maori smokers (6), non-Maori non-smokers (7) and non-Maori smokers (11). Qualitative data analysis and methodology was used, particularly content analysis to elicit common themes.

The common feelings that emerged from all of the groups were confusion surrounding the process of implementation. All groups felt disempowered although little

opposition was expressed to the WDHB going smokefree. The two non-smoking groups expressed happiness at perceived improvements in health while expressing empathy for their smoking counterparts. The smoking groups however, felt targeted and isolated.

Recommendations were that in future more consultation and submissions be undertaken with a diverse group of staff to enable staff ownership in the development of policy and greater knowledge of implementation.

**Implementing significant event management in general practice; potential barriers and solutions. S Lillis[1], H Gaddes[2]. [1]Waikato Clinical School, University of Auckland, Hamilton; [2]Pinnacle.**

Significant event management (SEM) offers general practice teams a process for reviewing significant events in a way that focuses on how and why defences against error can fail rather than the historical approach of blaming individuals for error. A series of focus groups were held with general practice teams in the Waikato with the purpose of understanding the barriers to implementing significant event reporting systems in general practice.

Factors that hinder and promote effective SEM related to the practice environment, the SEM process and external factors. Relating to the practice environment are barriers such as those created by a hierarchical structure, time constraints and financial costs. Effective leadership and a horizontal structure may assist practices to break down these barriers to promote better organisation learning and patient care through SEM.

The SEM process must be adaptable to meet the individual needs of practices. This may help avoid problems such as practitioners forgetting to follow through the process and promote confidence in the SEM process.

External barriers include fear of litigation and negative publicity as well as a perceived inability to effect change when a third party such as a hospital is involved. An umbrella organisation, such as a PHO, may be in a position to assist in the successful implementation of SEM by providing methods and motivation for practices and co-ordinate sharing of important SEM information between practices. Despite a number of barriers recognised by practices, there was strong support for the process of significant event management.

**Mechano-Growth Factor (MGF), but not mature IGF-IEa, reduces the severity of myocardial infarction. C D McMahon*[1], G P Devlin[2], K G Matthews[1], J Jensen[2], S P Stuart[1], P H Goldspink[4], S Y Yang[3], S K S Srai[3], B Ramesh[3], J V Conaglen[2], J J Bass[1] and G Goldspink[3]. [1]AgResearch Ltd., Hamilton, NZ; [2]Waikato Clinical School, University of Auckland, Hamilton, NZ; [3]Royal Free and University College Medical School, London, UK; [4]University of Illinois (Chicago), USA.**

We sought to determine whether MGF, a splice-variant of IGF-I containing a novel E domain, could reduce the extent of ischaemic damage following myocardial infarction (MI).

MI was induced by occluding the left circumflex coronary artery of sheep. In the first experiment, ewes received one of four protein treatments (n=6 per group) delivered into the circumflex artery: vehicle (saline), 200 nM mature IGF-I, 200 nM MGF E domain, or 200 nM of full MGF (domains B, C, A and D of mature IGF-I plus the E domain of MGF). In the second experiment, 18 ewes were randomised to receive either empty vector (control), IGF-IEa, or MGF plasmid DNA into the occluded artery (n=6 per group). Left ventricular function was assessed with echocardiography before MI (baseline), and at days 1, 2 and 6 post-MI.

Cardiac ejection fraction was reduced by 40% (P<0.001) at d 1 in all sheep, but was increased by 4% at d 6 only in sheep treated with MGF peptide only (E or full peptide; at least P<0.05). Sheep were killed on d 8 and the coronary artery perfused with 0.15% Evans blue dye to distinguish the area 'at-risk' from the viable and necrotic areas. Hearts were sectioned transversely into 1 cm slices, digitally photographed, and the respective areas assessed. In the first experiment, only MGF (E domain and full MGF) reduced the area 'at-risk' (58%, P<0.01) and increased the viable area (3.2 fold, P<0.01) compared with controls. Similar changes were observed in the second experiment, but of lower magnitude suggesting that early presence of the protein is essential.

These data suggest that acute administration of protein is more effective than gene therapy at reducing the area 'at-risk' and furthermore that the E domain of MGF, but not mature IGF-I or the Ea domain, may prevent expansion of the infarct and remodelling of the myocardium.


**Health Waikato Teledermatology Trials Phase 6. A Oakley, J Bennett, S Holmes. Waikato Hospital, Hamilton.**

The incidence of melanoma and non-melanoma skin cancer in New Zealand is among the highest in the world. Patients referred to specialist dermatologists often wait months to be seen for diagnosis and management. Teledermatological triage may allow us to offer a better service. A study was designed to find out if there was any advantage to adding images of the skin lesion to the standard referral.

Referral data and images of 109 lesions from 73 unselected patients attending lesion diagnosis clinics were included in the study. Thirty-eight dermatologists (the 'teledermatologists') were presented with online referral text and images for 5 skin lesions, images alone for 5 lesions and text alone for 5 lesions. Diagnoses, management plans and referral priority made online using non-standardised equipment were compared with those made histologically and/or face to face by a specialist dermatologist or plastic surgeon at routine clinics.

The referring GPs made the same diagnosis as the face-to-face specialist for 31% of lesions. The teledermatologists made the same diagnosis for 56% lesions given images but only 42% with text alone, and were much less confident about the diagnosis than the face-to-face specialists, especially when provided with text alone. The concordance of diagnosis ranged from 20 to 100% of lesions and was more consistent in more experienced dermatologists using high quality computer equipment.

In conclusion, high quality images help experienced dermatologists in the triage of patients with skin lesions but face-to-face consultations may be necessary for the majority of them.

### Changing Pattern of Gastroschisis. S Heap, U Samarakkody, S Brown, A Kukkady. Dept of Paediatric Surgery, Waikato Hospital, Hamilton.

**Introduction:** There is a global increase of the incidence of gastroschisis. We have looked at the incidence of gastroschisis and other associated demographics of the patients treated in a tertiary paediatric surgical centre.

**Aim:** To document the recent incidence of gastroschisis in the mid north island of New Zealand and compare it with the national figures.

**Method:** All patients born with Gastroschisis treated in the unit during a period of seven years are analysed. The case notes of patients at Waikato hospital and referring hospitals are reviewed. The incidence, ethnicity, maternal age, associated abnormalities and the method of delivery are analysed. The indications for Caesarean section and the effect it has on the outcome are critically reviewed. The surgical procedure and postoperative complications are reported. The results are compared with the national statistics obtained through Statistics New Zealand.

**Results:** There is a gradual increase in the incidence of gastroschisis. The Maori ethnicity is disproportionately represented. The maternal age is lower than the national average. The gestational age and birth weight are lower than average. Indications for Caesarean section seem to be not objective.

**Conclusion:** The incidence of Gastroschisis is increasing in the Mid north island of New Zealand.

### Complicated intestinal atresias need individualised approach. R Sakalkale, U Samarakkody, S Brown, A Kukkady. Dept of Paediatric Surgery, Waikato Hospital, Hamilton.

**Aims:** Retrospective study of complicated intestinal atresias.

**Methods:** We studied retrospectively, two groups of complicated and uncomplicated jejuno-ileal intestinal atresias respectively, presenting to us in the last five years. The study parameters were birth weight, gestational age, maternal age, antenatal factors, chromosomal and associated systemic abnormalities, surgical diagnosis and management.

**Results:** A total of 13 neonates presented to us with jejuno-ileal atresias in the last five years. Nine were males and four females. Six patients (5 male, 1 female) had complicated intestinal atresias associated with antenatal perforation (2), gastroschisis (1^ + 1*), patent vitello-intestinal duct* (1), posterior urethral valves (1), congenital short bowel^ and meconium ileus/CF (1). There was no difference in these two groups regarding birth weight, maternal age and antenatal factors. In the complicated atresia group all except one, who had a definitive anastamosis, had stomas fashioned

initially followed by definitive closure several weeks later. There was one death in this group, namely the child with short bowel syndrome, at 9 months of age due to combined liver and bowel failure.

**Conclusions:**

**1.** Current embryological concepts fall short of explaining some of these complicated atresia associations

**2.** Almost all patients in the complicated atresia group had more morbidity, prolonged hospital stay and multiple procedures.

**Learning Outcomes for Medical Student Radiology Teaching. R M Subramaniam[12] , J Sherriff[1] , K Holmes[1] , M C Chan[1]. [1] Department of Radiology, Waikato Hospital, Hamilton; [2]Department of Radiology, Waikato Clinical School, University of Auckland, Hamilton.**

**Objectives:** To establish a set of learning outcomes for medical student radiology teaching from non-radiology clinicians perspective.

**Methods:** A single tertiary centre study was conducted and a questionnaire was sent to consultant clinicians in all specialties, except radiology. Each learning outcome was graded on a scale of 1 (very strongly disagree) to 6 (very strongly agree). Participants also graded a list of 14 common radiological investigations in terms of the importance for students to observe during their training. Opportunity was given for clinicians to put forward any suggestions other than those presented on the questionnaire and grade them.

**Results:** 45 out of 90 questionnaires were returned. All educational learning outcomes scored above an average of 4 (agree). The five highest ranking learning outcomes in order of importance were: viewing chest films, viewing abdominal films, viewing bone and joint films, distinguishing normal structures from abnormal and identifying gross bone or joint abnormalities. Clinicians agreed for observing computed tomography (CT) and possibly for Magnetic Resonance Imaging (MRI), abdominal ultrasound (US) and chest films during medical student radiology training.

**Conclusion:** A set of learning outcomes for medical student radiology teaching was established from a clinicians' perspective.

## Proceedings of the 175th meeting of the Otago Medical School Research Society, 11 November 2004

**Investigative studies of dysgalacticin, a novel antimicrobial protein produced by _Streptococcus dysgalactiae_. H Baird, N Heng, J Tagg, R Jack. Department of Microbiology and Immunology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Dysgalacticin is a novel 21 kDa, heat-labile, acid-stable, anionic antimicrobial protein produced by the gram-positive bacterium _Streptococcus dysgalactiae_ subsp. _equisimilis_. The inhibitory spectrum of dysgalacticin includes predominantly streptococci belonging to Lancefield serogroups A, C, and G. These three serogroups contain essentially all the major streptococcal pathogens of humans. Dysgalacticin is of interest because of its ability to kill the human pathogen _Streptococcus pyogenes_ by an unknown mechanism that does not involve cell lysis. Studies on how dysgalacticin exerts its inhibitory activity may provide insight into novel methods for the control of _S. pyogenes_ infections. The aim of this study was to determine the structural features of dysgalacticin essential for its inhibitory activity.

Analysis of the primary amino acid sequence of dysgalacticin revealed a putative disulphide bond between Cys132 and Cys186. To ascertain if this disulphide bond is critical for activity, recombinant dysgalacticin was expressed in _Escherichia coli_. The inhibitory spectrum of recombinant dysgalacticin was indistinguishable from the native protein but its biological activity was lost following reductive alkylation using 2-mercaptoethanol and 4-vinylpyridine. In addition, an engineered dysgalacticin variant (Cys186Ala-dysgalacticin), unable to form a disulphide bond, was also expressed and found to be biologically inactive. Both these independent findings confirm that a disulphide bond is indeed present and essential for biological activity of dysgalacticin.

In conclusion, this study has provided essential information on the structure-function relationship of dysgalacticin. Future studies will focus on elucidating the mode of action of dysgalacticin and will involve (i) determining the effects of dysgalacticin on sensitive _S. pyogenes_ cells as well as (ii) identifying the dysgalacticin target (receptor) using transposon mutagenesis.

**Proliferation and apoptosis in the ovarian surface epithelium, cyst epithelium and rete ovarii of mice of different ages and total lifetime ovulation number. C Beaugié, H J McQuillan, J S Fleming. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.**

The risk of developing epithelial ovarian cancer correlates with age and total number of ovulations experienced in a lifetime. Over 90% of ovarian cancers are epithelial and thought to be derived from invagination of ovarian surface epithelium (OSE) within the stroma to form inclusion cysts. Incessant ovulation increases inclusion cyst

numbers in mice, as well as increasing the number of cysts derived from rete ovarii (RO) tubules. The present study investigated how proliferation and apoptosis rates within these ovarian epithelial compartments change with age and total lifetime ovulation number.

Incessant ovulation was induced in Swiss Webster mice (n = 10/group) by housing them in screen-divided cages alongside a male, until 3, 6, 9, and 12 months of age. Mice housed away from males, with a lower ovulation number, were used as age-matched controls, as were 6- and 9-month breeding animals. All animals were injected with 3 x 30 μg of bromodeoxyuridine (BrdU) per g body weight i.p. at 2 hour intervals and ovaries collected 2 hours later. Incorporated BrdU was determined by immunohistochemistry, with diaminobenzidine (DAB) visualisation. Apoptosis was determined by active caspase-3 immunohistochemistry with DAB visualisation and with TdT-mediated dUTP nick end labelling. A significant decline in proliferation with age was observed in OSE, but not cysts (negative binomial regression model; $p < 0.0001$). OSE proliferation rates were 1.16, 1.27, 0.45 and 0.33% for the 3, 6, 9 and 12 month-old animals respectively. Proliferation was highest in cysts (1.42%) and lowest in RO (0.20%). Incessantly ovulated ovaries showed significantly more proliferation than those with lower ovulation number ($p < 0.03$). Extremely low numbers of apoptotic cells were seen in all epithelial compartments.

We conclude apoptosis occurs infrequently in all ovarian epithelia studied. Unlike OSE cells, cyst cell proliferation does not decline significantly with age, suggesting cell cycle control is different in these cells.

**Protective effects of melatonin against secondary cardiac dysfunction to ischaemic stroke. J A Heaney, A V Tramoudanas, K K Narayan, R M A Rahman, S M Nair, J C Harrison, I A Sammut. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Secondary cardiac dysfunction to ischaemic stroke represents the primary complication in patients with a history of stroke. Inflammation and increased sympathetic activity during the initial ischaemic episode induce oxidative stress in peripheral tissues, including the cardiovascular system. Melatonin has recently been indicated to possess anti-inflammatory, anti-adrenergic and anti-oxidative properties. Furthermore, melatonin has been shown to display efficacy in attenuating pathological processes associated with cardiotoxic drugs. The potential protective effects of melatonin on myocardial function and energetics were investigated in a clinically relevant animal model of ischaemic stroke.

Male Sprague-Dawley rats (265-295 g, n = 32) underwent transient middle cerebral artery occlusion (MCAO) surgery. Melatonin (10 mg/kg, i.p., n = 8) was administered at 1-h post-ischaemic insult and then once every 24 h. At 72 h, animals were sacrificed and hearts perfused with Krebs-Henseleit buffer on a Langendorff system to assess haemodynamic function. Intact ventricular mitochondria were prepared through homogenisation and differential centrifugation. Flavin adenine dinucleotide (FAD)-linked respiratory function and mitochondrial enzyme kinetic analysis was

performed to determine bioenergetic capacity. Comparisons amongst treatment groups were performed using One-way analysis of variance, followed by Bonferroni $t$-tests.

MCAO surgery reduced sinus coronary flow ($p < 0.05$) and cardiac FAD-linked respiration ($p < 0.001$), however, ventricular function was not found to be compromised. Mitochondrial enzyme kinetic analysis also showed reduced bioenergetic capacity following MCAO surgery. Melatonin administration led to improved ventricular contractility and sinus coronary flow compared to vehicle treated animals. Melatonin treatment significantly improved FAD-linked respiration ($p < 0.001$) and led to improved mitochondrial enzyme kinetics.

These results demonstrate for the first time that, in the MCAO model used, cardiac mitochondria and sinus coronary flow are both significantly compromised. Melatonin administration resulted in the maintenance of mitochondrial activity and may oppose the pathophysiological processes resulting in secondary cardiac dysfunction to ischaemic stroke; potentially reducing cardiovascular complications.

**The human gene *PEG10* utilises backwards ribosomal frameshifting. M Jänicke, W P Tate. Department of Biochemistry, Otago School of Medical Sciences, University of Otago, Dunedin.**

Ribosomal frameshifting is a rare step in protein synthesis, in which the translating ribosome shifts its reading frame on the messenger RNA and continues synthesis of the protein in a new reading frame. Backwards (-1) frameshifting is widely used by viruses, such as HIV-1, during normal replication and is therefore a target in viral biology that can be exploited for new antiviral therapeutics. It is important to know if there are human genes expressed in adult tissue that utilise -1 frameshifting and must therefore be considered in any anti-viral therapy targeting this mechanism. The human paternally expressed gene 10 *(PEG10)* has been found to possess two overlapping reading frames with the second in a -1 reading frame. This study investigated whether *PEG10* utilises -1 frameshifting for expression in mammalian cells.

Bioinformatic analysis revealed highly conserved homologues of *PEG10* in nine other mammalian species (e.g. chimpanzee, cat, dog and mouse), with the seven nucleotide slippery sequence and secondary structure necessary for a -1 frameshift being 100% conserved. To investigate translation of *PEG10* in cell culture, the single open reading frames as well as the full-length gene were cloned into a mammalian expression vector. In addition, a control construct expressing both reading frames as a fusion protein was used to determine the size of the full-length protein. Constructs were transfected into monkey kidney (COS-7) cells. Specific antibodies against an N-terminal Flag-tag and a C-terminal His-tag revealed that two proteins are produced, a ~50 kDa protein that is the product of the first open reading frame and a ~100 kDa protein that is the product of both reading frames.

These results indicate that the human gene *PEG10* utilises a -1 frameshift mechanism for protein synthesis and expresses two proteins in mammalian cells. This is the first human gene identified that uses -1 frameshifting for its expression.

**A role for *Pax2* in polycystic kidney disease. C Li[1], C Stayner[1], A Jeffs[1], P Goodyer[2], J Zhou[3], M R Eccles[1]. [1]Department of Pathology, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand; [2]Pediatric Nephrology, Montreal Children's Hospital, QC, Canada; [3]Brigham and Women's Hospital, Harvard Medical School, Boston, USA.**

Autosomal dominant polycystic kidney disease (ADPKD) is characterized by cystic growths in the kidneys, pancreas, and liver. Over 85% of ADPKD cases are caused by disruptions of *PKD1*, encoding polycystin-1. Over-expression of the transcription factor, Pax2, is also associated with kidney cyst formation. To assess a role for *Pax2* in an animal model of ADPKD, mice carrying a disrupted *Pkd1* gene were crossed with *Pax2* heterozygous mutant mice. At embryonic day 18.5 (E18.5), *Pkd1* homozygous mutant (*Pkd1[-/-]*) embryos had a kidney mass ($12.6 \pm 0.93$ mg) more than twice that of wild-type litter mates ($5.9 \pm 0.53$ mg). The presence of a *Pax2* mutation in *Pkd1[-/-]* mice resulted in a significant reduction in kidney mass ($4.2 \pm 0.61$ mg, $p < 0.001$) when compared to *Pkd1[-/-]* mice, and attenuation of cyst development. To identify genes that may participate in cystogenesis, the expression levels of eight candidate genes, in the polycystin-1 signaling pathway, including *c-Myc*, *Egf* and ***b-catenin,*** were quantitated in mRNA from wild-type and mutant E18.5 kidneys, using realtime PCR.

There was no significant change in the mRNA levels of these genes, suggesting that (1) there is no role for these genes during cystogenesis in this mouse model of ADPKD, or (2) changes in gene expression levels may occur at an earlier developmental stage, or in specific kidney structures, or (3) post-transcriptional processing may alter the function of these genes during cystogenesis. In immunohistochemical studies β-catenin was retained at the lateral membrane of cystic cells in the *Pkd1[-/-]* kidneys, but cytoplasmic β-catenin staining was lost. The cytoplasmic localization of β-catenin was restored in kidneys of *Pkd1[-/-]* mice with a *Pax2* mutation, coincident with attenuation in cyst formation.

These observations suggest that persistent *Pax2* expression in ADPKD may be linked to changes in the localization of β-catenin, which has previously been suggested to form a complex with polycystin-1.


**Efficacy of a micronutrient-fortified seasoning powder on biochemical and functional outcomes in Thai primary school children. MS Manger[1], P Winichagoon[3], T Pongcharoen[3], S Gorwachirapan[3], A Boonpraderm[3], KB Bailey[1], RA Cook[1], J McKenzie[2], Andrew Gray[2], BA Ryan[4], RS Gibson[1]. [1]Department of Human Nutrition and [2]Preventive and Social Medicine, University of Otago, New Zealand; [3]Institute of Nutrition, Mahidol University, Thailand; [4]Department of Family Relations and Applied Nutrition, College of Social and Applied Human Sciences, University of Guelph, Canada.**

Deficiencies of iron, iodine, vitamin A, and zinc co-exist in children in northeast Thailand and may contribute to impairments in growth, immune competence, and cognitive function.

The aim was to determine the efficacy of a seasoning powder fortified with iron, iodine, vitamin A and zinc served with noodles or rice consumed for school lunch on biochemical status, morbidity, cognition, and growth in rural northeast Thai children.

A double blind, randomised controlled trial of children (n = 566) aged 6-12 years recruited from ten rural schools in Ubon Ratchanthani province was done. Children were stratified by age and gender, and then randomly assigned to receive either an unfortified or a fortified seasoning powder containing iron (5 mg), iodine (50 µg), zinc (5 mg) and vitamin A (270 µg) per serve on each school day for 31 weeks. Baseline and final micronutrient status, and haemoglobinopathies were assessed from blood and urine. Symptoms of diarrhoea-related and respiratory-related disease were recorded daily. Cognitive function was assessed at follow-up by visual memory and digit span tests. Anthropometrical measurements were carried out at baseline and follow-up.

At follow-up, the odds of zinc and iodine deficiencies in the fortified group were 0.63 (95% CI 0.42, 0.95) and 0.52 (0.38, 0.71) times those in the unfortified group, respectively, and haemoglobin was 1.70 g/L (0.45, 2.96) higher. Treatment had no effect on serum retinol or growth, but resulted in a significantly lower incidence of respiratory-related symptoms [ratio 0.82 (0.70, 0.96)], and a significantly higher number of items visually recalled [difference 0.56 (0.15, 0.99)], compared with children in the unfortified group.

In conclusion, a micronutrient-fortified seasoning powder reduced the incidence of zinc and iodine deficiency, increased haemoglobin concentration, reduced morbidity, and improved cognitive function after 31 weeks. It is therefore a promising vehicle for improving the micronutrient status of NE Thai school children.

**Interleukin-6 stimulates both STAT3 and ERK1/2 phosphorylation in adrenal chromaffin cells. B Milne, S Bunn. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Chromaffin cells of the adrenal medulla function as part of the stress response by releasing catecholamines. Stress can come in many forms including infection and tissue damage. It is therefore possible that activation of the immune system could be involved in regulation of the adrenal medulla. This project investigated whether adrenal chromaffin cells respond to stimulation by an immune signal, interleukin-6 (IL-6). IL-6 is known to play a major role in the inflammatory response.

Chromaffin cell cultures from bovine adrenal medullae were incubated with or without IL-6 (3 separate cultures in each case). For immunofluorescence microscopy and for Western blotting, antibodies were employed for phospho-STAT3 and phospho-ERK1/2. These phosphoproteins belong to two independent intracellular signalling cascades that are activated by IL-6 in other cells. An antibody for tyrosine hydoxylase (TH), the rate-limiting enzyme in catecholamine production, was employed for dual-labelled immunofluorescence.

IL-6 caused time- and concentration-dependent responses in phosphorylation of the STAT3 proteins. At 1 nM, IL-6 induced a 147 ± 8% (mean ± SEM) change from basal levels, with the maximal response after 15 min. The immunofluorescence supported this by showing increased levels of phospho-STAT3 in TH-positive cells, plus evidence of translocation to the nucleus, another indicator of STAT activation. Western blotting also showed increased levels of phospho-ERK1/2, with a maximal response by 1 nM IL-6 after 5 min.

This work shows for the first time that chromaffin cells respond to stimulation by IL-6. IL-6 activation of both the STAT3 and ERK1/2 proteins may have a role in catecholamine release, by altering the activity of TH. STAT3 may induce gene transcription, potentially including the transcription of TH. ERK1/2 causes phosphorylation of TH in other cells, and may play a similar role here. Therefore, through the actions of IL-6, the immune system may have a role in the stress response.

**Cardio-mitochondrial protection by haem oxygenase products: CO and bilirubin. K K Narayan, A V Tramoundanas, P Zhu, J C Harrison, I A Sammut. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Inducible haem oxygenase (HO) has been suggested to function as an effective antioxidant system in various types of cells thus conferring protection against oxidative stress/ischaemia-reperfusion injury both *in vivo* and *in vitro*. The byproducts of HO catalysed haem degradation, biliverdin and its metabolite bilirubin, demonstrate antioxidant properties whilst carbon monoxide (CO) possesses potent vasodilatory/cytoprotective effects. Therefore, this study investigated the individual effects of bilirubin and a CO-releasing molecule, tricarbonyldichlororuthenium (TCDR) on cardiac haemodynamic and mitochondrial function in an isolated, perfused rat heart model subjected to ischaemia-reperfusion injury.

Male Lewis rats (250-300 g) were anaesthetised with diethyl ether, and sodium heparin was injected intravenously. Hearts were excised, rapidly cannulated and Langendorff perfused with Krebs-Henseleit buffer containing either vehicle or drug; TCDR (20 µM) or bilirubin (0.5 and 1 µM). Hearts were then subjected to a 30-min period of warm global ischaemia followed by 1-h reperfusion. Cardiac haemodynamic parameters including left ventricular developed pressure (LVDP), heart rate (HR) and sinus coronary flow rates were measured at various time points (pre-ischaemia, 20, 40, 60 minutes into reperfusion). Intact cardiac ventricular mitochondria were isolated and purified through homogenisation and differential centrifugation. Mitochondrial respiratory function was measured in intact organelles, using a water-jacketed Clark-type oxygen electrode.

Significant recovery in LVDP at 10 mmHg and coronary flow rates were observed for all the treatment groups in comparison to the corresponding vehicle controls (n = 10, $p < 0.05$). Mitochondrial state 3-respiration rate (measure of oxygen consumption during phosphorylation) and FAD-linked respiration (marker of oxygen consumption) were also markedly improved in all treated groups versus vehicle controls (n = 10, $p < 0.05$ as analysed by one-way ANOVA for repeated measures followed by Dunn's post hoc test).

The byproducts, TCDR and bilirubin, protect cardiac haemodynamic and mitochondrial function against ischaemia-reperfusion injury, therefore replicating the protection obtained following HO-1 upregulation.

**Oral administration of EGCG modulates CYP450 enzymes in the female BALB/c mouse. M Scandlyn, M Goodin, R Rosengren. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Epigallocatechin gallate (EGCG) is the predominant polyphenolic catechin found in a variety of foods, including green tea, chocolate and fruits. It has demonstrated chemo-preventative activity both *in vitro* and *in vivo*, however the mechanism underlying this activity has not been conclusively proven. We have previously demonstrated that EGCG (12.5 and 25 mg/kg, i.p.) can alter the activity of key cytochrome P450 (CYP450) enzymes involved in the synthesis and metabolism of estrogens in the female Swiss Webster mouse. However, 50 mg/kg i.p. EGCG induced considerable mortality and hepatotoxicity. The aim of this study was to examine the ability of EGCG, when administered orally, to modulate CYP450 activity.

Female BALB/c mice (10 mice/group) were administered EGCG (25 and 50 mg/kg) or saline (5 ml/kg) by oral gavage for 7 days. On day 8, mice were euthanased by carbon dioxide inhalation, and ovarian and hepatic microsomes were prepared. Aromatase (CYP19) catalytic activity was determined in ovarian microsomes, and CYP1A, CYP3A and CYP2E1 catalytic activities were determined in hepatic microsomes.

Hepatotoxicity testing, assessed by plasma alanine aminotransferase (ALT) activities, demonstrated that EGCG was well tolerated. Ovarian aromatase activity, determined by the release of $[^3H]$-$H_2O$ from $[^3H]$-androstenedione, was reduced by 31% after 25 mg/kg and 46% after 50 mg/kg EGCG administration ($15 \pm 2$, $10 \pm 1$, $8 \pm 1$ pmol/mg/h for saline, 25 mg/kg and 50 mg/kg, respectively). Aniline hydroxylation was used to determine CYP2E1 activity, which increased 2-fold following 50 mg/kg EGCG ($0.45 \pm 0.05$ and $0.91 \pm 0.06$ nmol/mg/min for saline and 50 mg/kg EGCG, respectively). CYP1A and CYP3A activities, however, were not altered by treatment with EGCG.

These results demonstrate that oral administration of EGCG is capable of modulating aromatase and CYP2E1 activities without accompanying toxicity. This suggests that orally administered EGCG may have a potential role as an aromatase inhibitor for the treatment of hormone-sensitive cancers.


**Is the signal produced by the arterial baroreceptors in the rabbit affected when arterial pulse pressure increases as occurs, for example, during exercise? M Turner, C Bolter. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Mean arterial pressure (MAP) is the primary signal encoded in the nerve activity sent to the brainstem from the arterial baroreceptors. During exercise MAP is regulated at a higher value than at rest (baroreflex resetting). Arterial pulse pressure (PP) also increases considerably from ~40 to ≤100 mmHg during exercise in humans. This study investigated whether an increase in PP could contribute to the resetting of MAP during exercise.

Whole nerve activity was recorded from either the aortic depressor nerve (ADN) or carotid sinus nerve (CSN) in rabbits (2.5-4.5 kg) anaesthetised with sodium

pentobarbitone. Arterial pressure was recorded from either the aortic arch or a branch of the external carotid artery. Heart rate was held constant throughout the experiment by atrial pacing. Controlled inflation and deflation of cuffs secured around the vena cava and aorta produced ramps of arterial pressure (40-130 mmHg). To increase PP a mixture of isoprenaline (5 μg/ml) and adrenaline (5 μg/ml) was infused (0.4 ml/min) intravenously. For each arterial pressure ramp, the relationship between mean nerve activity and MAP (activity-pressure curve) was fitted with a third order polynomial expression.

For both the ADN and CSN recordings, an increase in PP was associated with a right shift of the activity-pressure curve. To quantify this shift we have expressed the response in terms of change from the control values at a MAP of 90 mmHg. Data are presented as the mean ± SD. Statistical comparisons were performed using the paired *t*-test. In recordings from the ADN (n = 7), PP increased 11.9 ± 6.8 (p = 0.004) and the response curve shifted by 5.9 ± 2.2 mmHg (p = 0.0004). In recordings from the CSN (n = 6) PP increased 16.8 ± 3.5 (p < 0.0001), and the response curve shifted by 7.7 ± 4.0 mmHg (p = 0.01).

Our results indicate that, at typical values of MAP, mean baroreceptor activity decreases when pulse pressure increases. A right shift in the activity-pressure curve when pulse pressure is increased may contribute to the upward resetting of mean arterial pressure that occurs during exercise.

## Metastatic melanoma causing small bowel obstruction

Jeremy Rossaak, Angus Watson

### Patient presentation

A 40-year-old female patient presented with a 3-week history of colicky abdominal pain, abdominal distension, and constipation. Abdominal X-ray confirmed the diagnosis of a small bowel obstruction. The patient had a past history of a 1.6 mm (Clarke level IV) malignant melanoma excised from her right shoulder 16 months prior to that admission. Fourteen months later, after a staging computer tomogram (CT) showed no other metastases, the patient underwent a right axillary dissection for a single 6 cm metastatic melanoma deposit. A further 28 lymph nodes were clear of melanoma.

The patient was recently readmitted to hospital with abdominal pain. A CT scan (Figure 1) demonstrated two pulmonary metastases and a small bowel intususseption with collapsed bowel beyond the obstruction.

**Figure 1. Computed tomography scan of the chest demonstrating metastatic tumour deposits in both left and right lung bases (arrowed)**
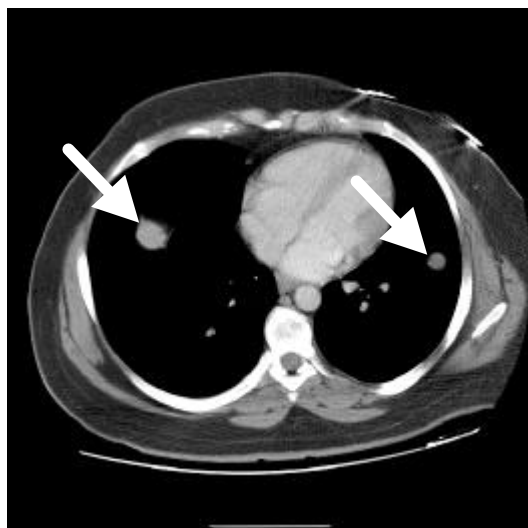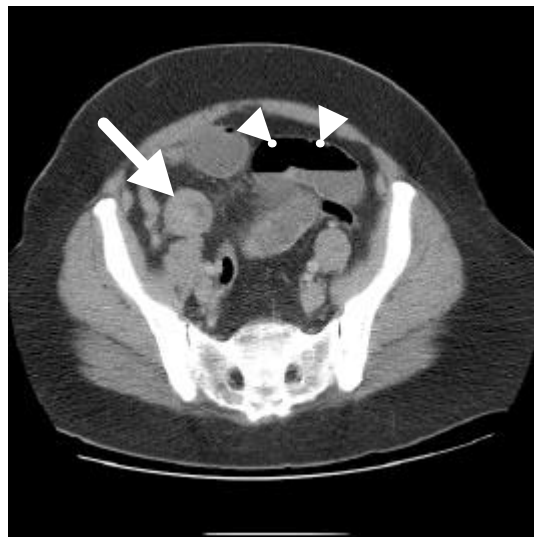
**Figure 2. Computed tomography scan of the abdomen showing the site of the intussusception (white arrow), with dilated small bowel proximally (arrowheads)**



A laparotomy was performed for small bowel obstruction presumed to be secondary to a melanoma deposit. A terminal ileum intussusception was identified (Figures 3 and 4). Histology confirmed melanoma deposit (Figure 5).

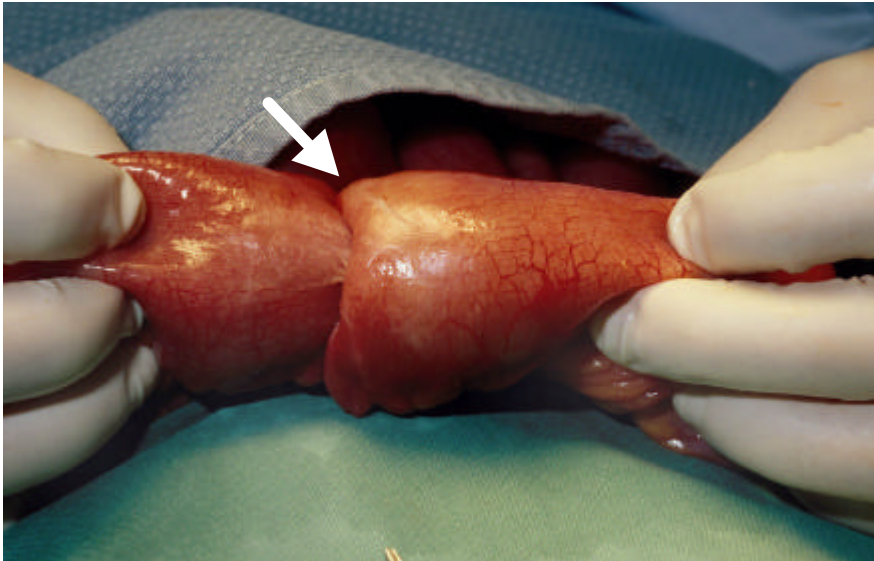**Figure 3. Operative specimen of the intussusception (arrowed)**



**Figure 4. Pathology specimen with the small bowel opened out, demonstrating the melanoma and the intussusception (arrowed). (It was surprising that the melanoma was not pigmented, however S-100 immunohistochemical staining confirmed melanoma.)**
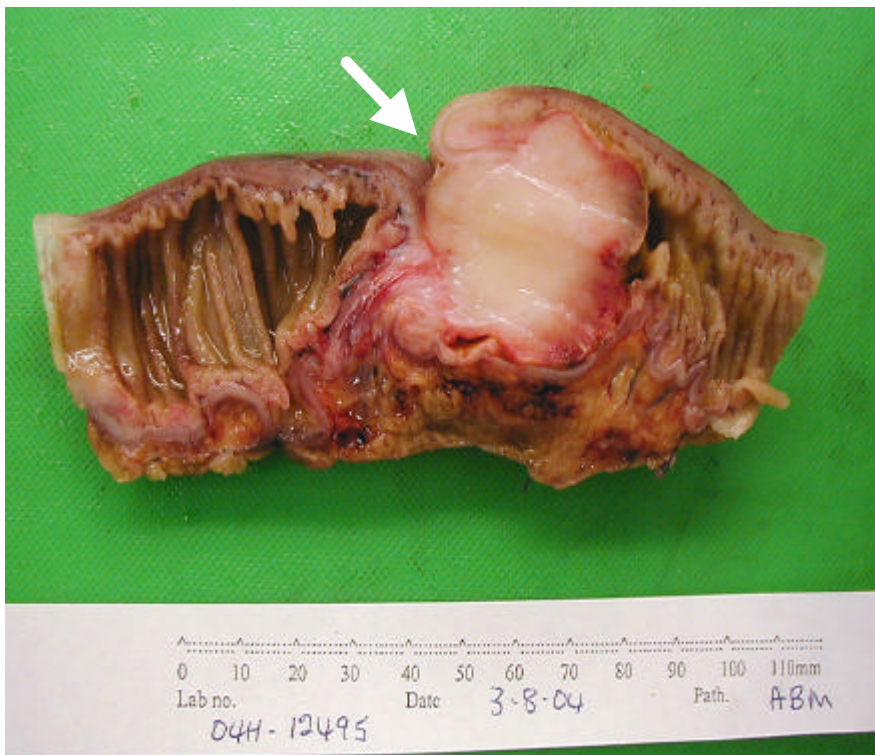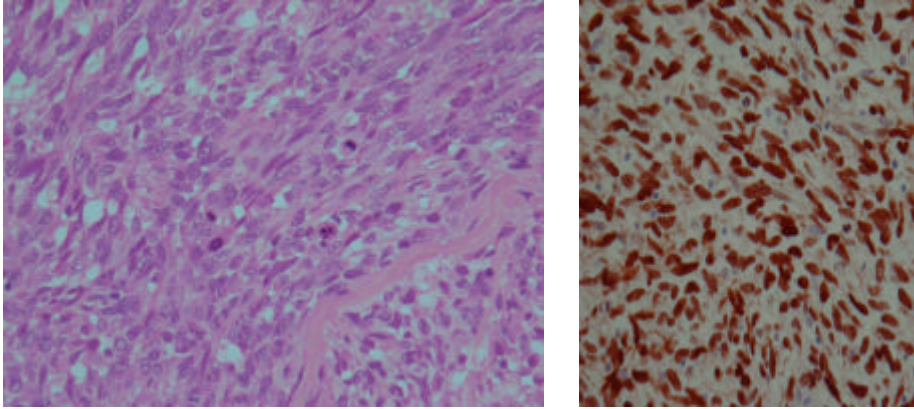
**Figure 5. Haematoxylin and eosin stained histology specimen demonstrating melanoma (left) and S-100 staining, which was strongly positive, consistent with melanoma**



## Discussion

This patient demonstrates rapid dissemination of malignant melanoma (MM), presenting with small bowel obstruction.

Systemic metastases from MM carry a poor prognosis ±6% survival at 5 years).[1] The most common sites of metastases are lung (42%), skin (18%), and brain (14%) with the gastrointestinal tract less commonly affected.[2] Chemotherapy, immunotherapy, and biologics have had limited effect on overall survival.[2] Surgical resection of isolated metastases may provide palliation and improved survival in a subgroup of patients.[2]

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## Keep getting your flu shots every year

The effectiveness of influenza vaccination has been reported to decrease in high-risk persons. Annual influenza revaccination has been proposed as a strategy to increase vaccination effectiveness.

But how effective is this approach? In a recent report from Holland on a study involving 26,071 people aged 65 years or older it has been shown to be beneficial. In this cohort, who were revaccinated annually, the subsequent all-cause mortality was reduced by 15% when compared with their first immunisation.

JAMA 2004;292:2089–95

## Beta blockers and hypertension

Atenolol has been around a long time and is one of the most widely used β-blockers, particularly in the management of hypertension. It has often been used as a reference drug in randomised controlled trials of hypertension.

Recently, however, its value has been challenged by a team from Sweden. Using the Cochrane Library, they have identified four studies that compared atenolol with placebo or no treatment and found no outcome differences between atenolol and placebo in terms of cardiovascular or all-cause mortality or myocardial infarction. In a meta-analysis of five other trials in which atenolol was compared with antihypertensives, there were no major differences in blood pressure lowering between the treatment arms.

They concluded that these results cast doubts on atenolol as a suitable drug for hypertensive patients. Moreover, they challenge the use of atenolol as a reference drug in outcome trials in hypertension. This has prompted the Cardiac Society of Australia and New Zealand to point out in a circular letter that there is no evidence for class effect—other beta-blockers have not been impugned.

Lancet 2004;364:1684–9

## Management options for critical carotid artery stenosis

Patients with severe carotid artery stenosis are at high risk for stroke. They may be treated medically or surgically but endarterectomy is more effective in the prevention of stroke. But what about those who are not fit enough for surgery? Could stenting do the trick?

In a recent randomized trial comparing carotid artery stenting (with the use of an emboli-protection device) with endarterectomy, stenting was found to be not inferior with respect to clinical outcome. Therefore, the less invasive approach may be an acceptable alternative among patients with high-risk carotid artery stenosis.

N Engl J Med 2004;351:1493–501

## How to treat Bell's palsy?

In a recent BMJ paper it was recommended that patients with acute hemifacial paralysis (Bell's palsy) should all be treated with steroids and antiviral drugs, and preferably be referred to a specialist. This prompted 24 responses, 10 of them critical of the views on treatment.

Critics stated that the authors were wrong to recommend early treatment with steroids or antiviral agents, or both, because the supportive evidence they offered was inconclusive and flawed; they ignored the best evidence (two systematic reviews) and selected other trials to support their own opinion; neither treatment is harmless, and antiviral agents are expensive; and they glossed over other potentially useful treatments.

**And the author's (weak) response—**"Although systematic reviews do not show statistically significant benefits, they are at least suggestive of benefit."

**And—**"We accept that there is no evidence that referral will lead to better outcomes but suggest that any patient is best cared for by a practitioner with an interest."

BMJ 2004;329:1103–4.

## Bug, drugs and the marketplace

Vancomycin-resistant gut bacteria first showed up in 1986. Then in 1997, the discovery of partially resistant strains of *Staphylococcus aureus*, which causes serious wound and surgical infections, shattered the hope of maintaining vancomycin as the ultimate weapon against the worst hospital-acquired infections. Two years ago, the first cases of fully vancomycin-resistant *S. aureus* were reported in the United States.

"If they can become resistant to vancomycin, they will become resistant to everything" says Chris Walsh, a molecular pharmacologist at Harvard Medical School in Boston. So we need new antibiotics—but will we get them? Maybe not.

There has been a 56% decline in the number of antibiotics approved annually by the US Food and Drug Administration (FDA) over two decades. According to a study published in the Journal of Infectious Diseases this year (2004;38:1279–86), only 6 of 506 drugs in late-stage clinical testing by the world's 15 largest pharmaceutical companies are new antibacterials—and all are derivatives of known antibiotics.

Companies have more incentive to bankroll research into treatments for chronic conditions such as high cholesterol or rheumatoid arthritis, for which patients take the drug over years or a lifetime, rather than for just a week or two.

Nature 2004;431:892–3.

## Regarding 'Mortality, morbidity, and asbestosis in New Zealand: the hidden legacy of asbestos exposure'

Whilst I agree with the conclusion that lung cancer in asbestos-exposed workers is significant, and frequently attributed only to smoking rather than the asbestos exposure, I do not agree that this is associated with asbestosis as described by Dr Pamela Smartt in her article (N Z Med J. 2004;117(1205). URL: http://www.nzma.org.nz/journal/117-1205/1153).

Regrettably, evidence of asbestos exposure by the finding of asbestos-related pleural plaques is often called asbestosis, even by radiologists. Asbestosis has come to mean any asbestos-related disease where it really refers to the quite specific interstitial lung disease caused by asbestos exposure.

I suspect the method used in this paper will over-estimate the prevalence of asbestosis, whilst not negating the overall premise that asbestos exposure is under-reported as a cause of lung cancer deaths.

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## Perinatal information

Congratulations to Dr Rosemary Reid on her recent editorial.[1] It amazes me that she should still need to point out deficiencies in this country's perinatal information systems.

In 1988, a widely-representative working party, convened by the then Department of Health, produced a report[2] which made a host of recommendations. It was totally ignored. This, among other frustrations, I suspect, led to the loss of several people from the Department, who might have otherwise played an important part in implementing recommendations contained in that report.

Among the recommendations was the establishment of a national perinatal epidemiology unit along the lines of units in Australia and the UK. The latter have proved their worth. Had a counterpart been set up in New Zealand at that time, Dr Reid's complaints about lack of in-depth study should not have been needed now.

Although it was almost usual for expert reports on health topics in the past to be quietly shelved, that one came at an especially bad time. The country was in the depths of Rogernomics and the powers that were in control of the health service had become obsessed with means, largely ignoring the ends to which the means were supposed to be directed. I suspect they still are, although perhaps less rabidly. I found it hard to believe even then that the country, preoccupied as it was with cost-cutting and managerialism, could spend hundreds of millions of dollars a year on maternity services with no good system for analysing their results and suggesting ways the money might be more effectively spent.

That report was by no means the first effort to improve perinatal information systems in this country. The working party documented earlier published recommendations back to 1969, and from his arrival in 1964 Dennis Bonham worked tirelessly. Well before that, obstetric hospitals in many places here and overseas produced annual statistical reports. For myself, the fact that obstetricians (in contrast to surgeons and physicians of the time) took some systematic interest in the outcome of their work was one of the things that attracted me into the newborn field in 1962. Progress has been made in the past 40 years, but I am distressed that it is so little in comparison with what might have been.

We should have had a better system over 20 years ago. There is a lot of leeway to be made up. Why not now?

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## Prognostic value of erythrocyte sedimentation rate in patients with decompensated heart failure

Chronic heart failure (CHF) is one of the most frequent source of morbidity and mortality in the elderly.[1] Different studies have searched for markers of prognosis in this disease. Erythrocyte sedimentation rate (ESR) is an easily available and cheap marker—although the outcomes of studies about its prognostic value have been contradictory.[2,3]

The purpose of our study was to assess the usefulness of ESR in patients with CHF and acute cardiac decompensation. We studied (retrospectively) ESR values in 77 hospitalised patients, admitted consecutively, whose diagnosis of heart failure was confirmed by echocardiography. Patients with other causes that can modify ERS (infection, neoplasm, and connective tissue diseases) were ruled out. Two years later, we assessed their survival rate.

Baseline characteristics of the studied population are presented in Table 1. All patients were in III or IV NYHA class. Differences were not found in ESR by age or sex. In patients with systolic dysfunction, ESR was lower than in ones with diastolic dysfunction (24.45±17.39 mm vs 35.59±26.06; p=0.037).

**Table 1. Baseline characteristics of patients**

| Variable | n (%) |
|---|---|
| **Age** | |
| • ≤ 70 years | 21 (27.3%) |
| • > 70 years | 56 (72.7%) |
| **ESR** | |
| • < 20 mm | 31 (40.6%) |
| • ≥ 20 mm | 46 (59.4%) |
| **Sex** | |
| • Male | 39 (50.6%) |
| • Female | 38 (49.4%) |
| **Prescription of ACE inhibitors** | |
| • Yes | 60 (77.9%) |
| • No | 17 (22.1%) |
| **Type of dysfunction** | |
| • Systolic | 33 (42.9%) |
| • Diastolic | 44 (57.1%) |
| **Aetiology** | |
| • Ischaemic disease | 26 (33.8%) |
| • Hypertension | 23 (29.9%) |
| • Others | 28 (36.4%) |

Eighteen patients (23.4%) died during follow-up period. The Kaplan-Meier survival method showed in systolic dysfunction group that ERS under 20 mm tended to greater survival, without differences statistically significant (p=0.065). In patients with diastolic dysfunction, no differences were found.

The Cox proportional risks model was used, and the predictor of death at 24 months was 'ESR higher than 20 mm' (OR 6.75; 95% CI 1.373–33.33; p=0.017). Age, sex, type of dysfunction, aetiology of heart failure, associated diseases (hypertension, diabetes, COPD, and cerebrovascular diseases) and treatment with ACE inhibitors were not significantly associated with 2-years mortality by multivariate analysis.

Chronic inflammation may play a role in the pathogenesis of atherosclerosis—so different markers of inflammation have been studied in cardiac dysfunction.[4] In our study, ERS was increased in patients who died during the follow-up period. Previous studies had reported that a low ESR was associated with severe cardiac decompensation and unfavourable prognosis.

ESR was related with clinical and haemodynamic status of patients.[2] On the other hand, Sharma et al demonstrated the relationship between low ERS and better prognosis in patients with systolic dysfunction and stable CHF.[3] In this study, ERS was identified as a predictor of survival in patients with decompensated heart failure.

In a variable percent of patients, prognostic value of ESR is limited, because decompensation is caused by diseases that increase ESR (such as infection). We did not find differences in ERS values among the several causes of heart failure, but the groups were too small and no clear conclusion can be drawn. Although the study is limited by a small number of patients this finding is consistent with prior reports.

Our results suggest that ESR could be a useful marker of mortality in patients with CHF and acute decompensation. Indeed, because it is a cheap and easily available, ERS could be a tool for monitoring patients at risk after discharge.

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## CSF bilirubin measurement for xanthochromia

A recent article argued that visual inspection of cerebrospinal fluid (CSF) for xanthochromia should be replaced by spectrophotometric analysis[1] as proposed in guidelines[2] published from the UK. Following the publication of these guidelines, the rate of visual assessment of CSF has fallen from 24% to 6% in the UK.[1] Although, we routinely employ scanning spectrophotometry in the suggested manner,[2] it is not necessarily suitable for use in all clinical laboratories and remarkably most laboratories in the United States still rely on visual inspection.[3]

We recently described the measurement of CSF bilirubin, using a modification of our routine serum bilirubin assay on the Aeroset analyzer (Abbott Laboratories), providing an alternative more practical approach.[4] This approach can be potentially be adapted for use on other automated analysers.

To determine a reference range, we measured CSF bilirubin levels on samples from patients in which SAH was not suspected,[4] and which was macroscopically clear and had normal CSF protein values ($\leq$ 400 mg/L). 172 samples were analysed with a mean CSF bilirubin of 234 nmol/L ranging from 110–359 nmol/L ($\pm$ 2SD).

In 144 CSF samples with a request for xanthochromia testing, 23 were positive by spectrophotometry, with elevated net bilirubin absorbance.[4] The ability of CSF bilirubin measurement to correctly identify the presence of xanthochromia assigned in this way was investigated. At our upper reference limit for CSF bilirubin of 359 nmol/L, negative predictive value was 100%, suggesting that subarachnoid haemorrhage can be reliably excluded at values below this level.[4]

We propose to prospectively validate our decision limit in further studies with the intention that CSF bilirubin measurement should become the routine method for xanthochromia detection. Provided that laboratories validate their decision limits, CSF bilirubin measurement may be a robust practical alternative to spectrophotometry.

Christopher Florkowski, Jacobus Ungerer, Sandi Southby, Peter George
Canterbury Health Laboratories, Christchurch

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## Professional Discipline – Indecent Assault Conviction

**Charge:**

The CAC charged that Dr Karunasekera had been convicted by the District Court in Invercargill of three offences of indecent assault under Section 135(1)(a) of the Crimes Act 1961, each being an offence punishable by imprisonment for a term of three months or longer. The CAC charged that the circumstances of the offences reflected adversely on Dr Karunasekera's fitness to practise medicine.

**Background:**

Dr Karunasekera arrived in New Zealand with his wife and two young children in February 2002 on a work visa. During his time at Grey Hospital, Dr Karunasekera was involved in a conflict with a senior consultant who was critical of his work although the matter was dealt with by the hospital administration.

Towards the end of 2002 Dr Karunasekera and his family went on holiday to Queenstown over the New Year period and it was at this time that the offending took place. There were three separate incidents that were the subject of the charges. The first two incidents occurred in two different shops. In each of those shops, Dr Karunasekera pulled a shop assistant's hands down to the penile region of his trousers. In the third incident Dr Karunasekera ran his fingers against the shop assistant's breasts and held her breasts for a period of approximately two seconds.

Dr Karunasekera pleaded guilty to the charges and was convicted and sentenced as follows:

- 12 months supervision; and

- Referral for assessment at the Adult STOP Programme and, if found suitable, to undertake the programme as directed and to the satisfaction of his probation officer; and

- Referral for Department of Corrections psychological assessment and treatment as directed by and to the satisfaction of his probation officer; and

- Payment of reparation (emotional harm) of $500.00 to each of the three complainants.

There was no dispute that Dr Karunasekera had been convicted of an offence that is punishable by imprisonment of a term of three months or longer.

Similarly, there was no dispute that the circumstances of the offences reflected adversely on Dr Karunasekera's fitness to practise medicine.

**Finding:**

The Tribunal found that Dr Karunasekera had been convicted of offences punishable by a term of imprisonment of three months or longer and that the circumstances of the offending reflected adversely on his fitness to practise.

**Penalty:**

The Tribunal ordered that Dr Karunasekera be censured; pay 15% of the costs and expenses of the prosecution and hearing costs of the inquiry. The costs were at the lower end of the scale as Dr Karunasekera accepted the charge and showed co-operation and commitment to his rehabilitation.

The Tribunal recommended to the Medical Council that should Dr Karunasekera wish to re-register, that re-registration should not occur until he has completed the STOP programme. The Tribunal further recommended that prior to re-registration Dr Karunasekera undertake the Sexual Misconduct Assessment Test (SMAT). The Tribunal considered registration and conditions of practice which might be imposed following the SMAT should address matters such as practising within a team or with a chaperone and issues of supervision.

It further ordered publication of the decisions of the Tribunal in the New Zealand Medical Journal.

The full decisions relating to the case can be found on the Tribunal web site at www.mpdt.org.nz Reference No: 04/121C.

## Maurice White Falloon

Maurice Falloon's death on July 25, 2004 ended a life of determination and hard work, a life which also included many friendships and recreational interests.

Maurice was said to be always a gentleman, who was calm and courteous even under pressure.

A surgeon at Wanganui Base Hospital for 32 years, he was passionate about keeping services in the city, and was instrumental in having wings and operating theatres added to the hospital.

Maurice was born into a farming family near Masterton in 1921, the youngest of seven children. At the age of 14 he suffered a burst appendix and nearly died. He spent a month in hospital, which began his interest in medicine.

His secondary education took place at Wairarapa High School, then Wellington College. He studied hard as well as playing cricket and rugby. After 5 years of medical study at Otago University, he went to Palmerston North to work as a house surgeon. There he met a stylish and vivacious nurse, Patricia Brooking, whom he married in 1949.

The two shifted to Kaitaia, where Maurice was medical superintendent. But his wife recognised his ability and she encouraged him to specialise. They went to England so that he could study in London, and he worked their passage as a ship's doctor. While in London, Mrs Falloon supported them by working as a dental nurse in a Harley Street practice, and they both enjoyed trips within Europe.

Maurice's study under top surgeons was demanding, but he was successful. He became a Fellow of the Royal College of Surgeons in 1953. Their first child was also born that year, and the family soon returned to New Zealand to Wanganui, where Mrs Falloon's family lived. Maurice worked as a surgeon at Wanganui Base Hospital from 1954 to 1986, becoming head of the department in 1980. He spent a term chairing its medical advisory committee.

At the same time, he maintained a private practice, which carried on after he retired from the hospital. He was also a director of the private Belverdale Hospital from 1955 to 1986, and a foundation trustee of what began as the Eye Care Trust.

He had to be on his feet and to concentrate a lot—and his son, Roger, remembered his father often fell asleep in his chair after a hard day of surgery. Despite being busy, he had strong interests and played golf, owned a small farm in Rapanui Road, went to race meetings, and bred racehorses. He had a lifelong interest in racing and breeding racehorses, but never struck it lucky. He and his wife also enjoyed entertaining, and there were often large gatherings at their house after race meetings.

In 1993, Maurice established the Wairere Probus Club and became its first president. He was a long-time member of the Wanganui Jockey Club, and its president from 1975 to 1977. And he was a life member of the Marton, Rangitikei, and Waverley racing clubs.

Maurice was awarded a Paul Harris Fellowship for 21 years of faithful membership at the Wanganui Rotary—and he lent a hand with the city's Sommerville Centre, Cancer Society, and Hospice.

For the last 6 years he was the main caregiver for his wife, who had had a stroke. He showed new depths of strength and determination in keeping the two of them together and independent.

Maurice became frailer, and died on July 25 when his heart failed. He was 83, and is survived by his wife, 5 children, and 12 grandchildren.

Maurice will be remembered as an excellent surgeon, who dressed well, was a very good colleague, and was cool under pressure. He will be sadly missed by his colleagues and many friends, who were present at his funeral.

We are grateful to Dr Neil de Zoysa (Senior Physician, Wanganui Hospital) for this obituary.

## Medical Benevolent Fund

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

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
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## Hyperlipidemia (2nd edition): Fast Facts Series

Paul Durrington, Allan Sniderman. Published by Health Press Ltd (distributed by Elsevier), 2002. ISBN 1903734231. Contains 139 pages. Price A$35.20

This small booklet is an easily readable guide to lipid disorders and management. It is most valuable for specialist clinics within institutions doing lipid disorders management. It is essential reading for registrars, clinic nurses and dietitians who are attached to such units. The book should also be read by all other professionals who undertake cardiovascular risk factor management (e.g. diabetes clinics, and cardiological services).

The first publication is in 2000 and this was revised in November 2002 but is only made available late in 2004. The booklet covers 12 sections ranging from basic physiology of lipids and lipoproteins, the specialist genetic disorders through to sections on dietary and drug management. Both authors are very well known lipidologists. Despite its size (139 pages), it packs in a lot of information. A number of the chapters, for example, include important but rare conditions of familial hypercholesterolaemia and type III dyslipidaemias that will not be of much relevance to many clinicians who are prescribing lipid modifying agents on a frequent basis. However the sections on basic physiology and treatments, both diet and drug therapy, are essential reading for any prescriber of the widely used lipid-modifying agents.

Although the book is already a couple of years old, the only chapters which reflect this age are the sections on guidelines. Since the end of 2002 there have been major changes in the guidelines for management of cardiovascular risk. The booklet thus needs to be read in the context of the New Zealand guidelines publication "The Assessment and Management of Cardiovascular Risk" completed in December 2003.

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## Colorectal Cancer (2nd edition): Fast Facts Series

Irving Taylor, Julio Garcia-Aguilar, Stanley Goldberg. Published by Health Press Ltd (distributed by Elsevier), 2002. ISBN 1903734088. Contains 75 pages. Price A$35.20

This book is a gem. In only 75 pages, which outline most relevant aspects of current knowledge available on colorectal cancer, the authors cover epidemiology; pathophysiology; clinical presentation, diagnosis, and staging; screening; and treatment of primary disease, advanced disease, and recurrent disease. This book is easy to read (you can read it from cover to cover in one evening), well put together, and the information is current and correct.

This series covers a wide range of topics from allergic rhinitis to urinary stones. The authors (two from the USA and one from the UK) are internationally well-known for their work on colorectal disease. This adds to the very good balance in the presentation of information.

The book is small, soft covered, well bound, and makes good use of colour in layout and in figures. The font style and size are excellent. There is a colour-coded index along the side of the page. The one criticism I would have of presentation is in regard to the photos; these are poor quality and need to be improved before the next edition.

I think this is an excellent book for anyone who needs to know more about colorectal cancer. It would be of great value to medical students, advanced trainees in general surgery and oncology, or another doctor who wishes to know more about colorectal cancer.

Frank A Frizelle
Editor, NZMJ (and Colorectal Surgeon, Christchurch Hospital)

## Surgery Minitext

John Lumley, Paul Sroden, Ramanathan Visvanathan (eds). Published by Arnold (Hodder Headline Group), 2004. ISBN 0340809779. Contains 830 pages. Price $85.00

This pocket-sized book is a concise surgical encyclopedia—surgery of the head, breast, thorax, abdomen, vascular, transplantation, orthopaedics, spine, and peripheral nerves are all covered in hundreds of small pages (9.5cm x 17.5cm).

In contains basic information of topics including postoperative complications, nutrition, HIV, and other infections. It is the sort of book that used to be carried around by medical students and house staff while attached to surgery rotations. The book appears to cover the normal topics expected by medical students. This book alone would however be inadequate to be their surgical textbook, and is no doubt aimed to be a companion to a larger textbook.

The quality of the presentation of the book is excellent. The book has a soft plastic cover, appears well bound, and its pages appear to be made of good quality paper. The font size and colouring are easy to read, and it is small enough to be carried in a jacket pocket.

There are two main faults with this book, the first problem is that the content of the chapters appears well out-of-date for a new book. While most books are in fact out-of-date when published, the text (on the topics I know best) appears at least 10 years out-of-date. While this possibly reflects current UK practice, I expect, however, that real problem lies with the authors.

The other problem is that most of the students now have moved away from books such as this and have the information in their pocket computer (e.g. palm pilot) thus allowing easier updating. So I am surprised this book doesn't come with the ability to load the book into a computer. This would add little to the cost and make it more user-friendly.

In summary this is a nice pocket book, but out-of-date in content and presentation. I think are better alternatives on the market at present.

Frank A Frizelle
Editor, NZMJ (and Colorectal Surgeon, Christchurch Hospital)

## Emergency Medicine: The principles of practice (4th edition)

Gordian Fulde (ed). Published by Churchill Livingstone, 2004. ISBN: 0729537471. Contains 642 pages. Price A$75.00

The First Edition was published in 1998 and the latest edition reflects the huge development in Emergency Medicine. There has been substantial expansion of this publication, which is now over 600 pages in length. It is a multi-authored text with the majority of authors being Fellows of the Australasian College for Emergency Medicine and several chapters authored by specialists in related fields.

The book is a very positive addition to the rapidly developing field of Emergency Medicine. Chapters are thoroughly researched with a "Recommended Reading" section at the conclusion of each chapter. Many chapters also have an "Editor's Comment" allowing Associate Professor Fulde to add his views and wisdom. Whilst not intending to be a complete analysis of all subjects, there is sufficient to make this text highly recommended for the doctor new to Emergency Medicine.

Whilst it is probably of most value to the House Officer/Registrar, I found all chapters to be useful. It is an easy book to pick up and read a chapter in a spare 10 minutes. I am sure many Specialists in Emergency Medicine will find this compulsive reading and an ideal update.

The only criticism I would make is that the amount of knowledge has almost outdone the size of the pages. Whilst it is still described as a pocket-sized book, I think few doctors would be able to carry this book (>3 cm thick) in their pocket. Because of its thickness, opening the book to a page and holding open (one-handed) can be a little cumbersome. I wonder if the publishers will move to a larger page format in future. There are no photographs but there are a several diagrammatic illustrations.

Everyone working in this field will derive benefit from reading this book. This should be compulsory reading for doctors before commencing work in an Emergency Department.

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## Unification of Western Medicine & Traditional Chinese Medicine

Lonnian Lee. Published by Lee Chongwi Press Ltd, 2004. ISBN 0473096269. Contains 431 pages.

This book aims to "bring the greatest revolution in the history of medicine", by providing the basis for unifying western medicine (WM) and traditional Chinese medicine (TCM), without clashes of theories and practice. Lee notes similarities between early WM and TCM- theories were based on concepts of the elements and humours—then traces the evolution of WM as knowledge advanced in anatomy, physiology, and chemistry. Following Descartes, WM attempted to explain and treat symptoms in terms of measurable pathology.

Meanwhile TCM largely retained a nonscientific framework, which Lee describes in some details. Anatomical terms such as kidney symbolise the various "energy body systems", without implying pathology in the actual organ. A new nomenclature for TCM is proposed, to avoid confusion with western medical terms, when the two are unified.

This unified "Synergetic Medicine" is apparently needed due to oft-voiced dissatisfaction with WM, unable to explain and treat all symptoms. It is claimed that TCM is complementary, able to diagnose and treat subclinical diseases, and symptoms unresponsive to WM. However, the evidence provided amounts to little more than anecdotes and statements of beliefs. Further detractions are the author's inclination to verbosity and repetition, frequent diversions into discussions on logic, and the use of algebra to depict ideas.

Despite having access to nearly 1000 publications listed in the bibliography—on medicine, philosophy, and logic—Lee's case for unification is unconvincing. It has been said that the twain of East and West will never meet. In this instance, TCM has not met the standard of proof of WM. Practitioners of WM wishing to expand their repetoire for helping patients with their symptoms, are likely to find the time and effort spent reading this book poorly rewarded.

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## Netdoctor: Osteoporosis

Dan Rutherford. Published by Hodder & Stoughton, 2004. ISBN 0340862661. Contains 110 pages.

This pocket book from Netdoctor is targeted at patients, relatives, nursing staff, and some junior doctors. There is good general overview of osteoporosis and a basic section on bone biology, which is easily digestible. The issue of bone mass and bone density is covered very adequately with some very sensible suggestions about repeat bone density. The key point section at the end of every chapter is excellent, providing concise highlights and key points.

In regards to treatment, this section is aimed at treatment available in the United Kingdom as we unfortunately do not have access to SERMs, Strontium, Teriparatide, Residronate, etc.

The section on hormone replacement therapy is a little weak and should have come down with a few recommendations on HRT. Comments about the lack of fracture prevention efficacy with HRT should have been included. A section on osteoporosis in men is welcome as is the Appendix section, which gives some very simple advice on exercise, diet, etc.

Overall this is a handy book spoilt by some typographical errors and little information as to who Dan Rutherford is.

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