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A Scott, R Toomath, D Bouchier, R Bruce, N Crook, D Carroll, P Dixon, J Doran, P Dunn, C Hotu, M Khant, M Lonsdale, H Lunt, E Wiltshire, D Wu

Diabetes is known to cause long-term complications which can be reduced by maintaining good control of blood glucose levels. In this study, the largest survey of diabetes care amongst children and young adults across New Zealand, data on 1282 people from 9 hospitals were pooled and analysed. Overall blood glucose control (measured using HbA1c) was poor but some centres achieved better levels than others. These findings are similar to those reported in this age group, by other countries such as the UK. Complications such as eye and kidney problems (retinopathy and nephropathy) were common particularly after more than 10 years of diabetes. Māori and Pacific Islanders were more likely to have evidence of kidney damage than New Zealand Europeans.

Ethnic differences in Type 2 diabetes care and outcomes in Auckland: a multiethnic community in New Zealand
T Robinson, D Simmons, D Scott, E Howard, K Pickering, R Cutfield, J Baker, A Patel, J Wellingham, S Morton

Māori and Pacific people with Type 2 diabetes are more likely to have bad health outcomes than other New Zealanders with diabetes. This study asks whether some of these differences may be due to differences in general practice care. The care provided to nearly 6000 people with Type 2 diabetes was studied. We found that although GPs were providing equally intensive care to Māori and Pacific people they were not achieving the same outcomes of care (for example smokefree, blood pressure control, blood sugar control).

Health status of New Zealand European, Māori, and Pacific patients with diabetes in 242 New Zealand general practices
A Tomlin, M Tilyard, A Dawson, S Dovey

In this paper, we compare indicators of health between New Zealand European as well as Māori and Pacific people presenting with diabetes at general practices in the South Island of New Zealand. Practices and patients were participating in the Government’s Get Checked programme providing free annual diabetes health checks. Ethnic inequalities were noted in seven of nine health status measures; Māori and Pacific people were more likely to be at risk for diabetes complications.
The Dunedin Multidisciplinary Health and Development Study: are its findings consistent with the overall New Zealand population?
R Poulton, R Hancox, B Milne, J Baxter, K Scott, N Wilson

The health of 26-year-old participants in the long-running Dunedin Multidisciplinary Health and Development Study was found to be very similar to people of the same age in the nationally representative New Zealand Health and National Nutrition Surveys. This suggests that the findings from regional cohort studies such as the Dunedin Study are likely to be relevant to people in other parts of New Zealand. It also suggests that the health of the Dunedin Study members has not been changed by repeated assessments throughout their lives. These findings have important implications for the design of the proposed national Longitudinal Study of New Zealand Children and Families.

Metabolic equivalent (MET) intensities of culturally-specific physical activities performed by New Zealanders
K Moy, R Scragg, G McLean, H Carr

This study’s aim was to collect objective information on physical activities typically performed by New Zealanders, including Māori and Pacific cultural activities. Information about the type of activities performed, and the physical effort or ‘intensity’ associated with executing such activities, were collected from 186 adults. Data were converted to metabolic equivalents (METs) to classify activities as light, moderate, or vigorous intensity, and compiled to create a New Zealand-specific compendium of physical activities. This culturally-specific information can be incorporated into physical activity and health-related questionnaires to improve the quality of information collected from respondents in both clinical and public health settings.

Do snacks of exercise lower blood pressure? A randomised crossover trial
R Elley, E Bagrie, B Arroll

We know that regular exercise, such as brisk walking for 30–40 minutes per day, can help lower blood pressure, particularly in those with hypertension. However, with busy lives, people can not always set aside 30–40 minutes per day to achieve this. Completing the required amount in ‘snacks of walking’ spread throughout the day would make it easier for some people to gain the benefit of exercise. Previously, there was little evidence that 10-minute snacks of exercise could achieve blood pressure reductions as effectively as continuous walking. This study found that 4×10-minute snacks of walking are as effective as 40 minutes continuous walking per day at reducing blood pressure, when compared with doing no exercise. This reinforces the message that walking can help control hypertension and can be achieved in 10-minute snacks throughout the day.
Dietary patterns of New Zealand European preschool children
R Theodore, J Thompson, C Wall, D Becroft, E Robinson, P Clark, J Pryor, C Wild, E Mitchell

Little is known about what preschool New Zealand children eat. The aims of this study were to describe the dietary patterns of New Zealand (NZ) European preschool children and to compare these with NZ Ministry of Health (MOH) food and nutrition guidelines. Food frequency information was collected on 549 New Zealand European children aged 3.5 years. Notable proportions of children were not eating fruit (27%), vegetables (52%), and bread and cereal (93%) at levels recommended by the MOH.
Diabetes epidemiology in New Zealand—does the whole picture differ from the sum of its parts?

Juliet Berkeley, Helen Lunt

At a time when most chronic diseases are showing a slowing of growth in prevalence and/or incidence, the diabetes epidemic in New Zealand continues unabated. If current trends continue, the incidence of both Type 2 and Type 1 diabetes is predicted to double in around 15 years.\(^1\)\(^2\)

The rapid rise in the number of patients suffering from diabetes is consistent with environmental factors playing a major pathogenic role. The environmental factors responsible for the rise in Type 1 diabetes are elusive. In contrast, the impact of risk factors for Type 2 diabetes that are present in our current ‘obesogenic’ environment is well known, even if the best way of improving these environmental risk factors has yet to be determined. How much relevance does this epidemic have for New Zealanders?

The paper by Joshy and Simmons in this issue of the Journal argues that the cost of treating Type 2 diabetes, a potentially preventable condition, is taking health dollars away from other important conditions.\(^3\) Indeed, one estimate suggests that the annual cost of treating Type 2 diabetes will be more than 1,000 million dollars, by 2021.\(^4\)

What New Zealand-specific elements form part of a local diabetes clinical epidemiologist’s job description? Epidemiology measures the determinants and distribution of disease. New Zealand has a unique ethnic mix, unique culture, and unique obligations regarding the health and welfare of its indigenous people. The impact of diabetes and its complications on Māori, in particular, has been a focus of many of the papers cited by Joshy and Simmons. These authors also highlight the impact of diabetes on Pacific Island and Asian people, living in New Zealand.

Epidemiological tools help us solve problems related to disease prevention and control, as well as help in the evaluation of health service delivery. Overseas studies have begun to address some of these issues in Type 2 diabetes; for example the impact of lifestyle intervention and therapy with metformin has been quantified in pre-diabetic populations,\(^5\) but the transferability of these ideas to the New Zealand setting is currently unknown.

Several local interventional projects have recently started, for example the Waikato’s Te Wai O Rona – Diabetes Prevention Strategy and Counties Manukau’s Lets Beat Diabetes project. These projects hope to address the question of the best lifestyle interventional approach required to prevent diabetes, from a regional perspective. Results should start to come through in the next couple of years.

The burden of Type 1 diabetes falls most heavily on the New Zealand European population. Type 1 prevalence is around 10% that of Type 2 diabetes. The mix of a relatively uncommon condition in a patient subgroup that has a genetic and environmental make up similar to that of Type 1 populations in Europe, Australia, and North America has meant that local epidemiological research in Type 1 diabetes has
tended to explore points of similarity, rather than points of difference, with populations in other geographic regions. New Zealand research on the prevention and control of Type 1 diabetes is now focussed on international collaboration through the TrialNet series of studies. A second article in this journal, by Scott et al, examined outcomes of care in young New Zealanders with diabetes. Their paper highlights the importance of undertaking a nationwide approach to evaluating health service delivery in Type 1 diabetes, as they found marked regional differences in glycaemic control, which have yet to be explained. They also reached a similar conclusion to the paper by Joshy and Simmons, in their finding that young Māori and Pacific Islanders have a disproportionate burden of Type 2 diabetes and its complications.

Accurate diabetes epidemiological data that is New Zealand-specific is clearly a necessity for the development of local solutions to the Type 2 diabetes epidemic. Regional researchers have given us a snapshot of their local epidemiological findings and these findings are broadly consistent from region to region. However local clinicians and epidemiologists have struggled to find the resources required to collate accurate, up-to-date national statistics.

Robust national data is needed as a baseline against which the impact of both current initiatives, such as the free annual Get Checked diabetes check, and future national initiatives and interventions can be assessed. As an example, Budget 2006 earmarked 76.1 million dollars over 4 years to combat obesity in New Zealand. How can we know if this money has an impact on people with impaired glucose tolerance and diabetes, if we have no baseline data?

The 1996/1997 New Zealand Health Survey obtained only limited diabetes-specific data and is now 10 years out of date, yet forms the background for the only major statistical modelling undertaken of the diabetes epidemic, including the number of people suffering from diabetes in New Zealand.

The maturing of medical information technology lead to the hope that collation of primary care statistics might provide valuable epidemiological data for chronic disease management, for example through regional and national Get Checked data. Unfortunately Get Checked has not lived up to its promise on this count. This is partly because of lower than anticipated levels of patient recruitment but also because of our inability to give an accurate estimation of denominatory data, especially the number of Pacific Island peoples with diabetes; both points have been highlighted by Joshy and Simmons.

Obtaining accurate, relevant national data on both diabetes and impaired glucose tolerance should not, however, be beyond our reach. Looking across the Tasman to Australia, the recently completed AusDiab study is an example of a relatively low cost nationwide survey of the prevalence and risk factors for diabetes, which included an oral glucose tolerance test for all individuals surveyed who did not have known diabetes.

In summary, the New Zealand population has a unique mix of ethnicities, resulting in a unique interaction between the environment and those individuals and population subgroups that are susceptible to Type 2 diabetes and its complications. We have energetic researchers addressing regional epidemiological questions.
A glance at the reference list accompanying Joshy and Simmons’ article shows that we also have the benefit of a history of collaboration between researchers and individual clinicians, on the few diabetes projects that have been undertaken with a national focus. Despite these advantages, we still have no clear, up-to-date idea on whether regional diabetes data really does reflect what is occurring, nationally.

Does the sum of the regional parts in this diabetes epidemiology picture accurately reflect the whole (national) situation in New Zealand? We do not know the answer to this question, even though we should. The time for a detailed national survey of diabetes is overdue.

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References:


First national audit of the outcomes of care in young people with diabetes in New Zealand: high prevalence of nephropathy in Māori and Pacific Islanders

Adrian Scott, Robyn Toomath, David Bouchier, Raymond Bruce, Nic Crook, David Carroll, Rick Cutfield, Paul Dixon, John Doran, Peter Dunn, Cheri Hotu, Maunt Khant, Maureen Lonsdale, Helen Lunt, Esko Wiltshire, Denise Wu

Abstract

Background Diabetes is an important cause of morbidity and mortality amongst young people. Despite improvements in technology, maintenance of good glycaemic control is hard to achieve.

Methods In July 2003, 12 paediatric and adult hospital-based diabetes services across New Zealand were invited to take part in an audit of the process and outcomes of care. By March 2004, 9 centres had submitted data on 1282 (1117 with Type 1 diabetes, 105 with Type 2) children and young people born after 1 January 1978.

Results There were significant centre differences in terms of glycaemic control, rates of microvascular complications and complication screening. The group mean HbA1c was 9.1±0.3%. Amongst 789 people aged 16–25 years, the prevalence of retinopathy was 12.8% (range 0–26%); nephropathy was 17.1% (range 7–28%). Of those with a duration of diabetes >10 years, 25% had retinopathy and 27% nephropathy. Over the age of 12, microalbuminuria was more common amongst Māori and Pacific Islanders (43.8%) compared to Europeans (17%) or Others (17.8%). This was independent of the type of diabetes.

Conclusions This is the largest study of young people with diabetes undertaken in New Zealand. The results confirm the difficulty of achieving good glycaemic control in children and young adults. Microvascular complications were common, particularly in those of long duration, and cardiovascular risk factors were present in many young adults. The difference in average HbA1c% between centres was highly significant and independent of other factors. Type 2 diabetes mellitus in young people was associated with early onset nephropathy and dyslipidaemia (almost from diagnosis), thus suggesting the need for earlier diagnosis.

New Zealand (NZ) has a population of 4 million people with approximately 20% identifying themselves as Māori, and 5% as Pacific Islanders. In 2000, there were an estimated 115,000 people with known diabetes (NZ Ministry of Health data)—predominantly Type 2 diabetes mellitus (T2DM).

Under the age of 26 years, most people with diabetes have Type 1 diabetes mellitus (T1DM)—although with the rising tide of obesity, more teenagers and young adults are found to have T2DM. The estimated number of people with diabetes under age 26 in NZ is uncertain, but Christchurch data indicate an increase in T1DM over the last 30 years.1,2
Children diagnosed with diabetes have greater morbidity and mortality at all ages compared to their non-diabetic counterparts. Moreover, compared with adult-onset diabetes patients, the risk of developing renal or retinal complications is greater if diabetes is diagnosed under 15 years.

It is over 10 years since the Diabetes Control and Complications Study reported the beneficial effects of tight glycaemic control in adults and adolescents. The care of young people with diabetes is challenging, and recent studies from Europe and Japan have illustrated the difficulties of achieving and maintaining good glycaemic control. The studies also highlight the high prevalence of complications and the wide range of glycaemic control between centres (unrelated to patient selection or choice of insulin regimen). Nevertheless, some centres consistently have mean HbA1cs as good if not better than the intensive arm of DCCT, without the increased risk of hypoglycaemia.

Little is known about the prevalence or progression of Type 2 diabetes in children and young adults in New Zealand, but with the increase in obesity, the picture is likely to mirror that of the rest of the World.

**Methods**

**Study design**—This audit had approval from the local ethics committee of each participating district health board (DHB).

The all-NZ young person’s diabetes audit was begun in July 2003 when 14 centres (adult and paediatric diabetes services in each) covering 2.3 million of the NZ population were invited to participate in an audit of the process and outcomes of care of young people with diabetes up to the age of 26. By April 2004, 1 centre in South Island and 8 in the North (8 adult and 6 paediatric) had submitted data. Data from the Waikato has been published in detail elsewhere.

If they had attended a diabetes centre at least once in the previous 3 years, any person with diabetes born after 1 January 1978 was eligible for inclusion in the study. Up to 45 data items (including date of birth, duration of diabetes, last weight, height and BMI, lowest HbA1c during first year after diagnosis, last HbA1c (and mean of last 3), presence of microvascular complications, and details of treatment regimen were collected from either paper health records or electronic diabetes registers. Pathology laboratory databases were searched for missing test results. The hospital number was used to eliminate or combine duplicates where individuals had attended more than one centre over the last 3 years.

HbA1c was measured by a variety of methods but all were Diabetes Control & Complications Trial (DCCT)-aligned. Microalbuminuria was defined as an early morning urine with an albumin:creatinine ratio of >2.5 in males (>3.5 females) on more than one occasion. Where available the number of abnormal urines was recorded.

**Statistics**—Statistical analysis was performed using SPSS for Windows (version 12, SPSS Inc., Chicago, IL) software. Univariate ANOVA was used to compare centre differences (using HbA1c as the dependent variable), with age and duration of diabetes as covariates.

Chi-squared was used to compare ethnic differences and type of diabetes. Pearson correlation coefficient was used to explore the relationship between early glycaemic control and recent HbA1c. The significance value was set to 5%

**Results**

There were 1282 (1251 after duplicates removed) people with diabetes under the age of 26 as of 1 July 2003. The number, gender, ethnicity, and type of diabetes by centre can be seen in Table 1. Of Europeans with diabetes, 90.4% had T1DM compared to 66% of non-Europeans (p<0.0001).
Table 1. Characteristics of young people with diabetes (0 to 25 years) by centre

<table>
<thead>
<tr>
<th>Diabetes centre</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total patient numbers</td>
<td>164</td>
<td>252</td>
<td>150</td>
<td>71</td>
<td>37</td>
<td>109</td>
<td>62</td>
<td>45</td>
<td>392</td>
<td>1282</td>
</tr>
<tr>
<td>Prevalence/1000</td>
<td>0.7</td>
<td>0.8</td>
<td>0.4</td>
<td>0.4</td>
<td>0.2</td>
<td>0.6</td>
<td>0.6</td>
<td>0.4</td>
<td>0.8</td>
<td>0.6</td>
</tr>
<tr>
<td>Gender (no. Female)</td>
<td>90F</td>
<td>135F</td>
<td>74F</td>
<td>40F</td>
<td>17F</td>
<td>38F</td>
<td>27F</td>
<td>26F</td>
<td>194F</td>
<td>641F</td>
</tr>
<tr>
<td>T1DM (%)</td>
<td>93.3</td>
<td>94.4</td>
<td>90.0</td>
<td>90.1</td>
<td>91.9</td>
<td>94.5</td>
<td>80.6</td>
<td>94.5</td>
<td>80.6</td>
<td>90.4</td>
</tr>
<tr>
<td>T2DM (%)</td>
<td>6.1</td>
<td>5.2</td>
<td>9.3</td>
<td>4.2</td>
<td>8.1</td>
<td>5.5</td>
<td>17.7</td>
<td>4.4</td>
<td>12.6</td>
<td>8.7</td>
</tr>
<tr>
<td>European (%)</td>
<td>83.5</td>
<td>84.1</td>
<td>94</td>
<td>81.7</td>
<td>94.6</td>
<td>82.6</td>
<td>69.4</td>
<td>80.0</td>
<td>70.2</td>
<td>80.1</td>
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<tr>
<td>Māori (%)</td>
<td>1.8</td>
<td>10.7</td>
<td>1.3</td>
<td>14.1</td>
<td>2.7</td>
<td>7.3</td>
<td>21</td>
<td>13.3</td>
<td>5.9</td>
<td>7.3</td>
</tr>
<tr>
<td>Pacific Islander (%)</td>
<td>4.3</td>
<td>2.0</td>
<td>0.7</td>
<td>1.4</td>
<td>2.7</td>
<td>1.8</td>
<td>0.0</td>
<td>2.2</td>
<td>11.7</td>
<td>5.0</td>
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<tr>
<td>Other (%)</td>
<td>10.4</td>
<td>3.2</td>
<td>4.0</td>
<td>2.8</td>
<td>0.0</td>
<td>8.3</td>
<td>9.7</td>
<td>4.4</td>
<td>12.2</td>
<td>7.6</td>
</tr>
<tr>
<td>Age (SE)</td>
<td>16 (0.4)</td>
<td>17.7 (0.3)</td>
<td>19.1 (0.3)</td>
<td>17.8 (0.5)</td>
<td>20 (0.4)</td>
<td>15.5 (0.6)</td>
<td>22.9 (0.6)</td>
<td>14.3 (0.9)</td>
<td>17.8 (0.2)</td>
<td>17.9 (0.5)</td>
</tr>
<tr>
<td>Duration (SE)</td>
<td>6.6 (0.4)</td>
<td>7.3 (0.3)</td>
<td>7.9 (0.4)</td>
<td>5.2 (0.5)</td>
<td>7.6 (0.8)</td>
<td>7.3 (0.9)</td>
<td>7.6 (0.6)</td>
<td>5.9 (0.7)</td>
<td>6.8 (0.3)</td>
<td>6.9 (0.6)</td>
</tr>
</tbody>
</table>

DHB pop is the estimated population of the district health board (DHB) serving the centre.
Prevalence/1000 is the prevalence of diabetes (in young people 25 years and younger) per 1000 population.
T1DM (%) is percentage of total with Type 1 diabetes.
T2DM (%) is percentage of total with Type 2 diabetes.
The mean Hba1c was 9.1±0.1%. After correcting for gender and ethnicity, there were significant differences in mean Hba1c between the 2 centres, with the highest values (9.6±0.2%) and lowest (8.5±0.12%) values (p=0.000). There were significant differences in Hba1c between age groups (p=0.034) (Figure 1).

**Figure 1: Glycaemic control by age group (T1DM). Using ANOVA there are significant differences in Hba1c between age groups (p=0.034)**

![Hba1c by Age Group](image)

When type of diabetes was included as a variable, age group remained significant (p=0.06). For T1DM alone, age group (p=0.017) and centre (p<0.0001) were highly significant.

There were no ethnic differences in glycaemic control either during the first year after diagnosis, or the latest or the mean, HbA1c. An HbA1c measured during the first year after diagnosis was available in 274 (27%) of the subjects. The mean of the lowest recorded HbA1c during that year was 8.2±0.1%.

Of those who had a recorded HbA1c during this period, 12.5% were in the normal range. There was a positive correlation between the lowest HbA1c during the first year after diagnosis and future glycaemic control (Figure 2).

There were 662 subjects with Type 1 diabetes between the ages of 16–25 years. Over 50% were on multiple injection therapy (4 or more injections per day); 23 patients were on pumps (predominantly from 1 centre). There was no correlation between number of injections and glycaemic control. The prevalence of microalbuminuria and
retinopathy varied considerably from centre to centre, and increased with increasing duration of diabetes (Figure 3 and Figure 4).

Figure 2: Relationship between lowest HbA1c during first year after diagnosis and latest HbA1c (mean of last 3) in a sub-sample of 274 who had HbA1c results available from the first year after diagnosis. Pearson's correlation is significant (p<0.01) (2-tailed)

![Figure 2: Relationship between lowest HbA1c during first year after diagnosis and latest HbA1c (mean of last 3) in a sub-sample of 274 who had HbA1c results available from the first year after diagnosis. Pearson's correlation is significant (p<0.01) (2-tailed)](image-url)

There were 105 subjects with T2DM; 63% were non-European. The mean age was 20±0.4 y with duration of 3±0.3 y. All were overweight (mean±SE BMI 35±0.8). The majority were managed with diet alone, 19% insulin treated and 8% on Metformin or Acarbose with or without insulin.

The mean HbA1c was 8.5±0.2%; 20% were hypertensive (blood pressure [BP] > 130/80 mmHg), 72% had microalbuminuria (of whom 19% were treated with ACE-inhibitors), 4% had background retinopathy, and 4% had sight-threatening retinopathy.

The mean total cholesterol was 5.5±0.1 mmol/L, HDL cholesterol 1.2±0.05 mmol/L, triglycerides 3.5±0.5 mmol/L. Amongst those with T2DM, hyperlipidaemia was common with 61.5% having a total cholesterol >5.0mmol/L, 36% an HDL < 1 mmol/L, and 52.6% with triglycerides > 2.0 mmol/L. Only 3% were on lipid-lowering drugs.
Figure 3. Prevalence of microalbuminuria in 16–25 year olds with T1DM (left bars), including those with diabetes for 10 years (y) (right bars)

Over the age of 12 years, microalbuminuria were more common amongst Māori and Pacific Islanders (43.8%) compared to Europeans (17%) or Others (17.8%). This was independent of the type of diabetes.

Figure 4. Prevalence of retinopathy in 16–25 year olds with T1DM and effect of duration of diabetes (Centre 6 reported no retinopathy)
Discussion

This is the first study to look at the outcomes of care of children and young adults with diabetes across NZ and involved 8 adult and 6 paediatric diabetes services in 9 centres. The results demonstrate a disappointing picture of poor glycaemic control and moderately high rates of microvascular complications, as seen in other studies. Despite widespread use of multiple injection therapy, at all ages, few people achieved satisfactory control (only 22% had a recent HbA1c < 8%).

Although glycaemic control in each centre was poor, it is similar to other published studies in Europe of unselected young people with diabetes.\textsuperscript{8–12} Little data on young people with diabetes in NZ is available. In 2002, the Christchurch group reported a mean HbA1c for females aged between 13 and 20 years was 10.2% and 9.5% for males.\textsuperscript{19}

The type of insulin regimen (including use of pumps) did not appear to have much impact on glycaemic control. Our finding of a relationship between the HbA1c during the first year after diagnosis and future glycaemic control is consistent with data from Australia which suggested that poor control in childhood led to poor control in adolescence and beyond.\textsuperscript{20}

Other studies have suggested that poor early control is associated with a four-fold increase in the subsequent prevalence of nephropathy.\textsuperscript{21} An intriguing observation of the DCCT collaborators was that tight control initiated a year after diagnosis was associated with preservation of islet cell function for a greater period than the group randomised to conventional (poor) control.\textsuperscript{22}

There have been a few small studies (but no long-term randomised studies) looking at the impact (on beta cell function) of intensive normalisation of glycaemic control from diagnosis, with conflicting results.\textsuperscript{23,24}

Recent twin studies may offer an alternative explanation for the association between early and long term glycaemic control, which suggest that 62% of the population variance in HbA1c levels is genetically determined and independent of the genes influencing fasting blood glucose.\textsuperscript{25}

This is unlikely to be the explanation, since both in our study and in a UK study\textsuperscript{26} there were marked differences between centres in the number of children with a normal HbA1c during the first year after diagnosis. This implies differences in both expectation and training of the person with diabetes rather than differences in genetics.

Access to health is not always equitable and socioeconomic factors may explain some of the differences between centres, though in the Scottish study, age, sex, insulin regimen, BMI, season, social circumstances, and family history were all associated with glycaemic control but not with deprivation score based on post code.\textsuperscript{8}

The DCCT trial also suggested a period of improved control during adolescence is associated with long-term improvements in risk of complications—although HbA1c became similar in the intensive management and control groups soon after the end of the DCCT, the benefits of intensive management on microvascular complications persisted.\textsuperscript{27} This suggests adolescence is a critical period for future risk of complications.
The success of the DCCT, and the difficulties in obtaining similarly improved control outside the clinical trial setting (together with the association we have observed between metabolic control soon after diagnosis and future metabolic control) suggest that more intensive effort in diabetes education, support, and motivation (as occurred in the intensive arm of the DCCT along with intensive insulin management) may be particularly important during the first year after diagnosis as well as during adolescence.

The prevalence of retinopathy amongst those screened was similar to published series, but the difference in screening methods between centres (from direct ophthalmoscopy without pupillary dilatation to retinal photography with mydriasis) makes comparisons between centres difficult.

Microalbuminuria rates showed less variation which makes the finding of no retinopathy in one centre (where screening was with direct ophthalmoscopy) suspect, and emphasises the need for standardised screening methods across the country.

Some centres did not begin retinal screening until 16 whereas the International Society for Paediatric and Adolescent diabetes (ISPAD) guidelines are to start screening for microvascular complications from either 5 years after diagnosis or age 11 (whichever is earlier) with pre-pubertal diagnosis or from 2 years from diagnosis with pubertal onset. The extremely high prevalence of microalbuminuria in those with T2DM, and the known high incidence of renal failure and increased mortality in Māori and Pacific Islanders with the metabolic syndrome, make early diagnosis and intervention essential. This ethnic predisposition to nephropathy was apparent in both T1DM and T2DM.

Although the cause of nephropathy was not confirmed by renal biopsy in our study, adult studies in obese patients with T2DM suggest that persistent microalbuminuria is associated with either diabetic nephropathy, or obesity related focal and segmental sclerosis.

Use of ACE inhibitors is reasonable in those with confirmed nephropathy, although in part this depends on the criteria for diagnosis (in one centre, of 13 young people with 3 or more abnormal results, 11 were on ACE inhibitors. None of those with just 2 abnormal results were treated with ACE-inhibitors).

A recent publication found that up to 60% of people with T1DM have spontaneous resolution unrelated to ACE inhibitor use. This finding, and concerns about using ACE inhibitors in young women of child bearing age, may be the reasons why they are not frequently used.

Sub-optimal lipid profiles were very common especially in those with type 2 diabetes, yet only 3% were receiving any lipid lowering therapy. Cardiovascular Risk charts underestimate risk and are inappropriate for this age group. Only the American Diabetes Association has published specific guidelines for young people with T1DM.

With the knowledge that most will die prematurely from a vascular accident, and that vascular disease is even more common in T2DM (especially in Māori and Pacific Islanders), 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 36% of those with T1DM over 16 yrs, and 100% of those with T2DM, are overweight. This reflects obesity in the community as in 1997 approximately 25% of 15 to 18 year olds and one in three 19 to 24 year olds were overweight or obese. In addition, weight gain (especially in girls) on intensive insulin therapy can sometimes be spectacular, and likely to be a disincentive to better glycaemic control.

A limitation of our study is that the exact number of people under 26 years of age with diabetes in NZ is unknown. However, a careful prevalence study from Christchurch, New Zealand estimates that there are approximately 2540 with T1DM in this age group in the country.

Using their estimates of prevalence for the participating centres we appear to have identified 92% of predicted. Of those young people ‘lost to follow-up’, the published literature suggests they have worse control and greater risk of complications.

Another limitation is that not all centres in New Zealand participated in the study. Of concern, some were unable to gather the data. On the basis of studies from countries with similar socioeconomic circumstances as New Zealand, it is unlikely that the addition of data from these remaining centres would have altered our conclusions, but it is vital that all centres (caring for children and young adults with diabetes) audit their services and we urge them to do so.

In summary, this multicentre study of nearly 50% of the children and young people with diabetes in NZ has revealed large numbers with poor glycaemic control and a disturbing prevalence of early microvascular disease, despite introduction of intensive insulin therapy. Nevertheless, there are highly significant differences between centres independent of other factors suggesting opportunities for improvement.

The factors influencing success or otherwise in achieving good glycaemic control need to be investigated further. There needs to be greater adherence to management guidelines in screening for complications. T2DM in young people is becoming a major problem and is associated with early onset nephropathy and dyslipidaemia (almost from diagnosis), thus suggesting the need for earlier diagnosis, which is likely only going to be achieved by targeted screening of high risk children and young adults.

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References:


Ethnic differences in Type 2 diabetes care and outcomes in Auckland: a multiethnic community in New Zealand

Tom Robinson, David Simmons, David Scott, Eileen Howard, Karen Pickering, Rick Cutfield, John Baker, Ashwin Patel, John Wellingham, Sara Morton

Abstract

Introduction In New Zealand, Māori and Pacific (mostly of Samoan, Tongan, Niuean, or Cook Islands origin) people with Type 2 diabetes are more likely to suffer poor outcomes than other New Zealanders. Responsibility for addressing this outcome differential is falling on primary care and general practice in particular. This paper compares the general practice care provided to people with Type 2 diabetes in South and West Auckland, according to ethnicity.

Method An external audit of general practice diabetes care is carried out in South and West Auckland by the Diabetes Care Support Service. The results of 5917 routine patient audits carried out in 2003 are included in this study. Number of visits, recording of important information, risk factors, and treatments are compared between different ethnic groups.

Results Māori and Pacific people with diabetes who attend a regular GP had a higher average number of consultations than Europeans (5.7, 5.4, and 4.8 visits per year respectively). They were as likely as Europeans to have undergone important regular examinations and investigations. Māori were more likely than Europeans to be on some treatments. However, Māori and Pacific people were more likely to have a range of adverse risk factors for diabetes complications than Europeans. These include being a smoker (35, 18, and 13% respectively), having an HbA1c greater than 8% (50, 56, 23%), and having microalbuminuria (55, 50, 27%).

Discussion Although there were no large differences in the process measures of general practice diabetes care provided to different ethnic groups in South and West Auckland, Māori and Pacific people were not achieving the same outcomes of care in terms of risk factors for diabetes complications. Many of these risk factors are influenced by other factors in the wider community; however the New Zealand health system needs to consider how it can better address these differences.

New Zealand has a multiethnic community with 76.9% of the population being European, 14.1% being Māori, 6.2% being Pacific (mostly of Samoan, Tongan, Niuean, or Cook Islands origin), and 6.4% being Asian (mostly Chinese or Indian).

1 The prevalence of known diabetes is higher in non-European populations than New Zealanders of European descent.2–5

As in most other parts of the World, the prevalence of Type 2 diabetes in New Zealand is increasing alarmingly. Indeed, the total number of people with Type 2 diabetes is expected to increase by 78% between 1996 and 2011.6 The increase in numbers of Māori and Pacific people with diabetes is predicted to be even greater.
There is already a rapid increase in the burden of complications of diabetes including premature renal disease and diabetic foot disease.

Several studies have provided strong evidence that assertive management of Type-2 diabetes leads to reduced microvascular and macrovascular complications and better patient outcomes. However, achieving these improved outcomes requires clinical teams to provide thorough and systematic care including strict attention to risk factors such as hyperglycaemia, hypertension and dyslipidaemia, and aggressive drug treatment.

Maori and Pacific people with diabetes have much poorer outcomes than Europeans. For example, although Maori men are 3.5 times more likely to develop diabetes than European men, they are 6.5 times more likely to die of diabetes. Moreover, Maori and Pacific people with diabetes have higher rates of diabetes-related renal, foot, and eye disease complications than Europeans.

One possible contributing factor to the high morbidity and mortality amongst Maori and Pacific people with diabetes is different quality of care provided by health services. New Zealand studies in the fields of asthma and cardiac interventions have suggested that Maori and Pacific people do not always receive equal healthcare for equal need. In a study in South Auckland of people with diabetes, Maori were most likely to have no ongoing care, while Pacific peoples had comparable access to other New Zealanders.

This cross-sectional study examines the care provided to people with Type 2 diabetes by general practitioners in South and West Auckland participating in the annual clinical audit service provided by the Diabetes Care Support Service (DCSS). The aim of the study is to establish whether there are important differences in care provided to different ethnic groups.

**Methods**

Since 1994, the DCSS has carried out audits of the general practice care of patients with diabetes in South and West Auckland. This has been described in a previous publication. South and West Auckland have a predominantly urban population, with a high proportion of Maori and Pacific people, and a large number of lower socioeconomic (poorer) areas.

The DCSS audit is provided free to general practices who wish to participate. Confidentiality between DCSS and the general practice is assured and DCSS never contacts patients directly. Posters informing patients of the purposes of the audit are displayed in all practice waiting rooms, and general practitioners and practice nurses discuss the audit with patients. Patients are free to have their notes excluded from the audit procedure. The North Health Ethics Committee approved the DCSS as an ongoing audit in 1993.

The DCSS’s audit nurses visit participating general practitioners and examine the patient notes of all patients who have diabetes. To ensure that no patients with diabetes are missed from the audit, the audit nurses use several ways of searching for patients in addition to using the practice’s disease register. For example, they search for patients on oral hypoglycaemic medication, insulin, patient’s prescribed capillary glucose testing strips, and patients who have had an abnormal HbA1c result.

Once all patients with diabetes are identified, 111 items of clinical data are collected on each patient. This includes demographic data (e.g. date of birth, sex, ethnic group); anthropometric characteristics (e.g. weight, height); diabetes history (e.g. year of diagnosis, type of diabetes); risk factors for complications (e.g. smoking status, glycaemic control, blood pressure, lipids, foot care, microalbuminuria/proteinuria); treatment (e.g. medication and referrals); and diabetic tissue damage (e.g. blindness or retinopathy, leg amputations, end-stage renal failure [ESRF], myocardial infarction). For most items that reflect routine care, nurses record the most recent measurements that have been made during a 1-year audit period.
All general practitioners in the two districts were invited to participate in the audit. The data in this cross-sectional study covers all audits completed with time periods that finished between 1 January 2003 and 31 December 2003. During this time period, the practice populations of 205 general practitioners were audited. Although the audit covers both Type 1 and Type 2 diabetes, the results reported here includes only patients identified as having Type 2 diabetes.

All statistical analysis was done using EpiInfo™ Version 3.3 (Centre for Disease Control, 5 August 2004). All tests are two-tailed with p<0.05 taken as statistically significant. Differences in proportions between populations were tested using Chi-squared tests and ANOVA tests were used for testing for inequalities in population means.

Where significant p-values are recorded, they indicate that there is significant variation between ethnic groups (rather than one ethnic group being compared with another). We used linear and logistic regression to calculate odds ratios with 95% confidence intervals for outcomes in circumstances when we wished to control for age and gender.

A high urinary albumin:creatinine ratio was defined as greater than or equal to 2.5 mg/mmol for men and 3.5 mg/mmol for women. People who were recorded as having abnormal pulses, sensory change, foot deformities, or a history of foot ulcers are classified as having at-risk feet.

**Results**

**Completeness of audit**—During the period covered, audit nurses identified 8754 patients with Type 2 diabetes. However, of these, audits could be done on only 5917 patients (67.6% of the total). In 92% of identified patients who were not audited, the reason given for not auditing was patients having moved into or out of the practice during the audit period or being registered with the practice but not seen (Table 1).

<table>
<thead>
<tr>
<th>Reason</th>
<th>N</th>
<th>Proportion of all people with Type 2 diabetes identified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Administrative reasons</td>
<td>28</td>
<td>0.30%</td>
</tr>
<tr>
<td>Died during audit period</td>
<td>191</td>
<td>2.20%</td>
</tr>
<tr>
<td>Registered with practice but not seen in audit period</td>
<td>408</td>
<td>4.70%</td>
</tr>
<tr>
<td>Moved out of practice</td>
<td>473</td>
<td>5.40%</td>
</tr>
<tr>
<td>New to practice</td>
<td>1724</td>
<td>19.70%</td>
</tr>
<tr>
<td>Totally under specialist care</td>
<td>13</td>
<td>0.10%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2837</strong></td>
<td><strong>32.40%</strong></td>
</tr>
</tbody>
</table>

The proportion of identified people who were audited varied by ethnicity (Europeans 70.1%, Other Asians 63.8%, Māori 64.6%, Pacific people 65.6%, Indians 66.9%, Others 70.0%; p<0.001). After adjusting for differences in age and gender between ethnic groups, Māori, Pacific, and Other Asians with Type 2 diabetes were still less likely than Europeans to have an audit completed (all p<0.001).

Table 2 shows that 82.6% of patients identified were Europeans, Māori or Pacific people. European patients were, on average, 10 years older than Māori and Pacific patients and were slightly more likely to be male. Patients had had diabetes for an average of 8 years.

**Number of diabetes consultations**—Audit nurses counted the number of consultations each person had related to their diabetes. This included any GP consultation with a component of diabetes care, and any practice nurse consultation where diabetes education was given. Patients had on average consulted their general practice for diabetes 5 times during the audit year. Māori and Pacific people had the...
highest mean number of diabetes consultations and Other Asian and Indian people the fewest (Table 3).

Table 2. Demographic details of the audited patients

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>N</th>
<th>% of total</th>
<th>Mean age at time of audit (SD)</th>
<th>Duration of diabetes in years (SD)</th>
<th>% male</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>2360</td>
<td>39.9%</td>
<td>65.8 (12.3)</td>
<td>8.9 (7.5)</td>
<td>52.9</td>
</tr>
<tr>
<td>Māori</td>
<td>898</td>
<td>15.2%</td>
<td>54.1 (12.1)</td>
<td>9.0 (7.2)</td>
<td>47.6</td>
</tr>
<tr>
<td>Pacific</td>
<td>1630</td>
<td>27.5%</td>
<td>55.8 (11.8)</td>
<td>7.2 (6.1)</td>
<td>42.6</td>
</tr>
<tr>
<td>Other Asian</td>
<td>257</td>
<td>4.3%</td>
<td>58.4 (12.9)</td>
<td>7.2 (5.8)</td>
<td>55.6</td>
</tr>
<tr>
<td>Indian</td>
<td>354</td>
<td>6.0%</td>
<td>55.5 (11.9)</td>
<td>8.6 (6.3)</td>
<td>52.0</td>
</tr>
<tr>
<td>Other</td>
<td>418</td>
<td>7.1%</td>
<td>62.0 (13.6)</td>
<td>8.1 (6.3)</td>
<td>57.4</td>
</tr>
<tr>
<td>Total</td>
<td>5917</td>
<td>100.0%</td>
<td>60.1 (13.3)</td>
<td>8.5 (6.9)</td>
<td>49.5</td>
</tr>
</tbody>
</table>

P value   <0.001   <0.001   <0.001

Table 3. Mean number of consultations for diabetes in the audit year

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Mean number of diabetes consultations (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>4.8 (2.9)</td>
</tr>
<tr>
<td>Māori</td>
<td>5.7 (4.1)</td>
</tr>
<tr>
<td>Pacific</td>
<td>5.4 (3.5)</td>
</tr>
<tr>
<td>Other Asian</td>
<td>4.1 (2.4)</td>
</tr>
<tr>
<td>Indian</td>
<td>4.2 (2.5)</td>
</tr>
<tr>
<td>Other</td>
<td>4.3 (2.2)</td>
</tr>
<tr>
<td>Total</td>
<td>5.0 (3.2)</td>
</tr>
</tbody>
</table>

P value   <0.001

Linear regression analysis was carried out to adjust for differences in age and gender composition between populations. Māori and Pacific people had a higher number of diabetes consultations than Europeans (Māori 1.2 more [95% CI 1.0–1.5], Pacific 0.8 more [95% CI 0.6–1.1] more). Other Asian and Indian people did not have significantly fewer consultations than Europeans in this analysis.

Recording of examinations and investigations and adverse risk factors—

Recording of significant examinations and investigations in patient notes was incomplete (Table 4). Whereas over 80% of all ethnic groups had a HbA1c, systolic blood pressure and total/HDL cholesterol ratio recorded in their notes, the recording of smoking status, body mass indices (BMIs), foot examinations, and urinary albumin:creatinine ratios was less complete and more variable. There were statistically significant differences in the recording of these latter items between ethnic groups.

Risk factors for macrovascular and microvascular complications are also shown in Table 4. For people who had smoking status recorded, 18% of all people, and 35% of Māori, were smokers. Over 50% of all people audited were obese, and very significant proportions of people had unsatisfactory blood pressure, lipid, and glycaemic control, and high urinary albumin:creatinine ratios.

A higher proportion of Māori and Pacific people had adverse risk factors in many categories. Exceptions were elevated systolic blood pressure and at-risk feet. Adjusting for age and gender using logistic regression showed that Pacific people, but not Māori or Indian, were less likely than Europeans to have a systolic blood pressure...
above 140 mmHg. Odds ratios for elevated systolic blood pressure were 1.05 (0.87–1.271) for Māori, 0.80 (0.67–0.94) for Pacific, and 0.85 (0.64–1.12) for Indian.
Table 4. Recording of examinations/investigations and adverse risk factors (percentages with 95% confidence intervals)

<table>
<thead>
<tr>
<th>Recorded in notes in audit year</th>
<th>European</th>
<th>Māori</th>
<th>Pacific</th>
<th>Other Asian</th>
<th>Indian</th>
<th>Other</th>
<th>Total</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking Status</td>
<td>81.0</td>
<td>84.9</td>
<td>84.2</td>
<td>73.2</td>
<td>77.1</td>
<td>65.1</td>
<td>80.8</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>79.3–82.5</td>
<td>82.3–87.1</td>
<td>82.4–86</td>
<td>67.3–78.5</td>
<td>72.4–81.4</td>
<td>60.3–69.6</td>
<td>79.7–81.8</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>73.7</td>
<td>78.3</td>
<td>82.5</td>
<td>68.1</td>
<td>76.0</td>
<td>62.2</td>
<td>75.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>71.9–75.5</td>
<td>75.4–80.9</td>
<td>80.5–84.3</td>
<td>62–73.7</td>
<td>71.2–80.3</td>
<td>57.3–66.8</td>
<td>74.8–77</td>
<td>0.000</td>
</tr>
<tr>
<td>HbA1c</td>
<td>88.7</td>
<td>85.6</td>
<td>87.1</td>
<td>87.5</td>
<td>88.7</td>
<td>87.1</td>
<td>87.6</td>
<td>0.243</td>
</tr>
<tr>
<td></td>
<td>87.3–89.9</td>
<td>83.1–87.8</td>
<td>85.3–88.6</td>
<td>82.9–91.3</td>
<td>84.9–91.8</td>
<td>83.4–90.1</td>
<td>86.7–88.4</td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>95.0</td>
<td>92.1</td>
<td>93.9</td>
<td>94.6</td>
<td>92.9</td>
<td>93.3</td>
<td>94.0</td>
<td>0.041</td>
</tr>
<tr>
<td></td>
<td>94.1–95.9</td>
<td>90.1–93.7</td>
<td>92.6–95</td>
<td>91–97</td>
<td>89.6–95.3</td>
<td>90.4–95.4</td>
<td>93.4–94.6</td>
<td></td>
</tr>
<tr>
<td>TC:HDL ratio</td>
<td>83.2</td>
<td>82.0</td>
<td>83.4</td>
<td>86.4</td>
<td>83.6</td>
<td>81.6</td>
<td>83.1</td>
<td>0.593</td>
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<tr>
<td></td>
<td>81.6–84.7</td>
<td>79.3–84.4</td>
<td>81.5–85.2</td>
<td>81.6–90.3</td>
<td>79.3–87.3</td>
<td>77.5–85.1</td>
<td>82.1–84.1</td>
<td></td>
</tr>
<tr>
<td>Foot Examination</td>
<td>59.3</td>
<td>60.5</td>
<td>60.4</td>
<td>49.8</td>
<td>50.8</td>
<td>54.3</td>
<td>58.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>57.3–61.3</td>
<td>57.2–63.7</td>
<td>58–62.8</td>
<td>43.5–56.1</td>
<td>45.5–56.2</td>
<td>49.4–59.1</td>
<td>57.2–59.8</td>
<td>0.000</td>
</tr>
<tr>
<td>Urinary albumin:creatinine ratio</td>
<td>65.6</td>
<td>68.2</td>
<td>74.3</td>
<td>66.5</td>
<td>66.4</td>
<td>59.6</td>
<td>68.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>63.6–67.5</td>
<td>65–71.2</td>
<td>72.1–76.4</td>
<td>60.4–72.3</td>
<td>61.2–71.3</td>
<td>54.7–64.3</td>
<td>66.8–69.2</td>
<td>0.000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Adverse risk factors (of those with records)</th>
<th>European</th>
<th>Māori</th>
<th>Pacific</th>
<th>Other Asian</th>
<th>Indian</th>
<th>Other</th>
<th>Total</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current smokers</td>
<td>13.1</td>
<td>34.9</td>
<td>17.8</td>
<td>17.6</td>
<td>7.7</td>
<td>17.3</td>
<td>18.0</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>11.6–14.7</td>
<td>31.5–38.4</td>
<td>15.9–20</td>
<td>12.4–23.8</td>
<td>4.8–11.5</td>
<td>13–22.3</td>
<td>17–19.2</td>
<td></td>
</tr>
<tr>
<td>BMI&gt;30</td>
<td>46.6</td>
<td>76.0</td>
<td>73.7</td>
<td>12.6</td>
<td>24.9</td>
<td>48.1</td>
<td>56.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>44.2–48.9</td>
<td>72.6–79</td>
<td>71.2–76</td>
<td>8–18.4</td>
<td>19.9–30.5</td>
<td>41.9–54.3</td>
<td>55.3–58.2</td>
<td>0.000</td>
</tr>
<tr>
<td>HbA1c&gt;8.0</td>
<td>22.7</td>
<td>49.5</td>
<td>55.7</td>
<td>30.2</td>
<td>44.9</td>
<td>25.0</td>
<td>37.6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>21–24.6</td>
<td>46–53.1</td>
<td>53–58.3</td>
<td>24.3–36.7</td>
<td>39.3–50.6</td>
<td>20.7–29.8</td>
<td>36.2–38.9</td>
<td>0.000</td>
</tr>
<tr>
<td>Systolic BP&gt;140 mmHg</td>
<td>32.6</td>
<td>27.6</td>
<td>23.7</td>
<td>28.4</td>
<td>24.0</td>
<td>36.4</td>
<td>29.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>30.7–34.6</td>
<td>24.6–30.8</td>
<td>21.6–26</td>
<td>22.8–34.5</td>
<td>19.6–29.1</td>
<td>31.7–41.4</td>
<td>27.8–30.2</td>
<td>0.000</td>
</tr>
<tr>
<td>TC:HDL ratio&gt;4.5</td>
<td>27.3</td>
<td>46.3</td>
<td>35.4</td>
<td>33.8</td>
<td>34.8</td>
<td>31.4</td>
<td>33.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25.4–29.3</td>
<td>42.7–50</td>
<td>32.9–38.1</td>
<td>27.6–40.4</td>
<td>29.4–40.5</td>
<td>26.5–36.6</td>
<td>32.1–34.8</td>
<td>0.000</td>
</tr>
<tr>
<td>At-risk feet</td>
<td>36.5</td>
<td>33.0</td>
<td>23.2</td>
<td>31.6</td>
<td>24.2</td>
<td>36.8</td>
<td>31.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>34.2–38.9</td>
<td>29.4–36.8</td>
<td>20.8–25.7</td>
<td>24.5–39.5</td>
<td>18.7–30.4</td>
<td>30.9–43</td>
<td>29.9–32.7</td>
<td>0.000</td>
</tr>
<tr>
<td>High urinary albumin:creatinine ratio</td>
<td>27.4</td>
<td>55.2</td>
<td>50.4</td>
<td>34.4</td>
<td>36.6</td>
<td>24.8</td>
<td>39.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25.4–29.5</td>
<td>51.5–58.8</td>
<td>47.8–53</td>
<td>28.1–41.2</td>
<td>31.1–42.5</td>
<td>20–30.2</td>
<td>37.9–40.7</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Note: Significant p-values indicate that there are significant differences between ethnic groups
Pacific people, but not Māori or Indian were less likely to have at-risk feet than Europeans (odds ratios 0.69 [0.57–0.84], 1.21 [0.96–1.51], and 0.76 [0.53–1.10] respectively).

**Pharmacologic management** - 41% of patients were on aspirin and 43% were on statins (Table 5). One-quarter of patients were on diet therapy alone whilst 17% were on insulin, either as a monotherapy or in combination with an oral hypoglycaemic.

Table 5. The percentages of patients on various therapies (95% confidence intervals)

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Aspirin</th>
<th>Two or more antihypertensives</th>
<th>Statin</th>
<th>Diet only</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>43.9 (41.9–45.9)</td>
<td>42.2 (40.2–44.2)</td>
<td>44.8 (42.8–46.8)</td>
<td>28.8 (27.0–30.7)</td>
<td>16.8 (15.3–18.4)</td>
</tr>
<tr>
<td>Māori</td>
<td>42.1 (38.8–45.4)</td>
<td>40.1 (36.9–43.4)</td>
<td>44.0 (40.7–47.3)</td>
<td>23.4 (20.7–26.3)</td>
<td>19.2 (16.7–21.9)</td>
</tr>
<tr>
<td>Pacific</td>
<td>37.3 (35–39.7)</td>
<td>26.7 (24.6–29)</td>
<td>39.3 (36.9–41.7)</td>
<td>17.9 (16.0–19.8)</td>
<td>17.9 (16.1–19.9)</td>
</tr>
<tr>
<td>Other Asian</td>
<td>31.1 (25.5–37.2)</td>
<td>23.3 (18.3–29)</td>
<td>38.1 (32.2–44.4)</td>
<td>27.2 (21.9–33.1)</td>
<td>8.2 (5.1–12.2)</td>
</tr>
<tr>
<td>Indian</td>
<td>40.7 (35.6–46.0)</td>
<td>30.2 (25.5–35.3)</td>
<td>43.8 (38.6–49.1)</td>
<td>19.8 (15.8–24.4)</td>
<td>19.8 (15.8–24.4)</td>
</tr>
<tr>
<td>Other</td>
<td>39.5 (34.8–44.4)</td>
<td>36.4 (31.8–41.2)</td>
<td>44.3 (39.5–49.2)</td>
<td>28.5 (24.2–33.1)</td>
<td>16.0 (12.7–20.0)</td>
</tr>
<tr>
<td>Total</td>
<td>40.7 (39.5–42.0)</td>
<td>35.7 (34.5–36.9)</td>
<td>42.8 (41.5–44.0)</td>
<td>24.3 (23.2–25.4)</td>
<td>17.2 (16.3–18.2)</td>
</tr>
</tbody>
</table>

**Note:** Significant p values indicate that there are significant differences between ethnic groups.

Table 6 shows the odds ratios for patients being on different therapies according to their ethnic group. Logistic regression was used to adjust for age and gender differences between groups. Māori are more likely to be on Aspirin and 2 or more antihypertensives than Europeans. Pacific people and Other Asians are less likely to be on 2 or more antihypertensives and Pacific people were less likely to be on statins than Europeans. Europeans were more likely to be on diet only therapy for glycaemic control than Māori, Pacific people, or Indians.

Table 6. Odds ratios for patient being on different therapies, adjusted for age and gender (95% confidence intervals)

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Aspirin</th>
<th>Two or more antihypertensives</th>
<th>Statin</th>
<th>Diet only</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Māori</td>
<td>1.49 (1.25–1.76)</td>
<td>1.54 (1.30–1.83)</td>
<td>1.05 (0.89–1.24)</td>
<td>0.79 (0.66–0.95)</td>
<td>1.16 (0.94–1.43)</td>
</tr>
<tr>
<td>Pacific</td>
<td>1.14 (0.99–1.31)</td>
<td>0.76 (0.65–0.87)</td>
<td>0.86 (0.75–0.99)</td>
<td>0.56 (0.47–0.65)</td>
<td>1.07 (0.89–1.27)</td>
</tr>
<tr>
<td>Other Asian</td>
<td>0.74 (0.56–0.99)</td>
<td>0.55 (0.40–0.74)</td>
<td>0.79 (0.61–1.03)</td>
<td>0.96 (0.72–1.28)</td>
<td>0.44 (0.28–0.70)</td>
</tr>
<tr>
<td>Indian</td>
<td>1.31 (1.03–1.66)</td>
<td>0.91 (0.71–1.17)</td>
<td>1.03 (0.82–1.29)</td>
<td>0.64 (0.48–0.84)</td>
<td>1.21 (0.91–1.61)</td>
</tr>
<tr>
<td>Other</td>
<td>0.94 (0.76–1.18)</td>
<td>0.90 (0.72–1.13)</td>
<td>1.00 (0.81–1.23)</td>
<td>1.01 (0.80–1.27)</td>
<td>0.95 (0.71–1.26)</td>
</tr>
</tbody>
</table>

**Discussion**

This study provides a description of the care provided and outcomes achieved for 5917 patients of different ethnicities attending South and West Auckland general practices. The population studied is similar in age and duration of diabetes to a large
community survey of diabetes that was undertaken in South Auckland, thus suggesting it is representative of the general population. Important information is provided about access to care, quality of care, and outcomes of care for these ethnic groups.

Māori and Pacific people (that were audited) visit their general practice at a rate that is greater than other ethnic groups. This finding is consistent with information from the general population which showed that Māori and Pacific people had a higher mean number of visits to GPs than Europeans, and it is reassuring given the known higher health needs of people with diabetes from these two ethnic groups.

It is also pleasing that general practice teams seem to perform important examinations and investigations on Māori and Pacific patients as frequently as they do on Europeans.

In contrast, Other Asian people (i.e. non-Indian Asians) do seem less likely to have some results recorded such as smoking status, BMI, and foot examinations. Whilst it is not possible from our results to say why this is, it may be due to cultural and/or language barriers or because Other Asians are perceived by GPs as a relatively low-risk group. We feel that recording of this information, whilst it could be improved, is well done. They compare with similar New Zealand audits previously reported and with a recent British study. For example, HbA1c was recorded in 88% of patients in this audit, in 90% of patients in Otago, 84% in North Canterbury, and 92% in United Kingdom. Similarly, foot examinations were recorded in 60% of patients in this audit, in 36% of patients in Otago, and 76% in North Canterbury, whilst foot pulses were checked in 53% of patients in United Kingdom.

There are large ethnic differences in risk factors for microvascular and macrovascular disease, with Māori and Pacific people being more likely to be at high risk in every category except for blood pressure measurements. Māori had very high rates of smoking as is seen in the general population. Māori and Pacific patients are much more likely to be obese than Europeans whilst Indian and Other Asian people are less likely to be so.

Again, these differences mirror the general population differences, although the rates of obesity for all ethnic groups in this study of people with diabetes are markedly higher than in equivalent groups in a general population survey. Māori and Pacific (and to a lesser extent Indian) people are also more likely to have high HbA1cs, cholesterol, and microalbuminuria. These large differences in risk factors are of great concern and undoubtedly contribute to poor outcomes. How much they can be attributed to medical care or to social, economic, and cultural differences between groups is uncertain however.

Just over 40% of patients were on statins and aspirin. Given that New Zealand guidelines recommend that most people with diabetes who are at high risk of cardiovascular disease (CVD) should be on these medications, there is room to increase the use of these medications.

More Māori were being treated with aspirin and two or more antihypertensives than European people (after controlling for age and gender). This is likely to be appropriate, since Māori are known to be at higher risk of serious complications of diabetes and high proportions of Māori had poor risk factors.
Pacific people are less likely to be on two or more antihypertensives than Europeans but they are also less likely to have raised blood pressure. However, the fact that they are also less likely to be on statins (despite being more likely to have elevated TC:HDL ratios) is concerning.

The major limitation of this paper is that it only provides information on a proportion of patients with Type 2 diabetes; those that have ongoing regular care in a general practice. It is known that around 6% of people with diabetes in South Auckland do not have ongoing care\textsuperscript{16} and that Māori are more likely to be in this group.

It is also noteworthy that people with diabetes from some ethnic groups were more likely to not be included in this study because they were not audited. The main reasons for not being audited were moving into or out of the practice or being lost to follow-up. It seems likely that these groups are less likely to have good continuity of care and may therefore be at greater risk of adverse outcomes.

Another limitation of this study is that statistical analysis was done assuming random sampling of individuals whereas in fact the sampling of individuals has been achieved through obtaining clusters of patients at the level of the general practice. This means that the p values and confidence intervals given are over precise although the fact that over 200 general practitioners patient were audited reduces this limitation.

A more significant limitation is that these GPs were not a random sample of the GPs in South and West Auckland but were instead those that self-selected themselves by agreeing to be audited. We have no way of knowing whether the care they provide is different from those GPs who did not participate, but it may be that GPs who are interested in participating in a quality process may provide better care than those that do not. Since many GPs have participated for some years we believe that the audit would have helped them achieve better standards of care than non-participating GPs. A further limit on generalising from this study is that care provided in West and South Auckland may not reflect care provided in other parts of New Zealand.

A difficulty in assessing care is that intensity of care and prescribing should be based upon clinical need. For example, it is known that Māori and Pacific people with diabetes have worse outcomes than European people with diabetes. It would therefore be appropriate if Māori and Pacific with diabetes were provided with a higher intensity of care to Europeans with diabetes. These differences in clinical need between ethnic groups are very difficult to take into account fully and it is therefore difficult to say whether a particular intensity of care for an ethnic group is appropriate or not.

In summary, most people who access their general practice team regularly for management of their diabetes seem to be receiving methodical review. There is some evidence that Māori patients are receiving more assertive care, which is appropriate to their risk. However, Māori and Pacific people with diabetes are not achieving the outcomes in risk factor management that other groups (particularly Europeans) are. There will be many reasons for this, some of which are outside the control of general practice teams. However, these risk factors will be contributing significantly to the poor long-term outcomes Māori and Pacific people with diabetes face.

Medical management with well-proven therapies is one important way that these poor outcomes need to be addressed. The challenge for primary care and the New Zealand
The health system is to ensure that all people with diabetes, particularly those groups who currently have poor outcomes, are appropriately supported to more aggressively manage their condition. Whilst national programmes such as Care Plus and Get Checked and local initiatives such as Counties-Manukau’s Chronic Care Management Programme will no doubt assist, there is clearly an urgent need for the health sector to address these issues.

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**References:**

Health status of New Zealand European, Māori, and Pacific patients with diabetes in 242 New Zealand general practices

Andrew Tomlin, Murray Tilyard, Alexander Dawson, Susan Dovey

Abstract

Objective To compare the care and health status of different ethnic groups attending general practices with diabetes.

Method We analysed information about 13,281 patients with any type of diabetes, collected by 242 general practices in the first visit of the Southlink Independent Practitioner Association’s Get Checked program. These patients constituted about 60% of patients with diabetes in the South Island of New Zealand.

Results 13,196 (99.4%) patients had Type 1 or Type 2 diabetes. Of these, 11,911 (90.3%) were Europeans, and 759 (5.8%) were Māori or Pacific Islanders (mostly of Samoan, Tongan, Niuean, or Cook Islands origin). There was no difference between ethnic groups in total cholesterol, proportions on oral therapy or statins, or having a foot check.

Māori and Pacific Islanders had poorer glycaemic control (HbA1c > 8.0 for 41.5% of Māori or Pacific Islanders versus 23.8% of New Zealand Europeans; 95% confidence interval for the difference [CI]: 14.0, 21.1), and were less likely to have retinopathy screening (71.9% versus 77.9%; CI: -9.2, -2.6). In patients with Type 2 diabetes (and compared with Europeans) Māori and Pacific Islanders were younger, had higher mean body mass indices (males 33.9 versus 29.5; CI: 3.9, 5.0 and females - 34.6 versus 30.7; CI: 3.2, 4.6) and diastolic blood pressures 82.4 mmHg versus 78.7 mmHg (CI: 2.9, 4.5), and were more likely to smoke (27.5% versus 10.9%; CI: 13.3, 19.9). Overall, Māori and Pacific Islanders were more likely to be at high risk for microvascular complications (9.0% versus 4.4%; CI: 2.5, 6.6).

Conclusions In this study, Māori and Pacific Island patients had a demographic profile suggesting greater health vulnerability (especially for those with Type 2 diabetes) yet similar routine diabetes care (especially for those with Type 1 diabetes). Ethnic inequalities were noted in seven of nine health status measures.

Implications The Get Checked program aims to increase the health of all patients with diabetes but whether it accentuates or diminishes ethnic disparities is not yet known.

Diabetes registers and practice-based research networks monitoring diabetes care have been established in New Zealand and many other countries with the aim of systematically monitoring the health status of patients with diabetes and supporting implementation of clinical guidelines for improved diabetes care.1–4

These registers are an essential component of quality improvement systems to minimise the burden of diabetes and its complications. Diabetes registers are established in many New Zealand general practices, and primary care organisations have recently supplemented practice registers with centralised rolls as a key...
component of the *Get Checked* quality programme launched by the New Zealand Ministry of Health in 2000.

The *Get Checked* program aims to monitor and continuously improve both care and outcomes for people with diabetes. Southlink Health (an independent practitioner organisation with 475 general practitioner members) launched *Get Checked* reviews in August 2000 for their own practices, and for any of the 22 general practices in the South Island that wished to participate but were not part of any primary care organisation.

Internationally, ethnic differences in the health status of patients with diabetes have been well documented.5–9 In New Zealand, previous research has shown that Māori and Pacific (i.e. mostly of Samoan, Tongan, Niuean, or Cook Islands origin) people are younger at diagnosis, are more likely to be obese, and have poorer glucose control and more end-stage renal failure and blindness than New Zealand Europeans. Indeed, in 1996, the prevalence of diabetes in Māori and Pacific people was nearly three times that of Europeans.10

For South Island general practitioners, these statistics seem remote—at the 2001 census, 14% of the New Zealand population were Māori but nearly 90% of Māori lived in the North Island; 6% of the New Zealand population were of Pacific Island origin, and 94% of Pacific Islanders lived in the North Island.

General practitioners in the South Island therefore see relatively few Māori or Pacific Island people among their patients.

In this descriptive analysis we report on the demography and health status of people with Type 1 and Type 2 diabetes when first registered in the Southlink Health database as part of the *Get Checked* program. Up to this point, all patients had received “routine” diabetic care. We aimed to determine whether there were differences in either the routine diabetic care patients had received or differences in health status measures between patients of New Zealand European, Māori, and Pacific ethnicity.

**Methods**

People listed on the diabetes registers of 242 general practices (N=16,557) were specifically invited to participate in the *Get Checked* programme by attending their practice for a free diabetes check. Reasons for non-response by 3276 patients (19.8%), shown in Table 1, were recorded in the *Get Checked* master database. At the initial check, patients gave written informed consent for the data used in this analysis to be sent to Southlink to be aggregated and used for population health research and practice feedback.

Data were collected from all 242 practices participating in the Southlink *Get Checked* program. Every practice in the South Island outside Christchurch city (the main urban centre), and 14 practices within Christchurch were involved. The total number of people on these practices’ diabetes registers represents 97.9% of the Ministry of Health’s estimates of the population prevalence of diagnosed diabetes.11 All records of the first *Get Checked* review (occurring between August 2000 and May 2003) were examined.

A doctor or practice nurse entered demographic and clinical data concerning the patient and their diabetes measures on standard paper data collection forms. Demographic data included sex, date of birth, and self-identified ethnicity. Patients’ height and weight were also recorded but whether they were measured or self-reported is not known as there were no specific instructions to providers in this regard.

Body mass index (BMI) was calculated as weight (kg) divided by height (cm) squared. Clinical data included type of diabetes; year of diagnosis; smoking history; blood pressure; the latest glycosylated
haemoglobin (HbA1c) level recorded within the previous 6 months; albumin/creatinine ratio; fasting total cholesterol, triglycerides and high density lipoprotein (HDL) levels; diabetic therapy; and whether the patient was taking an angiotensin converting enzyme inhibitor (ACE inhibitor), HMG-CoA reductase inhibitor (statin), or medication other than a statin to control lipids.

It also included details of the last foot check, and the most recent retinal screening or ophthalmologist examination. We adjusted for wrong assignment to the Type 1 group by reassigning to the Type 2 group those reported Type 1 patients who were not treated with insulin.

Incomplete forms were followed up with the relevant practice. Consultations for the Get Checked program were free for patients. The Ministry of Health paid general practitioners for this consultation, instead of the usual payment made by patients. Payment was not made if the form was not completed as far as possible.

The European Diabetes Policy Group Guidelines were used to classify patients as being either “low” or “high” risk for microvascular complications. Patients with cholesterol <4.8 mmol/L, triglycerides <1.7 mmol/L and HbA1c <6.5% were classified as at low risk. Those with cholesterol >6.0 mmol/L, triglycerides >2.2 mmol/L and HbA1c >7.5% were classified at high risk.

Female patients with an albumin:creatinine ratio ≥3.5 mg/mmol and male patients ≥2.5 mg/mmol were classified as having microalbuminuria. The target blood pressure (<140/80 mmHg) was based on the targets in the Scottish Intercollegial Guidelines Network evidence-based guideline for the management of diabetes. Data from patients reported to have Type 1 diabetes were reassigned to the Type 2 group if their reported diabetes treatment was “diet alone” or “oral medication alone” (N = 44; 3.7% of the reported Type 1 group). Data from patients of Māori and Pacific ethnicity were combined in the analysis due to the small number of Pacific people in the register and the similar clinical characteristics of patients with diabetes in the two ethnic groups.

The analytic approach was first to describe the Get Checked database, including patients with all types of diabetes. We then focused on patients with Type 1 and Type 2 diabetes only, and we specifically studied patients of New Zealand European, Māori, or Pacific Island ethnicity (excluding data from patients of other or unlisted ethnicity).

We framed the analysis according to the three categories:

- **Demography** (“given” or patient-controlled characteristics);
- **Care delivered** (diabetes-related health care provided by general practitioners); and
- **Health status** (measures of health attributable at least in part to a combination of “demography” and “care delivered”).

Statistical comparisons between ethnic groups used the Chi-squared test for differences in proportions for categorical data and unpaired-t or Mann-Whitney tests for continuous data.

**Results**

Mainly because of inter-practice transfers and deaths (Table 1), 19.8% of patients with diabetes listed in the diabetes registers of the 242 study general practices did not have a first Get Checked annual review. The first Get Checked review assessed 13,281 people. Ninety percent of patients (12,000) had Type 2 diabetes and 9% (1196) had Type 1 diabetes. In addition, 12 patients had gestational diabetes and 73 patients had diabetes of ‘other’ type (including unconfirmed diabetes).

Most patients indicated they were New Zealand Europeans (11,993; 90.3%), with smaller proportions of Māori (624; 4.7%) and Pacific (136; 1.0%) patients. A further 314 (2.4%) were of ‘other’ ethnicity and 214 (1.6%) declined to provide ethnicity details. Ethnicity data indicating their eligibility for this analysis was provided by 11,553 (96.3%) patients with Type 2 diabetes and 1117 (93.4%) patients with Type 1 diabetes. This last group of patients defined the study group for this analysis (Table 2).
Table 1. Patients with diabetes listed in practices’ diabetes registers but not reviewed in the Get Checked program

<table>
<thead>
<tr>
<th>Reason not reviewed</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transferred to another practice</td>
<td>1333</td>
</tr>
<tr>
<td>Deceased</td>
<td>913</td>
</tr>
<tr>
<td>Patient residing at rest home</td>
<td>420</td>
</tr>
<tr>
<td>Declined</td>
<td>193</td>
</tr>
<tr>
<td>Patient seen by specialist clinic only</td>
<td>161</td>
</tr>
<tr>
<td>Contacted – no response</td>
<td>135</td>
</tr>
<tr>
<td>Blood sugar monitoring only</td>
<td>93</td>
</tr>
<tr>
<td>Terminally ill</td>
<td>28</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>3276</strong></td>
</tr>
</tbody>
</table>

**Demography**—Māori and Pacific patients with Type 2 diabetes were significantly younger than New Zealand Europeans (mean age for Māori = 56.8 years versus 66.7 years for New Zealand Europeans; p<0.001). Figure 1 shows age distributions by ethnicity and diabetes type.

Figure 1. Diabetes patients by age group and ethnicity

![Age distribution chart](chart.png)

Note: Age distribution of Type 1 Māori and Pacific Island patients not included because of small numbers

The small number of Māori and Pacific people with Type 1 diabetes in our population (47) precludes the calculation of meaningful age distributions for this study group. Among patients with Type 1 diabetes, there was no significant difference in age between the two study groups, but New Zealand Europeans had more years on average with diagnosed Type 1 diabetes (p<0.01).
Table 2. Demography and care delivered by diabetes type and ethnicity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Type 1 diabetes (N=1,117)</th>
<th>Type 2 diabetes (N=11,553)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NZ European (N=1070) Māori/Pacific (N=47)</td>
<td>NZ European (N=10,841) Māori/Pacific (N=712)</td>
</tr>
<tr>
<td>Demography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age in years (SD)</td>
<td>42.4 (17.6) 38.4 (18.7) (-1.1, 9.2)</td>
<td>66.7 (12.0) 56.8 (11.6) (9.1, 10.9)</td>
</tr>
<tr>
<td>Sex M/F (%)</td>
<td>55.5/44.5 51.1/48.9 (-10.2, 19.0)</td>
<td>50.2/49.8 52.7/47.3 (-1.3, 6.3)</td>
</tr>
<tr>
<td>Years with Diabetes (SD)</td>
<td>19.7 (13.8) 13.0 (10.8) (2.6, 10.7)</td>
<td>7.0 (7.3) 6.9 (7.2) (-0.5, 0.6)</td>
</tr>
<tr>
<td>Smokers (%)</td>
<td>19.9 25.5 (-7.1, 18.3)</td>
<td>10.9 27.5 (-13.3, -19.9)</td>
</tr>
<tr>
<td>BMI (SD) Males</td>
<td>25.5 (4.2) 26.3 (4.3) (-3.9, -1.1)</td>
<td>29.5 (5.0) 33.9 (6.0) (-5.0, -3.9)</td>
</tr>
<tr>
<td>Females</td>
<td>26.8 (6.2) 28.0 (6.3) (-5.0, 0.8)</td>
<td>30.7 (6.6) 34.6 (6.9) (-4.6, -3.2)</td>
</tr>
<tr>
<td>Care delivered</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes therapy (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin only</td>
<td>97.5</td>
<td>93.6</td>
</tr>
<tr>
<td>Insulin + oral meds</td>
<td>2.5 6.4</td>
<td>3.9 3.7</td>
</tr>
<tr>
<td>Oral meds only</td>
<td>0.0 0.0</td>
<td>48.0 61.3</td>
</tr>
<tr>
<td>Diet only</td>
<td>0.0 0.0</td>
<td>p=0.11 37.3 24.2 p&lt;0.001</td>
</tr>
<tr>
<td>ACE inhibitor use (%)</td>
<td>29.3 42.6 (-1.2, 27.6)</td>
<td>46.6 51.5 (-1.1, -8.7)</td>
</tr>
<tr>
<td>Statin use (%)</td>
<td>12.1 12.8 (-9.1, 10.4)</td>
<td>19.2 16.2 (0.2, 5.8)</td>
</tr>
<tr>
<td>Foot check (%)</td>
<td>94.1 87.2 (-2.8, 16.5)</td>
<td>95.1 93.0 (0.2, 4.0)</td>
</tr>
<tr>
<td>Retinal exam (%)</td>
<td>87.2 76.6 (-1.7, 22.9)</td>
<td>77.0 71.6 (2.0, 8.8)</td>
</tr>
</tbody>
</table>

*For the difference in proportions or mean difference between ethnic groups.
Table 3. Health status measures by diabetes type and ethnicity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Type 1 diabetes</th>
<th>Type 2 diabetes</th>
<th>(95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NZ European (N=1070)</td>
<td>Māori/Pacific (N=47)</td>
<td>(95% CI)*</td>
</tr>
<tr>
<td></td>
<td>NZ European (N=10,841)</td>
<td>Māori/Pacific (N=712)</td>
<td>(95% CI)*</td>
</tr>
<tr>
<td>HbA1c % (SD)</td>
<td>8.6 (1.8)</td>
<td>9.2 (1.7)</td>
<td>-1.2, -0.2</td>
</tr>
<tr>
<td>≤ 7.2 (%)</td>
<td>20.7</td>
<td>12.8</td>
<td>p &lt; 0.01</td>
</tr>
<tr>
<td>&gt; 7.2, ≤ 8.0 (%)</td>
<td>24.5</td>
<td>8.5</td>
<td></td>
</tr>
<tr>
<td>&gt; 8.0, ≤ 9.0 (%)</td>
<td>24.0</td>
<td>27.7</td>
<td></td>
</tr>
<tr>
<td>&gt; 9.0 (%)</td>
<td>30.7</td>
<td>51.1</td>
<td></td>
</tr>
<tr>
<td>Blood pressure (mmHg) (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>127.0 (19.2)</td>
<td>123.3 (17.0)</td>
<td>-2.0, 9.3</td>
</tr>
<tr>
<td>Diastolic</td>
<td>73.6 (10.1)</td>
<td>73.0 (12.0)</td>
<td>-2.4, 3.6</td>
</tr>
<tr>
<td>BP &lt; 140/80 (%)</td>
<td>49.7</td>
<td>54.3</td>
<td>-10.0, 19.4</td>
</tr>
<tr>
<td>Albumin/creatinine (mg/mmol)</td>
<td>9.3 (36.4)</td>
<td>28.8 (85.5)</td>
<td>-46.2, 7.3</td>
</tr>
<tr>
<td>Males &gt; 2.5 (%)</td>
<td>27.8</td>
<td>33.3</td>
<td>-14.8, 26.2</td>
</tr>
<tr>
<td>Females &gt; 3.5 (%)</td>
<td>24.8</td>
<td>47.6</td>
<td>-1.0, -44.5</td>
</tr>
<tr>
<td>Cholesterol mmol/L (SD)</td>
<td>5.2 (1.1)</td>
<td>5.3 (1.3)</td>
<td>-0.0, 0.3</td>
</tr>
<tr>
<td>&lt;4.8 (%)</td>
<td>37.6</td>
<td>34.1</td>
<td></td>
</tr>
<tr>
<td>&gt;4.8, &lt;6.0 (%)</td>
<td>42.6</td>
<td>48.8</td>
<td></td>
</tr>
<tr>
<td>&gt;6.0 (%)</td>
<td>19.8</td>
<td>17.1</td>
<td>p = 0.73</td>
</tr>
<tr>
<td>HDL mmol/L (SD)</td>
<td>1.5 (0.5)</td>
<td>1.4 (0.5)</td>
<td>0.0, 0.3</td>
</tr>
<tr>
<td>Triglycerides mmol/L</td>
<td>1.2 (0.8)</td>
<td>1.6 (1.4)</td>
<td>-8.8, 0.1</td>
</tr>
<tr>
<td>&lt;1.7 (%)</td>
<td>80.9</td>
<td>73.2</td>
<td>-6.0, 21.5</td>
</tr>
</tbody>
</table>

*For the difference in proportions or mean difference between ethnic groups.
Smoking was more common among Māori and Pacific patients with Type 2 diabetes (27.5% versus 10.9%; p<0.001), but at similar levels for both study group patients with Type 1 diabetes. Māori and Pacific males with Type 1 diabetes, and males and females with Type 2 diabetes, had significantly higher BMIs than New Zealand Europeans of the same sex and diabetes Type (p<0.001).

**Care delivered**—Diabetes treatment for 36.5% of Type 2 patients consisted of diet only, 48.8% were treated with oral hypoglycaemics, 10.8% were on insulin, and a further 3.9% on insulin and an oral hypoglycaemic agent.

Māori and Pacific Island Type 2 patients were less likely than New Zealand Europeans to be treated by diet alone (24.2% versus 37.3%; p<0.001), but correspondingly more likely to be on oral medication without insulin (61.3% versus 48.0%; p<0.001). There were no significant differences between study groups in treatment of Type 1 diabetes although 6.4% of Māori and Pacific Islanders, and 2.5% of New Zealand Europeans, were on oral medications as well as insulin (p= 0.11).

Significantly more New Zealand Europeans than Māori and Pacific Islanders with Type 2 diabetes had both foot checks (95.1% of New Zealand Europeans and 93.0% of Māori and Pacific Islanders; p<0.05) and retinal examinations (77.0% of New Zealand Europeans and 71.6% of Māori and Pacific Islanders; p<0.001), but similar proportions of both study group patients with Type 1 diabetes received these checks.

More Māori and Pacific Islanders with Type 2 diabetes used ACE inhibitors to control blood pressure and/or nephropathy risk than New Zealand Europeans (51.5% versus 46.6%; p= 0.01), but more New Zealand Europeans were prescribed statins to control blood lipid concentrations (19.2% versus 16.2%; p=0.05). There was no difference between study groups of patients with Type 1 diabetes in ACE inhibitor or statin use.

**Health status**—Mean HbA1c was 8.6% (standard deviation, 1.9) for all Type 1 patients and 7.3% (standard deviation, 1.5) for Type 2 patients (Table 3). Māori and Pacific Island patients were less likely to achieve satisfactory glycaemic control (HbA1c ≤8.0%) for patients with both Type 1 (21.3% versus 45.2%; p=0.01) and Type 2 diabetes (61.0% versus 79.4%; p<0.001). Mean HbA1c decreased with age for both Type 1 and Type 2 diabetics (Figure 2).

Mean systolic blood pressure for Type 1 and Type 2 patients with diabetes was 127 mmHg and 141 mmHg respectively, and mean diastolic blood pressure 74 mmHg and 79 mmHg respectively. Similar proportions of New Zealand European, Māori, and Pacific Islanders with Type 1 diabetes (p=0.53) had blood pressure readings of less than 140/80 mmHg. Amongst Type 2 patients, there were more New Zealand Europeans with blood pressure less than 140/80 mmHg (p<0.05).

Mean total cholesterol, HDL, and triglyceride levels for patients with diabetes were 5.4 mmol/L, 1.30 mmol/L, and 1.93 mmol/L. Acceptable levels of total cholesterol (<4.8 mmol/L) were recorded for 27.5% of all patients, with no difference between study groups.

Within the previous 6 months, 42.2% of Māori and Pacific females and 58.2% of Māori and Pacific males with Type 2 diabetes had an albumin:creatinine result suggestive of microalbuminuria, compared with 26.0% of New Zealand European females and 35.1% of New Zealand European males (p<0.001 for all comparisons).
Figure 2. Mean HbA1c by age group

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Type 1 European</th>
<th>Type 2 European</th>
<th>Type 2 Māori/Pacific</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-29</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40-49</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50-59</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60-69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70+</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Distribution for Type 1 Māori and Pacific patients not included because of small numbers

6.5% of all New Zealand European patients (779), and 2.5% of Māori and Pacific patients (19) met the criteria for low risk of microvascular complications; 4.6% of European patients (549) and 9.0% of Māori and Pacific patients (68) met the criteria for high risk (p<0.001 for both comparisons).

Discussion

In this analysis of measures of health status and diabetes-related care provided by general practices in the South Island of New Zealand to a large cohort of patients with diabetes, we found the demography of Māori and Pacific Islanders indicated greater vulnerability to poor health in three out of four measures for patients with Type 2 diabetes and in one out of four measures for patients with Type 1 diabetes.

Five indicators of care affirmed a non-discriminatory approach for patients with Type 1 diabetes, but the reverse for patients with Type 2 diabetes. Seven of nine measures of health status indicated poorer health among Māori and Pacific Islanders, especially for patients with Type 2 diabetes.

These results are consistent with earlier research showing that Māori people with diabetes are younger, more likely to be obese, and more likely to smoke than New Zealand Europeans with diabetes. This study adds to existing knowledge about
diabetes in New Zealand by providing up-to-date information about a large cohort of South Island general practice patients with diabetes.

Type 2 Māori and Pacific patients were more likely than Type 2 New Zealand Europeans to be overweight, to smoke, have a higher HbA1c, have a higher diastolic blood pressure, and higher triglyceride levels. There was some evidence of lower systolic blood pressure but total cholesterol was not significantly different.

The difference in diastolic blood pressure (mean of 82.4 vs 78.7 mmHg respectively) may not be of clinical significance, and smoking prevalence is likely to be the greatest absolute population-health contribution of the risk factors derived from Framingham.\textsuperscript{14}

A single albumin:creatinine ratio result cannot be used to establish the diagnosis of microalbuminuria, but if confirmed by diagnostic investigations, these data highlight microalbuminuria as a relatively common condition in people with diabetes.

An albumin:creatinine ratio above the threshold for microalbuminuria was present in more than half of Māori/Pacific males and nearly half of Māori/Pacific females with Type 2 diabetes (compared with one-third and one-quarter respectively of New Zealand Europeans).

Cardiovascular mortality is increased by two- to four-fold in people with microalbuminuria, and about one-third of people with Type 2 diabetes and microalbuminuria die within 5 years. ACE inhibitors are recommended as first-line therapy in people with microalbuminuria, and this may explain why more ACE inhibitors overall were prescribed to Māori/Pacific people (51.0\%) than New Zealand Europeans (45.1\%).

A proportion of those not on ACE inhibitors may be on angiotensin II receptor blockers (offering equivalent renal protection). It is possible that the number of patients with microalbuminuria has been overestimated. Guidelines for management of diabetes disseminated to practitioners advised that albumin:creatinine ratios should be measured from early morning urine samples. However, no data were available as to the time of day at which samples were taken, and measurements may be higher for a patient if the sample is taken later in the day.

There are few published population health studies with which these results can readily be compared: few countries have primary care diagnosed diabetes databases with as comprehensive coverage of the population as the Southlink Get Checked database (sampling methods affect results from audit programs); there is little standardisation in clinical measures; and even recently published studies have used data collected prior to 2000.

The National Diabetes Register in Sweden,\textsuperscript{1} for example, used data from 1996–99. The Swedish Register included 29,769 individuals: for patients with Type 2 diabetes, mean HbA1c and blood pressures were 6.7\% and 147/80 mmHg in 1999—comparable with 7.2\% and 141/78 mmHg for New Zealand Europeans in the Southlink database.

The Mayo Health System Diabetes Translation Project reported the results of a community-based diabetes planned care program at three sites, supplemented at two of the sites by a comprehensive diabetes electronic management system.\textsuperscript{15} Performance measures (mainly completeness of care) were all substantially better for
the Southlink patients at this first annual review (baseline) than they were for the Mayo group even after 2 years of planned care and management by their electronic systems. However, similar measures of metabolic control at baseline were reported in this study and in the report by the Mayo project.

In New Zealand, the Otago Diabetes Team has monitored changes in diabetes care from 1998 to 2003 using an Otago Diabetes Register holding information on 3387 patients in the region. Their research indicates that implementation of guidelines to general practitioners for better management of diabetes care has resulted in significant improvements in patient outcomes, including blood pressure and lipid control.

Prevalence estimates for the denominator population of people with diabetes in South Island district health board areas were provided by the Ministry of Health. The diabetes cohort in this study constituted approximately 60% of all South Island people with diabetes, and 80% of patients with diagnosed diabetes after excluding diabetes patients not participating in the Southlink Get Checked program. However, the number of Māori/Pacific people with diabetes participating in the program represented less than 30% of South Island prevalence estimates for these ethnic groups, and the data may not be as representative of these populations as for New Zealand Europeans.

Our study has several important strengths. The diabetes patients in this study represent a large cohort by international standards. There were few missing data and data collection was standardised across practices. Study measures of care delivered and health status reflect the current 21st century position in the South of New Zealand, providing a baseline from which improvements might be measured.

As a result of this investigation, Southlink Get Checked clinical indicators are being updated to provide more focus on microalbuminuria, overt nephropathy, and diabetic renal disease as predictors of both renal failure and cardiovascular outcome.

Weaknesses in this study include the likelihood of some error in the assignment of patients to the Type 2 diabetes group. Wrongly assigned Type 1 patients could not be differentiated in the database from correctly assigned Type 2 patients being treated with insulin. We estimate the error associated with misassignment as small (less than 4%) and no Type 2 patients on diet or oral medications only, should be in the Type 1 group.

We may also have under-reported the number of Type 1 diabetes patients, as some of these may only receive care from hospital-based clinics. Collating data from multiple practice diabetes registers into the single Get Checked database revealed weaknesses in individual practices’ lists (such as the 1333 patients who had transferred to other practices and the 913 who had died) that might not otherwise have been known.

The small number of Māori and Pacific people living in the South Island means that we had to combine data from these two different ethnic backgrounds into one study group. Even so, this combined group was also small relative to the group of New Zealand European people with diabetes and in the analysis this may mask some real differences between the groups.

This analysis provides information on the characteristics, care, and health status of a large population of diabetic people in the South Island of New Zealand at the start of a Government-funded program aiming to improve both care and outcomes. It confirms
that ethnic disparities exist in this patient group, despite the small proportion of people from ethnic minority groups among these patients with diabetes. Future studies will use these data to measure the impact of the Get Checked programme on the care and health outcomes of patients with diabetes in the South Island and to assess changes in ethnic disparities attributable to the programme.

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**References:**


5. Davis ME, Cull CA, Holman RR. Relationship between ethnicity and glycaemic control, lipid profiles, and blood pressure during the first 9 years of Type 2 diabetes. Diabetes Care. 2001;24:1167–74.


The Dunedin Multidisciplinary Health and Development Study: are its findings consistent with the overall New Zealand population?

Richie Poulton, Robert Hancox, Barry Milne, Joanne Baxter, Kate Scott, Noela Wilson

Abstract

Aims To compare the health of the Dunedin Multidisciplinary Health and Development Study members with people of the same age in the nationally representative New Zealand Health and National Nutrition Surveys.

Method Where similar information was obtained, means or proportions and confidence intervals were generated for both the age 26 assessment of the Dunedin sample and for the 25–26 year old participants in the national surveys. The populations were considered to differ when confidence intervals did not overlap.

Results For smoking habit, body mass index, waist-hip ratio, general practitioner and medical specialist consultations, and hospital admissions, the findings of the Dunedin Study were not significantly different to the nationally representative surveys. The Dunedin Study members also did not differ from their national counterparts on SF-36 subscales measuring physical functioning, bodily pain, general health, vitality, and mental health. They had better scores on the three interference subscales of the SF-36 compared to the national sample, and men in the Dunedin Study spent a little more time doing vigorous physical activity.

Discussion For most outcomes, the Dunedin Study members were very similar to the nationally representative samples. There was little evidence that the repeated assessments in the Dunedin Study had significantly altered the Study members’ health, either in terms of responses to questionnaires or on physiological measures of health status. Findings from the Dunedin Study are likely to be generalisable to most young New Zealanders. However, the Dunedin Study is under-representative of Māori and Pacific peoples, so these findings need to be interpreted with caution in this context. Implications for the proposed national Longitudinal Study of New Zealand Children and Families are discussed.
health status of the Dunedin Study members with those participating in nationally representative surveys.

Two issues arise. First, are the Study members, who were all born in Dunedin, similar to other New Zealanders of the same age? Second, have the health behaviours of Dunedin Study members changed (due to being intensively studied throughout their lives) to the point where they are no longer representative of the original population from which they were drawn (the so-called “Hawthorne effect”). These are not trivial matters. Despite information to the contrary, misperceptions about Dunedin Study sample persist and at times they raise questions about the value of the Dunedin Study data for policy-making in the New Zealand context.

The Dunedin Study members are now 32 years old, and they are undergoing a further assessment as we prepare to study the positive and problematic aspects of the transition from young adulthood to mid-life. This represents an opportune time to revisit the question of whether the findings from the Dunedin Study are generalisable to other New Zealanders.

In addition, there is another reason to do this review now as the New Zealand Ministries of Social Development, Health, and Education as well as Treasury and The Families Commission are planning to embark upon a national Longitudinal Study of New Zealand Children and Families. Because multi-site studies tend to be more costly, logistically-demanding, and risk greater threats to internal validity (e.g. standardisation of procedures) than single (or perhaps two) site studies, knowledge about the generalisability of findings from regionally-based studies like the Dunedin Study may help to plan the optimal sampling strategy for the National cohort study.

To address these questions about generalisability, we directly compared the Dunedin Study members from their most recently completed assessment in 1998–1999 (when they were all aged age 26) to 25 and 26 year-olds participants in the cross-sectional New Zealand Health Survey in 1996/97 and the National Nutrition Survey in 1997. Comparisons were conducted wherever the same or very similar data were collected in the Dunedin Study and the national surveys.

Methods

Sample characteristics

Dunedin Study—This analysis involved 499 male and 481 female members who participated in the Dunedin Study assessment at age 26 years (mean age = 26.0 years, SD = 3 months). The background to the study and Study members are described in detail elsewhere. Briefly, the Dunedin Study is a longitudinal investigation of the health, development, and behaviour of 1037 children born in Queen Mary Maternity Hospital, Dunedin between April 1972 and March 1973. The sample has been assessed with a diverse array of medical, psychological, and sociological measures with high rates of participation at age 3 (n = 1037), age 5 (n = 991), age 7 (n = 954), age 9 (n = 955), age 11 (n = 925), age 13 (n = 850), age 15 (n = 976), age 18 (n = 993), age 21 (n = 992), and age 26 (n = 980, 96% of the living cohort). Seventy-three (7.5%) Study members self-identified as Māori and 15 (1.5%) as Pacific people at age 26.

The age-26 assessments took place at the Dunedin Unit between March 1998 and June 1999. A small number (27/980, 3%) of participants who were unable to attend the Unit were assessed in the field. The assessment took a full day lasting from 8.30am to 5.15pm and involved interviews and physical examinations.

Of those who participated at the age-26 assessment, 41% (404) were still resident in Dunedin at the time of interview, 21% (202) were resident in other parts of the South Island and 17% (168) were...
resident in the North Island. Hence, 774 (79%) were resident in New Zealand at the time of interview. Of the remainder, 11% (108) were resident in Australia, 7% (66) were resident in the United Kingdom, and 3% (32) were resident elsewhere.

**New Zealand Health Survey (“Health Survey”)**—The 1996/1997 Health Survey used a clustered stratified design based on geographic areas to obtain a sample with characteristics that were representative of the entire New Zealand civilian population. To obtain more reliable estimates for Māori and Pacific peoples, a proportionately greater sample of these ethnic groups was included. A total of 7862 adults (aged 15 years and over) participated, thus representing a 73.8% response rate. This analysis included the 292 respondents who were aged 25 or 26 at the time of the survey. Of these, 64 (21.9%) identified themselves as Māori and 34 (11.6%) as Pacific people.

**National Nutrition Survey (Nutrition Survey)**—At the conclusion of the Health Survey, participants were asked if they would undergo further assessment for the 1997 National Nutrition Survey. A total of 4636 adults completed the Nutrition Survey, of which 146 aged 25 or 26 years are included in this analysis. Twenty-eight (19.2%) identified themselves as Māori and 12 (8.2%) as Pacific people.

**Comparison measures**

**Self-reported health status**—For both the Dunedin Study and Health Survey samples, self-reported health status during the previous 12 months was measured by the Australian/New Zealand adaptation of the SF-36 survey—a 36-item questionnaire measuring eight aspects of health. These included physical functioning, role physical (the impact of physical health on performance of everyday roles), bodily pain, general health, vitality, social functioning, role emotional (the impact of emotional health on performance of everyday roles), and mental health. This instrument has been shown to be a reliable and valid measure of the health status of New Zealanders.

**Body size measurements**—The Dunedin Study and Nutrition Survey measured height without shoes and weight in light clothing to calculate body mass index (BMI) in kg/m$^2$. Waist and hip circumference were measured to calculate the waist:hip ratio—an index of central adiposity. All body size measurements in both the Dunedin Study and Nutrition Survey were taken twice.

**Physical activity**—Participants in the Dunedin Study were asked if they had done any physical activities that caused them to “breathe hard or puff a lot” in the past 4 weeks and, if so, how much time per week they spent doing these activities in a normal week. This was taken as the time spent per week doing vigorous physical activities. Vigorous physical activity in the Health Survey was taken as the time they reported that they had spent doing physical activities in the past 7 days that had made them “breathe hard or sweat”. This question was prompted by a list of likely activities.

**Smoking status**—In the Dunedin Study, those who currently smoked one or more cigarettes per day and had smoked daily for at least 1 month in the last year were deemed to be current smokers. Study members who were not current smokers but had smoked daily for as long as a year at some time in their lives were deemed to be ex-smokers. In the Health Survey, those who reported that they smoked one or more cigarettes daily were to be deemed current smokers. Ex-smokers were those who had smoked in the past but were not current smokers.

**Health service utilisation**—Dunedin Study members were asked whether and how many times they had used a general practitioner (GP), or a medical specialist (e.g., cardiologist, gastroenterologist, obstetrician/gynaecologist, urologist, orthopaedic surgeon, nephrologist, dermatologist, neurologist, ear, nose & throat specialist, ophthalmologist, respiratory specialist, oncologist, endocrinologist, rheumatologist) in the past year. Study members were also asked whether they had spent any time in hospital in the past year for a physical health (not mental health) problem. Participants in the NZ Health Survey were asked how many times they had visited a general practitioner in the past year, and were also asked if they had seen a medical specialist but were not prompted by a list of possible specialists.

**Statistical methods**—Comparisons were conducted between the Dunedin Study members and 25–26 year old participants in the Health and Nutrition Surveys. For all measures, either means (e.g. SF-36 scale scores) or prevalences (e.g. current smokers) are presented together with 95% confidence intervals (CIs).

Sample survey weights were applied for the Health Survey based on each individual’s probability of being selected for the survey to provide estimates consistent with the New Zealand population. The Dunedin Study was considered to be significantly different from either of the national samples on a measure if the 95% CIs of the samples did not overlap.
Results

Comparisons between the Dunedin Study and the Health and Nutrition Surveys are shown in the following Tables. Because not all participants in the studies consented to every assessment, the numbers included in the tables vary slightly.

Self-reported health status—There were no significant differences between the Dunedin Study and the Health Survey on SF36 subscales measuring physical functioning; bodily pain; general health; vitality; and mental health (Table 1). On the subscales measuring interference with physical and emotional task roles, members of the Dunedin Study scored better than their Health Survey counterparts. They also reported higher social functioning scores, indicating that they experienced less interference in social activities as a result of a physical or emotional problem.

Body size measures—The Dunedin Study and the Nutrition Survey participants were very similar on measures of Body Mass Index and waist:hip ratio (Table 2). These measures were also similar if the comparison was restricted to Māori [Dunedin Study: mean (95%CI) BMI 25.5 (24.6–26.6), mean waist:hip ratio 0.799 (0.784–81.5); Nutrition Survey: mean BMI 27.5 (23.7–31.3), mean waist:hip ratio 0.808 (0.756–0.861)]

Physical activity—Overall, there were no significant differences in the time spent doing vigorous activity in the participants in the Dunedin Study and the Health Survey (Table 3). However, more men in the Dunedin Study spent more than 300 minutes per week doing vigorous activity and fewer of the Dunedin Study men did no vigorous activity.

Smoking status—The Dunedin Study had a slightly greater proportion of current smokers (37.1% vs 33.0%), and a slightly lower proportion of ex-smokers (11.5% vs 16.6%) than the Health Survey, although neither of these differences were significant (Table 4). Approximately half of both samples had never smoked.

Health service use—Similar proportions of the Dunedin Study and the Health Survey had used a GP in the previous 12 months (78.6% and 76.5%, respectively) and were admitted as an inpatient in the previous 12 months (9.7% and 7.8%, respectively) (Table 5). A slightly, though not significantly, greater proportion of the Health Survey participants had used a medical specialist, as compared to the Dunedin Study (29.2% and 20.5%, respectively). There were no differences between the samples in terms of frequency of GP use.
Table 1. SF-36 Health Survey results. The mean and 95% confidence intervals are presented for men and women in the Dunedin Study at age 26 and 25-26 year-olds in the New Zealand Health Survey for each of the eight subscales. Higher scores represent better health. Significant differences between the Dunedin Study and Health Survey are highlighted in bold.

<table>
<thead>
<tr>
<th>SF36 scales</th>
<th>Dunedin Study</th>
<th>New Zealand Health Survey</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male (n=499)</td>
<td>Female (n=480)</td>
</tr>
<tr>
<td></td>
<td>Male (n=98)</td>
<td>Female (n=194)</td>
</tr>
<tr>
<td>Physical functioning</td>
<td>94.4 (93.5–95.4)</td>
<td>91.1 (89.9–92.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>89.9 (85.5–94.3)</td>
</tr>
<tr>
<td></td>
<td>Role physical</td>
<td>91.5 (89.3–93.6)</td>
</tr>
<tr>
<td></td>
<td>89.4 (87.7–91.1)</td>
<td>79.6 (65.8–93.4)</td>
</tr>
<tr>
<td></td>
<td>Bodily pain</td>
<td>80.1 (78.4–81.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>78.9 (77.6–80.2)</td>
</tr>
<tr>
<td></td>
<td>General health</td>
<td>77.5 (76.1–78.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>77.4 (76.3–78.4)</td>
</tr>
<tr>
<td></td>
<td>Vitality</td>
<td>68.8 (67.5–70.1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>65.5 (64.5–66.5)</td>
</tr>
<tr>
<td>Social functioning</td>
<td>90.8 (89.4–92.1)</td>
<td>87.7 (86.1–89.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>88.0 (83.3–92.6)</td>
</tr>
<tr>
<td></td>
<td>Role emotional</td>
<td>93.5 (91.6–95.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>91.3 (89.8–92.8)</td>
</tr>
<tr>
<td></td>
<td>Mental health</td>
<td>80.4 (79.3–81.5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>78.8 (77.9–79.6)</td>
</tr>
</tbody>
</table>

Table 2. Body Mass Index (BMI) and waist:hip ratio means (95% CI) for the Dunedin Study members at age 26, and for 25 & 26 year olds from the National Nutrition Survey. Data from pregnant women are excluded.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dunedin Study</th>
<th>National Nutrition Survey</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male (n=494)</td>
<td>Male (n=49)</td>
</tr>
<tr>
<td></td>
<td>Female (n=445)</td>
<td>Female (n=97)</td>
</tr>
<tr>
<td></td>
<td>All (n=939)</td>
<td>All (n=146)</td>
</tr>
<tr>
<td>BMI</td>
<td>25.2 (24.8–25.5)</td>
<td>25.7 (23.9–27.4)</td>
</tr>
<tr>
<td></td>
<td>n=489</td>
<td>n=47</td>
</tr>
<tr>
<td>Waist:hip ratio</td>
<td>0.849 (0.846–0.853)</td>
<td>0.860 (0.832–0.888)</td>
</tr>
<tr>
<td></td>
<td>n=489</td>
<td>n=47</td>
</tr>
</tbody>
</table>
Table 3. Time spent in vigorous activity during a typical week by the Dunedin Study members at age 26 and the 25–26 year olds in the New Zealand Health Survey (percentage and 95% CI of sample). Significant differences between the Dunedin Study and Health Survey are highlighted in bold.

<table>
<thead>
<tr>
<th>Time interval</th>
<th>Male (N=496)</th>
<th>Female (N=476)</th>
<th>All (N=972)</th>
<th>Male (N=98)</th>
<th>Female (N=194)</th>
<th>All (N=292)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>% (95% CI)</td>
<td>n</td>
<td>% (95% CI)</td>
<td>n</td>
<td>% (95% CI)</td>
</tr>
<tr>
<td>0 mins</td>
<td>138</td>
<td>27.8 (23.9–32.0)</td>
<td>183</td>
<td>38.4 (34.1–42.9)</td>
<td>321</td>
<td>33.0 (30.1–36.1)</td>
</tr>
<tr>
<td>&lt;150 mins</td>
<td>95</td>
<td>19.2 (15.8–22.9)</td>
<td>109</td>
<td>22.9 (19.2–26.9)</td>
<td>204</td>
<td>21.0 (18.4–23.7)</td>
</tr>
<tr>
<td>150–300 mins</td>
<td>110</td>
<td>22.2 (18.6–26.1)</td>
<td>128</td>
<td>26.9 (23.0–31.1)</td>
<td>238</td>
<td>24.5 (21.8–27.3)</td>
</tr>
<tr>
<td>&gt;300 mins</td>
<td>153</td>
<td>30.8 (26.8–35.1)</td>
<td>56</td>
<td>11.8 (9.0–15.0)</td>
<td>209</td>
<td>21.5 (19.0–24.2)</td>
</tr>
</tbody>
</table>

Table 4. Smoking status (percentage and 95% CI) of the Dunedin Study members at age 26 and the 25-26 year olds in the New Zealand Health Survey sample.

<table>
<thead>
<tr>
<th>Smoking status</th>
<th>Male (N=499)</th>
<th>Female (N=481)</th>
<th>All (N=980)</th>
<th>Male (N=98)</th>
<th>Female (N=194)</th>
<th>All (N=292)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>% (95% CI)</td>
<td>n</td>
<td>% (95% CI)</td>
<td>n</td>
<td>% (95% CI)</td>
</tr>
<tr>
<td>Never smoked</td>
<td>266</td>
<td>53.3 (48.8–57.8)</td>
<td>241</td>
<td>50.1 (45.5–54.7)</td>
<td>507</td>
<td>51.7 (48.6–54.9)</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>54</td>
<td>10.8 (8.2–13.9)</td>
<td>64</td>
<td>13.3 (10.4–16.7)</td>
<td>118</td>
<td>12.0 (10.0–14.2)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>179</td>
<td>35.9 (31.7–40.3)</td>
<td>176</td>
<td>36.6 (32.3–41.1)</td>
<td>355</td>
<td>36.2 (33.2–39.3)</td>
</tr>
</tbody>
</table>

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Table 5. Twelve-month health service use (percentage and 95% CI of sample) by the Dunedin Study members at age 26 and the 25–26 year olds in the New Zealand Health Survey

<table>
<thead>
<tr>
<th>Health service use</th>
<th>Dunedin Study</th>
<th></th>
<th></th>
<th>New Zealand Health Survey</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male (N=498)</td>
<td>Female (N=479)</td>
<td>All (N=977)</td>
<td>Male (N=98)</td>
<td>Female (N=194)</td>
<td>All (N=292)</td>
</tr>
<tr>
<td>Used GP</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Used specialist</td>
<td>331</td>
<td>66.5 (62.1–70.6)</td>
<td>429</td>
<td>89.6 (86.5–92.3)</td>
<td>760</td>
<td>77.8 (75.2–80.4)</td>
</tr>
<tr>
<td>Admitted as inpatient</td>
<td>39</td>
<td>7.8 (5.6–10.6)</td>
<td>55</td>
<td>11.5 (8.8–14.8)</td>
<td>94</td>
<td>9.6 (7.8–11.4)</td>
</tr>
<tr>
<td>Used GP once</td>
<td>136</td>
<td>41.1 (35.7–46.4)</td>
<td>66</td>
<td>15.4 (12.1–19.2)</td>
<td>202</td>
<td>26.6 (23.5–29.9)</td>
</tr>
<tr>
<td>Used GP twice</td>
<td>74</td>
<td>22.4 (18.0–27.2)</td>
<td>104</td>
<td>24.2 (20.3–28.6)</td>
<td>178</td>
<td>23.4 (20.5–26.6)</td>
</tr>
<tr>
<td>Used GP 3–5 times</td>
<td>81</td>
<td>24.5 (19.9–29.5)</td>
<td>149</td>
<td>34.7 (30.2–39.4)</td>
<td>230</td>
<td>30.3 (27.0–33.7)</td>
</tr>
<tr>
<td>Used GP 6–11 times</td>
<td>27</td>
<td>8.2 (5.4–11.6)</td>
<td>72</td>
<td>16.8 (13.4–20.7)</td>
<td>99</td>
<td>13.0 (10.7–15.6)</td>
</tr>
<tr>
<td>Used GP &gt;11 times</td>
<td>13</td>
<td>3.9 (2.1–6.6)</td>
<td>38</td>
<td>8.9 (6.3–12.0)</td>
<td>51</td>
<td>6.7 (5.0–8.7)</td>
</tr>
</tbody>
</table>

* Data from two members of the Health Survey sample not available
Discussion

Dunedin Study members were similar to their age matched peers in the national Health and Nutrition samples on most of the health measures we compared. This included five of the eight subscales of self-reported health status from the SF36; smoking behaviour; physical activity; two physical measurements (BMI and waist:hip ratio); and use of general practice and specialist health services.

There were significant differences between the Dunedin Study members and the nationally representative samples on three of the eight subscales of the SF36. The SF36 is a widely-used, validated, and reliable instrument that provides a multidimensional assessment of health. The 8 subscales measure physical, emotional, and social factors and the SF36 is used to provide a reasonable overall assessment of a person’s health in the context of large Health Surveys. However, despite its usefulness, the SF36 remains a self-report measure and has the accompanying limitations. For example, reports can be confounded by mood or certain personality traits.9 The three subscales on which the Dunedin Study members differed from their peers in the Health Survey were “role physical”, “social functioning”, and “role emotional” (only differed in women).

Interestingly, the Dunedin Study members tended to score higher (better health) on these scales than the participants in the Health Survey. If the repeated interviews of the Dunedin cohort had altered their perception of their health (the “Hawthorne effect”), we might have predicted that they would become more sensitised to their health problems. In fact, the Dunedin Study members reported less interference in their roles than participants in the national studies. It is possible that these minor differences arose because of a selection bias of more health-focussed individuals among the 74% of people who agreed to participate in the Health Survey.

For some measures, slightly different methodologies were used by the Dunedin Study and the national surveys. For example, members of the Dunedin sample were considered smokers if they had smoked one cigarette a day for at least a month of the previous year AND they currently smoked at least one cigarette a day—whereas members of the Health Survey were only asked if they currently smoked at least one cigarette a day (they need not have smoked for a month). Also, members of the Health Survey weren’t required to have smoked for a year to be considered ex-smokers. Despite this, the proportions of smokers and ex-smokers in the samples were quite similar. For the health service utilisation measures, the methods used by both studies seem comparable except that members of the Dunedin sample were given a checklist of specialists who they may have visited in the previous year—whereas members of the Health Survey were not. Similarly the physical activity measures were similar, although worded slightly differently and the Health Survey respondents were shown a checklist of activities. Taken together these measures indicate that the samples are broadly comparable in terms of health-risk behaviours and lifestyle factors (smoking and physical activity) and health problems indexed by health service use. It is notable that on the comparison of the two objective physical health markers (body mass index and waist:hip ratio), the Dunedin Study members and the National Nutrition sample were almost identical.

Have we changed people? It would appear not. Participants in the Dunedin Study look the same as research-naïve participants in the national studies in almost all respects.
These findings are consistent with an earlier study comparing respiratory symptoms among Dunedin Study participants at age 21 with those of 20–22-year-old participants in the New Zealand section of the European Community Respiratory Health Study. This was a once-only postal questionnaire conducted in Auckland, Wellington, Christchurch, and Hawke’s Bay which used virtually identical questions to those used in the Dunedin Study. There was no difference in the prevalence rates of any of the reported symptoms or asthma medication-use between the samples. Thus, in two comparisons we have found little evidence that the health status of our study members has been altered by virtue of their involvement in a longitudinal study.

These findings have several implications for planning of future cohort studies, including the New Zealand Longitudinal Study of Children and Families. The first relates to the need to distinguish between ‘representativeness’ and ‘generalisability’. Classically, representativeness refers to sampling methods that faithfully represent all members of the target population (in a New Zealand nationally representative study this would mean the whole of the country), whereas generalisability refers to the ability to extrapolate findings to the wider population, despite imperfect representativeness. Deriving a sample that is perfectly representative of the major population groups of interest (in terms of socioeconomic status and geographic location for example) is resource intensive and costly. Moreover, for a longitudinal study, generalisability is more important than representativeness. By its nature, a cohort study cannot remain truly representative of the population of interest. Thus, although the Dunedin Study sample appears to be broadly representative of New Zealand children born in 1972/1973, they will not necessarily be representative of children born in 1992 or 2002. Nevertheless, it is today’s New Zealand children that are most likely to benefit from the lessons that we have learned from the Dunedin Study. The value of a cohort study is that it provides a means of testing hypotheses about the importance of early influences and the sequence of events in growth and development. Unless there are good reasons to suspect otherwise, the findings are likely to be generalisable to other people in similar circumstances.

Second, a potentially greater threat to study validity is from non-random loss to follow-up. In the context of a new national cohort study, a strong argument can be made for resources being spent on maintaining cohort retention, and ensuring high quality measurements, especially if the generalisability of findings to the wider population from a single site can be demonstrated, as appears to be the case here. In support of this argument, Youth 2000, a nationwide survey of health and wellbeing amongst New Zealand secondary school students has been analysed by region. Although there were minor differences between the 15 regions, the conclusions drawn about health, risk behaviour, and health service needs for each region were identical. The findings provide broad support for the generalisability of findings from the Dunedin Study (and by implication similar studies such as the Christchurch Health & Development Study) to other New Zealanders. However, the cross-sectional comparisons presented here risk underplaying the ways in which longitudinal designs enhance generalisability compared to cross-sectional studies. For example, prospective-longitudinal studies provide better estimates of lifetime exposures than cross-sectional, retrospective studies.

Longitudinal studies also permit casual inferences about a range of exposures and outcomes, and it is this information that is most useful for policy-making.
regard, it is noteworthy that both the Dunedin and Christchurch longitudinal studies produce highly replicable findings in the international context, which given the similarities between countries such as Australia, USA, Canada, and the UK is perhaps not surprising. Indeed it is precisely this generalisability that has resulted in significant investment in the Dunedin Study by the U.S. National Institutes of Health, and more recently by the UK Medical Research Council.

However, there are some limitations to our findings, particularly in the capacity of this analysis to inform on the generalisability of findings to specific ethnic groups (Māori, Pacific, and European/Other). We have not been able to examine whether health outcomes within specific ethnic groups (Māori, Pacific, European/Other) are comparable between the Dunedin Study and the national surveys. Summary data for the individual ethnic groups in the New Zealand Health Survey for this age-group were not available. There may also be limitations due to small numbers of Māori and Pacific ethnic groups in both the Dunedin Study and in the national surveys, which may reduce the precision of estimates making statistical comparison difficult.

Finally, Māori, and Pacific people are under-represented in the Dunedin Study when compared with the National Surveys (where data is weighted to match the census population). Given differences in health status between ethnic groups, this may impact on comparisons between the total Dunedin cohort and the New Zealand Survey populations. For these reasons we need to be cautious about concluding that the Dunedin cohort findings are able to be generalised on the basis of ethnicity and this issue needs to be investigated further. Nevertheless, with regards to Māori, it is noted that 73 Dunedin Study members self-identified as Māori at age 26. By comparison, there were 64 Māori participants in the 25 and 26 year-old age range in the New Zealand Health Survey. Hence, although the Dunedin Study may under-represent Māori as a proportion of its total sample, it actually has a larger number of Māori participants of this age than the nationally representative sample.

In conclusion, there appear to be few important differences in self-reported and objectively measured health between participants in the long-running Dunedin Study, and participants of similar ages in nationally-representative surveys. This suggests that the Dunedin Study members have not been changed by undergoing repeated assessments throughout their lives, and that findings from the Dunedin Study are likely to be broadly generalisable to the wider New Zealand population. These findings may be relevant to the design of future New Zealand cohort studies.

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We are grateful to the Dunedin Study members and their parents for their continued support and to the participants in the New Zealand Health and National Nutrition Surveys. We thank Karen Blakey for help in compiling data from the National Health Survey, and Professor David Fergusson for helpful comments. We also wish to thank Dr Phil A Silva, the Dunedin Study founder.

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References:

2. Gale EA. The Hawthorne studies--a fable for our times? QJM. 2004;97:439–49.
Metabolic equivalent (MET) intensities of culturally-specific physical activities performed by New Zealanders

Karen Moy, Robert Scragg, Grant McLean, Harriette Carr

Abstract

Aims This study’s purpose was to objectively measure the intensity, expressed as metabolic equivalents (METs), of free-living physical activities (PAs) performed by New Zealanders.

Methods A sample of 186 European/Other (n=60), Māori (n=61), and Pacific (n=65) males and females (mean age 48.6±16.4 yrs) underwent 3 days of minute-by-minute heart rate monitoring (HRM) with individual calibration on a cycle ergometer. Mean METs were derived from average heart rate readings and compared to published equivalents from the United States (US) Compendium of Physical Activities.

Results Although New Zealand-derived METs were slightly higher, comparison to the US instrument showed strong correlations (R²=0.62). Overall intensities for Māori kapahaka PAs ranged from 4.3 to 7.1 METs, and were generally classified as vigorous- and moderate-intensity for males and females, respectively. In addition to 12 New Zealand Māori and Pacific activities, 5 PAs captured during HRM are not found in the US Compendium, and New Zealand-derived METs for 9 PAs were classified differently from the US instrument.

Conclusions Availability of New Zealand-specific MET intensities increases the precision of estimating energy expenditure and PA levels when direct measures are not possible. PA surveillance in New Zealand is further enhanced by the ability to substitute culturally-specific examples of intensity when necessary.

Intense is a dimension of physical activity (PA) that signifies the level of physical exertion associated with executing the activity. Absolute intensity represents the overall rate of energy expenditure (EE) while performing the PA, and metabolic equivalents (METs) are universally accepted units for expressing EE relative to an individual’s body weight. One MET represents the rate of oxygen consumption (VO₂), approximately 3.5 ml·kg⁻¹·min⁻¹, for an average adult sitting quietly. Thus, an individual performing an activity of 4 METs has a VO₂ 4 times higher than that at rest. PA intensities are categorised in absolute terms as ‘light’ (<3 METs), ‘moderate’ (3–6 METs) or ‘vigorous’ (>6 METs), in accordance with guidelines set forth by the Center for Disease Control (CDC) and the American College of Sports Medicine (ACSM).

Due to influencing factors such as an individual’s age, gender, and fitness level, exact MET intensities can only be obtained through direct measures. Published tables of energy costs, such as the Compendium of Physical Activities created in the United States (US), serve as references for estimating MET levels when direct measures are impractical. The US Compendium is an internationally accepted compilation of published and unpublished data that classifies an expansive range of PAs by MET
intensities. This instrument was designed to facilitate coding of PAs obtained from questionnaires, interviews, diaries or logs, and to promote comparison between intensities of different PAs. Although the US compendium is widely used, the MET intensities are specific to Western populations where PAs were predominantly performed by young adults. The EE data may therefore be inappropriate for other cultures,\textsuperscript{8,9} and could have a tendency to overestimate MET levels for middle-aged and older adults.\textsuperscript{10}

An instrument that reflects PAs performed by New Zealand cultures, and segregates MET levels by age and gender subgroups, is desirable and advantageous for several reasons.

- Establishing a list of baseline METs would serve as a foundation for PA researchers in New Zealand to build upon, in terms of sample size and the continued expansion of listed PAs;
- The segregation of MET intensities by age and gender groups is advantageous for PA-related research with limited or specifically-targeted samples;
- A list of New Zealand-specific METs could be utilised in both clinical and public health settings;
- Increased accuracy of daily EE would enhance PA and dietary recommendations prescribed by health professionals, such as Green Prescriptions and daily energy intake, and could contribute significantly to decreasing obesity rates in Māori and Pacific populations in New Zealand;
- Culturally-specific METs support New Zealand PA guidelines and enhance PA data obtained by questionnaires;\textsuperscript{11} and
- These PAs and associated MET values could contribute to and be included in an updated version of the US Compendium, if its creators so desire.

The aim of this study was to objectively measure and list MET intensities of culturally-specific PAs performed by New Zealanders, in an effort to enhance PA assessments among the New Zealand population. MET intensities were obtained during a study which validated two New Zealand physical activity questionnaires (NZPAQs) against each other, as well as an international physical activity questionnaire (IPAQ-long).

This study, conducted in 2002–2003, was jointly funded by Sport and Recreation New Zealand (SPARC) and the Ministry of Health. The long form (NZPAQ-LF) will be incorporated into SPARC’s New Zealand Sport and Physical Activity Survey 2006–2007. The short form (NZPAQ-SF) has been included in the Ministry of Health’s New Zealand Health Survey and is designed for inclusion in other omnibus surveys.

**Methods**

Ethical approval was granted by the Auckland Ethics Committee. Participants were recruited from community organisations and a general practitioner register to ensure PA patterns were representative of free-living adults. Each participant received verbal and written explanations of the study protocol prior to providing written informed consent. The sample consisted of 186 participants, aged 19–86 years. Sample sizes were approximately equal across gender, age (18–39 yrs, 40–59 yrs, 60+ yrs) and ethnic groups (European/Other, Māori, Pacific) to enable subgroup comparisons.
An initial visit for physical and physiological measures took place at a local community site near the participants’ residence or workplace. Age, height, and weight were measured prior to fitting of a Polar S-610 heart rate (HR) monitor and Metamax gas analyser. Participants lay prone for a minimum of 5 minutes prior to recording resting HR and blood pressure (BP). Following resting measures, a submaximal cardiorespiratory fitness (CRF) test on a Monark cycle ergometer (Model 824E) was performed.

Three protocols for determining workloads were designed according to age, gender, and fitness level, adapted from the YMCA protocol, which is commonly used to predict maximum oxygen consumption (VO\textsubscript{2max}) by extrapolating data to the individual’s age-predicted maximum HR (HR\textsubscript{max}). Average HR and VO\textsubscript{2} were measured during multiple stages of submaximal work rates, consisting of a 5-minute warm-up stage, followed by 4-minute stages at increased work rates. Additional 1-minute stage extensions were performed if steady state HR was not achieved within 4 minutes. The CRF test protocols were designed to elicit HR responses equivalent to “moderate-” and “hard”-intensity exercise, defined by the ACSM as 40%–50% and 60%–70% of heart rate reserve (HRR), respectively. Target HR ranges were calculated by the following formula:

\[
\text{Target HR range} = [(\text{HR}_{\text{max}}-\text{RHR})] \times \%\text{HRR} + \text{RHR}
\]

Where HR\textsubscript{max} = 220 – age (yrs), and

\%HRR is the individual’s average HR over 5 consecutive minutes of rest, and

Tests were terminated when steady-state HR was achieved during hard-intensity cycling, or by request due to unreasonable discomfort or volitional fatigue. HR monitors were programmed to average and record minute-by-minute HR over 3 consecutive days. Participants received written and verbal instructions pertaining to heart rate monitoring (HRM) and simultaneous completion of PA logs, which commenced the following day. HRM took place during waking hours only, recording immediately following the morning shower to just before bedtime. Participants completed PA Logs at the end of each day, but were encouraged to immediately record the type of PAs performed.

Visit 2 was conducted at the subject’s home or workplace within 24 hours of HRM completion (4 days after Visit 1). HRM data was downloaded using the Polar IR Infrared Serial Port Interface and Polar Precision Performance 3.0 software, and manually searched for periods of at least 10 minutes where the individual’s HR was consistently above 40% HRR. The exact duration and average HR of each PA was recorded, and PA logs were verified for corresponding time periods. Actual MET values for PAs reported and analysed during HRM were derived from corresponding relative VO\textsubscript{2} (ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) for each average HR, using the following calculation:

\[
\text{METs} = \frac{\text{VO}_2}{3.5}\text{ml·kg}^{-1}\text{·min}^{-1}
\]

Mean METs were compared to published METs from the US Compendium. In an effort to facilitate comparisons, PAs captured in this study were categorised into major headings found in the US Compendium (Sport and Recreation, Ball Games, Water Activities, Transportation, Occupation, Other/Incidental, Home, Lawn and Garden Activities). Although very specific PAs are listed in the US instrument, METs corresponding to ‘general’ activities were chosen to facilitate comparisons to New Zealand-derived METs. If necessary, more detailed information was collected for the most accurate reflection of intensity, and each PA was classified as either moderate- or vigorous-intensity, and assigned the appropriate MET value from the updated Compendium of Physical Activities.

The majority of PAs captured from the New Zealand sample but not included in the US Compendium were cultural activities performed by Māori participants, who were also members of Te Roopu Manutaki, an Auckland-based kapahaka group that conducts weekly practice sessions in preparation for national competitions. HRM data were used to calculate individual and group EE levels (METs) for kapahaka practice and each individual activity. Descriptions of each activity (Appendix 1) combine input from a member of Te Roopu Manutaki, personal observation by the first author, and definitions from an appropriate reference. HRM data from Māori participants outside of Hoani Waititi Marae, who performed the same activities, were also used in the calculations.

The data were analysed using the SAS statistical package. CRF and HR data were collected as continuous measures, used to estimate VO\textsubscript{2max} and total EE, respectively. The means (95%CI) were reported for the total sample and by age, ethnicity, and gender. Correlation coefficients were classified as poor (r<0.30), moderate (r=0.31–0.50) and strong (r≥0.50).
Results

Total sample size by gender, ethnicity, and age subgroups is presented in Table 1. Overall, the total sample of 186 apparently healthy participants had a mean age of 48.6 yrs, and a mean BP reading of 127/79 mmHg.

Table 1. Characteristics of study sample size by gender, ethnicity, and age

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>European/Other</th>
<th>Māori</th>
<th>Pacific*</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>F</td>
</tr>
<tr>
<td>18–39</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>40–59</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>60+</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>35</td>
</tr>
</tbody>
</table>

*Comprising mostly Tongans and Samoans.

Approximately 36% and 40% of the total sample was classified as overweight and obese, respectively. Height (p<0.0001) and weight (p=0.001) were the only observed gender differences that reached statistical significance, as males were taller and heavier than females. Although the proportion of male smokers (27%) was higher than females (16%), this difference was not statistically significant (p=0.07).

In general, European/Others (n=60) displayed significantly lower body weight and body mass index (BMI) values (p<0.0001), compared to Māori (n=61) and then Pacific (n=65), and significantly lower BP levels compared to the Pacific sample. Pacific and Māori participants had the highest rates of obesity (55% and 44%, respectively) and being overweight (32% and 43%, respectively), compared to European/Other participants.

Compared to the 18–39 yrs age group, participants in the 40–59 yrs and 60+ yrs age groups showed significantly higher systolic BP (p=0.008 and p=0.0004, respectively) and diastolic BP (p=0.0002 and p=0.0005, respectively). Additionally, the 60+ yrs age group was significantly shorter than the 18–39 yrs (p=0.003) and 40-59 yrs (p=0.01) age groups.

Six participants (3 male, 3 female) had unusable heart rate (HR) data for various reasons. HR monitors were unable to detect one participant’s HR, despite 2 separate attempts. Two Māori participants (1 male and 1 female aged 60+ yrs) had abnormally high HR readings throughout all 3 days of HRM, and HR data was lost for 3 European/Other participants (1 male aged 40–59 yrs, 2 females aged 18–39 yrs) due to computer problems.

Mean MET levels for sport and recreation and non-sport and recreation PAs (transportation, occupation, other/incidental, home, lawn and garden), compared to US METs, are presented in Tables 2 and 3, respectively. New Zealand activities are broadly classified, while the US Compendium provides MET values for very specific PA modes. In some cases, mean METs from multiple corresponding US Compendium PAs were calculated to enable comparisons of EE rates with New Zealand-derived PAs. Generally, METs from this study sample showed good correlation (R²=0.62) to those listed in the US Compendium. Overall, New Zealand MET values were slightly higher, although still strongly correlated with the US Compendium (Figure 1).
### Table 2. New Zealand (NZ) vs. US mean METs—sport and recreation activities

<table>
<thead>
<tr>
<th>SPORT &amp; RECREATION ACTIVITIES (number of activity sessions)</th>
<th>NZ Code</th>
<th>Mean METs (95% CI)</th>
<th>US Code</th>
<th>US METs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aerobics (12)</td>
<td>1</td>
<td>6.6 (6.3–6.9)</td>
<td>03015</td>
<td>6.5</td>
</tr>
<tr>
<td>Cycling – competitive (3)</td>
<td>10</td>
<td>10.0 (9.6–10.4)</td>
<td>01050</td>
<td>12.0</td>
</tr>
<tr>
<td>Cycling – recreational (not mountain biking) (1)</td>
<td>11</td>
<td>8.7</td>
<td>01015</td>
<td>8.0</td>
</tr>
<tr>
<td>Exercise classes/Gym (other than aerobics work)/Weight training (26)</td>
<td>12</td>
<td>5.0 (4.6–5.4)</td>
<td>02060, 02130, 02050</td>
<td>5.5, 3.0, 6.0</td>
</tr>
<tr>
<td>Exercising at home (11)</td>
<td>13</td>
<td>3.8 (3.7–3.9)</td>
<td>02030</td>
<td>3.5</td>
</tr>
<tr>
<td>Rowing (2)</td>
<td>22</td>
<td>4.8 (4.6–5.0)</td>
<td>02070</td>
<td>7.0</td>
</tr>
<tr>
<td>Running/Jogging/Cross-country (21)</td>
<td>26</td>
<td>7.4 (7.0–7.8)</td>
<td>12150, 12020, 12140</td>
<td>8.0, 7.0, 9.0</td>
</tr>
<tr>
<td>Walking for enjoyment or exercise (10-30min) (47)</td>
<td>40</td>
<td>4.4 (4.2–4.6)</td>
<td>17160, 17250, 17200</td>
<td>3.5, 3.5, 3.8</td>
</tr>
<tr>
<td>Walking for enjoyment or exercise (&gt;30min) (33)</td>
<td>41</td>
<td>3.7 (3.4–4.0)</td>
<td>17160, 17250, 17200</td>
<td>3.5, 3.5, 3.8</td>
</tr>
<tr>
<td>Martial Arts/Dancing (2)</td>
<td>46</td>
<td>2.7 (2.5–2.9)</td>
<td>15670, 15430, 03010</td>
<td>4.0, 10.0, 4.8</td>
</tr>
<tr>
<td>Boxing (punching bag) (1)</td>
<td>48</td>
<td>5.4</td>
<td>15110</td>
<td>6.0</td>
</tr>
<tr>
<td><strong>BALL GAMES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basketball (5)</td>
<td>5</td>
<td>5.8 (5.4–6.2)</td>
<td>15050</td>
<td>6.0</td>
</tr>
<tr>
<td>Bowls – outdoor/lawn (2)</td>
<td>6</td>
<td>3.2 (3.18–3.22)</td>
<td>15570</td>
<td>3.0</td>
</tr>
<tr>
<td>Bowls – indoor (1)</td>
<td>7</td>
<td>3.7</td>
<td>15090</td>
<td>3.0</td>
</tr>
<tr>
<td>Golf (2)</td>
<td>16</td>
<td>5.3 (5.0–5.6)</td>
<td>15255</td>
<td>4.5</td>
</tr>
<tr>
<td>Rugby – union (2)</td>
<td>23</td>
<td>8.3 (8.1–8.5)</td>
<td>15560</td>
<td>10.0</td>
</tr>
<tr>
<td>Rugby – touch (2)</td>
<td>25</td>
<td>6.4 (5.6–7.2)</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Soccer (5)</td>
<td>29</td>
<td>7.0 (6.7–7.3)</td>
<td>15610</td>
<td>7.0</td>
</tr>
<tr>
<td>Tennis (3)</td>
<td>35</td>
<td>5.8 (5.5–6.1)</td>
<td>15675</td>
<td>7.0</td>
</tr>
<tr>
<td>Volleyball (1)</td>
<td>38</td>
<td>3.0</td>
<td>15720</td>
<td>3.0</td>
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<tr>
<td><strong>WATER ACTIVITIES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aquarobics (2)</td>
<td>2</td>
<td>5.9 (5.5–6.3)</td>
<td>18355</td>
<td>4.0</td>
</tr>
<tr>
<td>Swimming (12)</td>
<td>34</td>
<td>5.9 (5.5–6.3)</td>
<td>18310, 18240, 18230</td>
<td>6.0, 7.0, 10.0</td>
</tr>
</tbody>
</table>
Table 3. New Zealand (NZ) vs. US mean METS—non-sport and recreation activities

<table>
<thead>
<tr>
<th>NON-SPORT &amp; RECREATION ACTIVITIES (number of activity sessions)</th>
<th>NZ Code</th>
<th>Mean METs (95% CI)</th>
<th>US Code</th>
<th>US METs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TRANSPORTATION ACTIVITIES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking (4)</td>
<td>200</td>
<td>2.6 (2.5, 2.7)</td>
<td>17161</td>
<td>2.5</td>
</tr>
<tr>
<td><strong>OCCUPATION ACTIVITIES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carrying light loads (walking) (2)</td>
<td>300</td>
<td>3.4 (3.3–3.5)</td>
<td>11795, 11800, 11810</td>
<td>3.0, 4.0, 4.5</td>
</tr>
<tr>
<td>Moving/Lifting light loads (standing) (4)</td>
<td>301</td>
<td>5.0 (4.8–5.2)</td>
<td>11610</td>
<td>3.0</td>
</tr>
<tr>
<td>Moving/Lifting/Carrying heavy loads (12)</td>
<td>302</td>
<td>4.2 (4.0–4.4)</td>
<td>11050, 110630</td>
<td>8.0, 4.0</td>
</tr>
<tr>
<td>Walking (13)</td>
<td>303</td>
<td>3.5 (3.4–3.6)</td>
<td>11791, 11792, 11793</td>
<td>2.0, 3.3, 3.8</td>
</tr>
<tr>
<td>Light/Moderate cleaning (3)</td>
<td>304</td>
<td>2.3 (2.2–2.4)</td>
<td>11125</td>
<td>3.5</td>
</tr>
<tr>
<td>Other (lawn mowing, planting) (4)</td>
<td>305</td>
<td>3.9 (3.6–4.2)</td>
<td>08095, 08140, 08150</td>
<td>5.5, 4.5, 4.5</td>
</tr>
<tr>
<td><strong>HOME ACTIVITIES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General cleaning (31)</td>
<td>400</td>
<td>2.9 (2.7–3.1)</td>
<td>05030</td>
<td>3.0</td>
</tr>
<tr>
<td>Walking - light, non-cleaning (ready to leave, shut/lock doors &amp; windows) (15)</td>
<td>401</td>
<td>3.4 (3.2–3.6)</td>
<td>05165</td>
<td>3.0</td>
</tr>
<tr>
<td>Self care (13)</td>
<td>403</td>
<td>3.9 (3.5–4.3)</td>
<td>13020, 13040</td>
<td>2.0, 2.0</td>
</tr>
<tr>
<td>Moving furniture (1)</td>
<td>404</td>
<td>5.7</td>
<td>05120</td>
<td>6.0</td>
</tr>
<tr>
<td>Multiple household activities (moderate) (4)</td>
<td>405</td>
<td>4.3 (4.0–4.6)</td>
<td>05026</td>
<td>3.5</td>
</tr>
<tr>
<td>Home repair (2)</td>
<td>410</td>
<td>5.7 (5.5–5.9)</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Childcare (2)</td>
<td>412</td>
<td>2.8 (2.7–2.9)</td>
<td>05185, 21016, 05186, 21017</td>
<td>2.5, 2.5, 3.0, 3.0</td>
</tr>
<tr>
<td>Kitchen activity - cooking, washing dishes (8)</td>
<td>418</td>
<td>3.4 (3.2–3.6)</td>
<td>05041, 05050</td>
<td>2.3, 2.0</td>
</tr>
<tr>
<td><strong>OTHER/INCIDENTAL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Playing with children (3)</td>
<td>407</td>
<td>2.2 (2.1–2.3)</td>
<td>05170, 05171</td>
<td>2.5, 2.8</td>
</tr>
<tr>
<td>Carrying light to moderate loads (4)</td>
<td>408</td>
<td>4.5 (4.0–5.0)</td>
<td>--</td>
<td>2.0, 3.3, 3.8</td>
</tr>
<tr>
<td>Carrying heavy loads (4)</td>
<td>409</td>
<td>4.0 (3.9–4.1)</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Coaching sport (1)</td>
<td>413</td>
<td>8.0</td>
<td>15140</td>
<td>4.0</td>
</tr>
<tr>
<td>Emotion/Stress/Sport spectator (4)</td>
<td>414</td>
<td>3.3 (3.1–3.5)</td>
<td>09115</td>
<td>1.5</td>
</tr>
<tr>
<td>Socialising/Eating (15)</td>
<td>415</td>
<td>3.4 (3.1–3.7)</td>
<td>13030, 09100, 13035</td>
<td>1.5, 1.5, 2.0</td>
</tr>
<tr>
<td>Religious/Church activity (7)</td>
<td>416</td>
<td>3.4 (3.2–3.6)</td>
<td>20005, 20020</td>
<td>1.5, 2.0</td>
</tr>
<tr>
<td><strong>LAWN AND GARDEN</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gardening (22)</td>
<td>15</td>
<td>4.0 (3.7–4.3)</td>
<td>08245</td>
<td>5.0</td>
</tr>
<tr>
<td>Lawn mowing (3)</td>
<td>406</td>
<td>3.9 (3.5–4.3)</td>
<td>08095</td>
<td>5.5</td>
</tr>
</tbody>
</table>
Table 4 lists PAs which were classified into different intensity categories when compared to the US instrument. Generally, sport and recreation PAs were performed at lighter intensities, while Other/Incidental/Household PAs were performed at higher intensities. The mean age of participants in this study (48.6 yrs) is not comparable to the US Compendium, as the latter is a compilation of findings from several studies.
Table 4. Major discrepancies between US and New Zealand (NZ) METs

<table>
<thead>
<tr>
<th>Major heading</th>
<th>Physical Activity</th>
<th>US METs</th>
<th>NZ METs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sport/Recreation</td>
<td>Martial Arts</td>
<td>6.3</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>Rowing</td>
<td>7.0</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td>Tennis</td>
<td>7.0</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>Trampoline</td>
<td>6.1</td>
<td>3.5</td>
</tr>
<tr>
<td>Other/Incidental/Household</td>
<td>Coaching Sport</td>
<td>4.0</td>
<td>8.0</td>
</tr>
<tr>
<td></td>
<td>Kitchen Activity</td>
<td>2.2</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>Religious/Church Activity</td>
<td>1.8</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>Self Care</td>
<td>2.0</td>
<td>3.9</td>
</tr>
<tr>
<td></td>
<td>Socialising/Eating</td>
<td>1.8</td>
<td>3.4</td>
</tr>
</tbody>
</table>

Subgroup analyses were performed on seven sport and recreation PAs. Females and participants aged 60+ yrs performed four and five PAs at lower mean MET levels, respectively, compared to males and the younger age groups (Table 5). Consequently, three PAs were classified into lighter intensity categories when performed by females or participants aged 60+ years: aerobics, exercise classes-going to the gym/weight training, and swimming.

Two occupational PAs (‘Moving/Lifting/Carrying heavy loads’ and ‘Walking’) were performed frequently enough to warrant subgroup analyses. Males and participants in younger age groups performed both PAs at higher MET levels.

‘Moving/Lifting/Carrying heavy loads’ was captured as a moderate-intensity activity for males, and light-intensity for females. Amongst different age groups, intensity categories remain unchanged.

The following Other/Incidental, Household, Lawn, and Garden PAs were performed at higher intensities by males and younger participants (<60 yrs): Gardening, General Cleaning, Self care, Socialising/Eating, Walking (light), and non-cleaning (ready to leave, shut/lock doors, close windows). Self care and socialising/eating were classified as moderate-intensity PAs compared to light-intensity on the US Compendium, and were classified as vigorous-intensity when performed by the 18–39 yrs age group.

PAs captured during HRM which were not listed in the US Compendium include petanque, touch rugby, carrying light/moderate or heavy loads, home repair, and New Zealand cultural PAs. In addition to assigning a MET level to the overall kapahaka practice performed by Te Roopu Manutaki at the Hoani Waititi Marae, METs for 11 individual kapahaka activities were captured during HRM, and are listed in Table 6.

A description of New Zealand Māori activities captured in this study is listed in Appendix 1. Cook Island Dance was performed once by one female participant, providing a baseline of 4.3 METs (moderate-intensity) for this activity. Another female participant performed Tongan Dance on two separate occasions during her HRM period, averaging 1.9 METs (light-intensity). The three sessions of Cook Island and Tongan dance were combined into a ‘Pacific Island Dance’ category and classified as light-intensity activity (2.7 METs) (Table 6).
Table 5. New Zealand METs (95% CI) by age and gender subgroups

<table>
<thead>
<tr>
<th>PHYSICAL ACTIVITIES BY CONTEXT</th>
<th>MEAN METs (95% CI) ASSESSED IN NEW ZEALAND (n)</th>
<th>18-39</th>
<th>40-59</th>
<th>60+</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age Group (yrs)</td>
<td>Gender Group</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>18-39</td>
<td>40-59</td>
<td>60+</td>
<td>Male</td>
<td>Female</td>
<td></td>
</tr>
<tr>
<td><strong>SPORT &amp; RECREATION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aerobics</td>
<td>7.0 (6.7, 7.3)</td>
<td>5.3</td>
<td>3.5</td>
<td>7.4 (7.2, 7.6)</td>
<td>5.7 (5.4, 6.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(10)</td>
<td>(1)</td>
<td>(1)</td>
<td>(6)</td>
<td>(6)</td>
<td></td>
</tr>
<tr>
<td>Exercise classes/Going to the gym (other than aerobics work)/Weight training</td>
<td>5.3 (5.2, 5.4)</td>
<td>6.2 (5.9, 6.5)</td>
<td>2.5 (2.4, 2.6)</td>
<td>5.9 (5.7, 6.1)</td>
<td>4.4 (4.0, 4.8)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(7)</td>
<td>(12)</td>
<td>(7)</td>
<td>(16)</td>
<td>(10)</td>
<td></td>
</tr>
<tr>
<td>Exercising at home</td>
<td>3.9 (3.9, 3.9)</td>
<td>3.4 (3.4, 3.4)</td>
<td>4.1 (4.0, 4.2)</td>
<td>4.6 (4.5, 4.7)</td>
<td>3.6 (3.6, 3.6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(2)</td>
<td>(5)</td>
<td>(4)</td>
<td>(2)</td>
<td>(9)</td>
<td></td>
</tr>
<tr>
<td>Running/Jogging/Cross-country</td>
<td>7.5 (7.0, 8.0)</td>
<td>7.1 (7.0, 7.4)</td>
<td>6.9 (6.2, 7.6)</td>
<td>7.1 (6.7, 7.5)</td>
<td>7.7 (7.2, 8.2)</td>
<td></td>
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<tr>
<td></td>
<td>(15)</td>
<td>(4)</td>
<td>(2)</td>
<td>(12)</td>
<td>(9)</td>
<td></td>
</tr>
<tr>
<td>Walking for enjoyment or exercise (10-30min)</td>
<td>4.4 (4.1, 4.7)</td>
<td>4.2 (4.1, 4.3)</td>
<td>4.4 (4.1, 4.7)</td>
<td>4.4 (4.1, 4.7)</td>
<td>4.4 (4.2, 4.6)</td>
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<tr>
<td></td>
<td>(27)</td>
<td>(6)</td>
<td>(14)</td>
<td>(15)</td>
<td>(32)</td>
<td></td>
</tr>
<tr>
<td>Walking for enjoyment or exercise (&gt;30min)</td>
<td>4.7 (4.5, 4.9)</td>
<td>3.9 (3.6, 4.2)</td>
<td>3.3 (3.1, 3.5)</td>
<td>3.8 (3.5, 4.1)</td>
<td>3.7 (3.5, 3.9)</td>
<td></td>
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<tr>
<td></td>
<td>(7)</td>
<td>(7)</td>
<td>(19)</td>
<td>(17)</td>
<td>(16)</td>
<td></td>
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<tr>
<td>Swimming</td>
<td>7.1 (6.9, 7.3)</td>
<td>6.8 (6.6, 7.0)</td>
<td>3.0 (2.8, 3.2)</td>
<td>6.6 (6.3, 6.9)</td>
<td>2.6 (2.5, 2.7)</td>
<td></td>
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<tr>
<td></td>
<td>(3)</td>
<td>(6)</td>
<td>(3)</td>
<td>(10)</td>
<td>(2)</td>
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<tr>
<td><strong>OCCUPATION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moving/Lifting/Carrying heavy loads</td>
<td>5.5 (5.4, 5.6)</td>
<td>3.5 (3.4, 3.6)</td>
<td>–</td>
<td>4.5 (4.3, 4.7)</td>
<td>2.9 (2.8, 3.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(4)</td>
<td>(8)</td>
<td></td>
<td>(10)</td>
<td>(2)</td>
<td></td>
</tr>
<tr>
<td>Walking</td>
<td>--</td>
<td>3.7 (3.6, 3.8)</td>
<td>3.1 (3.0, 3.2)</td>
<td>3.5 (3.4, 3.6)</td>
<td>3.3</td>
<td></td>
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<td></td>
<td></td>
<td>(8)</td>
<td>(5)</td>
<td>(12)</td>
<td>(1)</td>
<td></td>
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<tr>
<td><strong>HOME</strong></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>General Cleaning</td>
<td>4.3 (4.0, 4.6)</td>
<td>3.5 (3.4, 3.6)</td>
<td>2.5 (2.3, 2.7)</td>
<td>3.2 (2.9, 3.5)</td>
<td>2.8 (2.6, 3.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3)</td>
<td>(8)</td>
<td>(20)</td>
<td>(11)</td>
<td>(20)</td>
<td></td>
</tr>
<tr>
<td>Walking: light, non-cleaning (ready to leave, shut/lock doors &amp; windows)</td>
<td>5.3</td>
<td>3.6 (3.4, 3.8)</td>
<td>2.7 (2.6, 2.8)</td>
<td>4.9 (4.8, 5.0)</td>
<td>3.2 (3.0, 3.4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1)</td>
<td>(10)</td>
<td>(4)</td>
<td>(2)</td>
<td>(13)</td>
<td></td>
</tr>
<tr>
<td>Self Care</td>
<td>6.4 (6.1, 6.7)</td>
<td>4.0 (3.7, 4.3)</td>
<td>2.3 (2.2, 2.4)</td>
<td>4.6 (4.1, 5.1)</td>
<td>3.6 (3.3, 3.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3)</td>
<td>(5)</td>
<td>(5)</td>
<td>(4)</td>
<td>(9)</td>
<td></td>
</tr>
<tr>
<td><strong>LAWN &amp; GARDEN</strong></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Gardening</td>
<td>5.0 (4.8, 5.2)</td>
<td>3.8 (3.5, 4.1)</td>
<td>3.9 (3.6, 4.2)</td>
<td>4.1 (3.8, 4.4)</td>
<td>3.8 (3.6, 4.0)</td>
<td></td>
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<tr>
<td></td>
<td>(2)</td>
<td>(3)</td>
<td>(18)</td>
<td>(16)</td>
<td>(7)</td>
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</tbody>
</table>
Table 6. New Zealand METs—Māori activities

<table>
<thead>
<tr>
<th>MĀORI ACTIVITIES*</th>
<th>Overall METs (n)</th>
<th>MEAN METs (95% CI) ASSESSED IN NEW ZEALAND (n)</th>
<th>Gender Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Age Group (yrs)</td>
<td>Male</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18–39 (yrs)</td>
<td>40–59 (yrs)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Male (n)</td>
</tr>
<tr>
<td>Haka</td>
<td>7.1 (6.8–7.4) (10)</td>
<td>7.9 (7.6–8.2) (6)</td>
<td>5.9 (5.7–6.1) (4)</td>
</tr>
<tr>
<td>Haka Pōwhiri</td>
<td>5.8 (5.2–6.4) (11)</td>
<td>6.6 (5.8–7.4) (5)</td>
<td>5.2 (4.8–5.6) (6)</td>
</tr>
<tr>
<td>Haka Tūtūngurahu</td>
<td>5.4 (4.9–5.9) (21)</td>
<td>6.3 (5.7–6.9) (11)</td>
<td>4.4 (4.0–4.8) (10)</td>
</tr>
<tr>
<td>Kapahaka</td>
<td>4.9 (4.6–5.2) (27)</td>
<td>4.2 (4.0–4.4) (16)</td>
<td>3.8 (3.6–4.0) (10)</td>
</tr>
<tr>
<td>Mōteatea</td>
<td>4.3 (3.9–4.7) (13)</td>
<td>4.8 (4.4–5.2) (7)</td>
<td>3.7 (3.4–4.0) (6)</td>
</tr>
<tr>
<td>Poi</td>
<td>4.6 (4.4–4.8) (15)</td>
<td>4.9 (4.7–5.1) (10)</td>
<td>4.0 (3.7–4.3) (5)</td>
</tr>
<tr>
<td>Taiaha</td>
<td>6.4 (6.1–6.7) (22)</td>
<td>6.9 (6.6–7.2) (16)</td>
<td>5.0 (4.9–5.1) (6)</td>
</tr>
<tr>
<td>Waiata-ā-ringa</td>
<td>4.9 (4.5–5.3) (35)</td>
<td>5.4 (5.0–5.8) (20)</td>
<td>4.3 (4.0–4.6) (15)</td>
</tr>
<tr>
<td>Waiata Tira</td>
<td>5.0 (4.6–5.4) (21)</td>
<td>5.4 (5.0–5.8) (12)</td>
<td>4.4 (4.1–4.7) (9)</td>
</tr>
<tr>
<td>Whakaeke</td>
<td>5.4 (4.9–5.9) (20)</td>
<td>6.0 (5.3–6.7) (11)</td>
<td>4.6 (4.2–5.0) (9)</td>
</tr>
<tr>
<td>Whakawātea</td>
<td>5.5 (5.1–5.9) (23)</td>
<td>5.8 (5.3–6.3) (14)</td>
<td>5.1 (4.8–5.4) (9)</td>
</tr>
<tr>
<td>Whakawhititi</td>
<td>5.7 (5.3–6.1) (12)</td>
<td>5.8 (5.2–6.4) (7)</td>
<td>5.4 (5.2–5.6) (5)</td>
</tr>
</tbody>
</table>

* Refer to Appendix 1 at the end of this paper for the English translations.
Table 6 also includes subgroup analyses for Māori activities, and reveals a more strenuous kapahaka performance by males (6.2 METs) compared to females (3.9 METs). All male kapahaka PAs were classified as vigorous-intensity (>6 METs), with the exception of Waiata-ā-ringa (5.9 METs), Mōteatea (5.0 METs), and Waiata Tira (5.4 METs), and female kapahaka PAs were of moderate-intensity (3-6 METs), with the exception of Haka Tūtūngurahu (2.7 METs), classified as a light-intensity activity. No kapahaka performers were aged 60+ yrs, although a 78-year-old female participant (not a Te Roopu Manutaki member) performed kapahaka in a different setting, which was classified as light-intensity (2.0 METs). Mean METs were consistently higher in the 18–39 yrs age group compared to the 40–59 yrs age group, with an average difference of 1.2 METs for all kapahaka activities.

Discussion

This study is the first to report population-specific MET values in New Zealand, and is advantageous in that age- and gender-specific MET values for a range of PAs are included. In this study, energy costs of PAs were objectively measured by HRM. Individual calibration processes determined relationships between HR and VO$_2$, which were used to convert mean HR values into METs, an intensity of metabolic activity relative to resting conditions. The objective method of HRM involves an individual calibration procedure that determines HR vs VO$_2$ relationships for each participant, resulting in increased accuracy of individual EE estimates. Most physiological responses to exercise are dictated by the relative intensity, and influenced by factors such as age, gender, weight, disability, and fitness level. An objective physiological measure such as HR can indicate the actual intensity each individual was working at, relative to their age, fitness, and PA level. Furthermore, the equation used in this study to predict VO$_{2\text{max}}$ from individual calibrations yielded a mean difference of -0.32 ml·kg·min$^{-1}$ (-1.1%), compared to measured values, whereas Strath et al reported a deviation of ±15% between predicted VO$_{2\text{max}}$ values and those measured by calorimetry in 61 subjects. This suggests a high level of accuracy for New Zealand-derived MET values.

Energy costs associated with an activity can vary substantially within and between individuals, depending on influencing factors such as the person’s age, sex, body mass, movement patterns, skill, and level of fatigue. Measured METs from this study were compared to those listed in the US Compendium of PAs, and showed strong correlations.

This study provided the opportunity to create a New Zealand-specific compendium of PAs, and included age-, gender-, and culturally-specific MET levels, as several traditional activities performed by the Māori population were captured during HRM. Age-specific MET levels are an important contribution to PA research, as younger individuals perform PAs at higher MET levels, compared to older adults. This data should be used in future PA research conducted in New Zealand, in an effort to build on the instrument and provide more accurate EE levels by age, gender, and ethnicity. This study is the first to report MET values for traditional Māori activities. Although the sample of Māori participants engaging in regular kapahaka practice was relatively small, these results provide baseline measures for 12 kapahaka activities. PA intensity
and rate of EE varies within and between individuals for several reasons. For example, some individuals are more familiar with the different activities, and perform them at the intended level of intensity, while newer members tended to stay in the back and focus on learning proper execution of the PAs. Additionally, each individual’s level of motivation to perform the activities at a competitive level is a factor. Although males and females perform many of the same activities, the EE of each gender’s role should be kept separate, as the male roles are generally of vigorous-intensity while female roles are of moderate-intensity. Similarly, MET levels are consistently higher for the 18–39 yrs age group compared to the 40–59 yrs age group. Finally, Te Roopu Manutaki holds kapahaka practices year-round, and the intensity of these PAs will surely increase as competition nears and individuals are chosen to represent their kapahaka group. Future studies should endeavour to assess these activities in greater numbers.

Due to the invasiveness and burden associated with gold standard procedures, small, convenient samples are typically selected, and therefore caution should be used when extrapolating validation study results to other populations. The respondent burden associated with this study’s design was high. Three face-to-face visits were required from each participant, which entailed a physically demanding exercise test and administration of 4 different PA questionnaires. Additionally, the 3-day period of HRM involved simultaneous completion of daily PA Logs, requiring participants’ full cooperation and availability.

This study used non-random, convenience sampling in an effort to avoid low participation rates, and therefore may not be representative of the general population. Participants were recruited from several different community settings in an effort to ensure the sample would be representative of the Auckland population. Our sample size was relatively large, compared to other studies using the HRM technique to assess PA or validate PAQs. However, numbers for subgroup analyses were comparatively small, and may limit the validity of within-group associations.

This study’s sample consisted of volunteers interested in taking part in a health-related research study, or who had the desire to obtain a free cardiovascular risk profile or individually tailored exercise prescription, and met the criteria for age, gender, and ethnic groups. These individuals were therefore more likely to maintain a higher level of compliance and commitment to the study.

This sample included a wide range of ages and ethnicities as well as both genders. The usual age, ethnic, and gender differences were observed in terms of BP and BMI, as participants who were older and of Polynesian descent demonstrated increased cardiovascular risk from these variables. However, in terms of PA, overweight, or smoking habit, this sample may not be representative of the New Zealand population. In relation to BMI, approximately 40% of males and 30% of females in New Zealand are classified as overweight, while 15% and 19%, respectively, are obese. In this sample, male (40%) and female (31%) prevalences of being overweight matched previous findings, while male (38%) and female (42%) obesity in this sample was substantially higher.

In 2002, 25% of individuals aged 15+ yrs in New Zealand smoked cigarettes, with the highest and lowest prevalence found in people aged 25–34 yrs and 55+ yrs, respectively. The proportion of smokers in our study sample (21%) was similar to...
the population level reported previously, and our participants aged 60+ yrs had the
lowest smoking rate compared to younger age groups. However, prevalence of
smoking was similar between European/Others (20%), Māori (21%), and Pacific
(22%), whereas higher values were expected for Māori (49%) and Pacific (35%)
ethnicities. These findings also support the earlier statement that the sample of 186
participants limits the power of subgroup comparisons. The representativeness of this
subsample to the wider New Zealand population is therefore questionable.

Measured METs from this study were compared to those listed in the US
Compendium of PAs, and showed strong correlations. This study provided the
opportunity to create a New Zealand-specific compendium of PAs, and included age-,
gender-, and culturally-specific MET levels, as several traditional activities performed
by the Māori population were captured during HRM. Age-specific MET levels are an
important contribution to PA research, as younger individuals typically perform PAs
at higher intensities, compared to older adults. Similar data should be captured in
future PA research conducted in New Zealand, in an effort to build on this instrument
and provide more accurate EE levels by age, gender, and ethnicity.

Potential benefits of a New Zealand-specific compendium of PAs exist in both
clinical and public health settings. For example, there are increasing expectations for
general practitioners (GPs) in New Zealand to promote participation in PAs to reduce
risk of obesity and cardiovascular disease, for example by using Green Prescriptions.
Age-, gender-, and culturally-specific MET intensities reported in this paper serve as
valuable resources to GPs for prescribing participation in specific types and intensities
of PAs to improve patients’ health status.

Culturally-specific PA data highlights the contribution these activities make towards
total PA and EE levels, and the baseline MET values can be utilised to increase
accuracy of self-reported measures in this context. In particular, the MET values for
Māori cultural PAs have the potential to assist public health workers engaged in
obesity prevention initiatives with Maori communities. Furthermore, providing
culturally-specific exercise prescriptions may enhance compliance rates in higher-risk
populations, although further exploration is necessary.

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**References:**

Appendix 1. Description of Auckland Manutaki’s kapahaka activities:

<table>
<thead>
<tr>
<th>Activity</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haka</td>
<td>The most physically demanding activity, involving hands, feet, legs, body, voice, tongue and eyes to express passion and vigour. In haka the whole body comes into play, particularly the facial expressions, which can illustrate the meaning of the words quite graphically. Haka is normally performed by males, although females lend vocal support.</td>
</tr>
<tr>
<td>Haka Pōwhiri</td>
<td>The haka of welcome. Although less intense than the Haka, the Haka Pōwhiri is performed vigorously by both males and females.</td>
</tr>
<tr>
<td>Haka Tūtūngurahu</td>
<td>The haka of war. A confrontational form of the Haka performed by males and females.</td>
</tr>
<tr>
<td>Kapahaka</td>
<td>Refers to the entire set of Māori traditional performing arts activities, and the group they are performed by. The term ‘kapa’ refers to ranks or rows, and combined with ‘haka’ it means ranks or rows doing the haka forms. In a contemporary sense it is held to mean a group that practices the Māori performing arts.</td>
</tr>
<tr>
<td>Mōteatea</td>
<td>A traditional chant, emphasising simultaneous pronunciation and interpretation expressed through bodily movements. Although a number of chants exist, they do not normally require much physical effort, and therefore involve a low to moderate energy level.</td>
</tr>
<tr>
<td>Poi</td>
<td>A dance performed solely by females, involving movements with one or two poi to express song interpretation. Poi refers to a string with a ball at one end, and can be single or double short or single, double or quadruple long. Poi is performed in unison as a group, and requires more mental focus then physical exertion, as the motion primarily originates from the wrists and elbows.</td>
</tr>
<tr>
<td>Taiaha</td>
<td>Teaches the art of combat and involves literally hundreds of combined movements of the feet, hands and long club weapon. It is very physical and requires moderate to high levels of energy. Note: The term ‘Mau Rakau’ may be used, referring generally to any (wooden, stone, or bone) Māori weapon.</td>
</tr>
<tr>
<td>Waiata-ā-ringa</td>
<td>Referred to as the action song, involves group movements in unison. A medium level of energy is required.</td>
</tr>
<tr>
<td>Waiata Tira</td>
<td>Fairly motionless singing and harmonising with a number of parts (i.e. base, tenor, soprano, descant). This may involve some stage formations, but requires little physical effort, as the focus is on the music and the song. Recently, Waiata Tira has been used by some groups either as a warm-up or as a humorous piece.</td>
</tr>
<tr>
<td>Whakaeke</td>
<td>Performed as a means of entering the stage, making an impression on the audience and grabbing their attention. The entrance can include singing or 'Haka' type activity, although usually a combination of the two are performed. This can be a vigorous-intensity activity, and requires a medium to high energy level.</td>
</tr>
<tr>
<td>Whakawātea</td>
<td>This activity is similar to the 'Whakaeke', but refers to exiting the stage. It is the final opportunity to impress the audience and is often a combination of Waiata-a-ringa and Haka activity, requiring medium to high levels of energy.</td>
</tr>
<tr>
<td>Whakawhiti</td>
<td>A short, transitional activity performed to simultaneously move the males and females to the front and rear of the stage, respectively, so that males can perform the Haka. This is a moderate to vigorous energy activity.</td>
</tr>
</tbody>
</table>
Do snacks of exercise lower blood pressure? A randomised crossover trial
Raina Elley, Emma Bagrie, Bruce Arroll

Abstract

Aim To assess whether four 10-minute ‘snacks’ of exercise per day are as effective at lowering blood pressure as 40 minutes of continuous moderate exercise, when compared with no exercise.

Method Single blind randomised crossover trial of three ‘exercise’ regimes in general practice. Participants—35 hypertensive adults without complications. Interventions—regimes included 4×10-minute episodes of brisk walking per day, 40 minutes continuous brisk walking per day, and no brisk walking. Each regime lasted 4 days with 10 days of no exercise in between. Outcomes—change of systolic and diastolic blood pressure.

Results Mean age 53 years and mean baseline blood pressure 166/103 mmHg. Systolic blood pressure changed by: –7.5 mmHg (95%CI: –8.9, –6.0) with 40-minutes regime; –7.3 mmHg (95%CI: –8.7, –5.8) with 4×10-minutes regime; and +1.0 mmHg (95%CI: –0.4, 2.5) with ‘no brisk walking’ regime (p<0.001). Diastolic blood pressure reduced by –4.0 mmHg (95%CI: –5.0, –3.0) with 40 minutes regime; –5.4 mmHg (95%CI: –6.4, –4.4) with 4×10 minutes regime; and –0.2 mmHg (95%CI: –1.2, 0.8) with ‘no brisk walking’ regime (p<0.001).

Conclusion Four 10-minute snacks of brisk walking were as effective as 40 minutes of continuous brisk walking per day at reducing blood pressure. This has implications for public health messages and advice to patients with hypertension.

Evidence shows that moderate-intensity physical activity (such as walking) can help control blood pressure,¹⁻⁴ and that blood pressure reductions with exercise are greatest amongst those with hypertension.⁵,⁶

While 30 to 40 minutes of continuous moderate-intensity physical activity on several days per week is adequate to reduce blood pressure in the medium term, 10 minutes is inadequate.⁷

Additional evidence shows that cardiorespiratory fitness (VO₂ max) can improve as much with several short episodes as with one continuous episode of moderate exercise (when energy expenditure remains constant),⁸ although to date the same has not been confirmed for change in blood pressure. Specifically, previous studies of normotensive adults have compared three episodes of 10 minutes against 30 minutes of continuous brisk walking per day but they were not able to demonstrate significant changes in blood pressure compared with a control.⁹,¹⁰ In addition, further research into the effects of ‘fractionisation’ of exercise was recommended.⁸

Time is a major barrier for people achieving regular exercise for health benefit.¹¹ However, if regular moderate exercise could be achieved in ‘snacks’ throughout the
day, recommended levels of exercise for blood pressure and other health benefit may be more achievable, particularly for those people with pre-existing risk factors such as hypertension.

Our study aims to determine whether four 10-minute ‘snacks’ of exercise are as effective at reducing blood pressure in hypertensive participants as 40 minutes of continuous exercise per day (when compared with no exercise).

Methods

Design—A randomised crossover design was used within one primary healthcare practice with three general practitioners in Auckland, New Zealand. The Auckland Ethics Committee approved the study protocol and written informed consent was obtained from all participants.

Subjects—All adults with the diagnosis of hypertension were identified by the practice. Those fulfilling eligibility criteria and considered suitable for participation by their usual general practitioner, were invited to take part in the study.

Inclusion criteria consisted of a systolic blood pressure of at least 140 mmHg or diastolic blood pressure of at least 85 mmHg 10 days after withdrawal of any antihypertensive medication; and able to do ‘no exercise’ during the 10-day run-in period prior to the study.

Exclusion criteria consisted of a cardiovascular or unstable condition, progressive or debilitating medical condition, acutely unwell, physically active occupation, unable to understand English, or considered unsuitable by their general practitioner to participate in the study or withdraw temporarily from their antihypertensive medication.

Following the protocol used in a previous study, 7 participants were asked to reduce their antihypertensive medication under the supervision of their general practitioner so that they would be receiving no antihypertensive medication 10 days prior to the start of the study. The usual general practitioner was notified if the blood pressure exceeded 160 mmHg systolic or 95 mmHg diastolic during the run-in period and throughout the trial.

The recommendation to enrol or continue in the study in these circumstances was made by their general practitioner. Usual antihypertensive medication was recommenced after the 6-week study.

Study protocol—Following a 10-day period of no exercise, the participants were randomised if they complied with the no-exercise run-in protocol and fulfilled eligibility criteria. Eligible participants undertook three ‘exercise’ regimes in an order randomly determined using numbered opaque envelopes. Each sealed envelope was handed to the participant who was told not to disclose the order to the assessor. A different researcher undertook prior computer-randomisation and prepared the envelopes.

The intervention regimes included advice given by the researcher based in primary care to achieve 4 x 10-minute episodes of brisk walking per day, 40 minutes continuous brisk walking per day, and no brisk walking. Participants were encouraged to set a pace that made them ‘puff’ and increased their heart rate. Participants were also asked not to do any other exercise outside of the regimes and not to change their diet over the course of the study.

Primary outcome measures were change in systolic and diastolic blood pressure with each regime. A researcher who was blind to allocated random order carried out all measures. The usual general practitioners were also blind to allocation of randomisation.

A calibrated Speidal and Keller OSZ5 electronic sphygmomanometer was used to measure all baseline and follow-up pulses and blood pressures. On each occasion, these measures were taken after at least 5 minutes of sitting quietly. Three readings were taken and the average of the second and third readings was used for analysis, according to a previously used research protocol.12 Each regime lasted 4 days with 10 days of no exercise in between each regime. Blood pressure was measured before, and the day after, each regime—at least 12 hours after any exercise and at a similar time of day.

To provide an indication of intensity of exercise undertaken during the regimes, heart rates were recorded electronically immediately following a short ‘brisk walk’ and compared with resting heart rate after completion of the trial.
Sample size calculations—Twenty-five participants were required to detect a statistically significant difference in change of 7 mmHg systolic or diastolic blood pressure when comparing results of either exercise regime with those of control. Achievable change in blood pressure following 4 days of 40-minutes of moderate intensity exercise was obtained from previous research. The standard deviation of change in blood pressure with exercise (11 mmHg) was also obtained from previous research.

Statistical analysis—The differences between blood pressure measurements before and after each exercise regime were compared for the three regimes using a generalised linear model in SPSS (version 11.5) statistical software. Differences between each pair of regimes were analysed using post-hoc Student-Newman-Keuls and Bonferroni tests that allowed for multiple comparisons. All participants were analysed according to randomisation sequence. Only those that completed the study were included in the analysis. A conservative intention to treat sensitivity analysis was also undertaken where no change in blood pressure was assumed for all regimes of randomised participants who did not complete the study.

Intensity of usual brisk walking was estimated using percent estimated maximum heart rate, equal to (exercise heart rate − resting heart rate)/(estimated maximum heart rate − resting heart rate)*100. Maximum heart rate was estimated using the formula (220-age) for males and (226-age) for females. Percent heart rate reserve is closely numerically related to VO2 reserve and can be used to estimate intensity of exercise. It has been suggested that less than 25% represents very light activity; 25–44% is light activity; 45–59% is moderate activity; and 60–84% is hard activity.

Results

Figure 1 shows the recruitment process. Of the 165 patients that were potentially eligible to participate in the study, 105 patients declined involvement or did not adhere to the 10-day run-in protocol of no exercise. A further 25 were excluded by the general practitioner because of medical exclusion criteria or concern about elevated blood pressure prior to enrolment. Thirty-five patients were randomised and 31 completed the study.

Table 1 shows the baseline characteristics of the participants by randomised initial regime and overall. Fifty-four percent (19/35) were female and 97% (34/35) were on antihypertensive medication prior to the study.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>40 minutes first (SD*) [N=12]</th>
<th>4x10 minutes first (SD*) [N=11]</th>
<th>No exercise first (SD*) [N=12]</th>
<th>Overall (SD*) [N=35]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>56 (12)</td>
<td>51 (14)</td>
<td>52 (9)</td>
<td>53 (12)</td>
</tr>
<tr>
<td>Systolic blood pressure in mmHg</td>
<td>164 (13)</td>
<td>165 (12)</td>
<td>170 (14)</td>
<td>166 (13)</td>
</tr>
<tr>
<td>Diastolic blood pressure in mmHg</td>
<td>107 (11)</td>
<td>100 (8)</td>
<td>101 (5)</td>
<td>103 (8)</td>
</tr>
<tr>
<td>Body mass index in kg/m²</td>
<td>27.7 (4.2)</td>
<td>27.5 (4.0)</td>
<td>27.6 (4.5)</td>
<td>27.6 (4.1)</td>
</tr>
</tbody>
</table>

* Standard deviation
Figure 1. Process of recruitment, randomisation and follow-up of the trial

Table 2. Mean blood pressure and pulse at enrolment baseline and before and after each regime (N=31)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Systolic blood pressure mmHg (SD*)</th>
<th>Diastolic blood pressure mmHg (SD*)</th>
<th>Heart rate beats/min (SD*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>165.7 (13.4)</td>
<td>103.0 (8.2)</td>
<td>77.6 (7.2)</td>
</tr>
<tr>
<td>Before 40 min regime</td>
<td>166.0 (13.4)</td>
<td>102.6 (8.2)</td>
<td>78.2 (7.3)</td>
</tr>
<tr>
<td>After 40 min regime</td>
<td>158.6 (13.1)</td>
<td>98.6 (8.2)</td>
<td>74.8 (8.2)</td>
</tr>
<tr>
<td>Before 4x10min regime</td>
<td>165.7 (13.2)</td>
<td>102.0 (8.6)</td>
<td>77.3 (6.9)</td>
</tr>
<tr>
<td>After 4x10min regime</td>
<td>158.4 (13.2)</td>
<td>96.6 (8.9)</td>
<td>76.6 (7.4)</td>
</tr>
<tr>
<td>Before ‘rest’ regime</td>
<td>165.3 (13.5)</td>
<td>102.6 (7.7)</td>
<td>77.4 (7.8)</td>
</tr>
<tr>
<td>After ‘rest’ regime</td>
<td>166.4 (14.0)</td>
<td>102.4 (7.6)</td>
<td>78.0 (8.1)</td>
</tr>
<tr>
<td>Immediate post-exercise</td>
<td>176.2 (11.9)</td>
<td>102.8 (7.5)</td>
<td>100.6 (8.5)</td>
</tr>
</tbody>
</table>

* Standard deviation; *‘rest’ regime refers to the 4 days of no exercise.
Table 2 shows mean heart rate and blood pressures at enrolment, prior to and following each regime, and immediately post-exercise. Blood pressure returned to baseline levels between regimes after 10 days of no exercise.

Table 3 shows the changes in systolic and diastolic blood pressure with each regime. There was a significant difference between the three regimes in change of systolic blood pressure (p<0.001) and change in diastolic blood pressure (p<0.001).

Table 3. Changes in systolic and diastolic blood pressure for three daily exercise regimes

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Change in blood pressure with each walking regime</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>40 minutes: Mean (95%CI*) [N=31]</td>
</tr>
<tr>
<td>Systolic blood pressure in mmHg</td>
<td>−7.5 (−8.9, −6.0)</td>
</tr>
<tr>
<td>Diastolic blood pressure in mmHg</td>
<td>−4.0 (−5.0, −3.0)</td>
</tr>
</tbody>
</table>

* Confidence interval.

A Student-Newman-Keuls post-hoc test showed that there was no significant difference between the effect of 40 and 4×10 minutes of exercise on systolic blood pressure (p=0.9) although diastolic blood pressure dropped significantly more in the 4×10 minutes regime than in the 40-minute regime (p<0.05).

Both exercise regimes produced significantly greater drops in systolic and diastolic blood pressure than with ‘no exercise’ (p<0.05). Mean intensity of brisk walking was estimated to be light to moderate. Mean heart rate following exercise was 68% (standard deviation 6.0) of maximum heart rate. Mean percent heart rate reserve was 41% (standard deviation 9.4).

Apart from one participant who sprained her ankle just prior to commencing the first regime, there were no other reports of adverse events during the study period or during the 2 months subsequent to the study.

Table 4 shows a conservative Bonferroni analysis comparing each pair of regimes to produce incremental estimates. Table 4 also includes an intention to treat sensitivity analysis, where no change in blood pressure was assumed for all regimes of the four who did not complete the study. Significant differences between the groups (p<0.001) were found but with lower estimated incremental change.
Table 4. Bonferonni analysis of incremental effect on blood pressure of different exercise regimes, including sensitivity analysis

<table>
<thead>
<tr>
<th>Systolic blood pressure mmHg</th>
<th>Per protocol analysis Mean (95% CI*) [N=31]</th>
<th>Sensitivity analysis Mean (95% CI*) [N=35]</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 minutes vs No exercise</td>
<td>-8.5 (-11.1, -6.0)</td>
<td>-7.5 (-10.0, -5.1)</td>
</tr>
<tr>
<td>4x10 minutes vs No exercise</td>
<td>-8.3 (-10.9, -5.6)</td>
<td>-7.4 (-9.8, -4.9)</td>
</tr>
<tr>
<td>40 minutes vs 4 x 10 minutes</td>
<td>-0.2 (-2.8, 2.4)</td>
<td>-0.2 (-2.6, 2.3)</td>
</tr>
<tr>
<td>Diastolic blood pressure mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40 minutes vs No exercise</td>
<td>-3.8 (-5.5, -2.1)</td>
<td>-3.4 (-4.9, -1.8)</td>
</tr>
<tr>
<td>4x10 minutes vs No exercise</td>
<td>-5.2 (-6.9, -3.5)</td>
<td>-4.0 (-6.2, -2.2)</td>
</tr>
<tr>
<td>40 minutes vs 4x10 minutes</td>
<td>1.4 (-0.3, 3.1)</td>
<td>1.2 (-0.3, 2.8)</td>
</tr>
</tbody>
</table>

* Confidence interval; † An intention to treat analysis, where no change in blood pressure was assumed for missing data.

Discussion

This study has demonstrated that four 10-minute snacks of brisk walking are as effective as 40 minutes of continuous brisk walking per day at reducing blood pressure in the short term when compared with no exercise amongst adults with hypertension. This has implications for advice to patients with hypertension and public health messages.

The intervals between the 10-minute episodes of brisk walking were not known, nor were the rates of adherence with the respective protocols. Despite this, significant blood pressure reductions were found in both exercise regimes compared with no exercise. The intensity of exercise was estimated from immediate post-exercise pulse, rather than heart rate monitoring during exercise, which may have underestimated the intensity. However, the walking pace achieved in this study represented that which the participants considered ‘brisk’ and was manageable during their every-day lives, which enhances generalisability of results to a real-life setting.

Many hypertensive participants did not participate because they did not want to do ‘no exercise’ for most of the 6-week study period. Others did not want to stop their medication temporarily, were too busy to participate, or were considered unsuitable by their general practitioner. The resulting low rate of participation may limit external validity of results. However, this is an efficacy trial and there is no obvious reason to believe results would have been different for those who declined participation.

The randomised crossover design used in the study was appropriate, and an unbiased assessment was ensured by blind evaluation of blood pressure and the use of electronic sphygmomanometers. A conservative intention to treat analysis did not change results overall, and ‘snacks’ and ‘continuous’ exercise lowered systolic and diastolic blood pressures by similar amounts.

The time course of this study was appropriate and adequate to demonstrate clinically significant reductions in blood pressure and has been used previously. It is not clear whether these blood pressure reductions are acute or chronic effects of exercise. Previous studies of single episode light or moderate exercise amongst hypertensive subjects have recorded acute reductions of blood pressure that can last between 12
and 22 hours.\textsuperscript{15,16} Whether acute or chronic response, regular moderate intensity walking is likely to be useful in blood pressure management of hypertensive patients.

Blood pressure reductions that last beyond the acute phase can be achieved after as few as three exercise sessions and disappear within 1–2 weeks of no exercise.\textsuperscript{3} This is consistent with findings from the present study, which showed that baseline blood pressures were regained 10-days after each exercise regime.

Reduction of blood pressure was achieved with light to moderate intensity exercise, which is consistent with previous research where energy expenditures as low as 40% maximum capacity have reduced blood pressure both acutely and in the medium term.\textsuperscript{3,5} Indeed, there is some suggestion that light to moderate exercise (35%–79% maximum heart rate) may be more effective at lowering blood pressure than high intensity exercise.\textsuperscript{6}

Health practitioners may feel more comfortable advising short episodes of light to moderate exercise to hypertensive patients, as these patients are likely to be at higher risk of cardiovascular events during vigorous exercise.\textsuperscript{6} Lighter intensity exercise is also associated with fewer musculoskeletal adverse effects. In addition, shorter episodes of lighter exercise may be more achievable and sustainable by patients than longer or more vigorous exercise.

The magnitude of blood pressure reductions with moderate exercise was also consistent with previous studies.\textsuperscript{6,7,10} If snacks of exercise could be achieved on a regular basis and if the resulting blood pressure reduction of 8.3/5.2 mmHg could be sustained, an individual’s cardiovascular risk would be reduced markedly, and the potential population effect would be significant if the message was delivered effectively.

In a review of nine major observational studies including 420,000 individuals with baseline diastolic blood pressures of between 70 and 110 mmHg, a reduction of 5 mmHg diastolic blood pressure was associated with reductions of 34% in the incidence of stroke and 21% in the incidence of coronary heart disease.\textsuperscript{17}

Adding physical activity to the treatment regime of hypertensive patients may also reduce the costs and adverse effects of anti-hypertensive medications as well as improving quality of life of patients.\textsuperscript{6} There is evidence that 10-minute episodes of brisk walking are adequate to improve psychological wellbeing.\textsuperscript{9}

The trend of greater reductions in diastolic blood pressure with short episodes of exercise than with long continuous episodes is interesting and not inconsistent with previous findings, although we do not know the regime adherence rates. Greater increases in VO$_2$ max were achieved with 3×10 minutes compared with 30 minutes of brisk walking several times per week in a previous crossover trial.\textsuperscript{9}

The implication for clinicians and public health advisers is that four 10-minute ‘snacks’ of moderate exercise daily are sufficient to lower blood pressure at least in the short term. The positive results from this study may encourage otherwise inactive individuals (who cannot do 40 minutes of continuous activity) to increase their physical activity and hence reduce their blood pressure with resulting health gains.
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References:


Dietary patterns of New Zealand European preschool children

Reremoana Theodore, John Thompson, Clare Wall, David Becroft, Elizabeth Robinson, Phillipa Clark, Jan Pryor, Chris Wild, Ed Mitchell

Abstract

Aims To record and describe the dietary patterns of a large group of New Zealand (NZ) European preschool children and to compare these with NZ Ministry of Health (MOH) food and nutrition guidelines.

Methods Mothers were interviewed when children enrolled in the Auckland Birthweight Collaborative (ABC) study were seen at 3.5 years of age. Approximately half of the children in the study were born small for gestational age (SGA ≤10th percentile) and the remaining were born appropriate for gestational age (AGA >10th percentile). Food frequency information was collected on 549 New Zealand European children. The analysis utilised weighting to allow for the disproportionate sampling of children born SGA.

Results Compared with nutritional guidelines, 27% and 54% of preschool children did not eat the recommended two or more servings of fruit per day or two or more servings of vegetables per day, respectively; 93% of children did not eat breads and cereals the recommended four or more times a day.

Conclusion A notable proportion of children were not eating fruit and vegetables at levels recommended by the MOH. Preschool children’s food frequency patterns were, however, similar to patterns reported for school-aged children in the National Children’s Nutrition Survey.

Diet during the early childhood years is important for growth, development, and health. Studies have found that undernutrition and vitamin deficiencies can lead to developmental problems including lower cognitive functioning\(^1\) and poor growth.\(^4\)\(^,\)\(^5\) Studies have also suggested that diets high in fat and sugar are related to increasing rates of obesity and obesity-related diseases in children and adolescents.\(^6\)\(^,\)\(^7\) The prevalence of overweight and obesity has increased worldwide in children and adolescents over the last 30 years.\(^8\)\(^-\)\(^10\) Lack of physical activity, as well as dietary patterns, are considered to be likely contributors to this global rise.\(^11\)\(^,\)\(^12\) Furthermore, childhood dietary habits are associated with later adult diet and health.\(^13\)\(^-\)\(^15\)

Nutritional research in New Zealand has focused on the first 2 years of life,\(^16\) or on children over 5 years of age.\(^17\) Little is known about what preschool New Zealand children eat. The New Zealand Ministry of Health (MOH) recommendations for preschool children include daily consumption of a variety of fruits and vegetables, lean meats and pulses, breads and cereals, and milk and dairy products.\(^18\) There is no information, however, on whether preschool children’s dietary patterns meet with MOH recommendations.
The aim of this study was to describe the dietary patterns of preschool children and compare these with the recommended daily intakes of key food groups. Dietary patterns of children in this study will be discussed in relation to the findings from the NZ 2002 National Children’s Nutrition Survey (CNS) on school-aged children.

Methods

Sample—Children in the study were those enrolled in the Auckland Birthweight Collaborative (ABC) study, which was principally a case-control study of risk factors related to being born small-for-gestational age (SGA). The ABC study design has been previously described in detail. In brief, approximately half of the children in the study were born SGA, weighing less than, or equal to, the sex-specific 10th percentile for gestational age. Controls were born appropriate-for-gestational age (AGA), weighing greater than the sex-specific 10th percentile for gestational age. All children were born at term, defined as 37 or more weeks of completed gestation.

Children were born between October 1995 and November 1997. The ABC study is a longitudinal study. At birth, 1714 mothers and children enrolled in the study. In data collected when the children were born, 871 mothers identified as being New Zealand European. Data have been collected at birth (Phase 1), at 1 year of age (Phase 2) and 3.5 years of age (Phase 3).

At Phase 3 of the study, 550 NZ European mothers and children were interviewed. The response rate for Māori, Pacific Island, and other non-European participants was low at 3.5 years. Analysis of the results of children in these groups was considered to be unrepresentative of children in the overall population. Analysis was therefore restricted to NZ European participants attending at Phase 3.

The ABC study was approved by the North Health Research Ethics Committee.

Food frequency information—An interviewer-administered food frequency questionnaire (FFQ) examining the frequency of consumption of a wide variety of commonly eaten foods was completed. The FFQ had been previously validated against a 4-day weighed food record and biochemical measures and showed good short-term repeatability. The FFQ was then adapted for children at 3.5 years of age and was comparable to the FFQ used in the CNS.

The majority of the questionnaire examined how often a child had eaten a food in the previous four-week period.

Response options were:
- Never;
- < Once per month;
- 1–3 times per month;
- 1 time per week;
- 2–4 times per week;
- 5–6 times per week;
- Once per day; and
- 2 or more times per day.

Information was also collected on the number of consumed daily standard servings of fruit and vegetables. Serving size examples used in the study were comparable to serving sizes defined in the NZ Ministry of Health guidelines—e.g., 1 apple, ½ cup of stewed fruit. Data collection over a 2-year period allowed for the seasonal variability in intake of food.

Statistical analysis—Analyses of the total sample employed weighting to adjust for the disproportionate sampling of children born SGA. The weighting accounts for the unequal selection probabilities of the SGA and AGA infants in this study, thus making the results representative of the total population of New Zealand European children aged 3.5 years. Food frequency information on 88 individual foods or drinks was converted to times eaten per month and combined to create overall food groups (e.g. fruit).

To convert data on foods into groups, the mid-point of the frequency options was taken for options such as 1–3 times per month, which was calculated as 2 times per month.
The percentage of children consuming (or not consuming) the New Zealand Ministry of Health (MOH) recommended intake for fruits and vegetables based on serving sizes was calculated.\textsuperscript{18}

The percentage of children consuming the following food groups in line with MOH recommendations were calculated: breads and cereals (including rice and pasta); meat, fish, chicken or eggs; milk and dairy products. For other food groups, the percentage of children eating from a food group daily or weekly was calculated.

The most commonly eaten type of food (e.g. potatoes) in overall food groups (e.g. vegetables) was calculated by ranking the percentage of children consuming each food per week. To assess the different variety of fruit and vegetable eaten weekly, individual fruits or vegetables eaten at least weekly were added.

The differences in food frequency between SGA and AGA children, between genders, and between those children taking, and not taking, vitamin and/or mineral supplements daily were assessed for 40 food variables, listed in Table 2, using $\chi^2$ statistics. The procedure ‘surveyfreq’ in SAS v9.1 (SAS Institute, Cary, NC) was used for analyses. Proc surveyfreq can be used for single-stage or multistage designs, with or without unequal weighting, and with or without stratification. This procedure uses the Taylor expansion method to estimate sampling errors of estimators based on complex sample designs (SAS statistics online manual).

Results

The anthropometric characteristics of the children at Phase 3 are shown in Table 1. Characteristics of the parents have been described previously.\textsuperscript{19} In brief, for mothers who attended Phase 3 of the study, mean maternal age at birth of the subject was 32 years of age; 14% of mothers smoked during pregnancy; mean maternal height was 166.4 cm (SE=0.35); mean maternal weight was 64.7 kg (SE=0.70); and mean maternal BMI was 23.2 (SE=0.24).

Table 1. Physical characteristics of the children in the study

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Male (n=261) Mean (SE)*</th>
<th>Female (n=288) Mean (SE)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>103.3 (0.33)</td>
<td>102.4 (0.29)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>17.6 (0.18)</td>
<td>17.1 (0.16)</td>
</tr>
<tr>
<td>BMI</td>
<td>16.4 (0.11)</td>
<td>16.3 (0.10)</td>
</tr>
</tbody>
</table>

*Weighted to account for disproportionate sampling; BMI=Body mass index (kg/m\textsuperscript{2}).

There was no difference in food frequency between SGA and AGA children for major food groups. SGA children were significantly less likely than AGA children to eat processed meats weekly (SGA 30% vs AGA 51%, $\chi^2 = 7.85$, p=0.005) and to drink water two or more times per day (SGA 22% vs AGA 36%, $\chi^2 = 4.46$, p=0.03). Food frequency results are shown for the total sample of children adjusted for disproportionate sampling.

Seventy-three percent of preschool children were reported as eating the recommended two or more servings of fruit per day, not including fruit juice (Table 2). In relation to food frequency information, 68% of preschool children ate fruit two or more times a day (Table 2). The MOH recommended vegetable servings of two or more a day were consumed by 46% of all children (Table 2). Males were significantly less likely than females to consume two or more servings of vegetables per day (males 41% vs females 52%, $\chi^2 = 3.98$, p=0.05). In relation to food frequency, 77% of preschool children had vegetables two or more times a day (Table 2). Males were significantly
less likely than females to eat two or more vegetables per day (males 71% vs female 81%, $\chi^2 = 4.96$, $p=0.03$).

Table 2. The percentage of children consuming specific types of foods or drinks by frequency

<table>
<thead>
<tr>
<th>Type of food</th>
<th>Frequency</th>
<th>% of children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruit*</td>
<td>≥2 servings a day</td>
<td>73</td>
</tr>
<tr>
<td>Fruit*</td>
<td>≥2 a day</td>
<td>68</td>
</tr>
<tr>
<td>Vegetables</td>
<td>≥2 servings a day</td>
<td>46</td>
</tr>
<tr>
<td>Vegetables</td>
<td>≥2 a day</td>
<td>77</td>
</tr>
<tr>
<td>Meat, chicken, fish, or eggs</td>
<td>≥1 a day</td>
<td>88</td>
</tr>
<tr>
<td>Red meat†</td>
<td>≥2 a week</td>
<td>73</td>
</tr>
<tr>
<td>Processed meat†</td>
<td>≥1 a day</td>
<td>14</td>
</tr>
<tr>
<td>Chicken</td>
<td>≥1 a week</td>
<td>90</td>
</tr>
<tr>
<td>Eggs</td>
<td>≥1 a week</td>
<td>73</td>
</tr>
<tr>
<td>Fish¶</td>
<td>≥1 a week</td>
<td>70</td>
</tr>
<tr>
<td>‘Oily’ fish***</td>
<td>≥1 a week</td>
<td>18</td>
</tr>
<tr>
<td>All milk†† and dairy products</td>
<td>≥2 a day</td>
<td>86</td>
</tr>
<tr>
<td>All milk††</td>
<td>≥1 a day</td>
<td>85</td>
</tr>
<tr>
<td>Drinking milk</td>
<td>≥1 a day</td>
<td>61</td>
</tr>
<tr>
<td>Standard milk</td>
<td>≥1 a day</td>
<td>41</td>
</tr>
<tr>
<td>Reduced fat and low-fat milk‡</td>
<td>≥1 a day</td>
<td>9</td>
</tr>
<tr>
<td>Dairy products§§</td>
<td>≥1 a day</td>
<td>75</td>
</tr>
<tr>
<td>All cereals, rice, pasta, and breads</td>
<td>≥4 a day</td>
<td>7</td>
</tr>
<tr>
<td>Breakfast cereals</td>
<td>≥1 a day</td>
<td>44</td>
</tr>
<tr>
<td>Bread</td>
<td>≥1 a day</td>
<td>79</td>
</tr>
<tr>
<td>Rice</td>
<td>≥1 a week</td>
<td>71</td>
</tr>
<tr>
<td>Pasta</td>
<td>≥1 a week</td>
<td>65</td>
</tr>
<tr>
<td>Butter</td>
<td>≥1 a day</td>
<td>34</td>
</tr>
<tr>
<td>Margarine</td>
<td>≥1 a day</td>
<td>48</td>
</tr>
<tr>
<td>“Treat” foods¶¶</td>
<td>≥1 a day</td>
<td>85</td>
</tr>
<tr>
<td>“Treat” foods¶¶</td>
<td>≥2 a day</td>
<td>40</td>
</tr>
<tr>
<td>“Treat” foods¶¶</td>
<td>≥3 a day</td>
<td>12</td>
</tr>
<tr>
<td>Chips</td>
<td>≥1 a week</td>
<td>68</td>
</tr>
<tr>
<td>Candy bars</td>
<td>≥1 a week</td>
<td>52</td>
</tr>
<tr>
<td>Muesli bars</td>
<td>≥1 a week</td>
<td>55</td>
</tr>
<tr>
<td>Biscuits &amp; cakes</td>
<td>≥1 a day</td>
<td>56</td>
</tr>
<tr>
<td>Water</td>
<td>≥1 a day</td>
<td>82</td>
</tr>
<tr>
<td>Water</td>
<td>≥2 a day</td>
<td>60</td>
</tr>
<tr>
<td>Fruit juice</td>
<td>≥1 a day</td>
<td>30</td>
</tr>
<tr>
<td>Cordial</td>
<td>≥1 a day</td>
<td>36</td>
</tr>
<tr>
<td>Soft drinks</td>
<td>≥1 a day</td>
<td>4</td>
</tr>
<tr>
<td>Soft drinks</td>
<td>≥3 a week</td>
<td>24</td>
</tr>
<tr>
<td>Dietary supplements ***</td>
<td>≥1 a day</td>
<td>24</td>
</tr>
<tr>
<td>Dietary supplements ***</td>
<td>≥1 a week</td>
<td>39</td>
</tr>
<tr>
<td>All nuts†††</td>
<td>≥1 a week</td>
<td>24</td>
</tr>
</tbody>
</table>

* Does not include fruit juice; † MOH recommended number of servings
Eighty-eight percent of children ate meat, fish, eggs, or chicken at least daily, as recommended by MOH (Table 2). Seventy-three percent of children had red meat at least twice weekly.

Eighty-six percent of children consumed dairy products or milk at least twice daily, in line with MOH recommendations (Table 2). Milk was consumed daily by 85% of children and dairy products by 75% of children.

Seven percent of children ate breads, cereals, rice, or pasta at least four times a day as recommended (Table 2). The percentage of children eating bread and breakfast cereals at least daily was 79% and 44% respectively.

Total treat foods (including cakes, biscuits, chips, candy bars, and muesli bars) were consumed at least daily by 85% of children (Table 2). Twelve percent of children ate treat foods three or more times daily.

Eighty-two percent of children drank water daily (Table 2). Fruit juice and cordial were consumed daily by 30% and 36% of children respectively. Soft drinks were consumed three or more times a week by nearly one-quarter of children (24%).

Despite the recommendation that dietary supplements should not be generally given to children, nearly one-quarter of children (24%) were taking vitamin and/or mineral supplements daily, and 39% were taking a dietary supplement at least once a week (Table 2). Children taking dietary supplements daily were significantly less likely (than those not taking dietary supplements daily) to consume milk or dairy products at least two times per day (79% vs 88%, $\chi^2 = 3.71$, $p=0.05$).

The most commonly consumed foods are described in Table 3. Apples and pears were the most commonly eaten fruit, consumed weekly by 95% of children (Table 3). The most commonly eaten vegetable was potato (Table 3). Standard milk (approximately 3% fat) was the most commonly consumed milk drink (Table 3).

There was no difference between the proportion of male and females drinking standard milk, however, females were significantly more likely to drink reduced-fat (1.5% fat) and low-fat (0.5% fat) milk than males (females 12% vs males 6%, $\chi^2 = 4.39$, $p=0.04$).

The mean number of different types of fruit consumed per week was 4.65 (SE=0.10). The mean number of different types of vegetables eaten per week was 6.53 (SE=0.16).
Table 3. Food frequency information showing the percentage of children consuming specific types of foods or drinks at least once per week

<table>
<thead>
<tr>
<th>Type of food</th>
<th>%</th>
<th>Type of food</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruit</td>
<td></td>
<td>Meat</td>
<td></td>
</tr>
<tr>
<td>Apples/pears</td>
<td>95</td>
<td>Chicken</td>
<td>90</td>
</tr>
<tr>
<td>Bananas</td>
<td>79</td>
<td>Beef/pork/lamb as main dish</td>
<td>75</td>
</tr>
<tr>
<td>Oranges</td>
<td>67</td>
<td>Bacon and ham</td>
<td>67</td>
</tr>
<tr>
<td>Dried fruit</td>
<td>60</td>
<td>Processed meats (e.g. salami)</td>
<td>64</td>
</tr>
<tr>
<td>Grapes</td>
<td>33</td>
<td>Beef/pork/lamb as part of dish</td>
<td>55</td>
</tr>
<tr>
<td>Kiwi</td>
<td>26</td>
<td>Hamburger</td>
<td>32</td>
</tr>
<tr>
<td>Stone fruit (e.g. plums)</td>
<td>26</td>
<td>Liver</td>
<td>1</td>
</tr>
<tr>
<td>Canned fruit in juice</td>
<td>24</td>
<td>Breads</td>
<td></td>
</tr>
<tr>
<td>Berries</td>
<td>23</td>
<td>White</td>
<td>78</td>
</tr>
<tr>
<td>Canned fruit in syrup</td>
<td>13</td>
<td>Mixed grain</td>
<td>60</td>
</tr>
<tr>
<td>Feijoas</td>
<td>11</td>
<td>Omega-3</td>
<td>5</td>
</tr>
<tr>
<td>Pineapple</td>
<td>10</td>
<td>Dairy products</td>
<td></td>
</tr>
<tr>
<td>Vegetables</td>
<td></td>
<td>Cheese</td>
<td>91</td>
</tr>
<tr>
<td>Potatoes</td>
<td>92</td>
<td>Yoghurt</td>
<td>80</td>
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<tr>
<td>Carrots</td>
<td>86</td>
<td>Ice cream</td>
<td>75</td>
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<tr>
<td>Broccoli</td>
<td>68</td>
<td>Milk drinks</td>
<td></td>
</tr>
<tr>
<td>Peas</td>
<td>58</td>
<td>Standard milk (3% fat)</td>
<td>56</td>
</tr>
<tr>
<td>Tomatoes</td>
<td>49</td>
<td>Milkshakes and flavoured milk</td>
<td>50</td>
</tr>
<tr>
<td>Sweet corn</td>
<td>41</td>
<td>Reduced fat (1.5% fat) milk</td>
<td>14</td>
</tr>
<tr>
<td>Cauliflower</td>
<td>36</td>
<td>Low fat (0.5% fat) milk</td>
<td>5</td>
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<tr>
<td>Kumara</td>
<td>35</td>
<td>Drinks</td>
<td></td>
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<tr>
<td>Other green leafy vegetables</td>
<td>35</td>
<td>Water</td>
<td>94</td>
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<tr>
<td>Beans</td>
<td>33</td>
<td>Juice</td>
<td>71</td>
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<td>Pumpkin</td>
<td>33</td>
<td>Cordial</td>
<td>53</td>
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<tr>
<td>Mixed vegetables</td>
<td>31</td>
<td>Soft drinks</td>
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<td>Cucumber</td>
<td>30</td>
<td>Tea</td>
<td>4</td>
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<td>Spinach</td>
<td>24</td>
<td>Coffee</td>
<td>2</td>
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<tr>
<td>Peppers (all colours)</td>
<td>17</td>
<td>Dietary supplements</td>
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<tr>
<td>Celery</td>
<td>13</td>
<td>General multivitamins</td>
<td>22</td>
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<tr>
<td>Sprouts</td>
<td>4</td>
<td>Vitamin C</td>
<td>22</td>
</tr>
<tr>
<td>Breakfast cereal</td>
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<td>Iron</td>
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<tr>
<td>Cereal (e.g. cornflakes)</td>
<td>89</td>
<td>Halibut oil</td>
<td>1</td>
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<tr>
<td>Porridge</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muesli</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High-bran weetbix</td>
<td>5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Does not include drinking milk

Discussion

This study provides a unique description of what NZ preschool children eat, and research has not been previously undertaken on this scale within this particular age group.

In this study, more than one-quarter of preschool children were not eating the recommended daily intake of fruit servings, similar to the number of children reported as not eating fruit two or more times per day. The small discrepancy between the reported servings of fruit consumed and reported fruit frequency (5%) may be due to children eating less commonly consumed fruit that were not listed in the FFQ.
The proportion of children in this study eating fruit at least twice daily was higher than that reported for school-aged NZ European and other children (non-Māori and non-Pacific Island) in the CNS. Our results are consistent with the finding that younger children consume fruit more regularly than older children. More than half of preschool children were not consuming the recommended daily intake of vegetable servings. A higher percentage of children (77%) were reported as eating two or more vegetables per day based on food frequency information. This finding suggests that not all children are consuming whole servings of vegetables. Of concern is the small proportion of children eating breads or cereals (including pasta and rice) at recommended levels. These foods are high in energy and are a significant contributor of dietary folate and iron for NZ children. The CNS found that although bread intake did not vary with age, the frequency of intake of breakfast cereals declined with age. Most children ate meat, fish, chicken, or eggs daily. The most frequently eaten meat was chicken, a finding consistent with the 2002 nutrition survey. Nearly two-thirds of children drank milk daily, a higher proportion than NZ school-aged children; this is consistent with the CNS finding that milk consumption decreases with age. Several studies have found a positive relationship between soft-drink consumption and obesity, and body mass index. Soft-drink consumption has been found to be positively related to increased daily energy intake and may displace the consumption of other drinks, such as milk and juice. Of concern, nearly 25% of preschool children in this study were drinking soft drinks three or more times a week. Limiting the consumption of these drinks in preschool children may be important given the increasing rates of childhood obesity in New Zealand. NZ guidelines recommend that “treat” foods, such as muesli bars and potato chips, be eaten only occasionally. Australian guidelines are more specific for these “extra” foods, recommending no more than one or two servings per day. We found that 12% of preschool children ate these foods three or more times a day. These foods tend to be energy dense and low in micronutrients. In this study, preschool boys were less likely to eat vegetables at recommended levels than girls. Boys were also less likely than girls to consume reduced-fat milk and low-fat milk. These gender differences in dietary patterns are similar to those found in NZ adults. SGA children were less likely than AGA children to eat processed meats weekly and to drink water two or more times per day. Due to numerous comparisons, caution should be taken in interpreting these results. This study found no differences in dietary patterns between those children taking dietary supplements, and those not taking supplements, except for milk and dairy product consumption. (Supplements are generally not recommended for New Zealand children.) This study’s limitations need to be addressed. Firstly, diet is strongly associated with socioeconomic status and parental education. Previous ABC study research has found that NZ European mothers attending at Phase 3 of the study were more likely to have higher socioeconomic status, a tertiary education, be older, and less likely to have smoked during pregnancy than non-respondents. It is therefore likely that our findings on food frequency are conservative. The proportion of children in the general...
population eating fruit, vegetables, breads, and cereals at recommended levels is likely to be lower than reported in this study. Secondly, these findings are restricted to NZ European children. Future research is needed to examine the diet of New Zealand preschool children of other ethnic groups.

In conclusion, these results suggest that preschool children are not eating the recommended number of vegetables, fruits, and breads/cereals. However, there is limited information on preschool nutrition in New Zealand, and interpretation of these results should be undertaken with some caution until further studies have been completed.

Assessing and describing the diet and nutrient intakes of New Zealand preschool children is an area that needs attention, as diet in early childhood is likely to impact on later adult diet and health.

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**References:**


Epidemiology of diabetes in New Zealand: revisit to a changing landscape

Grace Joshy, David Simmons

Abstract

Aim The aim of this review is to describe the evolution of the burden of diabetes, its risk factors and complications in New Zealand, and the current national strategies underway to tackle a condition likely to impact on the national ability to afford other health services.

Methods The MEDLINE database from 1990 was searched for New Zealand-specific diabetes studies. The Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) Reports from 1990–2004 and Ministry of Health (MoH) publications and reports were also reviewed. Key contact people working in the field of diabetes care in every district health board (DHB) were contacted, and information on current initiatives for diabetes control and prevention were collected.

Results The prevalence of diabetes (known and undiagnosed), impaired glucose tolerance (IGT) / impaired fasting glucose (IFG) and gestational diabetes are tabulated by ethnic group. The latest New Zealand Health Survey (NZHS) result of known diabetes: European 2.9%, Māori 8%, Pacific 10.1%, Asian 8.4%. Diabetes risk factors have been examined and the reported rates have been compiled. Māori and Pacific people have a particularly high prevalence of diabetes risk factors (e.g. obesity, physical inactivity, insulin resistance, metabolic syndrome) compared with Europeans. The profile of diabetic patients in New Zealand has been summarised using publications on their clinical characteristics. The latest available data on ethnic specific clinical characteristics are a decade old. With the suboptimal participation in the Get Checked program: 63% Europeans/Others, 27% Māori, 92% Pacific (possibly overestimated) people in 2004, the results may not be representative. The burden of diabetes complications and diabetes related mortality has been reviewed. A high proportion of Māori and Pacific dialysis patients and new renal disease patients from the ANZDATA registry have diabetes comorbidity. The inadequacy of official statistics in New Zealand and the scarcity of indepth studies across the country, including ethnic perspectives, has been clearly demonstrated.

Conclusions While the diabetes epidemic has continued to impact increasingly on New Zealanders and its health services over the past 5 years, a growing number of Government and DHB-funded initiatives are in place to prevent diabetes and its complications. A nationally agreed strategic plan is now urgently needed on how best to monitor and control the increasing incidence and prevalence of diabetes in the New Zealand population as well as the proportion with undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose.

Almost a decade has passed since Simmons'¹,² painted the portrait of diabetes epidemiology in New Zealand and warned about the increasing risk of diabetes and its complications, especially for Māori and Pacific peoples. When Moore and Lunt ³ re-
examined the situation in 2000, they found the burden of diabetes and its complications escalating, especially end-stage renal failure (ESRF). They also noted the ageing population structure, increasing Pacific population (mostly of Samoan, Tongan, Niuean, and Cook Islands origin), and the obesity epidemic. Since this time, New Zealand’s population has continued to age (median age has increased 2.5 years over 10 years). Furthermore, it has grown by 6%, with a 40% increase in the Asian population (2001–2005). These figures point to an increasing Type 2 diabetes burden for New Zealand.

The New Zealand Ministry of Health has responded to the growing diabetes epidemic with a diabetes strategic plan in 1997, a Diabetes Implementation Plan in 2000, and a “Diabetes Toolkit” for district health boards (DHBs) in 2001. The latter included the establishment of Local Diabetes Teams at DHB level and the free annual Get Checked programme for diabetes patients. A set of guidelines for the management of Type 2 diabetes were released in 2003. A Ministry of Health/Health Research Council grant was put out to tender in 2001 and again in 2003, which was subsequently awarded to the Te Wai o Rona: Diabetes Prevention Strategy team in the Waikato/Lakes districts.

Results from a large number of important studies have been published since the last review, which have confirmed the picture of a disease increasing in numbers, especially at a younger age and consistent with a lowering of the age at onset of Type 2 diabetes.

The aim of this review is to describe the current burden of diabetes and the current district-based strategies underway to tackle a condition likely to impact on the ability of New Zealand to afford other health services.

Methods

A comprehensive review was undertaken using MEDLINE database, reviewing diabetes prevalence or complications studies/surveys reporting New Zealand-specific figures. Experimental intervention trials have been excluded. The Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) Reports from 1990–2004, the MoH publications/reports, and New Zealand Society for Study of Diabetes conference abstract books from 2000 have been reviewed.

The latest unpublished results from the Get Checked programme, being the only national diabetes surveillance tool, were obtained from the MoH. The diabetes teams in all DHBs were consulted via email regarding current (unpublished) initiatives on diabetes control and prevention (10/21, 48% response). While a comprehensive attempt has been made to include current unpublished diabetes initiatives, there could be a limitation on the number of such initiatives included in this article due to the limited response.

Prevalence of diabetes

As with many other countries, currently there are no up-to-date national diabetes prevalence data for New Zealand. The only area with comprehensive epidemiological data is South Auckland, where between 1991 and 1995 a household survey of 100,000 residents was undertaken with a nested study of those with undiagnosed diabetes undertaken thereafter.

Table 1 shows the prevalence of diagnosed and undiagnosed diabetes in different population-based surveys by ethnic group. As no significant and consistent gender differences in prevalence have been found, prevalence data have been integrated.
### Table 1. Prevalence (%) of known diabetes, undiagnosed diabetes, and IGT/IFG in New Zealand by ethnicity

<table>
<thead>
<tr>
<th>Variable</th>
<th>European</th>
<th>Māori</th>
<th>Pacific</th>
<th>Asian</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prevalence (%) of known diabetes—all ages</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Christchurch Workforce Survey</td>
<td>1982-83&lt;sup&gt;1&lt;/sup&gt;</td>
<td>≥15</td>
<td>2.78%</td>
<td>11.27%</td>
</tr>
<tr>
<td>SADP household survey&lt;sup&gt;11&lt;/sup&gt;</td>
<td>1992–95&lt;sup&gt;*&lt;/sup&gt;</td>
<td>All ages</td>
<td>1.86%</td>
<td>5.21%</td>
</tr>
<tr>
<td>New Zealand Health Survey&lt;sup&gt;12&lt;/sup&gt;</td>
<td>1996/97&lt;sup&gt;†&lt;/sup&gt;</td>
<td>≥15</td>
<td>3.10%</td>
<td>8.30%</td>
</tr>
<tr>
<td>New Zealand Health Survey&lt;sup&gt;13&lt;/sup&gt;</td>
<td>2002/03&lt;sup&gt;‡&lt;/sup&gt;</td>
<td>≥15</td>
<td>2.9%</td>
<td>8%</td>
</tr>
<tr>
<td>Pacific Study&lt;sup&gt;14&lt;/sup&gt;</td>
<td>1996&lt;sup&gt;*&lt;/sup&gt;</td>
<td>≥20</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Ngatai Porou Hauora Register&lt;sup&gt;15&lt;/sup&gt;</td>
<td>2003&lt;sup&gt;*&lt;/sup&gt;</td>
<td>≥25</td>
<td>–</td>
<td>7.1%</td>
</tr>
<tr>
<td>Northland Survey&lt;sup&gt;16&lt;/sup&gt;</td>
<td>2003</td>
<td>6% (no ethnic specific data reported)</td>
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<th>Pacific</th>
<th>Asian</th>
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<tr>
<td><strong>Prevalence (%) of known diabetes—40+ age group</strong></td>
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<td></td>
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<tr>
<td>Auckland Workforce</td>
<td>1990&lt;sup&gt;a&lt;/sup&gt;</td>
<td>40–64</td>
<td>1.06%</td>
<td>5.26%</td>
</tr>
<tr>
<td>Christchurch elderly</td>
<td>1991</td>
<td>≥65</td>
<td>10%</td>
<td>–</td>
</tr>
<tr>
<td>Auckland Surgical Ward&lt;sup&gt;19&lt;/sup&gt;</td>
<td>1990–91</td>
<td>40–59</td>
<td>6.0%</td>
<td>18.3%</td>
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<tr>
<td></td>
<td></td>
<td>60–69</td>
<td>7.9%</td>
<td>31.7%</td>
</tr>
<tr>
<td>SADP household survey&lt;sup&gt;11&lt;/sup&gt;</td>
<td>1992–95</td>
<td>40–49</td>
<td>1.5%</td>
<td>6.8%</td>
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<td>50–59</td>
<td>3.8%</td>
<td>13.1%</td>
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<td>60–69</td>
<td>5.6%</td>
<td>15.0%</td>
</tr>
<tr>
<td>Pacific Study&lt;sup&gt;14&lt;/sup&gt;</td>
<td>1996</td>
<td>40–49</td>
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<td>–</td>
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<td>50–59</td>
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<th>Pacific</th>
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<td><strong>Prevalence of diabetes in other subgroups</strong></td>
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<td></td>
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<td></td>
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<tr>
<td>Christchurch, Type 1</td>
<td>2005</td>
<td>&lt;25</td>
<td>274</td>
<td>81</td>
</tr>
<tr>
<td>(prevalence/100,000)</td>
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<td></td>
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<tr>
<td>Gestational diabetes&lt;sup&gt;21&lt;/sup&gt;</td>
<td>1994–95</td>
<td>3.3%</td>
<td>7.9%</td>
<td>8.1%</td>
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<tr>
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<th>European</th>
<th>Māori</th>
<th>Pacific</th>
<th>Asian</th>
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<tr>
<td><strong>Prevalence % of Undiagnosed Diabetes (percentage of total diabetes)</strong></td>
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<td></td>
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<tr>
<td>Dunedin General Practice&lt;sup&gt;22&lt;/sup&gt;</td>
<td>1990</td>
<td>50–69</td>
<td>(20)</td>
<td></td>
</tr>
<tr>
<td>Christchurch elderly&lt;sup&gt;18&lt;/sup&gt;</td>
<td>1991</td>
<td>≥65</td>
<td>4.0 (30)</td>
<td></td>
</tr>
<tr>
<td>Waikato Discover Diabetes</td>
<td>1993</td>
<td>40–59</td>
<td>0.8</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60–79</td>
<td>2.1</td>
<td>6.1</td>
</tr>
<tr>
<td>Auckland Workforce Survey&lt;sup&gt;17&lt;/sup&gt;</td>
<td>1990</td>
<td>40–64</td>
<td>0.8 (42)</td>
<td>4.64 (48)</td>
</tr>
<tr>
<td>SADP&lt;sup&gt;23&lt;/sup&gt;</td>
<td>1996</td>
<td>40–59</td>
<td>3.3 (30)</td>
<td>10.6 (48)</td>
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<td></td>
<td></td>
<td>60–79</td>
<td>2.7 (24)</td>
<td>7.9 (33)</td>
</tr>
<tr>
<td>Te Wai o Rona, Waikato&lt;sup&gt;24&lt;/sup&gt;</td>
<td>2004</td>
<td>45–64</td>
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<th>Māori</th>
<th>Pacific</th>
<th>Asian</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prevalence (%) of IGT/IFG</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland Workforce Survey&lt;sup&gt;17&lt;/sup&gt;</td>
<td>1990</td>
<td>40–64</td>
<td>1.93</td>
<td>7.40</td>
</tr>
<tr>
<td>SADP&lt;sup&gt;23&lt;/sup&gt;</td>
<td>1996</td>
<td>40–59</td>
<td>7.4</td>
<td>22.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60–79</td>
<td>22.1</td>
<td>22.3</td>
</tr>
<tr>
<td>Te Wai o Rona, 2004&lt;sup&gt;24&lt;/sup&gt;</td>
<td>2004</td>
<td>45–64</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

* Age standardised; † Crude prevalence, Europeans include Asians, Māori include Polynesians; ‡ Asians include others; † Europeans include others; SADP=South Auckland Diabetes Project.

The SADP survey<sup>11</sup> found a high prevalence of diabetes in the non-European populations of New Zealand (except in Chinese and Cambodians); the highest prevalence was found in South Asians—e.g. Asian Indians. (Figure 1). The
prevalence of diabetes among Chinese was also low on the Middlemore Hospital surgical wards at this time and has been shown in other Chinese populations. The NZHS 2002/03 results showed an increased diabetes prevalence of 8.4% among Asians living in New Zealand when compared with 1996/7, although South Asians were also included in the Asian category.

Figure 1. Prevalence (%) of known (Type 1 and Type 2) diabetes among 40–49 year olds in South Auckland (by ethnic group)

Source: South Auckland Diabetes Project (SADP) survey, 1992–95. Middle East=e.g. Iranians, Iraqis, Egyptians; South Asians=e.g. Indians, Sri Lankans, Bangladeshi.

Compared with Europeans aged ≥40 years, the prevalence of undiagnosed diabetes is more than three-fold among Māori and more than four-fold among Pacific peoples. HbA1c screening of 50,819 subjects aged 20+ years found that Māori, Pacific people, and Indians had particularly high rates of elevated HbA1c.

The age-standardised proportion of individuals with HbA1c >6% in these ethnic groups were increased six-fold. Preliminary results from Te Wai o Rona Diabetes Prevention Strategy in the Waikato are consistent with the South Auckland data, but the age-specific prevalence of undiagnosed diabetes was greater than predicted in the younger age groups.

Risk factor screening is still recommended in New Zealand, although many of those with undiagnosed diabetes (25.0%) and dysglycaemia (31.4%) have no diabetes risk factors. Past studies have indicated the earlier onset of Type 2 diabetes in Māori (8–10 years earlier) and Pacific people (5–9 years earlier) than Europeans. The NZHS 1996/97 figures are in agreement with the results from the SADP survey regarding age at diabetes diagnosis among Europeans (50–55.5 years), Māori (41–43
years), and Pacific (45–47 years)—but the NZHS 2002/03 results for Māori and Pacific are contradictory (50 and 51 years respectively).

About 10–15% of diagnosed diabetes is Type 1 diabetes among European New Zealanders; it is approximately 5% among other ethnic groups. Of concern, the incidence of Type 1 diabetes diagnosed before 20 years of age in Canterbury, New Zealand has increased 3.4-fold in 30 years—from 6.79 to 22.79 patients/100,000 per year starting from 1970. This increase is considered consistent with a worldwide increase in Type 1 diabetes.

In the most recent national study, Campbell-Stokes et al estimated the average annual incidence in 1999/2000 to be 17.9 per 100,000 (95% CI: 15.9–20.0) among children under 15 years. Unlike earlier studies, this study found that Māori, Pacific people, and Asians all had significantly lower incidence rates (both absolute and relative to their respective population proportions) than Europeans, although the basis of the ethnicity definition is not stated.

Although the prevalence of Type 1 diabetes was found to be lower in non-Europeans in a recent Christchurch study, they also noted the increasing number of Māori, Pacific, and Asian people with diabetes.

**Figure 2. The changing epidemiology of diabetes in New Zealand: 1991–2011**

*indicates numbers, and proportions by ethnicity, of people with diabetes*

**Sources:**
- 2003 – NZHS 2002/03 (*Europeans include Others).

Figure 2 shows the projected numbers with known diabetes by ethnic group across all surveys to date, although the cross comparisons are limited by the changing definitions of ethnicity and diabetes. Figure 2 also shows the different MOH diabetes forecasts for New Zealand (based upon the NZHS 96–97 and South Auckland Household Survey 91), which may be underestimates (e.g. the 2003 predictions were
already less than the prevalence of diabetes among Europeans, Pacific peoples, and Māori males in the NZHS 2002/03 survey).

The age at onset of Type 2 diabetes has also been dropping, with increasing numbers of children and adolescents with Type 2 diabetes and women with Type 2 diabetes in pregnancy. The Auckland Diabetes Centre has reported increasing prevalence of Type 2 diabetes in adolescents. The prevalence of Type 2 diabetes among the adolescent clinic attendees was 1.8% in 1996, and 11.0% in 2002.

Northland Diabetes Service has reported that Type 2 diabetes presents before the age of 30 years in 2.66% of Māori diagnosed with diabetes. Among South Auckland women with gestational diabetes mellitus (GDM), a high proportion (4.3% European, 21% Māori, 21% Pacific) of Polynesians had permanent diabetes postnatally.

Gestational diabetes

A review of 1994/95 hospital records in South Auckland showed high rates of GDM in Māori and Pacific women who attended oral glucose challenge tests compared with Europeans. This study found that Pacific women were more likely to be screened (68.5%) when compared with Māori (47.3%) when both have high rates of GDM and Type 2 diabetes.

Risk factors for diabetes

The prevalence of obesity in New Zealand has increased from 9.4% in 1977 to 19.9% in 2003 among males, and from 10.8% to 22.1% among females. Māori and Pacific people have a particularly high prevalence of obesity, physical inactivity, insulin resistance, and metabolic syndrome compared with Europeans (Table 2).

The association between body composition and central fat distribution with risk of diabetes appears to be independent of ethnicity. While Asians appear to have comparatively lower obesity, Rush et al have found high body fat composition for Asian Indians compared with Europeans for a given BMI.

Complications

Table 3 shows the risk factors for microvascular and macrovascular disease in the New Zealand studies to date. The poor glycaemic and lipid control among patients attending the clinic from 1992–95 appears to have continued into this century. The Otago register has reported a mean HbA1c of 7.2% for Type 2 patients; 50.1% had HbA1c result >7% in 1998. The results of the Get Checked programme showed that 63% of Europeans, 27% of Māori, and 92% of Pacific people with diabetes had a free annual check in 2004 (personal communication, MoH). But the denominators are derived from the MOH forecast estimates and actual percentage of Pacific people getting free checks may be much lower. The 2004 results show poorer metabolic control (HbA1c > 8%), for Māori (40%) and Pacific people (51%) with compared with European/Other (23%).
Table 2. Prevalence of risk factors for diabetes and its complications

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Study/Survey</th>
<th>European</th>
<th>Māori</th>
<th>Pacific</th>
<th>Asian</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic syndrome (%) by ATP III criteriaa</td>
<td>SADP 1996, 40–59 years:</td>
<td>24.6</td>
<td>52.8</td>
<td>48.5</td>
<td>45.5</td>
</tr>
<tr>
<td></td>
<td>Males</td>
<td>13.4</td>
<td>51.8</td>
<td>45.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Females</td>
<td>13.4</td>
<td>51.8</td>
<td>45.5</td>
<td></td>
</tr>
<tr>
<td>Insulin resistance (%)</td>
<td>East Coast 2003b; 25–29 years; 30–39 year</td>
<td></td>
<td>43</td>
<td></td>
<td>44</td>
</tr>
<tr>
<td>Sedentary (%)</td>
<td>NZHS 1996/97c</td>
<td>14.7</td>
<td>19.8</td>
<td>14.1</td>
<td>20.5</td>
</tr>
<tr>
<td></td>
<td>NZHS 2002/03d</td>
<td>11.2</td>
<td>12.6</td>
<td>17.8</td>
<td>22.3</td>
</tr>
<tr>
<td></td>
<td>SPARC Survey1997–01f: 5–17 years; ≥18 years</td>
<td>8.0</td>
<td>10.0</td>
<td>19.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Obesity (%)</td>
<td>NZHS 2002/03g, h</td>
<td>18.9</td>
<td>28.3</td>
<td>43.0</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>Church study</td>
<td></td>
<td></td>
<td>60</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking (%)</td>
<td>NZHS 2002/03i, j</td>
<td>21</td>
<td>47</td>
<td>33</td>
<td>11</td>
</tr>
</tbody>
</table>

*Age standardised.
European includes Other.
The ATP III criteria for metabolic syndrome were considered to have been met when 3 or more of the following factors were present: waist circumference >102cm for men or >88cm for women, treated hypertension or systolic blood pressure (sBP) ≥130mmHg and/or diastolic blood pressure (dBP) ≥85mmHg as mean of two readings, triglycerides ≥1.7mmol/L, HDL <1.04mmol/L for men or <1.29mmol/L for women, fasting blood glucose (FBG) ≥26.1mmol/L or diabetes.
Obesity is body mass index (BMI) ≥30 for European/Other/Asian, BMI ≥32 for Māori/Pacific.
Asian includes Other.

Table 3. Clinical characteristics of diabetes patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Waikato Diabetes Clinic1992-95</th>
<th>South Auckland Survey 1996</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Control</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>Type 1</td>
<td>Type 2 - Ist</td>
</tr>
<tr>
<td>Random blood glucose (mmol/L)</td>
<td>376 ± 78</td>
<td>321 ± 73</td>
</tr>
<tr>
<td>Fructosamine (mmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>5.2 ± 1.1</td>
<td>5.8 ± 1.2</td>
</tr>
<tr>
<td>HDL (mmol/L)</td>
<td>1.5 ± 0.5</td>
<td>1.1 ± 0.3</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.7 ± 1.4</td>
<td>3.1 ± 3.6</td>
</tr>
<tr>
<td>Renal Characteristics</td>
<td>Albumin creatinine ratio (mg/day)</td>
<td>2.18</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Systolic (mmHg)</td>
<td>141 ± 25</td>
</tr>
<tr>
<td>Diastolic (mmHg)</td>
<td>81 ± 12</td>
<td>84 ± 13</td>
</tr>
<tr>
<td>% on anti-hypertensive medication (complication free cohort)</td>
<td>44%</td>
<td>33%</td>
</tr>
<tr>
<td>Physical Characteristics</td>
<td>BMI (kg/m2)</td>
<td>25.6 ± 4.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30.5 ± 6.6</td>
</tr>
</tbody>
</table>

Data are mean ± SD unless otherwise stated; * Transferred from diet/pill to insulin; † Data are geometric mean.
Diabetes-related mortality

NZHIS mortality data has attributed 3% of deaths in 2000 to diabetes.\(^{55}\) Difficulties in the coding of diabetes have been recognised for many years,\(^{49}\) yet continue to be rediscovered\(^{56}\)\(^{57}\) with 45%–55% under-coding especially among non-insulin using (Type 2) patients.

In spite of inadequate mortality statistics, the standardised mortality rate for diabetes mellitus during 1999 were 62.5 per 100,000 in Maori versus 11 in non-Maori.\(^{58}\) A 10-year follow-up of the predominantly European Type 2 diabetic cohort in Canterbury showed increased mortality (standardised mortality ratio 2.17), the cause of death being predominantly attributable to cardiovascular disease (CVD) (69.8%).\(^{59}\)

The Canterbury insulin-treated Diabetic Registry has reported a CVD-related standardised mortality ratio of 4.48 for diagnosis age <30 years and 2.05 for diagnosis age ≥30 years among those who commenced insulin within 12 months of diagnosis.\(^{60}\)

The meta-analysis of studies from Asia Pacific region (including 10,326 subjects from New Zealand) revealed that the hazard ratio associated with diabetes was significantly higher for fatal cardiovascular disease (1.97), fatal coronary heart disease (2.19,) and fatal cerebrovascular disease (2.0).\(^{61}\)

Table 4 shows the ethnic specific death rates from ESRD and ischaemic heart disease in the SADP cohort age 40–79 years.\(^{49}\) The standardised mortality ratio for renal failure is 8.37%, estimated from the Canterbury insulin-treated Diabetic Registry.\(^{60}\) This reflects the renal failure rate in insulin treated diabetes patients in a registry that has predominantly European patients (97.7%).

Cardiovascular and cerebrovascular diseases

Very few reports relating to heart disease exist (Table 4). A review of records from Middlemore Hospital has reported significant ethnic differences in the prevalence of diabetes among in-patients aged 40+ with acute MI.\(^{52}\)

Diabetic nephropathy

Among the 449 new renal disease patients entering the ANZDATA registry in 2003,\(^{54}\) 45% had diabetes (23% of European patients, 65% Maori, 67% Pacific, 50% Asian).

Diabetic nephropathy (40%) was the most common cause of end-stage renal disease (ESRD) in New Zealand, followed by glomerulonephritis (26%) and hypertension (10%). Type 2 diabetes (non-insulin and insulin requiring) was identified in 94% of diabetic nephropathic patients on the registry.

From the prospective data from ANZDATA reports, the numbers with of diabetes-related ESRD in Maori population are the highest, but appear to have reached equilibrium (Figure 3). The incidence of diabetes related ESRD in Europeans while lower than other ethnic groups, has also doubled since 1992. The crude prevalence of proteinuria and ESRD were higher in Maori and Pacific people compared with Europeans in the SADS survey\(^{62}\) in 1990 (Table 4).
Table 4. Diabetes-related mortality and complications

<table>
<thead>
<tr>
<th>mortality</th>
<th>European</th>
<th>Maori</th>
<th>Pacific</th>
<th>Asian</th>
</tr>
</thead>
<tbody>
<tr>
<td>Five-year mortality rates among Type 2 diabetes patients aged 40–79 in 1991†</td>
<td>16.3%</td>
<td>26.2%</td>
<td>16.8%</td>
<td></td>
</tr>
<tr>
<td>For ischaemic heart disease</td>
<td>5.7%</td>
<td>6.3%</td>
<td>4.6%</td>
<td></td>
</tr>
<tr>
<td>For end-stage renal failure</td>
<td>0.8%</td>
<td>8.9%</td>
<td>2.9%</td>
<td></td>
</tr>
</tbody>
</table>

Renal complications

| Proteinuria in 1990 ‡† | 5.4% | 30.2% | 13.0% |
| Microalbuminuria in 1990 ‡‡ | 22.1% | 26.7% | 33.3% |
| End-stage renal failure in 1990 ‡‡ | 0.3% | 4.7% | 3.3% |

Crude incidence (per 100,000) of diabetes-related renal disease in New Zealand in 2001* | 1.5 | 18.2 | 19.8 | 3.8 |

Cardiovascular complications

| Self-reported known ‘heart attack’ in 1992–93 ‡‡ | 11% | 11% | 11% |

Eye complications

| Blindness in 1992-93 ‡ | 2.0% | 6.6% | 7.7% |
| Laser treatment in 1992-93 ‡ | 7.2% | 19.2% | 12.3% |
| Cataract in 1992-93 ‡ | 6.2% | 14.4% | 16.0% |

Vision-threatening retinopathy in 2002 ‡‡ | 2.5% | 4.3% | 4.9% | 4.6% |

Foot complications

| Self-reported leg/foot symptoms in 1992–93 ‡ | 37% | 42% | 29% |
| Amputation in 1990 ‡ | 2.2% | 2.8% | 1.0% |
| Foot ulcer in 1990 ‡ | 1.7% | 2.7% | 8.4% |

Prevalence of diabetes among cardiovascular and renal disease patients

| Among MI patients aged 40+ in 1992–93 ‡ | 14.7% | 36% | 37.9% |
| Among patients with congestive cardiac failure ‡ | 17% | 34% | 36% |
| Among new renal disease patients in 2003 ‡ | 23% | 65% | 67% | 50% |

*Estimated from ANZDATA Registry 2001 and Census 2001; † Age and sex standardised; ‡ Age adjusted to total diabetes population; MI=Myocardial infarction.

A familial predisposition to renal disease was suggested from one study showing that the predisposition to diabetic nephropathy in Polynesians was associated with a family history of renal disease (rather than a family history of diabetes), yet associated with diabetes through relative hypoinsulinaemia and hyperglycaemia. Diabetic nephropathy among children and young adults with Type-I diabetes was reportedly 19% in the Waikato area.
Figure 3. Numbers of dialysis patients with diabetic primary renal disease and new renal disease patients with diabetes (by ethnicity)

![Graphs showing numbers of dialysis patients and new renal disease patients with diabetes](image)

Source: ANZDATA Reports 1998–2004

**Other diabetes-related complications**

Few studies of diabetic eye and foot disease have been undertaken. A summary is shown in Table 4. The SADP study in 1992–93 found significant ethnic differences in the rates of blindness, laser treatment, and cataract among people with diabetes: Maori and Pacific people having double the proportions as those of European descent.\(^{46}\)

Retinopathy was present in 41% of a Type 2 diabetes cohort in Canterbury at baseline.\(^{59}\) A decline in the rates of vision-threatening diabetic retinopathy from 11.5% in 1993 to 1.5% in 2002 has been reported in diabetes patients in the Waikato area, but Maori had a high failure-to-attend-screening rate (32.3%) compared with the overall rate of 18.7%.\(^{50}\) *Get Checked* results for 2004 indicated low eye-screening rates of less than 70% overall, with less than 60% for Maori and Pacific groups. The rate in those aged under 26 was 13%.\(^{64}\)

The prevalence of hospital discharges for diabetic foot disease in New Zealand increased from 13.56 in 1980 to 25.79 in 1993.\(^{65}\) The total inpatient cost for the management of diabetic foot disease in New Zealand (population 3.3 million) for 1993 was estimated to be in the range of NZ$10–11 million.

The SADP study found significantly higher numbers of Pacific peoples with major lesions (amputation or ulcer/blister) compared with European or Maori diabetes patients (Table 4).\(^{51}\) The Ministry of Health estimated that Pacific people have more than double rate of lower limb amputation (43.6 per 100,000) in adults aged 25+ compared with the total New Zealand average (17.4)\(^{66}\) in 2004.

The Auckland Leg Ulcer Study in subjects aged 40+ years showed that 18% of cases had diabetes as a comorbidity whereas only 5.5% of controls had diabetes.\(^{67}\)
Conclusions

While the diabetes epidemic continues to impact increasingly on New Zealanders and its health services during the past 5 years, a growing number of Government and DHB-funded initiatives are in place to prevent diabetes and its complications (e.g. Lets Beat Diabetes and Diabetes Projects Trust in Counties-Manukau, Ngati Porou Hauora Ngatai and Healthy Programme in Taiwawhiti, Te Whai Matauranga o te Ahua Noho lifestyle program in Otago, and Te Wai o Rona: Diabetes Prevention Strategy in Waikato/Lakes).

Moreover, several district diabetes registers are in place or are under development (e.g. in Otago, Canterbury, Waikato and South/West Auckland), and these are complemented by the Get Checked data. ANZDATA renal and the emerging Australasian Diabetes in Pregnancy Society diabetes in pregnancy registers, along with a several eye screening registers also contribute to our understanding of diabetes in New Zealand.

The Get Checked dataset is apparently due to be extended, and this may help provide a more detailed and comprehensive view of diabetes and its care. Work is now needed on how best to monitor the incidence and prevalence of diabetes as well as the proportion of people with undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose. How else will we know that the growing resources directed towards lifestyle change are having an effect?

To date, the data gathered relating to metabolic control and complications are patchy, however they suggest that New Zealand needs to do more to reduce the impact of diabetes on cardiovascular, renal, eye, foot, and pregnancy related complications. This is particularly the case for Maori and Pacific peoples, whose metabolic control remains poorer than that for European New Zealanders.

More aggressive blood pressure, glycaemic, and lipid control would appear to be needed, and the development of ways to deliver this within the context of New Zealand (i.e. to its people and its health service) are urgently required. Such increases in medication use and services (in both primary and secondary care) are likely to cost more initially and yet little data exists to guide such development.

PricewaterhouseCoopers Ltd estimated that the Type 2 diabetes cost in 2001 approached NZ$400 million and was predicted a rise to more than NZ$1000 million by 2021. They also estimated that the total cost of diabetes could be reduced over 20 years if existing services are increased as soon as possible (by $10 million each year in their enhanced services model). The models used are not perfect, yet more complete than the earlier Health Funding Agency report. It is surprising that more detailed economic data is not available.

While there have been a relatively large number of publications relating to diabetes in New Zealand over the last 5 years, a significant proportion were from South Auckland in the 1990s and these data are now ageing. More importantly, while services are developing in primary and secondary care, evaluation has rarely been sufficiently robust to lead to publication in peer-reviewed journals. Indeed, funding for such “diabetes translational research” has been uncommon and fits poorly into the existing research funding paradigm.
If we are to develop more complex models of care, and increase access to modern pharmaceuticals and devices, then it is also clear that we need more research into the impact of such service developments on the incidence, prevalence, and costs of diabetes and its complications. While this will not come cheaply, it will be cheaper than the alternative.

A nationally agreed strategic plan is now urgently needed on how best to monitor and control the increasing incidence and prevalence of diabetes. In addition, major national surveys are required now to ascertain the proportion of those people living in New Zealand with impaired fasting glucose or impaired glucose tolerance, as well as those with undiagnosed diabetes.

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**References:**


Extramedullary plasmacytoma of the thyroid
Sheng-Fong Kuo, Hung-Yu Chang, Chuen Hsueh, Jen-Der Lin

Abstract
Plasmacytomas of the thyroid are uncommon. A 19-year-old female presented with a palpable, 3.5-cm-diameter mass in the right thyroid lobe. Fine-needle aspiration cytology (FNAC) showed that lymphoma cells were likely. The patient underwent a right lobectomy. Final haematoxylin and eosin (HE) staining of the tumour confirmed a diagnosis of thyroid plasmacytoma. Immunohistochemical staining showed that plasma cells were stained strongly with IgA antibody. To date, this is the youngest patient with thyroid plasmacytoma in the literature. Diagnosis of thyroid plasmacytoma by fine-needle aspiration cytology is typically difficult, as it was for this patient. Currently, no treatment standard exists for thyroid plasmacytoma.

Case report
A 19-year-old female visited the Metabolic Clinic at Chang Gung Memorial Hospital (CGMH) due to a right neck mass persisting for about 6 months. Physical examination revealed a 3.5-cm nodule in the thyroid gland. The mass was non-tender, elastic in consistency, and movable after swallowing.

All other physical examination findings were normal. Thyroid function; haemoglobin, serum calcium, phosphate, total protein (7.6 g/dl) and albumin (4.9 g/dl) levels; immunoelectrophoresis; and chest X-ray results were normal. Thyroid ultrasonography with fine-needle aspiration cytology (FNAC) was performed.

Figure 1 shows the results of real-time thyroid ultrasonography with the 10 MHz transducer probe. There was a 3.5×2.8×1.6 cm hypo-echo mass in the right thyroid.

Figure 2 presents the patient’s cytological data. Tentative diagnosis by FNAC was thyroid lymphoma. The patient underwent surgery and lobectomy for the right thyroid tumour to obtain a histological diagnosis.

Figure 3 shows the histology of the tumour, composed of diffuse infiltration of sheets of plasmacytic cells in mature and immature forms.
Figure 1. Thyroid echo showed a 3.5×2.8×1.6-cm hypoechoic mass in the right lobe of the thyroid gland.

Figure 2. Fine-needle aspiration cytology results indicated thyroid lymphoma. (Liu’s stain, original magnification ×400.)
Figure 3. Final histological examination showed diffuse infiltration of mature and immature plasma cells in thyroid (haematoxylin and eosin stain, original magnification ×200).

The final diagnosis of thyroid plasmacytoma was made and further confirmed by immunohistochemical stains for IgA, IgG, IgM, light chains-kappa (κ), and lambda (λ).

Figure 4 shows the positive staining result with a striking intracytoplasmic staining for the IgA antibody. The light chains were negative immunohistochemically. A diagnosis of extramedullary plasmacytoma of the thyroid gland was therefore made.

Figure 4. Immunohistochemical staining demonstrated strong positivity for IGA (avidin-biotin complex, original magnification ×100)
The patient was well and had no bone pain. She did not undergo bone marrow examination or further treatment, and has been well for 3 years without tumour recurrence.

**Discussion**

Plasma cell neoplasm is a malignancy typically afflicting elderly populations. In contrast to multiple myeloma, plasmacytoma is a localised proliferation of plasma cells in the bone marrow and less frequently in extraosseous organs.\(^1\,^2\) Primary plasmacytomas and plasma cell granulomas are unusual thyroid disorders.\(^3\,^4\)

A review of primary plasmacytoma cases identified similar gender distribution in Western nations and Japan. In an average 5-year follow-up period, over 70% of the patients were alive with no evidence of disease.\(^6\) Long-term follow-up is recommended due to possible progression to multiple myeloma.\(^6,\,^7\)

Although FNAC has been widely used in diagnosing nodular thyroid disorders,\(^8,\,^9\) limited experience exists for preoperative diagnosis of thyroid plasmacytomas.\(^5,\,^6,\,^10\) A thyroid plasmacytoma can be mistaken as thyroid lymphoma and even medullary carcinoma by FNAC;\(^5,\,^6,\,^10\) in this case, the thyroid plasmacytoma was mistaken for thyroid lymphoma.

In contrast to extramedullary thyroid plasmacytoma, multiple myelomas with thyroid involvement are rarer. Multiple myelomas have reportedly involved the thyroid in its advanced stage.\(^11\) Fewer than 10 reported cases have the thyroid as the first presenting site of multiple myelomas.\(^11\)

In our case, the possibility of multiple myeloma with thyroid involvement could not be completely excluded, however, a literature review revealed that all patients diagnosed with primary thyroid plasmacytoma were over 35 years old, as was the patient with multiple myelomas with thyroid involvement. Our case is the youngest patient reported with thyroid plasmacytoma.

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**References:**


Cutaneous paraneoplastic syndrome (acrokeratosis paraneoplastica) preceding squamous cell carcinoma of the glottic larynx

Gorkem Aksu, Ahmet Karadeniz

Abstract

Paraneoplastic syndromes that occur in the minority of cancer patients are the produced signs and symptoms at distant sites from the tumour or its metastases. These syndromes may occur due to the production of substances by tumoural lesions that directly or indirectly cause distant symptoms or depletion of normal substances or host response to the tumours. A paraneoplastic syndrome may be the first sign of a malignancy so its recognition may be critical for early cancer detection. Most of the paraneoplastic syndromes associated with head and neck tumours are endocrinologic or neurologic; dermatologic syndromes are less common. Head and neck cancers also have occasionally been reported in association with paraneoplastic syndromes and to date there are only a few cases in the literature about the presence of a cutaneous paraneoplastic syndrome as the first manifestation of a laryngeal cancer, especially glottic larynx cancer. A wide variety of cutaneous syndromes are associated with malignancies and these syndromes may precede, follow, or be concurrent with the underlying malignancy.

In this report we present a case with cutaneous syndrome of acrokeratosis paraneoplastica preceding squamous cell carcinoma of glottic larynx, and review the other cutaneous paraneoplastic syndromes reported in the literature.

Paraneoplastic syndromes that occur in the minority of cancer patients are the produced signs and symptoms at distant sites from the tumour or its metastases. These syndromes may occur due to the production of substances by tumoural lesions that directly or indirectly cause distant symptoms or depletion of normal substances or host response to the tumours.

The best characterised paraneoplastic syndromes are those producing ectopic hormones such as parathyroid hormone (PTH) or adrenocorticotropin (ACTH)—in such cases the treatment of the underlying malignancy leads to the disappearance of the hormone and the syndrome.1–3

A paraneoplastic syndrome may be the first sign of a malignancy, so its recognition may be critical for early cancer detection. Also, the secreted proteins that cause paraneoplastic syndromes can be used as tumour markers during the therapy and follow-up for the evaluation of treatment response or recurrence.2,3

Head and neck cancers have occasionally been reported in association with paraneoplastic syndromes and to date there are only a few cases in the literature about the presence of a paraneoplastic syndrome as the first manifestation of a laryngeal cancer.1,4–6
In this report we present a case in which cutaneous paraneoplastic syndromes preceded the histopathological diagnosis of glottic laryngeal carcinoma, and we review the literature.

**Case report**

A 62-year-old man with a 35-pack per year smoking history referred to dermatology department of our hospital for skin lesions developing 3 months previously. On physical examination, hyperkeratotic psoriasiform plaques were present in both of his hands and soles. Both of the dorsal and palmar aspects of the fingers and nail folds were involved and his toenails were also thickened and dystrophic (Figure 1).

**Figure 1. Hyperkeratotic psoriasiform plaques on the left hand of our case**

Laboratory findings were within normal limits, and the only abnormal finding in physical examination (except skin lesions) was significant voice hoarseness. The patient claimed that skin lesions had preceded voice hoarseness. With these findings, an endoscopic examination of larynx was performed that showed a tumoural lesion in the left posterior ventricle extending up to the vocal cord.

A biopsy was taken from the lesion and the histopathological diagnosis was squamous cell carcinoma of the larynx. Computed tomography (CT) showed that the lesion was limited to the glottic region and there was no lymphadenopaties in the neck. CT of chest also demonstrated no pathology.

The patient was diagnosed as having glottic larynx cancer and acrokeratosis paraneoplastica, and he was treated with 66 Gy external radiotherapy with 2 Gy daily fractions. The skin lesions (except the nail dystrophy) significantly resolved after the completion of radiotherapy, and the patient is still alive 3 years later with no evidence of disease.

**Discussion**

A wide variety of cutaneous syndromes are associated with malignancies. These syndromes may precede, follow, or be concurrent with the underlying malignancy. The most critical point is that once a potential cutaneous paraneoplastic syndrome has been diagnosed, an extensive systemic evaluation emphasising the malignancies most strongly associated with that type skin lesion shall be undertaken.
The physicians especially should be aware of some cutaneous lesions that are uncommon and usually associated with cancer. Acrokeratosis paraneoplastica is a typical example of such lesions since it is one of the most rare cutaneous paraneoplastic syndromes and is typically present in patients with squamous cell carcinoma of the oesophagus, head, and neck or lungs. The eruptive lesions are characteristically hyperkeratotic, resembling psoriasis and favouring acral sites and nails.

In most of cases, including ours, acrokeratosis paraneoplastica is the first sign of the underlying occult malignancy. Antigenic cross-reaction of basement membrane and tumour antigens, and the secretion of some growth factors such as insulin-like growth factor-1 (IGF-1) or transforming growth factor-alpha (TGF-alpha), are thought to be possible mechanisms causing this syndrome—as squamous cell carcinomas have been shown to synthesise and secrete these autoimmune growth factors.

Head and neck squamous cell carcinomas are the most frequently associated malignancy with this syndrome, and oropharynx or larynx cancers make up more than 60% of the cases reported in the literature. Bazex described three stages for cutaneous lesions of acrokeratosis paraneoplastica; he reported that fingers, toes, soles, and (in some cases) the ear helices and nose are affected usually in a symmetrical fashion.

Bazex also mentioned that the average time between the appearance of the cutaneous lesions and the detection of the underlying tumour is about 11 months, but this interval is shorter in some of the reported cases in the literature, including our case. The skin eruptions may resolve with the treatment of underlying malignancy but in some cases the lesions persist or reappear with the recurrence of the tumour. UV-A phototherapy or retinoids and oral psoralen have been used in some cases with the reportage of limited benefits.

Seborrhoeic keratosis which is generally seen in elderly patients can also appear as a paraneoplastic cutaneous syndrome. The Leser-Trelat sign which is characterised by the sudden appearance and rapid increase in the number and size of seborrhoeic keratoses is important since these lesions can be associated with internal malignancies. The most common malignancy is adenocarcinoma of the stomach but the syndrome can also be present in patients with breast cancer as well as squamous cell carcinomas of head, neck, and lung.

As described above, early diagnoses of these lesions is critical because, with our case, these cutaneous lesions may precede or be concurrent with an underlying malignancy. Therefore paying careful attention to such lesions may lead earlier detection of at least some malignancies.

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References:


PHARMAC and Herceptin for early-stage breast cancer in New Zealand: Herceptin or deception?

Martin Rosevear

Abstract
Pressure to fund Herceptin (trastuzumab) for use in early-stage breast cancer is a welcome development for those patients who may benefit. However such a decision would have major implications since the health gains made by trastuzumab come at a very high cost (when compared to health gains achieved by other drugs currently funded on PHARMAC’s schedule). The budget for trastuzumab (estimated to be NZ$30m/annum but this is currently being negotiated) will be funded from district health board (DHB) budgets, which will impact other patients unless DHB budgets are appropriately increased. In comparative terms, this proposed expenditure is almost the same as what is currently being spent on all other oncology agents together, and is similar to the total cost of hospital services in New Zealand regions such as Wairarapa and Marlborough.

Drug
Herceptin® (trastuzumab) (Roche)

Indication
Reduces disease progression in breast cancer with an over-expressed HER2 gene, representing approximately 15–20% of women with breast cancer.\(^1\)

Patients with HER2-positive breast cancer are a relatively vulnerable group. Patients with metastatic cancer and expressing HER2 have a 50% shorter survival time relative to other patients with breast cancer, and approximately 70% of breast cancers will eventually develop into the metastatic form.\(^3\)

Recommended dose & duration
Trastuzumab for the indication of early HER2-positive breast cancer is provisionally licensed for use subsequent to surgery and adjuvant chemotherapy. Following adjuvant chemotherapy (or in combination with chemotherapy with adjusted dose), an initial dose of 8mg/kg followed by 6mg/kg body weight every 3 weeks for 1 year.\(^1\)

Clinical efficacy
Trastuzumab as an adjuvant treatment in early breast cancer when used sequentially to standard chemotherapy has been shown in the interim results of one published open label clinical trial to improve the disease free survival after 2 years from 77.4% to 85.8%.\(^1\)
Background

Trastuzumab is a recombinant monoclonal antibody against HER2 and has been available for advanced (metastatic) stage cancer since 2001. In March 2006, New Zealand became the first country to give trastuzumab provisional approval for the aggressive HER2 form of early breast cancer.

Media and public interest in the trastuzumab debate has been strong, both in New Zealand and internationally. A recent high court decision in the UK found the local NHS trust acted ‘irrationally and unlawfully’ when it refused to pay for Ann Marie Rogers’ treatment. The three Court of Appeal judges said there was no “rational basis” for “preferring one patient to another.” They ruled that the focus should be on what a doctor felt was right for their patient. The ruling does not mean primary care trusts will be forced to provide the drug but it does set the precedent that giving the drug to some women but not others is unlawful.

New Zealand Government policy

Government policy seeks to make cost-effective drugs and other treatments available to patients on a fully subsidised basis where health benefits have been proven in high quality evidence based trials. We understand that the district health boards (DHBs) will fund Herceptin. Therefore the DHBs must balance their existing budgets to fund this service (presumably by achieving additional efficiencies or reducing services), or DHB budgets must be increased.

Current situation

On 23 March 2006, Medsafe announced provisional approval for trastuzumab for the treatment of women with early breast cancer who test positive for the HER2 gene once they have had surgery and completed adjuvant chemotherapy. The provisional approval limited treatment to those women who have a normal heart function before treatment starts and requires women using trastuzumab to have their heart function checked by echocardiogram every three months during treatment. PHARMAC is currently developing its policy on trastuzumab and negotiating the terms of support. Based on costs of NZ$70,000/patient, this could see a NZ$30m/annum bill covering 430 patients. This should be compared to PHARMAC’s total budget in 2003 for community pharmaceuticals of $568m, and $47m spending by PHARMAC or DHB hospitals on all cancer agents under the ‘cancer basket’. In addition, it is noted that it costs approximately $30m/annum to provide hospital services to communities of 30,000+ people such as Wairarapa and Marlborough.

The decision to enter into negotiation over the subsidy of trastuzumab appears to be driven by public pressure rather than strong scientific evidence. The decision may also have implications for other users of PHARMAC’s drug schedule, who may not be as well-resourced to wage a media campaign to support their needs, as the supporters of trastuzumab are.
Economic analysis

There is one published economic analysis of trastuzumab in early breast cancer,¹² but its results are difficult to interpret in the New Zealand setting; and other analyses relate to advanced disease. In 2001, the UK’s National Institute for Clinical Excellence (NICE) provided estimates of the cost of trastuzumab for advanced breast cancer ranging from £38k/QALY to £19k/QALY depending on treatment.⁵ A US investigation into metastatic cancer estimated US$125k/QALY.⁶ Currently PHARMAC’s budget enables it to fund drugs nominally up to NZ$20k/QALY, although some such as antipsychotic drugs are purchased in quantity at NZ$43k/QALY² and small quantities are purchased at higher rates. However we understand that while PHARMAC uses cost/QALY results as part of its funding consideration, it has no cost/QALY threshold which automatically ensures funding.

The recent results reported from trials (Piccart-Gebhart et al (HERA trial)¹ and Romond et al)⁷ involving early breast cancer indicate possibly stronger health gains. However PHARMAC’s PTAC Advisory Committee has indicated the following concerns with these results:⁸

- The HERA results were interim, and both the benefit and safety data for early breast cancer are premature;
- Both papers have omitted to publish all results;
- The Romond paper omitted to publish the treatment arm that was directly relevant to this indication. Therefore they considered efficacy results of the paper were found to be of limited value; and
- The risk of cardiotoxicity had to be especially managed and in the case of early disease, the addition of trastuzumab could put at risk patients who would otherwise have survived.

However in an effort to provide some estimate of trastuzumab’s impact on early-stage breast cancer, we have used data from the Romond paper to provide a hypothetical estimate of cost/QALY results. Survival results at four years have been combined with the effects of aging as observed in the life expectancy for women in the general population¹⁰ to extrapolate a very simplistic picture by the authors of this paper:
Indications are:

- 50 years is the average age at diagnosis for HER2-positive breast cancer patients who have an average life expectancy of perhaps 8 years, versus 35 years calculated life expectancy for women of the same age in the general population.

- The trastuzumab arm shows a hypothetical benefit of approximately 1.6 years, versus patients receiving standard treatment only. This benefit is the area between the two treatment curves in the graph above.

But this simplistic analysis raises a number of questions:

- How long does the therapeutic value of trastuzumab last? Does the differential reported in the trials persist, or are their latent toxic effects yet to be observed which could reduce or totally remove any persistent benefits for trastuzumab? For instance it is known that trastuzumab is cardiotoxic, with approximately 20% discontinuing trastuzumab treatment due to cardiac problems in the Romond trials, and a further 14% discontinuing for other reasons (noting that Romond results were for a different regimen). Will the death rate from heart failure or other toxic side-effects remove short-term benefits in the longer term?

- Will additional courses of trastuzumab be required to increase persistence?
Therefore when estimating a $/QALY for early breast cancer, uncertainty exists in the following issues:

- Increase in life expectancy due to trastuzumab in the long-term, and associated savings due to delays in recurrence or terminal cares;
- The quality of life (relative to a normal population) that is provided during those extended years following treatment. Adjustments between 30% to 50% to convert extended life in metastatic cancer to the normal population have been used;\(^5\)
- The duration of treatment required to obtain long-term benefits; and
- NZ$/patient cost of a course of trastuzumab.

Given the uncertainties above, it can be estimated that the cost per QALY could lie somewhere between:

NZ$88k and NZ$100k+

…where the lower bound has been estimated using the analysis above (without adjusting for other costs/savings nor quality of life) and the upper bound has been estimated using the published results for metastatic cancer. Note that this estimate has not allowed discounting of future costs and benefits (standard for such analyses), which would likely mean an increase in the cost/QALY.

**Comment**

Evidence suggests trastuzumab can bring benefit to early-stage breast cancer in the short-term. However, these benefits come at a high cost, which appear to be above the margin at which PHARMAC has historically subsidised drugs. As such, funding of trastuzumab could come at a high cost to other users of publicly funded pharmaceuticals and other health services.

**Disclosures:** None.

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**References:**


9. Survival results of Romond paper can be found at: http://content.nejm.org/cgi/content/full/353/16/1673


Odds and ends of a year’s surgery. A case of appendicitis with general suppurative peritonitis. (Sudden death on eleventh day.)

This case report was written by Philip James, F.R.C.S., Wellington and published in the New Zealand Medical Journal 1906, Volume 5 (19), p39–47

A young healthy man, aged 26, was admitted into Hospital on the evening of the 7th November. I saw him early on the morning of the 8th, and decided to operate at once, as the diagnosis was obvious, and the man was rapidly becoming moribund. On opening the peritoneal cavity a large quantity of fluid containing pus and several faecal concretions escaped. The appendix was gangrenous and had perforated. The abdomen was thoroughly washed out with hot saline and a rubber drain inserted. For many days he was desperately ill, but he gradually improved, pain became less, tongue, which had been like a burnt chip, became moist, and he felt fit to get up when I last saw him on the eighth day after the operation.

I was then absent from the Hospital for three days, owing to illness, and was thunderstruck when I saw in the morning paper on the 12th day that he was dead. He had been seen by the Medical Superintendent the previous evening, and was feeling and looking so well that Dr. Ewart put him down for a liberal diet for the following day.

The nurse in charge reports that he slept until 3.25 a.m., when his breathing suddenly became embarrassed, he lost consciousness, and perspired profusely. He asked if he had had a faint, and said that he felt very weak. After an interval of a few minutes his breathing again became much louder, pupils dilated. Temp., 97° Fahr.; pulse, uncountable. These attacks occurred at intervals of a few minutes, and he died shortly before 4 a.m. For the last ten minutes he had been getting more cyanosed, and was unconscious.

I regret that I did not see the partial post-mortem that was made, but the Assistant House Surgeon reported about 3 oz. or 4 oz. of a brownish fluid in the peritoneal cavity, so that it would seem that the peritoneum had come off the victor in its phagocytic contest. There was a patent foramen ovale, and all the cavities were filled with blood. By some curious oversight the lungs were not examined. This malformation had never been suspected in him, as he was always so strong and healthy, although a brother of his suffered in the same way. I find it impossible to believe that this had anything whatever to do with his death.

To my mind, a much more likely explanation is that he had thrombosis of a large vein in the abdomen, and died of pulmonary embolism. The patent foramen ovale would account for the cavities on both aides of the heart being full of blood.
Stalk failure

Sarah Mathai, Krishna Sudeep, Mathew John

A 3-year-old boy was referred to Endocrine Services for evaluation of recurrent episodes of hypoglycaemia. On examination he was noticed to have micropenis, a prominent forehead, and midfacial hypoplasia. The fontanelles were closed. He was 84 cm in height (less than the 5th centile).

A magnetic resonance imaging (MRI) scan of the brain was performed and the T1-weighted non-contrast coronal and sagittal images are presented in Figure 1.

Figure 1

Questions—What is the abnormality on the MRI? (arrowed). What is the name of the syndrome and its implications?
Answers

The anterior pituitary (lower arrow) is small and the infundibulum is not seen. The hyperintensity of the posterior pituitary is located ectopically in the region of the hypothalamus (upper arrow).

The patient has stalk interruption syndrome associated with posterior pituitary ectopia.

The patient has combined pituitary hormone deficiency.

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Advanced-care practitioners

Apparently there is a workload crisis in the UK National Health Service (NHS)—too many patients and not enough nurses and doctors. A proposed answer to this problem—the creation of advanced-care practitioners, new professionals in the UK health-care system. The duties of these practitioners include some aspects of direct clinical care that have previously only been done by doctors in the UK—eg, the administration of an anaesthetic.

And their training? It will be “broadly based and will take at least 90 weeks, equivalent to a 3-year degree course, with a minimum of 1600 h of clinical learning.” Already there are accusations of dumbing down of medicine and risking standards of patients’ care.

And what about the views of the other recent innovation—the specialist nurse? One would expect them to be unenthusiastic. Maybe it would be better just to train more real doctors.

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Antiplatelet therapy—a place for dual treatment with clopidogrel plus low-dose aspirin?

Antiplatelet therapy with low-dose aspirin, an irreversible inhibitor of platelet cyclooxygenase, has earned its rightful place as a cornerstone of treatment for reducing cardiovascular and cerebrovascular events in patients with established vascular disease.

Clopidogrel, by inhibiting the adenosine disphosphate P2Y_{12} receptor, offers a distinctly different mechanism to reduce platelet activation and aggregation. It has an established role when used with aspirin in reducing ischemic events in patients with unstable angina, myocardial infarction with or without ST-segment elevation as well as those undergoing angioplasty and stenting.

A recently reported trial (involving 15,603 subjects) randomised patients to low-dose aspirin or aspirin plus clopidogrel 75 mg daily. The results—“clopidogrel plus aspirin was not significantly more effective than aspirin alone in reducing the rate of myocardial infarction, stroke, or death from cardiovascular causes.”

It is speculated that clopidogrel is only effective when the patient has acute vascular injury.

Hip protectors versus femoral fracture

Hip fracture is the commonest reason for admission of elderly people to an acute orthopaedic ward and is generally the result of a fall. As most hip fractures affect the greater trochanter of the femur, it would seem to be good common sense to protect that site from trauma. Enter the hip protector—a padded device first used about 50 years ago. So, it was pleasing when Cochrane Database reviewers reported in 1999 the conclusion that hip protectors seemed to reduce the risk of hip fractures among institutionalised elderly people. But wait! The same reviewers have had a second look and their current conclusion is that “accumulating evidence indicates that hip protectors are an ineffective intervention for those living at home and that their effectiveness is an institutional setting is uncertain.” Non-compliance may be a relevant issue?

Osteonecrosis of the jaw (ONJ) associated with the use of new generation bisphosphonates—a new disease

The use of bisphosphonates is well established for the use of treatment of patients with metastatic bone disease, osteoporosis, Paget’s disease and hypercalcaemia. This class of drug inhibits specific enzymes targeting cellular pathways within the osteoclast, thus leading to osteoclast inactivation. Etidronate, the earliest of them, is relatively weak in action. The newer generations of bisphosphonates contain the nitrogen moiety, creating drugs with much higher potencies—pamidronate, alendronate, and zolendronate—the latter being $\times 1000$ more powerful than etidronate. This paper reports on 23 such cases associated with long-term bisphosphonate usage. Your scribe is aware of 7 cases in his parish—a probable incidence rate between 1 and 2% of those who have been treated for not less than 4 years. Great class of drug, but no free lunch.

Croup and humidity treatment

Viral croup (acute laryngotracheobronchitis) is a common childhood complaint. In this paper from Toronto it is claimed that it is the commonest cause of acute upper airway obstruction in children, and is diagnosed in up to 5% of children younger than 6 years, of whom approximately 1% are hospitalized.

Children with croup are often treated with humidity—yes I can remember that—an unpleasant illness with an unpleasant treatment. Hence a 3-arm randomised trial. And the result—100% humidity with particles specifically sized to deposit in the larynx failed to result in greater improvement than 40% humidity or humidity by blow-by technique. The blow-by technique is the standard of care in Toronto and in their opinion confers no benefit (ie, equal to placebo). So—this study does not support the use of humidity for moderate croup for patients treated in the emergency department.
New Zealand guidelines for early management of meningococcal disease—time for revision?

Invasive meningococcal disease is extremely challenging to diagnose and treat. General practitioners (GPs) have perhaps the most difficult task of all—identifying cases early in the disease course, at a time when the clinical picture is often indistinguishable from common self-limiting conditions such as colds and flu.

Current advice and practice in New Zealand

The Ministry of Health has produced guidelines for GPs to aid rapid diagnosis and treatment of suspected cases.

The Immunisation Handbook gives advice as follows:¹

Prior to transfer to hospital, practitioners should administer parenteral antibiotics to:

- All suspected cases of meningococcal disease in whom there is any haemorrhagic rash.
- All other suspected cases for whom the delay to assessment in hospital is likely to be greater than 30 minutes.

The Handbook also advises:

If the practitioner has considered the diagnosis and decided that the clinical features do not merit assessment in hospital, caregivers should be warned to seek urgent medical help, no matter what the time, if there is significant deterioration in the individual’s condition or if any petechial or purpuric lesions develop.

We would argue that this advice is not consistent with current evidence in the literature, and is likely to lead to under-treatment. In fact, figures released by the Ministry of Health show that in 2004, although 61.4% of meningococcal cases were seen by a GP prior to admission, only 34.5% of these received pre-hospital antibiotics.²

Pre-hospital antibiotics—the evidence

Interestingly, the medical literature provides at best conflicting evidence that pre-hospital antibiotics are effective in reducing case fatality rates. The most frequently cited study reporting improved outcome with administration of pre-hospital antibiotics did not achieve statistical significance using 95% confidence intervals, although a strong trend was noted.³ Other studies reporting improved outcomes have been based on small sample sizes.⁴⁻⁶

By contrast, a large study from Denmark,⁷ and a recent UK study published in the British Medical Journal,⁸ report that case fatality rates were actually worse amongst patients who had received pre-hospital antibiotics.

One explanation for this seemingly paradoxical result is that studies addressing this question are observational and hence subject to confounding. For example, patients who are very ill on presentation to their GP are more likely to be diagnosed and treated—but they are also more likely to have an adverse outcome.
Treatment criteria—early vs late signs

Another explanation is that “classical” signs such as haemorrhagic rash indicate an advanced stage in the disease course. The severe morbidity and mortality of meningococcal disease derive from the profound inflammatory reaction to meningococcal endotoxin; thus, antibiotics administered at this point may be too late to be of benefit.

In a recent study published in the Lancet, the authors have identified three early symptoms of meningococcal disease in children which were consistently noticed by parents and were present at the first GP consultation: leg pains, cold hands and feet, and abnormal skin colour.

They also note that symptoms evolved very rapidly, particularly in younger children, and as a result they strongly recommend early (within 4–6 hours) review of any cases where the diagnosis cannot be excluded at first examination.

Distance to hospital

If it is hard to demonstrate benefit from the use of pre-hospital antibiotics, it is even more difficult to comment on the relevance of travel time to hospital in the decision to give antibiotics. There is certainly no evidence from the literature regarding 30 minutes’ travel time as an appropriate treatment criterion.

Given the potential of meningococcal disease to progress rapidly over a timeframe of minutes to hours, travel time may well be an important consideration in determining management and outcome, particularly in New Zealand.

A study based on 2001 census data estimated that 167,295 residents of New Zealand had a travel time of more than 1 hour to reach their nearest hospital. This figure increased to over a million when only tertiary hospitals were included in the analysis; an important consideration in meningococcal disease, as many patients require a high level of care.

However, there is currently no research to indicate precisely how travel time should influence management decisions in meningococcal disease.

Conclusion

Despite the lack of objective evidence, it is still overwhelmingly likely that early antibiotic treatment is beneficial to outcome, and that pre-hospital antibiotics should be given wherever feasible.

On the assumption that this is a reasonable treatment goal, and given recently available evidence in the literature, we would suggest the following changes to the guidelines:

- That the treatment criteria should be widened to include early signs such as leg pains, cold hands and feet, and abnormal skin colour in children.
- That whenever there is concern about possible meningococcal disease at first presentation, the patient should be brought back for clinical review within 4–6 hours.
• That 30 minutes’ travel time to hospital is not an appropriate treatment
criterion, as there is no evidence to support this. Until further evidence is
available to clarify this question, suspected cases should be treated regardless
of travel time.

In addition, there is a clear and urgent need for further New Zealand-based research to
provide the best possible information for GPs managing this devastating disease.
Priorities for research could include special features of the New Zealand environment
such as the impact of travel time.

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References:


2. Martin D, McDowell R. The Epidemiology of Meningococcal Disease in New Zealand in
2004. Report prepared for the Ministry of Health by the Institute of Environmental Science

3. Cartwright K, Reilly S, White D, Stuart J. Early treatment with parenteral penicillin in

4. Strang JR, Pugh EJ. Meningococcal infections: reducing the case fatality rate by giving
penicillin before admission to hospital. BMJ. 1992;305:141–3.


meningococcal disease and case fatality: a Danish population-based cohort study. J Infect.
2002;45:144–51.

meningococcal disease before hospital admission: case-control study. BMJ. 2006 Mar 24;
[Epub ahead of print]


Geogr. 2002;1:3.
Response to McNaughton and colleagues regarding their article—Interferon beta, PHARMAC, and political directives: in the best interests of people with multiple sclerosis?


Figures provided by PHARMAC show that funding Betaferon and Avonex cost NZ$5,309,012 in 2005, for the benefit of 364 patients with multiple sclerosis (MS) at an average cost of $14,585 each.

Given their belief in insufficient evidence of effectiveness and value for money, McNaughton et al suggest the key motivation behind PHARMAC’s funding of interferons is political, rather than scientific, and that PHARMAC is ignoring the greater cost-effectiveness that may be delivered by channelling the $5 million spent on interferons into alternate health and disability services that benefit a greater number of people with MS.

However, Multiple Sclerosis Society of New Zealand (MSSNZ) has found there is good data available on the effectiveness of interferons for the treatment of MS; our 1999 campaign to have interferon funded was based on this premise. A more recent study by the American Academy of Neurology found that interferons could be prescribed with confidence. As McNaughton et al acknowledge, New Zealand neurologists are comfortable prescribing these medications. Thus, one can assume the body of clinical evidence is robust enough to consider interferons an effective MS treatment.

Whether this effectiveness comes at a reasonable cost is clearly dependent on which figures are to be used. The NICE study contradicts other British studies, but neither is really relevant to the New Zealand health system. Currently, there are no figures available on the cost-effectiveness of MS treatment in New Zealand, and this is a key information gap.

However, MSSNZ knows from our members that those for whom interferons do not work, or those for whom the side effects prove too straining, they discontinue the treatment. Thus, we assume people with MS who are currently receiving interferons are satisfied that the medication works for them. In the absence of data on cost-effectiveness, the Society believes the evidence of effectiveness (as shown in clinical studies and illustrated by the ongoing involvement of 364 patients in the regime) is enough to justify the ongoing cost.

Furthermore, the effectiveness of interferons is likely to be increased if PHARMAC expands the eligibility criteria to reflect studies showing that immediate prescription of disease-modifying drugs (DMDs) upon diagnosis can delay disease progression. Emerging studies also show that prescription of interferons upon evidence of lesion damage can prevent the emergence of clinically definite MS altogether. If PHARMAC revise its policies to reflect these studies and make interferons available...
earlier in the MS experience, then hospitalisations and the use of other health services will be reduced.

Although the actual cost-benefit ratio will remain a mystery until dedicated research is implemented, logic leads us to believe that decreased use of tertiary health services saves money and increases the financial return on the expanded interferon investments.

Of course, medical treatment does not exist in a vacuum. As the authors note, there are many additional approaches to MS management that have proven moderately effective (e.g. physical rehabilitation, the use of adaptive equipment, and psychosocial support), but the current delivery of support for these approaches is inadequate and dysfunctional. MSSNZ agrees with the authors wholeheartedly on this criticism and points out that, until such time as services are improved, people with MS have little alternative besides adopting a mostly pharmacological approach to their condition.

The authors suggest that the inadequacy of disability services could be somewhat alleviated by applying the $5 million spent on interferons to better ensuring a multidisciplinary approach to MS management is available. They also make the point that this type of treatment benefits more patients and thus provides better value for money. However, MSSNZ knows from experience that current funding for health and disability services is woefully inadequate, and an additional $5 million will do nothing to address the underlying problems with the sector. Furthermore, patients currently receiving interferons do so because they find them effective and MSSNZ believes it is unethical to take funding from these patients to make very little difference to another aspect of the health service.

The problem is not that $5MILL is spent on a sole therapeutic approach to MS treatment used by a limited number of patients in the absence of proven value for money within the NZ health system. As we have shown, this is debatable. Instead, the greater issue is that total government funding for all avenues of MS treatment for all people affected by the condition fails to scratch the surface of what is required to achieve “long-term support centred on the individual”, as promised in their Disability Strategy. Thus, MSSNZ strongly argues that funding for MS medications should be maintained; treatment eligibility increased, and these policies accompanied by adequate funding for other necessary health and disability services to ensure a total MS management package is available to those in New Zealand affected by the condition.

In addition, MSSNZ recognise the difficulties faced in providing adequate evidence of the cost-effectiveness of interferons in the New Zealand health environment. Only rigorous research into the social and economic costs of MS in New Zealand will make this debate possible, and will benefit all those with a vested interest in MS, McNaughton et al included.

Nola Rawson
National Director
Multiple Sclerosis Society of New Zealand
References:


3. Ibid.
Metastatic thyroid carcinoma: a case from Australia

We read with interest the recent case presentation published in the *New Zealand Medical Journal* [Kaya H, Barbaros U, Erbil Y, et al. Metastatic thyroid carcinoma. 2005;118(1224). URL: http://www.nzma.org.nz/journal/118-1224/1705/]. This case is similar to one of our own described below.

An 84-year-old man presented with a 6-month history of a solitary lump in the right side of his neck. His major complaint was progressive dysphagia. On examination, there was a solitary nodule with a smooth non-tender surface that moved with swallowing. CT of the thyroid confirmed the there was a swelling of the right side of the thyroid gland that displaced the carotid artery and jugular vein laterally, some areas of reduced density was seen within the mass. He underwent a right hemithyroidectomy and made an uneventful recovery. Histology showed a dominant nodule that was thinly encapsulated.

Much of the nodule had solid sheets, anastomosing trabeculae, or clusters of cells with clear cytoplasm. There was scattered areas of necrosis. The tumour cells were immunoreactive for CD10 and there was no staining for thyroglobulin or TTF-1. The morphology and immunoprofile was consistent with metastatic renal cell carcinoma. CT chest, abdomen, and pelvis showed no other evidence metastatic spread. This man had undergone a left nephrectomy for renal cell carcinoma 10 years prior and had been well with no recurrence before this presentation.

Based on our own literature search, which agrees with the NZMJ published case report, this patient has a more favourable prognosis than that published by van der Poel et al. In that study they quote that the percentage of patients free of disease more than 36 months after initial diagnosis of a metastasis is 11%. This favourable prognosis is based on several factors as outlined by your case report including: a long interval between primary tumour resection and the development of a metastasis; evidence of a solitary site of spread; and demonstrated necrosis in the resected specimen.

We would like to point out the extensive data collected by Heffess et al not mentioned in your article. They presented 36 cases of metastatic RCC to the thyroid gland from the Endocrine Registry at the Armed Forces Institute of Pathology from 1959 to 1998. These 36 cases were identified in a review of 37,158 benign and malignant thyroid tumours seen in consultation between 1959 and 1998. From the total of 36 cases, 13 were the initial presentation of an underlying renal cell carcinoma. The 23 cases with patients that underwent nephrectomy presented with a thyroid mass at a mean time of 9.4 years (range 2 to 21.9 years). Of these 23 cases, half died with widely disseminated disease at a mean time of 5.4 years. The other half remained disease free or died of other causes.

We would like to highlight this pathologically interesting, although rare, presentation and recognise the contribution of the above article to this topic.
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References:


Neurotoxic reaction to citalopram

We present the case of an acute neurotoxic reaction with severe extrapyramidal features associated with commencement of citalopram in a patient without a prior history of Parkinson's disease or neuroleptic exposure.

A 77-year-old woman was admitted to a rural hospital with dehydration secondary to antibiotic-associated diarrhoea following treatment for a chest infection. Her past history included type two diabetes mellitus on insulin, a transient ischaemic attack, parotid carcinoma with left facial paralysis, ischaemic heart disease, atrial fibrillation, and mastectomy for breast carcinoma with resultant lymphoedema of her right upper limb.

There was no definite prior history of Parkinsonism but subtle extrapyramidal features were present on admission with reduced mobility, swallowing difficulty, psychomotor slowing, and increased tone with cogwheeling in the left upper limb. Modified Mini-Mental State examination revealed cognitive impairment with a score of 64/100. Medications on admission consisted of digoxin, enalapril, simvastatin, Penmix insulin, aspirin, and cefuroxime. She improved with rehydration with normal saline and cessation of her antibiotics.

Ten days after admission, she was commenced on citalopram10 mg daily for depressive symptoms. Over the next 4 days, she deteriorated significantly, with hypoactive delirium and markedly worsened extrapyramidal signs. She developed marked bradykinesia and rigidity of the limbs, associated with decreased coordination of swallowing, monosyllabic speech, and decreased self-cares. She was no longer able to follow requests.

She continued to deteriorate with decreased level of consciousness, increased rigidity with cogwheeling, and sustained clonus at the ankles with positive Babinski responses. There was no myoclonus, hyper-reflexia, fever, or autonomic dysfunction. Her citalopram was stopped 3 days after commencement. She was commenced on lorazepam for possible catatonia.

Investigations showed normal electrolytes and creatine kinase but mildly raised liver function tests. Full blood count and erythrocyte sedimentation rate were normal. C-reactive protein was 10 mg/L. She was transferred to a tertiary hospital for further investigation and treatment.

A magnetic resonance imaging (MRI) brain scan showed generalised cortical, posterior fossa, and brainstem atrophy with no sign of ischaemic or neoplastic processes or hydrocephalus. Lumbar puncture was attempted twice but was unsuccessful. Over the next 3 days she developed dystonic posturing of the upper limbs. She continued to have rigidity in both upper limbs. A psychiatry of the elderly specialist did not find any evidence to suggest major depression or catatonia, and the lorazepam was stopped 5 days after commencement. A normal electroencephalogram (EEG) excluded status epilepticus.
Over the following 7 days, the patient improved, becoming more communicative and alert. Tone remained increased in the upper limbs. She was commenced on levodopa, which had no noticeable effect. This was discontinued.

The patient was transferred back to the peripheral hospital 10 days later where she continued to recover over the next month becoming alert and orientated. She also communicated freely and was mobile with a low frame under supervision. She was noted to have persisting increased tone in the upper limbs, although this was improving. Unfortunately at this time (when she appeared to have returned almost to her pre-morbid level of functioning) she had an acute left middle cerebral artery infarct with a right hemiparesis and severe global aphasia. MRI scan confirmed a large left inter parietal/temporal lobe infarct.

Although extrapyramidal motor disorders are well reported with serotonin uptake inhibitors (SSRIs)\(^1\)\(^-\)\(^4\) there have been very few reports with citalopram\(^5\)\(^-\)\(^7\) being thought to have a low potential for extrapyramidal side effects.\(^8\) The extrapyramidal effects of SSRIs are thought secondary to an indirect modulatory effect of dopaminergic function through inhibitory serotonergic input.\(^3\)

This appears to be a case of a neurotoxic reaction with severe extrapyramidal features (Parkinsonism and dystonia) and delirium probably resulting from citalopram. There were no other features to support serotonin syndrome. The relatively rapid onset of extrapyramidal symptoms following commencement of an SSRI is consistent with other reports.\(^2\)\(^-\)\(^4\) The patient may possibly have had pre-existing cerebral Lewy body disease, a condition in which toxic reactions to neuroleptic drugs (thought to involve brain dopaminergic-serotonergic dysfunction) are common.\(^9\)

Clinicians need to be aware of possible extrapyramidal reactions from SSRIs, as early recognition and management is essential to prevent potentially significant adverse outcomes.

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References:


Expected versus demonstrated skills of postgraduate year 1 (PGY1) doctors in New Zealand

Old, Naden, and Child\(^1\) highlight a significant discrepancy between the skills expected of graduates at the end of postgraduate year 1 (PGY1) as indicated by the Medical Council of New Zealand (MCNZ) and those attained.

This topic has been highlighted in a number of educational and professional fora in recent years, including pre-vocational medical education meetings (2001–2005) and the Australian and New Zealand Association of Health Professional Education 2002 to 2004 conferences. We support Old et al with their call to the MCNZ to review its list of indicative skills (in consultation with all stakeholders) including those responsible for undergraduate and postgraduate education and supervision as well as health providers and employers.

We would also like to share our unpublished, but similar, data from Christchurch as well as local response to the findings. We conducted a survey to identify the skill levels and needs of PGY1 doctors commencing employment at the Canterbury District Health Board (CDHB) and to direct training accordingly.

Our survey comprised questions relating to confidence and experience in clinical management and practical skills based on the indicative list from the MCNZ. All Year 1 House Officers starting at Christchurch Hospitals in November 2002 were asked to complete the survey at orientation and again 10 months later during their final attachment.

We found considerable variability in the reported experience of skills. More than 10% of PGY1 doctors reported having no experience in 14 of the 49 emergency and acute clinical scenarios and more than 25% had never attempted 14 of the 37 practical skills at the start of their PGY1 year.

More than 10% of PGY1 doctors reported no experience in 7 of the 49 emergency and acute clinical scenarios, and more than 25% reported no experience in 14 of the 37 skills after 10 months of clinical experience. Conversely a small percentage of PGY1 doctors reported considerable experience.

Given the very broad range of indicative skills listed by the NZMC including skills often not experienced by house officers in their first year, we have introduced a self-assessment questionnaire regarding practical skills experience for house officers commencing work in the CDHB. Included in the questionnaire are 14 core practical skills from the MCNZ list (identified through a questionnaire sent to senior medical staff asking them to rank the skills they thought most important to be taught), in addition to cardiopulmonary resuscitation, language, and communication skills, computer skills, and knowledge of the New Zealand and local healthcare system.

The information from these questionnaires is used to assist in supervised and self directed learning exercises in these areas. These self assessments again show that experience is variable.
Recognising that skills acquisition will be one of learning and building on experience, a national curriculum should be more clearly defined as to what clinical and practical skills should be taught and to what level of competency prior to graduation, prior to registration for general scope of practice, and what skills should subsequently be targeted in the PGY2 year.

A national curriculum that recognises the continuum of medical education with an approach that allows junior doctors to build skills and document those attained across these 3 years with accountability and flexibility would be optimal.

Given that experience and expertise aren’t synonymous, agreement as to how competency in those skills is achieved and assessed must also be addressed. As identified by Ardagh, sufficient resources need to be allocated for the early stages of acquisition of skills as well as adequate processes for the assessment of competency and credentialling. Recognition of the importance of adequate resourcing for clinical teaching time will be essential. Greater research into the potential benefits of clinical simulation scenarios and clinical skills laboratories will also be required.

Some of these issues have begun to be addressed in the “Foundation Programme” in the United Kingdom, and Australia, and might guide the Trainee Intern, PGY1, and PGY2 curriculum in New Zealand.

We acknowledge the work of the NZMC in its initiation of discussion in the area of clinical skills, and we support the call for a coordinated approach between the NZMC, the education providers, and the DHBs to ensure that all medical graduates have the necessary skills for safe and competent medical practice in New Zealand.

**Acknowledgment:** We thank Kirsten Gaerty (Resident Medical Officer, Canterbury District Health Board) and Caroline Jewels (Resident Medical Officer, Canterbury District Health Board) for their assistance in collecting and analysing data from our original study.

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**References:**


Alcohol advertising in New Zealand

It is good to see that the NZMA Council recognises the importance of tackling the causes of the youth alcohol culture of this country. We do not treat cholera just with drugs, we examine the water supply.

The severely worsening statistics for alcohol harm, especially for women and young teenagers, call for radical thinking about the multiple causes, and their long-term solution, not just regulatory, pricing, or policing measures, important though they are.

Our Group Against Liquor Advertising, actioned largely by doctors, wonders if most of your Council or general members realise the mechanism of the situation. The liquor industry worsens the youth alcohol culture, by linking alcohol with sexuality, and with heroes of the young via sport. This is directly against their voluntary code. That code soothes the public and regulatory bodies, and helps to sustain the industry’s place, and that of the advertisers, at the policy table.

Highly sophisticated advertising, some electronic, is largely “beneath the parental radar,” and promotes youth alcohol culture. Scottish researchers have found that the industry knows the distinct preferences of even 11–14 year olds (who like inexpensive sweet drinks and colourful, wacky packaging) and 15–17 year olds (who prefer sophisticated adult brand names).\(^1\) Compelling evidence of brand identity entering the culture of young people, including Maori, is found in articles by McCreanor and others.\(^2,3\)

We invite those concerned, especially about young people, to visit our website (http://www.gala.org.nz). We are not prohibitionists, nor are we against the enjoyment of civilised drinking. Our main policy statement, listed on the homepage, summarises this large subject, and provides links to evidence-based research.

Perhaps some will want to help a small unfunded body tackling a hugely financed Goliath, a situation similar to that of the early tobacco activists. Already we alone have the distinction of achieving a forthcoming Officials’ Review on Alcohol Advertising and Sponsorship. This year is an important one for the NZMA, to achieve policy to match the statements of world leaders in this matter, the WHO, and AMA.

But again, neither your members, nor most journalists, would realise how undemocratic alcohol politics can be. The recently appointed “independent” members of the Review Committee had to be approved by the first appointees, one of whom had financial interests and whose performance was being judged. Alcohol advertising policies have been determined by reviews run by the advertisers for years. On the last occasion, they increased the television alcohol advertising time, making it earlier in the evening.

Harold Coop
Committee Member, Group Against Liquor Advertising (GALA)
Auckland
References:


Sir John Staveley

Blood transfusion pioneer. Died aged 91.

The path towards John “Jock” Staveley’s knighthood for his role as a pioneer in the field of blood transfusion began with five years of war.

Newly married to cellist Elvira Wycherley, he volunteered for active service abroad in World War II and served in Greece, escaping to Crete, then Syria, Egypt, Libya, and Italy.

After he recovered from wounds received in an ambush in Greece, he was made malarial control officer with Lieutenant-General Sir Bernard Freyberg’s New Zealand division in Syria.

“Most of the initial symptoms of malaria show up in blood manifestations, so I became interested,” he said. Severely wounded twice, a prisoner of war twice, he returned with a Military Cross and a determination to work in the fast-developing field of blood transfusion. “Having seen on such a scale [in field hospitals] what could be achieved by blood transfusion, my interest never wavered,” he wrote.

Following post-graduate study in London and Edinburgh, he was appointed pathologist and then haematologist to the Auckland Hospital Board and set about establishing a blood bank run by the board, and laboratories and donor rooms in all the major hospitals.

The next two decades were exciting. The demand for blood and blood products grew dramatically for not only Green Lane Hospital’s famous 1960s pioneering of cardiac bypass surgery, but also for orthopaedics, obstetrics and gynaecology. Blood diseases and the mounting road toll contributed to the challenge, as did forensic work.

A mobile blood collection unit was established, the Auckland Blood Transfusion Centre was opened in 1968 and New Zealand's work in transfusion medicine, largely through Sir John, became known and admired in Britain, the United States and Australia.

Research into Maori blood groups was triggered by the possibility that different blood groups might indicate differing places of origin. With the help of the Maori Affairs Department he traced more than 500 pure-blooded Maori, and compared their blood types with those of a variety of Pacific Islanders. Results were inconclusive, and he concluded that the distribution of blood groups throughout the Pacific was more geographical than racial.

Sir John’s high standards and integrity were unquestioned. He recognised that he and the blood service were often seen as backroom boys, and he remained fiercely loyal to his vision, his patients and his staff. He frequently locked horns with the Wellington health bureaucracy and the medical Establishment.
Eye problems followed a small brain haemorrhage in the early 1960s, which permanently slowed his reading and hindered his international activities. It was a cruel, hidden disability for someone famed for his quickness of thought in debate and decision-making. His movements slowed, especially during his regular walks through Newmarket for more than 30 years on his way to and from work.

Knighted in 1979, having taken early retirement at 62, he served as medical director of the Blood Foundation of New Zealand for 10 years and enjoyed a lengthy retirement in Taupo and Auckland, as patron of the Haemophilia Foundation and the Ngauruhoe Ski Club.

Sir John was born in Hokitika and educated at Timaru Boys’ High School. As a teenager he climbed with some of Southern Alps’ best. But the influence of his early mentor Dr Harry Buchanan swung the decision towards medicine, and in 1938 he graduated from Otago Medical School to become a junior resident medical officer at Auckland Hospital.

Despite an old war knee injury and a dicky ticker, he climbed well into his 70s, studied astronomy, avidly followed politics and composed songs and light music into his 90s.

The devoted grandfather to six and great-grandfather to two was widowed in 1992. Self-reliant nearly to the end, he died at his Northcote home on May 14, only days after writing to the Haemophilia Foundation congratulating it on the apology and compensation recently won from the Government regarding hepatitis C testing.

As summed up by a former close laboratory colleague, everything he did, he did for others, never himself.

The New Zealand Blood Service, which he envisioned in the 1950s and saw finally established in 1998 with its headquarters in Auckland, is his legacy.

This obituary was written by Tessa Duder (nee Staveley). It first appeared in the 27 May 2006 issue of the New Zealand Herald.
How to pass. The insider’s guide to the RACP examination

Ingrid Naden, Zoe Raos. Published by The Clinical Education and Training Unit (order from lmaskell@adhb.govt.nz), Auckland District Health Board, 2006.
Contains 149 pages. Price $40.00

Examinations are important milestones in our careers and passing them is a skill not always taught well. Every year, medical registrars across New Zealand sit written then clinical exams in March and July. This short book, written by two recently examined Auckland registrars, collects words of wisdom from fellow examinees and consultants as an aid to this specific group. Using a relaxed style of prose, with short chapters, the authors cover every aspect of the exam process, from when to start studying to what it is like to pass. Cartoons and quotations add to the readability of the book.

The shorter first section includes sensible advice on general study technique as well as more specific insights on topics to study. A comparison of available textbooks and resources presents their pros and cons but doesn’t come to specific conclusions. More informative is a chapter on useful websites and a list of revision courses available in Australasia. The remainder of the book is devoted to the clinical exam covering general advice, book and revision course reviews, long and short cases. Tips and exam techniques are presented in well-organised and readable chunks, and two chapters summarise past candidates’ exam cases. The long-case section is a strength, but there is one notable omission from the introduction to this section: the authors do not mention that examiners in the long case can award “+” and “-” grades, as well as integers, for example “4+”.

There are already a number of books aimed at this market, with Talley and O’Connor’s text the most well-known, and much of “How to Pass” is covered already. The major contributions of this book are the reviews of local resources available to the potential candidate and the understanding that these “insiders” have of what it does feel like coming up to this milestone. Candidates from centres with less active teaching programmes may find this collected experience from Auckland registrars and consultants helpful.

John Young
Neurology Registrar
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John Fink
Director of Physician Training
Christchurch Hospital
The good writing guide


I thought this book was a mixed bag. It is a small New Zealand-produced paperback with a simple title, and it is a useful introduction to the basics of good writing. I found some sections more useful and interesting than others—in general, the second half of the book (giving specific advice and useful templates) is better than the first half which starts with grammar (a loathsome, boring subject for many that would be better placed at the end). Indeed, it may discourage some readers from continuing with or buying the book.

To recognise and write good English, sticking to the three c’s (clarity, conciseness, and consistency) is far more important than an in-depth knowledge of grammar principles and terminology I believe, while two p’s (punctuation and paragraphing) are under-rated in importance.

The book covers technical terms; sentence structure; punctuation; paragraphing; words to watch; commonly confused words (e.g. affect versus effect); language flow; referencing; quantitative information; strengths to build into your writing; and sample formats/templates—including writing essays, reports, various letters (e.g. complaints), proposals, and memos. I thought the final section (sample formats/templates) was particularly interesting and useful, especially for people in a hurry.

Sometimes the obvious is stated (e.g. “two is a number, too means also”) which suggests the book is primarily aimed at writers whose first language is not English as well as error-prone native English writers who need a lot of help. (The author previously taught at Universiti Putra Malaysia and at Chuo University in Japan. She now teaches part time at Waikato and Massey, and conducts training sessions for New Zealand organisations.)

People who are already reasonably proficient at writing but want to polish and build on their skills would be better advised to get the 500-page Write Edit Print: Style Manual for Aotearoa New Zealand. And for a book of comparable size and price to McLaren’s, then Strunk and White’s old classic The Elements of Style may be a better choice if you can find it (although it doesn’t show templates).

Brennan Edwardes
Production Editor, NZMJ