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Body of the paper – there should be a brief introduction (no heading) followed by sections for Methods, Results, Discussion, Acknowledgements and Correspondence.

References – in the text use superscript numbers for each reference. Titles of journals are abbreviated according to the style used by Index Medicus. For articles and journals the format is: Bratvedt GD. Outcome of managing impotence in clinical practice. NZ Med J 1999: 112: 272-4. For book chapters the format is: Marks P. Hypertension. In: Baker J, editor. Cardiovascular disease. 3rd ed. Oxford: Oxford University Press; 1998, p567-95. Note all authors where there are four or less; for five or more authors note only the first three followed by ‘et al’. Personal communications and unpublished data should also be cited as such in the text.

Tables should be on separate sheets with self-explanatory captions. Footnote symbols must be used in a set sequence (* † ‡ § etc).

Figures must be glossy prints or high quality computer printouts. Since these are likely to be reduced in size when printed, use large type and approximately twice column size for the figure.

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New Zealand Medical Journal 26 April 2002
New Zealand’s HIV infected population under active follow-up during 2000

Graham Mills, Infectious Diseases Physician, Waikato Hospital; Anne-Marie Yardley, Medical Student, Auckland School of Medicine; Mark Thomas, Infectious Diseases Physician, Auckland Hospital; Tim Blackmore, Infectious Diseases Physician, Wellington Hospital; Alan Pithie, Infectious Diseases Physician, Christchurch Hospital; Bryan Schroeder, Scientific Officer, Virology Laboratory, Auckland Hospital; Nigel Dickson, AIDS Epidemiology Group, Department of Preventive and Social Medicine, University of Otago, Dunedin.

Abstract

Aim. To audit New Zealand’s HIV infected population currently under active follow-up.

Methods. Multiple sources were used to determine anonymously the demographic and management characteristics of HIV infected individuals being monitored with HIV viral load measurements and/or receiving antiretroviral therapy during 2000.

Results. 593 people (480 males and 113 females) were under active follow-up. The most common transmission risk was male homosexual contact (56%) followed by heterosexual contact (28%), injecting drug use (3%) and mother to infant transmission (1%). Ethnicity data showed a disproportionate number of Africans (13%) compared to recent census figures. Anti-retroviral therapy was used in 71% of the cohort of whom 62% had HIV viral load measurements below 400 copies/mL. An upper estimate of diagnosed HIV individuals living in New Zealand at 30/9/2000 was 801.

Conclusions. This is the first time that the demographic and clinical state of HIV infected individuals has been assessed throughout New Zealand. The results suggest a slightly lower number of HIV infected individuals currently living in New Zealand than previously estimated. Anti-retroviral therapy is being used effectively within the HIV infected population. The changing demographics, with a higher proportion of people under care from Africa, increasing numbers of females, and an increase in the proportion with heterosexual risk factors are particular challenges.

HIV infection prevalence in New Zealand is currently low by world standards with the rate estimated by UNAIDS at the end of 1999 to be 6.2 per 10,000 adults aged 15-49 years. This rate is less than half the estimate for Australia (14.7 per 10,000), very much lower than the United States (61 per 10,000) and almost pales into insignificance when compared with South Africa where it is estimated that 20% of the 15-49 year age group are infected. Nevertheless, the effect of HIV infection in New Zealand has a major impact on the lives of those infected, their families and friends and those close to the more than 550 people who have already died as a consequence of HIV/AIDS.

The last six years have seen dramatic changes in the management of HIV infection, with the advent of highly active anti-retroviral therapy (HAART). HAART, defined as combination therapy with three or more anti-retroviral drugs, has led to marked falls in the incidence of opportunistic infections and increases in life expectancy for those with HIV infection. HIV medical care in New Zealand is provided by a limited number of infectious diseases physicians, sexual health specialists and some general practitioners (GPs) with high HIV caseloads. This small number of providers has enabled us to undertake a national audit of New Zealand’s HIV infection population receiving ongoing follow-up.

The primary aims were to determine the number and demographic characteristics of those being followed during the first nine months of 2000 through linking HIV surveillance data to the immunological, viral load and treatment regimen status of individuals. In addition these data were used to estimate the total number of people with diagnosed HIV living in New Zealand.

Methods

This clinical audit was undertaken as a partnership between clinicians involved in the care of patients with HIV infection and the AIDS Epidemiology Group. An anonymous database was established of all living individuals at 30/9/2000 who had at least one plasma HIV viral load performed within New Zealand between January and September 2000 and/or were dispensed anti-retroviral therapy between July and September 2000. These individuals were considered to be under active follow-up. The project was undertaken by linking data elements from currently existing databases using the same anonymous code. These databases included HIV viral load results from the four New Zealand laboratories that are equipped to undertake this test; special authority approvals for the prescription of anti-retroviral drugs; prescription dispensing records between July and September 2000; mortality data from the New Zealand Health Information Service; and surveillance data held by the AIDS Epidemiology Group. Information was collected on the current age, sex, ethnicity, mode of transmission, location, the date and result of the latest HIV viral load and CD4+ lymphocyte counts, current anti-retroviral regimen, and the treating clinician.

To estimate the total number of people diagnosed with HIV currently living in New Zealand (both those under and not under active follow-up), a review was undertaken of the 330 enhanced surveillance reports - introduced in 1996 for new diagnoses of HIV infection - received between January 1996 and December 1999. The notifying clinicians of those people without evidence of being followed and who were not known to have died, were contacted in September 2001 to determine whether they were still in New Zealand, and to ascertain their current status with respect to clinical care. Statistical analysis was undertaken using EpiInfo™ 2000. Means of HIV RNA were calculated after log transformation. The Kruskal-Wallis test and Mantel-Haenszel Chi squared test were used where appropriate.

Results

Characteristics of the cohort. A total of 593 individuals met the definition of being under active follow-up. Of these, 480 (81%) were male and 113 (19%) female (Table 1). The mean
Untreated individuals had CD4+ lymphocyte counts below this value with no evidence for any barrier to treatment by gender, ethnicity or mode of transmission.

**Treated individuals.** 419 (71%) of the cohort were currently on any anti-retroviral therapy with 373 (89%) of these receiving HAART. Among those treated, the latest HIV viral load result was below 400 copies/mL (<2.6 log10) in 62% (Table 2). Response to therapy did not show a gender difference as reflected by mean log10 HIV viral load (male 2.6 vs female 2.5; p = 0.46) and mean CD4+ lymphocytes/mm3 (male 455 vs female 476; p = 0.58). CD4+ lymphocyte count and HIV viral load differences were not observed after stratification by ethnicity and mode of transmission (data not shown). Of the 196 individuals with CD4+ lymphocyte counts below 350/mm3, 158 (80.6%) were on anti-retroviral therapy.

**Untreated individuals.** 174 individuals were not receiving anti-retroviral therapy. Among the untreated, men had higher mean log10 HIV viral load than women (4.2 vs 3.8; p = 0.003) while differences were not seen in mean CD4+ lymphocyte counts between men and women (507/mm3 vs 462/mm3; p = 0.32). Table 3 shows the demographic characteristics of untreated individuals by CD4+ lymphocyte count. The proportion with a CD4+ lymphocyte count below 350/mm3 is shown because current guidelines suggest limiting treatment to-infant transmission (1%) was low. There were considerable differences across ethnic groups with 89% of African men and 48% of Asian men acquiring HIV infection through heterosexual contact compared with 7% of European men. The clinical care of 93% of the 593 individuals occurred in four locations: Auckland (56%), Wellington (19%), Christchurch (12%) and Hamilton (6%).

**Table 1. Demographic characteristics and presumed mode of transmission for the cohort.**

<table>
<thead>
<tr>
<th>Age Group (years)</th>
<th>Male n=480</th>
<th>Female n=113</th>
<th>Total n=593</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;16</td>
<td>4 (1%)</td>
<td>6 (5%)</td>
<td>10 (2%)</td>
</tr>
<tr>
<td>16-29</td>
<td>46 (10%)</td>
<td>33 (29%)</td>
<td>79 (13%)</td>
</tr>
<tr>
<td>30-39</td>
<td>191 (40%)</td>
<td>49 (43%)</td>
<td>240 (41%)</td>
</tr>
<tr>
<td>40-49</td>
<td>143 (10%)</td>
<td>18 (16%)</td>
<td>161 (27%)</td>
</tr>
<tr>
<td>&gt;49</td>
<td>96 (20%)</td>
<td>7 (6%)</td>
<td>103 (17%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Male n=480</th>
<th>Female n=113</th>
<th>Total n=593</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>317 (66%)</td>
<td>42 (37%)</td>
<td>359 (61%)</td>
</tr>
<tr>
<td>Maori</td>
<td>42 (9%)</td>
<td>7 (6%)</td>
<td>49 (8%)</td>
</tr>
<tr>
<td>Pacific Is</td>
<td>11 (2%)</td>
<td>7 (6%)</td>
<td>18 (3%)</td>
</tr>
<tr>
<td>African</td>
<td>42 (9%)</td>
<td>17 (13%)</td>
<td>59 (10%)</td>
</tr>
<tr>
<td>Asian</td>
<td>29 (6%)</td>
<td>10 (9%)</td>
<td>39 (7%)</td>
</tr>
<tr>
<td>Other</td>
<td>11 (2%)</td>
<td>1 (1%)</td>
<td>12 (2%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>28 (6%)</td>
<td>9 (8%)</td>
<td>37 (6%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mode of Transmission</th>
<th>Male n=480</th>
<th>Female n=113</th>
<th>Total n=593</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male homosexual contact</td>
<td>334 (70%)</td>
<td>-</td>
<td>334 (56%)</td>
</tr>
<tr>
<td>Heterosexual contact</td>
<td>84 (18%)</td>
<td>83 (74%)</td>
<td>167 (28%)</td>
</tr>
<tr>
<td>Injecting drug use</td>
<td>11 (2%)</td>
<td>6 (5%)</td>
<td>17 (3%)</td>
</tr>
<tr>
<td>Mother to child</td>
<td>3 (1%)</td>
<td>3 (3%)</td>
<td>6 (1%)</td>
</tr>
<tr>
<td>Receipt of blood/products</td>
<td>12 (2%)</td>
<td>3 (5%)</td>
<td>15 (3%)</td>
</tr>
<tr>
<td>Other/undetermined</td>
<td>36 (7%)</td>
<td>18 (16%)</td>
<td>54 (9%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location of Care</th>
<th>Male n=480</th>
<th>Female n=113</th>
<th>Total n=593</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auckland</td>
<td>266 (55%)</td>
<td>64 (57%)</td>
<td>330 (56%)</td>
</tr>
<tr>
<td>Hamilton</td>
<td>24 (5%)</td>
<td>9 (8%)</td>
<td>33 (6%)</td>
</tr>
<tr>
<td>Wellington</td>
<td>91 (19%)</td>
<td>24 (21%)</td>
<td>115 (18%)</td>
</tr>
<tr>
<td>Christchurch</td>
<td>63 (13%)</td>
<td>9 (8%)</td>
<td>72 (12%)</td>
</tr>
<tr>
<td>Other</td>
<td>36 (7%)</td>
<td>7 (6%)</td>
<td>43 (7%)</td>
</tr>
</tbody>
</table>

Ethnicity of New Zealand Population: European 79.9%, Maori 14.7%, Pacific Is 6.4%, African <0.2%, Asian 6.6%. NZ Census Data 2001.
Table 2. Relationship between antiretroviral treatment, HIV viral load and CD4\(^+\) lymphocyte count.

<table>
<thead>
<tr>
<th>HIV viral load (log(_{10}) copies/mL)</th>
<th>Untreated (n=174)</th>
<th>Treated (n=119)</th>
<th>Total (n=593)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.7</td>
<td>5 3%</td>
<td>218 52%</td>
<td>223 38%</td>
</tr>
<tr>
<td>1.7-2.6</td>
<td>5 3%</td>
<td>43 10%</td>
<td>48 8%</td>
</tr>
<tr>
<td>2.6-3.3</td>
<td>16 9%</td>
<td>35 8%</td>
<td>51 9%</td>
</tr>
<tr>
<td>3.3-4.0</td>
<td>41 24%</td>
<td>40 10%</td>
<td>81 14%</td>
</tr>
<tr>
<td>4.0-4.8</td>
<td>68 39%</td>
<td>49 12%</td>
<td>117 20%</td>
</tr>
<tr>
<td>&gt;4.8</td>
<td>39 22%</td>
<td>32 8%</td>
<td>71 12%</td>
</tr>
<tr>
<td>Missing</td>
<td>2 2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Log(_{10})</td>
<td>4.1</td>
<td>2.6</td>
<td>3.1</td>
</tr>
</tbody>
</table>

Table 3. Characteristics of the 174 untreated individuals by CD4\(^+\) lymphocyte count (per mm\(^3\)).

<table>
<thead>
<tr>
<th>CD4&lt;150 (n=38)</th>
<th>CD4 (\geq 150) (n=118)</th>
<th>Missing CD4 (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>27 85%</td>
<td>11</td>
</tr>
<tr>
<td>Female</td>
<td>11 35%</td>
<td>7</td>
</tr>
<tr>
<td>European</td>
<td>20 67%</td>
<td>8</td>
</tr>
<tr>
<td>Maori</td>
<td>5 18%</td>
<td>2</td>
</tr>
<tr>
<td>Pacific Is</td>
<td>1 3%</td>
<td>1</td>
</tr>
<tr>
<td>African</td>
<td>7 23%</td>
<td>1</td>
</tr>
<tr>
<td>Asian</td>
<td>3 10%</td>
<td>1</td>
</tr>
<tr>
<td>Other</td>
<td>0 2%</td>
<td>0</td>
</tr>
<tr>
<td>Unknown</td>
<td>2 10%</td>
<td>5</td>
</tr>
<tr>
<td>Male homosexual contact</td>
<td>18 51%</td>
<td>6</td>
</tr>
<tr>
<td>Heterosexual contact</td>
<td>12 45%</td>
<td>6</td>
</tr>
<tr>
<td>Injecting drug use</td>
<td>3 3%</td>
<td>0</td>
</tr>
<tr>
<td>Mother to child</td>
<td>0 1%</td>
<td>0</td>
</tr>
<tr>
<td>Receipt of blood/products</td>
<td>1 5%</td>
<td>0</td>
</tr>
<tr>
<td>Other/undetermined</td>
<td>4 15%</td>
<td>6</td>
</tr>
</tbody>
</table>

Discussion

This national audit is the first attempt to ascertain the number, demographic characteristics and clinical state of diagnosed HIV infected individuals under active follow-up and currently living in New Zealand. The approach taken reduces the potential for bias that may be associated with reports from more selected cohort populations. In defining active follow-up for our study population, the assumption was made that HIV viral load testing would be undertaken as part of care at least once during the first nine months of 2000. This practice would be consistent with current guidelines\(^7\) that recommend the measurement of plasma HIV viral load levels every 3-4 months. The observation that only two individuals receiving anti-retroviral therapies did not have an HIV viral load result during the study period suggests that New Zealand clinicians are following these recommendations for treated individuals. The requirement for an HIV viral load test to be undertaken within the first nine months of 2000 for inclusion in our cohort has excluded a small number of untreated subjects as indicated by the ‘reappearance’ of seventeen individuals in the twelve months after September 2000.

This study reflects the epidemiology and medical management of the diagnosed HIV infected population alive during 2000 and highlights a major shift in the characteristics of New Zealand’s HIV infected population in recent years. Compared with AIDS notification data from the first seven years of the epidemic (1983-89)\(^4\) the proportion of males under care for HIV infection is 81% (previously 97%), while the proportion of European, Maori or Pacific Island combined has fallen from 93% to 72%. Although male homosexual contact remains the major mode of transmission, this has fallen from 88% to 56% over the last decade. The increase in number of women diagnosed with HIV will have an ongoing impact on the nature of New Zealand’s epidemic and will require new preventive strategies including efforts to avert perinatal HIV transmission. The number of HIV infected migrants from high prevalence countries has risen considerably in recent years.\(^3\) This has created a number of challenges for both the clinicians providing clinical care and the associated support services as a result of cultural, religious and language differences.

The optimal time to initiate antiretroviral therapy remains uncertain with ongoing vacillation between aggressive and conservative approaches. Current guidelines\(^8\) suggest that patients with fewer than 350 CD4\(^+\) lymphocytes/mm\(^3\) should be offered therapy. In our cohort, 71% were currently receiving anti-retroviral therapy, which is slightly lower than the 78% treatment uptake rate during 2000 within the Australian HIV Observational Database\(^6\) but higher than the 62% on therapy during 2000 within the Royal Free Hospital, London cohort of over 1000 patients (C Sabin, personal communication.) Evidence of barriers to therapy related to gender, ethnicity or mode of transmission were not seen with uptake rates among those with CD4\(^+\) lymphocyte counts below 350/mm\(^3\) being similar. The sex difference in HIV viral load results among untreated patients has been noted in previous studies and is considered to have a biological basis.\(^9\) Of those currently on treatment in New Zealand, 62% have HIV viral loads of less than 400 copies/mL. HIV virological control did not differ by gender, ethnicity or among subjects with differing modes of transmission. The reduction in HIV virological burden in New Zealand’s treated population is comparable to cohort data from both an individual and public health perspective for those themselves from clinical care. There is considerable concern however have suggested that some individuals have excluded themselves from clinical care. There is considerable concern from both an individual and public health perspective for those with diagnosed HIV infection who are not receiving ongoing clinical care.

At the end of 1998, based on the number of individuals diagnosed with HIV and those with AIDS who had died or left the country, a calculation of 770 people living with diagnosed HIV in New Zealand was made.\(^3\) The estimate for 30/9/2000 using the same calculation was 915 (AIDS Epidemiology...
Depression in patients in an Auckland general practice

Bruce Arroll, Associate Professor; Felicity Goodyear-Smith, Senior Lecturer; Trevor Lloyd, Senior Lecturer; Division of General Practice & Primary Health Care, Faculty of Medical & Health Sciences, University of Auckland.

Abstract

Aim. To measure the rate of detected and undetected depression in patients attending an Auckland general practice.

Method. At their consultation conclusion, general practitioners (GPs) asked all consecutive patients over sixteen years attending for consultation to participate in a health and mood questionnaire. A researcher administered the Beck Depression Inventory (BDI) to consenting participants. The GPs previously recorded whether they considered these patients depressed.

Results. Response rate among patients was 81% (253/314). The BDI found a 13.8% (35/253) 95% CI (9.6-18.5) depression prevalence among patients. GPs picked up 51% of cases (sensitivity 0.51 and specificity 0.91). Māori patients were no more likely to be depressed than non-Māori but they were less likely to be receiving or have received treatment with antidepressants.

Conclusion. The rate of depression in this practice was higher than an earlier study suggesting the true rate may be >10%. GPs see more depressed patients than other health professionals, therefore improvement in detection and management of depression in primary care is important. More work is needed on the difference between Māori and non-Māori in the use of antidepressants.

Depression is a common and costly mental health problem seen frequently in general practice and general medical settings. Researchers at Harvard University estimate that by 2020 unipolar depression will be second only to ischemic heart disease as the leading cause of disability adjusted life years. When self rated depression scores are used, between 5.5% and 65% of participants are thought to be depressed depending on where the threshold values are set for the self-rating scale. This wide range of prevalence estimates indicates a need for high quality studies about depression set in primary care. The annual economic burden of depression in the US (including direct care costs, mortality costs and morbidity costs) has been estimated to total almost $44 billion.

Major depressive disorder can result in serious sequelae. The suicide rate in depressed persons is at least eight times greater than an earlier study suggesting the true rate may be >10%. GPs see more depressed patients than other health professionals, therefore improvement in detection and management of depression in primary care is important. More work is needed on the difference between Māori and non-Māori in the use of antidepressants.

levels associated with major chronic medical conditions such as diabetes, hypertension or coronary heart disease. Also, depressed persons frequently present with a variety of physical symptoms (three times the number of somatic symptoms compared to controls in one study), leading to excess utilisation of medical services.

The prevalence of depression in New Zealand general practice has not been clearly established. The WaiMedCa study of general practice patients in the Waikato found that 4.4% of patients received a ‘psychological’ diagnosis. Depression was reported as being 0.5% of all new problems and 0.9% of existing problems. The WaiMedCa study attempted only to identify the one main reason for presentation at the consultation, and this may partly explain the low result. A cross-sectional population study undertaken in Christchurch showed a 3.7% rate for the two week prevalence of depression and a 12.6% rate for the one year prevalence. The WaiMedCa results have long been regarded as a low estimate given the findings in overseas studies and the Christchurch study. The recent MaGPte study found that GPs thought that 20.7% of their patients described symptoms that were partially or fully psychological within the current consultation, a sizeable increase on the WaiMedCa findings. There is considerable evidence that GPs miss cases of depression and it would be helpful to have an estimate of that situation. The aim of this study was to measure the rate of detected and undetected depression in general practice patients. The term screening is usually used for assessment in asymptomatic patients whereas patients with undiagnosed depression will have symptoms. Thus we shall use the term case finding.

Methods

This study was undertaken at an Auckland practice with approximately five full time equivalent doctors (four full time one half time and one three tenths). The practice is located in South Auckland and has 25-33% of its patients describing themselves as being of Māori ethnicity. Consecutive patients over the age of sixteen years were asked by their GP if they would participate in a survey about their health and mood at the conclusion of their consultation. Those who consented were referred to a research interviewer. The interviewer obtained written consent and administered the Beck Depression Inventory (BDI) in a separate office following the consultation. Prior to this the GPs had made a note on a piece of paper as to whether or not they thought these patients were depressed. If they were considered suicidal, they could signal the interviewer that this issue had been addressed.

Patients were excluded from the study if they were cognitively impaired or unlikely to read English. Scores from the inventory were reported back to the patients as soon as they had completed it. Those patients who gave any positive responses to the suicidal feeling questions on the inventory were asked by the interviewer to return to see the doctor. Others who were depressed but not suicidal were asked to return to see the GP in the near future.

It was decided to use the BDI to identify cases of depression. Mulrow et al assessed nine case finding instruments, including the BDI, in eighteen studies of depression in primary care. Their interpretation was that all the instruments had reasonable operating characteristics, and selection of a particular instrument was dependent on issues such as feasibility, administration and scoring times and the instrument’s ability to serve additional purposes such as monitoring severity or response to therapy. The BDI intentionally does not include items on physical symptoms such as decreased appetite, decreased sleep and agitation as these are very common in the general population. The National Health Committee guidelines recommended the Centre for Epidemiological Studies – Depression (CES-D) or the Hamilton Depression Inventory (HAM-D). The CES-D does not have a question about suicide and the HAM-D requires it to be interviewer administered. As a validated, short, self-administered tool, the BDI does not have a question about suicide and the HAM-D requires it to be interviewer administered. The BDI was chosen for this study because it focuses on psychological rather than physical aspects of depression and hence this was used to dichotomize the group in order to measure the sensitivity and specificity.

There were 253 consecutive patients found to be depressed female, with a median age of 40 years (range 18-70). Table 1 shows the range of BDI scores for different cut-points and by gender and Māori and non-Māori. The choice of ranges is to facilitate comparison with other studies that used different cut-points. The rate of depression (using >16 as the cut point) is 16%, 9%, 11% and 15% for women, men, Māori and non-Māori respectively. The rate of depression (using >10 as the cut point) is 34%, 20%, 33%, 28% for women, men, Māori and non-Māori respectively.

Discussion

This is the first study looking specifically at depression in New Zealand general practice patients and suggests that rates

<table>
<thead>
<tr>
<th>Range of BDI scores*</th>
<th>Number of patients</th>
<th>Female patients n(%)</th>
<th>Male n(%)</th>
<th>Māori n(%)</th>
<th>non-Māori n(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10 normal</td>
<td>179</td>
<td>109 (66%)</td>
<td>70 (80%)</td>
<td>43 (67%)</td>
<td>136 (72%)</td>
</tr>
<tr>
<td>11-13 mild mood disturbance</td>
<td>17</td>
<td>14 (9%)</td>
<td>3 (3%)</td>
<td>3 (5%)</td>
<td>14 (7%)</td>
</tr>
<tr>
<td>14-16 mild mood disturbance</td>
<td>22</td>
<td>15 (9%)</td>
<td>7 (8%)</td>
<td>11 (17%)</td>
<td>11 (6%)</td>
</tr>
<tr>
<td>17-20 borderline clinical depression</td>
<td>10</td>
<td>9 (5%)</td>
<td>1 (1%)</td>
<td>1 (2%)</td>
<td>9 (5%)</td>
</tr>
<tr>
<td>21-30 moderate depression</td>
<td>14</td>
<td>8 (5%)</td>
<td>6 (7%)</td>
<td>4 (6%)</td>
<td>10 (5%)</td>
</tr>
<tr>
<td>31-40 severe depression</td>
<td>10</td>
<td>9 (5%)</td>
<td>1 (1%)</td>
<td>2 (3%)</td>
<td>8 (4%)</td>
</tr>
<tr>
<td>Greater than 40 extreme depression</td>
<td>1</td>
<td>1 (1%)</td>
<td>0</td>
<td>0</td>
<td>1 (1%)</td>
</tr>
</tbody>
</table>

* The uneven size range of BDI scores is to enable comparison with other studies. Table 2 shows the study results according to Māori and Non-Māori. There was a significant difference between those who were Māori, had a BDI score > 10 (mild mood disturbance) and who had been or were on antidepressants 4% (1/26) and those whose ethnicity was non-Māori 31% (3/977). There was no difference in the proportion of Māori and non-Māori with depression in the BDI range >16. There was no difference in the average BDI for all Māori compared with all non-Māori.
are almost certainly higher than previously measured and are similar to those found overseas. A strength of our study was the 81% response rate. Another strength was the inclusion of ethnicity. We believe this is the first New Zealand study to make comparisons of Māori and non-Māori in terms of depression. While Māori are no more likely to be depressed in this study they were significantly less likely to be treated with antidepressant medication than non-Māori. We cannot link this to the high suicide rate in young Māori, as there were very few adolescents in the study. There was a non-significant difference between the proportion of Māori (71%) and non-Māori (64%) in terms of having a community services card. This information suggests that there is a gap in prescribing of antidepressant medication to Māori patients. We cannot tell from this study if this is an issue on the part of the GPs or an issue to do with patients not wanting to take medication. If it is the former then this is further evidence of a health gap between Māori and non-Māori.

Table 2. Māori non-Māori comparisons.

<table>
<thead>
<tr>
<th>BDI &gt; 10 and now or ever been on antidepressants*</th>
<th>Māori</th>
<th>non-Māori</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI &gt; 10*</td>
<td>21</td>
<td>53</td>
</tr>
<tr>
<td>BDI &gt; 16/Māori or non-Māori†</td>
<td>7/64</td>
<td>28/189</td>
</tr>
<tr>
<td>Average BDI (age) [7]</td>
<td>8.07 (0.74) [64]</td>
<td>8.64 (0.498) [189]</td>
</tr>
<tr>
<td>Have CSC card§</td>
<td>46</td>
<td>122</td>
</tr>
<tr>
<td>Do not have CSC card§</td>
<td>18</td>
<td>67</td>
</tr>
</tbody>
</table>

*p=0.007 Fisher’s exact. †Chi squared statistic p= 0.43. ‡t test 0.55. §Chi squared statistic p=0.28.

A weakness in this study was the use of the BDI as the measuring tool, whereas the gold standards are interviews with psychiatrists. However, the BDI is a validated, short self-administered instrument feasible to use in GP settings for research.

The prevalence of depression in this study, as measured by the BDI, is similar to that in other studies. In one Australian study in a primary care clinic they found 25.1% of women and 16.6% of men were depressed when the cut point of >10 was used. Our study found 34% of women and 20% of men would be depressed at that cut–point. A Health Maintenance Organisation in middle class Wisconsin found 18.3% of patients were suffering from depression when they used the cut-point of >13 while in our study the figure is 23%. Our findings are very similar to those in a World Health Organisation study of 25 916 primary care patients using ICD-10 criteria which found 10.4% of patients had depression and 2.1% had dysthymia.” This suggests that the WaiMedCa study underestimated mental health conditions in general and depression in particular.

The fact that many depressive illnesses were missed by their GPs is a common finding in overseas studies of screening/case finding for depression in primary care. Other studies have found similar figures to ours for rates of missed depression. There is evidence that missed depression does not have adverse consequences but in view of the poor prognosis of depression (60% still meet the criteria for caseness at one year) improving compliance with treatment may be a more important aim. This is controversial as another study found a greater reduction in symptoms on the GHQ at three months but not at twelve months in a World Health Organisation study of Psychological Problems in General Health Care. There are many reasons why primary care physicians and psychiatric diagnostic instruments may differ in assessment of depression. These include physician factors such as beliefs in the effectiveness of treatment, comfort with psychological views, perceived time and role responsibility and skills in acquiring information and assessing non-verbal skills. Patient factors include absence of self-awareness, co-morbid medical illness, physical symptoms, and degree of somatisation, sub-threshold depression and factors such as shame, guilt and hopelessness. Certain key skills in the consultation have been identified that are both teachable and associated with increased rates of recognition. However, teaching better consultations skills leads to only a modest increase in detection rates yet primary care physicians who are better at detection also have better management skills.

The patient initiates most consultations in primary care. The content of the typical primary care consultation and its outcome will be influenced by what the patient chooses to present and how he or she chooses to present it. Many patients with psychological disorders present to their GP with common somatic symptoms – which are the currency of general practice.

While the BDI is a useful research tool, it is too cumbersome and time consuming for routine use, and requires a copyright fee of about $5.00 to be paid each time it is used. The BDI Fast Scan, a short (seven question) version requires a copyright fee of about $1. Validated in at least four different studies, it asks only psychological questions, which facilitates its use in a medical environment where appetite and sleep disturbance may be due to medical disorders. Our choice of one instrument was out of concern that patients may find additional questionnaires tedious and hence not consent or not complete all the questions. This was not the case and in other and future studies we are using more than one questionnaire. Our choice in future would be the short BDI for Primary Care now known as the BDI Fast Scan rather than the 21 question BDI. It has seven questions and has been validated in a number of settings including general practice and medical outpatients. This makes a point of focusing on psychological aspects of depression in a primary care setting so that issues of appetite loss, weight loss and sleep disturbance, which can be symptoms of physical illness, do not cloud the picture.

A number of studies have shown benefit from treatment in primary care settings. They have usually involved some process such as psychological treatment in addition to the usual pharmaceutical management of depression. At least 80% of the New Zealand population visit their GP each year. GPs see the majority of patients with depressive conditions. There is evidence that if GPs make the diagnosis themselves the patients are more likely to be given antidepressants.

There is a need to improve detection of depression in primary care in order to ensure appropriate patients are offered treatment. Improved understanding of the prevalence and prognosis of depression in general practice is also necessary. Given the degree of undetected depression uncovered by our study, the utility of very short screening tools requires further evaluation. Further work is also required on the discrepancies in antidepressant use between Maori and non-Maori.

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Ethnic and gender differences in the use of coronary artery revascularisation procedures in New Zealand

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Abstract

Aims. To examine ethnic and gender variations in the use of coronary artery revascularisation procedures in New Zealand and to determine whether the introduction of priority scores affected intervention trends.

Methods. Analysis of the National Minimum Database for coronary artery bypass graft (CABG) and percutaneous transluminal coronary angioplasty (PTCA) intervention rates for New Zealand Pacific, Maori and other men and women aged 40 years and over during the decade 1990-1999.

Results. Coronary artery revascularisation rates were lower in women than in men in all ethnic groups and in Pacific and Maori men compared with other New Zealand men. Compared to all men, the mean age-standardised CABG and PTCA intervention rate ratios in all women were 0.34 and 0.36. Compared to other New Zealand men, the mean age-standardised CABG and PTCA intervention rate ratios were 0.64 and 0.25 in Pacific and 0.40 and 0.29 in Maori men respectively. Compared to other New Zealand women, the rate ratios for CABG and PTCA were 0.73 and 0.21 in Pacific and 0.74 and 0.43 in Maori women respectively. Introducing priority scores was neither associated with reduced cardiac procedures nor significantly reduced variation in procedures across all ethnic groups.

Conclusions. Although Pacific and Maori peoples had higher rates of coronary artery disease morbidity and mortality, revascularisation rates were lower in both groups. Strategies beyond the use of priority scores are needed to address ethnic and gender disparities in coronary artery revascularisation procedures in New Zealand.

Coronary artery disease is the leading cause of morbidity and mortality in New Zealand adults. It contributes to about 40% of all deaths and 11% of all hospitalisations each year.1 Hospital admissions for congestive heart failure alone are estimated to cost at least $50 million or 1% of the total health annual budget.2 Coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA) are well-established methods of myocardial revascularisation which, when applied to suitable individuals can significantly reduce morbidity and mortality associated with coronary artery disease.3,4 The recent increase in use of coronary artery stents has improved the safety and success rate of these procedures and increased the range of people who can benefit from cardiovascular interventions.5,6 Mortality from coronary artery disease in New Zealand declined by 3-9% each year during the 1990s. This decline, however, was slower than in other developed countries during the same time period.7 About half the coronary artery disease mortality rate decline in New Zealand has been attributed to medical therapies and half to reductions in major risk factors.7 Risk factors for heart disease are more prevalent among Pacific and Maori peoples compared with other New Zealanders.8 The Auckland Coronary Artery and Stroke (ARCS) Study found that morbidity rates from coronary artery disease were similar in all ethnic groups9 but other studies have shown that hospitalisation rates were higher among Pacific and Maori peoples compared with other New

Zealanders.\textsuperscript{10,11} Pacific and Maori peoples have higher mortality rates from coronary artery disease and the rate of decline in mortality rates has been slower compared with other New Zealanders.\textsuperscript{3} Despite the higher burden of illness from coronary artery diseases, Pacific and Maori peoples are lower users of preventive and primary health care services. Per capita consultation rates with general practitioners and uptake of preventive services are lower among Pacific and Maori peoples compared with other New Zealanders.\textsuperscript{12,13}

In 1996, in order to ensure consistency and transparency in prioritising patients for surgery, the New Zealand government introduced numerical scores based on the individual's biological and social risk factors for coronary artery disease.\textsuperscript{14} To date, observational studies examining utilisation rates of coronary artery revascularisation procedures in New Zealand have not included data on patient ethnicity.\textsuperscript{15,16} This study examines ethnic and gender patterns in coronary artery revascularisation procedures and investigates whether introducing priority scores affected intervention rates. The findings may assist policy development in planning health care services.\textsuperscript{17} Assessing and monitoring activities designed to reduce disparities in the use of health care services between Pacific, Maori and other New Zealanders.

Methods

The New Zealand Health Information Service (NZHIS) National Minimum Database (NMDS) contains information on all public and private hospitalisations in the country coded according to the International Classification of Disease Version 9 (ICD9). We obtained data from the NMDS for all coronary artery bypass graft (ICD 361) and percutaneous transluminal coronary angioplasty (ICD 360) events reported from 1990 to 1999. All people who received one or more CABG procedures during the study period were counted once. Similarly, all people who received one or more PTCA procedures were counted once. People who received both a CABG and a PTCA procedure were counted once for each procedure type.

We calculated procedure rates using the 'usually resident' annual population estimates aged 40 years and over provided by Statistics New Zealand for Maori and the total New Zealand population. Information on the Pacific population was available for 1991 and 1996. Estimates of the Pacific population for the intervening years were calculated from the census data assuming linear population growth. Assuming linear population growth is subject to error because the Pacific population is among the fastest growing groups in the country. Error is likely to underestimate actual Pacific population growth and reduce intervention rate even further.

We obtained mortality rates from the ARCS study in Pacific, Maori and other New Zealanders aged 35-64 years for 1990-1992 and calculated coronary artery revascularisation rates for people aged 40 years and over for the period 1990-1999. Comparing intervention rates in the latter part of the 1990s with mortality data from the early part of the decade in different age groups is subject to error associated with different rates of decline in mortality across ethnic groups.

Ethnicity for both numerator and denominator data was self-defined. Patients who nominated more than one ethnic category including Maori, were included in the Maori group for analysis. Patients who self-identified themselves as both Pacific and other non-Maori ethnicity were included in the Pacific peoples group. Rates were calculated for each ethnic group by summing the number of people with each procedure on an annual basis in the ten-year period and dividing by the population of adults 40 years and over for each ethnic group during the same period. As ethnic differences in event rates may result from differences in the age distribution of the population, rates were standardised using Segi's World population. To examine the impact of priority scores on coronary artery revascularisation rates, we compared CABG and PTCA growth rates between 1990 and 1995 with those for 1995 and 1999 for each ethnic group. Because these rates are derived from the entire population and not a sample, we did not perform statistical tests of comparison.

Results

During the decade 1990-1999, 10 413 CABG procedures were performed on men and 3500 in women in New Zealand. Of these, 155 were performed on Pacific men, 56 in Pacific women, 321 in Maori men and 166 in Maori women. During the same period, 9419 PTCA procedures were performed on men and 4248 in women. Of these, 70 were in Pacific men, 23 in Pacific women, 232 in Maori men and 120 in Maori women.

CABG intervention rates per 100 000 population were 32.9 in Pacific men, 20.5 in Maori men and 51.2 in other New Zealand men. CABG intervention rates were 10.6 in Pacific women, 10.7 in Maori women and 14.5 in other New Zealand women. PTCA intervention rates were 12.1 in Pacific men, 13.9 in Maori men and 48.1 in other New Zealand men. PTCA intervention rates were 3.4 in Pacific women, 7.2 in Maori women and 16.6 in other New Zealand women.

Figure 1 shows age-standardised coronary artery disease mortality rates between 1990 and 1992, CABG and PTCA intervention rates between 1990 and 1999 for New Zealand men by ethnic group and Figure 2 shows the same information for women. Mortality rates were lowest in other New Zealand men and women and highest in Maori men and women. Mortality rates, CABG and PTCA intervention rates were lower in women than in men across all ethnic groups. CABG rates were lower in Pacific and Maori men compared with other New Zealand men (rate ratios were 0.64 and 0.40 respectively) and in Pacific and Maori women compared with other women (rate ratios 0.73 and 0.74 respectively). PTCA rate ratios were 0.25 in Pacific and 0.29 in Maori men compared with other men and 0.21 in Pacific and 0.43 in Maori women compared with other New Zealand women. Ethnic differences in PTCA intervention rates were more marked than ethnic differences in CABG intervention rates.

Use of coronary artery revascularisation procedures increased in all ethnic groups but CABG and PTCA event rates were lower in Pacific and Maori peoples throughout the study period. Ethnic differences in PTCA intervention rates narrowed slightly towards the latter half of the study period but differences in CABG event rates remained constant throughout.

Table 1 shows CABG and PTCA annual rate of growth in intervention rates by ethnic group per 100 000 population before and after the introduction of priority scores. The rate of change in CABG interventions decreased in Pacific peoples but increased among Maori and other New Zealanders while the rate of change for PTCA interventions increased in Pacific and other New Zealanders but decreased slightly among Maori. Introducing priority scores was neither associated with a decrease in the overall use of cardiac procedures nor a significant reduction in the differential use of the procedures across all ethnic groups.

![Figure 1. Age standardised mortality (1990-1992), CABG and PTCA intervention rates (1990-1999) per 100 000 population by ethnicity group.](image-url)
Discussion

Significant ethnic and gender differences in coronary artery revascularisation intervention rates were apparent in New Zealand throughout the 1990s. Intervention rates were lowest in Pacific and Maori peoples despite higher coronary artery disease mortality and morbidity rates. Given the lower overall coronary artery revascularisation intervention rates in New Zealand compared with other OECD nations, our findings suggest that substantial numbers of Pacific and Maori men and women from all ethnic groups may not have received necessary cardiac care. The findings may also partly explain the slower rate of decline in coronary artery mortality rates in New Zealand compared with other OECD nations.

Our findings are similar to those of a study in the northern region of the North Island where coronary artery revascularisation rates were lower among Pacific and Maori peoples compared with other New Zealanders. Ethnic and gender differences in coronary artery revascularisation rates in New Zealand were also consistent with findings reported from other countries. In the USA, many studies have shown that women and minority groups are less likely to undergo invasive cardiovascular procedures for coronary artery disease even after adjusting for co-morbidities and contraindications to procedures. Secondary prevention strategies with proven benefits, including revascularisation, were significantly underused in women, especially in minority groups. Gender differences were partly attributed to women having a higher incidence of complications and risk of operative death after CABG than men. Studies suggest that underuse of coronary artery revascularisation procedures is common, and between 22-41% of patients where expert panels deemed the intervention appropriate and necessary did not undergo the procedure. Ethnic differences were due mainly to underuse of these procedures in minority groups. As profound as the racial and ethnic differences are in the US, they are less than those found in this New Zealand study.

There is insufficient information in the NMDS to explain ethnic and gender differences in invasive cardiology procedures in New Zealand. Variations in coronary artery revascularisation procedures may be due to ethnic and gender differences in coronary artery disease prevalence and severity; patients’ health seeking behaviour; preferences for cardiac procedures or medical treatment; physician practice styles, and patient-doctor interaction. Higher prevalence of diabetes and early onset of cardiac disease among Maori and Pacific peoples may influence the choice of treatment with respect to medical, angioplasty or coronary artery bypass grafting. Higher morbidity and mortality rates among New Zealand’s ethnic minority populations suggests that ethnic differences in disease prevalence are unlikely explanations for lower coronary artery revascularisation intervention rates in these groups. On the other hand, Maori and Pacific peoples are low users of primary care and preventive services. Since elective CABG and PTCA interventions are dependent on referrals from primary care, lower rates of revascularisation procedures may be a result of poor access or use of primary care services by New Zealand’s ethnic minority groups. Studies elsewhere have shown that women and ethnic minority groups with coronary artery disease are less likely to be referred for assessment and treatment. It was also unclear whether Maori and Pacific peoples referred for cardiology assessment received timely diagnostic and treatment procedures. Co-morbidities, contraindications to procedures and patient refusal to accept invasive medical treatment are other possible reasons for the observed differences. Discrimination against ethnic minorities has been suggested as a possible reason for ethnic disparities in the use of these procedures in the USA. Discrimination might exist in New Zealand as well. Referral patterns, patient characteristics and in-hospital processes-of-care issues are useful areas for further research to determine if lower CABG and PTCA rates in Pacific and Maori peoples are due to factors operating outside, within the hospital, or both. Studies are also needed to determine if ethnic disparities in the use of coronary artery revascularisation procedures in New Zealand are due to underuse in Pacific and Maori peoples, overuse in other New Zealanders, or both.

After priority scores were introduced, CABG intervention rates increased at a faster rate in Maori and other New Zealanders but decreased in Pacific peoples. PTCA intervention rates increased at a faster rate in Pacific and other New Zealanders but decreased slightly among Maori. Increasing use of PTCA relative to CABG procedures was a worldwide phenomenon and unlikely to be related to the introduction of priority scores in New Zealand. While coronary artery revascularisation rates have improved in New Zealand over time, availability of these procedures remains limited for all New Zealanders. Strategies beyond the use of priority scores are needed to address ethnic and gender disparities in the use of coronary artery revascularisation procedures in New Zealand. Consideration should be given to improving cardiac surgical capacity for the whole country as well as supporting effective heart disease prevention programmes targeted at Pacific and Maori peoples.

Underuse of appropriate coronary artery revascularisation procedures is now regarded as a failure to provide recommended standards of care and problems with the overall quality of care within the health system. Furthermore, as quality of care initiatives gain momentum,
cardiovascular intervention rates may be suitable indicators for monitoring equity in the use of health care services in the country. Ethnic disparities in the use of these procedures may provide useful information on the progress being made on the government's programme to reduce health disparities between various groups in New Zealand.

Our study findings should be interpreted with care because of its observational nature and lack of detailed information about patient characteristics and health system factors that may help explain the disparities identified. Under-reporting and misclassification of Maori and Pacific peoples is a recurring problem which may affect the validity of these figures. However, a liberal definition of Pacific and Maori peoples would inflate population numbers and lower intervention rates even further. Despite these limitations, our study findings showed significant ethnic and gender differences in the use of coronary artery revascularisation procedures in New Zealand. Further research should be conducted to determine the reasons for the differences in the use of coronary artery revascularisation procedures.

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Vitamin D is a steroid pro-hormone of importance for the maintenance of bone strength due to its actions in the gut and in bone and through its inhibitory effects on parathyroid hormone secretion. In countries where food is not fortified, including Australia, it is difficult to obtain sufficient vitamin D in the diet particularly for older people in whom fat soluble vitamin D absorption is reduced. The skin, via exposure to ultraviolet irradiation is the main source of the precursors for vitamin D which is sequentially hydroxylated in the liver to 25-hydroxyvitamin D (25 OHD) and kidney to 1,25 dihydroxyvitamin D (1,25 OHD). Older people are more likely to have relative vitamin D deficiency due to inadequate exposure to sunlight, decreased production of vitamin D within the dermis, decreased renal capacity for production of 1,25 OHD and a decrease in intestinal responsiveness to 1,25 OHD occurring with age, which may potentiate the reduced availability of 1,25 OHD.

There is a high prevalence of vitamin D deficiency in frail and institutionalised older individuals both overseas and in Australia. Younger groups and active older groups have not been found to be vitamin D deficient. A seasonal fluctuation to serum vitamin D indices, with the nadir in late winter and early spring, has been described. A seasonal variation in the incidence of hip fractures has also been reported that cannot be explained by falls on wet icy pavements as the majority of falls occur inside the home.

There are two pre-requisites for most hip fractures; low bone fragility and a fall. Type II muscle fibre atrophy and pain in vitamin D deficient states may predispose older individuals to falls. If bone strength fluctuates with serum vitamin D levels a fracture following a fall would be more likely at the end of winter. Seasonal increases in fracture rate are reported to correlate with the degree of histologically proven osteomalacia. The reported seasonal fluctuation in bone mineral density in post-menopausal women has been prevented by vitamin D supplementation. However, the seasonal variation in admissions with a hip fracture. The majority of patients (68%) either lived in institutional care or were dependent on a carer and 43% reported going outdoors less than once a week.

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Results
There were 91 patients, 66 being female. The mean age was 81.3 yrs (SD = 7.9) with a range of 58-101 years (Figure 1). Three patients reported taking vitamin D supplements but none had received anti-epileptic medication, had undergone gastrectomy or had a history of malabsorption. 68% of subjects had vitamin D deficiency, defined as a serum 25 OHD level less than 28 nmol/L. Mean serum 25 OHD for the whole group was 25.9 nmol/L (SD = 13).

Patients were stratified according to their level of dependence into three groups (Table 1). Subjects in Group 1 lived independently at home (Independent). Group 2 required hostel level of care either in a hostel or from a carer (Low Care) and Group 3 were from nursing homes (High Care). Although there was no significant difference in vitamin D (serum 25 OHD) level and body mass index between the three groups, there was a trend to lower vitamin D levels in subjects who were more dependent. More dependent groups were significantly older and had decreased sunlight exposure (Outdoor Score) and were more dependent in walking and activities of daily living. Overall at least 68% of subjects required some assistance with activities of daily living and only 57% reported going outdoors at least once a week. Only 50% were able to walk without aids.

Table 1. Results grouped by level of dependence.

<table>
<thead>
<tr>
<th>Residence</th>
<th>Independent</th>
<th>Low care</th>
<th>High care</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>34</td>
<td>28</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Vit D(nmol/L)</td>
<td>27.5 ±15</td>
<td>26.1 ±10</td>
<td>23.6 ±15</td>
<td>p = 0.5</td>
</tr>
<tr>
<td>Age (Yrs)</td>
<td>77.6</td>
<td>80.3</td>
<td>86.5</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.8</td>
<td>22.3</td>
<td>22.2</td>
<td>p = 0.8</td>
</tr>
<tr>
<td>Outdoor Score = 0</td>
<td>33%</td>
<td>39%</td>
<td>62%</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>Walk Score = 0</td>
<td>29%</td>
<td>57%</td>
<td>68%</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>ADL Score = 0</td>
<td>15%</td>
<td>36%</td>
<td>64%</td>
<td>p &lt; 0.05</td>
</tr>
</tbody>
</table>

The high prevalence of vitamin D deficiency in patients with hip fracture was not expected. Studies from Western Australia found vitamin D deficiency in only 44% of patients admitted with a hip fracture, but, nursing home patients were excluded from that study. A study from Adelaide in patients with fractured hips showed subjects to have a significantly lower vitamin D level.16

Discussion
The absence of seasonal fluctuation in serum vitamin D (25 OHD) levels and fractures in this group of subjects is surprising. It is likely to reflect the fact that patients who sustain a hip fracture are generally more frail than the general population and are less likely to go outside at any time during the year and are therefore less likely to have seasonal variations in sunlight exposure. Our results concur with other studies that have suggested that markers of poor nutrition and functional disability are important associations of vitamin deficiency and that patients who present with hip fracture are more likely to be housebound prior to their fracture.28 Our Outdoor Score is only a crude measure of sunlight exposure and does not include the length of time spent outdoors or the surface area of the skin exposed to direct sunlight that is fundamental to vitamin D metabolism. The lack of seasonal difference may also be due to the “floor effect” as these patients have inadequate sunlight exposure throughout the year.

The high prevalence of vitamin D deficiency in patients with hip fracture was not expected. Studies from Western Australia found vitamin D deficiency in only 44% of patients admitted with a hip fracture, but, nursing home patients were excluded from that study. A study from Adelaide in patients with fractured hips showed subjects to have a significantly lower vitamin D level.
have a mean serum vitamin D level of 39.2 nmol/L and the subgroup of patients from nursing homes had a mean level of 35 nmol/L. The obvious difference between Tasmania and the other States is the climate, which discourages people from going outdoors, and the reduced skin area exposed to direct sunlight. However, frail older individuals, even in sunnier climates are at risk of vitamin D deficiency as it is access to direct sunlight that appears to be most important. A previous study in our Department showed vitamin D deficiency in 17% of older volunteers living in the community but deficiency in 67% of older patients admitted to a Geriatric Rehabilitation ward. The age distribution of the community volunteers makes direct comparison with our community dwelling fracture group difficult. However all patients over 70 years can be compared. There were 38 subjects over 70 years in the “well volunteer group” with a mean age of 78.2 and a mean vitamin D of 44.3 nmol/L. This is significantly different from the studied ‘fracture’ group where the mean age was 77.6 years and the serum vitamin D only 27.5 nmol/L (p = 0.00029). This may indicate that patients with a hip fracture come from a group of community residents who are already housebound and do not get enough sun exposure. Furthermore, vitamin D deficiency appears to be a marker for frailty resulting in hospitalisation with fractures or other medical problems.

From our data we are not able to show any causal role between vitamin D fluctuations and the incidence of hip fracture. However, given the possible role of vitamin D in maintaining muscle strength and its known essential role in bone metabolism, and the high incidence of vitamin D deficiency in frail older individuals in Southern Tasmania is a cause for concern. Our findings suggest that all frail individuals with hip fracture, those admitted with medical problems and those living in institutional care or dependent in their mobility or activities of daily living, should be screened for vitamin D deficiency and offered replacement treatment. Studies show targeting supplementation with cholecalciferol 800 IU per day with adequate calcium contributes vitamin D deficiency and the biochemical markers of increased bone turnover, increases bone mineral density, and reduces fractures. A randomised controlled trial in Southern Tasmania should be considered as vitamin D deficiency does have far reaching musculoskeletal consequences with significant morbidity, mortality and public cost – all of which are likely to increase in line with our ageing population.

Acknowledgements. We thank the staff of Royal Hobart Hospital, the Biochemistry Department and the Endocrinology Department, and Miss CA Edwards for typing this manuscript.

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Mortality and cancer incidence in New Zealand pulp and paper mill workers

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Abstract

Aims. To evaluate mortality and cancer incidence in a cohort of workers employed in the New Zealand pulp and paper industry, and to identify the exposures responsible for any increased risk.

Methods. A total of 8456 workers employed for at least one year in three pulp and paper mills between 1978 and 1990 were followed up until 1992. The observed number of deaths and cancer cases was compared with expected numbers calculated using five-year age-specific rates for the New Zealand population.

Results. Vital status was determined for 81% of the cohort, and for 93% of the total person-years at risk. Mortality from all causes (standardised mortality ratios (SMR) = 0.80, 95% confidence intervals [CI] 0.71-0.89; 314 deaths), and from all malignant neoplasms (SMR = 0.95, 95% CI 0.78-1.15, 103 deaths), was lower than expected. Mortality from lung cancer (SMR = 1.33, 95% CI 0.94-1.83, 37 deaths) was marginally increased.

Conclusions. No overall increase in mortality from cancer or other causes was observed in this cohort. A small increase in lung cancer risk is suggested, although this was not statistically significant. Numbers of cases were too small for detailed analyses of associations between disease and specific exposures.
women) met the criteria for inclusion in the study and of these 6465 (77%) were still alive at the end of the study period, 314 (4%) had died, 115 (1%) had emigrated, and 1562 (18%) had incomplete follow-up. Cohort members had been employed in the industry on average for 12.1 years, and the average length of follow-up was 10.4 years. A total of 87549 person-years of observation were accumulated, ie, 93% of the total possible number of person-years if all subjects had been followed through until 31/12/1992.

**Table 1. Characteristics of the New Zealand pulp and paper mill workers cohort.**

<table>
<thead>
<tr>
<th>Year of birth</th>
<th>Male (n=1911-1973)</th>
<th>Female (n=1917-1973)</th>
<th>Total (n=1911-1973)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>1948</td>
<td>1953</td>
<td>1949</td>
</tr>
<tr>
<td>Status at end of follow-up:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alive</td>
<td>5726 (77%)</td>
<td>719 (75%)</td>
<td>6465 (76%)</td>
</tr>
<tr>
<td>Dead</td>
<td>1014 (4%)</td>
<td>13 (1%)</td>
<td>1144 (4%)</td>
</tr>
<tr>
<td>Incomplete follow-up</td>
<td>1143 (18%)</td>
<td>219 (22%)</td>
<td>1362 (18%)</td>
</tr>
<tr>
<td>Emigrated</td>
<td>105 (1%)</td>
<td>1 (1%)</td>
<td>106 (1%)</td>
</tr>
<tr>
<td>Person-years of observation:</td>
<td>78 614</td>
<td>8915</td>
<td>87 499</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Year</th>
<th>Range</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-9</td>
<td></td>
<td>5782 (17 784)</td>
<td>911 (6042)</td>
<td>6693 (41 827)</td>
</tr>
<tr>
<td>10-14</td>
<td></td>
<td>6655 (16 416)</td>
<td>498 (1658)</td>
<td>5153 (18 094)</td>
</tr>
<tr>
<td>15-19</td>
<td></td>
<td>1042 (10 263)</td>
<td>234 (697)</td>
<td>1276 (10 960)</td>
</tr>
<tr>
<td>20-24</td>
<td></td>
<td>1997 (6768)</td>
<td>94 (311)</td>
<td>2091 (7099)</td>
</tr>
<tr>
<td>25+</td>
<td></td>
<td>1264 (7383)</td>
<td>48 (186)</td>
<td>1312 (7569)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Duration of employment:</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-4</td>
<td>4661 (25 109)</td>
<td>821 (4741)</td>
<td>5482 (30 050)</td>
</tr>
<tr>
<td>5-9</td>
<td>3876 (18 234)</td>
<td>461 (2128)</td>
<td>4337 (20 362)</td>
</tr>
<tr>
<td>10-14</td>
<td>11277 (13 615)</td>
<td>260 (1355)</td>
<td>1387 (14 750)</td>
</tr>
<tr>
<td>15-19</td>
<td>2179 (8602)</td>
<td>118 (315)</td>
<td>2297 (9946)</td>
</tr>
<tr>
<td>20-24</td>
<td>1546 (6463)</td>
<td>56 (290)</td>
<td>1602 (6753)</td>
</tr>
<tr>
<td>25+</td>
<td>897 (4591)</td>
<td>21 (84)</td>
<td>920 (4675)</td>
</tr>
<tr>
<td>&gt;10</td>
<td>321 (1520)</td>
<td>5 (23)</td>
<td>326 (1542)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Range</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-4</td>
<td>1-44</td>
<td>1-38</td>
<td>1-44</td>
</tr>
<tr>
<td>Mean</td>
<td>12.7</td>
<td>7.6</td>
<td>12.1</td>
</tr>
<tr>
<td>Average length of follow-up:</td>
<td>10.5</td>
<td>9.1</td>
<td>10.4</td>
</tr>
</tbody>
</table>

*Number of subjects and person-years.

The findings of the mortality analyses are shown in Table 2. Overall mortality was decreased, with a total of 314 deaths observed versus 394.3 expected yielding an SMR of 0.80 (95% CI, 0.71-0.89), with deficits for deaths from diseases of the circulatory system (SMR=0.78, 95% CI 0.64-0.93) and from external causes (SMR=0.55, 95% CI 0.39-0.75), in particular traffic accidents (SMR=0.55, 95% CI 0.39-0.75), although excess risk was observed in females (SMR=1.83, 95% CI 0.94-1.83, 37 cases). Other significant excess deaths due to specific cancers, although an elevation in lung cancer mortality is suggested (SMR = 1.33, 95% CI 0.94-1.83, 37 cases). Other cancers for which a non-significant excess mortality was observed include oesophagus (SMR = 1.88, 95% CI 0.69-4.09, 6 cases), liver (SMR = 2.40, 95% CI 0.78-5.60, 5 cases), pleura (SMR = 5.44, 95% CI 0.66-19.7, 2 cases), breast (SMR = 1.94, 95% CI 0.53-4.97, 4 cases), testis (SMR = 3.55, 95% CI 0.73-10.4, 3 cases) and bladder (SMR = 2.14, 95% CI 0.58-5.49, 4 cases).

Analysis of cancer incidence showed a similar reduction in overall risk (SIR=0.86, 95% CI 0.74-1.00, 175 cases) with only lung cancer showing an elevation approaching statistical significance (SIR = 1.35, 95% CI 0.97-1.84, 41 cases). Non-statistically significant excesses were observed in men for cancer of the liver (SIR = 2.70, 5 cases) and pancreas (SIR = 1.69, 7 cases), and among women for cancer of the breast (SIR = 1.64, 10 cases) and cervix uteri (SIR = 2.14, 3 cases).

The results of analyses for risk by time since first exposure and duration of employment are shown in Table 3. The SMR for overall mortality increased significantly with increasing time since first employment, but not with increasing duration of employment. No other significant trends were evident.

Results for mortality from all causes, and for incidence of all malignant neoplasms and lung cancer, are shown in Table 4 for workers ever exposed to selected specific agents and in Table 5 by department of employment. Statistically significant excesses for lung cancer were observed among those categorised as ever exposed to pulp and paper dust (SIR = 1.45) and talc (SIR = 1.51, 95% CI 1.02-2.15, 30 cases) for those workers who had “only worked” in the non-production department, with a suggestion of a trend with increasing duration of employment, and for those who had “ever worked” in the non-production department (SIR = 1.44, 95% CI 1.01-1.99, 36 cases). This department includes workers responsible for operation and maintenance of plant, but also includes administration and other white-collar workers. When specific exposures were evaluated, elevated lung cancer risks appeared to be associated with ever having been exposed to talc (SIR = 3.15, 95% CI 1.27-6.50, 7 cases), pulp and paper dust (SIR = 1.45, 95% CI 1.00-2.04, 33 cases), fungal spores and bacteria (SIR = 1.39, 95% CI 0.95-1.96, 32 cases) and combustion products (SIR = 1.38, 95% CI 0.95-1.93, 33 cases).

While a finding of an increase in lung cancer risk in this industry is in agreement with previous reports for maintenance workers, workers in Kraft pulping mills, or those exposed to inorganic dusts and wood dust, other studies have found elevated risks only for those working in sulphite mills or have found no increase in lung cancer at all. The lack of information on smoking makes it impossible to rule this out as the cause of any excess in this cohort, although confounding by smoking in occupational cancer studies is often overestimated. It would normally account for no more than about a 20-25% increase in comparisons with the general population, whereas the increase seen here exceeds this. In addition, while mortality from some smoking-related cancers (oesophagus, pancreas and bladder) is marginally elevated, it is lower than expected from non-malignant respiratory disease and diseases of the circulatory system. It is possible, therefore, that part of the observed increase in lung cancer could be attributed to occupational factors.

Asbestos was used extensively in the past throughout this type of industrial plant, although all three New Zealand mills reported that asbestos was never used as an ingredient in stock...
incidence among those ever employed in the non-production dept (SIR = 1.44, 95% CI 1.01-1.99, 36 cases). The excess mortality from pleural cancer (SMR = 5.44, 95% CI 0.66-19.7), although based on only two cases, is consistent with previous findings in this industry and is likely to reflect past asbestos exposure.

preparation and that external contractors did virtually all installation or removal of asbestos material. The most likely exposure within this cohort, therefore, would be to maintenance workers who would have incidental exposures in the plant during work that disturbed asbestos insulation or friction materials, and there was evidence of excess lung cancer

### Table 2. Mortality by cause in the New Zealand pulp and paper mill workers cohort compared with national rates.

<table>
<thead>
<tr>
<th>Causes of death (ICD 9th revision)</th>
<th>Observed</th>
<th>Expected</th>
<th>SM R*</th>
<th>95% Confidence intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALL CAUSES</td>
<td>314</td>
<td>394.28</td>
<td>0.80</td>
<td>[0.71-0.89]</td>
</tr>
<tr>
<td>ALL MALIGNANT NEOPLASMS (140-208)</td>
<td>103</td>
<td>108.37</td>
<td>0.95</td>
<td>[0.78-1.15]</td>
</tr>
<tr>
<td>Oral cavity &amp; pharynx (140-149)</td>
<td>2</td>
<td>2.85</td>
<td>0.70</td>
<td>[0.08-2.53]</td>
</tr>
<tr>
<td>Oesophagus (150)</td>
<td>6</td>
<td>3.19</td>
<td>1.88</td>
<td>[0.69-4.09]</td>
</tr>
<tr>
<td>Stomach (151)</td>
<td>4</td>
<td>5.72</td>
<td>0.70</td>
<td>[0.19-1.79]</td>
</tr>
<tr>
<td>Colon (153)</td>
<td>7</td>
<td>10.31</td>
<td>0.68</td>
<td>[0.27-1.40]</td>
</tr>
<tr>
<td>Rectum (154)</td>
<td>4</td>
<td>7.03</td>
<td>0.57</td>
<td>[0.16-1.46]</td>
</tr>
<tr>
<td>Liver (155)</td>
<td>5</td>
<td>2.08</td>
<td>2.40</td>
<td>[0.78-5.60]</td>
</tr>
<tr>
<td>Gallbladder (156)</td>
<td>1</td>
<td>0.66</td>
<td>1.51</td>
<td>[0.04-8.42]</td>
</tr>
<tr>
<td>Pancreas (157)</td>
<td>6</td>
<td>4.34</td>
<td>1.38</td>
<td>[0.51-3.01]</td>
</tr>
<tr>
<td>Lung (162)</td>
<td>37</td>
<td>27.86</td>
<td>1.33</td>
<td>[0.94-1.83]</td>
</tr>
<tr>
<td>Pleura (163)</td>
<td>2</td>
<td>0.37</td>
<td>5.44</td>
<td>[0.66-19.7]</td>
</tr>
<tr>
<td>Brain (171-175)</td>
<td>4</td>
<td>2.06</td>
<td>1.94</td>
<td>[0.53-5.97]</td>
</tr>
<tr>
<td>Cervix uteri (180)</td>
<td>1</td>
<td>0.45</td>
<td>2.21</td>
<td>[0.06-12.3]</td>
</tr>
<tr>
<td>Corpus uteri &amp; uterus unspecified (179, 181-182)</td>
<td>1</td>
<td>0.11</td>
<td>7.67</td>
<td>[0.19-42.7]</td>
</tr>
<tr>
<td>Ovary (183)</td>
<td>1</td>
<td>0.45</td>
<td>2.22</td>
<td>[0.06-12.4]</td>
</tr>
<tr>
<td>Testis (186)</td>
<td>3</td>
<td>0.83</td>
<td>3.55</td>
<td>[0.73-10.4]</td>
</tr>
<tr>
<td>Bladder (188)</td>
<td>4</td>
<td>1.87</td>
<td>2.14</td>
<td>[0.58-5.49]</td>
</tr>
<tr>
<td>Brain (191-192)</td>
<td>3</td>
<td>3.07</td>
<td>0.59</td>
<td>[0.12-1.73]</td>
</tr>
<tr>
<td>Thyroid (191)</td>
<td>1</td>
<td>0.19</td>
<td>5.39</td>
<td>[0.13-30.0]</td>
</tr>
<tr>
<td>Lymphatic and haematopoietic tissue (200-208)</td>
<td>5</td>
<td>9.65</td>
<td>0.52</td>
<td>[0.17-12.1]</td>
</tr>
<tr>
<td>BENIGN NEOPLASMS (S210-239)</td>
<td>1</td>
<td>0.71</td>
<td>1.34</td>
<td>[0.03-7.47]</td>
</tr>
<tr>
<td>DIS. OF ENDOCRINE SYSTEM &amp; BLOOD (240-289)</td>
<td>6</td>
<td>10.87</td>
<td>0.55</td>
<td>[0.20-1.20]</td>
</tr>
<tr>
<td>MENTAL DISORDERS (290-319)</td>
<td>3</td>
<td>2.54</td>
<td>1.18</td>
<td>[0.24-3.46]</td>
</tr>
<tr>
<td>DIS. OF NERVOUS SYSTEM (320-389)</td>
<td>2</td>
<td>6.01</td>
<td>0.33</td>
<td>[0.04-1.19]</td>
</tr>
<tr>
<td>DIS. OF CIRCULATORY SYSTEM (390-459)</td>
<td>119</td>
<td>153.42</td>
<td>0.78</td>
<td>[0.64-0.93]</td>
</tr>
<tr>
<td>DIS. OF RESPIRATORY SYSTEM (460-519)</td>
<td>22</td>
<td>23.08</td>
<td>0.95</td>
<td>[0.60-1.44]</td>
</tr>
<tr>
<td>DIS. OF DIGESTIVE SYSTEM (520-579)</td>
<td>10</td>
<td>9.19</td>
<td>1.07</td>
<td>[0.51-1.96]</td>
</tr>
<tr>
<td>DIS. OF GENITO-URINARY SYSTEM (580-629)</td>
<td>2</td>
<td>2.77</td>
<td>0.72</td>
<td>[0.09-2.61]</td>
</tr>
<tr>
<td>SYMPTOMS &amp; ILL-DEFINED CONDITIONS (780-799)</td>
<td>1</td>
<td>0.53</td>
<td>1.89</td>
<td>[0.05-10.5]</td>
</tr>
<tr>
<td>EXTERNAL CAUSES (E800-999)</td>
<td>39</td>
<td>70.85</td>
<td>0.55</td>
<td>[0.19-0.75]</td>
</tr>
<tr>
<td>Traffic accident (E810-819)</td>
<td>15</td>
<td>27.12</td>
<td>0.55</td>
<td>[0.31-0.91]</td>
</tr>
<tr>
<td>Suicide (E950-959)</td>
<td>9</td>
<td>19.81</td>
<td>0.45</td>
<td>[0.21-0.86]</td>
</tr>
<tr>
<td>Homicide (E960-969)</td>
<td>0</td>
<td>2.58</td>
<td>0.00</td>
<td>[0.00-1.43]</td>
</tr>
<tr>
<td>Other external causes (E800-807, E820-949, E970-999)</td>
<td>15</td>
<td>21.14</td>
<td>0.70</td>
<td>[0.19-11.6]</td>
</tr>
<tr>
<td>UNKNOWN CAUSES</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OTHER CAUSES</td>
<td>4</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Standardised Mortality Ratio.

### Table 3. Cause of death by years since first employment and duration of employment in the New Zealand pulp and paper mill workers cohort.

<table>
<thead>
<tr>
<th>Years since first employment</th>
<th>O/E*</th>
<th>SM R†</th>
<th>95% C I†</th>
<th>All cancer (140-208)§</th>
<th>O/E</th>
<th>SM R</th>
<th>95% C I</th>
<th>Lung cancer (162)</th>
<th>O/E</th>
<th>SM R</th>
<th>95% C I</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-9</td>
<td>51/97.11</td>
<td>0.54</td>
<td>0.40-0.71</td>
<td>16/19.80</td>
<td>0.81</td>
<td>0.46-1.31</td>
<td>2/1.57</td>
<td>0.56</td>
<td>0.07-2.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-14</td>
<td>46/61.64</td>
<td>0.75</td>
<td>0.55-1.00</td>
<td>15/16.50</td>
<td>0.91</td>
<td>0.51-1.50</td>
<td>9/5.75</td>
<td>2.40</td>
<td>1.10-4.55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15-19</td>
<td>65/60.09</td>
<td>1.08</td>
<td>0.83-1.38</td>
<td>24/17.54</td>
<td>1.37</td>
<td>0.88-2.04</td>
<td>5/4.58</td>
<td>1.09</td>
<td>0.35-2.55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-24</td>
<td>35/58.46</td>
<td>0.94</td>
<td>0.71-1.01</td>
<td>19/17.69</td>
<td>1.07</td>
<td>0.65-1.68</td>
<td>9/4.97</td>
<td>1.81</td>
<td>0.83-3.44</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25+</td>
<td>9/117.68</td>
<td>0.83</td>
<td>0.67-1.01</td>
<td>29/36.85</td>
<td>0.79</td>
<td>0.53-1.13</td>
<td>12/10.99</td>
<td>1.09</td>
<td>0.56-1.91</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Observed/Expected. †Standardised Mortality Ratio. §95% Confidence Interval. §ICD 9th Revision.

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Excess liver cancer has been reported twice previously in this industry, namely a nonstatistically significant elevation (SIR = 2.77, 95% CI 0.85-1.95, 18 cases) found in a British Columbian industry, namely a nonstatistically significant elevation (SIR = 2.86, 95% CI 1.18-5.97, 4 cases), and incidence (SIR = 1.64, 95% CI 0.79-3.02, 10 cases) was observed. A similar finding (SMR = 2.86, 95% CI 0.77-7.32, 4 deaths) has been reported in a Spanish pulp and paper worker cohort, while a US case-control study also found elevated incidence (but not mortality) in paper mill workers. All ten incident cases in our study were in the group categorised as having "ever worked" in the non-production department, and seven were categorised as having been exposed to reduced sulphur compounds (SIR = 2.92, 95% CI 1.17-6.02), sulphur dioxide (SIR = 2.86, 95% CI 1.15-5.90), pulp and paper dust (SIR = 2.44, 95% CI 0.98-5.03) and dyes (SIR = 2.66, 95% CI 1.07-7.67).

In summary we found no overall increased risk of mortality from all causes and all cancer in this group of workers, although a marginal increase in lung cancer incidence among those who had worked in the non-production department may be, in part, attributable to occupational factors. Much of this increase could be explained by smoking and asbestos exposure, but the effect of other exposures including t alc and pulp and paper dust cannot be excluded.

Acknowledgements. This study was funded by the Cancer Society of New Zealand, the Accident Compensation Corporation of New Zealand, and the Health Research Council of New Zealand. The Centre for Public Health Research is supported by the Health Research Council of New Zealand. This study was conducted as part of an International Agency for Research on Cancer (IARC) international collaborative study and we thank the other collaborators, and relevant IARC staff, for their assistance with this investigation.

### Table 5. Mortality and cancer incidence in the New Zealand pulp and paper mill workers cohort by department of employment.

<table>
<thead>
<tr>
<th>Department category ever employed in:</th>
<th>All causes</th>
<th></th>
<th>Lung cancer (162)</th>
<th></th>
<th>All cancer (140-208)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>O/E*</td>
<td>SMR†</td>
<td>95% CI‡</td>
<td>O/E*</td>
<td>SIR§</td>
<td>95% CI</td>
<td>O/E*</td>
<td>SIR§</td>
</tr>
<tr>
<td>Pulp production department</td>
<td>56/82</td>
<td>0.68</td>
<td>0.52-1.15</td>
<td>31/41</td>
<td>0.75</td>
<td>0.51-1.07</td>
<td>5/76</td>
<td>0.52</td>
</tr>
<tr>
<td>Recycled paper/paperboard production department</td>
<td>2/33</td>
<td>0.43</td>
<td>0.01-2.39</td>
<td>2/10</td>
<td>2.00</td>
<td>0.24-7.22</td>
<td>1/0</td>
<td>10.10</td>
</tr>
<tr>
<td>Paper/paperboard production department</td>
<td>27/51</td>
<td>0.52</td>
<td>0.34-1.40</td>
<td>21/25</td>
<td>0.81</td>
<td>0.50-1.24</td>
<td>6/37</td>
<td>1.78</td>
</tr>
<tr>
<td>Paper/paperboard product manufacture</td>
<td>7/24</td>
<td>2.91</td>
<td>1.17-5.99</td>
<td>2/12</td>
<td>1.62</td>
<td>0.20-5.86</td>
<td>2/0</td>
<td>9.73</td>
</tr>
<tr>
<td>Non-production department</td>
<td>27/83</td>
<td>0.89</td>
<td>0.78-1.26</td>
<td>14/16</td>
<td>0.87</td>
<td>0.73-1.02</td>
<td>6/24</td>
<td>1.46</td>
</tr>
<tr>
<td>Other/unknown department</td>
<td>12/33</td>
<td>0.36</td>
<td>0.19-0.63</td>
<td>11/16</td>
<td>0.77</td>
<td>0.41-1.31</td>
<td>2/2</td>
<td>0.92</td>
</tr>
</tbody>
</table>

*Observed/Expected. †Standardised Mortality Ratio. ‡95% confidence interval. §Standardised Incidence Ratio.
study and in particular the work on exposure assessment and data management. We also thank Regina Winkelmann for assistance with data management. This work was initiated when Neil Pearce was based at the Department of Public Health, Wellington School of Medicine.

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Inborn errors of metabolism are a group of individually rare yet collectively not uncommon diseases caused by single gene disorders. This molecular anomaly leads to a defect in an enzyme or less commonly a structural protein such as a transmembrane transporter. Decreased enzyme activity results in an accumulation of the biochemical substrate or a deficiency of a product. This can be particularly dangerous if the former happens to be toxic or the latter is essential for cellular function. These diseases usually affect children although increasingly adult phenotypes are being described. Due to the rarity of the individual conditions, the multitude of possible presentations and the perceived complexity of the investigations required, metabolic diseases are likely to be frequently undiagnosed. This is unfortunate as once suspected they are, in general, relatively easy to diagnose and subsequent treatment is often inexpensive yet effective. The lack of a specific metabolic service in New Zealand until recently has probably hindered the diagnosis and treatment of these conditions.

Most metabolic diseases can be classified into four main groups; small molecule diseases leading to a) a ‘sudden’, rapid accumulation of a toxic substance or b) a lack of ‘energy’, c) a slow accumulation of a large complex molecule or d) mitochondrial diseases (Table 1).

The ‘rapid accumulating’ small molecule diseases are best represented by the organic acidaemias and urea cycle defects. The former are the result of defects in the oxidation of protein-derived amino acids leading to an accumulation of organic acids. This occurs particularly during periods of increased protein turnover either from exogenous dietary sources or endogenous catabolism during times of intercurrent illness. Similar episodes occur in the urea cycle disorders, a group of conditions caused by enzymological defects in the production of urea and characterised by periods of severe hyperammonaemia. Both groups of conditions frequently present in the early neonatal period with lethargy, poor feeding, vomiting and eventually encephalopathy. These symptoms are often mistaken for sepsis although the finding of massive hepatomegaly and frequently severe acidosis should alert the clinician to the possibility of a metabolic disorder. Direct measurement of organic and amino acids will lead to a rapid and precise diagnosis. Acute treatment often involves haemofiltration with long-term therapy reliant on an individualised low protein diet and specific medications.

The glycogen storage diseases (GSDs) and fatty acid oxidation defects (FAODs) are examples of small molecule diseases leading to a lack of ‘energy’. In order to maintain blood glucose normal children, when fasted, rely initially on the breakdown of hepatic glycogen and later on the oxidation of fat. Children with GSDs can make glycogen but cannot effectively catabolise it. Glycogen is thus stored in huge quantities in the liver. During periods of starvation, such as that which occurs during an intercurrent viral illness, the children become hypoglycaemic and lethargic. Presentation in the first few months of life with hypoglycaemia and massive hepatomegaly is typical. Defects of fatty acid oxidation are more variable in their presentation. Because some children may not be exposed to significant catabolic stress during early life some patients with FAODs may not present until mid childhood or even adulthood. Presentation with hypoglycaemia and encephalopathy is typical although as skeletal and cardiac muscle is particularly reliant on fatty acids for cellular metabolism, initial symptoms may relate to rhabdomyolysis, cardiomyopathy and arrhythmias. Diagnosis of these conditions relies on a strong clinical suspicion and specialised tests such as enzymology in the GSDs and the measurement of various fatty acid derivatives (acylcarnitine profile) in the FAODs. The latter can be done quickly and cheaply on blood ‘spotted’ onto blotting paper (Guthrie card) making neonatal screening an exciting possibility. Treatment of these conditions is relatively easy, cheap and effective. Regular oral feeds are advised. The regular intake of a carbohydrate solution, either orally or intravenously, during periods of catabolic stress is essential.

Defects in the metabolism of large complex molecules present in quite a different manner. The best examples of these disorders are the lysosomal storage diseases (LSDs). Lysosomes are cell organelles important in the recycling of sphingolipids, mucopolysaccharides and oligosaccharides. These large complex molecules are made up of fatty acid chains, carbohydrate moieties and amino groups and are important as structural components of cells and organelles. They are catabolised in a stepwise fashion by a series of enzyme

### Table 1. Metabolic disease: general overview.

<table>
<thead>
<tr>
<th>Metabolic Type</th>
<th>Examples</th>
<th>Features</th>
<th>Diagnostic Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small molecule ‘intoxication’</td>
<td>Organic acidaemia, urea cycle disorders</td>
<td>Episodic lethargy, vomiting, ‘unwellness’</td>
<td>Urine amino and organic acids. Plasma ammonia</td>
</tr>
<tr>
<td>Small molecule – ‘lack of energy’</td>
<td>Fatty acid oxidation defects, glycogen storage disease</td>
<td>Sudden collapse, hypoglycaemia, hepatomegaly, cardiac/skeletal muscle dysfunction</td>
<td>Acylcarnitine profile. Enzyme measurement</td>
</tr>
<tr>
<td>Large molecule - slow deterioration</td>
<td>Lysosomal storage disease</td>
<td>Developmental regression, variable hepatomegaly and dysmorphic features</td>
<td>White cell enzymes</td>
</tr>
<tr>
<td>Mitochondrial disease</td>
<td></td>
<td>Neurological plus any other organ involvement!</td>
<td>Plasma and CSF lactate. Muscle biopsy for enzymology</td>
</tr>
</tbody>
</table>
Attacking the pneumococcus – a hundred years’ war

The battle with pneumococcus over the past century is reminiscent of the Hundred Years’ War, the struggle between England and France that was interrupted by truces and stalemates. Streptococcus pneumoniae was isolated by Pasteur in 1881 and was soon recognized as the commonest cause of lobar pneumonia. When Dochez and Gillespie divided S. pneumoniae into 4 types (there are currently 90) on the basis of capsular antigens, the fatality rate associated with untreated pneumococcal pneumonia was 33 percent. By 1936, the use of type-specific antiserum reduced mortality to about 18 percent.

Sulfonamides were introduced in the 1930s for the treatment of pneumococcal pneumonia. One third of the strains isolated before treatment were resistant by the mid-1940s, penicillin supplanted prior therapies because of its efficacy, the uniform susceptibility of pneumococcus to it (minimal inhibitory concentration [MIC], ≤0.02 µg per milliliter), and the rarity of toxicity. Twenty years later, the MIC of penicillin was 0.10 mg per milliliter or higher for 1 percent of S. pneumoniae isolates. A high degree of resistance to penicillin in S. pneumoniae emerged in the late 1970s, and worrisome multidrug-resistant strains then began to appear. In the late 1980s, penicillin resistance in S. pneumoniae reached a prevalence of 44 percent in Spain. Resistance soon became worldwide.

CASE REPORT

Positional upper airways narrowing and an apparent life threatening event

SL Tonkin, Medical Officer, New Zealand Cot Death Association; S Vogel, Radiologist, Starship Children’s Hospital; L Bennet, Senior Lecturer, The Liggins Institute; AJ Gunn, Senior Lecturer, Department of Paediatrics, University of Auckland, Auckland.

At Starship Children’s Hospital, Auckland, 43 infants were assessed over an eighteen month period after being admitted because they had been found apparently not breathing, or cyanosed. These events were considered to have been ALTEs (Apparent Life Threatening Events). Of these 43 infants, sixteen had no history of previous respiratory problems or any other diagnosable illness. They were completely normal to physical examination including head size. Seven infants were being held by caregivers at the time of the ALTE while the other nine were in various restraining devices.

The circumstances of each episode were elicited, and scene reconstruction was carried out with the caregivers to replicate the position and attitude of each baby when found cyanosed and/or not breathing. In every case the infant's head was markedly flexed on the body and it was suspected that the upper airway could have been restricted or obstructed. The importance of preventing excessive head flexion, particularly in sleep, was explained to the caregivers and none of these infants had any further episodes.

Case history

At the age of three weeks this 3.275 kg breast fed boy was carried from his bassinet to his mother by his ten year old sister. She had one hand under his head and the other under his napkin. His mother noticed that he was very “scrunched up” and was alarmed to see that his face was dark blue and that it seemed his breathing had stopped. She grabbed him from his sister and when she “straightened him out” there was rapid return to normal colour and breathing.

When assessed in hospital he was noted to have a relatively large head with a protuberant occiput. Physical examination was normal, and an overnight polygraph (Edentec Mallinckrodt Inc., Missouri, USA) with leads to record heart rate, respiration, nasal airflow, and pulse oximetry was completely normal. Inspiratory radiographs of the upper airway were taken, timed using a modified Graseby (Graseby Dynamics Limited, Herts, UK) MR 10-apnoea monitor with the capsule of the monitor taped to the infant’s lateral abdomen at the level of the umbilicus. In Figure 1a, taken when the infant’s head was in the neutral position, the upper airway was normally patent. The mother then gently flexed the infant’s head towards the position that he had been in during the episode, with his jaw impinging on his chest. It could be seen by eye that the jaw was being displaced by pressure on the infant’s own chest. His mother stated that the infant’s head was actually more flexed than this during the original episode. Marked narrowing of the upper airway was evident on the second radiograph (Figure 1b) which shows the head flexed 25°. The mandible is displaced cephalically and posteriorly to push the tongue and soft palate towards the posterior pharyngeal wall, and the airway space was halved.

Discussion

The danger of airway obstruction from pressure on the jaw in newborns was first noted as early as 1976, and some ALTEs have been related to the position in which infants were being held. We have previously shown that timed lateral neck radiographs provide a highly accurate, reproducible method of assessing upper airway dimensions in early infancy. The present case confirms that flexion of the infant’s head onto it’s own chest can produce severe airway narrowing. It is highly likely that this narrowing was responsible for the alarming cyanosis in this infant.

The underlying factor allowing such airway narrowing is the immaturity of the temporo-maxillary joint in newborns, which allows much more antero-posterior movement than in adults. Also the infant upper and lower jaws can readily be approximated as there are no teeth present to hinder this. The
likelihood of head flexion is greatly increased as the infant falls asleep and the neck muscles relax. It is important that infants are not placed or held in positions which allow over-flexion of the head on the neck.

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**VIEWPOINTS**

**Screening for type 2 diabetes in non-pregnant adults in New Zealand: practice recommendations**

Tim Kenealy, HRC Training Fellow, Department of General Practice and Primary Health Care; Geoff Braatvedt, Senior Lecturer in Medicine, University of Auckland, Robert Scragg, Senior Lecturer, Department of Community Health, University of Auckland, Auckland.

NZ Med J 2002; 115: 194-6

This article is written to help general practitioners (GPs), practice nurses and other primary care health providers in the early detection of diabetes. In preparing this article we used available systematic reviews and consulted widely with colleagues in New Zealand, however, the views remain our own.

**Need for new screening guidelines**

Most patients will have no symptoms from their diabetes, which can therefore be detected only by screening. This means performing a simple test to see if it is worth doing further diagnostic tests. However, the 1995 New Zealand Society for the Study of Diabetes (NZSSD) screening guidelines became outdated when the diagnostic criteria for diabetes changed in 1999. The earlier diagnostic fasting venous plasma glucose criteria of ≥7.8 mmol/L has been reduced to ≥7.0 mmol/L. This has caused an increase of nearly 20% in the number of people classified as having diabetes. Table 1 shows current estimated percentages of people with diabetes by age and ethnic groupings.

**Table 1.** Estimated prevalence of people with diabetes in New Zealand, by age and ethnic groups. Figures are percent diagnosed (additional percent undiagnosed).

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>European</th>
<th>Maori</th>
<th>Pacific</th>
<th>Asian</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-39</td>
<td>0.7 (1.2)</td>
<td>2.2 (5.5)</td>
<td>1.1 (1.8)</td>
<td>1.0 (1.6)</td>
</tr>
<tr>
<td>40-49</td>
<td>1.5 (2.3)</td>
<td>6.7 (10.8)</td>
<td>4.7 (7.5)</td>
<td>4.1 (6.6)</td>
</tr>
<tr>
<td>50-59</td>
<td>3.8 (6.0)</td>
<td>13.2 (21.1)</td>
<td>12.1 (19.3)</td>
<td>8.0 (12.9)</td>
</tr>
<tr>
<td>≥60</td>
<td>5.9 (9.4)</td>
<td>15.4 (24.6)</td>
<td>11.7 (18.7)</td>
<td>12.8 (20.5)</td>
</tr>
</tbody>
</table>

Figures for known prevalence are based on a community survey in South Auckland. Figures for undiagnosed prevalence are based on the known prevalence inflated by 1.6, a factor derived from re-analysis of a workforce survey in Tokoroa in New Zealand.

Furthermore, since 1995 there is new evidence that treating diabetes and its associated metabolic abnormalities prevents micro- and macro-vascular complications, making it even more important to use screening tests that miss few people with undiagnosed diabetes. This inevitably means screening more people, most of whom will not have diabetes. Nevertheless, many of those without diabetes may prove to have lesser degrees of impaired glucose metabolism, including impaired glucose tolerance (IGT). Recent studies have shown that treating IGT with lifestyle changes or drugs reduces the number of people going on to develop frank diabetes. Finally, screening for diabetes is likely to detect other associated and modifiable health risks including obesity, raised lipids, high blood pressure, smoking and sedentary lifestyles.

**Diagnosis of diabetes**

Formal diagnosis of diabetes is made by; either, characteristic symptoms of diabetes plus one diagnostic elevated glucose, or two diagnostic glucose values in the absence of symptoms. Characteristic symptoms of diabetes means one or more of: weight loss, blurred vision, excess tiredness, recurrent infections, excess drinking or excess urine volume - unless the symptoms have another explanation. Diagnostic glucose values, shown in Table 2, are a fasting venous plasma glucose ≥7.0 mmol/L, or ≥11.1 mmol/L on either a random venous plasma glucose or the 2 hour value of the oral glucose tolerance test (OGTT). (All routine New Zealand laboratory glucose tests on adults are done on venous plasma). When the person has no symptoms of diabetes, two diagnostic tests are required, on separate days.

**Table 2.** Venous plasma glucose values for diagnosis of diabetes mellitus and other categories of hyperglycaemia (mmol/L).

<table>
<thead>
<tr>
<th>Diabetes mellitus</th>
<th>Fasting</th>
<th>2 hour post-glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥7.0</td>
<td>≥11.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Impaired glucose tolerance (IGT)</th>
<th>Fasting (if measured)</th>
<th>2 hour post-glucose load</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≤7.0</td>
<td>7.8-11.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Impaired fasting glycaemia (IFG)</th>
<th>Fasting</th>
<th>2 hour post-glucose load (if measured)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6.1-6.9</td>
<td>≤7.8</td>
</tr>
</tbody>
</table>

The fact that two glucose values (fasting ≥7.0 and random OGTT ≥11.1 mmol/L) can be used to diagnose diabetes means that while some people with diabetes will have only a raised fasting glucose, some will have only a raised random or 2 hour value on the OGTT, and some may have both (Figure 1).

Those diagnosed by an elevated 2 hour glucose value may be more at risk of cardiovascular disease than those with only an elevated fasting glucose. Actual numbers in each category vary with the population tested. However, in an
elderly European population, one third of people with diabetes had a fasting value <7 mmol/L but a 2 hour value ≥11.1 mmol/L on an OGTT.11 In another study in a high-risk US population, one quarter of all people with diabetes had a fasting glucose <6.0 mmol/L.13 Furthermore, because the OGTT uses a larger glucose test meal than most people ever normally consume, a person's random glucose will rarely be as high as their 2 hour OGTT test value. These factors have important implications for choice of screening tests to use and how to interpret them.

**Who to test**

We recommend screening people who have a 5% or more risk of having undiagnosed diabetes (Table 1). While the choice of 5% is arbitrary, it is consistent with the 1995 NZSSD guidelines1 and with draft Australian guidelines.1 In addition, many people are at 'high risk' of undiagnosed diabetes because they have co-morbidities known to increase diabetes risk; obesity, high blood pressure, low HDL cholesterol, raised triglycerides, a parent or sibling with diabetes, cardiovascular disease, peripheral vascular disease, cerebrovascular disease or polycystic ovary syndrome. Therefore, we recommend screening for diabetes in Europeans age 50 years or more, non-Europeans age 40 years or more, and both groups ten years earlier if they are at 'high risk' as defined above.

Most of those screened will not have diabetes. Nevertheless, some 5% of those people without diabetes at initial screening will progress to diabetes within three years, so all those with a negative screening test should be recalled for re-screening three yearly. In contrast, the progression to diabetes is faster for people known to have IGT or impaired fasting glucose (IFG) or previous gestational diabetes, with some 5% developing diabetes every year. Therefore, we recommend screening this sub-group yearly.

**Which test?**

A 'fasting' glucose test means that the person has had no food or drink, except water, for 8 hours prior to the test.11 A fasting glucose should be done in the morning, as a test done in the afternoon can be as much as 1 mmol/L lower than in the morning,14 which could result in a falsely negative screening result. If the fasting glucose result is ≥7.0 mmol/L then the person has diabetes if they have characteristic symptoms or if they have a repeat glucose above the diagnostic level on another day. If the fasting glucose result is 6.0-6.9 mmol/L, the person has IFG. This should be followed up with an OGTT as he or she may have diabetes according to the 2 hour test. In the above example of the elderly European population,11 calling values <7.0 mmol/L a 'negative' screen, ie one requiring no further testing, would result in missing one third of all those who actually have diabetes (which is the same as saying the screening test had a sensitivity of 67%).

Even some people with a fasting glucose of 5.5-6.0 mmol/L will have diabetes on the 2 hour test of the OGTT. In the example of the high risk US population,14 calling values <6.0 mmol/L a 'negative' screen would result in missing one quarter of those with diabetes (which is the same as saying the screening test had a sensitivity of 75%). We therefore recommend an OGTT for this group if they are otherwise at 'high risk' as specified above. A person with a fasting glucose <5.5 mmol/L is highly unlikely to have current diabetes.

A 'random' glucose test is performed in no fixed relation to time since eating or amount of prior food or drink. A random glucose may therefore be more difficult to interpret than a fasting glucose, ie it can be more difficult to decide whether to send the person for further testing. A practitioner is entitled to make a judgement as to how closely the random glucose approaches the conditions of a 'fasting' glucose or those of a 2 hour OGTT, and decide follow-up accordingly. In mid-2000 we asked all private New Zealand laboratories for their 'normal range' for random glucose, and found that they varied widely (unpublished). We recommend using a cut-off, admittedly arbitrary, of 6.0 mmol/L, ie a random glucose ≥6.0 mmol/L warrants further testing, either with an OGTT if the person is at 'high risk' as defined above, otherwise with a fasting glucose.

The HbA1c test has been used for many years to monitor glucose control in people with known diabetes. However, over the past ten years there have been several studies assessing its usefulness as a screening test for diabetes, either used alone or used at the same time as a fasting glucose.15,17-20 The appeal is that HbA1c is not affected by when the person last ate or drank, and it may help identify the people who would have a raised 2 hour test on an OGTT despite a non-diabetic fasting glucose. Unfortunately, HbA1c is dogged by the fact that there can be clinically significant differences in results when the same blood sample is tested by different methods. There are currently two main methods used in New Zealand, each with minor variations. Furthermore, as for random glucose, laboratories around the country report different 'normal ranges', and current comments are designed to help practitioners interpret the tests when used for monitoring diabetes, not when used for screening. Nevertheless, many laboratories report so many low results - for example, one third under 5.5% (GB unpublished) - that it seems many GPs are already using HbA1c as part of their screening process for diabetes. No international body currently recommends screening using HbA1c. Further research is needed on the usefulness of using HbA1c as the primary test to screen for diabetes.

Many primary health care providers screen for diabetes using capillary blood testing ('finger-prick') meters and strips designed for people with known diabetes to monitor their glucose at home. These meters are simple and convenient, and some GPs and practice nurses comment that they prefer to test a patient 'on the spot' for patient convenience or to reduce the chance that the patient will not or cannot attend the laboratory if given a laboratory request form. While we accept that this is a judgement for practitioner and patient, unfortunately the meters are technically a poor substitute for a laboratory glucose. For example, when the 'true' venous plasma is 7.0

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Figure 1. Diabetes can be diagnosed by a fasting glucose ≥7.0 mmol/L, a 2 hour value on an glucose tolerance test (or random glucose) ≥11.1 mmol/L, or both. Impaired fasting glucose (IFG) is intermediary between normal and diabetes diagnosed on the fasting criterion. Impaired glucose tolerance (IGT) is intermediary between normal and diabetes diagnosed on the 2 hour criterion.
mmol/L, 95% of the readings of one of the best New Zealand meters will fall between 4.9 and 8.2, with a mean of 6.6 mmol/L. For comparison, 95% of the laboratory readings will fall between 6.8 and 7.2 mmol/L. While meter performance is adequate for home glucose monitoring, when used to screen people whose true fasting venous glucose is around 7.0 mmol/L, about half of them may be misclassified as having diabetes when they do not, or vice-versa. On the other hand, this is clearly not a problem when the true fasting glucose is, say, 9.0 mmol/L or more. We therefore recommend treating the results of capillary meter testing with considerable caution, especially if the result is within 2 mmol/L of the cut-off point being used to decide if further testing is warranted. Ideally, we recommend restricting meter use to screening patients who have symptoms characteristic of diabetes, and subsequently confirming results with a laboratory glucose.

**Where to test**

Diabetes screening is currently undertaken in a wide range of community, primary care and secondary care settings. However, general practice is the only setting in which 80-90% of people at risk of undiagnosed diabetes attend in any one year,27 is the setting most likely to have the complex information needed to identify people at ‘high risk’ and is the only setting with established systems capable of recalling people for follow-up screening in one or three years. Therefore, we see general practice or equivalent primary health care as the only appropriate setting for any form of systematic screening. To achieve this, however, requires more systematic use of reminders, recalls and related systems of care than are currently in use in most general practices - a challenge for all.

**Conclusion**

We have recommended who, how and where to screen for diabetes. We believe the evidence firmly supports the value of finding and treating diabetes, IGT and the associated metabolic and lifestyle disorders. The best opportunity for this in New Zealand is through what we call ‘systematic opportunistic screening’ in general practice or equivalent primary health care.

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**US “boutique medicine” could threaten care for the majority**

In south Florida last June Dr Robert Colton, an internist with more than 20 years’ experience, started a company called MDVIP with four other doctors. In return for a yearly membership fee of $1500 ($1050; £1700), the doctors contracted to provide patients with annual physical examinations, same day appointments, 24 hour doctor availability, co-ordinated referrals to specialists, and online access to their medical records. Dr Colton promised to limit his practice to 600 patients.

Now similar groups are springing up in Arizona and Washington state, including one in Seattle charging families $20 000 a year. Plans are afoot to create outfits similar to the US “boutique medicine” could threaten care for the majority

Cardiovascular disease and lipid management in New Zealand: progress at last!

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Epidemiological studies in the 1950s and 1960s established the crucial association between raised total cholesterol and the development of ischaemic heart disease. The Framingham study investigators coined the term ‘risk factor’. Initial attempts to demonstrate reductions in the rate of ischaemic heart disease through cholesterol reduction were hampered by the limited potency of the then available drugs and a relatively high level of patient side effects. Nonetheless, the Lipid Research Clinics Coronary Primary Prevention Trial using cholestyramine and the Helsinki Heart Study using gemfibrozil demonstrated a reduction in cardiac events in ‘high risk’ populations. The Coronary Drug Project using high dose nicotinic acid, also demonstrated benefit in patients with established ischaemic heart disease, although it took sixteen years for a statistically significant mortality endpoint to emerge. The development of the potent and safe ‘statin’ drugs allowed efficacy, angiographic and endpoint outcome studies to be initiated. The angiographic trials typically showed only minimal ‘regression’ of coronary atheroma, yet a significant decrease in the number of cardiovascular events for patients randomised to statin drugs. These observations consolidated the concept of endothelial stabilisation, resulting in reduced atheromatous plaque rupture and acute coronary syndromes.

Five major placebo-controlled statin trials were completed in the 1990s. Three were in populations with known coronary disease: the Scandinavian Simvastatin Survival Study (4S) using simvastatin, the Cholesterol and Recurrent Events (CARE) trial using pravastatin, and the Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID) study. Two trials were in populations without known coronary disease: the West of Scotland Coronary Prevention Study (WOSCOPS) using pravastatin and the Air Force/Texas Coronary Atherosclerosis Prevention Study (AFCAPS/TexCAPS) using lovastatin. All five trials showed similar relative reductions in vascular endpoints of myocardial infarction, stroke or cardiac death of approximately 30% over five years. After the presentation of 4S, the 1996 New Zealand Lipid Guidelines were formulated and then published and have been a valuable tool to help clinicians identify high-risk subjects in whom cholesterol reduction would confer cardiovascular benefit. Around the same time the Pharmacology Management Agency (PHARMAC) established its guidelines for statin approval, and these remain substantially unchanged in 2002.

More recent publications

Following the publication of the 1996 Guidelines, the CARE, the LIPID and AFCAPS/TexCAPS studies were published. CARE and LIPID demonstrated the benefit of statin therapy in patients with known ischaemic heart disease and cholesterol levels much lower than for 4S (≥4mmol/L). AFCAPS/TexCAPS extended the benefit of statins to a low-risk population with a total mean cholesterol of 5.7mmol/L and a relatively low mean high-density lipoprotein (HDL) cholesterol of 0.96mmol/L.

Other major studies have also been published since the 1996 New Zealand Lipid Guidelines. The Post Coronary Artery Bypass Graft (CABG) trialists study using lovastatin (and cholestyramine), and the Aggressive Lipid Lowering with Atorvastatin versus Revascularisation Treatments (AVERT) trial enrolled patients with prior revascularisation with CABG surgery or percutaneous coronary intervention (PCI) and randomised them to vigorous versus standard lipid management, as a major part of the trials. Both trials demonstrated that vigorous cholesterol lowering treatment to a level below 4mmol/L or a low-density lipoprotein (LDL) cholesterol well below the 2001 United States National Cholesterol Education Program (NCEP) Adult Treatment Panel 3 (ATPIII) guidelines of 2.6mmol/L resulted in greater clinical benefit than for less stringent lipid targets. The Myocardial Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL) Study; using 80 mg atorvastatin daily, demonstrated that treating patients with an acute coronary syndrome with a statin between 24 and 96 hours following hospital presentation was not only safe, but also beneficial for reducing vascular events by the fourth month of therapy. The recommendation of the 1996 Guidelines to delay statin treatment for 3-6 months in order to assess the effect of diet and lifestyle change is thus obsolete. Correspondingly the inclusion of this stand-down period in the PHARMAC approval system for statins up to date, has been against medical evidence, and has compounded short and long-term compliance treatment issues. Endpoint fibrate studies have also been published since 1996. The Veterans Affairs High-Density Lipoprotein Cholesterol Intervention Trial (VAHIT) using gemfibrozil and the BezaFibrate Infarction Prevention (BIP) trials have demonstrated that LDL cholesterol reduction by statins is not the only way of reducing vascular events. In an ischaemic, dyslipidaemic population whose principal problem was low HDL cholesterol and a raised triglyceride level, fibrate drugs significantly reduced the risk of myocardial infarction, stroke and death.

The heart protection study

By far the largest statin trial, the Heart Protection Study, has recently been presented at the American Heart Association Scientific Meeting in November 2001 in Anaheim, California and its results disseminated to the medical community on www.hpsinfo.org. 20 536 British subjects were enrolled: 13 379 with coronary disease and 7157 (35%) without overt coronary disease. Of these ‘non-coronary’ subjects, 1822 had cerebrovascular disease, 2185 peripheral vascular disease, and 3150 were selected because they were at ‘high risk’ of developing vascular disease, being treated for diabetes mellitus (n=2913) or hypertension (n=237). Patients with a total cholesterol of 3.5mmol/L and above were randomised to
simvastatin 40mg daily or placebo. The 2 x 2 factorial design of the study also randomised patients to 600mg Vitamin E, 250 mg Vitamin C and 20 mg beta-carotene daily versus placebo, but the addition of these supplementary antioxidant vitamins had no beneficial effects. However the simvastatin therapy was extremely well tolerated and safe, resulting in a 24% risk reduction of vascular events, including myocardial infarction, stroke and death. This study has provided a major extension to our statin knowledge. It indicates that patients aged between 40 and 80 years, with either coronary, cerebrovascular or peripheral vascular disease, or those at high risk of developing vascular disease due to pre-existing diabetes mellitus, and with cholesterol levels of 3.5mmol/L or above, would benefit from a statin drug.25 Those with pre-existing hypertension may also benefit, although only a small number of subjects (n=237) were enrolled in HPS.25 Further, as with all of the statin trials, the benefit, although only a small number of subjects (n=237) were enrolled in HPS.25

1. Perhaps the most long overdue change is with heart disease. While this is a welcome change, it carries concerns. Fluvastatin is immediately bench marked against simvastatin and will carry a considerable part charge and within two years the same may happen with atorvastatin. Withdrawal of these statins is a possibility in that circumstance leaving New Zealand with a sole statin agent on the market. That would compromise the care of many subgroups of patients. Furthermore it creates an impossible environment for the fund listing of the newer so called ‘super-statins’.

2. The 1996 New Zealand Lipid Guidelines correspondingly have been long overdue for an update and the reconvening of a group to do this is welcomed. The guidelines will undoubtedly reflect the available clinical trial data and it is assumed they will endorse more vigorous lipid management, in terms of total and LDL cholesterol reduction,26 as well as focussing attention on patients with low HDL and high triglyceride levels.27,28 The more important task, however, will be the difficult process of ensuring that the guidelines are implemented in primary and secondary practice, that priority treatment is applied to those with highest risk and that lower risk patients are not given statin treatment based simply on laboratory values of cholesterol.

3. It follows that clinicians will need to become guideline ‘wise’ and practice accordingly, adopting the messages from scientific trials more effectively than has been reported to date.19,21,25

4. The issue of patient compliance needs to be addressed both in New Zealand and overseas. Methods of improving patients’ uptake of medication must be explored,24,25 and include assessments of how the patients understand their illness.26,27 In the future, large numbers of patients and high-risk individuals will be prescribed lipid modifying medicines over prolonged periods of time, and will only gain a benefit if they comply with treatment.

In summary, we are now experiencing a paradigm shift in our understanding of the role of lipid management and cardiovascular disease. There are currently approximately 120,000 New Zealanders receiving lipid-modifying agents.29-31 A figure closer to 400,000 within five years would vastly improve patient outcomes for cardiovascular disease, the commonest cause of death and major morbidity in New Zealand.32-34 The impending changes to access if coupled with utilisation of the soon to be available guidelines will allow most high-risk patients to be more effectively treated. All doctors have a responsibility to use this clinical resource efficiently and wisely.

**Progress at last**

It has been abundantly clear to many that the clinical management of patients with vascular disease and those at high risk of developing vascular disease in New Zealand has needed to move forward in line with these scientific studies. Fortunately the Heart Foundation, individual clinicians and PHARMAC, under the auspices of the Guidelines Group, are currently addressing this need.

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25. Heart Protection Study (HPS). Late Breaking Clinical Trials. Session Anchale, USA: Tuesday 11/1;00, American Heart Association Scientific Conference 13/11/01.


