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This Issue in the Journal

A system of chronic pain: disability consequential to accidental injury
Margaret H Moon, Brian E Niven

Chronic pain, following accidental injury, is a disability costly to New Zealand because of work days lost, medical and compensatory costs, and altered roles and relationships. For 30 years, the Melzack Pain Questionnaire has been used as an assessment tool, but this test masks the manner in which ‘pain’ and ‘fear’ both contribute to the complexity of this disability. To understand this complexity, system theory is used, and analysis shows that ‘sleep disturbance’ as well as ‘fear’ and ‘pain’ are part of the disability that formerly had been attributed to the ‘pain’ alone. System theory lets terms like ‘copers’ and ‘catastrophisers’—that have emotive overtones (goodies and baddies) and are used by ‘pain experts’—to be defined objectively.

Diabetes eye screening in the Wellington region of New Zealand: characteristics of the enrolled population (2002–2005)
Lesley G Frederikson, Robert J Jacobs

Visual impairment is a complication of diabetes which can be reduced through better management of the diabetic disease. The chances of vision loss can be reduced by keeping blood glucose levels under control and by maintaining a healthy weight and fitness level. Regular eye-screenings for diabetic eye disease can assist with the management of the condition as early changes will be detected. Monitoring how diabetic eye disease develops and advances will enable timely treatment to be provided and can avoid the blindness that would otherwise result. The availability of eye screening services varies around the regions of New Zealand. Wellington was the first region to provide community based screening by optometrists. The two key strengths of the Wellington diabetes eye screening programme are (i) its routine collection of comprehensive information and (ii) the links it established between GPs, optometrists, and ophthalmologists. The high uptake of the Wellington eye screening service and the comprehensive data collected allowed us to make a thorough assessment of how retinopathy and maculopathy develop among those diagnosed with diabetes in New Zealand. The study highlighted the importance of blood sugar control and the need to monitor visual performance regularly. For people with diabetes affected by retinopathy it is important that they have ongoing vision care and the best visual correction and treatment available to make the most of their remaining vision.
Retention of patients in the Get Checked free annual diabetes review programme in New Zealand
Grace Joshy, Ross A Lawrenson, David Simmons

This paper looks at the retention of patients in the “Get Checked” free annual diabetes review programme in the Waikato region in New Zealand. Younger patients aged <40 years, those of Māori or Asian origin, and those with Type 1 diabetes were less likely to be retained in the programme with regular checks. Despite the programme being fully funded, a significant proportion of patients did not return for a second review within 1.5 years after initial review. Use of these data for policy purposes could be significantly biased unless there is a single reliable regional diabetes register based on the National Health Index number including all known patients.

Concordance and discordance between primary and secondary care health workers in perceptions of barriers to diabetes care
Steven Lillis, David Simmons, Judith Swan, Jarrod Haar

Those with diabetes may receive care from both a general practice and a hospital. There are some differences in beliefs between healthcare workers in hospitals and general practices concerning barriers to good diabetes care. Hospital healthcare staff rated appointment systems, inappropriate cultural messages, lack of community-based services, high prevalence of diabetes, and unhelpful health practitioners as barriers to care. Primary care health workers are more likely to rate motivation, self-belief, financial issues, lack of governmental funding, lack of public awareness of diabetes, and lack of symptoms as barriers to care.

Supporting pregnant women to quit smoking: postal survey of New Zealand general practitioners and midwives’ smoking cessation knowledge and practices
Marewa Glover, Janine Paynter, Chris Bullen, Kay Kristensen

This study examined smoking cessation (quitting) knowledge, messages given, and support offered to pregnant women who smoke by New Zealand general practitioners (GPs) and midwives. The information was obtained via a random postal survey of New Zealand GPs and midwives, undertaken between September and October 2006. The study found that GPs are in the ideal position to offer stop-smoking advice, because they usually confirm pregnancy. GPs are most likely to advocate stopping smoking completely; midwives are more likely to advocate cutting down with a view to quitting. Both GPs and midwives would benefit from improved knowledge of the full range of nicotine replacement therapy (NRT) products available so they recommend them to smoking pregnant women more.
Just how safe is the New Zealand health system?

Des Gorman, John Kolbe

The first annual report of the Government’s Health Quality Improvement Committee released on 10 February 2008 could be of great comfort; if we are compared by way of the cited data to the United Kingdom and North America, we would seem to be doing very well.\(^1\) Alternatively, the report could be of great concern if the relevant reporting rate and veracity is as low and poor as is likely.\(^3\) Most probably, it is of no use whatsoever, as even a rudimentary analysis of the integrity of the system used to collect the reported data shows it to be somewhat wanting.

Committee Chairman Mr Pat Snedden might have been better off quoting the old statistical aphorism about “rubbish in equals rubbish out.” Some attention is certainly warranted by Mr Snedden’s committee to modern perspectives of quality measurement and management in health;\(^6\)–\(^8\) best practice in this context is a far cry from the burgeoning data collection exercise undertaken by District Health Boards as part of their reporting obligations to the Ministry of Health. In the USA, a similar obsession with high quantity, poor quality “quality-data” is thought to have “paralysed” elements of the health services.\(^9\) We would refer readers to a very appropriate analogy in this context.

…Perhaps the culture of accountability that we are relentlessly building for ourselves actually damages trust rather than supporting it. Plants don't flourish when we pull them up too often to check how their roots are growing: political institutional and professional life too may not go well if we constantly uproot them to demonstrate that everything is transparent and trustworthy.\(^10\)

The increase in complaints cited by the Health and Disability Commissioner, Mr Ron Patterson,\(^11\) may reflect deterioration in the quality of our health services. However it might just demonstrate a growing “culture of complaint.” It needs to be remembered, by anyone interested in understanding just how safe our health system is, that the first thing to suffer in any culpability-based system of reporting is honesty.\(^2\)\(^,\)\(^12\)\(^,\)\(^13\)

“Beating up” on doctors predictably and understandably encourages doctors to engage in doctor-protective behaviour and this can be at the expense of patient safety. The unfavourable “safety” comparisons with the aviation industry, made recently by the United Kingdom’s chief doctor, Sir Liam Donaldson,\(^1\) overlook the blame-free nature of reporting that has led to such an enviable safety record for the airlines and general aviation.\(^14\)

There are good reasons why the New Zealand health system could be sick. Successive reforms have created a schism between managers, clinicians, and public health advocates. District Health Board key performance indicators are hospital-oriented and throughput-obsessed, such that a perverse “widget factory” culture pervades our hospitals.

Industrial relations between these health boards and their employees (such as evidenced by the current senior medical officer dispute) are poor. The
Industrialisation of the junior doctor workforce has largely dismantled the apprenticeship basis of continuing medical education for those in the early postgraduate years.\textsuperscript{15}

General medical practitioners—the sector of the medical community that is most capable of driving up health quality and driving down costs\textsuperscript{16}—have been significantly undervalued and undermined by Government and Ministries for several decades. The current relevant ideological obsession for primary care is for (incentive-free) capitation.

New Zealand never caught the Prime Minister’s knowledge wave; we are the most reliant country in the OECD on overseas-trained doctors.\textsuperscript{17} For example, only one in three of our practising rural doctors are New Zealand-trained. And we are no longer able to recruit from countries of equivalent medical educational standard, as witnessed by the recent failed attempt to lure several hundred United Kingdom graduates to New Zealand in the face of a supposed glut there—only about 20 were recruited.

Student debt is now a major determinant of career choice for our medical graduates,\textsuperscript{18,19} and (in this context) there is a strong financial incentive for them to either go overseas and or to take up specialist practice, which is rich in procedures and technology.

This has to be seen against a background of the pharmaceutical industry in the USA alone spending more money on direct-to-doctor propaganda than the combined budgets of all the medical schools.\textsuperscript{20,21} Somewhat flippantly, it is fair to say that doctors are not accidentally stupid.

There are a plethora of reasons why our health system should be performing badly. On face value, the data reported by Mr Snedden should encourage us to look for the reasons why in such an apparently pear-shaped system we are doing so well. What we actually need are meaningful data, and the nature of this report and the reaction to it are unlikely to get us to a better position.

If New Zealanders want some hard data to chew on, the average life expectancy of a Māori man is 9 years less than that of his Pākehā (New Zealand European) equivalent; a greater gap (by 2 years) than for Europeans living in North America compared to indigenous Americans.\textsuperscript{22,23} That fact alone should be a cause for discomfort as compared to the Quality Improvement Committee’s “data.”

**Competing interests:** None known.

**Author information:** Des Gorman, Head of the School of Medicine, The University of Auckland, New Zealand; John Kolbe; Head of the Department of Medicine, School of Medicine, The University of Auckland, New Zealand, and, Chair of the Adult Division of the Royal Australasian College of Physicians, Sydney, Australia

**Correspondence:** Professor Des Gorman, Head of the School of Medicine, The University of Auckland, Private Bag 92019, Auckland, New Zealand. Fax: +64 (0)9 3737599; email: d.gorman@auckland.ac.nz

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The chronic pain experience—a dynamic complex interaction

Edward A Shipton

The chronic pain experience is the product of a dynamic complex interaction of many factors including biological, social, psychological, environmental (such as patients' close social environments), and familial. This results in a non-linear relationship between the onset of chronic benign pain and its outcome.

In this issue of the Journal, Moon and Niven (A system of chronic pain: disability consequential to accidental injury; http://www.nzma.org.nz/journal/121-1270/2943) have undertaken a longitudinal study of a group of chronic pain sufferers within a community-based practice over 2 years. They view chronic pain as a system that includes measures of pain and interactions with a set of processes associated with chronic pain (depression, state and trait anxiety, sleep, and stress).

A set of null hypotheses (all rejected) was drawn from this theory, namely: there were no relationships between the measures of pain and associated processes with no changes over time; and there were no changes over time in any relationships between the measures of pain and the measures of the associated processes.

The process variables were then scrutinized. Forty-two subjects were initially tested at the start of the study, with the same 42 subjects being tested 2 years later. Measures used included the McGill Pain Questionnaire, the Self-Rating Depression Scale, the State-Trait Inventory, and the Social Readjustment Rating Scale. Let us consider some of these processes associated with chronic pain, namely depression, sleep disturbance, stress, anxiety and fear, and the disability created.

Numerous studies have shown depression to be highly prevalent among persons with chronic pain. In clinical studies, rates of current major depression can range from 30% to 54% (significantly higher than the rate of 5–8% found in the general population).

Sleep and rest significantly influence the quality of life of chronic pain patients. At least 50% of patients with chronic pain report simultaneous sleep disturbance. Although some factors (pain severity, depression, and pain beliefs) are consistently documented as predictors of disability, others such as sleep disturbance and sleep quality are less well understood. No instrument has been validated to measure sleep disturbance in chronic pain. However, scales like the Medical Outcomes Study Sleep Scale may, in future, be used to determine the interference of sleep with the perception of pain.

The relationship of chronic pain and post-traumatic stress disorder with increased impairment and distress is well documented in the literature. The severity of pain and emotional numbing are related to reductions in life satisfaction as well.
Interventions targeting physical pain and emotional numbing may prove central to improving the quality of life of many patients with comorbid chronic pain and post-traumatic stress disorder.

Pain is an ideal habitat for worry to flourish. The fear-avoidance model postulates that catastrophic misinterpretations of pain lead to fear of pain and promote a cycle of activity avoidance, disuse, and disability.\textsuperscript{4} Helplessness, fear of pain, and passive pain-coping strategies are all related to pain levels, disability, and depression.\textsuperscript{5} Even in children, anxiety sensitivity and fear of pain play important roles in the processes that maintain chronic pain and pain-related disability.\textsuperscript{6}

Cognitive-behavioural therapy and operant behavioural therapy treatments focus on factors like fear that exacerbate or maintain suffering in chronic pain.

Chronic pain patients can be thought of as trapped in a ‘perseverance loop’. Interventions should be aimed at enabling patients to break out of this ‘perseverance loop’ and change the problem framework. They may be more effective than interventions that appear to endorse the patient’s view of the problem as one that can only be solved by pain relief.\textsuperscript{7}

Pain severity, depression, and pain beliefs (such as pain-related fear) are related to disability in persistent pain conditions. This disability is costly both to the individual and to society. In a recent study looking at correlates of self-reported disability in patients with low back pain, pain and fear-avoidance beliefs primarily drove the emergence of disability.\textsuperscript{8}

Another study on chronic low back pain published in February 2008 found that anxiety, depression, fear-avoidance beliefs (relating to work), and back pain-related stresses predicted impairment in subsequent physical health-related quality of life and healthcare utilisation.\textsuperscript{9}

In an ingenious manner, Moon and Niven have teased out the importance of ‘fear’ as a correlate of chronic pain that had been masked by the manner of construction of the McGill Pain Questionnaire. As a result, ‘fear’ emerged as the only variable to predict measures of pain at both the start and finish of the 2-year test period. As the processing of pain takes place in an integrated matrix throughout the neuraxis, their study adds weight to the description of chronic pain as a system of pain, fear, and sleep. Adequate interventions are those able to break these loops and stabilise them.

If specific treatments (in addition to usual rehabilitation techniques) aimed at social stresses, depression, and maladaptive beliefs in chronic pain are given, would physical health-related quality of life and healthcare utilisation continue to improve?

The system model of Moon and Niven adds to our understanding of chronic (benign) pain.

\textbf{Competing interests:} None known.

\textbf{Author information:} Edward A Shipton, Academic Head and Chair, Department of Anaesthesia, Christchurch School of Medicine, University of Otago, Christchurch

\textbf{Correspondence:} Professor Edward Shipton, Department of Anaesthesia, Christchurch School of Medicine, University of Otago, PO Box 4345, Christchurch, New Zealand. Fax: +64 (0)3 357 2594; email: ted.shipton@cdhb.govt.nz
References:


A system of chronic pain: disability consequential to accidental injury

Margaret H Moon, Brian E Niven

Abstract

Aim The aim of this research is to see if system theory is a tool that can give a new understanding of chronic pain.

Method Over a 2-year period, a group of people with chronic pain was tested with measures of pain and the associated processes of depression, state and trait anxiety, sleep, and stress.

Results In the analysis, ‘fear’ emerged as a correlate of pain and a ‘pain factor’ was identified which comprised measures of pain, fear, and sleep. This factor could account for the disability associated with chronic pain, which is costly to the individual and society.

Conclusion This study showed the validity of describing chronic pain as a system that could be understood using measures of pain, fear, and sleep. As a consequence, a set of commonly used terms is defined objectively and questions posed in the literature are answered.

Although the terms ‘chronic pain’ and ‘persistent pain’ have tended to be used interchangeably, ‘chronic’ is used in this paper because the ‘passage of time’ is central to the analysis.

The form of chronic pain that is most frequently investigated is low back pain, but chronic pain is not homogenous. In low back pain, many different structures can be damaged while chronic pain occurs in many other sites and structures around the body.

The consequences of having chronic pain are unpredictable, with cause, treatment, and outcome not necessarily showing a linear relationship. The heterogeneity of causes, the multiplicity of interventions, and the unpredictability of the outcomes point to a non-linear relationship between the onset of chronic benign pain and its outcome.

The language of system theory in reference to chronic pain has been referred to in the literature for some years. When pain is considered clinically or theoretically, general system theory is often used (whether it is articulated or not). With a pain system as the system of reference, varied emphasis is placed on neuroanatomic, neurochemical, and on psychological sub-systems and interpersonal and social suprasystems.\(^1\)

For the chronic pain patient, system or control theory can describe the process or dynamics and address the change mechanisms of interventions.\(^2\) A system has been described as organised complexity,\(^3\) and is defined as a set of interacting elements. Interactions occur between systems. Cybernetics, involving feedback, is based on
communication (transfer of information) between the system and the environment, or within the system and control (including feedback) of the system’s function in regard to the environment.

Chronic pain can be described as a system that includes measures of pain and measures of a set of factors that are associated with pain, together with the mutual relationships (loops) between these processes.

A set of null hypotheses was drawn from this theory, namely there are:

- No relationships between the measures of pain and the measures of a set of processes associated with chronic pain;
- No changes over time within the measures of pain and the measures of a set of associated processes; and
- No changes over time in any relationships between the measures of pain, and the measures of a set of processes associated with chronic pain.

To test this theory, a group of chronic pain sufferers was observed. The common feature of this group was that they were attending the researcher’s private practice at the time of commencement of the study. The participants in this study are not a sample of a specific population of pain sufferers. The study is not about individual differences but seeks to substantiate a model suggested by scientific literature and clinical experience.

Measures of pain and of a set of processes that the literature associated with chronic pain were examined since it was the process variables that were being scrutinized; did the process variables interact? Did the relevant process variables and/or the interactions between them alter over time? Was there any indication that a system of processes relative to pain was independent of other pertinent systems or did they interact?

**Methods**

The process variables selected to test were measurements of pain, depression, state and trait anxiety, and stress. The following standard tests were used.

- McGill Pain Questionnaire (MPQ).  
- Self-Rating Depression Scale (SDS).  
- State-Trait Inventory (STAI).  
- Social Readjustment Rating Scale (SRRS).

All participants had been referred medically to the practice. Frequency of attendance throughout the test period was by their own choice and they were free to seek intervention from other sources. These variables were observed at two times for all participants:

- Time 1, the initial testing of 42 subjects at the start of the study, and
- Time 2, the testing of the same 42 subjects 2 years after the initial testing.

The software program SPSS (Statistical Package for Social Sciences) was used to analyse the data. The statistical procedures used were:

- Spearman’s correlation coefficients: to examine the relationship between variables.
- Wilcoxon matched-pairs signed-ranks test: to test for changes over time.
- Multiple regression, stepwise: to relate pain scores to predictor variables.
- Factor analysis with varimax rotation: to examine structural relationships among variables.
The assumptions required for all tests were checked during analysis, and found to be satisfactory.

The origin and descriptors of the key variables used in this study are shown in Table 1, with the abbreviations used in analyses at Time 1 and Time 2. Chronicity is measured by the number of months from the accident until Time 1.

Melzack used a rank-value method of scoring the MPQ. His instructions are that the word in each subclass of adjectives implying the least pain is given the value of 1, the next word up is given the value of 2, and so on. The values of the words chosen by the subject are then added up to obtain a score for each category and a total score for all the categories.

The categories are described as sensory, affective, and evaluative (factors), with an additional group of adjectives called supplementary. This score is referred to as the ‘total pain rank score (Rank)’.

Alternatively, Melzack scored the MPQ by counting the number of words (NWC) or total numbers of descriptive adjectives chosen by each subject.

Table 1. Key variables

<table>
<thead>
<tr>
<th>Process</th>
<th>Test</th>
<th>Score descriptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>MPQ</td>
<td>Total pain rank</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Number of words counted (NWC)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sensory (rank total)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Affective (rank total)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Evaluative (rank total)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Supplementary (rank total)</td>
</tr>
<tr>
<td></td>
<td>MPQ19</td>
<td>Total pain rank—fear (derived)</td>
</tr>
<tr>
<td>Chronicity</td>
<td>Passage of time</td>
<td>Months since accident</td>
</tr>
<tr>
<td>Fear</td>
<td>MPQ</td>
<td>Adjective group</td>
</tr>
<tr>
<td>Sleep</td>
<td>MPQ</td>
<td>Additional MPQ question*</td>
</tr>
<tr>
<td>Depression</td>
<td>ZUNG / SDS</td>
<td>Self-rating score</td>
</tr>
<tr>
<td>State anxiety</td>
<td>STAI-X1</td>
<td>“Here-and-now” anxiety</td>
</tr>
<tr>
<td>Trait anxiety</td>
<td>STAI-X2</td>
<td>Personality trait</td>
</tr>
<tr>
<td>Stress</td>
<td>SRRS</td>
<td>Environmental change</td>
</tr>
</tbody>
</table>

All participants were tested at two times:

**Time 1,** Initial testing, start of study and **Time 2,** Testing 2 years after the start.

*Version of the McGill Pain Questionnaire: An older version of MPQ was used at both Times 1 and 2 that included an additional categorical question on sleep; was it good, fitful, can’t sleep? More recent versions of the MPQ omit this question.

**Results**

The McGill pain questionnaire scores—The nature of the measures yielded by the McGill Pain Questionnaire were examined. The correlations between the scores for the total rank score and the separate scores given by the McGill Pain Questionnaire for the sensory, affective, evaluative (cognitive) factors and for the supplementary adjectives were examined. Almost all of the correlations were significant, p<0.0001. Therefore, because the scores of pain were not orthogonal, the Total Pain Rank Score only is used as the measure of pain in the analyses that follow.
A regression analysis of the scores on these adjectives on the Total Rank Pain Score (RANK) at Times 1 and 2 is made. From these analyses equations can be written:

**Time 1:**
\[ \text{Pain} = 8.0 \times \text{fearful} + 2.7 \times \text{pinching} + 2.3 \times \text{annoying} - 2 \ (R^2 = 0.41). \]

**Time 2:**
\[ \text{Pain} = 9.6 \times \text{fearful} + 0.20 \times \text{annoying} + 4.8 \times \text{tingling} + 3.0 \times \text{spreading} + 12.4 \times \text{jumping} - 16.2 \times \text{wretched} - 28.4 \ (R^2 = 0.62). \]

The regression analysis shows that the scores of *fear* and *annoyance* predict the rank score of pain at Time 1. The score of the adjective category *fear* predicts the rank score of pain at Time 2. The predictive values of adjectives other than *fear* are relatively small. *Fear* is one of the 20 sets of adjective descriptor groups used in the MPQ.

Subjects had 4 options:
1. Not to make a choice within this set;
2. To choose: *fearful*;
3. To choose *frightful*; or
4. To choose *terrifying*.

Thus, the *fear* group of adjectives was dominant over the other 19 groups of adjectives at both times 1 and 2. Because *fear* appears to behave differently from the other 19 groups of adjectives and because of its apparent dominance on the total pain rank score of the MPQ, the score on the *fear* (FEAR) group of adjectives was separated from the Total Pain Rank Score (RANK), leaving 19 sets of adjectives only to account for the score on pain. This adjusted rank score is named MPQ19.

Thus, MPQ19 = RANK (Total Pain Rank Score) – FEAR, at both Time 1 and Time 2.

Spearman’s correlation coefficients were calculated between the scores of RANK (total pain rank score), NWC (number of words counted), MPQ19 (adjusted rank score), and Fear (adjective group) at Times 1 and 2.

All the correlations RANK, NWC, MPQ19, and Fear were statistically significant at p≤0.001 level. Thus, the adapted pain score (MPQ19) correlates with Melzack’s two alternative methods of scoring of the full McGill Pain Questionnaire, Total Pain Rank Score (RANK), and Numbers of Words Counted (NWC) at both Times 1 and 2.

These three methods of scoring the pain all show statistically significant correlations with the score of *fear* at both times. Therefore, separated scores of MPQ19 (denoting the pain score derived from the MPQ) and *fear* (the one adjective set removed from the MPQ) were used in the remainder of this analysis. The two variables are measured distinctly.

**The relationships between pain and associated variables**—The means and standard deviations of measures at Time 1 and Time 2 with N=42 in all further calculations, are shown in Table 2.

Spearman’s correlation coefficients were calculated across the set of variables at both times. At Time 1, pain correlates with fear (0.57) and sleep (0.41); fear correlates with stress (0.48); trait anxiety correlates with depression (0.79), all at p≤0.001 level of significance.
Table 2. Descriptive data of process variables at Time 1 and Time 2

<table>
<thead>
<tr>
<th>Process</th>
<th>Time 1</th>
<th>Time 2</th>
<th>Time 1</th>
<th>Time 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Pain (total rank)</td>
<td>26.48</td>
<td>13.35</td>
<td>26.02</td>
<td>13.33</td>
</tr>
<tr>
<td>Pain (MPQ19)</td>
<td>25.05</td>
<td>13.76</td>
<td>24.38</td>
<td>12.78</td>
</tr>
<tr>
<td>Fear</td>
<td>1.43</td>
<td>0.94</td>
<td>1.64</td>
<td>0.93</td>
</tr>
<tr>
<td>Sleep</td>
<td>2.07</td>
<td>0.75</td>
<td>2.21</td>
<td>0.68</td>
</tr>
<tr>
<td>State</td>
<td>49.21</td>
<td>6.54</td>
<td>39.60</td>
<td>11.96</td>
</tr>
<tr>
<td>Trait</td>
<td>43.74</td>
<td>10.17</td>
<td>42.88</td>
<td>8.36</td>
</tr>
<tr>
<td>Depression</td>
<td>46.14</td>
<td>7.43</td>
<td>43.02</td>
<td>5.00</td>
</tr>
<tr>
<td>Stress</td>
<td>12.62</td>
<td>5.11</td>
<td>6.69</td>
<td>3.95</td>
</tr>
</tbody>
</table>

SD=standard deviation (of individuals); Min=minimum; Max=maximum; n=42

At Time 2, pain correlates with fear (0.63) and sleep (0.50); sleep correlates with fear (0.49), stress (0.49), state anxiety (0.45), and trait anxiety (0.49), all at p≤0.001 level of significance.

Changes that occur over time—The measures of pain and of the variables representing a set of processes associated with pain are inspected to look for changes over time. Because the data are not necessarily normally distributed a non-parametric test is required. The results, using the Wilcoxon matched-pairs signed-ranks test, are presented in Table 3.

Table 3. Changes over time: Wilcoxon matched-pairs, signed-ranks test

<table>
<thead>
<tr>
<th>Process</th>
<th>Mean rank</th>
<th>Number of cases</th>
<th>Z-score</th>
<th>2-tailed probability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>negative</td>
<td>positive</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Pain</td>
<td>22.19</td>
<td>17.08</td>
<td>18</td>
<td>20</td>
</tr>
<tr>
<td>Fear</td>
<td>10.13</td>
<td>7.96</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Sleep</td>
<td>5.50</td>
<td>7.00</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>State anxiety</td>
<td>18.12</td>
<td>26.25</td>
<td>33</td>
<td>4</td>
</tr>
<tr>
<td>Trait anxiety</td>
<td>21.50</td>
<td>17.50</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>Depression</td>
<td>24.11</td>
<td>15.62</td>
<td>23</td>
<td>17</td>
</tr>
<tr>
<td>Stress</td>
<td>22.08</td>
<td>7.33</td>
<td>38</td>
<td>3</td>
</tr>
</tbody>
</table>

By inspection of the probabilities, the pain score does not change across the 2 years. By inspection of Tables 2 and 3, both state anxiety and stress reduce significantly over time (p<0.0001). Depression showed a decrease, but with a lower significance level (p=0.052).

Factors of chronic pain—The interactions between the process variables in this study are examined further by using principal component factor analyses. Varimax normalised factor loadings are used. Tables 4 and 5 show that three factors are identified at both Time 1 and Time 2. The statistics and the interpretation of the factors are shown in Table 6.
Table 4. The rotated factor matrix of process variables at Time 1

<table>
<thead>
<tr>
<th>Time 1</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Months</td>
<td>0.826</td>
<td>0.189</td>
<td>-0.118</td>
</tr>
<tr>
<td>Stress</td>
<td>0.795</td>
<td>0.098</td>
<td>0.080</td>
</tr>
<tr>
<td>Zung</td>
<td>-0.011</td>
<td>0.953</td>
<td>0.078</td>
</tr>
<tr>
<td>Trait</td>
<td>0.164</td>
<td>0.888</td>
<td>-0.132</td>
</tr>
<tr>
<td>State</td>
<td>0.223</td>
<td>0.105</td>
<td>-0.668</td>
</tr>
<tr>
<td>Sleep</td>
<td>0.240</td>
<td>0.390</td>
<td>0.610</td>
</tr>
<tr>
<td>MPQ19</td>
<td>0.586</td>
<td>-0.042</td>
<td>0.594</td>
</tr>
<tr>
<td>Fear</td>
<td>0.576</td>
<td>-0.203</td>
<td>0.578</td>
</tr>
</tbody>
</table>

Table 5. The rotated factor matrix of process variables at Time 2

<table>
<thead>
<tr>
<th>Time 2</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>MPQ19</td>
<td>0.857</td>
<td>0.119</td>
<td>0.061</td>
</tr>
<tr>
<td>Fear</td>
<td>0.771</td>
<td>0.194</td>
<td>0.183</td>
</tr>
<tr>
<td>Sleep</td>
<td>0.634</td>
<td>0.347</td>
<td>0.365</td>
</tr>
<tr>
<td>State</td>
<td>0.375</td>
<td>0.850</td>
<td>-0.056</td>
</tr>
<tr>
<td>Trait</td>
<td>0.309</td>
<td>0.822</td>
<td>0.009</td>
</tr>
<tr>
<td>Months</td>
<td>-0.153</td>
<td>0.663</td>
<td>0.516</td>
</tr>
<tr>
<td>Zung</td>
<td>0.114</td>
<td>-0.099</td>
<td>0.789</td>
</tr>
<tr>
<td>Stress</td>
<td>0.284</td>
<td>0.179</td>
<td>0.647</td>
</tr>
</tbody>
</table>

Table 6. Statistics and interpretation of factors at Time 1 and Time 2

<table>
<thead>
<tr>
<th>Time</th>
<th>Label</th>
<th>Factor</th>
<th>Eigen value</th>
<th>% of variance</th>
<th>Cumulative % variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>External</td>
<td>1</td>
<td>2.634</td>
<td>32.9</td>
<td>32.9</td>
</tr>
<tr>
<td></td>
<td>Internal</td>
<td>2</td>
<td>1.889</td>
<td>23.6</td>
<td>56.5</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
<td>3</td>
<td>1.093</td>
<td>13.7</td>
<td>70.2</td>
</tr>
<tr>
<td>2</td>
<td>Pain</td>
<td>1</td>
<td>3.374</td>
<td>42.2</td>
<td>42.2</td>
</tr>
<tr>
<td></td>
<td>Worry</td>
<td>2</td>
<td>1.184</td>
<td>14.8</td>
<td>57.0</td>
</tr>
<tr>
<td></td>
<td>Depression / Stress</td>
<td>3</td>
<td>1.056</td>
<td>13.2</td>
<td>70.2</td>
</tr>
</tbody>
</table>

At both Times 1 and 2, three factors explain 70% of the variance of the scores of the measures of pain, chronicity, and the associated processes. At Time 1 the variance of the measures of pain and fear is shared between the three factors. With the passage of time, there is a sharper focus on the Pain Factor that is comprised of “pain, fear, and sleep.”

Predictors of pain scores—Stepwise multiple regression analyses were used at Time 1 and Time 2 to identify predictors of the pain score as measured by the score derived from the McGill Pain Questionnaire, i.e. the rank-score of the remaining 19 sets of adjectives after the score for the ‘fear’ set of adjectives had been removed. (MPQ19 = MPQRank – Fear).
Table 7. Summary of regression analyses at Time 1 and Time 2

<table>
<thead>
<tr>
<th>Time</th>
<th>Dependent variable</th>
<th>Independent variable</th>
<th>B</th>
<th>R²</th>
<th>F</th>
<th>F sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pain (MPQ19)</td>
<td>Fear Months</td>
<td>8.026</td>
<td>0.088</td>
<td>7.648</td>
<td>0.484</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Constant)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Pain (MPQ19)</td>
<td>Fear Sleep</td>
<td>5.647</td>
<td>6.069</td>
<td>1.560</td>
<td>0.403</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Constant)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 7 shows the results of these analyses for the 42 participants at both Times 1 and 2. The coefficient B is the slope of the least squares line relating each independent variable to the Pain score. R² is the coefficient of determination and measures how well the line fits the data.

These regression equations are derived from Table 7:

**Time 1:** Pain = 8.02*fear + 0.08*months + 7.65

**Time 2:** Pain = 5.65*fear + 6.07*sleep + 1.56

Thus, *fear* is a predictor of *pain* at both Time 1 and Time 2.

**Discussion**

The collection of data within a community-based practice rather than in an institution was a unique feature that would be difficult to replicate because of the many changes within the legislated compensatory system. This is a longitudinal study and there is a paucity of such studies in the literature on a group of sufferers with benign chronic pain in this context. There is no plan to generalise from this group of participants to a wider field. These results show that previously the importance of *fear* as a correlate of chronic *pain* has been masked by the manner of construction of the McGill Pain Questionnaire. Thus, both *pain* and *fear* may contribute to the disability that has been attributed to lasting pain alone; it is this disability that is costly to both the individual and to society. Fear has emerged as the only variable to predict measures of pain at both the start and finish of the 2-year test period.

Three null hypotheses are not supported because there are relationships between the measures of pain and the measures of a set of processes that are associated with chronic pain. There are changes over time within the measures of pain and the measures of a set of processes that are associated with chronic pain. There are changes over time in relationships between the measures of pain and the measures of a set of processes that are associated with chronic pain.

An interpretation of the results of the factor analyses indicates that there is a sharper focus on the pain factor, comprised of pain, fear, and sleep, after 2 years have passed. It could be the case that this focus is a necessary and perhaps a sufficient condition for adaptation to occur.

The scale used to measure sleep in this study was coarse; a sleep-quality instrument more finely tuned to sleep-related problems in patients with pain could be the Medical Outcomes Study (MOS) Sleep Scale. However, the finding on *sleep* is in line with
the results of a Danish multidisciplinary study of pain epidemiology and quality of life in 149 chronic, non-malignant pain patients. Sleep was interrupted by pain in 125 patients (83.9%).

This study of a group of chronic pain sufferers supports the theory that chronic pain can be described as “a dynamic system that includes measures of pain and measures of a set of factors that are associated with pain, together with the mutual relationships (loops) between these processes.” An operational definition of chronic pain is necessary to facilitate the diagnostic process and to ensure appropriate referral and treatment. This study shows the validity of describing chronic pain as a system of pain, fear, and sleep.

A homogenous factor common to all chronic pain sufferers’ situations, sought in the literature, is identified as “the form of dynamic organisation that occurs between measures of pain and measures of a set of processes associated with pain, together with the interactions between these measures.”

Therapeutic intervention, generally, must aim to bring about changes that will break the adverse loops between measures of pain and of other processes, thus establishing a benign, stable system. The essential ingredient of treatment, or intervention, therefore, is “the process of nullifying the mutual interactions between pain and other processes within a set of processes that constitute the chronic pain system.”

Previously, ‘adaptation’ has been defined as decreased anxiety and depression, lower pain severity, and lower somatisation ratings along with higher levels of social activity. Adaptation is re-defined as “a goal of intervention and can be used to describe the situation in which pain exists without any significant relationships (loops) between it and other processes. The chronic pain system will have stabilised in a state of well-being.”

‘Catastrophe’ is a term which describes the antithesis of adaptation. Catastrophe is defined as “the situation in which the measures of each process variable within the chronic pain system are high and adverse loops exist between the pain and other processes.”

‘Catastrophising’ is a term that has been used to refer to negative self-statements. Both coping and catastrophising are terms used by psychologists as if they were dichotomous but in reality they could be applied to points on a continuum, if one were to imagine a line drawn between two points labelled adaptation and catastrophe.

‘Copers’ is a term that can be applied to those people with lasting pain for whom loops between measures of pain and other process variables have been broken, or for whom the values of process variables have been reduced to a point where interactions between them are negligible. On the other hand, ‘catastrophisers’ can be the term used to describe those people for whom there are pronounced or significant adverse loops between measures of pain and many of the set of other process variables within a defined chronic pain system.

In conclusion, viewing benign chronic pain as a system clarifies its complexity and provides a model of understanding on which to base interventions. This is necessary to maximise the attainment of the desired goals of treatment, adaptation and rehabilitation.
Competing interests: None known.

Author information: Dr Margaret Moon practised in New Zealand as a physiotherapist and a psychologist. Since retirement she has worked as a volunteer in Vanuatu, India, and with the Tibetan Government-in-exile in Northern India. She is the author of “Pain Poppies” (ISBN 0-473-09342-1).

Mr Brian Niven, MSc, is a statistical consultant, working with students and staff from a wide range of disciplines at the University of Otago, Dunedin, New Zealand. His interests include general applied statistics and statistical computing.

Acknowledgements: The doctoral dissertation on which this paper is based was supervised by Professor Gareth Jones and Dr Susan Mercer of the Department of Anatomy & Structural Biology, and by Professor Vernon Squire of the Department of Mathematics & Statistics. This excellent supervision is fully acknowledged.

Correspondence: Dr Margaret Moon, c/o Professor Gareth Jones, Department of Anatomy & Structural Biology, University of Otago, PO Box 56, Dunedin, New Zealand. Email: mhmoon@es.co.nz

References:

Diabetes eye screening in the Wellington region of New Zealand: characteristics of the enrolled population (2002–2005)

Lesley G Frederikson, Robert J Jacobs

Abstract

Aims To profile the distribution of people screened across the diagnostic grades for retinopathy and to further investigate the relationship between retinopathy and other clinical signs such as maculopathy, HbA1c, and visual acuity.

Methods The study used all records of first screening visits by 11,977 people from 2002–2005 in a longitudinal cohort design.

Results The majority of people (68%) enrolled in the screening programme had no retinopathy in either eye; 20% had minimal NPDR; 8% had mild NPDR; 2% had moderate NPDR; and 0.5% had severe NPDR. Only 28 people (0.2%) had proliferative retinopathy. Maculopathy was detected in 1429 cases (12.3%). There was a significant relationship between the progression of retinopathy and incidence of maculopathy (Chi-squared=3642.77; p<0.01). The mean log(MAR) value for best visual acuity in the eye with worse retinopathy showed there was an overall reduction in visual acuity as retinopathy progressed. Higher levels of HbA1c were associated with more advanced progression of NPDR (Chi-squared= 389.266; p<0.01) and the presence of maculopathy (Chi-squared= 147.056; p<0.01). New Zealand Māori were under-represented in the screened population while Pacific Island (PI) people appeared to be well represented.

Conclusion The Wellington regional retinal screening programme for people with diabetes managed by the Wellington Independent Practice Association (WIPA) provides a quality service accessed by a high proportion of the people in the region having diabetes (92%). Two key strengths of the programme are its routine collection of comprehensive information and the links between general practice, optometry, and ophthalmology.

Visual impairment is a complication of diabetes which can be reduced through regular screening and timely treatment. Prevalence of known diabetes in New Zealand (NZ) is close to 3% of all Europeans and rates for Māori, Pacific Island, and Asian people are around three times higher than this.1

Prior to 2002, diabetes eye screening in the greater Wellington region of NZ (with a catchment of around 300,000 people) was offered in only a single location at the Wellington Hospital Eye Department. Approximately 400 people were screened per year. There were concerns among the general practitioners of the region that the Get Checked programme (free annual checks for people with diabetes) required regular eye screens but the availability of eye screening was limited.
During 2001, a joint project was undertaken by the NZ Association of Optometrists and the Wellington Independent Practitioners Association (WIPA, a general practice network) to develop a programme specification for optometry-based eye screening in the Wellington region. The programme was successfully launched at the end of 2001 with optometrists providing eye screening services for people with diabetes from five locations around the region—this was subsequently expanded to 12 locations by 2007.

Optometrists providing the service assessed the condition of the retina, detected coexisting eye pathology, and graded and reported clinical findings. Referrals were made to ophthalmology services when appropriate and all findings were reported back to the referring GP. There was an ongoing quality assurance process involving peer review and ophthalmology oversight.

The primary screening method was non-mydriatic digital retinal photography with the optometrist required to use mydriasis if necessary to obtain a satisfactory photograph of the fundus and to examine by ophthalmoscopy (direct and indirect methods) if photo quality was inadequate. The quality of the fundus view was recorded. A standardised retinopathy grading scale was adopted by the designated ophthalmologist for the programme and this was used to classify the findings.

The programme requested that the optometrist record latest HbA1c and cholesterol findings at the time of eye screening visit. These values were from the patient’s report or from the GP practice referral. Studies such as the UK Prospective Diabetes Study, the Oslo Study and the Diabetes, Control and Complications Trial have shown that long-term control of blood glucose is associated with decreased risk of retinopathy progression. However, previous studies indicate that wide variation in HbA1c levels exist among people with diabetes and tight glycaemic control is not always maintained.

From early on in its delivery, the community-based programme appeared to be providing tangible benefits. The number of people being screened in the community was greater than that achieved in the hospital setting, and the rates of non-attendance (DNAs) fell to an average of 7%. Anecdotal reports from the screening optometrists suggested that variations to the screening intervals could be used to increase efficiency of screening. This led to the current study which looks at the distribution of people screened across the diagnostic grades for retinopathy and seeks to further investigate the relationship between retinopathy and other clinical signs such as maculopathy, HbA1c, and visual acuity.

Methods

Research design—After ethics approval was obtained, the study used all records of screening visits from 2002–2005 in a longitudinal cohort design. Data included 16,676 records. These records contained the results of first visits and subsequent screens for a group of 11,977 people. The Ministry of Health estimated that there were 12,909 people with diagnosed diabetes in the Wellington region as at 2005.

Analysis of the first-visit data was undertaken to profile the participants in the screening programme and to determine cross-sectional relationships between years since diagnosis, HbA1c, and the extent of diabetic retinopathy. The study also explored the prevalence of maculopathy, the level of comorbidity between retinopathy and maculopathy, and the effects of each on visual acuity.

Data and variables—Each record set included information on: HbA1c, screening method, use of mydriatic, quality of screen (left and right), retinopathy grading (left and right), presence of...
maculopathy (left and right), pinhole acuity (left and right), habitual acuity (left and right), screening location, and time to next screen. Cholesterol levels were not available in the dataset.

Acuity measures were recorded in the format 6/5 to 6/120 and converted to logMAR using the formula log(size read/test distance).

Retinopathy grading criteria for non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR) were as follows:

**Minimal NPDR**
Isolated microaneurysms are present.

**Mild NPDR**
Microaneurysms and retinal haemorrhages are both present.

**Moderate NPDR**
Haemorrhages and microaneurysms are present in at least 1 quadrant and cotton wool spots or venous beading occur in 1 quadrant only.

**Severe NPDR**
One or more of the following:
- Intraretinal microvascular abnormalities in 1 or more quadrants.
- Venous beading in 2 or more quadrants.
- Haemorrhages / microaneurysms in all 4 quadrants.

**PDR**
One or more of the following:
- Peripheral new vessels (NVE).
- New vessels at the disc (NVD).
- Vitreous or preretinal haemorrhage with peripheral NVE.

For each case the grading for the worse eye was used as an indicator of disease progression. Best achievable vision was defined as the best acuity recorded for either pinhole acuity or habitual acuity.

SPSS (version 14) software was used for all statistical analyses.

**Results**

**Demographics**—The sample of 11,977 cases consisted of 6213 men and 5734 women with no sex reported for 30 cases. The majority recorded ethnicity as NZ European (60%); other recorded ethnicities were NZ Māori (11%), Samoan (7%), Cook Island Māori (2%), Tongan (0.7%), Niuean (0.1%), Chinese (3%), Indian (6%), and Other (11%). Ethnicity was not recorded in 558 (4.7%) of the cases.

Age at first screen date ranged from 7 to 100 years (mean±SD: 59.8±14.3; median: 61; interquartile range: 19). Age at diagnosis ranged from birth to 94 years (54.1±15.4; 55; 20). The time in years between diagnosis and screening ranged from 0 (first screen in year diagnosed) to 77 (5.7±7.0; 3; 7).

The sample was predominantly people with Type 2 diabetes (10,865; 91%) but also included people with gestational diabetes (14; 0.1%), and Type 1 diabetes (967; 8%). There were 131 records with no diabetes type noted (1%).

Screeners used a mydriatic to dilate the pupil in 4840 cases (40% of cases) and almost all of the participants (11,137; 93%) were screened using digital photography. Clinical examination was used for 808 people (6.7%) with 19 (0.2%) having digital photos taken plus a clinical exam. Analogue film was used for 13 people (0.1%).

The quality of the screen was good or adequate in 92.5% of the eyes screened (both left and right) with 3.6% of views being inadequate due to cataract. A further 2.6% of views were inadequate for other reasons and 1.2% of eyes were recorded as not screened. Eyes that had previously had treatment were not screened.

**Retinopathy grading**—Three-quarters (75%) of the eyes (left and right) had no signs of retinopathy (Table 1) and a further 16% of eyes were classified as having minimal non-proliferative diabetic retinopathy (NPDR). Proliferative diabetic retinopathy (PDR) was detected in only 0.1% of the eyes. From a total of 23,954 eyes screened, 439 were not graded—a failure rate of only 1.8%.
Table 1. Retinopathy grading

<table>
<thead>
<tr>
<th>Variables</th>
<th>Left eye number</th>
<th>Left eye (%)</th>
<th>Right eye number</th>
<th>Right eye (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Retinopathy</td>
<td>8839</td>
<td>75.3</td>
<td>8818</td>
<td>74.9</td>
</tr>
<tr>
<td>Minimal NPDR</td>
<td>1933</td>
<td>16.5</td>
<td>1924</td>
<td>16.3</td>
</tr>
<tr>
<td>Mild NPDR</td>
<td>707</td>
<td>6.0</td>
<td>757</td>
<td>6.4</td>
</tr>
<tr>
<td>Moderate NPDR</td>
<td>207</td>
<td>1.8</td>
<td>207</td>
<td>1.8</td>
</tr>
<tr>
<td>Severe NPDR</td>
<td>43</td>
<td>0.4</td>
<td>47</td>
<td>0.4</td>
</tr>
<tr>
<td>PDR</td>
<td>17</td>
<td>0.1</td>
<td>16</td>
<td>0.1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>11746</strong></td>
<td><strong>100.0</strong></td>
<td><strong>11769</strong></td>
<td><strong>100.0</strong></td>
</tr>
<tr>
<td><strong>Missing</strong></td>
<td><strong>231</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Disease progression—For 8157 people (68%) enrolled in the screening programme there was no retinopathy in either eye. A group of 2438 (20%) had minimal NPDR in the worse eye; 930 (8%) had mild NPDR; 270 (2%) had moderate NPDR; and 56 (0.5%) had severe NPDR. Only 28 people out of 11879 (0.2%) had proliferative retinopathy (Figure 1).

Figure 1. Progression of diabetic eye disease in those screened
Maculopathy—Maculopathy was detected in 1429 cases (12.3%) with 10,208 people screened clear for maculopathy at the first screen (Table 2). This can be compared to 8157 having no retinopathy.

Table 2. Maculopathy

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>10,208</td>
<td>87.7</td>
</tr>
<tr>
<td>Left only</td>
<td>368</td>
<td>3.2</td>
</tr>
<tr>
<td>Right only</td>
<td>454</td>
<td>3.9</td>
</tr>
<tr>
<td>Both eyes</td>
<td>607</td>
<td>5.2</td>
</tr>
<tr>
<td>Total</td>
<td>11,637</td>
<td>100.0</td>
</tr>
<tr>
<td>Missing</td>
<td>340</td>
<td>2.5</td>
</tr>
</tbody>
</table>

To explore the overall relationship between development of retinopathy and development of maculopathy we looked at the numbers of people within each grade of retinopathy who also had maculopathy (Table 3).

Table 3. Maculopathy by retinopathy grade

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total number in group</th>
<th>Number with no maculopathy</th>
<th>Number with some maculopathy</th>
<th>Percentage of group with maculopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Retinopathy</td>
<td>8021</td>
<td>7821</td>
<td>200</td>
<td>2.5</td>
</tr>
<tr>
<td>Minimal NPDR</td>
<td>2385</td>
<td>1908</td>
<td>477</td>
<td>20.0</td>
</tr>
<tr>
<td>Mild NPDR</td>
<td>889</td>
<td>389</td>
<td>500</td>
<td>56.2</td>
</tr>
<tr>
<td>Moderate NPDR</td>
<td>259</td>
<td>73</td>
<td>186</td>
<td>71.8</td>
</tr>
<tr>
<td>Severe NPDR</td>
<td>56</td>
<td>8</td>
<td>48</td>
<td>85.7</td>
</tr>
<tr>
<td>PDR</td>
<td>26</td>
<td>9</td>
<td>17</td>
<td>65.4</td>
</tr>
<tr>
<td>Total</td>
<td>11,636</td>
<td>10,208</td>
<td>1428</td>
<td>100.0</td>
</tr>
<tr>
<td>Missing</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

A Chi-squared test of the association between maculopathy and retinopathy showed there was a significant relationship between the progression of a person’s retinopathy and the likelihood of them developing some degree of maculopathy (Chi-squared=3642.77; p<0.01). Phi and Cramer’s V measures of symmetry were both equal at 0.560 and with a contingency coefficient of 0.488 all three measures are significant at the 0.01 level. The size of these measures indicates the association between the variables was relatively strong.

To explore the relationship between development of retinopathy and development of maculopathy within the same eye we looked at cases which had both retinopathy and maculopathy to see if the maculopathy was more likely to occur in the worse eye (Table 4).
Table 4. Cross-tabulation of maculopathy by retinopathy in the worse eye

<table>
<thead>
<tr>
<th>Retinopathy</th>
<th>Maculopathy</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left only</td>
<td>Right only</td>
<td>Both</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Worse Eye</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left (expected)*</td>
<td>196 (74)</td>
<td>30 (92)</td>
<td>63 (123)</td>
<td>289</td>
<td></td>
</tr>
<tr>
<td>Right (expected)</td>
<td>19 (85)</td>
<td>228 (105)</td>
<td>84 (140)</td>
<td>331</td>
<td></td>
</tr>
<tr>
<td>Same (expected)</td>
<td>101 (157)</td>
<td>133 (194)</td>
<td>374 (258)</td>
<td>608</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>316 (316)</td>
<td>391 (391)</td>
<td>521 (521)</td>
<td>1228</td>
<td></td>
</tr>
</tbody>
</table>

(*Expected numbers in brackets are the number of cases expected to occur in each cell if there was no association between the variables.)

The Chi-squared test showed that where maculopathy and retinopathy were both present the maculopathy was significantly more likely to occur in the eye with the worse retinopathy (Chi-squared=577.27, p<0.01; Phi=0.686, p<0.01; Cramer’s V=0.485, p<0.01; contingency coefficient=0.565, p<0.01).

There was a significant main effect for retinopathy grade (F=76.798; p<0.01). However, there was wide variation within the groups and some of those with no retinopathy had acuities of 6/120. Similarly, some of the eyes with severe NPDR and proliferative retinopathy were achieving 6/5 vision.

Analysis also showed a significant interaction between retinopathy and maculopathy (F = 25.820; p < 0.01). Mean log(MAR) values for best visual acuity tended to be greater within each group when maculopathy was present in the worse eye indicating poorer visual performance (Table 7). However, these results must be interpreted cautiously as there was again wide variation within the groups and some of the groups had very few participants included.

Measures of visual acuity—The median for best achievable vision (letter acuity) was 6/7.5 and the mode was 6/6 (Table 5).

Table 5. Best achievable vision

<table>
<thead>
<tr>
<th>Acuity</th>
<th>Number</th>
<th>Valid percent</th>
<th>Cumulative percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/5</td>
<td>1874</td>
<td>17.6</td>
<td>17.6</td>
</tr>
<tr>
<td>6/6</td>
<td>3278</td>
<td>30.9</td>
<td>48.5</td>
</tr>
<tr>
<td>6/7.5</td>
<td>1940</td>
<td>18.3</td>
<td>66.8</td>
</tr>
<tr>
<td>6/9</td>
<td>2124</td>
<td>20.0</td>
<td>86.8</td>
</tr>
<tr>
<td>6/12</td>
<td>926</td>
<td>8.7</td>
<td>95.5</td>
</tr>
<tr>
<td>6/18</td>
<td>297</td>
<td>2.8</td>
<td>98.3</td>
</tr>
<tr>
<td>6/24</td>
<td>80</td>
<td>0.8</td>
<td>99.0</td>
</tr>
<tr>
<td>6/36</td>
<td>48</td>
<td>0.5</td>
<td>99.5</td>
</tr>
<tr>
<td>6/60</td>
<td>21</td>
<td>0.2</td>
<td>99.7</td>
</tr>
<tr>
<td>6/120</td>
<td>32</td>
<td>0.3</td>
<td>100.0</td>
</tr>
<tr>
<td>Total</td>
<td>10620</td>
<td>100.0</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>245</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The mean log(MAR) value for best visual acuity in the eye with worse retinopathy increased from 0.083 log minutes of arc in the group with no retinopathy to 0.29 log
minutes of arc in the group with proliferative retinopathy reflecting an overall reduction in visual acuity as retinopathy progresses (Table 6).

Table 6. Comparison of best achievable vision across retinopathy groups using log(MAR) values

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number</th>
<th>Mean log(MAR)</th>
<th>Standard Deviation</th>
<th>Standard Error</th>
<th>95% Confidence Interval for Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Retinopathy</td>
<td>8154</td>
<td>.0834</td>
<td>.16283</td>
<td>.00180</td>
<td>.0799 – .0869</td>
</tr>
<tr>
<td>Minimal NPDR</td>
<td>2437</td>
<td>.1151</td>
<td>.18707</td>
<td>.00379</td>
<td>.1076 – .1225</td>
</tr>
<tr>
<td>Mild NPDR</td>
<td>930</td>
<td>.1703</td>
<td>.24083</td>
<td>.00790</td>
<td>.1548 – .1858</td>
</tr>
<tr>
<td>Moderate NPDR</td>
<td>270</td>
<td>.1996</td>
<td>.25279</td>
<td>.01538</td>
<td>.1693 – .2299</td>
</tr>
<tr>
<td>Severe NPDR</td>
<td>56</td>
<td>.2513</td>
<td>.30488</td>
<td>.04074</td>
<td>.1697 – .3330</td>
</tr>
<tr>
<td>PDR</td>
<td>28</td>
<td>.2867</td>
<td>.35996</td>
<td>.06803</td>
<td>.1472 – .4263</td>
</tr>
<tr>
<td>Total</td>
<td>11875</td>
<td>.1006</td>
<td>.18202</td>
<td>.00167</td>
<td>.0974 – .1039</td>
</tr>
</tbody>
</table>

NPDR=Non-proliferative diabetic retinopathy; PDR=Proliferative diabetic retinopathy.

Table 7. Mean log(MAR) value for groups with and without maculopathy

<table>
<thead>
<tr>
<th>Grade in worse eye</th>
<th>Maculopathy</th>
<th>Mean log(MAR)</th>
<th>Standard Deviation</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>No retinopathy</td>
<td>None</td>
<td>0.0772</td>
<td>0.15331</td>
<td>7819</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.2786</td>
<td>0.26725</td>
<td>92</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.0796</td>
<td>0.15659</td>
<td>7911</td>
</tr>
<tr>
<td>Minimal NPDR</td>
<td>None</td>
<td>0.1156</td>
<td>0.17516</td>
<td>1907</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.0948</td>
<td>0.19782</td>
<td>318</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.1127</td>
<td>0.17867</td>
<td>2225</td>
</tr>
<tr>
<td>Mild NPDR</td>
<td>None</td>
<td>0.1671</td>
<td>0.22369</td>
<td>389</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.1761</td>
<td>0.26415</td>
<td>318</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.1711</td>
<td>0.24259</td>
<td>707</td>
</tr>
<tr>
<td>Moderate NPDR</td>
<td>None</td>
<td>0.1407</td>
<td>0.17255</td>
<td>73</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.2189</td>
<td>0.27856</td>
<td>120</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.1893</td>
<td>0.24638</td>
<td>193</td>
</tr>
<tr>
<td>Severe NPDR</td>
<td>None</td>
<td>0.1081</td>
<td>0.11564</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.2891</td>
<td>0.33248</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.2529</td>
<td>0.30926</td>
<td>40</td>
</tr>
<tr>
<td>PDR</td>
<td>None</td>
<td>0.2438</td>
<td>0.43720</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.2074</td>
<td>0.19518</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.2246</td>
<td>0.32303</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>None</td>
<td>0.0884</td>
<td>0.16286</td>
<td>10205</td>
</tr>
<tr>
<td></td>
<td>Some</td>
<td>0.1678</td>
<td>0.25470</td>
<td>890</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>0.0948</td>
<td>0.17337</td>
<td>11095</td>
</tr>
</tbody>
</table>

Blood glucose measures—HbA1c values ranged from 2.0 to 16.7 with a mean of 7.56 (standard error=0.017) and a median value of 7.2 (Figure 2).
The median of 7.2 was taken as the value at which to define adequate control of diabetes within the study group as it had the advantage of splitting the group around its midpoint and was consistent with the use of the non-parametric median test of HbA1c control across the groups for retinopathy grade in the worse eye.

The proportion of controlled cases (HbA1c ≤ 7.2) in each grade decreases from No Retinopathy (58.4% controlled), Minimal NPDR (48.5%), Mild NPDR (31.6%), Moderate NPDR (22.7%), to Severe NPDR (16%). Of the cases with PDR, 48% had HbA1c ≤ 7.2.

Table 8 shows the numbers of cases at each retinopathy grade with HbA1c above the overall median (7.2) or equal to and less than the overall median (controlled). The final row shows the mean HbA1c for each separate group.
Table 8. Numbers of controlled cases at each level of retinopathy progression

<table>
<thead>
<tr>
<th>HbA1c</th>
<th>Retinopathy grade: worse eye</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No retinopathy</td>
</tr>
<tr>
<td>Controlled ≤7.2</td>
<td>3985</td>
</tr>
<tr>
<td>&gt;7.2</td>
<td>2834</td>
</tr>
<tr>
<td>Group mean HbA1c</td>
<td>7.4</td>
</tr>
</tbody>
</table>

NPDR=Non-proliferative diabetic retinopathy; PDR=Proliferative diabetic retinopathy.

The median test yielded a Chi-squared statistic of 389.266 (p<0.01) indicating that significantly different HbA1c levels were associated with the different levels of disease progression. The group means for HbA1c level increased with increasing severity of non proliferative retinopathy but the trend did not continue for the people with proliferative retinopathy who were present in relatively low numbers in the screening cohort.

Table 9. Numbers of controlled cases with and without maculopathy

<table>
<thead>
<tr>
<th>HbA1c</th>
<th>Maculopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No maculopathy</td>
</tr>
<tr>
<td>Controlled ≤7.2</td>
<td>4698</td>
</tr>
<tr>
<td>&gt;7.2</td>
<td>3854</td>
</tr>
<tr>
<td>Group mean HbA1c</td>
<td>7.5</td>
</tr>
</tbody>
</table>

The analysis was repeated for cases grouped by presence of maculopathy with a Chi-squared statistic of 147.056 (p<0.01) indicating that higher HbA1c levels were also associated with the presence of maculopathy. There were significantly more cases with HbA1c above the overall median (uncontrolled) than below in the group with maculopathy present (Table 9). In contrast, the majority of cases in the group with no maculopathy have HbA1c levels at or below the overall median (controlled).

Years since diagnosis—The mean time from diagnosis to screen for the entire sample was 5.7 years and the overall median was 3 years.

The retinopathy grading groups differed significantly on years since diagnosis (Chisquared=644.994, p<0.01) with longer time since diagnosis associated with more severe retinopathy (Table 10).
Table 10. Median test of years since diagnosis for grades of retinopathy

<table>
<thead>
<tr>
<th>Years since diagnosis</th>
<th>No Retinopathy</th>
<th>Minimal NPDR</th>
<th>Mild NPDR</th>
<th>Moderate NPDR</th>
<th>Severe NPDR</th>
<th>PDR</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤3</td>
<td>4711</td>
<td>945</td>
<td>229</td>
<td>73</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>&gt;3</td>
<td>3375</td>
<td>1476</td>
<td>693</td>
<td>196</td>
<td>43</td>
<td>19</td>
</tr>
<tr>
<td>Group mean years since diagnosis</td>
<td>4.4</td>
<td>7.5</td>
<td>10.9</td>
<td>10.1</td>
<td>10.5</td>
<td>10.8</td>
</tr>
</tbody>
</table>

Ethnicity—The ethnic make up of the sample group was compared to the regional population from the 2006 census,8 and the sample distribution of this study reflected the ethnic distribution within the population of the Wellington Region. However, the prevalence of known diabetes in Māori and Pacific peoples is more than twice as high as among NZ Europeans, suggesting these groups are under-represented in the screened population for the Wellington region. Compared with known diabetes estimates Māori were under-represented in the screened population while Pacific Island people appeared to be well represented (Table 11).

Table 11. Ethnicity of screening participants compared to the regional population

<table>
<thead>
<tr>
<th>Variables</th>
<th>Screening numbers</th>
<th>Screening %</th>
<th>2006 Census regional %</th>
<th>Expected percentage of people with known diabetes (2005)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NZ European</td>
<td>6886</td>
<td>60</td>
<td>63</td>
<td>18.1</td>
</tr>
<tr>
<td>NZ Māori</td>
<td>1257</td>
<td>11</td>
<td>11</td>
<td>10.3</td>
</tr>
<tr>
<td>Pacific Islander (PI)*</td>
<td>1050</td>
<td>9</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>948</td>
<td>8</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>1276</td>
<td>11</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>11,417</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

A mean grade for retinopathy was calculated using an ordinal scale from 0 for No Retinopathy to 5 for PDR to provide an indication of the comparative burden of retinopathy occurring within each ethnic group (Figure 3).

The NZ European mean grade was close to No Retinopathy, with NZ Māori just a little higher. For Samoan and Tongan people the mean grade was closer to Minimal NPDR.
The results of one way ANOVA indicated there were significant differences among the means for the different ethnic groups (F=12.71; p<0.01).

**Table 12. Presence of maculopathy according to participant ethnicity**

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Frequency</th>
<th>Group Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>NZ European</td>
<td>787</td>
<td>12</td>
</tr>
<tr>
<td>NZ Māori</td>
<td>137</td>
<td>11</td>
</tr>
<tr>
<td>Samoan</td>
<td>125</td>
<td>16</td>
</tr>
<tr>
<td>Cook Island Māori</td>
<td>22</td>
<td>14</td>
</tr>
<tr>
<td>Tongan</td>
<td>14</td>
<td>19</td>
</tr>
<tr>
<td>Niuean</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Chinese</td>
<td>33</td>
<td>11</td>
</tr>
<tr>
<td>Indian</td>
<td>84</td>
<td>13</td>
</tr>
<tr>
<td>Other</td>
<td>180</td>
<td>15</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>1382</strong></td>
<td><strong>12</strong></td>
</tr>
</tbody>
</table>

There were also differences in the proportions of people with maculopathy across the ethnic groups (Table 12). The Niuean group with only 13 people had no maculopathy even in those with Minimal NPDR (n=4) and Mild NPDR (n=2) but because of the
small numbers this result is reported for completeness only. The other Pacific Island groups showed a higher proportion of cases with maculopathy compared to NZ European and Māori.

Discussion

Two key aspects of screening for diabetic eye disease are coverage and quality. The Waikato mobile screening service covered 79% of the estimated 1996 diabetic population for at least one screen.9 The current research indicates that in its first 3 years the Wellington programme reached 92% of the expected number of people with diagnosed diabetes in the region. The breakdown of participation by ethnicity, indicates that Pacific Island peoples are reasonably represented in the screening group (based on expected percentage of all people with known diabetes) while NZ Māori are under-represented. NZ Europeans and other groups appear to be participating at an expected level. This raises questions about why some Māori people in the region are not accessing the service.

The proportion of non-assessed ocular fundi among the Wellington cohort was 1.8%. This compares very favourably with the 6.2% non-assessable images reported from the Waikato mobile programme9 and falls well below the maximum failure rate of 5% recommended by the British Diabetic Association.10 This level of actively graded eyes is achieved in the Wellington programme because the screening service is delivered by optometrists who are able to dilate and view the fundus at the screening visit in cases where the photograph is inadequate due to either cataract or technical failure.

Due to the varying classification systems used in prior research9,11,12 it is hard to compare the prevalence for particular levels of retinopathy but the present results indicate that in the Wellington region 32% of the screened group have some diabetic retinopathy and this is likely to reflect the actual incidence in the population. This is similar to the findings in Newcastle, Australia, where 35% of the diabetic participants has some evidence of retinopathy12 but is higher than the Waikato9 findings of 22% (those not included in the 78% with no retinopathy).

Measures of best achievable visual acuity in the present study showed an overall reduction in visual acuity as retinopathy increases and analysis also showed a significant interaction effect for retinopathy grade and maculopathy. There have been very few studies of impairments in visual acuity related to diabetic eye disease but Bailey and Sparrow13 reported high levels of visual symptomatology for patients with sight-threatening diabetic retinopathy in the UK and Voutilainen-Kaunisto and colleagues14 recorded a marked deterioration in best-corrected visual acuity over 10 years in Type 2 diabetic patients with maculopathy in Finland.

Overall, 12.3% of the Wellington group had some evidence of maculopathy and this also is consistent with previous findings. The frequency of maculopathy among a group of Finnish people with diabetes increased from 3.4% at diagnosis to 21% after 10 years14 and Lopes de Faria et al15 report 78% of participants with diabetic retinopathy also had macular oedema. In the present study, 51.5% of participants with retinopathy also had maculopathy plus they were significantly more likely to have maculopathy in the eye with the worse retinopathy grading.
The present study also found a significant association between the development of retinopathy and the incidence of maculopathy although this was most likely due to the effects of increasing HbA1c levels.\textsuperscript{14,16} The median test analyses supported this conclusion as increasing HbA1c levels were associated with worsening levels of retinopathy grading and a similar association was shown between increasing HbA1c level and the presence of maculopathy.

The findings that retinopathy grading is related to both HbA1c levels and years since diagnosis confirmed that both these factors are related to the development of retinopathy and comes as no surprise. The role of poor glycaemic control in predicting the progression of diabetic retinopathy has already been widely reported as has the effect of time since diagnosis. However, the variation within groups and the large number of people with advanced retinopathy within 3 years of diagnosis highlights the inadequacy of a focus on only these two factors and points to the need for further research to more accurately determine level of risk and refine recommendations for screening intervals based on risk of developing sight-threatening diabetic eye disease.

Another area that warrants further investigation is the finding from the present study that there are significant differences in mean retinopathy grading among the different ethnic groups. This finding is must be interpreted with caution as the retinopathy scale is ordinal and thus may not fully reflect the increase in severity of disease through progression from one grade of retinopathy to the next. There were also some indications that different ethnic groups may have a higher proportion of people with maculopathy as Pacific Island groups tended to have a higher proportion of people with maculopathy compared to all other groups.

In conclusion, the Wellington regional retinal screening programme for people with diabetes managed by WIPA provides a quality service accessed by a high proportion of the people in the region having diabetes. Two key strengths of the programme are its routine collection of comprehensive information and the links between general practice, optometry and ophthalmology. The sharing of information enables better understanding of the development and progression of diabetic eye disease together with knowledge of visual impairment and measures for key risk factors.

The programme shows there is a clear advantage to having an optometrist available to examine eyes in cases where a photographic view is inadequate as the programme has a very low percentage of grading failures (1.8%). The GP/optometry partnership is also important in respect of the association between impairment in visual performance and progression of both retinopathy and maculopathy. Making the best of available vision is a key aim when the retina is compromised and the programme could be extended to include a diabetes education and motivation role for optometrists supporting the efforts of general practice.

Competing interests: None known.

Author information: Lesley G Frederikson, National Director, NZ Association of Optometrists, Wellington; Robert J Jacobs, Associate Professor, Department of Optometry and Vision Science, The University of Auckland, Auckland

Correspondence: Dr Lesley Frederikson, National Director, NZ Association of Optometrists, PO Box 1978, Wellington, New Zealand. Fax +64 (0)4 4732322; email director@nzao.co.nz
References:


7. Correspondence from Dr S Dawson, Chief Clinical Advisor, Ministry of Health, 2007.


Retention of patients in the Get Checked free annual diabetes review programme in New Zealand

Grace Joshy, Ross A Lawrenson, David Simmons

Abstract

Aims To characterise the retention of patients in the Get Checked free annual diabetes review programme in the Waikato region of New Zealand.

Methods Retrospective review of Waikato Primary Health (WPH) registered patients who had at least one Get Checked review between 1 July 2000 and 30 Jun 2006.

Results 10,919 patients (69% Europeans, 18% Māori, 3% Pacific Islanders, and 4% Asian) had an initial review during the 5 years of this programme. In 2005/06, only 6100 (57%) of the estimated 10,600 diabetes patients enrolled with WPH utilised the free check. Younger patients aged <40 years, those of Māori or Asian origin, and those with Type 1 diabetes were less likely to be retained in the programme with regular checks, as indicated by their longer time to second review and lesser likelihood of return for a second or subsequent review.

Conclusions Despite the programme being fully funded, a significant proportion of patients did not return for a second review within 1.5 years after initial review. The loss of those with Type 1 diabetes and younger patients may reflect their greater contact with secondary care rather than GP services. Excess drop out among ethnic minorities need further investigation and intervention. Use of these data for policy purposes could be significantly biased unless there is a single reliable regional diabetes register based on the National Health Index number including all known patients.

The rising diabetes epidemic is a major problem in New Zealand, especially among the non-European ethnic groups. The cost of Type 2 diabetes alone is expected to reach NZD1.6 billion by year 2021. Many of those with diabetes face a multitude of barriers to quality diabetes care and self care, including financial barriers. Structured care has been shown to improve patient care and outcomes. One strategy used elsewhere to ensure that each person with diabetes received regular structured assessment has been the annual diabetes review.

The Get Checked free annual diabetes review programme in New Zealand—a Ministry of Health initiative which started in June 2000—was established to address both the need for structured care and to help overcome personal expenses as a barrier to diabetes care. This national programme is delivered free of charge to diabetes patients through primary care services.

The review form collects data on whether the following checks or tests were either done or booked to be done: retinal screening (within the last 2 years), foot check, blood pressure, HBA1c, cholesterol, height, bodyweight, and kidney function in the last 12 months. Available test results are also collected.
Patients are expected to return for review every year. Nationally, the percentage of people with diabetes enrolled in the Get Checked programme increased from 33% in 2001 to 59% in 2005, but still the figures are sub-optimal, especially for Māori.\textsuperscript{12,13} The data purportedly provide insight into diabetes care across New Zealand,\textsuperscript{14,15} but we have been concerned about drop out from the programme and the problem of interpreting data through the use of repeated cross sectional rather than longitudinal analysis. We therefore aimed to investigate the patient retention in this programme using data from the local primary healthcare organisation.

**Methods**

Waikato Primary Health (WPH) is the largest primary health organisation in Waikato area with a registered population of 294,510 in 2006. It covers 90\% of the 328,510 Waikato District Health Board population enrolled with a primary health organisation (PHO),\textsuperscript{16,17} with an estimated 10,604 people diagnosed with diabetes.\textsuperscript{18}

This research is a retrospective review of WPH registered patients who had at least one Get Checked review between 1 July 2000 and 30 Jun 2006, using the demographic variables from the Get Checked database (age, gender, latest recorded ethnicity, and type of diabetes). Year of diagnosis of diabetes was not available for analysis.

Mortality data were obtained from the Ministry of Health and were linked to WPH patient register using the national health index numbers. Some patients left WPH after their initial review and were not available for further reviews. The WPH registrations were recorded on a quarterly basis for each patient.

Survival analysis was employed to analyse the time to second review from the initial review. In order to look at continued participation beyond the second review, time to third review from second review, time to fourth review from third, and time to fifth review from fourth were also analysed in a similar fashion.

Those who died or left WPH before a second review and were considered “censored” for the analysis of time to second review. Those who did not return for a second review during follow-up time were censored on 30 June 2006. A similar approach to censoring was also used for the analysis of time to subsequent reviews. For censored patients, the time to event was the start date to date of death, migration date or end of follow-up, which ever was the earliest. Migration date was defined as 45 days after the last quarter of registration with WPH.

Kaplan-Meier survival curves for time to reviews are presented. Allowing a 6-month window to the ideal 1-year of interval between reviews, review rates at 1.5 years were examined. Survival curves for time to second review are presented by ethnicity, age group at first review, gender, and type of diabetes.

Odds ratios for the likelihood of a second review were estimated using Cox’s proportional hazard model. Potential predictors were identified by running a series of regression analyses. Ethnicity, age group at first review, gender, and diabetes type were included as predictors in a Cox’s regression analysis. Proportionality assumption was verified by testing the correlation between Schoenfeld’s residuals for a particular covariate and individual failure times. All statistical analyses were performed using SAS\textsuperscript{®} version 9.1 software (SAS Institute, Cary, NC, USA).

**Results**

A total of 10,919 patients were reviewed at least once during the 5-year period (Table 1). Ethnicity was recorded for 95\% of patients, showing 69\% Europeans, 18\% Māori, 3\% Pacific Islanders, and 4\% Asians.

Of the reviewed patients 87\% had Type 2 diabetes, 8\% had Type 1 diabetes, and 5\% had other or unclassified diabetes. At first review, Europeans patients were on average a decade older (65.1±14.1 years) than other ethnic groups.
In 2005/06, 6135 patients attended a review, including 933 (15%) Māori. Of these, 1345 were new patients, attending their first review. During the each year of this study, between 1300–2100 patients attended their first Get Checked review (Figure 1). The proportion of newly diagnosed patients among those attending their first review is unclear.

Table 1. Patient characteristics at first review by ethnicity

<table>
<thead>
<tr>
<th>Variables</th>
<th>European</th>
<th>Māori</th>
<th>Pacific Islander</th>
<th>Asian</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total of all years</td>
<td>7582 (69%)</td>
<td>1958 (18%)</td>
<td>309 (3%)</td>
<td>394 (4%)</td>
<td>10919 (100%)</td>
</tr>
<tr>
<td>Year of first review</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000–01</td>
<td>1136 (75%)</td>
<td>244 (16%)</td>
<td>17 (1%)</td>
<td>53 (3%)</td>
<td>1523 (14%)</td>
</tr>
<tr>
<td>2001–02</td>
<td>1379 (63%)</td>
<td>364 (17%)</td>
<td>60 (3%)</td>
<td>49 (2%)</td>
<td>2172 (20%)</td>
</tr>
<tr>
<td>2002–03</td>
<td>1135 (69%)</td>
<td>281 (17%)</td>
<td>69 (4%)</td>
<td>56 (3%)</td>
<td>1654 (15%)</td>
</tr>
<tr>
<td>2003–04</td>
<td>1451 (70%)</td>
<td>390 (19%)</td>
<td>69 (3%)</td>
<td>84 (4%)</td>
<td>2079 (19%)</td>
</tr>
<tr>
<td>2004–05</td>
<td>1509 (70%)</td>
<td>431 (20%)</td>
<td>54 (3%)</td>
<td>94 (4%)</td>
<td>2146 (20%)</td>
</tr>
<tr>
<td>2005–06</td>
<td>972 (72%)</td>
<td>248 (18%)</td>
<td>40 (3%)</td>
<td>58 (4%)</td>
<td>1345 (12%)</td>
</tr>
<tr>
<td>Age at first review, years</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65.1</td>
<td>(64.8–65.4)</td>
<td>55.8 (55.2–56.4)</td>
<td>56.0 (54.6–57.3)</td>
<td>55.4 (54.2–56.7)</td>
<td>62.6 (62.4–62.9)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3330 (44%)</td>
<td>814 (42%)</td>
<td>132 (43%)</td>
<td>157 (40%)</td>
<td>4761 (44%)</td>
</tr>
<tr>
<td>Female</td>
<td>3185 (42%)</td>
<td>876 (45%)</td>
<td>133 (43%)</td>
<td>160 (41%)</td>
<td>4651 (43%)</td>
</tr>
<tr>
<td>Diabetes type</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>687 (9%)</td>
<td>106 (5%)</td>
<td>13 (4%)</td>
<td>22 (6%)</td>
<td>890 (8%)</td>
</tr>
<tr>
<td>Type 2</td>
<td>6549 (86%)</td>
<td>1752 (89%)</td>
<td>286 (93%)</td>
<td>353 (90%)</td>
<td>9547 (87%)</td>
</tr>
</tbody>
</table>

Data are N (%) or Mean (95% confidence interval).
Row wise percentages for the four ethnic groups are presented for each cohort.
Percentages may not add up to 100 because of missing data.

Figure 1. Uptake of patients in the programme (January 2000–June 2005)
Survival analysis shows that 7142 patients returned for a second review within a median time of 1.17 years after initial review (Table 2, Figure2). The survival distribution function (SDF) at a time represents the proportion of patients who have not returned for a review up to that time.

At 1½ years after initial review (allowing a 6-month window to the ideal 1-year timeframe), 35% of eligible patients were yet to return for a second review. At 5 years after the first review, 15% had not returned for a second review. Those who continued participating in the programme after second review returned for subsequent reviews on a much more regular basis. At 1½ years after second review 75% of eligible patients had returned for a third review. High proportions of patients (35%–46%) were censored for each review. The proportion of patients censored due to death and migration were relatively small (Table 2).

Table 2. Survival analysis of time to review (from the previous review)

<table>
<thead>
<tr>
<th>Analysis variable</th>
<th>Reviewed</th>
<th>Censored</th>
<th>Median time to review (interquartile range)</th>
<th>Review rates at 1.5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to Review 2 <em>(from Review 1</em>)</td>
<td>7140 / 10919</td>
<td>3779 (35%)</td>
<td>1.17 (1.0–2.1)</td>
<td>65.0%</td>
</tr>
<tr>
<td>Time to Review 3 <em>(from Review 2</em>)</td>
<td>4183 / 7140</td>
<td>2957 (41%)</td>
<td>1.10 (1.0–1.5)</td>
<td>74.8%</td>
</tr>
<tr>
<td>Time to Review 4 <em>(from Review 3</em>)</td>
<td>2352 / 4183</td>
<td>1831 (44%)</td>
<td>1.09 (1.0–1.3)</td>
<td>79.1%</td>
</tr>
<tr>
<td>Time to Review 5 <em>(from Review 4</em>)</td>
<td>1070 / 2352</td>
<td>1282 (46%)</td>
<td>1.06 (1.0–1.2)</td>
<td>84.8%</td>
</tr>
</tbody>
</table>

*Conditional that patient attended this review.

Figure 2. Kaplan-Meier survival curves for the time to reviews (from the previous review, conditional that patients attended the previous review)
Table 3. Survival analysis of time to second review (from initial review)

<table>
<thead>
<tr>
<th>Variables</th>
<th>N</th>
<th>Patients who attended a second review</th>
<th>% Censored</th>
<th>Median time to second review (interquartile range)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>10919</td>
<td>7140</td>
<td>35%</td>
<td>1.17 (1.03–2.14)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>7582</td>
<td>5093</td>
<td>32%</td>
<td>1.13 (1.03–1.97)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Māori</td>
<td>1958</td>
<td>1091</td>
<td>44%</td>
<td>1.39 (1.07–3.71)</td>
<td></td>
</tr>
<tr>
<td>Pacific*</td>
<td>1091</td>
<td>201</td>
<td>35%</td>
<td>1.24 (1.06–2.25)</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>394</td>
<td>234</td>
<td>41%</td>
<td>1.35 (1.09–2.43)</td>
<td></td>
</tr>
<tr>
<td>Age at first review (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>724</td>
<td>359</td>
<td>50%</td>
<td>1.82 (1.14–4.88)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>40–65</td>
<td>5020</td>
<td>3220</td>
<td>36%</td>
<td>1.24 (1.05–2.27)</td>
<td></td>
</tr>
<tr>
<td>65+</td>
<td>5175</td>
<td>3561</td>
<td>31%</td>
<td>1.11 (1.02–1.83)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>4651</td>
<td>1484</td>
<td>32%</td>
<td>1.17 (1.03–1.15)</td>
<td>0.7103</td>
</tr>
<tr>
<td>Male</td>
<td>4761</td>
<td>1578</td>
<td>33%</td>
<td>1.18 (1.03–2.16)</td>
<td></td>
</tr>
<tr>
<td>Diabetes Type</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>890</td>
<td>565</td>
<td>37%</td>
<td>1.32 (1.06–3.27)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Type 2</td>
<td>9547</td>
<td>6329</td>
<td>34%</td>
<td>1.16 (1.03–2.07)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>482</td>
<td>246</td>
<td>49%</td>
<td>1.22 (1.04–2.86)</td>
<td></td>
</tr>
</tbody>
</table>

P values from Wilcoxon’s test for homogeneity of survival curves over strata; *Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Table 4. Odds ratios (95% confidence intervals) for a second review

<table>
<thead>
<tr>
<th></th>
<th>Univariate odds</th>
<th>Multivariate odds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years (OR=1 for 65+ years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40 years</td>
<td>0.508 (0.45–0.57)</td>
<td>0.565 (0.50–0.64)</td>
</tr>
<tr>
<td>40–65 years</td>
<td>0.804 (0.76–0.85)</td>
<td>0.862 (0.82–0.91)</td>
</tr>
<tr>
<td>Ethnicity (OR=1 for Europeans)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>0.690 (0.65–0.74)</td>
<td>0.728 (0.68–0.78)</td>
</tr>
<tr>
<td>Asian</td>
<td>0.739 (0.65–0.85)</td>
<td>0.786 (0.69–0.90)</td>
</tr>
<tr>
<td>Diabetes Type (OR=1 for Type 2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>0.729 (0.66–0.80)</td>
<td>0.868 (0.81–0.93)</td>
</tr>
</tbody>
</table>

Odds ratios from Cox’s Proportional Hazards Model.; The multivariate model included age group (<40, 40–65, 65+), ethnicity (European, Māori, and Asian), and type of diabetes (Type 1 and Type 2); Gender and Pacific ethnicity were found to be non-significant predictors in the multivariate analysis and were excluded from the final model.

Patients of Māori or Asian origin, younger patients, and those with Type 1 diabetes took a significantly longer time to return for a second review (Table 3). Survival curves for time to second review were significantly different for subgroups of ethnicity, type of diabetes and age at first review (Figure 3). No significant gender difference was found.

After covariate adjustment for age, ethnicity, and type of diabetes, younger patients aged<40 years were less likely to return for a second review compared with those aged 65+. Māori and Asian patients were less likely to return for a second review.
compared with Europeans, and those with Type 1 diabetes were less likely to return for a review compared with Type 2 patients (Table 4). Pacific ethnicity and gender were not found to be significant predictors of return for a second review.

Figure 3. Kaplan-Meier survival curves for time to second review (from initial review) by subgroups of ethnicity, age, gender and diabetes type

Discussion

The WPH Get Checked programme, which started in 2000, had 1300–2100 new patients on board every year. WPH estimates that in 2005/6 there should be 10,600 patients with known diabetes within the organisation network. As pointed out by the recent audit report, it is difficult draw detailed conclusions on the coverage of the programme since patient level information on all people with diabetes is not available.
A separate database held by the Waikato Regional Diabetes Service (WRDS), which provides secondary diabetes services and retinal screening to diabetes patients in the Waikato region, had 9936 patients registered in year 2005, with 2006 (21%) patients being Māori. Yet in 2005/06 only 6135 WPH patients turned up for a free diabetes check, of which 933 (15%) were Māori. The Get Checked data could not be merged with WRDS database since the Get Checked dataset provided for analysis was anonymous. Merging these two datasets would have been valuable to estimate the proportion of WRDS patients attending Get Checked review and to understand the profile of patients who never entered the Get Checked programme.

Patients coming for their first check in any one year include newly diagnosed, those who have been diagnosed for some time but new to the programme, and existing patients who recently moved in to the Waikato region. Data from the Get Checked programme is underestimating the number of people with diabetes in the region. A regional diabetes register will help to evaluate the coverage of the programme and characterise patients who are not using the free check. Data fed from local registers to a national register could potentially maximise the use of Get Checked data with regular reports to the Local Diabetes Teams, thus enhancing planning and provision of diabetes services.

Despite this programme being free to patients, a significant proportion of patients did not return for a second review within 1.5 years after initial review. The profile of patients who were retained in the programme with regular reviews was quite different from the irregular attendees and those who dropped out.

Younger patients aged <40 years, those of Māori or Asian origin, and those with Type 1 diabetes were less likely to be retained in the programme with regular checks, as indicated by the longer time to second review and lesser likelihood of returning for a second review. In the UK, predictors of attendance for review in general practice were older age, less comorbidity, and being of European ancestry.19 A South Auckland study in the early 1990s showed that patients who defaulted from diabetes care were younger, diagnosed at a younger age, more likely to be in paid employment, knew less about diabetes, and were less likely to require medication.20 Out-of-pocket expenses also impede diabetes self-care in New Zealand.3,5

Māori, Pacific, and Asian people have considerably higher rates of diagnosed diabetes compared with European people (European/Other 2.9%, Māori 8%, Pacific 10.1%, Asian 8.4%).21 They also have an earlier onset of Type 2 diabetes and higher rates of diabetes complications.1

Overall, the finding that Māori and Asians were less likely to attend a second review is a concern and it indicates these diabetes patients still have problems with access to appropriate health care. In order to minimise this inequality, remedial measures are needed to increase the uptake of this free review among ethnic minorities.

Younger Type 1 patients with complications may be visiting the WRDS regularly and may not deem it necessary to go to their GP for a separate annual check. In 2005, there were 1338 (13%) Type 1 patients registered with WRDS,20 but only 890 (8%) of the patients who had had a Get Checked review had Type 1 diabetes.
Of the patients reviewed in 2005-06, 405 had Type 1 diabetes. Thus these patients may in fact be receiving good follow-up care, although the results from this study indicate that these patients may have difficulty accessing their health services. However, a high proportion of younger patients are likely to be in the workforce and patients in paid employment are known be less likely to access care. They are also more mobile and could be getting treated elsewhere (perhaps managed by WRDS) but still registered with WPH.

It was outside the scope of this analysis to track continued participation of patients who migrated and changed to a different PHO, after completing their initial Get Checked review with WPH. A national diabetes register will be needed to make cross-regional comparisons. Migrated patients were censored in the survival analysis and were not a major issue. Data concerning year of diagnosis of diabetes was not available.

It would have been of considerable interest as the participation rates of those newly diagnosed with diabetes may differ from that of others. Similarly, data concerning geographical location of residence was not available. Physical access to care including transportation has been identified as a barrier to care in a previous study.

The Get Checked programme provides a beneficial service for many people with diabetes, but the data are significantly limited as a source of understanding of diabetes care in New Zealand. Although the programme is adequately funded and provides more consistent care for patients, practical challenges need to be dealt with and cost-benefit of the programme may need to be more clearly demonstrated to the patients.

Our longitudinal analysis reveals concerns regarding retention rates in the Get Checked programme, especially for Māori and patients with Type 1 diabetes. Further research aimed at understanding the lower retention rates for these groups may help to improve health outcome disparities.

Competing interests: None known.

Author information: Grace Joshy, Research Fellow in Diabetes Epidemiology, Waikato Clinical School, University of Auckland, Hamilton, New Zealand; Ross A Lawrenson, Head of Waikato Clinical School, University of Auckland, Hamilton, New Zealand; David Simmons, Professor, Institute of Metabolic Science, Cambridge University Hospitals NHS Foundation Trust, Cambridge, United Kingdom.

Acknowledgements: We thank Waikato Primary Health and Pinnacle Group Pte Ltd for providing the data for analysis and for their support. University of Auckland researchers Timothy Kenealy, Greg Gamble, and Steven Lillis are also acknowledged for their valuable input.

Correspondence: Grace Joshy, Research Fellow-Diabetes Epidemiology, University of Auckland, Waikato Clinical School, Waikato Hospital, Private Bag 3200, Hamilton, New Zealand. Fax: +64 (0)7 8343615; email: JoshyG@waikatodhb.govt.nz

References:
Concordance and discordance between primary and secondary care health workers in perceptions of barriers to diabetes care

Steven Lillis, Judith Swan, Jarrod Haar, David Simmons

Abstract

Aims To understand differences between primary care health professionals and secondary care health workers in their perceptions of barriers to good diabetes care.

Methods Practice nurses and general practitioners in the Waikato region of New Zealand were surveyed to ascertain their perceptions (as primary health care workers) of barriers to diabetes care; 315 replies were received (70% response rate). Secondary care health professionals working at Waikato Hospital were similarly surveyed; 123 replies were received (71% response rate).

Results Primary care health workers are more likely than secondary health care workers to rate motivation, self-belief, financial issues, lack of governmental funding, lack of public awareness of diabetes, and lack of symptoms as barriers to care. Secondary health care workers are significantly more likely to rate appointment systems, inappropriate cultural messages, lack of community-based services, high prevalence of diabetes, and unhelpful health practitioners.

Conclusions Better understanding of the respective differences in perceptions between primary and secondary care may assist the development of a more functional and unified health system. It is suggested that greater emphasis on individual diabetes education and a stronger focus on motivation and lifestyle changes at both the individual and community levels may improve outcomes.

Three factors interact to contribute to the outcomes of management in diabetes: the health care provider, the patient, and the health care system. Well-organised systems of care for chronic disease states are known to substantially improve outcomes in diabetes.1 This beneficial influence is true for both secondary and primary care.2

Knowledge of, and agreement with, evidence-based guidelines also positively influences practice.3 As would be expected, the knowledge of specialists in their area of expertise exceeds that of generalists and their use of ‘best practice’ interventions is higher.3 General practitioners and practice nurses, on the other hand, are in an environment characterised by ‘high uncertainty-low technology’ where multiple imperatives (such as comorbidities, social dysfunction, poverty, and preventative health initiatives) compete for attention. Patterns of disease burden, disease complexity, comorbidity, outcome, and resource utilisation have been shown to differ between secondary care and primary care in diabetes.5

The experiences of those working in secondary care and primary care are clearly different. It is probable, therefore, that there are also differences in perception as to what barriers exist in achieving good diabetes care and how important these barriers
are. This research explores areas of commonality and difference between two groups of health care workers in the same region: those involved in primary care and those involved in secondary care in the Waikato region of New Zealand.

Methods

Study design—The dataset utilised was the Barriers to Diabetes Care in the Waikato study. In 2003 a total of 232 general practitioners and 220 practice nurses from the Midland region of New Zealand were invited to participate in a postal survey. The Dillman method (study information and questionnaire in stamped, addressed envelopes with repeat request for non responders) was used to maximise returns. Replies were received from 166 general practitioners (72%) and 149 practice nurses (68%). A total of 173 hospital health care workers in the Midland region were invited to participate. Replies were received from 123 staff (71%) including doctors, nurses, and dieticians thus giving an overall response rate of 69%.

The respondents were asked for demographic data and their responses to these 5 questions:

- What do you feel prevents your patients from looking after their diabetes?
- How would you improve diabetes care in this region?
- Are you worried about diabetes care in this region?
- Why/why not?
- Do you have any other comments about ways which may improve services for you or others?

A validated coding system previously reported was used to code all replies. The coding system allows analysis of statements by allocating the statements to one of five broad categories: psychological, educational, internal physical, external physical (including administrative barriers), and psychosocial. As can be seen in Table 1, more detailed codes were available with each broad category. All statements were collated, reviewed, and separately coded by at least two of the researchers. Disagreements in coding were resolved by discussion.

Statistics—Data were analysed using SPSS for Windows (v14.0) software. The Z test of proportionality was used to analyse for significance as it was desired to compare the proportion of two different populations who responded in a similar way to the study questions. A sample of 50 random responses were recoded blind to the original codes to assess reproducibility of the coding process. A kappa coefficient of 0.68 was achieved on comparison of original coding and re-coding. This would indicate a satisfactory level of agreement.

Results

An overall response rate of 69% was obtained in this survey. A total of 4888 statements were available for coding: 3297 from primary care and 1591 from secondary care. A comparison of the proportion of statements by barrier code between primary care health workers and secondary care health workers appears in Table 2.

Areas of commonality between the two groups included the code “unsatisfactory/inappropriate diabetes care or education” (code 22) as the second most commonly reported barrier to good care in both professional groups. The three prominent and inter-related barriers of motivation, lifestyle change, and self efficacy (codes 4, 5, and 30 respectively) were ranked highly by both groups and will be discussed further.

Codes where secondary health care workers responses were significantly greater than primary care workers are given in Table 3 and indicate that secondary care workers are distinct from their primary care colleagues in reporting barriers of staffing levels, appointment systems, inappropriate cultural messages, insufficient community based
care, overwhelming workload, and previous unsatisfactory interactions with health professionals.

Table 1. Barriers to diabetes care coding framework

<table>
<thead>
<tr>
<th>CODE</th>
<th>PSYCHOLOGICAL BARRIER</th>
<th>DESCRIPTION (EXAMPLES)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Western health belief</td>
<td>Believe science/professionals should find a cure/do more</td>
</tr>
<tr>
<td>2</td>
<td>Spiritual health belief</td>
<td>Believe cause/cure should be sought spiritually/within</td>
</tr>
<tr>
<td>3</td>
<td>Alternative health belief</td>
<td>Prefers uses alternative health models/treatments, fatalism</td>
</tr>
<tr>
<td>6</td>
<td>Public health belief</td>
<td>Believes the public should bear more financial responsibility for health care, physical resources (e.g. buildings), screening costs, subsidy/salary, includes cost of GP visits</td>
</tr>
<tr>
<td>4</td>
<td>Self factors—motivation</td>
<td>Psychological—motivation, attitudes, laziness, denial, effort, stupid</td>
</tr>
<tr>
<td>5</td>
<td>Self factors—self efficacy</td>
<td>No confidence, external locus of control, low self-efficacy, lacks in-sight, support, encouragement, hope, role model, mentoring, lack of will = plus code 5.</td>
</tr>
<tr>
<td>23</td>
<td>No symptom cue</td>
<td>No physical symptoms, screening = plus code 6.</td>
</tr>
<tr>
<td>25</td>
<td>Priority setting</td>
<td>Others needs priority over own (e.g. children, elders)</td>
</tr>
<tr>
<td>29</td>
<td>Negative perceptions of time</td>
<td>Not enough time (education provided too quickly)</td>
</tr>
<tr>
<td>27</td>
<td>Emotional</td>
<td>Fear, shame emotion anxiety, worry—lack of hope, shyness, frightening</td>
</tr>
<tr>
<td>30</td>
<td>Precontemplative</td>
<td>Lifestyle issues, e.g. Strictness of regime, giving up things I enjoy, exercise, diet change, compliance = plus code 4.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CODE</th>
<th>EDUCATIONAL BARRIER</th>
<th>DESCRIPTION (EXAMPLES)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>Low diabetes knowledge</td>
<td>Lacks general/specific diabetes knowledge,</td>
</tr>
<tr>
<td>20</td>
<td>Low knowledge of service</td>
<td>Unaware of services available</td>
</tr>
<tr>
<td>37</td>
<td>Low education status</td>
<td>Low educational status</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CODE</th>
<th>INTERNAL PHYSICAL BARRIER</th>
<th>DESCRIPTION (EXAMPLES)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Self factors/other health conditions</td>
<td>Diabetes (e.g. amputation) and non-diabetes related (e.g. arthritis), smoking, cataracts, age group (e.g. youth)</td>
</tr>
<tr>
<td>18</td>
<td>Physical effects of treatment</td>
<td>Pain of glucose monitoring, drug side-effects, drugs not helping, needles</td>
</tr>
<tr>
<td>38</td>
<td>Obesity</td>
<td>Obesity, overweight, need to lose weight</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CODE</th>
<th>EXTERNAL PHYSICAL BARRIER</th>
<th>DESCRIPTION (EXAMPLES)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>Personal finance</td>
<td>Income in relation to costs</td>
</tr>
<tr>
<td>9</td>
<td>Service/physical access</td>
<td>Transportation, wheelchair entry, long distance to get to service</td>
</tr>
<tr>
<td>12</td>
<td>Limited range of services</td>
<td>No such service exists. Timing of format of services (e.g. evening clinics, home visits), medicine services (e.g. glitazones)</td>
</tr>
<tr>
<td>13</td>
<td>Appointment system/staffing levels</td>
<td>Insufficient staffing for adequate service e.g. no follow up</td>
</tr>
<tr>
<td>14</td>
<td>Lack of community-based services</td>
<td>No local clinic that is identified as ‘own’, mobile service, marae (Māori meeting house), primary care</td>
</tr>
<tr>
<td>24</td>
<td>Unhelpful health professional in past</td>
<td>Past encounter with health professional leading to conflict or without expected communication or clinical expertise. (Worse than 22)</td>
</tr>
<tr>
<td>36</td>
<td>Information management</td>
<td>GP education &amp; training, research, communication, audit, cohesion, identification of gaps, access to specialist, team approach</td>
</tr>
<tr>
<td>40</td>
<td>Diabetes epidemic</td>
<td>high incidence rates, huge burden, surge, increasing, high numbers</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CODE</th>
<th>PSYCHO-SOCIAL BARRIER</th>
<th>DESCRIPTION (EXAMPLES)</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>Unsatisfactory/inappropriate diabetes care or education</td>
<td>Wrong information provided or information provided in inappropriate way</td>
</tr>
<tr>
<td>26</td>
<td>Group pressure</td>
<td>Pressure from others not to adhere to advice</td>
</tr>
<tr>
<td>28</td>
<td>Prejudice (not reported in household survey)</td>
<td>Impression of discriminatory practice due to diabetes or for other reasons</td>
</tr>
<tr>
<td>19</td>
<td>Lack of public awareness of diabetes</td>
<td>Others behave without adequate knowledge or acceptance of diabetes, general public</td>
</tr>
<tr>
<td>15</td>
<td>Lack of family support</td>
<td>Family consumes diabetic food, resists change of lifestyle</td>
</tr>
<tr>
<td>16</td>
<td>Family demands</td>
<td>Pressure to spend time/money on the family rather than their diabetes</td>
</tr>
<tr>
<td>17</td>
<td>Unsupportive macroenvironment</td>
<td>Feeling of lack of support in the community, e.g. access to low fat foods, fast food tax, unemployment, primary prevention</td>
</tr>
<tr>
<td>10</td>
<td>Communication</td>
<td>Language differences (translation)</td>
</tr>
<tr>
<td>11</td>
<td>Inappropriate cultural messages</td>
<td>Attitude, ethnicity of workers, appropriateness of communication, minorities</td>
</tr>
<tr>
<td>Code</td>
<td>Description</td>
<td>Primary health care statements; % (95% CI range)</td>
</tr>
<tr>
<td>------</td>
<td>-------------------------------------------------</td>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>1</td>
<td>Western health belief</td>
<td>1.94 (1.5–2.4)</td>
</tr>
<tr>
<td>2</td>
<td>Spiritual health belief</td>
<td>0.06 (0.0–0.1)</td>
</tr>
<tr>
<td>3</td>
<td>Alternative health belief</td>
<td>0.36 (0.2–0.6)</td>
</tr>
<tr>
<td>4</td>
<td>Self factors—motivation</td>
<td>13.32 (12.2–14.5)</td>
</tr>
<tr>
<td>5</td>
<td>Self factors—self efficacy</td>
<td>2.4 (1.9–2.9)</td>
</tr>
<tr>
<td>6</td>
<td>Public health belief</td>
<td>7.1 (6.2–8.0)</td>
</tr>
<tr>
<td>7</td>
<td>Other health conditions</td>
<td>1.88 (1.4–2.3)</td>
</tr>
<tr>
<td>8</td>
<td>Personal finance</td>
<td>6.67 (5.8–7.5)</td>
</tr>
<tr>
<td>9</td>
<td>Service/physical access</td>
<td>1.64 (1.2–2.1)</td>
</tr>
<tr>
<td>10</td>
<td>Communication</td>
<td>0.24 (0.1–0.4)</td>
</tr>
<tr>
<td>11</td>
<td>Inappropriate cultural messages</td>
<td>2.79 (2.2–3.4)</td>
</tr>
<tr>
<td>12</td>
<td>Limited range of services</td>
<td>4.7 (4.0–5.4)</td>
</tr>
<tr>
<td>13</td>
<td>Staffing levels/appointment system</td>
<td>2.76 (2.2–3.3)</td>
</tr>
<tr>
<td>14</td>
<td>Lack of community based services</td>
<td>2 (1.5–2.5)</td>
</tr>
<tr>
<td>15</td>
<td>Lack of family support</td>
<td>0.91 (0.6–1.2)</td>
</tr>
<tr>
<td>16</td>
<td>Family demands</td>
<td>0.27 (0.1–0.5)</td>
</tr>
<tr>
<td>17</td>
<td>Unsupportive macro-environment</td>
<td>3.73 (3.1–4.4)</td>
</tr>
<tr>
<td>18</td>
<td>Physical effects of treatment</td>
<td>0.42 (0.2–0.6)</td>
</tr>
<tr>
<td>19</td>
<td>Lack of public awareness</td>
<td>1.94 (1.5–2.4)</td>
</tr>
<tr>
<td>20</td>
<td>Low knowledge of service</td>
<td>0.42 (0.2–0.6)</td>
</tr>
<tr>
<td>21</td>
<td>Low diabetes knowledge</td>
<td>9.37 (8.4–10.4)</td>
</tr>
<tr>
<td>22</td>
<td>Unsatisfactory care or education</td>
<td>9.28 (8.3–10.3)</td>
</tr>
<tr>
<td>23</td>
<td>No symptom cue</td>
<td>1.97 (1.5–2.4)</td>
</tr>
<tr>
<td>24</td>
<td>Unhelpful health professional in past Priority setting</td>
<td>0.33 (0.1–0.5)</td>
</tr>
<tr>
<td>25</td>
<td>Group pressure</td>
<td>0.52 (0.3–0.8)</td>
</tr>
<tr>
<td>26</td>
<td>Emotional</td>
<td>0.12 (0.0–0.2)</td>
</tr>
<tr>
<td>27</td>
<td>Prejudice</td>
<td>0.55 (0.3–0.8)</td>
</tr>
<tr>
<td>28</td>
<td>Prejudice</td>
<td>0.09 (0.0–0.2)</td>
</tr>
<tr>
<td>29</td>
<td>Negative perceptions of time</td>
<td>1.64 (1.2–2.1)</td>
</tr>
<tr>
<td>30</td>
<td>Precontemplative</td>
<td>9.19 (8.2–10.2)</td>
</tr>
<tr>
<td>31</td>
<td>No barriers</td>
<td>3.43 (2.8–4.0)</td>
</tr>
<tr>
<td>35</td>
<td>Information management</td>
<td>5.22 (4.5–6.0)</td>
</tr>
<tr>
<td>36</td>
<td>Low education status</td>
<td>0.24 (0.1–0.4)</td>
</tr>
<tr>
<td>37</td>
<td>Obesity</td>
<td>1.4 (1.0–1.4)</td>
</tr>
<tr>
<td>38</td>
<td>Diabetes epidemic</td>
<td>1.09 (0.7–1.4)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

*p <0.05; **p <0.01; ***p <0.001; ns=not significant.
Table 3. Barriers to diabetes care where secondary care perception of barrier was greater than primary care

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
<th>P value for Z</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Inappropriate cultural messages</td>
<td>0.018</td>
</tr>
<tr>
<td>13</td>
<td>Appointment system/staffing levels</td>
<td>0.000</td>
</tr>
<tr>
<td>14</td>
<td>Lack of community-based services</td>
<td>0.004</td>
</tr>
<tr>
<td>24</td>
<td>Unhelpful health professional in past</td>
<td>0.020</td>
</tr>
<tr>
<td>40</td>
<td>Diabetes epidemic</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Codes where primary health care workers gave significantly more responses than secondary are given in Table 4. Primary care providers perceived motivation (code 4), self efficacy (code 5), personal finance (code 8) and public health belief (code 6) as barriers more often than secondary care providers.

Table 4. Barriers to diabetes care where primary care workers perception of barrier was greater than secondary care

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
<th>P value for Z</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Self factors— motivation</td>
<td>0.000</td>
</tr>
<tr>
<td>5</td>
<td>Self factors—self efficacy</td>
<td>0.018</td>
</tr>
<tr>
<td>8</td>
<td>Personal finance</td>
<td>0.000</td>
</tr>
<tr>
<td>6</td>
<td>Public health belief</td>
<td>0.000</td>
</tr>
<tr>
<td>19</td>
<td>Lack of public awareness of diabetes</td>
<td>0.024</td>
</tr>
<tr>
<td>20</td>
<td>Low knowledge of service</td>
<td>0.038</td>
</tr>
<tr>
<td>23</td>
<td>No symptom cue</td>
<td>0.006</td>
</tr>
</tbody>
</table>

A more detailed analysis of statements concerning staff levels and appointment system (Code 13) by secondary care participants revealed that 52% of statements related to inadequate staffing levels in the hospital, 27% to poor systems of organisation to provide appointments, and the remainder (21%) reflected access difficulties to primary care appointments.

Discussion

Shared concerns—The response rate is unusually high for surveys\(^1\) and may indicate a significant level of concern over diabetes care by health workers in general as well as the effectiveness of the Dillman system for increasing the response rate from surveys.\(^1\)

The data indicates particular areas of concern to both primary and secondary health workers highlighting patient education and psychological barriers such as motivation as principal foci. Current interventions would seem not to impact adequately on these barriers. Indeed, it may be unrealistic to expect episodic interventions from health professionals to provide high levels of motivation when there are multiple agendas to cover in a brief consultation for a disease characterised by complexity and comorbidity.
Diabetes prevention and treatment in a social rather than medical construct facilitates the evolution of different solutions. Developing the motivation, social support systems, and self-belief to negotiate the lifestyle changes and restraints imposed by diabetes fits more comfortably into a social rather than medical agenda.

Successfully meeting this social agenda may require reconsideration of the ‘ownership’ of diabetes and the ‘responsibility’ for diabetes management; is it a disease that is diagnosed and treated by health professionals or is it a reflection of modern society? Quoting Chaufan on the sociology of diabetes: *Attempts, however well intentioned, to empower patients to choose a style of diabetes care that fits their needs or to fight for their rights and freedoms are problematic if choices and freedom are seriously limited by social and structural conditions and if this limitation is ignored.* If indeed, diabetes is fundamentally a sociological problem, it is likely that the best long-term solutions are sociological rather than medical in nature.

**Predominant secondary care perceptions**—There was clear discordance between primary and secondary care practitioners regarding staffing levels and appointment systems. These barriers were the most commonly noted barrier to diabetes care by hospital respondents, yet were ranked only 12th by primary care participants. Related to the staffing level and appointment statements by secondary care workers is the high number of statements regarding the epidemic of diabetes and the lack of community-based services.

It is likely that such statements reflect the tension created when demand for services significantly outstrips capacity, coupled with the belief that some of these services would be better provided in a community rather than a hospital setting. Similarly, concerns over health care education of those with diabetes may reflect inadequate capacity to deliver effective diabetes education.

Cultural concerns were significantly greater in the secondary care group. This may be explained by the selected nature of those attending secondary care for diabetes treatment. Prevalence, morbidity and mortality rates are higher for Māori than New Zealanders of European descent for diabetes including higher admission rates to hospital. Thus hospital health care workers have higher exposure to Māori who live with diabetes. An alternative explanation is that hospital-based health care workers are more culturally aware than those in primary care.

**Predominant primary care perceptions**—A notable factor in statements made from primary care is that financial issues were more commonly seen as barriers than is the case with hospital-based participants. These financial difficulties concerned both lack of government funding for diabetes care as well as personal financial barriers. Patient fees for primary care consultations as well as the more visible costs of medication in community-based care may be partly responsible for this.

**Comparative studies**—These findings accord well with other studies. A qualitative study that explored the experiences of 31 primary care physicians in America revealed that diabetes was considered particularly difficult to manage in comparison to other chronic diseases. The authors highlighted the inability to influence lifestyle factors, the asymptomatic nature of early disease, the expense of care and discrepancy between provider and patient sense of urgency in treatment as significant difficulties.
Lack of patient motivation was considered to be a major barrier to good diabetes care in a Belgian study of general practitioners. A Canadian study further emphasised psychological factors including denial and lack of motivation as well as financial cost of diabetes to the patient as barriers to management of the disease. Low levels of agreement have been found between patients and general practitioners regarding priorities of treatment in Type 2 diabetes, a factor that increases provider frustration.

A multinational study of patients and their providers (nurse, primary care physician and secondary care physician) reported that 61–72% of providers recognised psychological problems in their Type 2 diabetic patients but also reported being more able to recognise than meet these psychological needs.

Conclusions

The steadily increasing disease burden that diabetes represents to our health system may require a departure from traditional treatment models. Psychological and motivational barriers together with low diabetes knowledge and unhelpful health practitioners were strong areas of concern for both primary and secondary care health practitioners. Timely and appropriate education for those with diabetes must become a priority of treatment as this research would strongly indicate that current patient educational initiatives are inadequate and that insufficient workforce capacity is a major contributing cause.

This research would also suggest that tailored solutions are required to address the differences found between these two groups of health practitioners. For those with diabetes, primary health care workers perceive financial barriers as a major factor in accessing adequate primary health care, alongside the barriers of inadequate knowledge of available services and low public awareness of the disease.

Respondents working in secondary care indicate that further barriers include inadequate workforce capacity in the face of rapidly escalating incidence of diabetes, inadequate community-based services and inappropriate cultural messages.

Competing interests: None known.

Author information: Steven Lillis, Senior Lecturer in General Practice, Waikato Clinical School, Bryant Education Centre, Hamilton, New Zealand; Judith Swan, Associate Research Fellow, Dean's Office, Faculty of Medicine, University of Otago, Dunedin, New Zealand; Jarrod Haar, Senior Lecturer, School of Management, University of Waikato, Hamilton, New Zealand; David Simmons, Institute of Metabolic Science, Cambridge University Hospitals NHS Foundation Trust, Cambridge, England

Correspondence: Steven Lillis, Senior Lecturer in General Practice, Waikato Clinical School, Bryant Education Centre, Private Bag 3200, Hamilton, New Zealand. Email: lillis@waikatodhb.govt.nz

Acknowledgements: We acknowledge the Waikato Local Diabetes Team and the Waikato Medical Research Foundation for their financial support.

References:


Supporting pregnant women to quit smoking: postal survey of New Zealand general practitioners and midwives’ smoking cessation knowledge and practices

Marewa Glover, Janine Paynter, Chris Bullen, Kay Kristensen

Abstract

Aim This study examined New Zealand general practitioners’ (GPs) and midwives’ smoking cessation knowledge and support offered to pregnant women who smoke.

Method Postal survey of a random sample of 776 New Zealand GPs and midwives, undertaken between September and October 2006.

Results Responses were received from 39% (147/376) GPs and 57% (203/355) midwives. Almost all GPs indicated that they were involved in confirming pregnancy during the first trimester, compared with only 55% of midwives. There was high reported routine recording of smoking status (84.5% for GPs and 98.5% for midwives) and almost all participants thought it was consistent with their role to ask about smoking in pregnancy, discuss the effects of smoking, and ask if women who smoke wanted to stop smoking. Whilst 71% of GPs reported usually advising pregnant women who smoke to abstain completely only 11% of midwives said they do this. Midwives were much more likely to advise cutting down. Over 60% of participants said they usually provide cessation counselling to pregnant women. Reported recommendation of nicotine replacement therapy (NRT) was low. Only 34% of GPs and 31% of midwives were likely to recommend nicotine gum.

Conclusions GPs are in a pivotal position to offer stop smoking advice at the time of confirmation of pregnancy, when the motivation to quit is highest. Insufficient emphasis on the importance of early and complete smoking abstinence is being given by most midwives. Intermittent nicotine delivery mechanisms (such as the nicotine gum, inhaler, lozenge, or microtab) are not well known and need to be promoted more to pregnant women who smoke.

Smoking in pregnancy is the single most preventable cause of pregnancy complications such as miscarriage, pre-term birth, and stillbirth.\textsuperscript{1,2} Smoking increases the risk of sudden infant death syndrome and has adverse effects on children’s physical and mental development.\textsuperscript{3,4}

Smoking cessation programmes should be available in all maternity care settings because they have been shown to increase smoking cessation, reduce preterm birth, and increase birth weight.\textsuperscript{5} Indeed, the New Zealand Ministry of Health (MoH) considers that pregnant women are a priority group.\textsuperscript{6}

Despite strong evidence of the harms of smoking in pregnancy and of the benefits of stopping smoking, a survey in 2003 found that 22% of pregnant New Zealand women who smoked were smoking around the time of conception.\textsuperscript{7} The proportion of Māori women smoking at conception was twice this estimate (55%).\textsuperscript{7} The MoH has an ambitious goal to reduce this figure to 30% or lower by 2008.\textsuperscript{8}

Pregnancy offers a unique opportunity for smoking cessation interventions due to an increased motivation to stop and likely increased number of contacts pregnant women
have with health professionals. Motivation to quit is highest in the first trimester, but despite this approximately one-third of pregnant women smokers continue to smoke during pregnancy, and 21% of women who quit during pregnancy relapse prior to delivery.

One published survey of cessation services provided for pregnant women has been conducted in New Zealand. This survey, conducted in 2001, randomly sampled 274 GPs and 184 midwives—65% of GPs and 95% of midwives asked most of their pregnant patients about smoking. More than three-quarters of the respondents considered smoking cessation advice as an important part of their job.

Half of the GPs and midwives surveyed reported offering smoking cessation advice. Midwives were significantly less likely than GPs to feel comfortable or confident giving smoking cessation advice. Low numbers (24%) of midwives and GPs reported that NRT was appropriate for pregnant women.

Since the 2001 survey, smoking cessation support for pregnant women and smoking cessation practice has changed. The MoH now funds training programmes to improve the provision of smoking cessation counselling and support given by lead maternity carers (LMCs) and other health professionals. Culturally appropriate cessation support for Māori, Aukati Kai Paipa, has also been funded.

Smoking cessation guidelines were developed in 2000, revised in 2002, and have been revised again in 2007 to provide healthcare workers with up-to-date evidence-based guidance. Accordingly, in the third quarter of 2006 we undertook a survey of New Zealand health professionals providing care to pregnant women to identify their knowledge and practices in regard to smoking cessation advice and support. This paper reports on the findings from that survey.

**Method**

We obtained a sample of 376 GPs randomly selected by computer programme from a national database of all New Zealand GPs (Atlantis Ltd), and a random sample of 446 registered midwives from the Midwifery Council of New Zealand’s database.

The online White Pages® telephone directory website was searched to obtain address details for the selected midwives, but because of missing data a second random selection from the original list was required to obtain a final sample of 397 registered midwives with postal addresses.

We constructed a questionnaire around six domains of enquiry, comprising participant demographic characteristics and each of the ‘Five As’ (Ask, Assess, Advise, Assist, Arrange) recommended in the 2002 New Zealand Guidelines for smoking cessation. The questionnaire comprised closed- and open-ended questions, so participants could write comments if they desired, and was pre-tested for comprehension and ease of completion by two GPs and two midwives.

The questionnaire, participant information sheet, consent form, a competition entry form and prepaid reply envelope were mailed out to the selected participants in September 2006, and re-sent to those who had not responded one month later. We entered the responses in a Microsoft Excel spreadsheet and analysed the data using simple EpiInfo 2000 software. Multiple entries for ethnicity were reduced to a single category using Statistics New Zealand’s prioritisation standard (NZ Māori, Pacific Island, Asian, Pakeha/NZ European, Other).

We obtained approval to conduct the study from the University of Auckland Human Participants Ethics Committee and funding support from the MoH.
Results

Response rates and sociodemographic and practice characteristics—Responses were received from 147/376 (39%) of the GPs and 203/355 (57%) of the midwives, an overall response rate of 48%. Surveys returned unopened due to an incorrect address were removed from the midwives denominator. Two GPs and one midwife responded to the survey despite not having been invited. These were excluded from the response rate calculation but their results are included in the analyses because their data could not be separately identified and removed.

The demographic characteristics of the respondents are shown in Table 1. The vast majority of GPs and midwives were European/Pakeha, with very small numbers of Māori, Pacific, Asian, and other ethnicities. Most respondents (71%) were aged between 35–54 years with similar proportions of GPs and midwives in each age group.

Almost all (99%) of midwives were female compared to just under half (46%) of the GPs. Responses were received from GPs located in all District Health Board (DHB) regions throughout New Zealand with the exception of Wairarapa, and from midwives in all 21 DHB regions.

Employment arrangements were markedly different: almost all (91%) of the GPs, but only a small proportion (3%) of midwives, worked under the auspices of a primary health organisation (PHO). Twenty-two percent of GPs and 45% of midwives stated that they practiced independently. A small number of GPs (11%) but almost two-thirds of midwives (60%) worked under the auspices of a DHB. No midwives and only 7% of GPs worked for a Māori provider. Ten percent of GPs and almost 20% of midwives estimated that half or more of their clients were Māori.

Involvement in confirming pregnancy—GPs were significantly more likely to see women to confirm pregnancy than midwives (99% compared with 55% respectively; RR 1.66, 95% CI 1.48–1.86) and were more likely to see them in the first trimester (73% of GPs compared with 60% of midwives; RR 1.67, 95% CI 1.49–1.87). Twenty-three percent of midwives said they usually see women for the first time in the second trimester and 17% in the third trimester.

Asking about smoking—Almost all midwives (98.5%) and fewer, but most, GPs (84.5%) reported that the smoking status of their patients was routinely recorded on the patient’s record. High proportions of GPs (97%) and midwives (95.5%) reported that they considered asking about smoking in pregnant patients to be part of their role.

GPs were significantly more likely than midwives to ask about smoking status at the first visit (92% vs 82%; RR 1.12, 95% CI 1.03–1.21). A key reason given for not asking about smoking was the short time available, as there were always a number of important topics to discuss. Other respondents stated that they didn’t see pregnant women until late in pregnancy or postnatal so didn’t ask.

Several GPs commented that asking and recording smoking status was the responsibility of the practice nurse and some noted that they usually only see women once for confirmation of pregnancy.
Table 1. Demographic profile of GPs and midwives participating in the survey

<table>
<thead>
<tr>
<th>Variables</th>
<th>GP (N=147)</th>
<th>Midwife (N=203)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnicity</strong></td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>European</td>
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<td>89</td>
</tr>
<tr>
<td>Māori</td>
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<td>5</td>
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<tr>
<td>Pacific Islands</td>
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<td>1</td>
</tr>
<tr>
<td>Asian</td>
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<td>1</td>
</tr>
<tr>
<td>Other</td>
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<td>3</td>
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<td></td>
</tr>
<tr>
<td>Auckland</td>
<td>17</td>
<td>22</td>
</tr>
<tr>
<td>Bay of Plenty</td>
<td>6</td>
<td>8</td>
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<tr>
<td>Canterbury</td>
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<td>11</td>
</tr>
<tr>
<td>Capital &amp; Coast</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Hawke’s Bay</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Hutt Valley</td>
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<td>3</td>
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<tr>
<td>Lakes</td>
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<td>1</td>
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<tr>
<td>Mid Central</td>
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<td>2</td>
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<tr>
<td>Nelson Marlborough</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Northland</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Otago</td>
<td>6</td>
<td>3</td>
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<tr>
<td>South Canterbury</td>
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<td>1</td>
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<td>Waikato</td>
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<td>7</td>
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<td>0</td>
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<td>Waitemata</td>
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<td>2</td>
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<tr>
<td><strong>Age (years)</strong></td>
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<tr>
<td>16-24</td>
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<td>1</td>
</tr>
<tr>
<td>25-34</td>
<td>5</td>
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<td>35-44</td>
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<td>45-54</td>
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<td>55-64</td>
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<td>65+</td>
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<td>3</td>
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<td>Urban</td>
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<td>72</td>
</tr>
<tr>
<td>Rural</td>
<td>16</td>
<td>23</td>
</tr>
<tr>
<td>Both</td>
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<td>5</td>
</tr>
<tr>
<td><strong>Organisation</strong></td>
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<td></td>
</tr>
<tr>
<td>Public Health Organisation (PHO)</td>
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<tr>
<td>Independent</td>
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<td>45</td>
</tr>
<tr>
<td>Māori</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Pacific Island</td>
<td>11</td>
<td>60</td>
</tr>
<tr>
<td>DHB</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori clients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>&lt;50%</td>
<td>90</td>
<td>77</td>
</tr>
<tr>
<td>&gt;50%</td>
<td>10</td>
<td>20</td>
</tr>
</tbody>
</table>
Advising about stopping smoking—High proportions of both GPs (94.5%) and midwives (90%) reported usually asking pregnant patients who smoke if they wished to stop but there were important differences in the advice given.

GPs were significantly more likely than midwives (71% vs 11%; RR 6.50, 95% CI 4.32–9.77) to report advising such patients to stop smoking completely. Conversely, midwives were significantly more likely than GPs (80% vs 28%; RR 2.86, 95% CI 2.18–3.74) to advise cutting down initially with a view to stopping altogether.

A small proportion of midwives (6%) said that they only advise cutting down smoking. Similarly high proportions of both GPs and midwives (90% and 95% respectively) stated that they usually discuss the adverse effects of smoking during pregnancy with smoking patients at the first visit.

Discussing smoking with pregnant women—GPs were significantly more likely than midwives to give stop smoking advice to pregnant women who are known smokers at each antenatal visit as opposed to discussing it only when raised by the woman (69% vs 47%; RR 1.45, 95% CI 1.20–1.75). Five GPs (3%) and 21 midwives (11%) reported that they discuss smoking at a pre-arranged time set up for that purpose.

Arranging cessation support—GPs and midwives were equally more likely than not to provide cessation counselling (65% vs 61%; RR 1.07, 95% CI 0.91–1.26). Figure 1 shows their responses to a question about the effectiveness of various stop smoking medications.

Nicotine replacement (NRT) patch was considered by both GPs and midwives to be the most effective mode of treatment although almost half the GPs thought nicotine gum would be effective for pregnant women. More GPs than midwives considered pharmacotherapy as being effective treatments for pregnant women than midwives. Acupuncture and hypnosis were considered effective by about the same number of midwives that considered NRT patch and gum effective.

The number of GPs rating acupuncture and hypnosis as effective was somewhat lower than the number rating NRT patch and gum as effective but the proportion of GPs doing so was lower than for midwives. Of concern is that a considerable number of GPs (33) and midwives (74) indicated they knew little about the effectiveness for pregnant women of the list of cessation treatments.

GPs and midwives were also asked how likely they were to recommend a particular treatment. Figure 2 shows that the likelihood of recommending particular treatments compared favourably with those treatments considered to be most effective. There was no difference between GPs and midwives in their likelihood of recommending NRT patches to a pregnant woman who smokes (RR 1.01, 95% CI 0.80–1.27) but GPs were significantly less likely to refer pregnant smokers for acupuncture than midwives (34% vs 50.5% respectively; RR 0.67, 95% CI 0.50–0.90). Almost half of the midwives surveyed stated that they were likely or very likely to recommend acupuncture and hypnotherapy.
Figure 1. Perceived effectiveness in pregnant women of various smoking cessation treatments by GPs and midwives (with 95% confidence intervals)

Figure 2. Proportions of GPs and midwives likely or very likely to recommend a particular cessation treatment
Awareness of New Zealand Guidelines for Smoking Cessation—GPs (70%) and midwives (61%) were equally likely to be aware of the New Zealand Guidelines for smoking cessation.14

Arranging referral to specialist smoking cessation providers—Table 2 shows the cessation support services most likely to be recommended by respondents. GPs were significantly more likely than midwives, or very likely, to refer pregnant women to Quitline (RR 1.20, 95% CI; 1.09–1.31). The next preferred source of cessation support for GPs was to refer women to practice nurses (65%). Only 26.5% of GPs were likely or very likely to refer women to a Māori cessation provider, and almost one-third (31%) said they didn’t know of any such service. Few GPs were likely to refer women to a Pacific Island cessation provider or Seventh Day Adventist cessation programmes, but again a large proportion of the GPs did not know of these services. Some participants said that they referred clients to other DHB services or hospital run programmes. A few said they had used Quitline in the past but not all reported having had a good experience with this service. Smokechange was strongly supported by midwives but not by GPs. Smokechange is a personalised, motivational intervention programme designed to encourage pregnant women to cut down their smoking as a pathway to stopping smoking.16

Table 2. Likelihood (%) of referral to a selected cessation provider

<table>
<thead>
<tr>
<th>Cessation provider</th>
<th>Highly unlikely</th>
<th>Unlikely</th>
<th>Likely</th>
<th>Very likely</th>
<th>Don't know</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GP</td>
<td>MW</td>
<td>GP</td>
<td>MW</td>
<td>GP</td>
</tr>
<tr>
<td>Quitline (N=142)</td>
<td>7</td>
<td>5.5</td>
<td>12</td>
<td>13</td>
<td>32</td>
</tr>
<tr>
<td>Māori cessation provider (N=123)</td>
<td>20</td>
<td>6</td>
<td>21</td>
<td>21</td>
<td>18.5</td>
</tr>
<tr>
<td>Smokechange (N=117)</td>
<td>23</td>
<td>5</td>
<td>19</td>
<td>8</td>
<td>205</td>
</tr>
<tr>
<td>Pacific Island cessation provider (N=114)</td>
<td>26</td>
<td>14</td>
<td>21</td>
<td>19</td>
<td>3</td>
</tr>
<tr>
<td>7th Day Adventist Church (N=114)</td>
<td>30</td>
<td>24</td>
<td>21</td>
<td>11.5</td>
<td>1</td>
</tr>
<tr>
<td>Practice nurse (N=124)</td>
<td>13</td>
<td>31</td>
<td>18</td>
<td>41</td>
<td>26</td>
</tr>
</tbody>
</table>

GP=general practitioner, MW=midwife.

Smoking cessation-related training—The training experiences of GPs and midwives differed markedly. Almost two-thirds of GPs but just over one-third of midwives indicated that they had undertaken training in smoking cessation (Table 3). A quarter of GPs and 16% of midwives recalled undertaking training in the use of the Guidelines for smoking cessation.14

A third of GPs but fewer than 5% of midwives had received training in the provision of NRT. However, almost a half of the midwives surveyed stated that they had received training in provision of cessation advice to pregnant women who smoke. Sixty-seven respondents (19%) had completed other forms of training, 23 (9% of the total) naming courses provided by Education for Change Ltd. Some had attended courses provided by the New Zealand College of Midwives, PHO, or DHB providers and a few had attended courses in the UK and Australia, or had attended courses provided by the manufacturers of pharmaceutical products used in smoking cessation treatment.
Table 3. GPs and midwife participation in cessation-related training

<table>
<thead>
<tr>
<th>Training area</th>
<th>GPs (N=147)</th>
<th>Midwives (N=203)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief smoking cessation advice</td>
<td>92 (62.5%)</td>
<td>78 (38%)</td>
</tr>
<tr>
<td>Use of the NZ Guidelines for smoking cessation</td>
<td>37 (25%)</td>
<td>32 (16%)</td>
</tr>
<tr>
<td>Provision of Nicotine Replacement Therapy</td>
<td>46 (31%)</td>
<td>8 (4%)</td>
</tr>
<tr>
<td>Providing smoking cessation advice to pregnant women who smoke</td>
<td>18 (12%)</td>
<td>94 (46%)</td>
</tr>
<tr>
<td>Other cessation treatment method</td>
<td>21 (14%)</td>
<td>30 (15%)</td>
</tr>
</tbody>
</table>

Education for Change Ltd was the most frequently used provider of cessation training for midwives (46%) whereas the National Heart Foundation was used more by GPs (but only 9.5% reported attending the course). Nearly a quarter (24%) of GPs and 17% of midwives said that smoking cessation was covered in their basic training. Thirty-nine percent of GPs and 17% of midwives had undertaken smoking cessation-related training with providers other than those funded by the Ministry of Health. Only 14% of GPs and 3% of midwives indicated that they were registered Quitcard providers.

Discussion

Surveys of GPs and maternity health professionals in the UK have indicated that while most routinely ask about smoking at the first antenatal visit, far fewer advise pregnant smokers on how to stop, and even fewer monitor and review those still smoking.\(^9,17\)

In a 1995 UK study, 96% of health professionals stated that they asked about and recorded the smoking status of pregnant women and explained the risks of smoking to pregnant smokers when they saw them for the first time. However, fewer (67%) advised pregnant smokers on how to stop and less than half (47%) monitored and reviewed smoking status throughout pregnancy.\(^9\)

In a more recent survey in the UK, McEwan and White found that 96% of GPs and 99% of nurses accepted that intervening to support cessation was part of their role and routinely recorded the smoking status of patients. However, only 50% of GPs and 71% of nurses advised patients to stop smoking on most occasions.\(^17\)

This study has identified areas of progress in smoking cessation treatment for pregnant women, but also reveals some opportunities for improvement. The good news is that high proportions of midwives and GPs reported always asking about smoking. This is consistent with earlier studies which found that 65–74% of New Zealand GP obstetricians and 95% of midwives asked most or every woman about smoking status\(^13\) and suggests that more GPs may be asking about smoking than before.

We found that GPs were significantly more likely than midwives to ask about smoking status at the first visit for pregnancy. Nevertheless, almost all respondents saw it as part of their role to ask about smoking in pregnant patients.

Changes to maternity care funding over recent years has lead to a dramatic reduction in the number of GPs providing antenatal care. Despite this, we found that GPs are
still involved in confirming pregnancies and usually see pregnant women for this service during the first trimester. It isn’t until the second trimester that pregnant women nominate their LMC, usually a midwife.

Māori women who smoke are most likely to attempt to stop smoking within 2 weeks of finding out they are pregnant, usually in the first trimester and there is no reason to suspect that this is any different for non-Māori women. Health professionals involved in confirming pregnancy therefore need to be actively promoting smoking cessation and providing stop smoking assistance to pregnant women.

This study suggests that GPs are far more likely than midwives to be in this pivotal position, that is, upon confirmation of a pregnancy. Fortunately, this survey shows that GPs are also likely to offer appropriate advice—to stop smoking. Despite this, efforts to reduce smoking in pregnancy to date have largely focused on midwives of whom over half have not received training in this area.

Another concern is our finding that midwives were significantly more likely than GPs to advise women to cut down on their smoking with a view to quitting rather than to stop smoking completely, with a small number reporting that they do not advise reducing or quitting at all. While most GPs and midwives reported discussing the effects of smoking during pregnancy and effects on the fetus with pregnant women who smoke, GPs were significantly more likely than midwives to discuss smoking at every visit.

According to a recent literature review of cessation treatments, GPs and midwives should discuss smoking at every visit, advise their pregnant patients to stop smoking altogether and refer them to a dedicated cessation service. Cessation training and advice for midwives should give greater emphasis to the importance of advising and supporting pregnant women to stop smoking completely.

The most recent New Zealand Guidelines for smoking cessation recommend either referral to smoking cessation services, if the health professional has limited time and/or expertise, or providing support that incorporates setting a quit date, advising complete abstinence, arranging medication if appropriate and arranging a follow-up within a week. Our survey suggests that there has been an increase in the proportion of midwives providing cessation advice and counselling: 61% in our study compared with 55% in 2001.

This study supports other local research suggesting that practice nurses are an important provider of smoking cessation support. There is emerging evidence that nurses can have a similar impact to doctors when providing smoking cessation in primary care. As McLeod et al concluded this work should be adequately resourced, not only within practices but also at regional and national levels, to strengthen a commitment to smoking cessation in primary care.

Awareness of and referring women to the Quitline for cessation assistance appears to have increased markedly since an unpublished New Zealand survey by Cowan in 2000 when only around 60% of GPs and 44% of midwives had heard of Quitline. In our study only one GP and 3% of midwives said they did not know about the Quitline. Almost a third of GPs and midwives did not know of any Māori smoking cessation providers. Consistent with this, respondents indicated that they wanted more
information about these services. We also found low awareness of and referral to Pacific Island cessation services, but this is likely to be because only a few Pacific Island cessation providers exist and most of these are in the Auckland region.

Knowledge of the effectiveness of using NRT for pregnant women appears to have improved since 2001 when only 24% of GPs and midwives thought NRT was appropriate for use during pregnancy. In our survey, the form of NRT considered most effective and most likely to be recommended by both GPs and midwives was the patch. However, intermittent forms of NRT are now considered preferable for pregnant women because they deliver a lower total daily dose of nicotine than patches and are therefore less likely to lead to potential adverse effects on the fetus.

We found high support for the use of acupuncture and hypnosis among respondents, thus suggesting a widespread lack of knowledge of effective smoking cessation methods. GPs appeared to know more about a wider range of cessation products and treatments and were more likely to identify effective cessation methods than midwives, who were also significantly more likely than GPs to refer pregnant women who smoke for acupuncture to assist quitting, despite absence of evidence for this approach.

As was found in a 2001 survey, many respondents wanted better access to training, especially in the provision of cessation treatments such as NRT in pregnancy and during breastfeeding. Education for midwives based on the Guidelines for smoking cessation and an increased effort to raise the proportion of GPs and midwives who have read and practice according to the Guidelines has the potential to improve knowledge and use of cessation methods which are supported by evidence.

Our survey has a number of limitations. Firstly, the response rates were low, especially for GPs. This may reflect the extent to which GPs are being approached by researchers in general rather than a particular reluctance to respond to questions about their management of pregnant patients who smoke.

Some GPs may not have responded because they do not provide care for pregnant women. Despite this the study population was similar to the wider population of GPs and midwives with regard to age group and sex suggesting that it was representative in these factors at least. However, very few Māori GPs or midwives participated and even fewer were of Pacific Islands ethnic groups.

Midwives from larger urban centres may have also been under-represented due to the way the database was constructed. Secondly, our study relied on self reported responses so respondents may have provided the answers they expected the researchers to want, and engaged in smoking cessation interventions more or less often than they reported. However, as it was made clear to participants that the survey was anonymous we would not expect this to be a major source of bias.

With regards to strengths, the study methodology is consistent with earlier New Zealand surveys and has provided a useful and comparable snapshot of current provider knowledge, awareness and practices.
Conclusions

The knowledge and practices of New Zealand GPs and midwives with regard to many dimensions of smoking cessation appears to be improving, but there is still considerable scope for greater alignment with evidence-based practices.

Training in the provision of cessation advice has largely been targeted at midwives but women are still seeing GPs to confirm their pregnancy in the first trimester, the very time when they should be receiving advice and support to stop smoking or referral to smoking cessation providers.

Practice nurses are now playing an important role in smoking cessation and should have greater access to appropriate training. Training for midwives in cessation needs to be based on the current evidence for effective smoking cessation support. Whilst many midwives undervalue the importance of stopping smoking completely when pregnant, this study found there was a willingness to improve knowledge of effective smoking cessation treatments.

Competing interests: None known.

Author information: Marewa Glover, Director, Auckland Tobacco Control Research Centre, Auckland; Janine Paynter, Researcher/Policy Analyst, Action on Smoking and Health New Zealand, Auckland; Chris Bullen, Associate Director, Clinical Trials Research Unit, School of Population Health, University of Auckland, Auckland; Kay Kristensen, Research Consultant, Hamilton.

Acknowledgements: The study was funded by the Ministry of Health. We also thank the general practitioners and midwives who participated in this study; and Andrea King, Karin Batty, Billie Harbridge, Sarah Haines, Becky Freeman, Te Hotu Manawa Māori, Department of Pacific Health, University of Auckland, Ngati Whatua o Orakei Health, The Midwifery Council of New Zealand, Research Director, Department of Primary Healthcare and General Practice, Wellington School of Medicine and Health Sciences, Education for Change Ltd, Smoking Cessation Specialist, National Heart Foundation of New Zealand.

Correspondence: Dr Marewa Glover, Social and Community Health, School of Population Health, University of Auckland, Private Bag 92019, Auckland, New Zealand. Fax: +64 (0)9 3035932; email: m.glover@auckland.ac.nz

References:


Primary malignant melanoma in the anorectum: an uncommon cancer

Omprakash Damodaran, Anthony Morgan, Graeme Mendelsohn

Malignant melanoma of the anorectum is an uncommon and highly malignant condition. It represents 0.2%–3%\(^1\) of all malignant melanomas and 0.1%–4.6% of all anorectal tumours.\(^2,3\) The prognosis of this highly malignant condition is poor, with 5-year survival of less than 5% in several series.\(^1,3,5\) Anorectal melanomas are seldom suspected thus leading to delay in diagnosis and management. We report a case of this condition in a female patient.

Case report

A 76-year-old Caucasian female patient presented to hospital with a long history of rectal bleeding and constipation. There was no significant past medical history apart from hypertension. On physical examination, a partially fixed mass in the lower rectum was palpated. There was no clinical involvement of the inguinal nodes.

During colonoscopy a 4 × 5 cm sessile mass was identified protruding into the rectal cavity. This mass was approximately 2 cm above the dentate line and retractable outside the rectum as shown in the Figure 1. CT scan of the abdomen demonstrated multiple low density liver lesions, consistent with metastatic disease.

Figure 1. Rectal melanoma retracted outside the rectum
The patient underwent a local excision of the tumour at the time of the colonoscopy. Histopathological examination revealed malignant melanoma (Figure 2). The patient was referred to an oncologist and conservative management was planned.

**Figure 2. Histopathology demonstrating submucosal melanoma nodule**
* (haematoxylin & eosin; original magnification ×50)

**Discussion**

A melanoma is defined as a “primary rectal melanoma” when it occurs in the rectum above the dentate line. Although an uncommon cancer, it is still the third most common site for melanoma after the skin and eye. This condition shows a clear female predominance, with the percentage of females varying from 54% to 76%. The typical presentation of this tumour is rectal bleeding and sensation of a mass which is often misdiagnosed as haemorrhoids. Up to 60% of patients have metastatic disease at the time of diagnosis. The prognosis of anorectal melanoma is dismal.

The Mayo Clinic reported 5-year survival as 22% and disease-free survival as 16% in a population of 55 patients treated at the hospital. The absence of early clinical manifestation combined with overlapping symptoms and lack of suspicion leads to delay in diagnosis.

Primary treatment of malignant anorectal melanoma is surgery. There is controversy regarding the best surgical procedure which ranges from wide local excision (WLE) to abdominoperineal resection (APR). No statistically significant survival advantage has been demonstrated for APR over wide local excision when patients are compared by similar stages. Yap et al reviewed 17 large case series and concluded that APR should only be reserved for lesions not amenable to local excision or for palliative treatment of large obstructing tumours.

Anorectal melanoma is a relatively radiation insensitive tumour, but recently anecdotal responses to treatment have been reported. In view of the high failure rate...
with local excision, the option of adjuvant radiotherapy for local control should be considered. There are also individual case reports of successful palliation with intratumoural injections of interferon-beta in combination with chemotherapy in advanced anorectal melanoma.

In conclusion, this is an unusual mass causing recurrent rectal bleeding and malignancy as a possibility should always be considered. Anorectal melanoma is an uncommon condition with early metastasis and poor prognosis. Given the unfavourable prognosis of this condition, it is of paramount importance that the treatment is tailored to the patient’s quality of life.

Author information: Omprakash Damodaran, Resident Medical Officer, Department of Surgery, Liverpool Hospital, Sydney, Australia; Anthony Morgan, Advanced Trainee, Department of General Surgery, Liverpool Hospital, Sydney, Australia; Graeme Mendelsohn, Consultant-General Surgeon, Department of Surgery, Fairfield Hospital, Sydney, Australia

Correspondence: Omprakash Damodaran, Resident Medical Officer, Liverpool Hospital, Elizabeth Street, Liverpool, NSW 2170, Australia. Email: domprakash@hotmail.com

References:
A case of Wellens’ syndrome

Madan Joshi, Meera Kaphle

Wellens’ syndrome is a pattern of electrocardiographic T-wave changes associated with critical, proximal left anterior descending (LAD) artery stenosis.

Case report

A 45-year-old, otherwise healthy Caucasian male presented to the Emergency Department (ED) complaining of intermittent “band-like” chest discomfort primarily on the left side with occasional radiation to the neck and bilateral elbow regions.

The discomfort started after he was trying to lift a 15 kg car battery with his left hand. He initially felt a sharp, shooting pain from the neck down the left shoulder and arm. He later felt a band-like discomfort across his upper chest and became somewhat short of breath. He had no chest pain during the episode.

He was adopted and thus did not know history of premature coronary artery disease in his biological parents. He had no history of hypertension or diabetes. He was found to have high triglycerides and LDL cholesterol in a recent annual health check-up but was not taking any medications. He smoked 5–6 cigarettes per day and drank alcohol socially. There was no history of recreational drug abuse. He was an avid jet skier in the past and had injured his neck multiple times with subsequent development of cervical spine osteoarthritis.

He received intravenous morphine and sublingual nitroglycerin with relief of chest discomfort in the ED. Complete blood count, electrolytes, and cardiac injury panel were unrevealing. Chest X-ray was negative for any acute lung or cardiac pathology; 12-lead electrocardiogram (ECG) (Figure 1) revealed T-wave inversions in lead V2 and biphasic T-waves in leads V3 and V4. There were no prior ECGs available for comparison. He was given aspirin, beta blocker, a single dose of low molecular weight heparin, and admitted to the hospital to rule out acute coronary syndrome.

Serial cardiac enzymes measurements came back within normal limits. Fasting lipid panel showed elevated LDL cholesterol and triglycerides, and low HDL cholesterol. While in the hospital, he had two episodes of severe, shooting pain from the neck radiating to both upper arms associated again with the band-like chest discomfort. The pain took few minutes to subside after which he felt numbness and tingling in both hands.

A complete neurological exam was performed at this time and it did not show loss of power, reflexes or sensation in the involved extremities. The pain was not reproduced by neck movements. It was felt that his symptoms were primarily neurological especially due to cervical radiculopathy. Magnetic resonance imaging (MRI) scan of the cervical spine was performed in an urgent basis which showed cervical osteoarthritis at multiple levels. There was neural foraminal narrowing secondary to uncinate process spurs with impingement of the exiting C6 nerve root on the left.
Based on the MRI findings, the patient was started on nonsteroidals, muscle relaxant, and discharged home with spine clinic follow-up arrangements. An outpatient cardiac stress test was also set up to evaluate for coronary artery disease as his symptoms were quite atypical and he exhibited risk factors.

Over the next two days at home, the patient had several more episodes of similar pain with little relief from ibuprofen. He also began developing what he described as “smothering sensation” and was advised to return to the ED promptly.

On presentation to the ED, the patient was in distress because of severe chest discomfort, and was tachycardic and diaphoretic. A repeat ECG revealed deep T-wave inversions in precordial leads. New T-wave inversions were noted in leads V5 and V6 (Figure 2).

A stat bedside echocardiogram showed severe hypokinesis of the anterior wall, septum, and apex of the left ventricle. The patient was emergently taken to the Cardiac Catheterisation Laboratory for coronary catheterisation. Coronary angiogram revealed 99% occlusion of the proximal left anterior descending artery (LAD) (Figure 3). The lesion was successfully stented (Figure 4). He was admitted to the Coronary Care Unit (CCU) for further monitoring and follow-up. A repeat echocardiogram performed the subsequent day revealed normal left ventricular function with marked improvement in the previously noted regional wall motion abnormalities.

His chest discomfort improved substantially and he was discharged to home on standard medical therapy.

Figure 1. Initial ECG tracing
Figure 2. Readmission ECG

Figure 3. Coronary angiogram showing the high grade stenosis of the proximal LAD
Discussion

The patient in this case ultimately manifested the clinical findings consistent with Wellens’ syndrome. Wellens’ syndrome is a pattern of electrocardiographic T-wave changes associated with critical, proximal left anterior descending (LAD) artery stenosis.1 Syndrome criteria include T-wave changes plus a history of anginal chest pain without serum marker abnormalities, lack of Q waves, and significant ST-segment elevation with normal precordial R-wave progression. The T-wave abnormalities are persistent and may remain in place for hours to weeks.3

The initial ECG tracing (Figure 1) in this patient revealed T-wave changes in V2, V3, and V4 which was taken seriously and the patient was admitted to rule out acute myocardial infarction. However, the occurrence of radicular-like pain diverted the attention to cervical spine disease.

In retrospect, the initial ECG was quite classical for type 1 Wellens’ syndrome. This syndrome occurs in 25% of patients, and is characterised by subtle biphasic T waves in V2 and V3 and carries the same prognosis as type 2. Type 2 Wellens’syndrome presents with deep symmetrical T wave inversion in precordial leads.

In a study by de Zwaan et al, 18% of the patients admitted with the diagnosis of unstable angina show this pattern and 75% of those who did not get definitive intervention developed extensive anterior left ventricular infarction.2

The proposed management of outpatient stress test was dangerous in this patient as there have been reports of sudden cardiac death in patients with very similar ECG findings. A diagnostic dilemma did arise in the case because of the radiculopathy-like symptoms; however, vague, band-like chest discomfort were not clearly explainable with the MRI.

Figure 4. Coronary angiogram showing the successful opening of LAD lesion after PTCA and stenting
With further worsening of the symptoms and development of deep, symmetrical T-wave inversions in the precordial leads, it was obvious that the pain was cardiac in origin and ischaemic (type 2 Wellens’ syndrome).

The patient eventually underwent coronary catheterisation that showed the culprit lesion, and with the definitive intervention, his pain and discomfort subsided completely.

**Author information:** Madan Joshi, MD, Chief Resident; Meera Kaphle, MD, Senior Resident; Department of Internal Medicine, SUNY Upstate Medical University, Syracuse, New York, USA

**Correspondence:** Dr Madan Joshi, Department of Internal Medicine, SUNY Upstate Medical University, 750 E Adams Street, Syracuse, New York 13202, USA. Fax: +1 315 4645797; email: joshim@upstate.edu

**References:**


Screening, diagnosis and services for women with gestational diabetes mellitus (GDM) in New Zealand: a technical report from the National GDM Technical Working Party

David Simmons, Janet Rowan, Rosemary Reid, Norma Campbell; on behalf of the National GDM Working Party*

Abstract

Rates of gestational diabetes mellitus (GDM) and Type 2 diabetes in pregnancy are increasing with the epidemic of obesity. GDM is associated with significant perinatal morbidity and future risk of permanent diabetes in the mother and obesity and diabetes in the offspring. The recent Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) has shown maternal and perinatal benefits of managing GDM once diagnosed. The criteria for GDM are under review following the recent completion of the Hyperglycaemia and Adverse Perinatal Outcomes study (HAPO).

In New Zealand, the approach to identifying women with GDM or undiagnosed Type 2 diabetes has varied. The National GDM Technical Working Party reviewed the available data in the New Zealand context and recommend that (1) All pregnant women are offered screening for GDM backed up with relevant educational, systems and materials for health professionals and the women; (2) Criteria for GDM should remain unchanged pending further information (which should be actively sought); (3) Women at high risk of undiagnosed Type 2 diabetes in pregnancy should be screened at booking: the HbA1c was recommended as a practical initial screening test, but further research is needed; and (4) A structured, audited, population-based approach to managing women with GDM should be introduced in each district.

Background

Gestational diabetes (GDM) is associated with maternal (pre-eclampsia, caesarean section, and perineal trauma) and perinatal (macrosomia, stillbirth, shoulder dystocia, birth injuries, hypoglycaemia, respiratory distress, stillbirth, and jaundice) complications. GDM is also associated with an increased risk of later Type 2 diabetes in both the mother and the offspring. Pregnancy is the only time to identify women with GDM, and provides the opportunity to implement strategies to improve both pregnancy and long-term outcomes.

The diagnosis of GDM in New Zealand (NZ) is generally made using a two-step approach. An initial screening test involves a non-fasting 50 glucose challenge test (GCT) at 24–28 weeks’ gestation. Women are subsequently referred for a diagnostic 75g oral glucose tolerance test (OGTT) if the 1-hour glucose concentration is ≥7.8 mmol/L. Currently, GDM is diagnosed if the fasting glucose is ≥5.5 mmol/L and/or the 2-hour glucose concentration is ≥9.0 mmol/L. These diagnostic criteria have been used since
1992 and are unique to NZ. Many other countries use lower glucose levels to diagnose GDM.

The current criteria for diagnosing GDM are used throughout NZ, but the extent of screening for GDM varies markedly. Figure 1 shows the uptake of the 50g GCT by District Health Boards (DHBs) in 2005, ranging from approximately 20% to 89% of pregnancies. This is not surprising as, until the end of 2006, some organisations promoted screening for all pregnant women, while others recommended screening women with one or more of seven clinical risk factors. Similar discordance is also seen between different global screening recommendations.

There are several reasons for reviewing the current approach to screening for GDM. Firstly, since the most potent nihilistic view of GDM was published in 1993, a great deal has changed from both global and knowledge perspectives. The world now faces a pandemic of diabetes and obesity, which has resulted in growing numbers of young women with risk factors for GDM or undiagnosed Type 2 diabetes. Type 2 diabetes in pregnancy is associated with high rates of fetal loss, congenital malformations, and other adverse perinatal outcomes.

Figure 1. Proportion of births where the mother had a glucose challenge test for each District Health Board (DHB). Proportions are shown for women aged below and above 25 years.

Polycose (Glucose Challenge) Testing as a % of Live Births by DHB
Secondly, there is good evidence that the development of Type 2 diabetes in high risk populations can be prevented or delayed,\textsuperscript{15–17} thus providing women with GDM the chance to actively try to delay/reduce their chance of permanent diabetes. Interventions are potentially useful for their children, as the intrauterine and postnatal environment influence later health risks (including obesity) for the child.\textsuperscript{18–20}

Thirdly, two recent studies have made important contributions with respect to the value of treating women with GDM during pregnancy. These are the prospective, randomised Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS)\textsuperscript{21} and a case control study by Langer.\textsuperscript{22} Both studies showed significant benefit from the treatment of GDM and support other studies showing the benefits of tight glucose control during GDM.\textsuperscript{23,24} In addition, ACHOIS laid to rest concerns that a diagnosis of GDM could be associated with increased caesarean section rates or maternal psychological trauma. The caesarean section rates were not increased in women treated for GDM and maternal well-being scores were better in women treated for GDM.

With the combination of all these reasons it was timely to review the approach to screening, diagnosis and models of care for women with GDM in NZ. The Australasian Diabetes in Pregnancy Society (ADIPS) and the New Zealand College of Midwives (NZCOM) worked with representatives from other relevant organisations (Appendix 1) to develop a Technical Report to contribute to this debate. This paper describes the process undertaken and summarises the recommendations made.

**The National GDM Technical Working Party process**

An open national workshop hosted by ADIPS and NZCOM was held on 10 March 2006 to discuss screening for, and management of, GDM. Presentations included reviews of the current epidemic of obesity and diabetes in NZ. Presentations focussed on:

- Recent evidence confirming benefits of treating women with GDM;
- The inter-generational effect of exposure of the fetus to maternal diabetes;
- Interventions that reduce progression to Type 2 diabetes in high-risk populations;
- Potential long-term health benefits for women and their children by identifying and treating GDM; and
- The rationale for promoting a general, rather than selective, screening approach for GDM, and the controversies around the criteria for diagnosis of GDM and how these may be solved.

A smaller Technical Working Party was established with representatives from stakeholder organisations to develop a Technical Report. The first meeting was on 1 June 2006 to consider GDM within the unique circumstances of the NZ demography and health services. Four groups were formed to address the main issues that had been identified with respect to screening and diagnosis of GDM (described below). Each group provided a written summary of the evidence and made a number of...
recommendations. These contributed to the body of the Technical Report, which is available for use to ensure that appropriate care is available for women during pregnancy.

The draft report was circulated to the member organisations, which led to minor amendments being made. The final report is available on www.midwife.org.nz.

The four main issues are outlined below.

(1) Should all pregnant women without known diabetes be offered screening for GDM? If so, how?

There is consensus that screening for GDM should be offered in NZ, but a debate exists about whether to offer screening to all women or only those with risk factors. GDM fulfils the criteria for a condition warranting screening in NZ, as part of routine clinical care, not as a national screening programme (as occurs with cancers of the cervix and breast).

The benefits from screening for GDM are to:

- Reduce adverse pregnancy outcomes in women subsequently diagnosed and treated for GDM;
- Reduce risks in subsequent pregnancies by increasing the likelihood of preconceptual identification and management of undiagnosed diabetes; and to
- Provide education to women with GDM about their predisposition to Type 2 diabetes in association with advice about how to reduce this risk for themselves and (potentially) their children.

Risk factor-based approaches miss a sizable proportion of women with GDM. In NZ, even women with risk factors are not being screened—possibly due to the complexity of this approach. A routine offer of screening would reduce these issues.

When offering any screening test it is important that accurate information is provided so that women can decide whether to be screened or not. This includes information about the performance of the test itself, consistent with the Code of Health and Disability Services Consumers’ Rights.

Recommendations:

All women should receive an offer of routine screening for GDM

For this to occur, it is crucial that:

- Relevant health professionals understand the rationale behind the screening and diagnostic process, and are provided with resources to maintain currency so they are able to advise women and implement screening in a woman-focused manner;
- There is written information available about the implications of screening and diagnosis of GDM that is nationally consistent and easily understood by women;
• Women are informed about their management options should they be diagnosed with GDM and remain the central focus of the model of care provided;
• Women are informed about screening in a timely and appropriate way;
• There is documentation that screening for GDM has been discussed, relevant information provided to the woman and the woman has consented to screening; and
• There is a system to ensure that screening for and management of GDM are continuously assessed, to allow development of further improvements.

(2) What should the diagnostic criteria be for GDM in New Zealand?
Currently, there is no global consensus on criteria for diagnosing GDM.6 Glucose concentrations are a continuum with intra-individual variability, so there is no clear separation between a normal and an abnormal glucose level.

When deciding diagnostic criteria, a balance is needed so that women who would benefit from treatment are not missed but other women are not needlessly labelled/treated. While ACHOIS21 used a 2-hour cutoff of 7.8 mmol/L to diagnose GDM (also now recommended by the International Diabetes Federation29), the study was not large enough to show a “cut point” for benefit. However, in NZ, dropping from 9.0 mmol/L to 7.8 mmol/L (or 8.0 mmol/L as endorsed by the Australian branch of ADIPS) would increase the number of women diagnosed with GDM by an estimated 52%: numbers unable to be managed by existing diabetes in pregnancy services. Any change would need to consider resources and cost-effectiveness.

Moreover, a large study of 25,000 women across 15 countries (the Hyperglycemia and Adverse Pregnancy Outcomes study—HAPO) will report on the relationship between pregnancy outcomes and maternal glycaemia in 2007.30 The study is powered to relate the fasting, 1-hour, and 2-hour glucose levels to outcomes by 0.5 mmol/L increments, providing relevant data regarding diagnostic criteria for GDM.

Recommendations:
• Whilst there is evidence that a lower 2-hour glucose level during the 75 g OGGT is associated with a reduction in adverse pregnancy outcomes, there are no clear data demonstrating an optimal level. The Technical Working Party recommend that the status quo be retained and data reviewed again when the results of HAPO are published.
• Further NZ information should be collated:
  o To see if potentially different recommendations from HAPO are relevant to our population
  o To see what the impact of any change would be on the number of women diagnosed with GDM and resource implications.
  o To ensure that there are robust models of care that could be expanded to deal with the increase in numbers if any change to criteria was decided.
Currently, where NZ criteria for a diagnosis of GDM are not reached, but the 2-hour glucose is 8.0–8.9 mmol/L (the ADIPS-Australia criterion), and the clinician and woman have concerns, it would be reasonable to manage the pregnancy as for GDM.

(3) Should any pregnant women be offered earlier screening for GDM, and if so, who and when?

The issue of early screening (prior to 24–28 weeks’) is complex. Women with Type 2 diabetes have similar pregnancy risks to women with Type 1 diabetes, so it is logical to try and identify these women as early as possible during pregnancy. The aim of early screening would be to identify women with hitherto undiagnosed Type 2 diabetes, impaired fasting glucose (IFG), or impaired glucose tolerance (IGT). However, within the pregnancy population, the prevalence of abnormal glucose tolerance is low, so offering early screening to all women is not advocated. Also, the lack of a simple and accurate screening test in early pregnancy remains a difficulty.

The HbA1c may be the most practical test, as it can be performed with booking bloods. One difficulty is that a proportion of women with abnormal glucose tolerance will have an HbA1c within the reference range. The optimal way of identifying these women needs to be determined. The OGTT is currently the diagnostic test for Type 2 diabetes/GDM in early pregnancy.

While women with past GDM or glycosuria should be offered early pregnancy screening, other groups are harder to define but include:

- Polycystic ovarian syndrome (PCOS).
- Morbid obesity: (Ethnic-specific: European=BMI ≥ 35 kg/m², Polynesian=BMI ≥ 37 kg/m², Indian and Asian=BMI ≥ 32 kg/m²).
- Two first-degree relatives with diabetes.
- Previous unexplained stillbirth.
- Previous shoulder dystocia.
- Previous macrosomic baby (≥ 97th percentile based on customised birthweight chart). If there is no access to customised birth weight, ≥ 4700 g Polynesian, ≥ 4400 g European, ≥ 4000 g Asians (including South Asians).

Women with several weaker risk factors may also require early screening.

Recommendations:

- Women with known IGT/IFG or thought to have undiagnosed Type 2 diabetes should have an HbA1c requested at booking and be directly referred to the Diabetes in Pregnancy team for management.
- Women with a past history of GDM should have an HbA1c requested at booking (even if the postnatal OGTT for this woman was normal). If the result is above the reference range (≥ 6.0%), the woman should be referred immediately to the Diabetes in Pregnancy team. If <6.0%, an OGTT should be undertaken at the earliest opportunity, typically 14-16 weeks. If the OGTT is
normal, it should be repeated at 24-28 weeks (or earlier if clinical suspicion occurs).

- Women who have other high risks of GDM: The current practice of offering a diagnostic OGTT to women considered at sufficient risk of undiagnosed Type 2 diabetes should still continue. Measuring HbA1c as an initial screening test should be formally piloted and assessed to determine its role in this population.

(4) How should care be delivered for women with GDM?

The move to a universal offer of screening for GDM and the possible lowering of diagnostic thresholds for GDM are likely to increase demand for services for women with diabetes in pregnancy. Closer working relationships between the various health professionals involved with the women concerned could mitigate such an increase in demand. An approach to facilitate this is shown in Figure 2.

Figure 2. Proposed framework for care pathway for women with GDM in New Zealand
Recommendations for the provision of care for women with GDM:

- Care needs to maintain the focus on women becoming mothers, and on the birth of healthy babies, only part of which is the management of their GDM.
- All DHBs require a defined Diabetes in Pregnancy team.
- The process for screening for GDM should include:
  - The development and establishment of a programme to increase awareness of GDM in the population.
  - A comment on screening for GDM in general pregnancy information sheets.
  - All Lead Maternity Carers (LMCs) should have access to a Diabetes in Pregnancy team, with an agreed process for referral.
  - The development of a specific information sheet, written with extensive consumer consultation, containing balanced information, in the appropriate languages and at the appropriate educational level. This should be given to, and discussed with, each woman. Information relating to healthy eating and physical activity must be included. Ideally this should be available for women during early pregnancy, as it may guide their diet and activity and reduce later risk of GDM. It can be formally discussed at the time of the offer for a glucose challenge test. The sheet could include a graph of the optimal gestation to screen.
  - Screening being offered at the 24 weeks visit [unless earlier—see issue (3) above], and if agreed, to be completed between 26 and 28 weeks
but before the 28-week visit. Offers of screening should incorporate use of the information sheet and it should be documented that informed consent to screen was given by the woman.

- If the screening result is positive, the woman should be contacted by the person ordering the test to explain the result and refer for an OGTT. This test should be undertaken within one week and include the fasting and 2-hour glucose as a minimum.

- If the test results indicate GDM, the results will be explained by the person ordering the test, initial action should be initiated and the woman should be referred to the Diabetes in Pregnancy team.

- An ongoing continuing professional education programme should be provided to support primary care services and facilitate primary care and specialist service integration. Lab staff could be included in this in relation to screening

- A national ongoing monitoring system that monitors, at the DHB level, the proportion of women being screened, gestation at screening, gestation at OGTT, gestation at referral and gestation at first visit, linking with outcome data, should be in place. A system to ensure that women that have a homebirth are also included in the audit will need to be developed

- All DHBs should facilitate the local development and definition of a model of care that best suits their region that address the issues/principles raised in this report particularly:
  - All diabetes in pregnancy, including GDM, is high risk and needs careful monitoring (ultrasound, glucose, clinical).
  - All LMCs should have access to a Diabetes in Pregnancy Team and ultrasound scanning facilities.
  - A close relationship, particularly good communication, is needed between the woman’s primary healthcare team, the diabetes educator and LMC.
  - LMCs, primary healthcare, and the Diabetes in Pregnancy Team in each District should develop agreed standards of care and referral pathways based upon Australasian Guidelines.
  - The ability of midwives to provide dietary advice, glucose monitoring teaching, and management advice about diabetes in pregnancy is not a core competency for midwifery. This does not preclude that women need midwifery care and that some midwives have an interest in this area and will have additional education to provide care for women with GDM in conjunction with the diabetes in pregnancy service in that region.
  - The management of diabetes in pregnancy should be integrated with the woman’s primary healthcare team. This is essential to provide follow-up—e.g. annual/biannual OGTTs for women with past GDM
and they may be involved in initiation and community-based aspects of management of GDM.

- Those caring for women with diabetes in pregnancy need to be alert as the woman’s clinical condition can change rapidly.
- Those in primary care will need updating and ongoing education about GDM management including pregnancy-specific dietary, glucose monitoring, and overall information advice.

- Each district should consider participating in the ADIPS audit and benchmarking programme. All pregnancies complicated by GDM would be part of the audit programme as a result.

Comment

It is important for the Technical Report (including its recommendations) to be seen in the context of a constructive and collaborative response to the current need to address the diabetes epidemic as it impacts on pregnant women. Each participating organisation is reviewing the current situation, and each has its own priorities, whether this be clinical standards, informed consent, cost or other systems issues. We hope that this report places NZ in a position to improve the health of those women and their children affected by GDM.

Competing interests: None known.

Author information: David Simmons, Australasian Diabetes in Pregnancy Society, Hamilton; Janet Rowan, Australasian Diabetes in Pregnancy Society, Auckland; Rosemary Reid, Royal Australian and New Zealand College of Obstetricians and Gynaecologists, Christchurch; Norma Campbell, New Zealand College of Midwives, Christchurch; On behalf of the National GDM Working Party (see Appendix 1)

Acknowledgements: We are grateful to all study participants as well as the Ministry of Health, New Zealand College of Midwives, and Australasian Diabetes in Pregnancy Society for their support. Special thanks to Duanna Fowler for her assistance.

Correspondence: Professor David Simmons, Institute of Metabolic Science, PO Box 281, Cambridge University Hospitals NHS Foundation Trust, Cambridge CB2 0QQ, England. Email: dsworkster@gmail.com

References:


Appendix 1. Members of the National GDM Working Party

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<thead>
<tr>
<th>Representative</th>
<th>Organisation</th>
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<tr>
<td>David Simmons</td>
<td>Co-Chair</td>
</tr>
<tr>
<td>Norma Campbell</td>
<td>Co-Chair</td>
</tr>
<tr>
<td>Janet Rowan</td>
<td>Australasian Diabetes in Pregnancy Society</td>
</tr>
<tr>
<td>Margret Norris</td>
<td>DHB Maternity Managers Network</td>
</tr>
<tr>
<td>Pat Bent</td>
<td>Diabetes New Zealand</td>
</tr>
<tr>
<td>Barbara Beckford</td>
<td>Federation of Womens’ Health Councils</td>
</tr>
<tr>
<td>Lynda Williams</td>
<td>Maternity Services Consumer Council</td>
</tr>
<tr>
<td>Sandy Dawson</td>
<td>Ministry of Health</td>
</tr>
<tr>
<td>Peter Moore</td>
<td>New Zealand Society for the Study of Diabetes/Physician</td>
</tr>
<tr>
<td>Cat Wilson</td>
<td>New Zealand Society for the Study of Diabetes/Diabetes Nurse Specialist</td>
</tr>
<tr>
<td>Carol Perwick</td>
<td>New Zealand Society for the Study of Diabetes/New Zealand Dietetic Association</td>
</tr>
<tr>
<td>Estelle Mulligan</td>
<td>Ngā Maia o Aotearoa me Te Waipounamu</td>
</tr>
<tr>
<td>Nimisha Waller</td>
<td>NZ College of Midwives</td>
</tr>
<tr>
<td>Isabelle White</td>
<td>Pacifica Inc.</td>
</tr>
<tr>
<td>Jenny Valgrae</td>
<td>Parents Centre New Zealand Ltd.</td>
</tr>
<tr>
<td>Rose Elder</td>
<td>Perinatal Society</td>
</tr>
<tr>
<td>Rosemary Reid</td>
<td>Royal Australian and New Zealand College of Obstetricians and Gynaecologists</td>
</tr>
<tr>
<td>Don Simmers</td>
<td>Royal New Zealand College of General Practitioners</td>
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<tr>
<td>Kristen Berger</td>
<td>Women’s Health Action</td>
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The recent outbreak of plague in Auckland (part 3: autopsies)

Published in NZMJ 1908;6(26):7–12 and written by J S Purdy, District Health Officer, Auckland

(Continued from part 2 at http://www.nzma.org.nz/journal/121-1269/2935)

The following are the notes on the autopsies made by Dr. de Clive Lowe:—

Miss K.—Body that of a young female neither well developed nor well nourished. Age about 16 years. Post mortem, rigidity passing off, lividity well marked. No signs of external violence, Bile stained fluid oozing from mouth. Slight blood-stained discharge from vagina. At junction of right thigh with groin there appears swelling of tissues.

Right pleural cavity, 4 or 5 ozs. fluid, colour blood.
Left Pleural Cavity, 2 ozs. fluid, colour bloody.
Fluid in Pericardial Cavity, about 3 ozs., straw coloured.
Heart Flaccid: Left ventricle fairly full, right ventricle empty.
No Pericarditis to be seem.
Lungs healthy, nothing abnormal;
Liver normal in size; consistency and condition. Spleen very much enlarged, very soft and friable; small body probably accessory spleen found posterior and to left side of stomach.
Stomach over distended.
Heart: Small vegetations on Aortic Valve. Intestines and Appendix, normal; small intestines distended.
Uterus and ovaries, normal.
Inguinal Glands (right side): Superficial and deep in state of. haemorrhagic infiltration discovered; glands removed for examination by Health Officer.
Smears of blood from heart, spleen, and glands taken.
Right Extl. Iliac Glands, enlarged and haemorrhagic:
Left Extl. Iliac Glands, enlarged.
Left Inguinal Chain, enlarged and engorged.
No detectable enlargement of Popliteal glands.
Left Post. and Cervical Chain, enlarged and engorged.
Ant. Cervical Glands, enlarged and engorged.
Mesenteric Glands, enlarged and engorged.
Pancreas, not enlarged and haemorrhagic.

**Miss M.**—5ft. 5in. Badly nourished.
Aged appearance, 39
Expression of face, very drawn, cheeks sunken, pupils dilated.
On body ecchimosis on abdomen, upper part of thighs and arms,
Vaginal haemorrhage.
Distinct enlargement of right femoral and left cervical glands could be felt.
Nothing in Popliteal space.
Right pleural cavity, one ounce of fluid.
Left pleural cavity, three ounces of fluid.
Lung, fibroid patch left apex, otherwise normal.
Pericardium contains four ounces of straw-coloured fluid.
Heart removed, blood taken from left ventricle.
Liver, normal consistency, larger than usual.
Spleen removed, enlarged, friable.
Left kidney, normal.
Haemorrhage from right kidney into its capsule.
Pancreas, normal.
Glands Rt. Inguinal glands matted together, haemorrhagic, easily friable, enlarged, haemorrhage into adjacent tissues, especially femoral gland.
Right Inguinal Chain, enlarged.
Left Ext. Iliac Glands, enlarged.
Left Cervical Glands, post occipit and Cervical Chain slightly enlarged and engorged.
An occult infection in the setting of diabetes mellitus

George I Varughese, Abd A Tahrani, John H B Scarpello, Adrian B Walker

A 57-year-old man with a history of diabetes mellitus (DM) for 10 years, with suboptimal glycaemic control (HbA1c: 11.9%), presented with a history of pain on movements of his right shoulder joint of 3 days duration with no prior trauma.

Examination revealed an erythematous area over the shoulder joint and he had reasonable range of movements around the joint. He was initially febrile with a temperature of 38.1°C, but no temperature spike thereafter, and he was treated with intravenous benzylpenicillin, flucloxacillin, and later clindamycin. White cell count was raised at 18.9 × 10⁹/L (reference range: 4–11 × 10⁹/L), and C-reactive protein (CRP) levels were 399 mg/L (reference range: 0–5 mg/L) and remained persistently elevated. Blood cultures had grown group-B Streptococcus.

Magnetic resonance imaging (MRI) scan was performed after 2 weeks as his shoulder movements were not completely satisfactory.

Figure 1. MRI scan of the right shoulder joint

What is the diagnosis?
Multiple loculated shoulder abscesses

Discussion

MRI scan revealed an intact rotator cuff with multiple loculated abscesses which were interconnected within the soft tissue (Figure 1). These collections were mainly seen below the deltoid muscle extending anteriorly. Surgical drainage was carried out immediately and the patient remains well.

The subacromial/subdeltoid space should be evaluated in all cases of suspected glenohumeral pyarthrosis, and computed tomography (CT) or MRI may help to detect abscesses and indicate surgical therapy. X-ray findings can be normal in the setting of shoulder joint infections, and streptococcal infections can result in fatal complications such as necrotizing fasciitis.

Life-threatening infections have been reported in unusual sites in the setting of DM, and can very easily be overlooked. A high index of suspicion is required in such circumstances; more so in the setting of DM as any delay in diagnosis can have serious consequences.

Conclusion

The importance of a low threshold for further investigations in these patients cannot be emphasised more, as the potential for underlying spread of infection in patients initially presenting with what appears to be cellulitis is less commonly perceived in routine clinical practice.

Author information: George I Varughese, Specialist Registrar in Diabetes, Endocrinology & General (Internal) Medicine; Abd A Tahrani, Specialist Registrar in Diabetes, Endocrinology & General (Internal) Medicine; John H B Scarpello, Consultant Physician in Diabetes, Endocrinology & General (Internal) Medicine; Adrian B Walker, Consultant Physician in Diabetes, Endocrinology & General (Internal) Medicine

1. Queen’s Hospital, Belvedere Road, Burton-on-Trent, UK
2. University Hospital of North Staffordshire, Stoke-on-Trent, UK

Correspondence: Dr G I Varughese, Specialist Registrar in Endocrinology & General (Internal) Medicine, Diabetes Centre, Queen’s Hospital, Belvedere Road, Burton-on-Trent DE13 0RB, UK. Fax: +44 1283 593056; email: georgeiv@doctors.org.uk

References:


Occult inflammation

Sheng-Kang Chiu, Chih-Jen Cheng, Shih-Hua Lin

A 61-year-old woman has chronic kidney disease with anaemia and fever of unknown origin, but treatment for underproduction of erythropoietin is ineffective.

Gallium-67 citrate scintigraphy and colonoscopy (Figure 1 and 2) are the next steps.

**Figure 1.** Anterior view of gallium-67 citrate scintigraphy showing abnormal tracer uptake over the region of ascending colon (arrow) at the interval of 6 hours (left) and 24 hours (right)

**Figure 2.** Colonoscopy showing hyperaemic, nodular, friable mucosa including irregular ulcers with sharply defined margins

What is the diagnosis?
Diagnosis

The pathologic examination proved the diagnosis of colonic tuberculosis (Figure 3). Anti-tuberculous medication for 6 months cured the colonic tuberculosis.

Figure 3. Multiple caseous granulomas with epitheloid histiocytes, a few lymphocytes rimming the caseous necrotic area, and many scattered Langerhan’s giant cells throughout the mucosa to serosa (haematoxylin & eosin stain ×100)

Discussion

Impaired cellular immunity of uremic patients causes them a higher incidence of tuberculosis. Gallium-67 citrate scintigraphy is a reliable, useful, and non-invasive method of detection of an occult inflammatory process.

Colonoscopic findings of colonic tuberculosis are non-specific and include: hyperaemia, nodulation, friable mucosa, irregular ulcers with sharply defined margins, polyploid lesions, and cobble stoning. Diagnosis of colonic tuberculosis is difficult because symptoms are similar to those of Crohn’s disease, lymphoma, or gastrointestinal malignancy. In most cases of colonic tuberculosis, the diagnosis is made post-mortem or after exploratory laparotomy with surgical resection of the infected bowel.

Colonic tuberculosis can be detected by a gallium-67 scan and diagnosed by colonoscopic biopsy. In these cases, initiation of anti-tuberculosis therapy leads to rapid improvement.
Author information: Sheng-Kang Chiu, Chief Resident, Division of Infectious Disease and Tropical Medicine; Chih-Jen Cheng, Chief Resident, Division of Nephrology; Shih-Hua Lin, Professor, Division of Nephrology; Department of Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, ROC

Correspondence: Shih-Hua Lin MD, Division of Nephrology, Department of Medicine, Tri-Service General Hospital, No. 325, Section 2, Cheng-Kung Road, Neihu 114, Taipei, Taiwan, ROC. Fax: +886 2 87927134; email: shihhualin@yahoo.com

Reference:

Testosterone supplementation in older men

Serum testosterone levels decline significantly with aging. Testosterone supplementation in older men might beneficially affect the aging process.

In this study from the Netherlands, 237 healthy men between the ages of 60 and 80 years with a testosterone level lower than 13.7 nmol/L were randomly assigned to receive 80 mg of testosterone undecenote or a matching placebo twice daily for 6 months. The investigators reported that compared with placebo, testosterone supplementation was associated with an increase in lean body mass and decrease in fat mass but no improvement from baseline in functional mobility, muscle strength, cognition, or bone mineral density.

Another promising theory proven wrong.


Cytotoxic chemotherapy for oestrogen-receptor-negative breast cancer

It has been recognised for several decades that axillary node involvement and lack of oestrogen receptor activity predict a poor prognosis. Hence adjuvant chemotherapy. In this review of 96 relevant trials involving 20,000 women, the Early Breast Cancer Trialists’ Collaborative Group (EBCTCG) highlights the important achievements of the past 30 years in chemotherapy for oestrogen-receptor-negative early breast cancer. The chemotherapy involved was usually about six courses of CMF (cyclophosphamide, methotrexate, and fluorouracil) or fluorouracil, cyclophosphamide and doxorubicin, or epirubicin.

And the results in women younger than 50 years, and also between 50 and 69 years, demonstrate that these older adjuvant polychemotherapy regimens were safe (i.e. had little effect on mortality from causes other than breast cancer) and produced substantial and definite reductions in the 10-year risks of recurrence and death.

Possibly regimens involving taxanes and trastuzumab (Herceptin), when appropriate, may be even better.


Overuse of proton pump inhibitors?

Proton pump inhibitors are one of the most frequently prescribed classes of drug in the world because they combine a high level of efficacy with low toxicity. Although it might be assumed that overprescribing occurs mainly in primary care, evidence of inappropriate use of proton pump inhibitors in secondary care is abundant. In hospital inpatients taking proton pump inhibitors in Australia, Ireland, and the UK, 63%, 33%, and 67% of patients did not meet their country’s criteria for taking the drug.
And it is also happening here, as a report from New Zealand found that 40% of hospital inpatients were taking proton pump inhibitors inappropriately. A waste of money and exposing patients to unneeded adverse effects.


**Angiotensin-Converting-Enzyme Inhibitors (ACEi) versus Angiotensin II Receptor Blockers (ARB) for hypertension**

Yet another meta-analysis, this time comparing ACEi and ARB in the management of raised blood pressure. After reviewing 61 studies that directly compared these medications, the authors concluded that both have similar effects on blood pressure. They found no consistent differential effects for mortality, cardiovascular events, progression to diabetes, left ventricular function, or kidney disease. Cough was more frequent with ACE inhibitors than ARBs.

Good news, particularly as ARB prices are some 6 to 10-fold greater than the cost of ACEi.


**The internet and global warming**

This paper reports on a raft of studies which have highlighted the rocketing demands made by computers. One of them, a report from UK-based Global Action Plan, puts carbon dioxide emissions from information and technology on the same level as that of the aviation industry—2% of global emissions.

How could this happen? Partly because of energy wasters—for example a program that checks the email inbox 100 times per second even though the inbox only asks the server if there is new email every 5 minutes; a clock that updates every second even though it displays the time in minutes; etc.

Industry giants including Microsoft, Intel, Dell, IBM, and Sun Microsystems are worried and have forged a collaboration called the Green Grid to investigate.

SSRI antidepressants: Medsafe’s response to Professor Werry’s letter

I write in reply to Professor Werry’s open letter published on 14 December 2007.1

Professor Werry’s open letter reprises concerns raised with then-Health Minister the Hon Pete Hodgson and states in conclusion that he has not had any substantial response. However a response from the new Minister of Health the Hon David Cunliffe was sent to Professor Werry on the 19th of December.

In the interests of affording readers the same information as provided to Professor Werry I wish to respond to his concerns, as published in this journal, one by one.

Professor Werry wrote that he had contested the information contained in Medsafe’s media releases but had not had any response from Medsafe. Medsafe has no record of receiving any correspondence from him on this issue.

Medsafe and the Medicines Adverse Reactions Committee (MARC) have been monitoring the safety of SSRI antidepressants for at least the last 10 years. The potential for SSRI medicines to cause suicidal ideation, suicidal behaviour, or completed suicide has been monitored since 2002.

In early 2004, the MARC reviewed the available data regarding the efficacy and safety of SSRI antidepressants when used to treat Major Depressive Disorder in children and adolescents aged less than 18 years. The MARC concluded that the available data was inconclusive. While further data was awaited the following advice was issued to prescribers:

- Pharmacological treatment is second-line therapy in the treatment of Major Depressive Disorder in children. Specialist advice should be sought before prescribing any antidepressant for children or adolescents.
- Children and adolescents currently responding well to SSRI therapy should complete their course of treatment. Specialist advice should be sought if response is inadequate.
- SSRIs should not be stopped abruptly.
- All patients with depression should be monitored for the emergence or worsening of suicidal thoughts and behaviours.

In September 2004, the MARC reviewed further data on this issue including recommendations from the US Food and Drug Administration advisory committees, the New Zealand branch of the Faculty of Child and Adolescent Psychiatry, and published medical literature.
The MARC issued the following information and advice to prescribers:

- **SSRI risk/benefit in childhood and adolescent Major Depressive Disorder**—For childhood and adolescent Major Depressive Disorder the possible risk of suicidal ideation and behaviours with SSRIs generally outweighs the possible benefits. However there is some evidence of efficacy with fluoxetine and therefore it may have a favourable risk/benefit ratio.

- **Informed consent**—Use of SSRIs in children and adolescents may be warranted in particular circumstances. In these cases, individual risk/benefit discussions between doctor and patient/parent must be undertaken and informed consent obtained.

- **Monitoring**—All patients with Major Depressive Disorder should be monitored for the emergence or worsening of suicidal thoughts and behaviours regardless of whether they are taking an antidepressant medicine or not.

- **Specialist advice** should be sought before initiating, changing, or stopping any antidepressant therapy in children and adolescents.

The MARC’s advice did not prohibit the use of SSRIs in the treatment of adolescent Major Depressive Disorder.

Medsafe and the MARC have continued to monitor the risk of suicidal ideation and behaviours occurring in association with SSRI use. In June 2007 the MARC concluded that the new information available did not alter the risk/benefit ratio for the SSRI antidepressants and therefore no change was required to the advice communicated to prescribers in October 2004.

Medsafe notes that in March 2005 the Royal Australian and New Zealand College of Psychiatrists, along with the Royal Australasian College of General Practitioners and the Royal Australasian College of Physicians produced a clinical guideline on the use of antidepressant medications in children and adolescents. This guideline is consistent with the advice issued by Medsafe and the MARC.

Professor Werry contends that Medsafe’s position is depriving patients of choice. Medsafe has not received an application for the approval of any SSRI antidepressant, including fluoxetine, for the treatment of child or adolescent major depressive disorder. Therefore, no SSRI antidepressants are currently approved for use in New Zealand to treat child and adolescent Major Depressive Disorder.

Medsafe, as the regulator, cannot advocate the off-label use of SSRI antidepressants or any other medicine. However its advice to prescribers does not prohibit the use of SSRI antidepressants.

Professor Werry raises a concern that in the past year Medsafe has publicly indicated that all SSRI antidepressants have an unfavourable risk/benefit profile in the treatment of child and adolescent Major Depressive Disorder.

In December 2006 and February 2007 Medsafe released media statements summarising the MARC’s advice from October 2004 regarding the use of SSRI antidepressants in child and adolescent Major Depressive Disorder. The statement was designed to be simple and concise, hence the general statement “that when
treating children and adolescents with depression the risk of suicidal thoughts and behaviour with SSRIs generally outweigh the possible benefits from the medication” was considered to be appropriate. Medsafe does not consider this statement is misleading or indicates a change in Medsafe’s position from the October 2004 statement on the use of SSRI antidepressants in children and adolescents.

Professor Werry has asked that Medsafe issue a statement in response to five items. Medsafe’s responses are in italics following his comments.

1. Medsafe has misinformed the public about the use of SSRIs notably fluoxetine in adolescent depression, by overstating the risks and minimising the value.

_Medsafe, and its expert advisory committee the Medicines Adverse Reactions Committee, consider that the information disseminated to prescribers in October 2004 remains a fair reflection of the available evidence on the risks and benefits of treatment of child and adolescent Major Depressive Disorder with an SSRI. In addition, Medsafe’s position is in broad agreement with the position taken by other international regulators including the Australian TGA, the US Food and Drug Administration and the United Kingdom’s Medicines and Healthcare Products Regulatory Agency. In Medsafe’s opinion the studies you refer to in your letter are insufficient to reverse this position._

2. That its opinion was formulated knowingly over the objections of the child and adolescent psychiatrists in New Zealand.

_Medsafe has not received any recent correspondence from Professor Werry or any other member of the Faculty of Child and Adolescent Psychiatrists on this issue. It should be noted that the Faculty was consulted during the development of the “Dear Health Professional” letter distributed in October 2004._

3. That in the treatment of adolescent depression, an SSRI antidepressant fluoxetine has been shown to be effective with a clinically significant effect size.

_As indicated previously, while the Medsafe advice refers to evidence of efficacy for fluoxetine, Medsafe has not received an application to register fluoxetine for use in the treatment of adolescent Major Depressive Disorder. Therefore neither Medsafe nor the MARC has had an opportunity to fully evaluate the efficacy data for fluoxetine in the treatment of adolescent Major Depressive Disorder. As the Medicines Act 1981 forbids a company to promote a medicine for a use other than one approved by the Minister, it would be inappropriate for Medsafe to actively promote the off-label, that is, unapproved use of fluoxetine._

4. That CBT and fluoxetine are of probably comparable efficacy although at the moment the evidence favours the superiority of fluoxetine. Thus both treatments, either separately or in combination should be considered as the primary treatments of adolescent depression.

_As it is Medsafe’s role to evaluate the safety and efficacy of pharmacological treatments only, Medsafe has no current position on the use of CBT in the treatment of adolescent Major Depressive Disorder. In addition, Medsafe is_
unable to actively promote the off-label use of fluoxetine. As set out in the Medsafe advice of October 2004, prescribers should seek specialist advice before initiating treatment of major depressive disorder in children and adolescents, this will allow specialist assessment of the relative safety and efficacy of pharmacological and non-pharmacological therapies in that patient.

5. That some patients will prefer, be suited and have access to CBT but others will need medication as treatment of first choice because they prefer it, are unable to co-operate with CBT, have no access to CBT or have depression of such severity that CBT is vitiated.

This statement is entirely consistent with the Medsafe advice to prescribers namely that a prescriber should seek specialist advice on the most appropriate treatment option for that patient, including the option of pharmacological treatment, before treatment commences.

Medsafe has reviewed all the studies referred to in Professor Werry’s letter, although only the TADS study⁴ has been formally reviewed by the MARC. Medsafe is of the opinion that the studies provided, while supportive of the use of fluoxetine, do not resolve the issue of the safety or efficacy of the SSRI antidepressants in the treatment of MDD in children and adolescents.

Medsafe has provided MARC members with copies of these studies for their information at the MARC meeting in December 2007 and in addition has provided further studies for discussion at the MARC meeting in March.

In addition your readers will be aware of a recent meta-analysis published in PloS Medicine⁵ which concluded that SSRIs have limited efficacy even in the treatment of Major Depressive Disorder. As already indicated, only the manufacturer of fluoxetine can apply to Medsafe to have this medication approved for the indication of treating MDD in children and adolescents. Until this occurs, Medsafe cannot take a position of advocating the use of a medicine for an unapproved indication.

Stewart Jessamine
Manager
Medsafe
Wellington

References:

PHARMAC and cardiovascular health in New Zealand

The recent analysis by Stewart et al\(^1\) again demonstrates how socioeconomic factors are important determinants of health outcomes.\(^2\) The additional comparison between New Zealanders and Australians in the LIPID study is interesting, in that from the data available no single point of difference can be identified. It is unfortunate that the study design did not identify ethnicity, as it is well recognised that Māori and Pacific people (even in higher socioeconomic groups) do not fare as well as New Zealand Europeans.\(^3-6\)

It was therefore disappointing to see that the accompanying editorial (Ellis and Hamer)\(^7\) focused at least in part on a criticism of PHARMAC’s historical stance on the prescribing of statins. These criticisms have been answered on a number of occasions in the Journal,\(^8-11\) and these responses should be read as part of the debate.

Ellis et al use relative international pharmaceutical expenditure figures to argue that New Zealand spends too little on medicines. In fact this is a meaningless statistic, as the price of medicines varies enormously between countries and depends on their negotiating ability. They then congratulate PHARMAC for containing costs and insisting\(^12\) on best value for money.

The usage of statins in New Zealand was closely monitored between 1993 to 2002 using Special Authority approvals data. During that time, access was progressively widened; however at no time did we achieve a greater than 40% uptake for the targeted groups (20% at the time of the LIPID study). We have calculated the loss of life from non-uptake in eligible patients was 26% higher than that from road traffic crashes over that time.* If we are going to improve our population health status then the most fertile area is to ensure that those who are in need really do access appropriate care. By comparison, the potency of statins is a relatively minor issue.

Ellis et al refer to unnecessary death and morbidity. The price of statins in 1996 was so high that to treat everyone advocated by the 1996 NHF dyslipidaemia guidelines would have cost some an extra $147 million each year for 137,000 patients; more than one-quarter of all community pharmaceutical spending at that time.† For context, this level of spending is much higher than the entire cumulative year-by-year new investments PHARMAC has made over the last nine years—forgoing the health gains and costs savings to the rest of the health sector from funding gabapentin for neuropathic pain, beta-interferon, erythropoetin beta, atypical antipsychotics, venlafaxine, clopidogrel, lamivudine, low-dose aspirin, alendronate, and imatinib, to name but a few.

It should also be remembered that the original evidence\(^13\) for the use of statins was for secondary prevention, and good evidence for the benefits in primary prevention is relatively recent. It is not appropriate to use today’s evidence to judge yesterday’s standards.
Since 2003 there have been no restrictions on the use of simvastatin in New Zealand, and our usage now outstrips Australia—where there are still prescribing constraints (see graphs below).

We should remember that the LIPID study data is now 10 to 18 years old, and it is now time to look forward and address the problems. Our first priority should be addressing the equity issues, and to that end PHARMAC has already initiated the *One Heart Many Lives* campaign. This is a joint approach with DHBs and with close cooperation from the National Heart Foundation. It focuses on Māori and Pacific people and promotes general cardiac health, while at the same time using statin usage as a surrogate measure of outcome.

Ellis and Hamer quite correctly argue for greater clinical involvement in decision-making for heart disease. If well done, real health gains and efficiencies could be made. However, if clinicians do take a stronger role, they will also have to take responsibility for the budget, not only for cardiac disease but also negotiate with other areas of the Health Sector for a fair and equitable distribution—just like the managers have to do now.

Peter Moodie
Medical Director
PHARMAC
Wellington

**Graphs:**

![New Zealand and Australian use of statins since 2002](#)

Footnotes:
*Historical burden of disease from statin non-uptake in eligible patients, estimated from the gaps between estimated eligibility and actual dispensings, i.e. the estimated numbers of non-uptaking eligible people, and the consequent QALY losses from untreated cardiovascular disease (when compared with statin treatment, according to the eligibility criteria in place each month). Statin non-uptake over the 10-year period July 1991 to June 2001 meant that there were 6,930 ‘statistical deaths’ in New Zealand through missed opportunities to gain QALYs, from 115,000 potential QALY gains not realised. This number is 26% higher than the number of road deaths reported to the LTSA during the same time period (5,499).
†Year-by-year between 1998/99 and 2006/07 PHARMAC has invested $111 million for 173 new investments, for which $91 million for 65 investments initially benefitting some 286,000 patients are for medicines where we can estimate health gains, saving some 6,460 QALYs, with $38 million nominal savings elsewhere to the health sector (58% offsets) just in the initial 12 months of investments

References:


Documentation of cardiovascular risk factors

I thank Dr Rafter et al\(^1\) for their clear illustration that although an excellent concept—there is a great deal of work to be done with systematic documentation of cardiovascular risk factors before we can assess risks and then treat appropriately.

In late 2007 prior to the implementation of a cardiovascular risk assessment tool in our suburban general practice I conducted a similar audit.

Records of 10\% of the eligible male and female population in our practice were sampled.

Pleasingly 64.7\% of the men had all four risk factors recorded within the previous 2 years. However only 29.6\% of the women had the same levels of recording. One reason for the lack of documentation was that several of the audited records were for infrequent attenders. This audit provided an opportunity to recall those people for a cardiovascular risk assessment rather than relying on opportunistic screening.

Feedback to the practice team with regard to consistent recording of risk factors has also been done.

We look forward to a second cycle audit with improved levels of documentation.

Lynn McBain  
Senior Lecturer  
Department of Primary Health Care and General Practice  
University of Otago, Wellington  
and Brooklyn Medical Centre, Wellington  
(lynn.mcbin@otago.ac.nz)

Reference:

Meningococcal B immunisation in New Zealand: why haven’t we seen the data?

In mid-2004, the meningococcal B immunisation programme was introduced in Counties-Manukau followed by a progressive implementation throughout the country culminating in the nationwide programme by mid 2005. This roll-out had been preceded by several clinical trials. Achieving these impressive feats has required support, contributions and commitment toward the programme by many individuals, groups, and communities including governmental funding of approximately $200 million. Despite the support of the medical community, there has been only one publication in the *New Zealand Medical Journal* describing any of the outcomes of the trials or of the programme itself.¹

Recently, several reports have been published which provide some outcome data, but these reports are only likely to have been read by people with a special interest in the field. Martin et al. have published the comprehensive annual update of the epidemiology of meningococcal disease in New Zealand for 2006.² The incidence of meningococcal disease peaked in 2001 with 650 cases and has declined steadily since, as shown in Figure 1. 40-50% of cases occur in children under 5 years of age, and of those cases, 30% occur in children < 1 year and 20% in children aged 1–2 years.

**Figure 1.** Incidence of meningococcal disease in New Zealand from 1991–2006. The black shading represents the proportion of cases due to the epidemic strain between 2001 and 2006. Data from Martin et al.²
A seroresponder following immunisation is defined as a four-fold or greater rise in the titre of serum bactericidal antibody which is accepted as a surrogate marker of an effective immune response to immunisation. Oster et al reported data on the immunogenicity of the vaccine. These data are shown in Table 1 and suggest that, with the current administration schedule (6 weeks/3 months/5 months/10 months), 45% of infants are not seroresponders after 3 doses, increasing to 87% at the time of the fourth dose. It is not clear from these data how quickly immunogenicity wanes following the fourth dose.

Table 1. Proportion of seroresponders following 3 doses of meninogococcal B vaccine given every 6 weeks, and following a 4th dose. Data from Oster et al.

<table>
<thead>
<tr>
<th>Age at initial immunisation</th>
<th>% seroresponders 6 weeks after 3rd dose (age of child)</th>
<th>% seroresponders 4-5 months after 3rd dose (age of child)</th>
<th>% seroresponders 6 weeks after 4th dose (age of child)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-10 weeks</td>
<td>53% (6 months)</td>
<td>13% (8.5–9 months)</td>
<td>69% (10–11.5 months)</td>
</tr>
<tr>
<td>6-8 months</td>
<td>74% (10–12 months)</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
<tr>
<td>16-24 months</td>
<td>75% (20–28 months)</td>
<td>Not reported</td>
<td>100% (31.5-46.5 months)</td>
</tr>
</tbody>
</table>

Finally, Kelly et al reported that rates of meningococcal disease were 3.7 times higher in unvaccinated cases compared with vaccinated cases over the first 2 years of the immunisation programme. Models of disease incidence estimated that immunisation prevented 54 cases of meningococcal disease and 1.7 fatalities over this 2-year period. Taken together, these data, along with other data presumably gathered but not yet published, should allow important questions about the vaccine to be answered. These questions include:

- How does the observed efficacy of the vaccine compare with predictions of its efficacy?
- Is the current timing of immunisations appropriate and is/are subsequent dose(s) required?
- Given the decline in incidence of meningococcal disease, is nationwide administration appropriate or should immunisation be targeted at high-risk groups?
- How does the cost-effectiveness of the meningococcal B programme compare with other vaccines of proven efficacy that are currently not funded such as pneumococcal, varicella, and HPV vaccines?

Hopefully, the New Zealand medical community will be given the opportunity to see the results of the clinical trials and immunisation programme soon.

Mark Bolland
Research Fellow
The University of Auckland
Auckland
References:


The role of MeNZB vaccination in controlling the New Zealand meningococcal epidemic

New Zealand’s epidemic of meningococcal disease, which began in 1991, has now largely abated. An intensive immunisation campaign offered to all young New Zealanders under 20 years of age (2004–2006) with an impressive 80% coverage for 3 doses of vaccine has played an important role in this decline.\(^2,3\)

The role of vaccine in this process is likely to have been that of a “circuit breaker” with a large proportion of the population at greatest risk (the under 20 year olds) successfully immunised in a very short timeframe.

The reasoning behind the use of MeNZB, a strain-specific Outer Membrane Vesicle vaccine especially manufactured for New Zealand using the New Zealand serogroup B meningococcal epidemic strain was based on the following:

- If a group of individuals showed an antibody response, it was likely that that group would be protected by vaccination (the protective antibody level for an individual for serogroup B vaccines is still debated). This was based on published data from Brazil.\(^4\)
- Even very young children are able to mount a strain-specific antibody response and therefore could potentially be protected by a strain-specific vaccine.\(^5\)
- New Zealand’s epidemic was dominated (~80% at peak) by a single serogroup B meningococcal strain.

Three doses of MeNZB vaccine were recommended in most age groups knowing that 2 doses produced a short-lived effect, though by the nature of these vaccines it was unlikely that 3 doses would produce anything more than a short extension of protection.

Norway’s experience highlighted this issue: the first published report signalled a 57% vaccine efficacy (in school children) after 29 months\(^6\) but review by other investigators at an earlier time point (10 months) showed a better efficacy of 87%.\(^7\) Very young infants (at time of routine vaccines) required 4 doses of MeNZB to achieve an antibody response likely to protect.

Studies, ongoing at the time of the immunisation programme, have provided supportive information that indeed MeNZB vaccination is not likely to provide long-term protection.\(^8,9\) In the youngest age group studied (6–8 month old infants) only 12.5% were sero-responders, i.e. had antibody likely to protect at 7 months after the third dose of vaccine.

New Zealand has continued to offer vaccine to young infants who become eligible by age for the New Zealand vaccine immunisation schedule. Coverage for the fourth dose of vaccine necessary in this age group has been poor (less than 50%) (Ministry of Health data).
Thus, since late 2005, in the highest risk areas where the immunisation programme began (northern New Zealand), most children are likely not to have had antibody protection against the New Zealand meningococcal epidemic strain. Despite this, the decay of the epidemic continues.\textsuperscript{10} In addition, it is well documented that serogroup B meningococcal epidemics naturally decay over 10–15 years.\textsuperscript{10}

It therefore seems reasonable that MeNZB vaccination be withdrawn from the New Zealand infant immunisation schedule. The introduction of 7 valent pneumococcal conjugate vaccine in June 2008 could be an appropriate time. Invasive pneumococcal disease has now risen to number one as the most important vaccine-preventable invasive bacterial pathogen.\textsuperscript{11}

Diana Lennon
Principal Investigator Meningococcal B Vaccine Trials—Professor of Population Child & Youth Health—Paediatrician in Infectious Diseases
The University of Auckland
Auckland

References:

Sick notes

When, in 2006, it became clear that all was not well at Wanganui Hospital, we learned, amongst other things, that an employee had stuffed into a cupboard 166 referral letters sent by the general practitioners. There they lay lost and forgotten. Nobody seemed to notice, or even care. When this came to light, one of the GPs was asked why the doctors were not making use of private specialists in the town. He, or as it may be, she, explained that a private specialist was the “expensive option.” This comment makes the point that anything is expensive if there is a “free” alternative. Excepting, of course, an expensive surgical cock-up.

We do not know if patients complained as the months rolled by that nobody seemed to care much about their serious plight. However, patients seeking a taxpayer-funded sterilizing procedure who were not seen in consultation by the government-funded “gynaecologist” Dr Hasil may now be feeling that this administrative sloth was very much in their best interests. The bus fare to Palmerston North and the fee of a private specialist would have been a small price to pay to avoid Wanganui Hospital.

Patients will not pay for medical attention if they don’t have to. A recent study in the city of New York showed that a copayment of as little as $15 for a mammogram put some patients off having it done at all.

Specialist medical attention is too dear for most people, and private medical insurance is far beyond the reach of pensioners with limited resources. We like to direct a lot of flak at medical care in the United States, but at least they have Medicare for patients aged over 65. They cannot extend that scheme because it is going broke.

General practitioners cling to the concept of a variable copayment to be paid by the patient, but that fee will deflect a lot of people to the nearest free service. The government has thrown millions of dollars at general practice in the belief that if GPs charge lower copayment fees they will keep people out of hospital. What the new prosperity has done for GPs is shorten their working hours, exacerbate maldistribution, and raise their taxes. Patients are thronging Casualty Departments and Outpatient Departments in greater numbers than ever.

Once there, they have no choice of a medical provider. They seem to like it that way, and it is only a matter of time before another incompetent Hasil shows up in a full-time job somewhere else.

Roger M Ridley-Smith
Retired GP
Wellington
(r.sdekka@actrix.gen.nz)
Disgraceful Conduct (05/127C)

Charge

Dr Dhammika Pradeepa Dissanayake, of Christchurch, was charged with disgraceful conduct in relation to a patient (Ms B) whom he knew was diagnosed as suffering from psychiatric disorders.

The charge alleged that Dr D Dassanayake:

1. Had sexual intercourse with Ms B who was at the time, or had been until recently, his patient; and/or
2. Paid money in return for sexual services to Ms B who was at the time, or who had been until recently, his patient; and/or
3. Provided prescription only drugs to Ms B without prescription, and without proper medical reasons or justification for doing so and at the time when he was not Ms B’s medical practitioner and/or when he was not in a treating role with her; and/or
4. Gave Ms B advice on how to prepare a lethal dose of medication for her to use as a suicide tool; and/or
5. Following a complaint being made against him to the Health and Disability Commissioner in or about 2003 concerning his treatment of Ms B, paid to Ms B a sum of money in return for her not attending a planned interview she was to have with investigators from the Health and Disability Commissioner’s office in September 2003; and/or
6. Telephoned Ms B on the morning of the Complaint Assessment Committee’s interview of her (2 November 2004) in relation to the complaint made against him by a psychiatrist, and attempted to dissuade Ms B from meeting with the Complaints Assessment Committee in relation to that complaint.

Outcome

Particulars 1, 2, 5 and 6 were proved. Particulars 3 and 4 were dismissed as they were not proved to the requisite standard.

Background

Ms B first met Dr D Dassanayake in 1991 when she was in high school. He treated her for depression and anorexia.

When Dr D Dassanayake moved to another practice in 1992, Ms B and her family transferred to Dr D Dassanayake’s new practice. One of Ms B’s siblings subsequently died from a drug overdose. Ms B became very depressed and her anorexia very severe. Ms B started seeing Dr D Dassanayake once a week for counselling in around 1996/97.
In 1997 Dr D Dassanayake was convicted of a number of charges of fraud and other related charges and was sentenced to imprisonment for 12 months of which he served six months.

Ms B was an inpatient at a Psychiatric Hospital from July 1998 until April 2001. Dr D Dassanayake visited her in hospital once in 1999.

Other than one visit from Dr D Dassanayake at that time, Ms B did not see Dr D Dassanayake until around early May 2002 when she saw him at a local supermarket. They had a chat and he told her he was doing counselling. She said she would like to go back to counselling. He telephoned her at the Stepping Stone Trust residence where she was then living, saying he wanted to meet with her for coffee in a café.

They met at a café and Ms B said about a week later he telephoned her inviting her to his home that evening. Ms B said she went to his apartment that evening and Dr D Dassanayake made her a coffee while they talked for about one hour to one and a half hours about what had happened to her while he had been in jail. At that visit she said he hugged her and started kissing her.

Dr D Dassanayake denied that they had ever met at his house. He said he saw Ms B on a few occasions in a setting like the one in the coffee shop and that discussions they had were in relation to matters at Stepping Stone and other personal matters but that they did not discuss matters to do with past [family] issues as he felt that was a matter for Stepping Stone and its counsellors.

After the first visit, Ms B said she would have gone to Dr D Dassanayake’s apartment about once a week. She explained that on the first few occasions she went to his flat he would just hug and kiss her, but then later had sexual relations there.

When Ms B returned from her visits to Dr D Dassanayake’s flat she would tell the staff at Stepping Stone about her encounters with him.

She said that throughout their relationship he told her she should never leave any text messages he had sent her on her mobile phone and that she should wipe every trace of all of them. Ms B began to feel used by Dr D Dassanayake but although she was feeling used and, at times, angry with him, she kept on seeing him because every time she went to his flat he would say nice things to her.

On six or seven occasions when she was at his flat, Ms B said that Dr D Dassanayake gave her drugs always being clonazepam or temazepam. She said he would give her about six to eight tablets at a time and would make her take the medication at his flat saying it would relax her.

Dr D Dassanayake said he had never dated Ms B and had never had a sexual relationship with her nor had he given her drugs. Dr D Dassanayake said that all the meetings he had with Ms B were in his role as a support person and were prior to his re-registration and employment as a medical practitioner in late August 2002.

Around October 2002 Ms B got a severe kidney infection, and she decided to go and see Dr D Dassanayake at his practice in order to get a prescription for some antibiotics. Before she left his room he gave her a kiss. When they went out to reception he told the receptionist that he wanted her notes transferred to his practice.
She said they had not discussed this in his room but she was quite happy for that to be done.

During this time Ms B said she was still having sex with Dr D Dassanayake at his apartment about once a week.

During January 2003 Ms B said that Stepping Stone staff told her they were going to make a complaint to the Health and Disability Commissioner about Dr D Dassanayake, saying he was being unethical, that he was using her and that it was their role to make a complaint. She said this made her very angry because she did not want to get Dr D Dassanayake into trouble and told them she would have nothing to do with their complaint and would deny it all.

She reported this to Dr D Dassanayake one evening. When she told him that the staff were going to make a complaint she said his response was that Ms B and he would deny everything.

At the end of January 2003 Ms B was admitted to hospital after an overdose.

Ms B said Dr D Dassanayake was texting her and telephoning her most days and that after the complaint had been made by the Stepping Stone Trust she thought she saw him every day for a while. After a while she said they went back to seeing each other about once a week and were still having sex. She said Dr D Dassanayake kept telling her that she did not need the support of the staff and encouraged her to move out of the Stepping Stone residence. In early April 2003 Ms B moved out of Stepping Stone and went flatting.

On 10 April 2003, Ms B wrote to the Health & Disability Commissioner’s office informing them that she did not have any complaint about Dr D Dassanayake; that he had always acted professionally towards her; and that she did not agree with anything which the Stepping Stone Trust had said in its letter of complaint. Ms B told the Tribunal that Dr D Dassanayake told her what to write in this letter.

From around the time she left Stepping Stone in early April 2003 until early 2004, Ms B said her relationship with Dr D Dassanayake continued but on those occasions when she had sex with him he paid her for it at his insistence. On each occasion she said he would pay her about $30 cash and told her she had to keep quiet about their having sex otherwise she would not receive any more money.

Around the middle of 2003 Ms B was admitted to hospital again as she was suicidal. She was seeing her Therapist twice a week and was seeing her Psychiatrist about once a month. She started to tell them about Dr D Dassanayake and that he was paying her to have sex with him.

In August 2003 the Health & Disability Commissioner’s Office contacted her again and asked her for an interview as they were still investigating the Stepping Stone Trust’s complaint. She told them she would be interviewed.

Ms B said she met Dr D Dassanayake at a café where he got out his briefcase and helped her draft a letter to the Health & Disability Commissioner’s Office saying she did not want to be interviewed. She wrote down what he was dictating and that she told him she would post the letter but he insisted that he would post it himself. Ms B
said Dr D Dassanayake paid her cash to leave town on the day that she was supposed to be interviewed by the Health & Disability Commissioner’s Office.

Ms B said that Dr D Dassanayake continued to pay her for sex until early 2004 when she sent him a text message around March or April 2004 saying that she wanted to stop it.

As she had been quite open with the Stepping Stone staff (particularly at the earlier stages) about her relationship with Dr D Dassanayake, Ms B was similarly open with her Therapist and her Psychiatrist. She told them about the sexual nature of her relationship with Dr D Dassanayake and other associated events.

Around April 2004, her Psychiatrist told her she was going to make a complaint to the Medical Council about Dr D Dassanayake and later told Ms B she had made one. Ms B said by that time she was happy to support this complaint.

The evening before the interview with the CAC was to take place, Ms B stayed at Pathways Respite. The following morning while still at Pathways she said she received a call on her mobile phone from Dr D Dassanayake. She said he asked her if she were going to the interview. She said she was and that she had to be honest. She said he tried to dissuade her from meeting the CAC.

**Finding**

The Tribunal found Dr D Dassanayake guilty of disgraceful misconduct.

The Tribunal considered this case was essentially about credibility. The Tribunal found Ms B to be a truthful witness. The Tribunal was impressed with all of the Stepping Stone witnesses and Ms B’s Psychiatrist and found them to be credible and reliable witnesses. The Tribunal found Dr D Dassanayake to be an unreliable witness.

The Tribunal found Particular 1 and 2 were established and that Dr D Dassanayake had sexual intercourse with Ms B.

The Tribunal was not satisfied Particular 3 was established. The prosecution did not advance evidence as to how the doctor could have got the drugs or what access he had for them. In addition the Tribunal was aware from the evidence that Ms B would from time to time stockpile drugs. While Ms B’s account was not inconsistent with the rest of her evidence, it did not quite cross the threshold of proof and accordingly the Tribunal gave the benefit of the doubt to Dr D Dassanayake.

The Tribunal was not satisfied Particular 4 was established. The Tribunal was not satisfied that Dr D Dassanayake advised her on how to prepare a lethal dose for her use as a suicide tool.

The Tribunal was satisfied Particulars 5 and 6 were established. The Tribunal found Dr D Dassanayake paid Ms B money in return for her not attending an interview with the HDC in September 2003. It was further satisfied that on 2 November 2004 he attempted to dissuade Ms B from meeting with the Complaints Assessment Committee.

The Tribunal found that the conduct alleged in Particulars 1, 2, 5 and 6 either separately or cumulatively amounted to disgraceful conduct and was at the high end of it.
Penalty

The Tribunal ordered that:

- Dr D Dassanayake’s name be removed from the Register of Medical Practitioners pursuant to section 110(a) of the Medical Practitioners Act 1995.
- Dr D Dassanayake be fined $5,000.
- The Tribunal made no order as to costs. However, if the doctor had not been legally aided then the Tribunal would have ordered the doctor to pay 40% of the costs of the CAC investigation and prosecution and 40% of the costs of the Tribunal.
- A report of the Tribunal’s substantive decision and this decision be published in the New Zealand Medical Journal.

The full decisions relating to the case can be found on the Tribunal web site at www.mpdt.org.nz

Reference No: 05/127C.
Professional Misconduct Finding (04/123D)

Charge

Dr John Angus Marks was charged with professional misconduct pursuant to s802 and 109 of the Medical Practitioners Act 1995 (the Act) by the Director of Proceedings regarding his management of his patient (deceased) between 11 August 1999 and 16 October 1999. The particulars were as follows:

1. On or about 11 August 1999, or any time thereafter, he failed to:
   1.1 Undertake or document an adequate clinical assessment of his patient; and/or
   1.2 Undertake or document an adequate risk assessment; and/or
   1.3 Develop or document an adequate treatment plan;

And/or

2. On or about 10 September 1999, or any time thereafter, he failed to:
   2.1 Undertake or document a thorough and systematic review of his patient’s mental status; and/or
   2.2 Adequately formulate or document a diagnosis;

And/or

3. On or about 17 September 1999, or any time thereafter, he failed to undertake an adequate review and/or adjustment of his patient’s medication plan in light of his presentation;

And/or

4. On or about 8 October 1999 he failed to adequately communicate with his patient, and/or his patient’s partner, and or his patient’s parents regarding the advantages and/or disadvantages of admission to hospital.

Background

The patient was born in 1968. While he was at university and subsequently from 1987 through to January 1990 the patient’s illness began to manifest itself.

In February 1990 he suffered his first psychotic episode. He was diagnosed as having schizophreniform psychosis.

In April 1990 the patient attempted to hang himself. This was the first of a number of suicide attempts which the patient would make. There was some uncertainty about the patient’s diagnosis as he presented with both schizophrenic and affective disorders but while an inpatient he was diagnosed with schizophrenia with a differential diagnosis of bipolar disorder –depressed phase.
The patient made a further suicide in attempt in March 1991. In early July he was admitted for a third time, due to increasingly bizarre behaviour. The diagnosis of the patient’s illness was changed from schizophrenia to schizoaffective disorder.

For approximately two years the patient’s condition stabilised. He continued to live at home with his parents, working part time and was regularly seen as an outpatient.

On 31 May 1993 the patient was admitted to hospital. The sudden onset of psychotic symptoms followed the discontinuation of carbamazepine and a reduction in his dose of haloperidol earlier in 1993.

The patient made a further attempt at suicide on 16 June 1993 and was admitted to hospital. He was started on imipramine and continued on haloperidol. On 30 June 1993 he left the hospital without permission. That day the patient made two further serious attempts at suicide. He was re-admitted to hospital as a compulsory patient. The patient was continued on haloperidol by injections as a maintenance therapy for psychotic illness and daily carbamazepine in order to stabilise his moods.

On 22 October 1993 the patient was transferred to the community psychiatrist. Despite a family tragedy in 1996 the patient remained stable from the end of 1993 until July 1998. He was maintained on haloperidol and carbamazepine. During this period he was a happy and creative young man.

In 1998 a number of changes took place in the patient’s life. These included entering into a new relationship with a woman and suffering a break-in to his home during which he was the victim of a violent attack.

The patient also experienced a series of changes in relation to the health professionals involved in his care, including being changed to a new psychiatrist, Dr Marks.

By July 1998 Dr Marks had been assigned to take over the role of the patient’s treating psychiatrist. On 15 March 1999 the patient had his first consultation with Dr Marks. There were further consultations with Dr Marks on 9 April, 30 April, 28 May, 11 August, 10 September, 17 September, and 8 October 1999. The patient became increasingly unwell.

On 15 October 1999 the patient attempted suicide and died the following day as a result of his injuries.

Finding
The Tribunal found that the charge laid in all its particulars was established and that Dr Marks was guilty of professional misconduct.

Particular 1.1
All members of the Tribunal were of the view that Dr Marks failed not only to document an adequate clinical assessment of the patient but also failed to undertake an adequate clinical assessment of the patient at the consultation of 11 August 1999 or at the subsequent consultations.
Particular 1.2
The Tribunal agreed that a risk assessment is a fundamental requirement and a basic skill of a consultant psychiatrist, which it is critical to undertake. It agreed any failure in this respect, particularly where the patient has made suicidal threats, must be considered a very significant departure from the expected standard of care.

The Tribunal found that Dr Marks neither undertook nor documented an adequate risk assessment.

Particular 1.3
The Tribunal found on the evidence that Dr Marks neither developed nor documented an adequate treatment plan either on or about 11 August 1999 or at any time thereafter.

Particular 2.1
Dr Mark’s claimed that the patient’s condition would have been obvious to any other clinician perusing the records. The prosecutor submitted Dr Mark’s position seemed to be that the patient’s mental state could have been “worked out” by looking at the various entries in the notes and patching together an assessment of the patient based on the scant notes that were recorded. The Tribunal agreed with this submission and found that at no stage did Dr Marks undertake or document a thorough and systematic review of the patient’s mental state.

Particular 2.2
Dr Marks claimed that he had made a diagnosis of cycloid psychosis. However, there was no record in the notes when or how he came to that conclusion and there was no record of such a diagnosis or of a diagnosis of psychotic depression. The Tribunal accepted the evidence of the two expert witnesses who agreed that there was no evidence to suggest that Dr Marks ever appreciated the significance of the emerging psychosis despite the fact that Dr Marks claimed that he said the patient was suffering a psychotic depression.

The Tribunal found that at no time did Dr Marks adequately formulate or document a diagnosis.

Particular 3
The Tribunal accepted by the consultation on 17 September 1999 the patient’s depression was worsening and that he presented at the consultation with no improvement in his mental state.

At this consultation Dr Marks changed the patient’s medication regime by reducing the haloperidol and starting treatment with amitryptiline. The Tribunal found Dr Marks did not give any instruction regarding the titration of the amitryptiline, which he should have done. The Tribunal considered that Dr Marks did not appear to have recognised that the reduction in haloperidol which had occurred since Dr Marks took over the patient had led to a process of gradual deterioration into psychosis by the patient.
The Tribunal was satisfied that on or about 17 September 1999, or any time thereafter, Dr Marks failed to undertake an adequate review and/or adjustment of the patient’s medication in light of his presentation.

**Particular 4**

The Tribunal found the patient’s parents were certainly not left with the impression that Dr Marks considered the patient should be in hospital or that he was a high suicide risk or that he might kill himself if he kept taking the haloperidol.

The Tribunal found not only that Dr Marks failed to adequately communicate with the patient, his partner and his parents regarding the advantages and/or disadvantages of admission to hospital but that he did not communicate to them about hospital at all.

The Tribunal considered it was beyond question that had Dr Marks raised the issue of hospital admission and made it clear to the patient’s parents that the patient was a high suicide risk the patient’s parents would have done all they could to persuade the patient to enter hospital. The Tribunal found that Dr Marks was aware of the close relationship between the patient and his parents.

**Tribunal finding on matters which were not part of the charge**

Dr Marks blamed his employer for imposing conditions on him which did not allow him to treat his patient in the way he thought appropriate. He was directed to put into practice any advice which his supervisor gave him. In the Tribunal’s view, however, there was nothing to prevent Dr Mark’s from bringing his alleged concern that the patient was at high risk of suicide to the attention of his employer or his supervisor, or indeed to any responsible person (including the patient’s parents). He did not do so.

Dr Marks considered that but for the administration of haloperidol the patient may have survived. His supervisor directed that the patient should remain on haloperidol. The Tribunal accepted the evidence of the two expert witnesses that the standard treatment for a psychotic depression (which the facts established the patient had) was a dual treatment by the use of an anti-psychotic, such as haloperidol, in conjunction with an appropriate anti-depressant. Dr Marks did not present any persuasive or credible evidence to the contrary.

**Professional Misconduct or Conduct Unbecoming**

The Tribunal considered whether or not the charge, which was laid as professional misconduct, should be altered to conduct unbecoming. The Tribunal reached the view that the charge of professional misconduct was properly laid and the charge should not be altered.

**Penalty**

The Tribunal ordered Dr Marks:

- Be censured; and
- Fined $5000; and
• For a period not exceeding three years practise medicine subject to the following conditions:
  o That he be supervised and work in accordance with a supervision plan approved by the Medical Council of New Zealand.
  o That he be responsible for the costs associated with the supervision.
• Pay 30% of the costs and expenses of the investigation by the Health and Disability Commissioner and prosecution of the charge by the Director of Proceedings and 50% of the costs and expenses of the hearing by the Tribunal.

The Tribunal recommended that the Medical Council consider a further competence review of Dr Marks. If the Medical Council do undertake a further competence review the Tribunal was of the view that it should be in regard to the competency and safety of Dr Marks’ practice focusing on mental health assessment, management and documentation, and including:

• Management of patients with chronic psychosis;
• Clear communication of the rationale for his diagnostic and management decisions to the team with whom he works;
• Clear documentation of the rationale recording both negative and positive findings;
• Ascertaining and following the guidelines of his employer on clinical risk assessment and management.

The Tribunal also considered that should the Medical Council undertake a further review regarding Dr Marks it would be appropriate that any audit of Dr Marks’ files be selected at random and not ones which Dr Marks selected.

The Tribunal further ordered that a notice of the hearing be published in the New Zealand Medical Journal.

Appeal
Dr Marks appealed the substantive and penalty Decisions to the District Court. The appeal was partially successful.

When considering the particulars of the charge the District Court found as follows:

• Particular 1.1—the appeal succeeded as to failure to undertake, but failed as to failure to document.
• Particular 1.3—the appeal succeeded.
• Particular 2.1—the appeal succeeded as to failure to undertake, but failed as to failure to document.
• All other Particulars and sub-Particulars—the appeal failed.
The Court upheld the finding of professional misconduct. The Court upheld all the penalty orders except the order for supervision which was cancelled. (*Marks v Director of Proceedings* (District Court, Wellington, CIV-2005-001181, 25 September 2007, Broadmore DCJ)).

The full decisions relating to the case can be found on the Tribunal web site at [www.mpdt.org.nz](http://www.mpdt.org.nz)
Reference No: 04/123D.
Rosemary Angela Johnson

Dr Rosemary Johnson (Mrs Rosemary Colls), who died on 8 November 2007, was a paediatrician who was a pioneer in the field of newborn medicine. She was an enthusiastic at her subject and encouraged the careers of many of the current senior doctors in this country who now care for sick newborn infants.

Rosemary Johnson arrived from England, where she had trained at the Royal Free Hospital, to take up a post as a paediatric registrar in 1963.

In 1969 she was appointed as Resident Assistant Paediatrician with responsibilities for running the established Newborn Unit at Christchurch Women’s Hospital, under the leadership of Dr (later Professor) Fred Shannon.

She became one of a select group of largely single-handed neonatal practitioners, up all hours of day and night. She used to say that it was her love of the babies under her care, which kept her going.

Rosemary made particular contributions in the air-transport of sick newborn babies. Together with two enterprising Christchurch engineers, Tony Blackler and the late Bruce Lill, she modified equipment which could be carried by the Air-Force to retrieve babies from the West Coast and as far away as the Chatham Islands. Transport equipment was also modified to allow babies, sometimes needing assisted ventilation, to be flown with a team on ordinary commercial flights to the children’s cardiac unit at Green Lane Hospital, in Auckland. Many babies, who would not otherwise have survived, were transported in this way.

In the days before newborn units had their own technical staff, Rosemary Johnson would undertake much of the maintenance herself. She was often to be seen about the unit with screwdriver in hand. In the early days, the needs of sick newborn infants were not high on the priority lists of hospital managers and Rosemary battled long and hard for appropriate resources for her patients. She was involved in several earlier plans for a new Women’s Hospital with an upgraded Neonatal Unit and she was thrilled when the new hospital was finally built on the main hospital campus long after her retirement in 1990.

Those who worked with Rosemary Johnson particularly remember the way she treated the babies as individuals, talking to them and watching their responses. Dr Simon Rowley, now a paediatrician working in Auckland, said that she gave babies a dignity and humanity that was in many ways ahead of her time. It was this approach
to her calling which captured the imagination of trainee doctors and led some of them into the specialty of newborn medicine.

A hobbyist photographer, she used her retirement to develop this into a semi-professional pursuit. She did a diploma course in photography and over 10 years took and developed many pictures. She was a purist, who preferred artistic black-and-white photography. Her main subjects were nature, especially birds, but family affairs were also well documented.

She also took wedding and function photographs. She produced photographic essays on places she visited, some of which appeared in the travel sections of magazines. Her interest in aviation led her to take photographs of aircraft. A shot of an Air New Zealand jumbo jet taking off was bought by the airline for advertising purposes.

Rosemary married Dr Barry Colls, a Christchurch Hospital colleague, in 1968, and they had 2 children, Rebecca and Andrew. She is survived by them and 5 grandchildren.

Rosemary lived her life by the motto

There are only two lasting bequests we can hope to give our children. One is roots; the other, wings.

Professor Brian Darlow and Barry Colls wrote this obituary with some assistance by Mike Crean.
Neville William Hogg

(20/8/27–24/11/07)

Dr Neville Hogg was a true son of the North, where he was a respected general practitioner, a prominent Catholic layman, and a noted art collector as well as being deeply involved in Māori history and welfare.

Neville was born in Dargaville, the son of a motor vehicle dealer and a nurse. He was educated at St Joseph’s Convent Dargaville, Sacred Heart College in Auckland (when it was located at Ponsonby), and Otago University, where he graduated in medicine in 1951.

After 2 years in Auckland as a house surgeon he returned to his hometown to set up a general practice. This was initially solo, but in the mid-1960s Neville joined with Drs Maurice Matich and Phillip Barham to establish the Dargaville Medical Centre, something of a novel setup for rural districts at the time. Apart from his practice, Neville became involved in the health of the wider community, serving as a member of the Dargaville Hospital Board, including a term as Chairman.

His interest in the public field extended in particular to the Māori people of the North, among whom he was a well-known and trusted figure, deeply immersing himself in Māori history and spirituality as well as purely medical matters.

Neville was a notable art collector and he had a large collection, especially New Zealand art—his interest was born out of a fascination with New Zealand history.

Following retirement from his practice Neville travelled extensively including undertaking charity work for Mother Theresa’s sisters in Calcutta and elsewhere. In France he managed to locate the grave of Bishop Pompallier, and was instrumental in having the Bishop’s remains brought back to New Zealand.

Tragically Neville was laid low by a severe stroke late in 1992, and he was nursed and cared for from that time with great dedication by the Little Sisters of the Poor at their home in Auckland.

Funeral services were held in Auckland and Dargaville, and Neville was buried at the Kaihu Cemetery.

Dr Hogg is survived by two sisters, Mary Dunn and Mabel Hogg.

Dr Bill Brabazon, a Dunedin classmate of Neville, wrote this obituary from material provided by Mrs Dunn and Mrs Matich. The Dargaville District News, the NZ Catholic newspaper, and The Grapevine (published by the Little Sisters of the Poor) are also gratefully acknowledged.
Graeme Bertram Blake

Leading plastic surgeon Graeme Blake, who gave his services freely to disfigured children in Nepal, has died at his Christchurch home. He was 69.

Blake travelled eight times to Nepal, with his wife, Brenda, to do cleft lip and palate surgery in a remote clinic. Not only was the work voluntary, but Blake and others in the mainly Australian party contributed funds so patients could make the pilgrimage from their mountain homes to the clinic.

Brenda Blake says she and her husband came to love the place and the people and looked forward each year to going back. Her husband had always felt at home in the mountains, after being a Queen's Scout in his youth.

His favourite leisure pursuits included tramping, skiing, and fishing in the Southern Alps. The congregation at his funeral sang his favourite song, Climb Every Mountain.

Blake was born in Palmerston North but grew up mainly in Wellington, attending Kelburn Normal School and Wellington College. After his medical intermediate year at Victoria University, he studied medicine at Otago University from 1958 to 1961. He met Brenda, a fellow student, at Dunedin and they married in 1962. They had four children.

Blake completed his studies at Christchurch Hospital in 1962, then served there as a house surgeon. He returned south in 1963 and worked as a registrar at Dunedin Hospital for 3 years.

In 1968, the Blakes embarked on the ocean liner Southern Cross, bound for England and specialist surgery studies. Blake signed on for the voyage as the ship’s assistant surgeon, which gave him free passage.

He was concerned at the lack of openings in plastic surgery in Christchurch and considered a career in orthopaedics. However, in London he met a former Burwood Hospital plastic surgeon who convinced him of the future of plastic surgery in Christchurch, so he followed that course.

Blake completed specialist studies at Mount Vernon and Roehampton hospitals and was awarded a Commonwealth Medical Fellowship in 1971. This allowed him to continue working in England, where he carried out much cleft lip and palate surgery.

He and his family returned to Christchurch in late 1972. Blake was employed first as full-time plastic surgeon at Burwood Hospital. He opened a private practice in Merivale in 1974 and became a visiting surgeon to Burwood and to the South Canterbury Hospital Board at Timaru. He became a lecturer at the Christchurch Clinical School of Medicine in 1976.

As head of the Burwood Plastic Surgery Unit from 1987 to 1995, Blake did much to expand and upgrade it to international standard and maintain that standard. He was
influential in moving the unit to Christchurch Hospital, where better emergency support systems were available. He retired from the hospital service in 2002 and from private practice in 2006.

Blake chaired the NZ Association of Plastic Surgeons for a term and was an examiner for 8 years. He was a board member of the Royal Australasian College of Surgeons and Chairman of its NZ Training Committee for Plastic Surgery from 1989 to 1995.

His annual March-April visits to Nepal began in 1997, when Australian colleague and friend Charles Sharpe invited him to join a group working in a Seventh Day Adventist clinic. Sharpe had established the service and needed a lip and palate specialist.

Brenda Blake says the clinic was 2 hours by tortuous road from Kathmandu. Her husband operated mainly on children born with facial deformities that would normally have been corrected at birth in Western countries. He trained local medical staff to continue the work.

“The people (in Nepal) were grateful and gracious, we loved doing the work. It was a marvellous experience which we looked forward to each year.” After each stint at the clinic, the Blakes and some colleagues embarked on mountain trekking and camping expeditions to see more of the country. “It was fantastic. Camping was no bother,” Brenda Blake says.

Daughter Prue Blake says her father was an enthusiast for life and an eternal optimist. He had “boundless energy.” He loved people and was interested in what everyone was doing. He had a great sense of humour. His advice was often sought because he considered all sides of an issue and took a balanced view. Brenda Blake says he was keen but inexpert at golf and tennis.

His mechanical workshop would have been the envy of many. There he made toys for the grandchildren whom he doted on. He was “a frustrated architect”—if anyone mentioned building or altering a building, he would whip out his drawing board and begin designing the project.

Friend and colleague Dr Ted Mayell says “Blue” (as Blake was known) was a dedicated husband and family man. As a medical leader, “he was extremely positive and encouraged others to step outside their boundaries. He was a man of scrupulous ethics.”

Graeme Bertram Blake, born Palmerston North, December 28, 1938; died Christchurch, January 7, 2008. Survived by wife Brenda, daughters Prue and Katie, sons Andrew and James, and 10 grandchildren.

This obituary entitled Surgeon helped Nepal’s children appeared in The Press newspaper (Christchurch) on January 19 and was written by Mike Crean.
**Ernest (Tim) D L Marsden**

Born in Wellington, the only son of Sir Ernest and Margaret Marsden, Tim’s childhood was happy and included holidays at Raumati South, and he had a vivid memory of meeting Lord Ernest Rutherford when he was 8 years old. He was educated at Wellington College and Nelson College.

In 1942 he qualified BSc at Canterbury University and was then seconded to work as a Radar Officer in the RNZAF; stationed at Collingwood, North Cape, and Mokohinau Island. After the War he went to Dunedin and for 1 year worked under Professor Eccles on electrical equipment.

In 1947 he commenced his medical studies in Dunedin and a romance began with fellow medical student Adair, culminating in marriage in February 1951. In their final year the “twins” (as they were called) lived in a hospital flat in Cumberland Street.

They spent the weekends working at Seacliff Mental Hospital—and with tinned food provided by Seacliff, and hot water and electricity by Dunedin Hospital, they had a very economical year.

After a house surgeon year at Waikato Hospital, he “repaid” his bursary by working at Porirua Mental Hospital and then joined Adair in general practice at Titahi Bay.

One evening they were desexing a dog—ether for anaesthetic and diathermy to stop the bleeding—the inevitable happened: one dog on fire but the patient lived! The years of general practice at Titahi Bay were the most enjoyable. Home deliveries were the “norm”, and later, when his car was fitted with a radio-telephone, he attended many motor accidents on the Wellington Motorway. It was a real family practice, with children coming to the surgery with their friends and not always their parents (to patch up bumps and scrapes)!

In 1982 after doing a few locums, he bought a practice from Dr Grant in Devonport, Auckland where he worked until selling the practice to Dr Chris Diggle in 1984. In Devonport he was a member and then President (in 1982) of the local Rotary Club and served two terms as a Councillor of the Devonport Borough Council.

He worked at the Belmont Surgery (known as the Devonport Medical Centre) till 1987 when the practice was sold to Dr Greg Simmons. The following year the couple went to Australia and worked in large Medical Centres in Blacktown (Western Sydney) and Canberra till finally returning to New Zealand in 2003.

He was elected FRNZCGP in 1998, and in 2000 was awarded the “Gibbie Abercrombie Plate” by members of the North Shore Division of the NZMA.

He enjoyed people; he was quick witted but probably his greatest talent was his ability to show empathy and “crack a joke” without offending anybody. He relished maternity work, and became very adept at using forceps.
He was always learning new things and was an “early adopter” of computers, mobile phones, and digital cameras. He was a “gadget man” and seldom attended a Home Show without purchasing a new toy or two. At home he was a “Jack-of-all-trades” fixing everything about the house and garden—electrical, plumbing, and carpentry. There was usually a cat or dog around as he loved animals, however his main hobby was fishing and he always owned a power boat.

A family man, he was good natured and seldom got ruffled. He was very proud of his children and involved himself in his sons’ various activities. In his later years, he became very interested in history and classical books, an interest he shared with his daughter.

Following a stroke in September 2007, one thing led to another and he passed away peacefully on 9 January 2008 in his 86th summer, to be sadly missed by his friends and family (wife Adair, 3 children, and 5 grandchildren).

Dr Adair Marsden wrote this obituary.
Computerworld Excellence Awards 2008: Calling for Technology Entries from the Health Sector

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

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

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

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

For a full category listing, eligibility details and entry process information, please go to computerworld.co.nz/awards or contact Claire Baker on 09 375 6050 or email Claire_Baker@fairfaxbm.co.nz
Verbal Autopsy Standards: ascertaining and attributing cause of death


This manual is a consensus of a 3-year effort by an expert group of the World Health Organization (WHO) consisting of researchers, data-users, and others of the Health Metrics Network, to obtain mortality data from Third World regions.

A verbal autopsy is first obtained from family and associates of the recently deceased, by using standard questions for different age groups and types of disease. These templates are to facilitate the classification of deaths according to the International Statistical Classification of Diseases and Related Health Problems (ICD-10). This classification favours recording the underlying factors such as diabetes, war, HIV, etc before the precipitation of death, such as infarct or bullet.

Robust mortality statistics will lead to the better targeting of health funding towards the world’s poor from government and private organisations, e.g. the Gates’ Foundation.

The previous lack of health data was highlighted recently in a Lancet series of reviews The way forward.¹ Our system is based on that of Britain, which took three centuries to hone, yet our own champion of diabetes, Professor Don Beaven was dismayed by diabetes not being noted in half of our autopsy reports into death from cardiovascular disease.² The cause of death ascertained by clinicians with sophisticated diagnostic aids such as scans and biochemistry tests have been shown to vary from autopsy findings in about 30% of cases ³.

I admit bias as an ageing morbid anatomist with pathological paternalism but believe the questions may overwhelm the recently trained worker. The lists are extremely detailed. For a field worker in New Guinea with 400 different languages, the task will be daunting.

Collation of the data involves computers for rapid correlation but this depends on the accuracy of the data, and correlations do not necessarily imply cause and effect.⁴

Despite my negative feelings as to the immensity of the task, and that the proposed methodology is too detailed, the aim of the exercise is all important in improving World health.

Hopefully pilot studies will test and refine the data collection.

Robin Fraser
Pathologist
Anatomical Pathology
Canterbury Health
Christchurch
References:


