CONTENTS

This Issue in the Journal
3 A summary of the original articles featured in this issue

Editorials
5 Leprosy in the South Pacific
   Stephen T Chambers
8 Allowing violence in New Zealand hospitals is bad business
   Steven Kelly

Original Articles
10 Patient aggression experienced by staff in a New Zealand public hospital setting
   Nicola Swain, Chris Gale, Rachel Greenwood
19 Why do patients self-present to Middlemore Hospital Emergency Department?
   Vanessa Thornton, Annie Fogarty, Peter Jones, Nouran Ragaban, Catherine Simpson
31 Premature mortality in adults using New Zealand psychiatric services
   Ruth Cunningham, Diana Sarfati, Debbie Peterson, James Stanley, Sunny Collings
42 Risk factors for general medicine readmissions and association with mortality
   Manaf Aljishi, Ketna Parekh
51 Does seasonal level of serum 25-OH vitamin D correlate with the activity of Crohn's disease?
   Geogry Peter Kini, Brian Young, Peter Herbison, Michael Schultz
60 From ICU to hospital-wide: extending central line associated bacteraemia (CLAB) prevention
   Mary E Seddon, Catherine J Hocking, Elizabeth A Bryce, Jackie Hillman, Vicki McCoubrie

Viewpoint
72 Rising antimicrobial resistance: a strong reason to reduce excessive antimicrobial consumption in New Zealand
   Mark G Thomas, Alesha J Smith, Murray Tilyard
Clinical Correspondence

85  Endocrine encephalopathy
    King W Yong, Steven Soule, Penny Hunt

88  Medical image. A benign glossal lesion
    Tarun Gupta, Naveen Kumar Kansal

Letters

91  The leprosy problem in the South Pacific
    Colin Crawford

94  Chewing the saturated fat: should we or shouldn’t we?
    Simon Thornley, George Henderson, Grant Schofield

97  Performance of funded point-of-care capillary blood glucose meters at altitude
    Steven C M Miller

100 Years Ago in the NZMJ

102  Case of hydroa aestivale

Methuselah

104  Selected excerpts from Methuselah

Book Reviews

106  The Autistic Brain (T Grandin, R Panek)
    Liane Dixon

108  The Women’s Health Book. A complete guide to health and wellbeing for
    women of all ages (The Royal Women’s Hospital, Victoria)
    Anna J Fenton

110  Migration, Ethnicity, Race and Health in Multicultural Societies (2nd edition; RS Bhopal)
    Rebecca Pascoe

112  The Rise and Fall of National Women's Hospital. A history (L Bryder)
    Peter Sykes
This Issue in the Journal

**Patient aggression experienced by staff in a New Zealand public hospital setting**
Nicola Swain, Chris Gale, Rachel Greenwood

Most hospital staff experience violence or aggression from patients in New Zealand. This is worst for nurses but happens to most staff. More than one-third of staff get physically assaulted each year, resulting in physical injury for just under one-third of those. This is higher than our rates of domestic violence, but nothing is being done.

**Why do patients self-present to Middlemore Hospital Emergency Department?**
Vanessa Thornton, Annie Fogarty, Peter Jones, Nouran Ragaban, Catherine Simpson

Almost 25% of self-presenting patients had contacted their GP or a health professional prior to their Emergency Department (ED) presentation and were advised to attend Emergency Department. The most common reason for patients to self-present at Middlemore Hospital Emergency Department is the belief that a hospital emergency department is the appropriate service to treat acute sickness. Neither cost nor knowledge of the Shorter Stays in Emergency Departments Health Target featured as a reason for attendance.

**Premature mortality in adults using New Zealand psychiatric services**
Ruth Cunningham, Diana Sarfati, Debbie Peterson, James Stanley, Sunny Collings

This study linked routinely collected anonymised information on mental health service use with death records to compare rates of death before age 65 (premature death) between people using mental health services and the general New Zealand population. We found that those using mental health services were twice as likely to die prematurely, and had higher death rates from chronic medical conditions as well as from suicide and accidents. People who had been diagnosed with schizophrenia or bipolar disorder fared even worse, with three times the death rate.

**Risk factors for general medicine readmissions and association with mortality**
Manaf Aljishi, Ketna Parekh

Characteristics of patients who get readmitted back to hospital within a month of discharge have been examined in this study. It has been found that these patients tend to be older, use more medicines and have more serious medical conditions compared to the ones who have not been readmitted. There is a higher probability of death within 1 year if patients get readmitted multiple times for the same illness, which usually signals a patient’s deteriorating health condition.
Does seasonal level of serum 25-OH vitamin D correlate with the activity of Crohn’s disease?
Geogry Peter Kini, Brian Young, Peter Herbison, Michael Schultz

Vitamin D, the so-called sunlight vitamin, plays a crucial role in health and has been implicated in various diseases including Crohn’s disease, a type of inflammatory bowel disease. Earlier studies suggested that vitamin D deficiency could result in a more active Crohn’s disease. Whether vitamin D deficiency results in or is a consequence of more active Crohn’s disease remains unresolved.

From ICU to hospital-wide: extending central line associated bacteraemia (CLAB) prevention
Mary E Seddon, Catherine J Hocking, Elizabeth A Bryce, Jackie Hillman, Vicki McCoubrie

Infections associated with central lines are some of the most frequent and serious hospital infections, and can result in longer stays in hospital and even death. Previous work has shown that these infections can be largely prevented in the intensive care unit (ICU). This study (for the first time in Australasia) extends this work to wards and units outside the ICU. Using a simple checklist and standardising care we have significantly reduced these hospital-acquired infections from central lines in our hospital.
Leprosy in the South Pacific

Stephen T Chambers

The letter in this issue of the *Journal* from Colin Crawford gives some welcome attention to the ongoing problem of leprosy transmission in the Pacific region. While leprosy is no longer the scourge that it once was, it is comparatively recently that in 1991 the World Health Organization (WHO) Assembly took an important initiative and passed a resolution to “eliminate leprosy as a public health problem” by 2000. This was defined as a global prevalence to less than 1 case per 10,000 population, which is equivalent to fewer than 600,000 cases worldwide.

By 2005 the disease burden had fallen around 250,000 cases annually thanks in large part to the resounding success of the multidrug therapy programme. Unfortunately the decline has since stalled and the number of cases has remained fairly static since then. The current situation in the Pacific region is little different from that found elsewhere in the world. Some regions are reporting a reduction in cases but there are foci of transmission in the Pacific as well as in Africa (including Nigeria), the America and Asia.

Because of the success in achieving the WHO benchmark of ‘elimination of leprosy as a public health problem’ there has been the temptation for policy makers around the globe to pay much less attention to the quality of the leprosy programme. This has occurred whether or not the country has achieved elimination targets in part because of limited resources and the rising tide of other health issue demanding attention and resources. These include tuberculosis, obesity and diabetes.

Skill levels for leprosy control programmes are also very difficult to maintain in integrated services as there are a low number of cases, loss of awareness, high turnover of staff, and often a loss of focus on maintaining the quality of the service. Nevertheless, it is obvious that the long-term goal should be complete eradication of leprosy as has occurred in Europe, except perhaps from places such as the Southern United States where infected armadillos survive and are the source of sporadic cases.

These considerations have led to a recent reconsideration of how the strategy for the next phase of leprosy eradication should be undertaken. The key element of the strategy is to respond to the current epidemiological situation. The main risk of leprosy is in close contacts of new untreated cases whereas the risk in the general community is low.

Contacts need to be examined for signs of leprosy by competent staff, educated on the signs of early leprosy and to report suspicious lesions when they occur. Anecdotal evidence suggests some of the best informed people are those who have suffered from leprosy and they represent a pool of expertise who can alert others to the possible occurrence of disease.

Trial evidence demonstrate that a single dose of rifampicin (600mg) reduces development of disease by about 50% but contacts need to be reviewed as disease...
may still occur. This will require initiatives to ensure relevant record keeping and epidemiological mapping is maintained over prolonged periods.

What then is the role of non-governmental organisations (NGOs) such as the Pacific Leprosy Foundation (PLF)?

Firstly NGOs are guests in sovereign states and need to operate as such. They have a support role, and although providing assistance, are not responsible for activities such as national reporting to WHO or instituting policy at the national or operations level that is inconsistent with government policy.

Secondly NGOs must be true to their mission. The mission statement of the PLF is “The eradication of leprosy and the continued care of patients and their families with disability, or social and economic disadvantage due to leprosy in New Zealand and the Pacific.”

This is platform from which is solicits financial support from the public. There is plenty of evidence that this is what it is doing. Indeed the WHO awarded the PLF the Lee Jong-Wook Memorial Prize for Excellence in Public Health in 2012.

The primary goal is and remains eradication of the disease and to this end it supports field workers in Kiribati, the Solomon Islands, Samoa, Vanuatu, Fiji as well as the Twomey Leprosy Hospital in Fiji. The PLF also funds visits of WHO accredited leprologists over several weeks twice a year to high prevalence counties to provide expert clinical care, education, advocacy to governments and review of infrastructure. Engagement with policy to provide sustainable leprosy services as has also been well documented.

Thirdly, NGOs can have an important role in providing leadership in developing best practice as it changes. The PLF and other such organisations are aware of new policy initiatives, such as those advocated recently by ILEP and others, and actively promote local appropriate regional responses to these.

It is worth pointing out that NGOs have limited budgets and seek to provide ongoing service to areas where it can be sustained. This inevitably means negotiations to provide the best solution for each locality bearing in mind the resources and expertise available at each site.

The PLF is engaged with Kiribati, but not with the Federated States of Micronesia or the Marshall Islands, although it collaborates with other countries to develop relevant policy.

Leprosy programmes in the latter two countries are supported by directly by WHO. All these three countries have vast areas of ocean separating islands (e.g. Kiribati is spread over more than 100,000 square miles) and the logistics for providing services are very challenging. These countries consist mainly of atolls and the population is at risk from rising seas.

Many inhabitants may well migrate both for work and potentially as a refugees, carrying leprosy with them and risking reintroducing this organism to New Zealand. Government aid funding could be well spent in averting this problem by supporting leprosy and other infectious diseases programmes in these countries.
Competing interests: Nil.

Author information: Stephen T Chambers, Infectious Diseases Physician, Department of Infectious Diseases, Christchurch Hospital, Christchurch

Correspondence: Dr ST Chambers, Department of Infectious Diseases, Christchurch Hospital, PO Box 4345, Christchurch, New Zealand. Fax: +64 (0)3 3640952; email: Steve.Chambers@cdhb.health.nz

References:

Allowing violence in New Zealand hospitals is bad business

Steven Kelly

Hospitals are sometimes hazardous to health. Not only are patients sometimes exposed to a risk of healthcare-associated adverse events but employees are also exposed to risk. The risk of verbal, physical or sexual abuse to staff can occur either from other employees or from patients.

The epidemic of workplace bullying has been previously well covered in the New Zealand Medical Journal. Indeed, there is increasing evidence that violence directed towards healthcare workers is a significant problem.

The paper by Swain and colleagues in this issue of the New Zealand Medical Journal confirms that violence and aggression directed toward hospital staff is real and does occur in New Zealand hospitals. This finding, as they point out, has been well shown in the international literature. There are many reasons why this occurs. The patients may have drug, psychiatric or personality disorders. There may be an acute delirium due to a current medical condition. Or they may come from a social environment were violence is normal and where violent behaviour is a way for them to get what they want.

All public hospitals in New Zealand must have policies in place that reduce the risk to staff of violence from patients. There must be within all health work places a “zero tolerance of violence policy.” This should be actively promoted and acknowledged by all staff. This zero tolerance policy was introduced into the United Kingdom in 1999. All staff involved in patient interactions should have training in dealing with these situations.

Without appropriate staff training and processes put in place, staff may not know how to deal with an aggressive patient and disruptive behaviour is left unchecked.

There is another good reason why hospitals need to introduce these policies. It is a legal requirement under the Health and Safety in Employment Act 1992. Under this Act, all employers must take all practicable steps to reduce potential work place harm to employees. Therefore allowing violence in New Zealand hospitals is bad business. The New Zealand Government through the Department of Labour in 2009 produced a practice guide for all healthcare environments to manage violence.

For aggressive patients whom are competent to make decisions in regard to their healthcare they need to be given the chance to improve their behaviour. This can be done through de-escalation techniques to reduce the stress of the situation. However, if this fails then the police can be called and they can be removed from the hospital facility.

Aggressive patients and also their families must realise that the right to access free healthcare in New Zealand doesn’t include a right to abuse hospital staff and that it won’t be tolerated.
Competing interests: Nil.

Author information: Steven Kelly, General Surgeon, Christchurch Public Hospital, Christchurch

Correspondence: Steven Kelly, Department of Surgery, Christchurch Public Hospital, PO Box 4345, Christchurch, New Zealand. Email: steve.kelly@cdhb.health.nz

References:


Patient aggression experienced by staff in a public hospital setting

Nicola Swain, Chris Gale, Rachel Greenwood

Abstract

Aims Working in a healthcare environment is a known risk factor for violence. Patient aggression towards staff is often present in a hospital setting but the extent, type and variation among various occupations and roles are not known.

Method This research examines the type and frequency of aggression experienced by healthcare staff, using a previously used measure the POPAS-NZ, which is a short pen and paper survey. Responses were gathered from 227 people working in a single district health board.

Results Responses showed verbal anger was experienced by 93% of healthcare workers in the previous year and physical aggression was experienced by 65% of respondents. Also, 38% of staff reported experiencing a physical assault in the previous year. When analysed by role it was found that nurses and support staff experienced the greatest number of aggressive incidents compared to doctors and allied health staff. No effects of gender of the healthcare worker were found. Psychiatric units showed greater levels of destructive behaviour and attempted assaults but were similar to other areas of the hospital on all other measures.

Conclusion These results demonstrate many hospital staff, of all roles and workplaces experience aggression on a frequent basis. Implications for staff training are discussed.

Working in the healthcare environment is a risk factor for violence, including patient violence or aggression.1, 2 This is particularly well documented in nursing.3-6

Documented reasons for aggression include: factors attributable to the patient, the illness characteristics, the staff member, other people, poor communication and the environment.7

Aggression from patients is detrimental to health care workers and to the patient community, health care workers report high stress and dissatisfaction in their work,8 emotional exhaustion, depersonalisation and inefficacy9 and patients report a reduced level of care and autonomy when the relationship deteriorates.10

Research conducted in the UK comparing professional groups found nurses experienced more aggression than doctors.7 They reported high levels of aggression: 27% had been physically assaulted and 68% experienced verbal aggression.

The difference in physical assault by profession was striking, with 43% of nurses reporting physical assaults in the previous year compared to 14% of doctors. The same pattern was true for threatening behaviour. In both measures allied health staff reported the lowest levels of aggression. Allied health in this study was restricted to radiologists, physiotherapists and occupational therapists.
Patient violence has noted to be particularly high in certain clinical areas, specifically accident and emergency and psychiatric settings. One study reported 31% of nurses in an accident and emergency department had reported at least one physical assault in the preceding 6 months, another study found 56% had been assaulted in the previous 12 months.

Similarly in psychiatry, Nolan reported that around 75% of mental health nurses had been exposed to violence during the previous year and 50% of psychiatrists had experienced violence in the preceding year.

Research of workplace aggression in nursing reports that male nurses are more likely to experience acts of aggression than females the same is true of community support workers.

Little is known about the aggression experienced within a District Health Board (DHB) in New Zealand. Scales have been developed for the purpose of measuring workplace aggression including one that has been adjusted for use in New Zealand, the Perception of Patient Aggression Scale (POPAS-NZ).

Gale et al (2009) have previously used the POPAS-NZ to gather data on aggression in community support workers. The research reported that almost 20% of community support workers experienced verbal anger often/very often and nearly nine percent experienced physical aggression often/very often. Similarly, in a study of medical students it was reported that 67% had witnessed verbal aggression and 35% had witnessed physical aggression.

We set out to examine the levels of aggression experienced by DHB hospital staff and consider whether experienced aggression varied according to health workers’ roles and places of work.

**Methods**

We recruited participants by contacting general managers of hospital units. We sought out mental health wards and units and also included: emergency medicine, neonatal intensive care, district nursing, rehabilitation, public health, and old age services. No surgical or general medical wards were included.

Following approval, service managers were contacted, and through them departmental managers. Data collection occurred at a time arranged by a meeting with the department manager. Staff were usually invited to participate at staff handover meetings or multidisciplinary meetings.

The participants were asked to fill in survey forms at the time of the meeting; the forms were collected and taken away for coding entry into a database. Participants filled in the name, role, years of health employment, contact details and the POPAS-NZ survey. 100% participation was achieved from the 22 wards/units that were visited. See Figure 1.
**Figure 1. Recruitment within the District Health Board**

![Recruitment Diagram]

**Measures**—The POPAS-NZ is a modification of the POPAS, developed by Oud\(^7\). The main modification has been an extension of the types of violence to include stalking and vexatious litigation. Previous work indicated that this was reasonably internally reliable, with a Cronbach alpha in two surveys of around 0.91.\(^5\) The POPAS-NZ consists of 12 questions each asking the respondent to rate how often each event may have happened to them over the previous year. Response categories range from 1(never) to 5 (frequently).

**Statistics**—Data was entered into a spreadsheet and analysed using PASW (formerly SPSS). Descriptive statistics are presented along with chi-squared statistics. Chi-squared were chosen because it compares counts of categorical responses between independent groups. A Bonferroni adjustment is necessary when multiple pair-wise tests are performed on a single data set to avoid false positives.

**Results**

**Participants**—Recruitment is outlined in Figure 1. 227 participants were recruited. Table 1 shows numbers of participants by role and gender and also by years of service.
Table 1. Participation and average length of service by gender and role

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number of participants</th>
<th>Average length of service (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>227</td>
<td>18.5</td>
</tr>
<tr>
<td>Male</td>
<td>54</td>
<td>19.4</td>
</tr>
<tr>
<td>Female</td>
<td>173</td>
<td>18.3</td>
</tr>
<tr>
<td>Doctors</td>
<td>23</td>
<td>20.7</td>
</tr>
<tr>
<td>Nurses (total)</td>
<td>124</td>
<td>22.5</td>
</tr>
<tr>
<td>Registered</td>
<td>111</td>
<td>21.9</td>
</tr>
<tr>
<td>Enrolled</td>
<td>13</td>
<td>28.3</td>
</tr>
<tr>
<td>Allied health professionals</td>
<td>38</td>
<td>13</td>
</tr>
<tr>
<td>Clinical support staff</td>
<td>42</td>
<td>11.1</td>
</tr>
</tbody>
</table>

Prevalence and pattern of aggression experienced in the past year—Table 2 shows the frequency of the various types of patient aggression reported by staff. Responses have been summarised into occasionally/sometimes and often/frequently for ease of reading. Responses vary from 94% reporting experiencing verbal anger in the previous year to 10% reporting experiencing sexual assault.

Table 2. Frequency (%) distribution of respondents’ perception of the prevalence of various types of aggression

<table>
<thead>
<tr>
<th>Variables</th>
<th>Never</th>
<th>Occasionally/sometimes</th>
<th>Often/Frequently</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal anger</td>
<td>7</td>
<td>67</td>
<td>27</td>
</tr>
<tr>
<td>Verbal threat</td>
<td>21</td>
<td>69</td>
<td>10</td>
</tr>
<tr>
<td>Humiliation</td>
<td>35</td>
<td>52</td>
<td>13</td>
</tr>
<tr>
<td>Physical aggression</td>
<td>35</td>
<td>46</td>
<td>10</td>
</tr>
<tr>
<td>Destructive behaviour</td>
<td>44</td>
<td>35</td>
<td>8</td>
</tr>
<tr>
<td>Attempted assault</td>
<td>57</td>
<td>32</td>
<td>6</td>
</tr>
<tr>
<td>Assault</td>
<td>61</td>
<td>26</td>
<td>3</td>
</tr>
<tr>
<td>Injury</td>
<td>71</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Sexual harassment</td>
<td>60</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sexual assault</td>
<td>90</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Stalking</td>
<td>88</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Litigation</td>
<td>74</td>
<td>26</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 3 shows ranges, medians and modes for the reported aggressive acts. Verbal anger, verbal threat, humiliation, physical aggression and destructive behaviour have medians above 0, so might be considered the commonly experienced. The other measures have medians below 1, which means they are not commonly experienced.
Table 3. Number of times of various types of aggressive behaviours on the POPAS-NZ in the past year

<table>
<thead>
<tr>
<th>Variables</th>
<th>Range</th>
<th>Median</th>
<th>Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal anger</td>
<td>Never–frequent</td>
<td>Sometimes</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Verbal threat</td>
<td>Never–frequent</td>
<td>Occasionally</td>
<td>Occasionally</td>
</tr>
<tr>
<td>Humiliation</td>
<td>Never–frequent</td>
<td>Occasionally</td>
<td>Occasionally</td>
</tr>
<tr>
<td>Physical aggression</td>
<td>Never–frequent</td>
<td>Occasionally</td>
<td>Never</td>
</tr>
<tr>
<td>Destructive behaviour</td>
<td>Never–frequent</td>
<td>Occasionally</td>
<td>Never</td>
</tr>
<tr>
<td>Attempted assault</td>
<td>Never–frequent</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Assault</td>
<td>Never–frequent</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Injury</td>
<td>Never–frequent</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Sexual harassment</td>
<td>Never–often</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Sexual assault</td>
<td>Never–often</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Stalking</td>
<td>Never–frequent</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Litigation</td>
<td>Never–often</td>
<td>Never</td>
<td>Never</td>
</tr>
</tbody>
</table>

Aggression by gender—A Chi-squared ($\chi^2$) test was conducted between each POPAS question and gender: verbal anger ($\chi^2=2.5, p=.62$), verbal threat ($\chi^2=10.3, p=.04$), humiliation ($\chi^2=5.0, p=.29$), physical aggression ($\chi^2=3.8, p=.43$), destructive behaviour ($\chi^2=4.2, p=.38$), attempted assault ($\chi^2=12.5, p=.01$), assault ($\chi^2=12.6, p=.01$), injury ($\chi^2=7.4, P=.11$), sexual harassment ($\chi^2=3.5, p=.34$), sexual assault ($\chi^2=3.4, p=.34$), stalking ($\chi^2=1.1, p=.79$), litigation ($\chi^2=6.7, p=.08$). Although assault and attempted assault approach levels of significance, a Bonferroni correction suggests that we should use $p<.004$. Therefore, there appears to be no statistically significant relationship between each of the measures of aggression and gender.

Aggression by role—Participants were divided into one of four role groups: nurses, doctors, allied health and clinical support to see if different professional groups might experience differing levels of aggression.

Results suggest quite different patterns of aggression among the four occupational groups. For illustrative purposes the two highest frequency aggressive behaviours (verbal aggression and physical aggression) are shown.

Around a third of both nurses and clinical support workers experience verbal aggression often or frequently (see Table 4). Clinical support workers and nurses also reported the highest rates of physical aggression (see Table 5).

Table 4. Percentage POPAS-NZ scores by role for verbal aggression

<table>
<thead>
<tr>
<th>Role</th>
<th>Never</th>
<th>Occasionally/sometimes</th>
<th>Often/frequently</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physicians</td>
<td>4.5</td>
<td>81.9</td>
<td>13.6</td>
<td>22</td>
</tr>
<tr>
<td>Nurses</td>
<td>2.5</td>
<td>63.1</td>
<td>34.4</td>
<td>122</td>
</tr>
<tr>
<td>Allied health</td>
<td>18.0</td>
<td>74.0</td>
<td>8.0</td>
<td>50</td>
</tr>
<tr>
<td>Clinical support</td>
<td>9.1</td>
<td>57.6</td>
<td>33.3</td>
<td>33</td>
</tr>
</tbody>
</table>
### Table 5. Percentage POPAS-NZ scores by role for physical aggression

<table>
<thead>
<tr>
<th>Role</th>
<th>Never</th>
<th>Occasionally/sometimes</th>
<th>Often/frequently</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physicians</td>
<td>36.4</td>
<td>59.1</td>
<td>4.5</td>
<td>22</td>
</tr>
<tr>
<td>Nurses</td>
<td>26.2</td>
<td>58.2</td>
<td>15.6</td>
<td>122</td>
</tr>
<tr>
<td>Allied health</td>
<td>58.0</td>
<td>36.0</td>
<td>4.0</td>
<td>50</td>
</tr>
<tr>
<td>Clinical support</td>
<td>30.3</td>
<td>48.5</td>
<td>21.2</td>
<td>33</td>
</tr>
</tbody>
</table>

Chi-squared tests show statistically significant differences in responses by role for all measures of aggression except sexual harassment, sexual assault, destructive behaviour, and stalking (p<.004), which are among the lower frequency events. Verbal anger ($\chi^2=45.2$, p<.000), verbal threat ($\chi^2=74.3$, p<.000), humiliation ($\chi^2=54.1$, p<.000), physical aggression ($\chi^2=35.9$, p=0.03), destructive behaviour ($\chi^2=31.6$, p=0.011), attempted assault ($\chi^2=43.9$, p<.000), assault ($\chi^2=47.6$, p<.000), injury ($\chi^2=35.7$, p=0.03), sexual harassment ($\chi^2=12.1$, p=.44), sexual assault ($\chi^2=7.4$, p=.83), stalking ($\chi^2=15.4$, p=.22), litigation ($\chi^2=20.5$, p=.06).

**Aggression by department**—Data was divided into mental health wards and units (63%) and other hospital wards and units (37%). Chi-squared tests revealed differences between the two patient groups in destructive behaviour ($\chi^2=20.7$, p<0.001) and physical aggression ($\chi^2=24.2$, p<0.001).

All other questions relating to aggression experienced (including actual assault) do not systematically vary according to whether they are mental health units or not. A mental health ward/unit has more destructive behaviour and physical aggression but on all other measures is similar to other hospital wards/units.

### Discussion

The aim of this study was to report on aggression experienced by the healthcare workforce in New Zealand. This was done by using a standardised survey instrument, across a hospital setting. Results show significant levels of aggression reported by healthcare workers.

Interestingly, more aggression was reported in the hospital setting than had previously been reported by community support workers\(^{15}\) and medical students\(^{16}\) using this tool in New Zealand. The rates are considerably higher than those reported in a UK general hospital where 27% were physically assaulted over the preceding year,\(^2\) compared to 38% in the past year in the present study. In a review of international nursing studies, 64.4% reported ever being physically assaulted by a patient,\(^6\)

The rates of assault in the healthcare workplace in New Zealand are higher than the lifetime incidences of interpersonal violence in New Zealand, which has been recognised as a national priority. Lifetime rates of interpersonal violence reported by women range from 17–19%.\(^{18}\) That 38% of our healthcare workforce report experiencing an assault in the previous year could be seen as a situation to which urgent attention should be paid. Inexperience does not appear to be a causal factor with the average experience of the healthcare worker being 18 years.
Prevalence of assault over working lives might be much higher than our figure of 38% in previous year, (although recall bias can favour recall of the most recent past) as reported at 64% in the review by Spector, Zhou and Che (2013).

Consistent with previous studies there were also significant differences in the aggression experienced and the professional group surveyed. Nurses experience the greatest levels of aggression, followed by doctors and clinical support, with allied health experiencing the lowest levels. Winstanley and Whittington (2004) also reported that nurses experienced the most aggression, followed by doctors and then allied health.

This study has added the group of clinical support staff and found that they too are experiencing high levels of aggression. The present study had a wider definition of allied health than the previous study but it did not change its position as least likely role to report aggression. There may be some confounding with hours of patient contact, which could be controlled for in future studies. However, the between group differences are sufficiently large that this is unlikely to fully account for them.

There were no effects of gender of the healthcare worker on the experience of aggression. Previous research had suggested that male nurses experience more aggression than female nurses and likewise male community support workers experience more aggression than females. It might be that analysis of gender by occupation is needed to elaborate these differences.

There are also few effects of working in mental health compared to other areas of the hospital. The data suggest that destructive behaviour and physical aggression are higher in mental health wards/units but no other measures of aggression show significant differences. This is also in contrast to previous finding which mental health is a particularly risky area to work but again the relationship might be a more complex interaction of workplace by role as allied health have the lowest levels of aggression and many (e.g. Psychologists) are also working in mental health. Those wards not sampled (medical and surgical) could possibly represent lower risk workplaces.

The study was generally well received and supported by the staff. A good participation rate was achieved using the recruitment method of attending departmental meetings. However, there are several limitations of the present research. This survey did not reach all areas of the hospital and so does not represent the experience of all of the staff employed by the DHB. There are no medical and surgical wards included in this survey; coverage could be improved for future surveys.

In summary, this study used a tool (POPAS-NZ) specifically designed to measure aggression in the healthcare workforce in a hospital in New Zealand. Significant levels of workplace aggression were experienced in a DHB environment. When roles were considered it was found that nurses were most at risk of aggression, which suggests targeted training might be considered highest priority for nurses but should also be considered for those in support roles working in a hospital. Department and gender were found to be less important predictors of aggression.

While it is important that workplaces support staff who are experiencing aggression, it is also possible to improve training to reduce aggression. Previous research in New Zealand has correlated a communication style with risk of aggression. This is a
modifiable component of the patient interaction and as such has potential for intervention. Future research might examine if communication skill interventions for staff could reduce the experience of aggression.

Competing interests: Nil.

Author information: Nicola Swain, Senior Lecturer – Behavioural Science, Department of Psychological Medicine, Dunedin School of Medicine, University of Otago, Dunedin; Chris Gale, Senior Lecturer – Psychiatry, Department of Psychological Medicine, Dunedin School of Medicine, University of Otago, Dunedin; Rachel Greenwood, Department of Psychological Medicine, Dunedin School of Medicine, University of Otago, Dunedin

Correspondence: Dr Nicola Swain, Department of Psychological Medicine, Dunedin School of Medicine, University of Otago, Cumberland Street, Dunedin 9054, New Zealand. Email: nicola.swain@otago.ac.nz

References
17. Oud N. Internal report. POPAS Ervaringen van psychiatrische hulpverleners met agressief gedrag. 2001:1–2

Why do patients self-present to Middlemore Hospital Emergency Department?

Vanessa Thornton, Annie Fogarty, Peter Jones, Nouran Ragaban, Catherine Simpson

Abstract

**Aim** To determine the drivers for acute (Australasian Triage Scale Category 3–5) demand in patients who self-present to New Zealand’s Middlemore Hospital Emergency Department (MMH ED), we sought to establish a demographic profile of a sample of self-presenting patients and explore their reasons for presenting to ED rather than attending a primary care centre.

**Method** A prospective, observational study was undertaken of patients in Australasian Triage Scale Categories 3–5 (ATS 3–5) who self-presented to MMH ED over a 7 day period from 14 April 2011 to 21 April 2011. We studied two time periods, 0900–1200 and 1800–2200, to compare drivers for attendance to MMH ED during primary care service open hours and closed hours. A structured questionnaire was used to collect demographic data and outcomes. The cumulative 2011 demographic data for self-presentation to MMH was compared to the study data.

**Results** 500 patients were approached to participate and 421 met the inclusion criteria. The mean age of presenters was 37.6 years (SD of 24.6) with 48.2% (95%CI 44–53%) being male and 23% (95%CI 19–27%) employed. Of those who indicated they had a general practitioner (GP), 23% (95%CI 21–30%) had contacted their GP prior to presentation to MMH ED, with 73% (n=73) advised to attend ED. Of the 73 patients told by their GP to attend ED, 30 (41.1%; 95%CI 31–53%) were admitted, with two patients being transferred to another district health board (DHB), and the remainder discharged home.

Thirty-two percent of the self-presenting patients came to ED because they felt sick enough to require emergency care. Comparison of the data for the two time periods indicated only one significant difference: 14% of patients presented to ED in the morning because their GP was closed, whereas 28.7% of those who presented after hours did so for this reason.

**Conclusion** Almost 25% of self-presenting patients had contacted their GP or a health professional prior to their ED presentation and were advised to attend ED. The most common reason for patients to self-present at MMH ED is the belief that a hospital emergency department is the appropriate service to treat acute sickness. Neither cost nor knowledge of the Shorter Stays in Emergency Departments Health Target featured as a reason for attendance.

Presentations to all emergency departments in the greater Auckland area are increasing year on year. However, the increase in self-presentation in these departments is greater than the background population growth in the Auckland region.
The Counties Manukau population to which the District Health Board (DHB) provides healthcare is growing at 3.2% per year, while presentations to Middlemore Hospital (MMH) Emergency Department (ED) have been increasing at a rate of 5–8% per annum over the last 4 years. If this continues, services will be stretched well beyond capacity.

Middlemore Hospital Emergency Department is the busiest mixed (adult/paediatric) ED in Australasia, with an annual census of 98,110 patients in 2011. The hospital is a tertiary academic centre with all major subspecialties (except for cardiothoracic and neurosurgery).

Since 2009, MMH has focused on meeting the New Zealand Government’s target of discharging or transferring 95% of ED patients within six hours in compliance with the Shorter Stays in Emergency Departments Health Target (the Shorter Stays Target).

Through a number of hospital-wide strategies, MMH has successfully met and maintained the 95% benchmark for the Shorter Stays Target. However, with the unprecedented and sustained increase in patient presentations to MMH ED, consideration of new initiatives is now necessary to meet the demand for high quality, cost-effective, patient-centred care.

Existing research suggests that between 15% and 40% of patients in New Zealand emergency departments have been seen by, or had contact with, a GP prior to coming to the ED. Primary care professionals have asserted that patients in ATS 3–5 can be managed in primary care, and that cost of access to primary care is a factor in patient choice.

The high cost of after-hours services in the community and the copayments in primary care, in contrast to the free service offered in hospital emergency departments, have supposedly driven the use of emergency departments by patients. This is thought by some to account for the rising percentage of self-presenters. Furthermore, many have commented that increased efficiency under the Shorter Stays Target may have encouraged patients to attend MMH ED rather than primary care services.

In 1995, MMH ED performed a prospective survey to determine patients’ reasons for presenting to the ED and their primary care utilisation patterns. This was an unpublished study completed by Lynn Butler for the CEO in 1995. The study included 199 patients who presented to MMH ED on 6 days over a 2 week period – 10 April 1995 to 24 April 1995 – between 0800 and 2215 hours.

Patients in the resuscitation and observation area were excluded. Demographic data was collected by an interviewer using a questionnaire that included questions about why patients had come to the ED. This study identified that 20% of the patients were told by health professionals to come to ED and 23% perceived their condition to be worsening or to be an emergency. Eighteen percent found ED convenient and 10% of patients’ GPs were unavailable.

Successfully managing the increase in demand for emergency services within a health environment of constrained resources requires a deeper understanding of self-presenting patients. Elley et al discuss the need to focus our energy on the barriers to
obtaining primary care before the need for emergency care is required, rather than concentrating on the appropriateness once patients arrive in ED.11

The aim of our study is to establish a demographic profile of self-presenting Australasian Triage Scale Category 3–5 (ATS 3–5) patients and explore the reasons why these individuals decide to attend MMH ED instead of their GP. The findings from this study may help to identify and implement changes in how demand for service is managed while ensuring that the appropriate care is delivered to the right patients at the right place.

**Method**

**Design**—This was a prospective observational study with data collected on consecutive days over 1 week (from 14 April 2011 to 21 April 2011). Two time periods were identified to represent availability of primary care (in hours: 0900hrs–1200hrs and out of hours: 1800hrs–2200hrs).

**Participants**—All patients who self-presented to MMH ED and met the ATS 3–5 criteria during the study periods were invited to participate. Table 1 describes the inclusion and exclusion criteria used to identify participants.

**Table 1. Inclusion and exclusion criteria used for this research**

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Criteria</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Self-presentation</td>
<td>Any patient presenting to MMH ED who was not referred to an inpatient specialist team by their primary care provider</td>
</tr>
<tr>
<td></td>
<td>Australasian Triage Scale Category 3–5</td>
<td>All patients are classified on arrival according to the Australasian Triage Scale (ATS). The ATS is a scale of the triage nurse’s impression of a patient’s urgency to be seen by a treating clinician, with 1 being most urgent (see immediately) and 5 being least urgent (see within 2 hours).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exclusion criteria</th>
<th>Criteria</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Refusal to participate</td>
<td>Patient refused</td>
</tr>
<tr>
<td></td>
<td>Unable to give consent</td>
<td>Unable to consent to participate or unable to complete the questionnaire due to being discharged</td>
</tr>
</tbody>
</table>

**Data collection tool**—A structured questionnaire was developed to address the aims of the research (see Figure 1). To ensure the validity of the questionnaire and effective data collection, the draft questionnaire was reviewed by 5 MMH ED nurses and amended in light of feedback received.
In addition to the questions identified to address the primary aims of the research, patients were asked if they were aware of the Shorter Stays Target in order to gauge whether that had an impact on their choice to present to ED.

Patients were also asked about their knowledge of urgent after-hours care, when his/her GP was closed.
Procedure—Using the patient management systems available in the MMH ED, patients fitting the inclusion criteria described above were identified as possible participants to approach. A research nurse and/or the research assistant from the University of Auckland approached patients within 30 minutes of their arrival.

Due to the nature of ED and to ensure that completion of the questionnaire was uniform, the nurse/research assistant completed the questionnaire on the patient’s behalf by asking them the question and noting his/her response.

After each study shift was completed, a nurse/research assistant entered the data into a Microsoft Excel spreadsheet made to mirror the questionnaire (Figure 1).

Most of the information was collected at first point of contact with the patient or from the demographic data collected at time of registration by the clerks. Only the date of discharge, final diagnosis and test results (i.e. full blood count, electrolytes, X-rays, and ultrasound scan) were gathered at a later date using linked data from the hospital (Table 2).

Table 2. The outcomes measured and analysed for patients participating in this study

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td>Age, gender, ethnicity, residency status, employment</td>
</tr>
<tr>
<td>Triage category</td>
<td>Initial (when presenting) and final (when discharged)</td>
</tr>
<tr>
<td>Length of stay</td>
<td>Time spent in ED</td>
</tr>
<tr>
<td>Patient disposition</td>
<td>Proportions discharged, admitted or transferred to another department (outside of ED) or another DHB</td>
</tr>
<tr>
<td>Primary care access</td>
<td>Proportion who did or did not have a GP, whether a GP was contacted prior to coming to ED, advice given by the GP on whether to go to ED</td>
</tr>
<tr>
<td>Visit type</td>
<td>Reason for ED visit; referral or self-presenting</td>
</tr>
<tr>
<td>Investigations</td>
<td>Tests performed during ED visit</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Diagnosis on discharge</td>
</tr>
<tr>
<td>Shorter stays target</td>
<td>Whether patients were aware of the target for 95% of patients to be admitted, discharged or transferred within 6 hours of arrival in ED</td>
</tr>
</tbody>
</table>

Analysis—The study demographic data and admission data were compared with MMH ED self-presenting ATS 3–5 patients for 2011 and across the two time periods for this study. These data were compared with the unpublished MMH data by Lynne Butler completed for the CEO in 1995.

Simple statistical analysis, including frequencies, proportions with 95% confidence intervals (CI) and mean (SD) was undertaken to examine and describe the data gathered from the 421 participants and the self-presentation ED data for 2011. GraphPad QuickCalcs software was used to calculate the 95%CI.

Ethical approval was obtained from the Northern Regional Ethics Committee (NTX/11/EXP012).

Results

500 patients were approached to participate, with 421 (85%) being included in the analysis (95%CI 81–87). (See Figure 2 for reasons for non-inclusion.)
Figure 2. The study cohort and breakdown of eligible participants based on inclusion exclusion criteria

Table 3 outlines the demographic characteristics of the study cohort. The demographic characteristics of the study population were consistent with those of total presentations to MMH in the year 2011; thus the study group was representative of the ATS 3–5 patients that normally attend MMH ED.

Of the study cohort, 377 were New Zealand residents (89.5%). The age range was 15 days to 94 years, with a mean of 37.6 years (SD 24.6). Eighty patients were over 65 years old (19%) and 203 were male (48.2%).
Table 3. The demographic information for the sample of 421 patients included in this research study

<table>
<thead>
<tr>
<th>Demographic Information</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Number Eligible</td>
<td>421</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age</td>
<td>37.6 years</td>
<td>–</td>
<td>24.6</td>
</tr>
<tr>
<td>≤ 18 years old</td>
<td>96</td>
<td>22.8</td>
<td>–</td>
</tr>
<tr>
<td>19–64 years old</td>
<td>245</td>
<td>58.2</td>
<td>–</td>
</tr>
<tr>
<td>≥ 65 years old</td>
<td>80</td>
<td>19.0</td>
<td>–</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Gender</th>
<th>n</th>
<th>%</th>
<th>95% CI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>203</td>
<td>48.2</td>
<td>44–53</td>
</tr>
<tr>
<td>Female</td>
<td>218</td>
<td>51.8</td>
<td>47–57</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>n</th>
<th>%</th>
<th>95% CI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maori</td>
<td>92</td>
<td>21.9</td>
<td>18–26</td>
</tr>
<tr>
<td>NZ European</td>
<td>113</td>
<td>26.8</td>
<td>23–31</td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>128</td>
<td>30.4</td>
<td>26–35</td>
</tr>
<tr>
<td>Other</td>
<td>88</td>
<td>20.9</td>
<td>17–25</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Resident status</th>
<th>n</th>
<th>%</th>
<th>95% CI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resident</td>
<td>377</td>
<td>89.5</td>
<td>86–92</td>
</tr>
<tr>
<td>Non-resident</td>
<td>42</td>
<td>10.0</td>
<td>7–13</td>
</tr>
<tr>
<td>Information unavailable</td>
<td>2</td>
<td>0.5</td>
<td>–</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Employment</th>
<th>n</th>
<th>%</th>
<th>95% CI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employed</td>
<td>98</td>
<td>23.3</td>
<td>19–27</td>
</tr>
<tr>
<td>Unemployed</td>
<td>48</td>
<td>11.4</td>
<td>8–14</td>
</tr>
<tr>
<td>Student</td>
<td>92</td>
<td>21.9</td>
<td>18–26</td>
</tr>
<tr>
<td>Retired</td>
<td>66</td>
<td>15.6</td>
<td>13–20</td>
</tr>
<tr>
<td>Other</td>
<td>117</td>
<td>27.8</td>
<td>24–32</td>
</tr>
</tbody>
</table>

The most common reason for presentation was medical illness (74.8%), while trauma accounted for 24.9%. There were 139 admissions to hospital (33%). Of these admissions, 33% were Medical and 26.6% were to General Surgery. This was comparable to admission data for all self-presenting patients in 2011 for CMDHB.

Most patients (n=393; 93.3%) indicated that they had a GP (1 patient indicated ‘No Comment’). When those patients were questioned as to whether they had tried to contact their GP on the day of presentation, 99 (25.2%) stated they had contacted the GP either in person or by phone. For this group of 99 patients, 73 (73.7%) were told to attend MMH ED, and a further 20 (20.2%) were told that the practice was too busy for the GP to see them that day.

Of the 73 patients told by their GP to attend ED, 30 (41.1%) were admitted, with 2 patients being transferred to another DHB, and the remainder discharged home.

Of the 321 patients who had not tried to contact their GP, 88 (27.4%) thought their condition was too urgent to await the GP, while 71 (22.1%) said the GP was closed, and 56 (17.4%) were brought by ambulance. Cost was indicated as a factor in their decision by 6 patients (1.9%).
The remaining 100 patients (31.2%) indicated a variety of reasons for not contacting a GP prior to presentation, including ED being their preferred choice at the time (see Table 4). Data comparison of the 321 patients across the two time periods (0900–1200 and 1800–2200) indicated that 20 (14.0%) attended ED because their general practice was closed during the morning period compared with 51 (28.7%) in the evening period. This was a statistically significant difference between the two groups.

Table 4 presents a comparison of data between the two time periods concerning reasons why patients who did contact their GP chose to attend ED.

<table>
<thead>
<tr>
<th>Reason for presenting</th>
<th>Morning</th>
<th>Afternoon</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acuity</td>
<td>41</td>
<td>47</td>
<td>0.65</td>
</tr>
<tr>
<td>Brought by ambulance</td>
<td>29</td>
<td>27</td>
<td>0.23</td>
</tr>
<tr>
<td>ED is preferred choice</td>
<td>25</td>
<td>10</td>
<td>0.0007*</td>
</tr>
<tr>
<td>GP closed</td>
<td>20</td>
<td>51</td>
<td>0.0017*</td>
</tr>
<tr>
<td>Referred</td>
<td>8</td>
<td>12</td>
<td>0.67</td>
</tr>
<tr>
<td>Do not have a GP</td>
<td>5</td>
<td>9</td>
<td>0.50</td>
</tr>
<tr>
<td>International/out-of-town visitor</td>
<td>5</td>
<td>7</td>
<td>0.84</td>
</tr>
<tr>
<td>No comment</td>
<td>4</td>
<td>1</td>
<td>0.11</td>
</tr>
<tr>
<td>Cost</td>
<td>3</td>
<td>3</td>
<td>0.79</td>
</tr>
<tr>
<td>Sudden onset</td>
<td>1</td>
<td>1</td>
<td>0.88</td>
</tr>
<tr>
<td>Follow-up</td>
<td>1</td>
<td>0</td>
<td>0.27</td>
</tr>
<tr>
<td>If deterioration go to ED</td>
<td>0</td>
<td>5</td>
<td>0.04*</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>5</td>
<td>0.17</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>143</strong></td>
<td><strong>178</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Most patients (233; 55.3%) had not considered planning what to do in the event that their GP was unavailable. Of those who had a plan for after-hours care, 91 (21.6%) were told to attend MMH ED and a minority, 13 (3.1%), were told to attend an urgent care clinic.

Laboratory investigations of full blood count, urea and electrolytes were performed in 77% (n=325) of cases. The following radiological investigations were performed:

- Plain film (X-ray): n=130 (40%; 95%CI 35%–45%)
- Computed tomography (CT): n=36 (11%; 95%CI 8%–15%)
- Ultrasound scan (USS): n=25 (7.7%; 95%CI 5%–11%).

Of all CT and USS investigations done, 47% had clinically significant findings that assisted in guiding management: admission or discharge with appropriate follow-up. 362 patients, 86.0% (95%CI 82–89%), had not heard of the Shorter Stays Target.
Discussion

This study is the beginning of a journey towards understanding the reasons for increasing ED acute demand.

This study has shown that the group that self-presents to MMH ED is a relatively young group of patients. This is consistent with the population statistics for South Auckland. Pacific Islander and Maori patients are over-represented in this ED self-presentation group compared with background population statistics in South Auckland.

Current demographic data for Counties Manukau District Health Board’s catchment area show that Pacific Islanders make up 23% of the population but account for 33% of patients self-presenting to the ED, and Maori make up 17% of the total population and 21% of patients self-presenting to the ED.\(^1\) This cultural over-representation is consistent with overall patient presentations at MMH ED, and was similar to the patterns shown in Lynne Butler’s 1995 study.\(^10\)

Analysis of GP access provides an interesting contrast in data trends. In 2011, 93% of patients had access to a GP; in 1995, 97% did. This same data showed that in 1995 only 3% of non-residents presented to MMH, but our data had this figure as high as 10%.\(^10\) One of the interim governmental policy changes has been the introduction of capitation to GPs, which may explain this phenomenon.

Almost a quarter of patients considered to be self-presenting had contacted their GP either by phone or in person and had been referred to the ED. This group of patients had consulted a health professional prior to making a decision to attend MMH ED. This is consistent with previous papers, including Lynne Butler’s 1995 unpublished data, that suggest between 15% and 40% of patients in New Zealand emergency departments have seen or had contact with a GP prior to coming to the ED.\(^6–10\)

The admission rate in the 1995 study for self-presenting patients was 33%.\(^10\) The admission rate for all ATS 3–5 patients in 2011 (60,020 patients) was 24.5%. In our study the admission rate was 28.2%. There has been a reduction in the total admission rate for all self-presenting ATS 3–5 patients presenting to ED over 20 years. This may be explained in part by the change in model of care with the development of emergency medicine and the presence of senior medical officers in the ED. In addition, GPs appropriately perceive that MMH ED provides specialty services to a group of patients that can be effectively managed and discharged rather than referred for admission under an inpatient service. Examples of such services are management of toxicology patients, assessment of head injuries and treatment of fractures and dislocations.\(^12\)

Analysis of the reasons why patients had not contacted their GP is consistent with many other studies.\(^4,6,7\) The most common reason for deciding to attend ED rather than a GP was that patients MMH considered ED to be the appropriate place to go based on their condition at the time. Lack of access to the GP was the next most common explanation.

The influence of lack of GP access on ED self-presentation rates is confirmed by the higher number of patients citing this as their reason for self-presenting in the after-hours sample compared with the sample taken in the morning, when GPs were more
likely to be available. Ambulance referrals made up another 20% of the patients who did not contact their GP. It is likely that the decision to come to ED was guided by the ambulance responder for these patients. Lewis has commented in his 1988 paper on accessibility to care that one of the major reasons for ED attendance and opening hours was the main contribution to what was perceived as accessibility of medical care.\textsuperscript{8}

Although there can be a cost to the patient associated with ambulance services, these are not charged at the time of the service provision, in contrast to a GP visit. However cost did not feature highly in this study. This is consistent with previous studies in New Zealand of this nature.\textsuperscript{6–9}

Another important finding of this study was patients did not seem to be aware of other options for out-of-hours care. Most patients in the study had not planned how to access healthcare if their GP was closed and a family member was sick. Few patients (3.2%) considered attending after-hours urgent care services such as accident and medical clinics, and many had been told by their GP to attend ED. This supports Elley’s study, which found that primary care needs to be made readily available, and that patients should be encouraged to develop an alternative plan of care in the event that their GP is unavailable.\textsuperscript{11}

Our findings for admissions rates for ATS 3–5 patients are not consistent with the assumption that patients in these low acuity triage categories are better served in primary care. Among this cohort of low acuity (ATS 3–5) patients, over a quarter were admitted to hospital. Understanding the function of a triage category is important.

The triage category of a patient dictates how quickly a patient needs to be seen within the queue that exists in the ED, rather than the complexity of the medical condition. For example, a patient with a dislocated shoulder could be ATS Category 2, whereas a complex medical patient who has presented with fever could be ATS Category 4. Furthermore, almost half of the investigations done in ED resulted in positive findings that guided patient care and disposition from ED, supporting the acute need of investigations in many self-presenting patients.

The Shorter Stays Target has revolutionised the process of admission to Middlemore Hospital. MMH ED has achieved the target of 95% of patients being admitted, discharged or transferred from the ED with six hours consistently since 2009 through a whole-system hospital approach. Many late adopters of the Shorter Stays Target believed that patient awareness of increased ED efficiency would result in higher numbers of patients choosing to present to ED rather than to their primary care providers. This belief is not supported in this study, as most patients had no idea of the Shorter Stays Target policy.

**Study limitations**—Our data are biased in that our study population was confined to patients who presented to the ED; we have not considered patients who attended a GP or an accident and medical clinic and were satisfied with that care. However, Ministry of Health surveys of the whole population indicate similar results to the findings of this study.\textsuperscript{13}

The patients answer as to why they attended the ED was derived from responses to questions on the patients to access to their General Practice. Patients attending a GP
or an accident and medical clinic may have a clearer understanding of available after-hours care. We also did not specify the geographical location of the patients in attendance at the ED and the wide diversity of access to GPs that occurs in the area served by Counties Manukau District Health Board.

**Conclusion**—Understanding patients’ reasons for presenting to emergency departments is crucial for enabling hospitals to develop successful strategies to manage increasing patient demand. These reasons are complex.

The key findings of our study indicate that a significant proportion of patients assumed to be self-presenters have in fact contacted a GP prior to their presentation and been advised to attend the ED for medical review.

We also found that many patients believed their choice to attend the ED was appropriate due to the acuity of their condition, or that they had been unable to access primary care.

Middlemore Hospital is now electronically tracking patients’ reasons for attending the ED in order to assess future trends and the impact of new health initiatives on self-presentations.

**Competing interests:** Nil.

**Author information:** Vanessa Thornton, Clinical Director, Emergency Department, Middlemore Hospital, Auckland; Annie Fogarty, Clinical Nurse Director, Emergency Department, Middlemore Hospital, Auckland; Peter Jones, Director of Emergency Medicine Research, Auckland City Hospital, Auckland; Nouran Ragaban, PhD candidate, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland; Catherine Simpson, Clinical Director, Intensive Care Unit, Middlemore Hospital, Auckland

**Acknowledgements:** We thank the participating patients who took the time to be surveyed for this research.

**Correspondence:** Dr Vanessa Thornton, Clinical Director, Emergency Department, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland 1640, New Zealand. Fax: +64 (0)9 2760000; email: vthornton@middlemore.co.nz

**References:**

5. Buchanan, C. Utilisation of accident and emergency services in Otago. Dunedin: Department of Preventive and Social Medicine, University of Otago; 1985.
### Abstract

**Aims** People with experience of mental illness, in particular those accessing mental health services, have increased mortality compared to the general population, but no studies have examined the situation in New Zealand. This study uses a complete national dataset to estimate mortality rates from natural and external causes for adults using psychiatric services compared to the general New Zealand population.

**Methods** Routinely collected data on adults aged 18–64 using secondary mental health services between January 2002 and December 2010 were linked to death registrations over the same period. Indirect standardisation was used to estimate the mortality ratio (SMR) for those with any contact with mental health services over this period compared to the New Zealand population.

**Results** Both men and women using mental health services in New Zealand have more than twice the mortality rate of the total population [combined SMR 2.14 (95% CI 2.09–2.19)], with an increased risk of death from cancer and cardiovascular disease [SMRs=1.31 (1.24–1.37), and 1.69 (1.60–1.79) respectively], and external causes (suicide and accidents) [SMR 3.11 (3.00–3.23)]. People with a diagnosis of a psychotic disorder had three times the overall death rate of the population.

**Conclusions** This study confirms that those using mental health services in New Zealand are dying prematurely from both natural and external causes, and provides evidence which supports calls for coordinated action on this issue.

It has been called the “scandal of premature mortality”\(^1\): people who experience mental health problems, in particular those whose illness is severe enough to lead to contact with psychiatric services, are dying prematurely.\(^2\)

While this is in part due to higher rates of suicide, chronic medical conditions such as heart disease and cancer are also important contributors to premature deaths in this population.\(^3\) There is also evidence that the difference between the mortality of those using psychiatric services and that of the general population has not diminished over time despite major changes to psychiatric care in the past century.\(^4,5\)

Studies in multiple countries including Australia,\(^6\) the United Kingdom,\(^7\) and the United States\(^8\) have demonstrated this inequality in health outcomes. However, most published studies have been restricted to subnational data, either collected regionally or by specific mental health services (for example\(^3\)), whereas in New Zealand national level data are available on public inpatient and community psychiatric service use, providing an opportunity to investigate mortality on a complete national dataset. Despite this, mortality amongst those using mental health services in New Zealand has not been explored.
New Zealand has a public health care system in which primary care attracts a part-
charge at the point of access, but all public secondary services, including mental
health care, are provided free of charge.

Most mental health care, particularly for those with more severe illness, is provided
by the public and not for profit sectors, and is increasingly provided in the
community. Public services are designed to cater for the 3% of the population with
the highest mental health needs.9

This study examines mortality rates and causes of death for adults using psychiatric
services in New Zealand from 2002 to 2010 and compares them to the total New
Zealand population.

Methods
This study examines mortality in a cohort of adults in contact with specialist adult public psychiatric
services, both community and inpatient, over a 9-year period.

Participants
Adults who had any recorded contact with New Zealand adult public psychiatric services between
January 2002 and December 2010, and who were aged 18–64 at the time of contact with services, were
eligible for inclusion. People were excluded if they had a recorded principal psychiatric diagnosis of
dementia or another organic disorder, without a diagnosis of a non-organic psychotic disorder, or a
principal psychiatric diagnosis of intellectual disability without another principal psychiatric diagnosis.

On the assumption that some psychiatric service use is secondary to terminal illness, people were also
excluded if their first recorded psychiatric service use was within 3 months prior to their death from a
natural cause (excluding psychiatric causes), if they did not have a diagnosis of schizophrenia or
bipolar disorder (as these diagnoses imply a longer standing mental illness).

Data on mental health service use in 2001 were used to provide a look-back period for the purposes of
establishing whether there was mental health service use more than 3 months before a person’s date of
death for those who died in 2002.

Data sources
All data were extracted from collections held by the New Zealand Ministry of Health, which were
linked using the National Health Index (NHI) (a unique health identifier), and subsequently
anonymised. Data on psychiatric service use came from the Ministry of Health data sets on mental
health service use. The MHINC (Mental Health Information National Collection) was established in
July 2000. In July 2008 this was superseded by PRIMHD (Project for Integration of Mental Health
Data) and all MHINC data mapped into this new system.

Data on mortality and cause of death were drawn from the New Zealand Mortality Data Collection.
The 2006 New Zealand Census was used for the national denominator population for comparisons.

Variables
Demographic variables: age, sex, ethnicity and area of residence were drawn from the NHI master
record. For all analyses presented by sex, those with unknown sex (n=6) were excluded. Prioritised
ethnicity as recorded on the NHI record was grouped into the four principal ethnic groups in New
Zealand: Māori (the indigenous population); Pacific, Asian and European (including New Zealand
European). For the analyses presented here, these were collapsed into Māori and non-Māori (all other)
ethnic groups. The New Zealand Deprivation Index 2006 (NZDep2006)10 was used to assign a
depprivation score to the area of residence.

Prioritised diagnosis—Multiple psychiatric diagnoses can be recorded for each individual on
psychiatric service records, including principal, secondary and provisional diagnoses, using ICD 9,
ICD 10AM or DSMIV. Nevertheless, many individuals have no diagnostic information or “no
diagnosis” recorded. There is a requirement that some diagnostic information is entered after a person
has been in contact with services for 30 days, with the result that many of those with no diagnostic information are those with short term contact with services.

In order to identify a single primary diagnosis for each individual to allow comparisons of mortality between diagnostic groups, a prioritisation process was used. The prioritised order of diagnoses was:

1. Schizophrenia, schizoaffective disorder and other non-organic psychoses;
2. Bipolar affective disorder and other affective psychosis;
3. Organic disorders and dementia (excluded from the current study);
4. Depression and other mood disorders;
5. Anxiety and stress disorders;
6. Substance use disorders;
7. Mental retardation (excluded);
8. Other mental health diagnoses (includes personality disorders, eating disorders, etc); and
9. “No diagnosis” or “diagnosis deferred” recorded.

Principal diagnosis was used if available, otherwise provisional diagnosis information was used.

Mental health service type and extent: Those with any inpatient service use recorded in the 9-year study period were categorised as having received inpatient care. The number of calendar years in the time period in which contact with mental health services was recorded (not necessarily continuous) was categorised into three levels: 1 year, 2 to 4 years, and 5 or more years.

Cause of death—Underlying cause of death is recorded using ICD10, based on information from death certificates and coroners reports. Cause of death was grouped into categories based on the underlying cause of death—natural causes of death (all deaths not from external causes), split into cardiovascular causes (ICD10 I chapter), cancer (ICD10 C chapter), psychiatric causes (ICD10 F chapter, includes deaths attributed to dementia, eating disorders and other psychiatric conditions) and other natural causes; and external causes of death, split into self-inflicted and other external (accident and assault and undetermined intent).

Analysis

A descriptive analysis of those using adult mental health services between 2002 and 2010 was performed to provide context for the study.

Standardised mortality ratios (SMRs) were calculated by dividing the observed mortality in those using psychiatric services by the mortality that would be expected if those using psychiatric services had the same patterns of mortality as the total New Zealand population.

The national mortality data for 2005 to 2007 (the mid-point of the study), by cause and 5-year age groups, were used for the comparison. Deaths in those under 20 were excluded from the SMR calculations as their small numbers could lead to unstable results. Only deaths prior to age 65 were included in the calculations, for the purposes of comparison to the New Zealand population. As a sensitivity analysis, the overall SMR was estimated both with and without the exclusion of those with service use only in the 3 months prior to death.

SMRs were calculated for all those using adult mental health services, and then separately for (a) those with a diagnosis of schizophrenia and other non-organic psychoses or bipolar disorder (psychotic disorders) and (b) those with substance use disorders. SMRs for other diagnoses were not calculated because of the large amount of missing diagnostic information.

To examine cause of death, SMRs were also calculated for natural and external causes of death, and for deaths from cancer and cardiovascular disease as the two most common causes of death other than suicide. Standardised mortality ratios for Māori and non-Māori mental health service users, compared to all Māori and non-Māori in the New Zealand population, were also examined separately.

All analysis was performed using SAS software (version 9).

Ethical approval for this study was granted by the New Zealand Multi-region Ethics Committee (reference number MEC/12/05/046).
Results

393,444 people who had had contact with services between 2002 and 2010 were identified from the Ministry of Health PRIMHD data set. After exclusions, 266,093 people were eligible for the study and were included in the final data set.

Figure 1 shows the numbers at each step.

Figure 1. Cohort selection process

Table 1 shows the demographic and service use characteristics and the prioritised psychiatric diagnosis of the study population. Both men and women were relatively young, with 70% under the age of 45.

The majority was of European ethnicity, and 20% were identified as Māori. Those using psychiatric services commonly lived in relatively deprived areas with around 30% living in the most deprived quintile. Approximately half of those using psychiatric services had no diagnostic information available, and this was related to the length of service use (30% of those with service contact only in 1 year had diagnostic information available, while 92% of those with 5 or more years of service use and 87% of people who had been inpatients had a diagnosis).
One-fifth of those using psychiatric services had inpatient stays during the study period. Nearly half of those seen by psychiatric services had contact with services in only one calendar year over the 9-year study period.

**Table 1. Cohort characteristics: adults using mental health services in New Zealand between 2002 and 2010**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Women</th>
<th>%</th>
<th>Men</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total with MHS use 2002–2010</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>128450</td>
<td>48.3</td>
<td>137637</td>
<td>51.7</td>
<td></td>
</tr>
<tr>
<td><strong>Age (at 1/1/06)#</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18–29</td>
<td>37835</td>
<td>29.5</td>
<td>43199</td>
<td>31.4</td>
</tr>
<tr>
<td>30–44</td>
<td>49858</td>
<td>38.8</td>
<td>51885</td>
<td>37.7</td>
</tr>
<tr>
<td>45–64</td>
<td>32394</td>
<td>25.2</td>
<td>32721</td>
<td>23.8</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>86840</td>
<td>67.6</td>
<td>84456</td>
<td>61.4</td>
</tr>
<tr>
<td>Māori</td>
<td>25855</td>
<td>20.1</td>
<td>31009</td>
<td>22.5</td>
</tr>
<tr>
<td>Pacific</td>
<td>5222</td>
<td>4.1</td>
<td>8992</td>
<td>6.5</td>
</tr>
<tr>
<td>Asian</td>
<td>5780</td>
<td>4.5</td>
<td>4186</td>
<td>3.0</td>
</tr>
<tr>
<td>other and unknown</td>
<td>4753</td>
<td>3.7</td>
<td>8994</td>
<td>6.5</td>
</tr>
<tr>
<td><strong>NZDep Score (quintile)^</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (least deprived)</td>
<td>16502</td>
<td>12.1</td>
<td>13935</td>
<td>9.7</td>
</tr>
<tr>
<td>2</td>
<td>19272</td>
<td>14.2</td>
<td>18445</td>
<td>12.9</td>
</tr>
<tr>
<td>3</td>
<td>25119</td>
<td>18.5</td>
<td>25337</td>
<td>17.7</td>
</tr>
<tr>
<td>4</td>
<td>32897</td>
<td>24.2</td>
<td>36320</td>
<td>25.3</td>
</tr>
<tr>
<td>5 (most deprived)</td>
<td>34228</td>
<td>25.2</td>
<td>43038</td>
<td>30.0</td>
</tr>
<tr>
<td><strong>Prioritised diagnosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizophrenia, other psychoses</td>
<td>8973</td>
<td>7.0</td>
<td>14075</td>
<td>10.2</td>
</tr>
<tr>
<td>Bipolar affective disorder</td>
<td>5881</td>
<td>4.6</td>
<td>4042</td>
<td>2.9</td>
</tr>
<tr>
<td>Depression and other mood</td>
<td>26443</td>
<td>20.6</td>
<td>15621</td>
<td>11.4</td>
</tr>
<tr>
<td>Anxiety and stress disorders</td>
<td>12546</td>
<td>9.8</td>
<td>8734</td>
<td>6.4</td>
</tr>
<tr>
<td>Substance use</td>
<td>4484</td>
<td>3.5</td>
<td>3415</td>
<td>2.5</td>
</tr>
<tr>
<td>Other mental health diagnoses</td>
<td>9631</td>
<td>7.5</td>
<td>21747</td>
<td>15.8</td>
</tr>
<tr>
<td>“no diagnosis” or “diagnosis deferred”</td>
<td>38295</td>
<td>29.8</td>
<td>47078</td>
<td>34.2</td>
</tr>
<tr>
<td>No diagnostic information</td>
<td>22197</td>
<td>17.3</td>
<td>22925</td>
<td>16.7</td>
</tr>
<tr>
<td><strong>Service type</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any inpatient service use</td>
<td>24025</td>
<td>18.7</td>
<td>25683</td>
<td>18.7</td>
</tr>
<tr>
<td><strong>Years of service use</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 year</td>
<td>62056</td>
<td>48.3</td>
<td>66950</td>
<td>48.6</td>
</tr>
<tr>
<td>2–4 years</td>
<td>47418</td>
<td>36.9</td>
<td>49528</td>
<td>36.0</td>
</tr>
<tr>
<td>5+ years</td>
<td>18976</td>
<td>14.8</td>
<td>21159</td>
<td>15.4</td>
</tr>
</tbody>
</table>

* 6 were of unknown sex and are not included in this table.

# 5.7% were under 18 years of age at the midpoint and 1.1% were over 65 years.

^ 994 had missing NZDep information.
Table 2. Standardised mortality ratios (SMRs) by cause of death for adults (aged 18–64) using mental health services in New Zealand 2002–2010 compared to the New Zealand population

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Women (n)</th>
<th>SMR</th>
<th>95% CI</th>
<th>Men (n)</th>
<th>SMR</th>
<th>95% CI</th>
<th>Combined SMR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All natural causes</td>
<td>2092</td>
<td>1.89</td>
<td>1.81–1.97</td>
<td>2611</td>
<td>1.78</td>
<td>1.72–1.85</td>
<td>1.83</td>
<td>1.78–1.88</td>
</tr>
<tr>
<td>Cancer</td>
<td>805</td>
<td>1.26</td>
<td>1.18–1.35</td>
<td>759</td>
<td>1.29</td>
<td>1.20–1.38</td>
<td>1.27</td>
<td>1.21–1.34</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>399</td>
<td>1.95</td>
<td>1.76–2.15</td>
<td>816</td>
<td>1.59</td>
<td>1.48–1.70</td>
<td>1.69</td>
<td>1.60–1.79</td>
</tr>
<tr>
<td>Mental health</td>
<td>56</td>
<td>9.58</td>
<td>7.37–12.45</td>
<td>81</td>
<td>5.13</td>
<td>4.12–6.38</td>
<td>6.33</td>
<td>5.35–7.48</td>
</tr>
<tr>
<td>Other natural causes</td>
<td>832</td>
<td>0.75</td>
<td>0.69–0.85</td>
<td>955</td>
<td>0.65</td>
<td>0.61–0.69</td>
<td>0.69</td>
<td>0.66–0.73</td>
</tr>
<tr>
<td>All external causes</td>
<td>832</td>
<td>4.27</td>
<td>3.99–4.57</td>
<td>1864</td>
<td>2.78</td>
<td>2.65–2.91</td>
<td>3.11</td>
<td>3.00–3.23</td>
</tr>
<tr>
<td>Other external causes</td>
<td>343</td>
<td>3.04</td>
<td>2.74–3.48</td>
<td>789</td>
<td>2.00</td>
<td>1.86–2.14</td>
<td>2.23</td>
<td>2.10–2.36</td>
</tr>
<tr>
<td>All causes</td>
<td>2924</td>
<td>2.23</td>
<td>2.15–2.32</td>
<td>4475</td>
<td>2.08</td>
<td>2.02–2.14</td>
<td>2.14</td>
<td>2.09–2.19</td>
</tr>
</tbody>
</table>

Table 2 shows numbers of deaths and standardised mortality ratios by cause of death. Over 7000 adults who had used mental health services died before the age of 65 during the study period.

The majority of deaths for both women and men were due to natural causes (71% and 58% respectively), with cancer and cardiovascular disease accounting for most deaths in this category. Suicide accounted for 15% of deaths in women and 22% of deaths in men, and other external causes (mainly accidents) were also common.

Overall those using mental health services had an SMR of 2.14, more than twice the risk of death compared to the general population. This difference was greatest for intentional self-harm and other external causes (SMR=4.4 and 2.2 respectively), but was also substantial for all natural causes combined (SMR=1.83), and for both cancer (SMR=1.27) and cardiovascular disease (SMR=1.69).

When those with psychiatric service use only in the last 3 months of their life were not excluded, the overall SMR was slightly higher at 2.26 (95% CI 2.21–2.31: sensitivity analysis not displayed in Table 2).

Table 3. Standardised mortality ratios (SMRs) by diagnosis and setting for adults using Mental Health Services in New Zealand 2002–2010 compared to the New Zealand population

<table>
<thead>
<tr>
<th>Diagnosis/Setting</th>
<th>Female (n)</th>
<th>SMR</th>
<th>95% CI</th>
<th>Male (n)</th>
<th>SMR</th>
<th>95% CI</th>
<th>Combined SMR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychotic disorders</td>
<td>630</td>
<td>3.00</td>
<td>2.78–3.25</td>
<td>938</td>
<td>2.94</td>
<td>2.76–3.14</td>
<td>2.97</td>
<td>2.82–3.12</td>
</tr>
<tr>
<td>Substance use</td>
<td>308</td>
<td>3.48</td>
<td>3.12–3.90</td>
<td>733</td>
<td>2.32</td>
<td>2.16–2.50</td>
<td>2.58</td>
<td>2.43–2.74</td>
</tr>
<tr>
<td>Any inpatient care</td>
<td>1081</td>
<td>3.84</td>
<td>3.62–4.07</td>
<td>1569</td>
<td>3.52</td>
<td>3.35–3.70</td>
<td>3.64</td>
<td>3.51–3.79</td>
</tr>
<tr>
<td>Outpatient care only</td>
<td>1843</td>
<td>1.79</td>
<td>1.71–1.88</td>
<td>2906</td>
<td>1.70</td>
<td>1.64–1.77</td>
<td>1.74</td>
<td>1.69–1.79</td>
</tr>
</tbody>
</table>

Table 3 shows standardised mortality ratios by psychiatric diagnosis and psychiatric service setting. Both men and women with psychotic disorders had mortality rates three times that of the general population (combined SMR=2.97). Women with a
principle diagnosis of substance use had an even higher mortality rate relative to the population as a whole (SMR=3.48).

Men and women who had accessed any inpatient care over the 9-year study period had much higher mortality relative to the whole population than that observed for those who had only accessed outpatient care (combined SMRs=3.64 and 1.74 respectively).

Māori mental health service users had a mortality rate one third greater than that of the whole Māori population assuming the same age structure [combined SMR 1.36 (95% CI 1.30–1.43)], while non-Māori service users had an SMR of 2.39 compared to the non-Māori New Zealand population (95% CI 2.33–2.45).

The difference between Māori using mental health services and the whole Māori population was more marked for women than men [SMR 1.50 (1.38–1.62) vs. 1.29 (1.21–1.37) respectively].

Discussion

This study is the first to examine the mortality of those using mental health services in New Zealand, and demonstrates that those with mental illness are experiencing premature mortality here just as they are in other countries. Men and women using mental health services in New Zealand have more than twice the risk of death when compared to the New Zealand population after adjusting for age. Men and women with psychotic disorders have even higher mortality, three times that of the whole population.

While suicide and accidents were important contributors to the high death rates, both men and women using mental health services also had a significantly raised risk of death from natural causes such as cancer and cardiovascular disease. Māori using mental health services also have higher mortality compared to the Māori population as a whole, but the magnitude of the difference was less for Māori than for non-Māori.

A large body of international literature points to multiple reasons for the high mortality of those using mental health services. The patterns of mortality found are important for understanding possible causes. For example the elevated risk of death from cardiovascular disease for those using mental health services in New Zealand is likely to be caused, at least in part, by the use of antipsychotic medications, which have adverse metabolic and cardiac effects. A recent international review found that monitoring of the side effects of psychiatric drugs tends to be inadequate.

In addition, smoking rates remain high amongst those using mental health services, in part because mental health services have in the past facilitated smoking. Smoking is likely to impact both on rates of cardiovascular disease and cancer. There is also evidence from other countries that those who use mental health services are less likely to receive appropriate treatment for their cardiac disease, and this may also be contributing to unequal outcomes in New Zealand.

The metabolic effects of antipsychotic medications are also likely to be a cause of the higher mortality seen in people with psychotic disorders. The effect of discrimination may also help explain the higher mortality of people with psychotic disorders.
Experience of discrimination by health service providers has been reported by people using mental health services internationally including in New Zealand, and discrimination is thought to be related to a lack of adequate preventative care or treatment for physical health problems for people with mental illness.

Moreover discrimination in wider society can lead to difficulties securing long-term employment and housing, which in turn impact on health. While such discrimination can occur against anyone with mental illness, there is some evidence that discrimination is more commonly experienced by people with psychotic disorders compared to other mental illness diagnoses.

The high mortality of those with substance use diagnoses is likely to be related to the impacts of the substances themselves, in particular alcohol. Alcohol is the most commonly used recreational drug in New Zealand and has a major impact on health and mortality.

Social deprivation will also be contributing to the reported mortality gap. Mental illness is both caused by social disadvantage, and also a cause of such disadvantage through social selection. We found that those using mental health services were more likely to live in more deprived areas, and thus social circumstances will be driving some of the increased mortality risk for this group.

Māori in New Zealand have higher rates of morbidity and premature mortality when compared to non-Māori. It might be expected that Māori using mental health services would bear a double burden of disadvantage—experiencing both the disadvantage of ethnicity and of mental health status. However these findings show that the additional burden of mortality experienced by those using mental health services compared to those of the same ethnicity in the New Zealand population was not greater for Māori than for non-Māori. Similarly, Piatt found that African Americans (who also have a higher base line mortality) with severe mental illness did not have increased premature mortality compared with white decedents with severe mental illness.

Our findings are consistent with other studies of mortality in people using mental health services, which have almost universally found excess mortality across all psychiatric diagnoses, settings and ages, and both natural and unnatural causes of death. However as far as we are aware, this is the first study to look at the impact of excluding those whose psychiatric service use is likely to be secondary to a terminal illness. People who are referred to consultant liaison psychiatry by medical or hospice services have a very high mortality rate, and so their inclusion can bias the results of this type of study.

In a recent Australian study, a high and increasing risk of death from cancer was found in those with diagnoses of stress and adjustment disorders. It is likely that this finding reflects stress and adjustment disorders secondary to cancer rather than the reverse. Because we were not able to specifically identify those accessing consultant liaison services or accessing care because of a physical illness, contact with psychiatric services only in the last 3 months of life (excluding those who died from external or psychiatric causes) was used as a way of identifying those likely to be in this group. It is notable that removing this group from the analysis reduced the SMR estimate slightly but the large gap remains.
A particular strength of this study is that it used routine national data about all people using public mental health services, as well as some NGO services, in New Zealand over a 9-year period. It is likely that virtually all deaths in this group are recorded in the national mortality data, as reporting is mandatory and the emigration rate for this group is likely to be low. However using routine data has limitations in terms of data completeness.

No psychiatric diagnosis information was available for half of those included in the study, which limited the examination of the mortality of people with specific diagnoses. However most of those with no diagnostic information had brief contact with services, and it is likely that people who did have the primary diagnoses examined (psychotic disorders or substance use) would have more prolonged contact with services and have diagnostic information recorded.

There are also no outpatient mental health service use data prior to 2001, and so the use of psychiatric services in earlier periods could not be examined. Information on psychiatric service use for those aged 65 and older is not universally included in the national mental health service use collection. The age of those included in a study of this type will impact on the results, as deaths from unnatural causes typically occur earlier in life while deaths from medical causes occur later in life.²

There are two sources of bias that may result in this study in fact underestimating the differences in mortality between those using mental health services and those who are not. First, it was not possible to exclude those who have used mental health services from the comparison population. This means we are not comparing those who have used mental health services with those who are not, but with a group that includes people with the high mortality related to mental illness. Second, the study examines a cross section of people using mental health services (a prevalent cohort), and because those who access mental health services have the highest risk of death, particularly from suicide, in the first year after diagnosis,²⁶ it is likely that the mortality for those using mental health services is underestimated with this method. However using a prevalent cohort enables inclusion of those who have long-term experience of psychiatric illness and therefore may be more likely to suffer the chronic physical effects of medication use, substance use and socioeconomic deprivation.

There have been numerous calls to action on the physical health and mortality of people with mental illness internationally (for example²⁷), and as well as in New Zealand,²⁸,²⁹ and many health services are working to address this inequality.³⁰

Primary care providers have a particularly important role as they treat the majority of those with mental illness, including those who also have contact with secondary services. However more evidence about the causes of the mortality gap and the effectiveness of interventions, including interventions in primary care, is needed. In particular more research to illuminate the causes of unequal outcomes for natural causes of death including cancer and cardiovascular disease is needed to inform appropriate action. Moreover, very little research has examined this issue from the perspective of those using mental health services.

As we have shown, adults using mental health services in New Zealand experience at least twice the mortality rate of the total population, information not previously available.
The present study provides a baseline for ongoing monitoring of the physical health of people with mental illness, and will inform the policy and research needed to address these highlighted inequalities.

**Competing interests:** Nil.

**Author information:** Ruth Cunningham¹,²; Debbie Peterson¹; Diana Sarfati²; James Stanley¹; Sunny Collings¹

¹ Social Psychiatry and Population Mental Health Research Group, University of Otago, Wellington

² Cancer Control and Screening Research Group, Department of Public Health, University of Otago, Wellington

**Acknowledgements:** This study was supported by the Health Research Council of New Zealand, as part of a Clinical Research Fellowship awarded to Ruth Cunningham (Grant number 11/846).

**Correspondence:** Dr Ruth Cunningham, Department of Public Health, University of Otago Wellington, PO Box 7343, Wellington South, New Zealand. Fax +64 (0)4 3895319; email: ruth.cunningham@otago.ac.nz

**References:**


Risk factors for general medicine readmissions and association with mortality

Manaf Aljishi, Ketna Parekh

Abstract

Aims To investigate general medicine readmissions for risk factors and association with mortality.

Method A case control study was performed comparing the characteristics of 30-day general medicine patients readmitted between 1 January to 30 June 2012 to a general medicine service at Capital and Coast District Health Board (Wellington region, New Zealand) with an equal number of randomly selected patients not readmitted to the service during the same time period.

Results 197 patients discharged from general medicine were readmitted during the 6-month study period. There were no differences in the sex, ethnicity, residential care at admission, history of dementia, length of admission or weekend discharge of readmitted patients compared to non-readmitted patients. The mean age, number of medications and comorbidities score were higher in the readmission group. Readmission (even after controlling for age, polypharmacy, and comorbidities) was a strong predictor of 1-year all-cause mortality, with an odds ratio of 2.2. Twenty-one percent of readmission patients had more than one general medicine readmission, up to 30 days between each, with even higher mortality rate compared to one readmission (49% vs. 28%).

Conclusion Readmission to general medicine is strongly associated with older age, polypharmacy, and multiple comorbidities. Readmission is an independent strong risk factor for 1-year mortality, with this risk increasing after multiple readmissions. Readmissions can be a marker of deteriorating patient’s condition, and a discussion in relation to prognosis, ceiling of treatment, resuscitation status documentation and advance directive may be warranted.

Admissions to general medicine make up a large proportion of the total admissions in most hospitals. It is estimated that about one of every six patients presenting to the emergency department get admitted to general medicine.1 However, around 11–13% of these were previously discharged from hospital within the last month.2 In addition to their impact on recovery and health outcomes, early readmission to general medicine is a burden on resources and has been linked to poorer healthcare provision.

The risk of early readmission is increased by up to 55% when healthcare is of relatively low quality.3 Furthermore, it has been postulated that hospital readmission rate can be utilised as an expedient assessment tool for the quality of inpatient care.4,5 Although the validity of this metric is debatable, readmission is invariably an outcome that both patients and clinicians want to avoid. This problem is magnified in the general medicine department due to the large volume of patients, many of whom need
multidisciplinary involvement in the often complex and challenging process of discharge planning.

There are conflicting published data on the association between readmission rates and mortality. A New Zealand study found an association between all-diagnosis readmissions and 90-day mortality. In other studies, however, there was no consistent association between readmission and mortality. In fact, among patients with heart failure, myocardial infarction and pneumonia, readmission was found to be inversely related to risk-adjusted 30-day mortality.

Evidence also suggests that hospital readmissions can be predicted in high-risk patients. Internationally, commonly reported risk factors for hospital readmissions include: older age; male sex; impaired cognition; nursing home residence; polypharmacy; low socioeconomic status; length of stay; and some clinical conditions such as heart failure. In New Zealand, readmission of elective surgical patients is more common in males, elderly and New Zealand Māori. Among older patients in New Zealand, the risk of readmission was greater in Māori, Pacific people, men and people living in deprived areas. This topic remains insufficiently addressed in the New Zealand literature.

This is a case-control study that investigates the association between readmission and mortality by comparing the characteristics of patients readmitted to general medicine within 30 days with those of patients who were not readmitted.

**Method**

**Study design**—We performed a retrospective case control study of general medicine readmissions over 6 months. The study compared patient- and admission-level characteristics as well as overall mortality in readmission patients and patients not readmitted.

**Study setting**—The study was undertaken in the General Medicine department at Capital and Coast District Health Board (CCDHB) in New Zealand, which comprises of Wellington Hospital, which is a main tertiary 434-bed academic hospital affiliated with University of Otago Wellington Medical School, and Kenepuru Hospital, which provides rehabilitation care and secondary services catering to the communities north of Wellington, including Porirua and Kapiti. Patients admitted to specialised medical departments were not included.

The study was conducted in accordance with the ethical guidelines set out by the Health and Disability Ethics Committee for the Ministry of Health, according to which formal ethical approval or an ethics committee review were not required.

**Readmission group**—This included all patients admitted into general medicine on 1 January to 30 June 2012 and were readmitted into general medicine within 30 days of discharge. The 6-month period was chosen in order to include an adequate sample size.

**Control group**—This comprised of equal number of randomly selected patients admitted into general medicine during the same duration as the readmission group but were not readmitted into general medicine within 30 days of discharge. Patients who were deceased on discharge were excluded. A Control group equal in number to the readmission group was selected from the top of the full list of non-readmitted patients that had been randomly rearranged using the random function in Microsoft Excel 2010.

**Data**—The patient data were electronically retrieved from the Decision Support Unit in Wellington Hospital. The data included: patient-level (age, sex, ethnicity, residential care residence, history of dementia and comorbidities); admission-level data (diagnosis, length of stay, number of medications at discharge); and the 1-year all-cause mortality. Patients were assigned different comorbidity scores using the Age-adjusted Charlson Comorbidity Index (ACCI), a validated prognostic index to quantify patients’ comorbidities, prognosis and risk adjustment. It predicts mortality based on the sum of differently weighted comorbid conditions, which can be improved by adjusting for the age.
Dementia was defined as any long-term cognitive deficit documented in the patient’s clinical record, regardless of aetiology. The admission diagnosis was the main diagnosis given in the discharge summary of the admission, and does not necessarily include all clinical problems encountered during the admission. The length of admission included the entire hospital stay, including transfer to Kenepuru Hospital General Medicine or Rehabilitation Departments because this was considered a continuation of the same admission. The number of medications on discharge included both short-term and regular medications. One-year mortality was defined as death by 1 year from the index discharge. The mortality data were obtained from the CCDHB electronic records and the ministry of health registry.

**Statistical methods**—The statistical analysis was conducted using SPSS software (version 20). A comparison between the two groups using the Chi-squared test for the categorical variables and the 2-sample t-test for the numerical variables was performed. Another analysis was inside the readmission group investigating an association with the number of subsequent readmissions. This utilised the Chi-squared test for the categorical variables and analysis of variance for the continuous variables.

A multivariable logistic regression analysis was performed to find variables predictive of readmission and mortality. Variables included were age, sex, dementia, ACCI, admission diagnosis, length of stay, the need for residential care, and number of medications at discharge. The significance level was chosen to be a p-value<0.05.

**Results**

During the study period, 3964 patients were admitted to general medicine, from which 197 patients (4.97 %) were readmitted back to general medicine, most of which (53%) occurred during the first 10 days after discharge. 111 readmissions (56%) were for reasons similar or related to the first admission.

The readmission patients were generally older (mean age: 73 vs. 65 years, p<0.0005), with a similar female to male ratio to controls (2:1 in both groups). Dementia did not significantly affect the likelihood of readmission (p=1.0).

Readmission did not seem to be affected by the length of stay, residential care, number of medications and discharge at weekends. There were no differences in the discharge diagnoses of readmission patients versus single admission patients (p=0.39). The number of discharge medications was significantly higher in readmission group (p=0.001) (Table 1).

Similarly, readmission patients had more complex comorbidities manifested by higher mean ACCI (4.6 vs. 3.6, p=0.002) (Table 1). However, ACCI was no longer a predictor of readmission once age and number of medications were accounted for (Table 2).

**Table 1. Comparison between readmission and control patients**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Overall</th>
<th>Controls</th>
<th>Readmissions</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>250 (64%)</td>
<td>124 (63%)</td>
<td>126 (64%)</td>
<td>0.917</td>
</tr>
<tr>
<td>Male</td>
<td>144 (37%)</td>
<td>73 (37%)</td>
<td>71 (36%)</td>
<td></td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td>68.7 (68.60–68.80)</td>
<td>64.88 (64.78–64.98)</td>
<td>72.49 (72.41–72.58)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Mean (95% CI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td>0.172</td>
</tr>
<tr>
<td>European</td>
<td>306 (78%)</td>
<td>144 (47%)</td>
<td>162 (53%)</td>
<td></td>
</tr>
<tr>
<td>NZ Māori</td>
<td>27 (7%)</td>
<td>15 (56%)</td>
<td>12 (44%)</td>
<td></td>
</tr>
<tr>
<td>Pacific</td>
<td>27 (7%)</td>
<td>18 (67%)</td>
<td>9 (33%)</td>
<td></td>
</tr>
<tr>
<td>Indian</td>
<td>19 (5%)</td>
<td>9 (47%)</td>
<td>10 (53%)</td>
<td></td>
</tr>
<tr>
<td>Chinese</td>
<td>7 (2%)</td>
<td>5 (71%)</td>
<td>2 (29%)</td>
<td></td>
</tr>
</tbody>
</table>

NZMJ 23 May 2014, Vol 127 No 1394; ISSN 1175 8716
URL: http://journal.nzma.org.nz/journal/127-1394/6128/ ©NZMA
In addition, readmission was significantly associated with 1-year mortality (32% vs 14%, p<0.0005). Since ACCI is higher in patients with early readmission (as noted previously), this mortality association was tested for independence using multivariable logistic regression model (Table 2).

Controlling for the effect of higher ACCI, readmission remained a highly significant predictor of mortality, with an odds ratio of 2.18. Because ACCI incorporates age adjustment, age is no longer a significant independent risk factor for readmission. The strongest factor that correlated with mortality was ACCI, suggesting that high mortality is mediated by higher comorbidities.

Table 2. Multivariable logistic regression analysis for the association with readmission and 1-year mortality

| Variables                  | Readmission | Mortality |
|                           | P-value     | OR   | 95% CI    | P-value | OR   | 95% CI    |
|                           |             | 95% CI |            |         | 95% CI |            |
| Age                       | 0.25        | 1.01  | (0.99–1.02) | 0.62    | 1.01  | (0.98–1.04) |
| Number of medications     | 0.02        | 1.09  | (1.02–1.17) | 0.10    | 1.08  | (0.99–1.18) |
| Dementia                  | 0.85        | 1.07  | (0.84–2.13) | 0.89    | 1.06  | (0.45–2.44) |
| ACCI                      | 0.64        | 0.98  | (0.88–1.08) | <0.005  | 1.52  | (1.28–1.81) |
| Length of stay            | 0.22        | 0.98  | (0.96–1.01) | 0.31    | 1.02  | (0.99–1.05) |
| Diagnostic category       | 0.98        | –     | –          | 0.08    | –     | –          |
| Level of care             | 1.00        | –     | –          | 0.52    | –     | –          |
| Readmission               | –           | –     | –          | 0.02    | 2.18  | (1.13–4.21) |

*Note: Even controlling for patient’s age, number of medications, and ACCI, readmission is still a significant predictor of mortality. Abbreviations: OR: Odds ratio. CI: Confidence intervals. ACCI: Age-adjusted Charlson Index.*
Among the 197 patients readmitted, 156 (79%) were readmitted once only, 33 (16.8%) were readmitted twice and 8 (4%) were readmitted three or more times in the 30 days after the index admission. The risk of higher number of readmissions did not differ with patient’s sex, ethnicity, dementia history, residential care need or the length of stay of the index admission.

Elderly patients (p=0.001) or those with higher number of medications (p=0.001) were more likely to have multiple readmissions. Having multiple readmissions was a strong predictor of mortality (p<0.05), with almost half of these patients within 12 months from the index admission (Table 3).

Table 3. Comparison between patients with a single readmission, two readmissions and more than two readmissions

<table>
<thead>
<tr>
<th>Parameters</th>
<th>1 readmission</th>
<th>2 readmissions</th>
<th>&gt;2 readmissions</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Totals</td>
<td>156</td>
<td>33</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td>0.663</td>
</tr>
<tr>
<td>Females</td>
<td>103 (66%)</td>
<td>19 (58%)</td>
<td>4 (50%)</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>53 (34%)</td>
<td>14 (42%)</td>
<td>4 (50%)</td>
<td></td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Mean (95% CI)</td>
<td>70.85 (70.75–70.95)</td>
<td>78.18 (78.0–78.38)</td>
<td>81.00 (80.8–81.2)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td>0.737</td>
</tr>
<tr>
<td>NZ European</td>
<td>96 (62%)</td>
<td>18 (55%)</td>
<td>4 (50%)</td>
<td></td>
</tr>
<tr>
<td>Other European</td>
<td>26 (17%)</td>
<td>8 (24%)</td>
<td>1 (13%)</td>
<td></td>
</tr>
<tr>
<td>NZ Māori</td>
<td>10 (6%)</td>
<td>2 (6%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Indian</td>
<td>7 (5%)</td>
<td>1 (3%)</td>
<td>2 (25%)</td>
<td></td>
</tr>
<tr>
<td>Samoan</td>
<td>7 (5%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>10 (5%)</td>
<td>4 (12%)</td>
<td>1 (13%)</td>
<td></td>
</tr>
<tr>
<td><strong>Number of medications</strong></td>
<td></td>
<td></td>
<td></td>
<td>0.008</td>
</tr>
<tr>
<td>Mean (95% CI)</td>
<td>7.29 (7.27–7.31)</td>
<td>6.79 (6.75–6.83)</td>
<td>7.50 (7.40–7.60)</td>
<td></td>
</tr>
<tr>
<td><strong>Length of index admission (days)</strong></td>
<td></td>
<td></td>
<td></td>
<td>0.810</td>
</tr>
<tr>
<td>Mean (95% CI)</td>
<td>5.28 (5.25–5.32)</td>
<td>6.55 (6.45–6.65)</td>
<td>3.50 (3.44–3.56)</td>
<td></td>
</tr>
<tr>
<td><strong>Discharge level</strong></td>
<td></td>
<td></td>
<td></td>
<td>0.689</td>
</tr>
<tr>
<td>Home</td>
<td>132 (85%)</td>
<td>29 (88%)</td>
<td>8 (100%)</td>
<td></td>
</tr>
<tr>
<td>Rest home</td>
<td>16 (10%)</td>
<td>2 (6%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Hospital level care</td>
<td>8 (5%)</td>
<td>2 (6%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>ACCI</td>
<td>4.27 (4.26–4.29)</td>
<td>5.82 (5.79–5.85)</td>
<td>6.0 (5.93–6.07)</td>
<td>0.081</td>
</tr>
<tr>
<td>Dementia</td>
<td>22 (14%)</td>
<td>5 (15%)</td>
<td>1 (13%)</td>
<td>0.992</td>
</tr>
<tr>
<td>Mortality</td>
<td>43 (28%)</td>
<td>20 (49%)</td>
<td></td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Note: Analysis utilised analysis of variance (ANOVA). ACCI: Age-adjusted Charlson Index.

Discussion

The study examined early readmissions and found a strong link between them and mortality. This adds to the existing literature on this topic. This is particularly relevant to the New Zealand setting, where little literature on this topic has been published. The factors found to be associated with readmission were age, polypharmacy and ACCI. This is consistent with the evidence that advanced age is a risk factor for readmission. Another study found the same association with polypharmacy.
Association with polypharmacy in our study is not directly attributable to medication effects because the number of presentations due to direct medication effects in the study was low, although medications can have unrecognised indirect and subtle influence on readmission rates.

The higher ACCI is also consistent with a New Zealand study, which showed that readmission patients tend to have complex comorbidities. It is notable however, that the logistic regression analysis revealed that the ACCI was no longer associated with readmission once age and polypharmacy were accounted for (Table 2), suggesting that the effect of comorbidities on readmission is tightly linked to advanced age and polypharmacy. This is consistent with the finding that polypharmacy has been clearly shown to be associated with multiple comorbidities, especially heart failure, cardiovascular disease and diabetes mellitus. Hence, a special discharge process for elderly patients with polypharmacy may be warranted. While one would hope that medication rationalization serves as a potential means of preventing medical readmissions, a recent Cochrane Review concluded that it is uncertain whether medication review reduces mortality, hospital readmissions or the cost, although it does seem to reduce emergency department contacts.

We found that medical readmission was a very strong predictor of 1-year mortality, which is consistent with a New Zealand study that found an association between readmissions and 90-day mortality. This prediction persists after controlling for patient’s age, number of medications and comorbidities (Table 2), with an adjusted odds ratio of 2.18. This suggests an independent additive effect of readmission on mortality, independent of the patient’s comorbidity profile as represented by the ACCI, bearing in mind that the ACCI does not take into account how stable or severe these comorbidities are.

However, this study did not determine whether this is a causal effect (e.g. delays in diagnosis/treatment, iatrogenic harm) or not, or whether reducing readmissions would reduce mortality. The reality is probably that for most patients, readmission is marker of more serious underlying disease rather than directly causing the high mortality. This is suggested by finding that the strongest risk factor for mortality was ACCI, even when adjusting for readmissions. For example, evidence suggests that specific heart failure-targeted interventions significantly decrease hospital readmissions but do not affect mortality rates. More importantly, most of the readmission literature focuses on stratifying risk factors for readmission and developing predictive models. However, there are relatively fewer studies that examine the association between readmission and mortality. Our finding of the strong association with mortality reinforces the prognostic significance of models that predict readmission, although their role in reducing mortality is less clear.

In New Zealand, readmissions is almost twice as likely to result in a further readmission compared to the first admission and readmitted patients are almost twice as likely to die within hospital or soon after discharge. This has been also illustrated in this study, where a significant proportion (21%) of the readmission patients had multiple readmissions back to general medicine department with very high subsequent mortality, i.e. almost 50% within 12 months (Table 3). This is clinically important as it alerts clinicians to a possible rapid decline in the patient’s health. It may also
prompt discussions with patients regarding future planning, ceiling of treatment, advance directive, resuscitation status and possibly end of life arrangements.

The patient’s level of care is an indirect measure of frailty, suggesting the need for an assessment of the readmission rates of elderly patients transferred to care facilities compared with those discharged home. A recent study found that a nursing home residence is associated with medical readmissions. Our study found no such association, which could be partly explained by the level of medical attention received in residential care and the effectiveness of assessment and discharge planning made by the multidisciplinary team in the study setting.

The current study did not detect any sex or ethnicity associations with readmissions, despite recent New Zealand studies showing that the risk of readmission was greater in Māori, Pacific and males. This might be partly due to the smaller size and limited geographic area in our study. In addition, no association between length of stay and readmission was found. Our finding is in contrast to other studies which found such association. Inconsistency between studies might actually be expected given the wide variability in patient clinical care and discharge planning processes in different study settings. In addition, length of stay is affected by many factors, some of which are non-clinical.

Although a long admission might reflect both clinical and social complexities, it can also be an indicator of extensive multidisciplinary team input that feeds toward a safe discharge. The highest rate of readmissions occurred within the first 10 days of discharge, consistent with the findings of another study and reflecting the vulnerability and instability of patients’ health immediately after discharge. This should be taken into account when arranging follow-up with the general practitioner or community nurse.

The study has some limitations. The small size and setting of the study limits its generalisability to health services in other countries. As with any retrospective study, the quality of the data is dependent on the accuracy of the clinical assessments and electronic documentation. Another potential weakness is taking the first diagnosis from the discharge summary, while realising that in elderly patients they are often multiple comorbidities that impact on the admission to hospital. Furthermore, the study only considered readmissions to general medicine, while patients readmitted to other departments were not examined. Readmission-associated morbidity was not examined either. Finally, the study did not evaluate the economic burden of early readmission and the potential benefits of preventive interventions.

In conclusion, we have studied early medical readmission and found it to be associated with older age, polypharmacy and multiple comorbidities. Our study found a close relationship between early medical readmission and mortality, with multiple readmissions increasing the risk of mortality even further.

We suggest that patients who are readmitted to a general medicine department warrant careful review with attention to remediable factors behind the readmission such as polypharmacy. Multiple readmissions should prompt discussions with patients in relation to prognosis, ceiling of treatment, advance directive and resuscitation status documentation.
Competing interests: Nil.

Author information: Manaf Aljishi, House Officer, General Medicine Department, Wellington Hospital, Capital and Coast District Health Board (CCDHB), Wellington; Ketna Parekh, General Physician, General Medicine Department, Wellington Hospital, CCDHB, Wellington

Acknowledgements: We thank Dalice Sim (Statistical Consultant, School of Mathematics, Statistics and Operations Research, Faculty of Science, University of Victoria); Associate Professor Andrew Harrison (Clinical Leader of Clinical Trials Unit, CCDHB); and Marina Dzhelali (Service Leader of Clinical Trials Unit, CCDHB).

Correspondence: Dr Manaf Aljishi, RMO Unit, Wellington Hospital, Private Bag 7902, Wellington South, New Zealand. Email: Manaf.aljishi@gmail.com

References:


Does seasonal level of serum 25-OH vitamin D correlate with the activity of Crohn’s disease?

Geogry Peter Kini, Brian Young, Peter Herbison, Michael Schultz

Abstract

Background and aim Vitamin D has immune modulating effects and normal to high levels might be correlated with less severe Crohn’s disease (CD). We aimed to review seasonal vitamin D levels in CD patients in correlation with disease activity.

Methods CD patients were identified from an inflammatory bowel disease (IBD) database and given two questionnaires enquiring about vitamin D supplementation, sun exposure, sunblock application and symptoms to complete the CDAI. Participants were examined and serum 25-OH vitamin D [25(OH)D] levels and haematocrit were determined in winter (06/2011–09/2011) and summer (12/2011–03/2012). Patients taking vitamin D supplements or with extensive small bowel resection were excluded.

Results 32 patients (19 women, mean age 39±16 years, range 18–73 years), from Dunedin, New Zealand (45°52’ S, 170°30’ E) consented to participate in the study. Of these, three took vitamin D supplements and were excluded. In winter 76% of the participants had serum 25(OH)D levels classified as deficient (<50 nmol/L) and all of them had insufficient 25(OH)D levels (<75 nmol/L). In summer, serum 25(OH)D levels were deficient only in 10% but insufficient in 55% of the participants. Mean serum 25(OH)D level was 35.9 nmol/L (norm 50–150nmol/L) in winter (range 5–67, SD 17.5) and 69.6 nmol/L in summer (range 13–119, SD 19.0) (p <0.0005). There was no significant difference in the seasonal levels of serum 25(OH)D between male and female participants (p=0.601). Mean CDAI score was 103.9 in winter (range -10–262, SD 76.9) and 90.2 in summer (range -13–331, SD 84.0) (p=0.365). A mixed-effects regression analysis showed no statistically significant correlation between seasonal levels of serum 25(OH)D and CDAI (p=0.612) among our study participants.

Conclusion Suboptimal levels of serum 25(OH)D were found in the majority of our study participants particularly in winter and they would benefit from supplementation. Our study showed no statistically significant correlation between seasonal serum 25(OH)D levels and CD activity. Given the limitations of the study, the role of 25(OH)D as a predictor of disease activity could not be clearly concluded.

Interest on vitamin D has recently shifted from its traditional role on bone metabolism to its anti-inflammatory and immune-modulating properties. Vitamin D has been implicated as a key regulator of innate immunity in many disorders including multiple sclerosis, inflammatory bowel disease (IBD), colorectal cancer and diabetes.1–4 Epidemiological studies have noted a north–south disease gradient with high incidence rates of inflammatory bowel disease in northern Europe, the UK, North America, southern Australia and New Zealand.5–7 Lack of sunlight exposure and low serum 25(OH)D level have been proposed as among the common aetiological factors for IBD.8
Vitamin D deficiency is common among patients with Crohn’s disease. This is attributed to a number of factors which include reduced efficiency of intestinal absorption of vitamin D due to the inflamed bowel, a disrupted enterohepatic circulation of vitamin D and disease activity.9–12

Vitamin D deficiency may play a causative role in the development of Crohn’s disease by virtue of its role as a direct inducer for NOD2 expression which has been shown to be deficient in a proportion of patients with Crohn’s disease and is associated with ileal disease.13,14

Association studies on a large cohort in Christchurch, New Zealand confirmed the finding of others that show a link between single nucleotide polymorphisms of the vitamin D receptor gene with IBD.15 Polymorphisms in vitamin D receptor gene lead to reduced effects of vitamin D despite normal serum levels. These studies were however not linked with the severity of the disease nor seasonal effects but significant differences were seen between males and females (more males with Crohn’s disease carry vitamin D receptor variant Taq-1 polymorphism).16

Other factors which have been shown to influence the levels of serum 25(OH)D in healthy subjects as well as diseased state are age, sex, race, body mass index, smoking, sun exposure and vitamin D supplementation.17

Studies have shown that Crohn’s disease tends to relapse in the autumn and winter months.18 Furthermore, vitamin D levels have been reported to be lower in winter and late summer in patients with Crohn’s disease compared to age- and sex-matched healthy subjects.19

Joseph et al (2009) found disease activity in CD as assessed by the Harvey Bradshaw score correlated negatively with serum 25(OH)D levels; whether pre-existing vitamin D deficiency was the initiating event leading to disease severity, or whether vitamin D deficiency was the consequence of severe underlying disease was inconclusive.20 Moreover, whilst vitamin D deficiency has been noted in more severe Crohn’s disease, whether or not vitamin D plays a role in the seasonal relapse of Crohn’s disease is still unresolved. Our study attempted to investigate this probable correlation.

**Methods**

**Study design**—This observation study was conducted in the winter (June to September) and in the summer (December to March) of 2011–2012. We identified and recruited patients who were diagnosed with Crohn’s disease from the Dunedin Hospital Inflammatory Bowel Disease database. Dunedin Hospital is a tertiary referral centre which serves a population of over 325 000 in Otago region located in the South Island of New Zealand (Dunedin 45° 52’ S, 170° 30’ E).21

Patients who were taking vitamin D supplement or had had previous extensive small bowel resection were excluded from the study. Each participant was given two sets of questionnaire (see Appendix 1); the first questionnaire enquired about sun exposure and the use of sunblock when outside, and whether they took vitamin D supplement (Q.1). The second questionnaire enquired about the symptoms to complete the Crohn’s disease activity index score (CDAI) (Q.2).
The participants were reviewed at the Gastroenterology Day Unit at the beginning and the end of the study period, and had their blood taken for 25(OH)D levels [5 mL in BD Vacutainer, SSTII Advance (serum separator tube)] and haematocrit (5 mL in EDTA tube) in both seasons. Specimens for vitamin D were spun using Spectafuge 6C (Serial number C107413) at 45,000rpm over 15 minutes and frozen before being sent in batches to the Canterbury Central Laboratory. Shimadzu LC connected to a 4000ABI mass spectrometer was used to analyse serum 25(OH)D level.

The study was approved by the Central Regional Ethics Committee of New Zealand (Ethics Reference no. CEN/11/07/040).

Patients were considered to have active disease if the CDAI score was above 150. The optimal levels of serum Vitamin D in patients with inflammatory bowel disease are still unresolved. We defined Vitamin D level in our cohort based on previous epidemiological studies on bone health as deficient if level was <50 nmol/L and insufficient if <75 nmol/L.22

Statistical analysis—The statistical analysis was performed using the Stata v12 (College Station, Texas, USA). A mixed-effects regression model was used to estimate the correlation between seasonal levels of Vitamin D and CDAI scores while allowing for the two measurements per person. Values were reported as mean±standard deviation (SD) for continuous variables and number and percent for categorical variables. A p-value of <0.05 was considered significant. Possible confounders considered included sex, age, race, time spent outside, sunblock use, dietary practice, extent of Crohn’s disease and previous bowel surgery but the numbers were too small to formally test these.

Results

Thirty-two patients (19 female, mean age 39±16, range 18–73 years)—identifying themselves as New Zealand European and of fair skin type—consented to participate in the study and had their blood taken for 25(OH)D levels and hematocrit in both seasons. Three patients took vitamin D supplement hence were excluded in the final analysis. Of the 29 participants included in the final analysis one was a vegetarian. Two patients did not return the completed questionnaires but had their blood taken for vitamin D levels and hematocrit and were included in the final analysis. The mean duration of Crohn’s disease since the diagnosis was 12.3 years (range 1–35, SD 8.5 years).

Eight, 4 and 17 participants respectively had Crohn’s disease affecting the ileum, the colon and both the colon and the ileum (L1, L2 and L3 respectively according to the Montreal Classification of Crohn’s disease). A total of 12 participants had had bowel resection; 3 had complete or partial colectomy, 1 had partial/segmental ileal resection and 8 had both partial colectomy and ileal resection. The remaining 17 participants had not had bowel resection.

Participants’ demographic and clinical characteristics are summarised in Table 1. Of all the participants who completed and returned their questionnaires, the majority spent 10 hours or less a week outside in both seasons studied (28 in winter and 15 in summer). The majority and equal number of participants in both seasons reported that they sometimes applied sunblock cream when they were outside – see Table 2.
Table 1. Demographic and clinical characteristics of participants

<table>
<thead>
<tr>
<th>Demographic/clinical characteristics</th>
<th>No. (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>12 (41%)</td>
</tr>
<tr>
<td>Female</td>
<td>17 (59%)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>39 years</td>
</tr>
<tr>
<td>Range</td>
<td>18–73 years</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>16 years</td>
</tr>
<tr>
<td>Duration of Crohn’s disease</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>12.3 years</td>
</tr>
<tr>
<td>Range</td>
<td>1–35 years</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>8.5 years</td>
</tr>
<tr>
<td>#Extent of Crohn’s disease</td>
<td></td>
</tr>
<tr>
<td>Ileum only (L1)</td>
<td>4 (14%)</td>
</tr>
<tr>
<td>Colon only (L2)</td>
<td>8 (27%)</td>
</tr>
<tr>
<td>Ileum and colon (L3)</td>
<td>17 (59%)</td>
</tr>
<tr>
<td>*Extent of bowel resection</td>
<td></td>
</tr>
<tr>
<td>Ileum only</td>
<td>1 (4%)</td>
</tr>
<tr>
<td>Colon only</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>Ileum and colon</td>
<td>8 (27%)</td>
</tr>
<tr>
<td>None</td>
<td>17 (59%)</td>
</tr>
<tr>
<td>Diet</td>
<td></td>
</tr>
<tr>
<td>Vegetarian</td>
<td>1 (4%)</td>
</tr>
<tr>
<td>Non-vegetarian</td>
<td>28 (96%)</td>
</tr>
</tbody>
</table>

#According to the Montreal Classification of Crohn’s disease.
*Bowel resection refers to total or partial colectomy and/or partial/segmental resection of the ileum.

Table 2. Sun exposure and sunblock use among the participants

<table>
<thead>
<tr>
<th>Sun exposure</th>
<th>Hours per week</th>
<th>#Winter (n=27)</th>
<th>*Summer (n=28)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Less than 5</td>
<td>11 (41%)</td>
<td>3 (11%)</td>
<td>0.098</td>
</tr>
<tr>
<td></td>
<td>5–10</td>
<td>15 (55%)</td>
<td>11 (39%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>11–15</td>
<td>1 (4%)</td>
<td>6 (21%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>More than 15</td>
<td>0 (0%)</td>
<td>8 (29%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sunblock use</td>
<td>Never</td>
<td>7 (26%)</td>
<td>7 (25%)</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>Sometimes</td>
<td>16 (59%)</td>
<td>17 (61%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Always</td>
<td>4 (15%)</td>
<td>4 (14%)</td>
<td></td>
</tr>
</tbody>
</table>

#Two participants in winter and *one participant in summer did not return completed questionnaires.

In winter 76% of the participants had serum 25(OH)D levels classified as deficient (<50 nmol/L) and all of them had insufficient 25(OH)D (<75 nmol/L) in winter. In summer, serum 25(OH)D levels were deficient in 10% and insufficient in 55% of the participants.

Mean serum 25(OH)D level was 35.9 nmol/L in winter (range 5–67, SD 17.5) and 69.6 nmol/L in summer (range 13–98, SD 19.6) (p-value <0.0005). There was no significant difference in the seasonal levels of serum 25(OH)D between male and female participants (p-value=0.601).

Overall, 70% of the patients were in clinical remission (CDAI <150) in winter compared with 86% in summer. Mean CDAI score was 103.9 in the winter (range -10–262, SD 76.9) and 90.2 in the summer (range -13–331, SD 84.1), however the difference was not statistically significant (p=0.365)—Table 3.
A mixed-effects regression analysis showed no statistically significant relationship between the seasonal levels of serum 25(OH)D and the activity of Crohn’s disease among our study participants (p=0.612).

Table 3. Seasonal levels of serum 25-OH vitamin D and CDAI scores among the participants

<table>
<thead>
<tr>
<th>25(OH)D level, nmol/L</th>
<th>Winter</th>
<th>Summer</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>35.9</td>
<td>69.6</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Range</td>
<td>5 - 67</td>
<td>13 –98</td>
<td></td>
</tr>
<tr>
<td>Standard deviation</td>
<td>17.5</td>
<td>19.0</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CDAI score</th>
<th>Mean</th>
<th>Range</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Winter</td>
<td>103.9</td>
<td>-10–262</td>
<td>76.9</td>
</tr>
<tr>
<td>Summer</td>
<td>90.2</td>
<td>-13–331</td>
<td>84.1</td>
</tr>
<tr>
<td>P-value</td>
<td>0.365</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Discussion

The appropriate thresholds for vitamin D deficiency are debatable. One study shows that for all health-related endpoints, the most advantageous serum levels for 25(OH)D appear to be at least 30 ng/mL (75 nmol/L), and for cancer prevention desirable levels are between 36 and 48 ng/mL (90–120 nmol/L). However the amount of 25(OH)D essential to support the immune system is not clear.

In this study, all of the participants had suboptimal levels of serum 25(OH)D in winter (<75 nmol/L) despite being in clinical remission in the majority (70%) in regard to their CD as reflected by their CDAI scores. This was probably attributed to a number of factors including the lack of sun exposure as reflected by the number of hours spent outside, the use of sunblock when outside which prevented ultraviolet light from reaching the skin for 25(OH)D synthesis, lack of sunlight in Dunedin for most of the period under study due to its geographical location, and inflamed bowel. As aforementioned, none of the study participants had had extensive small bowel resection hence this did not account for the low serum 25(OH)D observed.

Although most of the participants in this study had higher levels of serum 25(OH)D and lower CDAI scores in summer compared with in winter, we found no statistically significant correlation between seasonal levels of serum 25(OH)D and CDAI scores (p=0.612).

Our study concurred with that of O’Neill R et al (2011) which showed no correlation between these two variables in their cohort of 335 IBD patients. However, our finding did not concur with the finding from earlier studies; Joseph AJ et al (2009) showed in their cohort that serum 25(OH)D levels were lower in those with more active Crohn’s disease and less sun exposure, Ulitsky et al (2011) showed in their cohort that 25(OH)D deficiency was independently associated with lower health-related quality of life and greater Crohn’s disease activity.

Whether pre-existing 25(OH)D deficiency is the initiating event leading to disease severity or 25(OH)D deficiency is the consequence of severe underlying disease is unknown. The truth is likely somewhere in between given that patients with more
active disease tend to get less exposure to sunlight and not absorb sufficient amounts of 25(OH)D from their diet.

The main strong points of this study were enrolment of patients was done using strict exclusion criteria excluding those with confounding factors that would affect serum 25(OH)D levels, sampling was performed in summer and winter in all patients, and validated scoring system—i.e. CDAI was used for the assessment of disease activity.

The main limitations of this study were its small size, that it was an uncontrolled observational study and that it was conducted at a single centre on a group of participants of European origin. Moreover, our study population was heterogenous in regard to previous bowel surgery. Another limitation was that the study did not compare 25(OH)D levels from the normal population who were not affected by Crohn’s disease.

In conclusion, serum 25(OH)D levels were suboptimal in the majority of our study participants and would benefit from supplementation. However, our study did not show statistically significant correlation between seasonal levels of serum 25(OH)D and the activity of Crohn’s disease.

Given the limitations of the study, the role of 25(OH)D as a predictor of disease activity could not be clearly concluded from the findings of this study. Future studies with a bigger sample size, recruiting participants from various ethnic backgrounds and comparing with serum 25(OH)D levels from the normal population without Crohn’s disease were recommended.

Competing interests: This study received research funding from the Dean’s Bequest Fund of Dunedin Medical School and the New Zealand Society of Gastroenterology Research Grants. There was no perceivable conflict of interest at the time of publication.

Author information: Geogry Peter Kini, Gastroenterology Advanced Trainee, Dunedin Hospital, Dunedin; Brian Young, Director, Research and Enterprise Office, University of Otago, Dunedin; Peter Herbison, Professor of Epidemiology, Department of Preventive and Social Medicine, Dunedin School of Medicine, Dunedin; Michael Schultz, Consultant Gastroenterologist and Senior Lecturer, Department of Medicine, Dunedin School of Medicine, Dunedin

Acknowledgements: The authors acknowledge the Dean’s Bequest Fund of Dunedin Medical School and the New Zealand Society of Gastroenterology Research Grants for the funding received as well as Dr Lim Ming Han for his assistance in the collection of data.

Correspondence: Dr Geogry Peter Kini, Gastroenterology, Dunedin Hospital, Great King Street, Dunedin 9054, New Zealand. Email: drgeogry00@yahoo.com

References:
Appendix 1

Q1. Questionnaire 1 enquiring sun exposure, sunblock use, Vit D supplement, diet and duration of CD.

---

Questionnaire 1

Seasonal levels of Vitamin D and correlation with Crohn’s disease activity

1. How many hours a week on average do you spend out in the sun?
   a. Summer (December – March)
      - Less than 5 hours
      - 5 to 10 hours
      - 10 to 15 hours
      - More than 15 hours
   b. Winter (June – September)
      - Less than 5 hours
      - 5 to 10 hours
      - 10 to 15 hours
      - More than 15 hours

2. Do you apply sunscreen when you are out in the sun?
   - Always
   - Sometimes
   - Never

3. Are you currently taking any supplement containing Vitamin D?
   - Yes
   - No

4. How often do you take the above vitamin supplement?
   a. Winter (June – September)
      - Daily
      - Weekly
      - Monthly
   b. Summer (December – March)
      - Daily
      - Weekly
      - Monthly

5. When did you start taking this vitamin supplement in relation to the diagnosis of Crohn’s disease?
   a. Before diagnosis
   b. After diagnosis

6. Are you a vegetarian?
   - Yes
   - No

7. When was your last Crohn’s disease flare-up?
   - Month
   - Year

Seasonal Vitamin D levels and correlation with Crohn’s disease activity
Version 3.0, April 2011

---
Q2. Questionnaire 2 enquiring symptoms to complete CDAI score

![Questionnaire 2](image)

**Calculating your Crohn’s Disease Activity Index (CDAI)**

The CDAI is used to gauge the progress of Crohn’s disease. Please fill in the section below which will help us to calculate your CDAI score.

**Week commencing:**

<table>
<thead>
<tr>
<th>Day</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of liquid or very soft stools</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal pain rating</td>
<td>0 = none, 1 = mild, 2 = moderate, 3 = severe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General wellbeing</td>
<td>0 = generally well, 1 = slightly under par, 2 = poor, 3 = very poor, 4 = terrible</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fever over 37.8°C during the past week</td>
<td>Yes ☐ No ☐</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have you taken Lomotil/Imodium/Loperamide/Opiates?</td>
<td>Yes ☐ No ☐</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg) :</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm) :</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Seasonal Vitamin D levels and correlation with Crohn’s disease activity
Version 4.0, April 2011
From ICU to hospital-wide: extending central line associated bacteraemia (CLAB) prevention

Mary E Seddon, Catherine J Hocking, Elizabeth A Bryce, Jackie Hillman, Vicki McCoubrie

Abstract

Aim To decrease hospital-wide central line associated bacteraemia (CLAB) by spreading the prevention programme beyond the intensive care unit (ICU) in a secondary care hospital in Auckland, New Zealand.

Method Over 15 months, four general surgical wards, five inpatient units, and surgical theatres adopted the quality improvement initiative, and were followed for a further 15 months. The initiative included central line insertion and maintenance checklists, a central line insertion pack, training in central line care, and a dedicated database. In addition, a checklist to assess the readiness of each new area was developed; data collection and analysis processes embedded, with rapid feedback to staff and in-depth review of all CLAB events.

Results Compliance measures improved significantly (compliance with insertion increased from a mean of 84% to 92% p=0.001; maintenance from 64% to 85%, p=0.002). The absolute numbers of CLAB fell hospital-wide from a mean of 2.3/month to 0.56/month. The rate of CLAB hospital-wide decreased from 7.04/1,000 line days to 1.37/1,000.

Conclusion We have demonstrated that the CLAB prevention work proven effective in the ICU can be successfully adapted and expanded to the rest of the hospital. As central lines are increasingly inserted in units outside the ICU, and maintained in general wards, this work provides some useful insights into tackling this larger problem.

Central line associated bacteraemia (CLAB) ranks as one of the most frequent and serious nosocomial infections with high mortality rates. It also puts an economic burden on healthcare systems with increased hospital stays and total hospital costs.

In 2004, Berenholtz et al showed that it was possible to significantly reduce CLAB rates in an intensive care unit (ICU), and later this group extended the work to over 100 intensive care units in Michigan state, USA. The improvement came through a multifaceted programme that included a checklist of evidence-based practices focussing on five key elements: maximal sterile precautions when central lines were inserted; hand hygiene, chlorhexidine skin antisepsis, optimal site selection for insertion and daily review of the need for a central line.

A review of this work identified that part of the success was the reframing of CLAB as a social problem and addressing it through a professional movement, strong networks, and “using several interventions that functioned in different ways to shape a
culture of commitment to doing better in practice.”

This challenged the conventional wisdom that viewed CLAB as an inevitable complication of intensive care.

We have previously published in this journal the results of the first New Zealand CLAB prevention initiative, which started in 2008. Our ICU CLAB rate was unacceptably high at 6.6/1,000 line days, and over the next 2 years—building on the Michigan work—we were able to reduce this to a mean rate of 1.3/1,000 and a median of zero.

In this paper we report on how we have extended this quality improvement initiative to the rest of the hospital, with significant reductions in CLAB rates.

Auckland’s Middlemore Hospital is a 900-bed secondary care hospital, providing some tertiary care including the National Burns Centre (NBC), serving a population of approximately 500,000. Although many central lines are inserted in the ICU, large numbers are also inserted in the Renal Service, Neonatal Unit (NNU), Theatre and Radiology. Radiologists predominantly insert peripherally inserted central venous catheters (PICC), though studies suggest that the rate of infection associated with these lines are similar to conventional central lines. Increasingly patients with central lines in situ are nursed on the general wards.

A review of CLAB throughout the Hospital indicated that the Neonatal Unit, Renal Service and Surgical wards were key areas where CLAB rates were high.

A sequential approach was taken over 15 months, starting with areas that expressed willingness to be involved, and extending to include all that inserted or maintained a large number of central lines. In some areas (e.g. Theatres and the Renal Service) the initiative centred on ensuring safe central line insertions, and in others (such as the general surgical wards), the focus was on safely maintaining the line.

The wider hospital was a greater challenge than the circumscribed ICU, and required a different approach.

Method

A working group met for 1 hour each week. The team included the ICU Quality Coordinator, an Infection Prevent & Control Nurse, IV Clinical Nurse Specialist and the Clinical Director for the Centre for Quality Improvement. As each ward or unit joined the initiative, they added representatives to this group.

Checklists

From October 2010 to February 2012, 10 clinical areas engaged with the CLAB prevention programme, starting with Emergency Care and ending with Radiology. A separate roll-out checklist was developed that identified critical aspects to address prior to implementation. This ensured that the leadership (clinical and managerial) in each unit was supportive, all staff were informed, and a clinical champion was identified.

Responsibility for data collection in each area was assigned and supported. Data on CLAB numbers were presented to meetings of clinician’s along with both local and international evidence of CLAB prevention success.

Working with the staff involved, the central line insertion and maintenance checklists developed in ICU were modified where necessary to incorporate any unit-specific
differences (see Figure 1). Changes included accommodating different insertion sites (e.g. umbilical for neonates) and nursing shifts (12 and 8 hour shifts). Definitions of what constituted a ‘high-risk’ line were refined (e.g. inserted under emergency conditions, line used for total parenteral nutrition) for each area. Each unit developed their own way of collecting the checklists and assigned staff to enter the data.

Figure 1. Maintenance Checklist

The insertion pack previously developed for the ICU was modified to suit different patient needs. The renal patients required a different sterile drape (with a larger aperture) and the neonatal unit needed a drape for their small patients (prior to this initiative they had made do with four small drapes held together with bull clips). Both these drapes were developed and procured.

The pack was designed to decrease the time taken by staff putting together all the equipment needed for each insertion, and to encourage compliance. On opening the
sterile pack, the first thing the clinician sees is the hat and mask, after putting these on, the pack opens further and the sterile gown is presented, before the insertion tray is found, including the appropriate skin preparation (e.g. for adults chlorhexidine 2% in 70% alcohol). The pack was entered into the hospital ordering system so that units could order their own supply.

The central line database developed for ICU was modified to allow each unit/ward to record line insertion and line maintenance data for their patients.

On the wards, at least two staff (usually nursing) were identified to input the central line data, with rapid summary results available (no more than two mouse clicks to produce graphs of outcomes and compliance).

Each unit placed a poster in a prominent place that recorded the number of CLAB-free days (see Figure 2)

**Figure 2. Daily feedback by staff on CLAB-free days**

![Figure 2. Daily feedback by staff on CLAB-free days](image)

Each CLAB was identified independently by a microbiologist and Infection Prevention & Control nurse specialist, using the CDC surveillance definition for CLAB. (10)

If a CLAB occurred, the clinical team were notified immediately. Each CLAB was treated as a sentinel event and the clinical staff were provided with a standardised electronic form to record their investigation findings. These were reviewed at the
working group meetings and learning’s from these investigations were shared with all areas, with an emphasis on quality improvement rather than criticism.

Data analysis

Process measures—Compliance with the insertion checklist was measured for units that only inserted lines (Theatre, Radiology, Emergency Care and Renal Services). The compliance with the maintenance checklist was measured for those units that did not insert lines, but only cared for patients with central lines already in situ (NBC, and Surgical wards). And both processes were measured for the NNU, which inserted and maintained lines.

Balancing measures—We retrospectively measured the number of blood cultures completed from the surgical wards, NBC and NNU. A significant drop in blood cultures could have affected the determination of a CLAB, which is reliant on adequate blood cultures being taken. We were unable to retrospectively measure blood cultures per 1,000 line days so have instead used a proxy measure of blood cultures/1,000 bed days.

Outcome measure—The absolute numbers of CLAB for each unit and the hospital overall (excluding ICU) were measured and combined with the total line days to get a rate of CLAB per 1,000 line days.

Mintab® was used to produce statistical process control charts. The process measures were plotted as an “I chart” (using % compliance) while the CLAB rate used a “U chart.” (11) A two-sampled t-test was used for the difference in means when a statistically significant shift was demonstrated. The blood culture rate over three years was evaluated using the non-parametric Mann-Whitney U test.

Results

Process measures

Compliance with the Insertion Checklist—This measured compliance in Theatre, NNU, Emergency Department, Renal Unit and Radiology. The ‘I chart’ demonstrates a statistically significant shift in January 2012, with an increase in the mean compliance from 84.22 to 92.38% (see Figure 3). The difference between these two means was significant at the 0.05 level (p=0.001).
Compliance with Maintenance Checklist—This included the NNU, the NBC, and four general surgical wards. The chart (figure 4) shows a statistical shift (p=0.002) in February 2012, with an increase in mean compliance from 63.5 to 85.1%. The degree of variation also decreased significantly with a SD of 21.2 in the first phase and 3.76 in the second.
Balancing measures—There was no statistically significant differences found between the blood culture rate per 1,000 bed days taken on the surgical wards, the NBC or the NNU in 2010 and 2011 (p=0.13), 2011 and 2012 (p=0.18) or 2010 and 2012 (p=0.86). See Figure 5.

Figure 5. Blood culture numbers/1,000 bed-days

<table>
<thead>
<tr>
<th>Ward</th>
<th>Blood cultures/1,000 bed-days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2010</td>
</tr>
<tr>
<td>Gen Surg A</td>
<td>35.24</td>
</tr>
<tr>
<td>Gen Surg B</td>
<td>21.53</td>
</tr>
<tr>
<td>Gen Surg C</td>
<td>23.36</td>
</tr>
<tr>
<td>Gen Surg D</td>
<td>24.96</td>
</tr>
<tr>
<td>National Burns Centre</td>
<td>26.46</td>
</tr>
<tr>
<td>Neonatal Unit</td>
<td>75.69</td>
</tr>
<tr>
<td>Median</td>
<td>33.14</td>
</tr>
</tbody>
</table>

Outcome measures—The absolute numbers of hospital-wide (excluding ICU), inpatient CLAB (Figure 6) fell significantly in March 2012, with 8 months in which there were zero in-hospital CLAB infections.

The NNU, that previously had 2 or 3 CLAB per month prior to the initiative, went several months without a CLAB. Likewise several surgical wards had over 200 days between CLAB infections.

Figure 6. Hospital-wide CLAB numbers
The rate of CLAB/1,000 days also showed a shift in March 2012 (Figure 7), which has been maintained for more than a year, with a mean of just over 1 CLAB/1,000 line days, down from 7.04/1,000. The degree of variation also decreased in the second phase (SD 6.30 to 1.5).

Figure 7. Rate of hospital-wide CLAB per 1,000 line days

Discussion

Central line associated bacteraemia is a serious healthcare associated infection, and our study has shown that the lessons learned in ICU CLAB prevention can be extended into other units and wards in the hospital.

We have shown a reduction in the rate of hospital CLAB to just over 1/1,000 line days. The improvement came after an increase in compliance with the insertion checklist, and a little later, an improvement in compliance with the maintenance bundle. This improvement and the decrease in CLAB rate has been maintained for over a year.

The effort to engage the ICU in CLAB prevention was considerable, as it challenged the attitudes of healthcare professionals, their wariness of standardised medicine, their reticence to use checklists, and their concerns about having their practice scrutinised by other staff members. The challenge was far greater outside the ICU with many more staff involved, and some of the same attitudes. Although we had shown that our checklists had been part of the improvement in the ICU, medical professionals in particular wanted to be shown the evidence for each component of the checklist and some routinely refused to comply (one clinician steadfastly refused to wear a hat and mask when inserting a central line meaning that none of his insertions were compliant).
One of the biggest challenges was having a system to measure line days. We had managed this in the ICU, but line days and indeed patients with central lines were not routinely measured on the wards. Systems also had to be developed to measure compliance with the maintenance of the line; this is considerably more complicated than compliance with line insertion which is a one-off event.

There is a cap on hiring administrative staff in New Zealand hospitals so this data entry tended to be done by frontline staff. Though not an ideal use of their time, it did mean that they could spot problems with compliance and discuss directly with medical or nursing staff. It also ensured clinical engagement and a clinical champion or champions in each area.

It took just over a year to engage all the wards/units and there were several false starts. This can be seen in both the insertion and maintenance compliance charts. Maintenance compliance improved from 40% to over 90% in the first few months; however, this was only sustained for four months before decreasing again to 20%.

Sometimes this was due to changing personnel (a supportive charge nurse resigning), new units coming on board and taking time to come up to speed, and sometimes the collective focus on the initiative was lost amongst all the other quality improvement initiatives going on in the hospital.

Perseverance from the working group, and a focus on providing support and education to staff, along with the identification of CLAB as a preventable sentinel event, helped to ensure high compliance for the last year with the insertion and maintenance checklists, and this is mirrored in the continued low rate of CLAB.

The CVL insertion compliance showed a statistical shift starting in January 2012. The improved compliance with the maintenance checklist started in February 2012 and the shift was confirmed in May. The CLAB rate shift started in March – at a time when both compliance rates were high.

Standardising the care of all central lines (“its what we do”) and incorporating the maintenance checklist into the record of nursing care was useful to build buy-in from the nursing staff.

The insertion pack also proved to be an advantage in spreading the improvement outside the ICU. The packs were very tangible symbols, and they were also designed to encourage good, safe central line insertion practice - it made doing the right thing the easy thing to do.

There have been few other studies reporting CLAB prevention initiatives outside the ICU. A recent Spanish study in a 350-bed hospital, estimated that more than 85% of CLAB occurred in the non-ICU setting and they were also able to demonstrate a decrease in CLAB rate from 15.1/1,000 line days to 10.1 after introducing an insertion checklist.

A Thai tertiary hospital, using a mixture of QI methods with a strong emphasis on hand hygiene, resulted in a decrease in CLAB rates from a high starting point (14/1,000 line days) to 1.4/1,000. They had an endemic rate of *Acinetobacter baumannii* in their ICU at the start of the project and initiated a number of interventions to get this under control.
Cincinnati Children’s Hospital—a quaternary centre—used a QI collaborative in 3 critical care units, oncology and bone marrow transplant units, and medical and surgical wards. They started with a low rate of catheter-associated bloodstream infections (3.0/1,000 line days) and decreased this further to less than 1.16

Another American paediatric tertiary hospital also showed a reduction in CLAB in their 18-bed paediatric oncology unit with the introduction of the maintenance bundle.17

These studies were carried out in tertiary/quaternary hospitals and included the ICU in their work. Our study was in a large secondary care hospital and excluded ICU. The studies mentioned above, used a variation on the Michigan work and our paper lends support to the evidence that such improvement work can be successful outside the ICU.

There are some limitations to our study. First, we cannot rule out that other unknown influences led to the reduction in CLAB rates outside the ICU. We have no data from other centres in New Zealand to compare ourselves with as no other hospital was collecting line days outside the ICU. Second, we were unable to measure blood culture rates in patients with lines in situ and had to use a proxy measure of blood cultures/1,000 bed-days. We were not able to detect a significant difference in these rates over the study period which gives us some confidence that the decrease in CLAB rates was not due to decreases in blood cultures taken, but it is only an approximation. Third, like the other studies in this area, this work was done in a single institution. The success may not be replicable at other centres with different organizational cultures.

The group is now working on decreasing CLAB further with a focus on outpatient haemodialysis and maintenance of central lines in theatre.

**Conclusion**

CLAB prevention strategies that have been shown to work in the ICU can be amended and successfully used in other hospital areas. The challenges in CLAB prevention are similar, albeit on a wider scale.

We have shown a significant reduction in CLAB rates for the last 12 months—we were able to decrease CLAB rates from 7.04/1,000 lines days down to 1.37 hospital-wide—and the CLAB prevention initiative is now business as usual in 10 units/wards.

**Competing interests:** Nil.

**Funding:** The study was supported by Counties-Manukau Health and there was no external funding.

**Author information:** Mary E Seddon, Clinical Director, Centre for Quality Improvement, Middlemore Hospital, Auckland; Catherine J Hocking, Quality Coordinator, Critical Care Complex, Middlemore Hospital, Auckland; Elizabeth Bryce, Infection Prevention & Control Specialist Nurse, Centre for Quality Improvement, Middlemore Hospital, Auckland; Jackie Hillman, Clinical Speciality Nurse, Surgery & Ambulatory Care Services, Middlemore Hospital, Auckland; Vicki McCoubrie, Clinical Nurse Manager, IV Access Team, Middlemore Hospital, Auckland
Acknowledgements: The authors acknowledge Prem Kumar and Alison M Pirret of Counties-Manukau Health for their help with statistical analysis.

Correspondence: Catherine J Hocking, Critical Care Complex, Middlemore Hospital, 100 Hospital Road, Otahuhu, Auckland 1640, New Zealand. Email: Catherine.hocking@middlemore.co.nz

References:


Rising antimicrobial resistance: a strong reason to reduce excessive antimicrobial consumption in New Zealand

Mark G Thomas, Alesha J Smith, Murray Tilyard

Abstract

Steadily rising rates of antimicrobial resistance, in a range of common bacterial pathogens, are a major threat to human health in New Zealand in the near future. The single largest contributor to this threat is the high level of antimicrobial consumption in New Zealand.

Antimicrobial consumption in New Zealand needs to be reduced if we are to slow the spread of antimicrobial-resistant bacteria. Reporting the per capita antimicrobial consumption within each District Health Board (DHB), in relation to targets for reductions from present levels of consumption, could provide an impetus for DHBs to address this threat to the health of their populations.

Antibiotic resistance is a growing problem in New Zealand

Resistance to commonly used antimicrobial medicines, in a wide range of bacteria responsible for common diseases, is rapidly emerging as a major threat to health in New Zealand. For example, in New Zealand during 2012, approximately 4,000 people suffered from infections due to strains of *Escherichia coli* (*E. coli*) or *Klebsiella pneumoniae* (*K. pneumoniae*) or other closely related organisms that produced an extended spectrum beta-lactamase (ESBL).\(^1\)

The production of an ESBL by these bacteria confers resistance to almost all penicillins and all cephalosporins. Associated resistance genes in these ESBL-producing bacteria frequently confer resistance to many other antimicrobials.

The epidemic of ESBL-producing bacteria in New Zealand, has grown rapidly in recent years. During 2006–2008 approximately 2.6% of *E. coli* isolated from blood cultures in New Zealand were ESBL positive; by 2009–2011 the proportion had risen to 3.8%—a 46% increase.\(^2\)

Experience in other countries suggests that ESBL-producing bacteria may become much more common in New Zealand in the next few years. For example, between 2000 and 2011, the proportion of isolates of *E. coli* that were resistant to third-generation cephalosporins (an approximate surrogate for the production of an ESBL) increased from 0.1% to 5.7% in the Netherlands, and from 3.6% to 14.9% in Greece.\(^3\)

The dramatic increase in ESBL-producing *E. coli* and *K. pneumoniae* is but one example of the growth in antimicrobial resistance in New Zealand in recent years. Resistance to mupirocin, the active component in Bactroban\(^\text{©}\) ointment, was present in <5% *Staphylococcus aureus* strains isolated from patients in Auckland in 1992, but by 2000 had risen to >20%.\(^4\) The proportion of strains of *Neisseria gonorrhoeae* (*N. gonorrhoeae*) that are resistant to ciprofloxacin has risen from 6% in 2002 to 40.6% in 2012.\(^5\) (Figure 1)
Increasing levels of antibiotic resistance cause inconvenience and risk for patients and increased costs for the health system

The emergence of high rates of resistance, in a wide range of bacterial species, during recent decades, has had significant impacts on patients and on the healthcare system. For most of the approximately 4000 patients who suffered from an infection due to an ESBL-producing E. coli or K. pneumoniae during 2012, the only reliable and safe treatment would have been a carbapenem antibiotic, such as meropenem or ertapenem. Because these antibiotics are not orally absorbed, patients with infections due to an ESBL-producing strain of E. coli or K. pneumoniae commonly require intravenous therapy, usually administered within hospital.

In contrast, patients infected with strains of E. coli or K. pneumoniae that do not produce an ESBL can commonly be treated with an oral agent, such as trimethoprim, amoxicillin plus clavulanate, ciprofloxacin or nitrofurantoin, and therefore usually do not require admission to hospital. Because of the high prevalence of resistance to ciprofloxacin, patients with gonorrhoea are now usually treated with intramuscular ceftriaxone, instead of oral ciprofloxacin.
Use of second-line antibiotics to treat these more resistant strains of *E. coli*, *K. pneumoniae*, and *N. gonorrhoeae* results in increased discomfort and inconvenience for the patient, and increased costs for the healthcare system.

**Completely untreatable bacterial infections are a rapidly emerging threat**

At present, for most infections due to antibiotic-resistant bacteria, a relatively effective treatment is available, even if this treatment has disadvantages with regard to cost or convenience or adverse effects. However strains of *E. coli* and *K. pneumoniae* that produce a carbapenemase, and so cannot be effectively treated with meropenem or ertapenem, have been isolated from relatively small numbers of patients in New Zealand, and there has been an alarming increase in their prevalence in hospitals overseas. These infections are often untreatable and cause high death rates.

A similar problem has recently emerged in Russia where excessive antimicrobial consumption has led to resistance to all available antimicrobial agents in over 5% of strains of *Pseudomonas aeruginosa* (*P. aeruginosa*) isolated from hospital inpatients.

If untreatable infections, due to *E. coli*, *K. pneumoniae*, *P. aeruginosa* or other completely drug-resistant organisms become common here, this will have major consequences for the New Zealand healthcare system. For example, the surgical implantation of prosthetic materials, and the potent immunosuppression required for successful solid organ or bone marrow transplantation, place many patients at risk of severe infection by a wide range of organisms, including *E. coli*, *K. pneumoniae* and *P. aeruginosa*.

At present, because of the very low rates of completely drug-resistant organisms in New Zealand, the infectious risks of these modern medical procedures are considered acceptable for most patients. However, if an increasing proportion of procedure-related infections are caused by untreatable bacteria, the infectious risks may be considered much less acceptable and the use of these procedures curtailed. Increasing antibiotic resistance therefore threatens a very wide range of current medical and surgical practices. It should be of great concern to the community and to all healthcare workers.

**New antibacterial drugs will not be available in the next decade**

Until about 15 years ago the emergence of resistance to a single antimicrobial, or even to all members of a class of antimicrobials, was merely an inconvenience. During the 1970s and 1980s, large numbers of new antimicrobials had been developed, and together these new drugs ensured that infections due to almost any bacteria could be readily cured. However, since the 1990s, pharmaceutical companies have devoted minimal resources to development of new antimicrobials and the pipeline of new drugs has dried up.

It seems very unlikely that new drugs will be developed in time to effectively treat infections caused by the increasing numbers of highly resistant bacteria. Consequently we must change our strategy from accepting the emergence of bacterial resistance as a minor inconvenience to doing all that is feasible to slow the emergence of resistance.
High levels of consumption of antibiotics is the primary cause of high rates of antibiotic-resistant bacteria

The emergence and proliferation of bacteria resistant to an antibacterial is directly related to the amount of that antibacterial in the organism’s environment.

As a result, the prevalence of strains of bacteria resistant to an antimicrobial class increases more rapidly in those countries where large amounts of antimicrobials within that class are consumed. For example, strains of *S. pneumoniae* with reduced susceptibility to penicillins are common in Spain and France, where large amounts of penicillins are consumed.\(^3,10\) In contrast, strains of *S. pneumoniae* with reduced susceptibility to penicillins are unusual in Germany and the Netherlands, where lower amounts of penicillins are consumed (Figure 2a).

Similarly strains of *E. coli* resistant to ciprofloxacin are common in Spain and Italy, where large amounts of fluoroquinolones are consumed\(^3,10\) In contrast, strains of *E. coli* resistant to fluoroquinolones are less common in Norway, Sweden and Finland, where lower amounts of fluoroquinolones are consumed (Figure 2b).

**Figure 2.** Consumption of penicillins (defined daily doses [DDDs]/1000 population/day) by community-based patients (i.e. not hospital inpatients) during 2010, in relation to the prevalence of reduced susceptibility to penicillin in strains of *S. pneumoniae* isolated during 2010; (a) and consumption of fluoroquinolones (DDDs/1000 population/day) by community-based patients during 2010, in relation to the prevalence of resistance to ciprofloxacin in strains of *E. coli* isolated during 2010; (b) for a number of large European nations.\(^5,10\)
Note: The per capita level of antimicrobial consumption by community-based patients is commonly measured in defined daily doses (DDDs) per 1000 population per day. The DDD is the weight of each medicine that has been agreed by an international panel to be the standard daily dose used when treating an otherwise healthy adult (e.g. oral amoxicillin: 1g; roxithromycin: 300mg; ciprofloxacin: 1g; doxycycline: 100mg; trimethoprim: 400mg).11

Antibiotic consumption in New Zealand has risen rapidly and is high by international standards

Antimicrobial consumption in New Zealand has been relatively prudent in the past, and consequently the prevalence of antimicrobial resistance in most organisms has, until recently, been relatively low.

However, in New Zealand during the 7 years between 2005 and 2012, annual per capita antimicrobial consumption by community-based patients increased by 43%, an average annual increase of just over 6% (Figure 3).
Figure 3. Annual per capita consumption of antimicrobials, by community-based patients in New Zealand, measured in DDDs/1000 population/day, between 2005 and 2012

Note: Community pharmacy dispensing data for antimicrobials was obtained from the National Pharmaceutical Collection, in the Ministry of Health,\textsuperscript{12} and the number of people resident in New Zealand during each year was obtained from Statistics New Zealand.\textsuperscript{13}

The overall level of consumption of antibiotics, by community-based patients in New Zealand in 2010, was less than that in Greece, Belgium, France and Italy, but was greater than that in Spain and most other European countries (Figure 4).\textsuperscript{10}

The level of antimicrobial consumption in New Zealand in recent years is most comparable with those European countries that are widely considered to have profligate levels of antimicrobial consumption, and that in consequence have high levels of antimicrobial resistance.
Programs to reduce antibiotic consumption in New Zealand are urgently needed

New Zealand needs to promptly institute a range of effective measures to reduce antimicrobial consumption. In the absence of such measures the prevalence of resistance in common pathogens will rise quickly and we will suffer the consequences outlined above.

Since 1999 New Zealand’s Pharmaceutical Management Agency (PHARMAC) has funded and managed the Wise use of antibiotics campaign that was associated with a dramatic decline in prescriptions for amoxycillin plus clavulanate during 1999 and 2000,14 and that probably also contributed to the slower rate of growth in overall consumption since 2009 (Figure 3).

The slower rate of growth of antimicrobial consumption in recent years may also be a result of increased prescriber compliance with the guidance provided by the Best Practice Advocacy Centre (BPAC)15 and attention by prescribers to the feedback from BPAC about their level of antimicrobial prescribing. However, despite these efforts to encourage prudent antimicrobial prescribing in New Zealand, the per capita consumption of antimicrobials has grown, rather than stayed level or declined.

Sustained reductions in antimicrobial consumption in New Zealand in the coming years will require recognition that this is a goal with major long-term benefits. Reducing the level of antimicrobial consumption is analogous to increasing the level of infant immunisation: effort expended now is more than repaid in the future.
In 2009, the Ministry of Health recognised the potential benefits of setting targets for the uptake of childhood immunisations, and regularly reporting the performance of DHBs in relation to these immunisation targets.\(^{16}\)

Subsequently there has been a significant improvement in immunisation uptake in all population groups throughout New Zealand. In a similar manner the Ministry of Health now should set targets for reductions in the per capita antibiotic consumption and regularly report the performance of DHBs in relation to these targets.

Figure 5 shows that the annual per capita consumption of antibiotics by community-based patients during 2012, (measured in DDDs/1000 population/day) varied between 18.93 in the West Coast DHB, and 28.13 in Counties Manukau DHB.

A number of factors, such as the proportions of the population comprised by children, and by people experiencing socioeconomic deprivation, will cause some variation between DHBs in the prevalence of infections that require antibiotic treatment.

Therefore it is not realistic to expect that the per capita level of antimicrobial consumption will be equivalent for all DHBs. However, it is inevitable that those DHBs with higher levels of antimicrobial consumption will experience more rapid growth in antimicrobial resistance than those DHBs with lower levels of consumption.

As a result, in the near future, DHBs with consistently high levels of antimicrobial consumption will more commonly confront the problem of patients with infections due to resistant organisms. Therefore, it should be a goal for all DHBs to reduce unnecessary antimicrobial consumption and slow the emergence of antimicrobial resistance within their population.

**Figure 5. Annual per capita consumption of antimicrobials by community-based patients, in the 20 New Zealand District Health Boards, during 2012, measured in DDDs/1000 population/day**

![Graph showing annual per capita consumption of antimicrobials by community-based patients in different DHBs during 2012.](https://example.com/graph.png)
Strategies to reduce antimicrobial consumption in New Zealand

In developed countries, a large proportion of antimicrobial treatment, often more than 50%, is prescribed for patients with conditions in which antimicrobial treatment makes no significant impact on mortality or morbidity. For example, despite widespread understanding that antimicrobials provide no benefit for patients with viral upper respiratory tract infections, it is disappointingly common practice in New Zealand for patients presenting with symptoms suggestive of such illnesses to be prescribed an antimicrobial.\(^{17,18}\) Other conditions for which antimicrobial treatment is commonly prescribed, with nil or negligible benefit, include otitis media, boils and most diarrhoeal illnesses.\(^{15}\)

Ideally treatment of infections should be as narrow spectrum as possible. For example, penicillin should be preferred over amoxicillin, cephalaxin or amoxicillin/clavulanate in the treatment of *Streptococcus pyogenes* or *S. pneumoniae* infection, and flucloxacillin should be preferred over cephalaxin or amoxicillin/clavulanate in the treatment of *Staphylococcus aureus* infection.

Antimicrobial treatment should be prescribed for the shortest duration necessary to achieve a significant impact on mortality or morbidity, and not prolonged unnecessarily.

Prescribers may be reluctant to select the most narrow spectrum antimicrobial agent for the treatment of infections, because other, more broad spectrum, agents may have advantages with respect to the palatability of the medicine or the convenience of the dosing regimen. For example, amoxicillin may be preferred over penicillin because there is no requirement for the medicine to be taken well away from meals, or cephalaxin may be preferred over flucloxacillin because it can be taken twice, rather than four times, daily.

The overall benefits and disadvantages of the various antimicrobials that might be used for the treatment of an infection should be carefully considered. The contribution of broad spectrum agents to speeding the emergence of antimicrobial resistance should not be ignored, or considered trivial in comparison to other considerations such as palatability or convenience.

Narrow-spectrum penicillins such as penicillin V and flucloxacillin are under-prescribed in New Zealand when compared with other more prudent countries. During 2012, consumption of narrow spectrum penicillins comprised only 21% (2.73/13.22 DDDs/1000 population/day) of total consumption of penicillins by community-based patients in New Zealand.

In contrast, during 2012, consumption of narrow spectrum penicillins comprised 57% and 77% of total consumption of penicillins by community-based patients in Denmark and Sweden.\(^{10}\) In these countries a much greater emphasis is placed on the use of narrow spectrum antimicrobials to slow the emergence of resistance.

Figure 6 shows that in New Zealand, as in other countries, the level of antimicrobial consumption is highest in children aged less than 5 years and in adults over the age of 80 years. However there was no age group that was dispensed less than 60 antibiotic
prescriptions per 100 population. This is consistent with a recent study of antibiotic consumption in Tairawhiti which found that, during a 12 month period in 2005 to 2006, at least one antibiotic prescription was dispensed to 60% of males and females aged 0–15 years, 33% of males and 48% of females aged 35–44 years, and 65% of males and 70% of females aged ≥85 years.¹⁹

These data suggest that strategies to reduce antimicrobial consumption in New Zealand should not be focused exclusively on paediatric prescribing but rather should be designed to influence prescribing of antibiotics across all age groups.

**Figure 6.** Per capita consumption of antimicrobials by community-based patients, in relation to patient age, during 2012, measured in prescriptions/100 population/year.

![Graph showing per capita consumption of antimicrobials by community-based patients, in relation to patient age, during 2012, measured in prescriptions/100 population/year.](image)

**Note:** Data for the number of antimicrobials dispensed by community pharmacies during 2012, for patients within each age range, was obtained from the National Pharmaceutical Collection;¹² and for the number of people within each age range resident in New Zealand during 2012 was obtained from Statistics New Zealand.¹³

**Programs that encourage reduced antimicrobial consumption are effective**

Various measures to reduce antimicrobial consumption, and thus slow the increase in the prevalence of resistance, have been used overseas.

In 2002, France conducted a nationwide campaign *Antibiotics are not automatic* that over the subsequent 5 years resulted in a greater than 25% per capita reduction in antibiotic prescriptions. ²⁰

In 2010, the Swedish Government set a target to reduce total antimicrobial consumption by 36% over 4 years, from 39 antibiotic prescriptions per 100 population per year, to less than 25 antibiotic prescriptions per 100 population per year.²¹
Systematic reviews suggest that a variety of educational strategies targeted at the prescribing doctor and/or their patients can result in an average 25% reduction in the proportion of patients who are prescribed an antibiotic. There is little evidence that one strategy is more effective than others, however “passive” education of prescribers appears to have relatively little effect and encouraging use of “delayed dispensing” appears to have a relatively large effect.

**Patients should be educated about the adverse effects of antibiotic consumption**

Patients commonly appreciate the dramatic benefits of antibiotic treatment of serious bacterial infections, but commonly are unaware of the potential harms of antimicrobial consumption. As a result patients may unrealistically exaggerate the benefits and neglect the disadvantages of antibiotic treatment for a wide range of relatively minor illnesses.

One disadvantage of an unnecessary antimicrobial treatment that patients may neglect is the increased risk that future infections will be due to an antimicrobial resistant bacterium. Antibiotic treatment significantly increases the risk that subsequent infection is due to an antibiotic resistant bacterium.

A meta-analysis of four studies has shown that antibiotic treatment for a urinary tract infection results in a 2.5 times greater risk that a subsequent urinary tract infection in the next three months is due to an antibiotic resistant *E. coli*. Similarly, antibiotic treatment results in an overall 2.4 times greater risk that a respiratory tract infection in the subsequent 12 months is due to an antibiotic resistant *S. pneumoniae*, *Haemophilus influenzae* or *S. pyogenes*.

Antibiotic treatment results in a 3 times greater risk that any staphylococcal disease in the next three months is due to MRSA. Patients deserve to be informed of these adverse effects, especially when seeking advice about a condition in which antimicrobial treatment makes no significant impact on mortality or morbidity.

Other risks of antibiotic use may be less obvious. Even brief courses of antibiotic treatment result in prolonged disruption of the normal flora of the gastrointestinal tract. Such antibiotic caused alterations in the normal intestinal flora may then result in alterations in the person’s metabolic pathways with an increased risk of diabetes and obesity.

Patients need to be educated that interactions with the bacteria present in their mouth and intestines, and on their skin represent their most intimate relationship with the environment. They should be encouraged to appreciate and protect their normal flora and avoid unnecessary use of antibiotics that disrupt it.

**New Zealand’s Ministry of Health should lead efforts to reduce antimicrobial consumption**

The New Zealand Ministry of Health needs to reinvigorate efforts to reduce antimicrobial consumption in New Zealand by setting targets for reductions in antimicrobial consumption and then reporting on progress towards these targets by each DHB.
New Zealand is fortunate in already having excellent data on antibiotic consumption. Regular reporting of this data in relation to targets will encourage health administrators throughout the country to allocate resources to local or national programs that encourage reductions in antibiotic use.

Residents of DHBs that consistently fail to meet targets should be encouraged to press their health administrators for the reasons that they are failing to take the steps necessary to ensure that antibiotic treatments will remain beneficial for their population.

Failure to act now to reduce antibiotic consumption will significantly harm the health of our population in the near future.

Competing interests: Nil.

Author information: Mark Thomas, Associate Professor, Department of Molecular Medicine and Pathology, Faculty of Medical and Health Sciences, University of Auckland; Alesha Smith, Best Practice Advocacy Centre, Dunedin, and Research Fellow, School of Pharmacy, University of Otago, Dunedin; Murray Tilyard, CEO, Best Practice Advocacy Centre, Dunedin, and Professor of General Practice, University of Otago, Dunedin

Correspondence: Dr Mark Thomas, Department of Molecular Medicine and Pathology, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand. Fax: +64 (0)9 3737492. Email: mg.thomas@auckland.ac.nz

References:

Endocrine encephalopathy

King W Yong, Steven Soule, Penny Hunt

Abstract

The diagnosis of Hashimoto’s encephalopathy is made when no other cause is found for an acute encephalopathic illness, in the presence of positive thyroid autoantibodies, and is supported by a response to steroid therapy.

A 59-year-old woman developed an encephalopathic illness with mixed aphasia, global weakness and generalised seizures requiring intubation and ICU admission. Extensive imaging and laboratory investigations looking for an underlying cause for the encephalopathy were unremarkable. Thyroid autoantibodies were strongly positive, raising the possibility of Hashimoto’s encephalopathy. Thyroid function testing showed profound primary hypothyroidism.

The patient was commenced on high-dose methyprednisolone, with prompt cessation of seizure activity. Thyroxine replacement was commenced, with the methyprednisolone switched to oral prednisone and slowly weaned. The patient had no further seizures and ultimately made a full recovery.

Hashimoto’s encephalopathy was first described in 1966 in a patient with seizures, disorientation, alternating hemiparesis and positive thyroid autoantibodies. The pathophysiology was thought to be an autoimmune encephalopathy related to the thyroid autoantibodies.

The diagnosis is made when positive thyroid autoantibodies are found in a patient with an unexplained encephalopathic illness, and is supported by a response to steroid therapy. It is a rare condition with a recent review identifying only 121 reported cases.

Case report

A 59-year-old woman, previously well, was admitted with 3 days of abdominal pain. Ascending and transverse colitis, presumed secondary to recent diclofenac usage, was found on CT abdomen on day 1 and subsequent colonoscopy. Her abdominal symptoms gradually improved, however she became acutely confused and incoherent on day 13. She had mixed aphasia and a generalised reduction in power with normal tone and reflexes. She proceeded to a generalised seizure with subsequent status epilepticus, requiring intubation and ICU admission. Recurrent seizures occurred over the next 6 days, despite treatment with three anticonvulsants (valproate, phenytoin and levetiracetam).

Extensive investigations including CT and MRI brain, CSF analysis for HSV, blood cultures, vasculitic screen and HIV serology were all unremarkable apart from mild elevation in CSF protein at 1.43 g/L (0.15–0.40). Plasma sodium ranged between 133 and 145 mmol/L (135–145) over the acute illness and there were no evidence of hypoglycaemia or hypothermia.
Thyroid autoantibodies were found to be strongly positive on day 20—anti-TPO 707 IU/ml (<10), anti-TG 91 IU/ml (<10), raising the possibility of Hashimoto’s encephalopathy. Thyroid function tests on day 21 showed profound primary hypothyroidism—free T4 6 pmol/L (10–24), TSH 180 mIU/L (0.4–4.0)—and endocrinology was consulted.

The patient was commenced on 1 gram methylprednisolone and 50 mcg thyroxine daily on day 21, with prompt cessation of seizure activity, successful extubation and transfer to a medical ward after 2 days. Methylprednisolone was switched to oral prednisone 40 mg daily after 5 days and thyroxine was increased to 100 mcg daily after 2 weeks.

The patient continued to improve, with no further seizure activity and ongoing recovery in neurological function. She was transferred to a rehabilitation facility 39 days after admission, on a reducing course of prednisone and valproate. She returned home on day 49. Prednisone was ceased after 3 months and valproate discontinued at 1 year. She remains well on thyroxine 100 mcg daily in the community 2 years later.

**Discussion**

Hashimoto’s encephalopathy is a rare diagnosis of exclusion, which usually presents with encephalopathic symptoms and seizures, positive thyroid autoantibodies, and characteristically responds to steroid treatment.

Thyroxine replacement alone was shown to improve symptoms in 8 of 47 patients with subclinical or overt hypothyroidism with Hashimoto’s encephalopathy.\(^2\) However, hypothyroidism alone is unlikely to account for the encephalopathy as not all patients respond to thyroxine therapy and patients have variable thyroid function at presentation.\(^2\) If thyroid dysfunction is present, it should be managed no differently to those patients without an encephalopathic illness.\(^4\)

Myxoedema coma may have a similar neurological presentation, with seizure and status epilepticus usually attributed to severe hyponatraemia.\(^5\) We believe Hashimoto’s encephalopathy is a far more likely diagnosis in our patient, given the absence of hypothermia (a cardinal feature of myxoedema coma),\(^5\) the minimally deranged plasma sodium and the prompt cessation of seizure with glucocorticoid and low dose thyroxine therapy.

We commenced thyroxine at a low-dose initially to reduce the risk of exacerbating occult coronary artery disease in our patient. A loading dose of thyroxine was not administered as the clinical presentation was not consistent with myxoedema coma.

An autoimmune pathogenesis for Hashimoto’s encephalopathy is supported by the finding of peri-arteriolar lymphocytic infiltration at brain biopsy,\(^6\) the identification of autoantibodies against the enzyme \(\alpha\)-enolase\(^7\) and the steroid responsiveness of the condition.

No CNS antigenic locus has been found for the thyroid autoantibodies and there is no correlation between the symptom severity and the titre of thyroid autoantibodies.\(^5\) The thyroid autoantibodies therefore are thought to be markers of autoimmunity\(^2,4\) and steroid-responsive encephalopathy associated with antibodies to thyroperoxidase (SREAT) is a designation used by some authors for this condition.\(^8\)
In summary, Hashimoto’s encephalopathy is an unusual condition which should be considered in the encephalopathic patient when more common metabolic, infective and toxic aetiologies are excluded. The disorder is associated with positive thyroid antibodies, variable thyroid function and is typically responsive to parenteral high dose glucocorticoid therapy.

**Author information:** King W Yong, Endocrine Registrar; Steven Soule, Endocrinologist; Penny Hunt, Endocrinologist. Department of Endocrinology, Christchurch Hospital, Canterbury District Health Board, Christchurch

**Correspondence:** King W Yong, RMO Unit, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand. Fax +64 (0)3 3641473; email: kingwei.yong@gmail.com

**References:**

A benign glossal lesion

Tarun Gupta, Naveen Kumar Kansal

Case—A 7-year-old boy presented with an asymptomatic red patch on tongue for last 1 year. As observed and stated by his anxious parents, the patch was ‘moving’ on the different areas of the tongue.

On examination, a circumscribed area of erythema was noted on left lateral border of the tongue with a distinct erythematous medial margin (Figure 1). The rest of the physical examination, including oral hygiene, was within normal limits.

Figure 1. Erythematous lesion on the left lateral border of the tongue

What is your diagnosis?
Diagnosis—**Geographic tongue.** The patient’s parents were counselled about the benign nature of the condition. As the lesion was asymptomatic, no active treatment was prescribed.

Discussion—Geographic tongue, also known as benign migratory glossitis, lingual erythema migrans, or geographic stomatitis is defined as a benign condition of the tongue with a ‘map’-like area(s) of erythema, which is not constant in its shape, size, or location.

It is a relatively common condition, affecting about 1–2% of healthy population.\(^1\) The condition may sometimes cause a sore tongue but is usually asymptomatic, as in our patient. The map-like areas can also affect other mucosal sites, e.g. lips or palate.\(^3\)

The diagnosis of this condition is based essentially upon typical history and presentation. However similar erythematous areas in the oral cavity may also represent erythroplasia, malignancy (e.g. squamous cell carcinoma, Kaposi’s sarcoma), candidiasis, lichen planus, lupus erythematosus or rarely hereditary mucoepithelial dysplasia;\(^1\) as some of these diseases have more guarded prognoses, geographic tongue should be clearly distinguished.

Treatment is neither required nor effective, however topical retinoic acid solution had been used for geographic tongue.\(^4\) For patients with soreness, benzydamine hydrochloride (0.15% spray or mouthwash) may provide symptomatic relief.

Patients need to be adequately counselled about the benign nature of condition to allay anxiety and to minimise unnecessary referrals.

---

**Learning points**

1. Geographic tongue: an asymptomatic, erythematous ‘map’-like lesion, with recurrences revealing changing, migratory, geographic patterns
2. Idiopathic; some cases associated with psoriasis, type 1 diabetes, atopy, reactive arthritis, lithium therapy and HIV infection
3. Diagnosis essentially clinical; patients may need to be properly counselled so as to allay undue concerns about ‘oral cancers’

---

**Author information:** Tarun Gupta, Consultant Dermatologist, Greater Noida, India; Naveen Kumar Kansal, Assistant Professor, Department of Dermatology and Venereology, Gian Sagar Medical College and Hospital, Ram Nagar, Patiala, India

**Correspondence:** Naveen Kumar Kansal, Department of Dermatology and Venereology, Gian Sagar Medical College and Hospital, Ram Nagar, Patiala–140601, India. Email: kansalnaveen@gmail.com

**References:**

The leprosy problem in the South Pacific

I am a New Zealander living in London. From 1966 to 1968, I was a medical officer, specialising in leprosy, in the northern region of Nigeria. Patients were treated here in villages by auxiliaries, using weekly doses of dapsone. In 1967, I carried out a population survey and found that the prevalence of leprosy had declined from 67 per 1000 to 2 per 1000 after 15 years of dapsone monotherapy.\(^{1}\) This information, together with other evidence, indicating that the disease can be eradicated, has recently been published.\(^{2}\)

Despite this optimism, leprosy remains a serious problem in the South Pacific. Recent data from the World Health Organization (WHO) shows that there have been 94 new cases in Kiribati, including 21 children.\(^{3}\) The incidence of childhood leprosy is very significant, as an indicator of ongoing transmission. The percentage of 22%, next to Micronesia and the Marshall Islands, is the highest in the world.

In contrast, in Vietnam, in a population of some 80 million and after a horrific civil war there were only 10 new children with the disease. It appears that the Pacific Leprosy Foundation continues to focus on managing patients after they have become crippled rather than providing early treatment with multidrug therapy, which would prevent the spread of the disease.

I produced maps of the area in Kaduna Province (Nigeria) where domiciliary treatment could be obtained.\(^{4}\) No such maps appear to be available in Kiribati, or where the focus of infection remains. Mobile clinics could be introduced through nautical means of transport. The successful decline in Nigeria proves that field workers should be only employed to treat leprosy and not ‘integrated’ with other diseases. This would stop workers being diverted to treat tuberculosis.

It appears that some workers have not been trained to recognise leprosy, although the ‘diagnosis and treatment is easy’ (WHO). It does not need highly qualified people to work in leprosy. Any school leaver can be trained. In fact, the best leprosy auxiliary I worked with in Nigeria could barely read or write, but he was always on time for the weekly administration of dapsone and knew all the patients. There should be a good response to treatment provided that the patients are not segregated.

Other countries listed in the Foundation’s website are Tonga, Fiji, Western Samoa and Vanuatu. In *The Weekly Epidemiological Record* (a WHO publication),\(^{5}\) there were three new cases in Fiji with no children; Samoa had eight with one child, but there were no returns for Vanuatu and Tonga. Surely it is the Foundation’s responsibility to ensure that all new cases are recorded, especially in children. In Tonga, there are apparently no new infections, but this has to be confirmed by examining the contacts of new child and multibacillary cases.

As there is now a Centre of International Health in Dunedin, I would suggest a collaboration with the Pacific Leprosy Foundation, especially as professionals are conducting surveys for tuberculosis.
A recent publication entitled *A strategy to halt leprosy transmission*,\(^4\) reinforces these points. For example, ‘Few countries now have a surveillance-response system that could provide the epidemiological data to map high-risk areas for leprosy, to monitor the changing epidemiological pattern of the disease and to implement the required interventions’ and ‘School surveys, too, might provide clues: the finding of school-age children with leprosy is a strong indicator of ongoing transmission’.

This approach should also be adopted in Micronesia and the Marshall Islands where the Pacific Foundation has recently taken over responsibility for leprosy. The high prevalence of leprosy has been recognised since 1971, but little appears to have been done here to prevent the spread of the disease. The Pacific Leprosy Foundation has been given an award which could be put to good use and I am sure that the New Zealand public would donate generously.

It has been claimed that the incubation period for leprosy is very long; at least 4–6 years and sometimes longer, but there is no evidence for this. Instead the decline in the Karamui study suggests that it is quite short. The authors of the *Lancet* publication write ‘A serious obstacle, however, to gaining the full potential of contact tracing is the absence of a diagnostic test for early-stage or sub-clinical infection in contacts’.\(^4\)

As you can see from the details in the chapter,\(^2\) we have shown that it is possible to reproduce the features of tuberculoid leprosy as a result of an autoimmune response to an antigen in peripheral nerve rather than a direct response to *Mycobacterium leprae*. A specific positive skin test will produce an epithelioid cell granuloma, thus reproducing the pathology of this form of the disease. This will define the incubation period and determine whether there is a subclinical infection. It will also determine whether transmission has ceased in a previously endemic area.

Money for research projects is available from the leprosy charities at info@leprosyresearch.org – I would strongly encourage New Zealand neuroscientists to apply for a grant to isolate the non-myelin antigen involved. Details of the procedure are available.\(^5,6\)

Nerve damage is the main reason why leprosy is a serious disease. I have emphasised that patients with non-lepromatous leprosy may develop acute sensory loss in all four limbs.

On the basis of this clinical finding, rabbits were injected with a homogenate of human sensory peripheral nerve plus adjuvant and electrophysiological recordings were taken from the hind limb by Jim Pascoe at University College London. There was a specific diminution of C fibre action potentials with preservation of A delta fibres.\(^7\)

This is also a good model to study pain mechanisms and diabetic neuropathy as well as leprosy, so physiologists should also consider applying for a grant to continue this work.

For any further information please contact me at clcraw66@outlook.com

**Colin Crawford**

London, UK
References:


Chewing the saturated fat: should we or shouldn’t we?

We respond to an NZMJ editorial by Te Morenga and colleagues about the issue of saturated fat. The authors call for quiet, after the link between saturated fat intake and cardiovascular disease (CVD) was questioned.

The editorial authors attack the validity of a recent meta-analysis by Chowdhury of cohort and trial studies which showed no significant association between saturated fat intake, or biomarkers thereof, and CVD. Instead, the editorial authors assert that saturated fat causes CVD, with support from another meta-analysis of cohort studies by Jakobsen which contains individual participant data.

The editorial raises a number of issues. When should statistical evidence that negates a hypothesis be believed, and the conviction overturned? If there are conflicting meta-analyses evaluating the evidence for a hypothesis, which should be selected? Clearly, with saturated fat and its influence on CVD, it is possible to prefer a summary study which supports one’s point of view. Less subjectively, the quality of one study over another may be ranked.

Bradford-Hill’s causal criteria are useful to apply to the evidence in question. Briefly, these principles suggest that an association is more likely to be causal if there is consistent evidence from different studies, the association is strong, a dose-response association is evident, and experimental randomised trial data supports the hypothesis. Temporality, analogy and biological plausibility are other considerations.

We believe the hypothesis that saturated fat causes cardiovascular disease fails at the first criterion. Summaries of the experimental evidence do not show a consistent association between saturated fat restriction (or substitution) and mortality endpoints.

If saturated fat is the strongest dietary factor that causes CVD, it would be expected that replacement with other types of fat would lead to reduced incidence. The Jakobsen study does not show consistent evidence of benefit from saturated fat avoidance. Rather, only one of the subgroup analyses returns a positive association. Similarly, if saturated fat reduces CVD without adverse effects on other outcomes, we would expect overall mortality to be reduced.

Death is measured with less error than any other disease-specific outcomes. Focus on overall mortality avoids the risk of concluding that an intervention improves one endpoint, but, in reality, is offset by harm to another. For example, a treatment may reduce CVD but increase cancer incidence, so that the effect on overall mortality is neutral. This is possible in the Jakobsen study, since only CVD endpoints are reported.

A number of meta-analyses now support the findings of Chowdhury, showing little backing for the idea that substituting saturated fat with other types reduces CVD. A Cochrane review of randomised studies, designed to test the hypothesis that saturated fat influences CVD, showed no association between treatment arm and overall mortality (pooled relative risk 0.98, 95%CI: 0.93–1.04, 71,790 participants, 4292 deaths).
With the high number of participants and deaths reported, a large effect of the intervention is unlikely to be missed. The funnel plot for this analysis showed some evidence of publication bias. That is that small studies which showed harm from saturated fat replacement were unlikely to be published. The reported pooled effect is, therefore, likely to overestimate the benefit of avoiding saturated fat.

So, we conclude, that randomised trial data, which is superior to the observational evidence offered by Jakobsen, does not support either limiting or altering saturated fat intake to improve survival. We also consider that this Cochrane review is less likely to be biased than the surrogate endpoint (low density lipoprotein cholesterol) and ecological studies referred to by the editorial authors. In an editorial that claims to present “the totality of the evidence”, we find this omission striking.

The editorial authors argue that the Jakobsen study should be preferred over that by Chowdhury, even though the latter includes randomised studies. Experimental trials are generally considered less biased than those which observe cohorts, due to the randomisation which balances confounders between the treated and control arms.

Other studies support the lack of statistical association between altering saturated fat intake, both from randomised and observational designs. One comparative meta-analysis ranks the statistical link between saturated fat and CVD amongst the poorest of a range of dietary factors.

We ask ourselves, “How much more evidence is needed before saturated-fat-based interventions are abandoned?” Popper stated that the hallmark of the scientific method is that a hypothesis is possible to falsify, should it lack supporting evidence.

In the absence of a strong indication of harm, we believe the public should be left to chew the saturated fat, and concern themselves with avoiding dietary factors which consistently cause ill health.

**Competing interests and funding:** Nil.

---

**Simon Thornley**  
Public Health Physician  
Section of Epidemiology and Biostatistics  
The University of Auckland  
[Email](mailto:s.thornley@auckland.ac.nz)

**George Henderson**  
Research Associate  
Auckland University of Technology, The Human Potential Centre  
Auckland

**Grant Schofield**  
Professor of Public Health  
Auckland University of Technology, The Human Potential Centre  
Auckland
References:
Performance of funded point-of-care capillary blood glucose meters at altitude

In March 2013 the choice of point-of-care blood glucose meter (and requisite test strips) subsidised by PHARMAC was restricted to those provided by a single manufacturer for the majority of individuals with diabetes in New Zealand. Systematic assessment showed that these new meters met international standards with respect to accuracy and precision, but many users reported apparent differences between blood glucose measurements made with old and new meters.

Formal comparison between the new meters and one particular meter commonly used prior to March 2013 revealed that there was on average a 0.6 mmol/l difference between meter readings (with the new meter giving the higher reading) when compared with laboratory standard samples. Although this difference between meters is not of clinical relevance, considerable concern remains from both consumers and clinicians regarding reliability of the new meters.

The performance of point-of-care blood glucose meters at altitude has previously been examined. The methods used by each glucose meter (glucose oxidase—or glucose dehydrogenase-based systems) is influenced to a varying degree by altitude and other environmental variables, but in an inconsistent manner to be able to predict either direction (over- or under-estimation) or magnitude of effect.

Although altitude leads to significant changes in insulin sensitivity, type 1 diabetes mellitus (T1DM) per se should not preclude travel to altitude. Recent advances that facilitate optimized glycaemic self-management in T1DM such as insulin pumps and continuous glucose monitoring (CGMS) have led to an increasing number of individuals with T1DM travelling to altitude, often in remote locations and to participate in strenuous activities such as snowsports, climbing and mountaineering. In this setting, an under-reading meter could lead the individual to believe blood glucose was in the target range when the true value would be hyperglycaemic, potentially resulting in impaired mental and physical functioning and reduced insulin dosing.

Of more pressing concern would be an over-reading meter than could mask a hypoglycaemic reading and risk the incipient dangers of hypoglycaemia. It is therefore essential that any such individual with T1DM understands the potential interaction between altitude and the accuracy and precision of their blood glucose meter to ensure personal safety and effective diabetes self-management.

Fasting and non-fasting capillary blood glucose (CBG) samples were obtained at sea level and at an elevation of 3441m from a healthy (non-diabetic) 39-year-old male (no medication, HbA1c 33mmol/mol). Each sample was simultaneously analysed on old (Optium Xceed and Accuchek Performa, glucose dehydrogenase-based method) and new (Caresens N, Caresens N Pop, glucose oxidase-based method) point-of-care blood glucose meters within the manufacturers’ recommended operating temperature range.
The results reveal that although there is no apparent difference in the measured glucose readings between the new or old meters at sea level; the new meters give higher readings than the old meters for both fasting and non-fasting glucose; and that the difference is both statistically significant and likely to influence insulin dose decisions (Figure 1).

**Figure 1.** Mean capillary blood glucose [CBG] (fasting and random) measured at sea level (1A and 1B) or at altitude (1C and 1D) using the meter indicated

Note: Error bars indicate ± 1 standard deviation. Results indicate no difference between sea level measurements, however the Caresens N and Caresens N Pop meters give significantly higher readings at altitude when compared to the Optium Xceed and Accu-chek Performa meter. (1A n=4, 1B n=17, 1C n=3 and 1D n=10).
Miller CBG at altitude: supplemental data

Random CBG at sea level

<table>
<thead>
<tr>
<th>Meter</th>
<th>Caresens N</th>
<th>Caresens N Pop</th>
<th>Optium Xceed</th>
<th>Accu-chek Performa</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>6</td>
<td>5.8</td>
<td>5.9</td>
<td></td>
</tr>
<tr>
<td>5.5</td>
<td>5.3</td>
<td>5.3</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>5.7</td>
<td>6</td>
<td>5.7</td>
<td>6.4</td>
<td></td>
</tr>
<tr>
<td>6.5</td>
<td>6.6</td>
<td>6.9</td>
<td>6.3</td>
<td></td>
</tr>
<tr>
<td>6.9</td>
<td>6.8</td>
<td>7.5</td>
<td>6.4</td>
<td></td>
</tr>
<tr>
<td>6.7</td>
<td>7.1</td>
<td>6.9</td>
<td>6.6</td>
<td></td>
</tr>
<tr>
<td>5.6</td>
<td>5.4</td>
<td>5.7</td>
<td>5.8</td>
<td></td>
</tr>
<tr>
<td>5.6</td>
<td>5.6</td>
<td>5.2</td>
<td>5.7</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>5.6</td>
<td>5.9</td>
<td>5.9</td>
<td></td>
</tr>
<tr>
<td>8.4</td>
<td>8.5</td>
<td>8.7</td>
<td>7.8</td>
<td></td>
</tr>
<tr>
<td>5.9</td>
<td>6</td>
<td>6.8</td>
<td>5.7</td>
<td></td>
</tr>
<tr>
<td>5.3</td>
<td>5.5</td>
<td>5.9</td>
<td>5.7</td>
<td></td>
</tr>
<tr>
<td>5.5</td>
<td>5.1</td>
<td>5.2</td>
<td>6.1</td>
<td></td>
</tr>
<tr>
<td>5.5</td>
<td>5.1</td>
<td>5.6</td>
<td>5.8</td>
<td></td>
</tr>
<tr>
<td>5.3</td>
<td>5</td>
<td>5.6</td>
<td>5.3</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>5.9</td>
<td>6.2</td>
<td></td>
</tr>
<tr>
<td>5.3</td>
<td>5.3</td>
<td>5.3</td>
<td>5.1</td>
<td></td>
</tr>
</tbody>
</table>

Mean: 6 5.9 6.1 6
Standard deviation: 0.79 0.90 0.95 0.65

Random CBG at altitude

<table>
<thead>
<tr>
<th>Meter</th>
<th>Caresens N</th>
<th>Caresens N Pop</th>
<th>Optium Xceed</th>
<th>Accu-chek Performa</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.8</td>
<td>6.7</td>
<td>5.6</td>
<td>4.7</td>
<td></td>
</tr>
<tr>
<td>6.7</td>
<td>6.7</td>
<td>5.7</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>7.3</td>
<td>7.1</td>
<td>5.7</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7.2</td>
<td>7.2</td>
<td>5.7</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>5.7</td>
<td>5.4</td>
<td>5.1</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>10.3</td>
<td>9.8</td>
<td>8.6</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>6.1</td>
<td>6.1</td>
<td>4.8</td>
<td>4.7</td>
<td></td>
</tr>
<tr>
<td>9.1</td>
<td>8.7</td>
<td>8.1</td>
<td>6.3</td>
<td></td>
</tr>
<tr>
<td>6.1</td>
<td>6.8</td>
<td>6.1</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>8.7</td>
<td>9.2</td>
<td>7.6</td>
<td>6.8</td>
<td></td>
</tr>
</tbody>
</table>

Mean: 7.4 7.4 6.3 5.6
Standard deviation: 1.50 1.41 1.31 0.95
Fasting CBG at sea level

<table>
<thead>
<tr>
<th>Meter</th>
<th>Caresens N</th>
<th>Caresens N Pop</th>
<th>Optium Xceed</th>
<th>Accu-chek Performa</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5.3</td>
<td>5.5</td>
<td>5.9</td>
<td>5.7</td>
</tr>
<tr>
<td></td>
<td>5.6</td>
<td>5.4</td>
<td>5.7</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>5.3</td>
<td>5</td>
<td>5.6</td>
<td>5.3</td>
</tr>
<tr>
<td></td>
<td>5.3</td>
<td>5.3</td>
<td>5.3</td>
<td>5.1</td>
</tr>
<tr>
<td>Mean</td>
<td>5.4</td>
<td>5.3</td>
<td>5.6</td>
<td>5.5</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.15</td>
<td>0.22</td>
<td>0.25</td>
<td>0.33</td>
</tr>
</tbody>
</table>

Fasting CBG at altitude

<table>
<thead>
<tr>
<th>Meter</th>
<th>Caresens N</th>
<th>Caresens N Pop</th>
<th>Optium Xceed</th>
<th>Accu-chek Performa</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6.8</td>
<td>6.2</td>
<td>5.5</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td>6.7</td>
<td>7.8</td>
<td>4.3</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>6.5</td>
<td>6.1</td>
<td>5.8</td>
<td>4.3</td>
</tr>
<tr>
<td>Mean</td>
<td>6.7</td>
<td>6.7</td>
<td>5.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.15</td>
<td>0.95</td>
<td>0.79</td>
<td>0.15</td>
</tr>
</tbody>
</table>

There are clear limitations of the pilot study data described. The small number of samples originate from a single individual without diabetes, and there are no laboratory standard controls. Further study would be required to confirm these findings and to explore the characteristics of the effect (threshold, linearity etc.). However, when considered alongside previous studies these data suggest that whilst the tendency for the new Caresens point-of-care meters to over-read capillary blood glucose levels is small at sea level, the effect is exaggerated with altitude.

The resulting apparent over-reading of glucose has potential to increase risk of hypoglycaemia in the austere environment, where consequences of hypoglycaemia would be greater.

Although most New Zealanders live at relatively low altitude (<1000m above sea level) there are a number of locations (mountain peaks, ski resorts, Great Walks [e.g. Routeburn Track]) on both North and South Islands at elevations approaching that of the present study to which people with diabetes are attracted for recreation.

Accordingly, consideration should be given to providing an alternative capillary blood glucose meter to any individual with diabetes who plans to test blood glucose at altitude. Alternatively, clinicians should advise such patients to expect higher than usual blood glucose levels, and adjust glucose targets appropriately.

Steven C M Miller  
Specialist in Endocrinology and Diabetes, and General Internal Medicine  
North Shore Hospital  
Auckland, New Zealand
References:


Case of hydroa aestivale


Leslie P., aged 8 years, came to Dunedin Hospital (out patients) complaining of the occurrence, of a rash which appeared from time to time on the exposed parts of his body, ears, face, and hands. This eruption occurred more frequently in summer. It had the appearance of crops of vaccinia. Different stages of development of the eruption were present.

Scars like old vaccination scars showed where previous eruptions had occurred. In December last year the boy ran about bare-legged with the result that the condition shown in the photograph developed.

He was admitted 12th December with a temperature of 103.8 F. Many of the vesicles were purulent, and the surrounding tissues were acutely inflamed. Fomentations rapidly reduced the inflammation. He was discharged on the 23rd December relieved. He was advised to wear a broad-brimmed hat and to avoid exposing himself to the hot sun without protection.
6th February, 1913—

His face and ears have since kept well. Previously there was almost constantly some eruption in his ears. He puts a newspaper over himself when he is in the sun. He has now only a few small vesicles on his hands. The disease is described in Norman Walker's book. Cultures from purulent but unruptured vesicles were sterile.

For this information I am indebted to Dr. Champtaloup. This is the second case of this disease which I have seen. In both instances the disease is said by the parents to date from vaccination.
Risk of a thrombotic event after the 6-week postpartum period

Pregnancy significantly increases the risk of thrombosis. This heightened thrombotic risk rises further during the postpartum period, which is conventionally defined as the 6 weeks after delivery. Over these 6 weeks there is a significant increase in the risk of venous thromboembolism, stroke and myocardial infarction as compared with non-pregnant women. This study considers whether this risk period may be longer than 6 weeks.

Data from women hospitalised for labour and delivery over a 5½ year period in hospitals in California was reviewed. Among the 1.687,930 women with a first recorded delivery, 1015 had a thrombotic event (248 cases of stroke, 47 cases of myocardial infarction, and 720 cases of venous thromboembolism) in the period of 1 year plus up to 24 weeks after delivery.

The researchers confirmed the heightened 6 week risk and noted a modest but significant increase in risk during the 7–12 week period compared with the same period 1 year later. However, the absolute increase in risk beyond 6 weeks was low.


Off-hour presentation and outcomes in patients with acute myocardial infarction

This meta-analysis from the Mayo Clinic reviews whether patients with acute myocardial infarction presenting to hospital during off-hours (weekends and nights) have higher mortality than those presenting during regular hours, and do patients with ST elevation myocardial infarction (STEMI) have longer door to balloon time during off-hours than in regular hours?

The meta-analysis included 48 cohort studies with fair quality enrolling 1,896,859 patients. The results were that there was a 5% relative increase in mortality in hospital and at 30 days in those presenting in off hours. There was a delay of nearly 15 minutes in door to balloon time in this cohort.

The authors note that the difference in mortality between off-hours and regular hours may be confounded by patients’ clinical characteristics. High heterogeneity reduces the validity of the study findings, and the pooled effect size of this study should be viewed as an average estimate expected across a range of different settings.

BMJ 213;347:f7393.
Day-patient treatment after short inpatient care versus continued inpatient treatment in adolescents with anorexia nervosa

Guidelines of European countries and the USA consider inpatient treatment (IP) as the treatment of choice for moderately or severely ill adolescent patients with anorexia nervosa or those who have not improved with outpatient treatment. This is costly and relapse and readmission rates are high. Day patient treatment (DP) is less expensive and might avoid problems of relapse and readmission by easing the transition from hospital to home.

This theory is tested in this randomised non-inferiority multicentre German trial. 172 patients were randomly allocated to treatment: 85 to IP and 87 to DP. DP was found to be non-inferior to IP with respect to the primary outcome, an increase in body-mass index at 12 months. Treatment-related serious adverse effects were similar in both groups, 8 in the IP and 7 in the DP groups.

The researchers recommend DP as it is effective, safe and less expensive than IP.

The Autistic Brain


The Autistic Brain is a soft-covered book with an eye-catching display of colour on the front cover with a realistic retail price of $29.99.

It is broken into two main sections with Part 1 looking at the Autistic Brain and Part 2 Rethinking the Autistic Brain. Each part consists of 4 chapters that are well laid out in an easy to read font.

Each chapter is clearly labelled with a bold title making it easy to navigate the book. Part 1 has a few illustrations and Part 2 has more illustrations along with exercises to attempt.

At the end of the book there is a comprehensive notes section and a well set out index, all of which are in black and white.

It is easy to read and therefore will appeal to a wide audience both those working with autistic people, personally or professionally as well as the general public. Importantly it should be taken into account that this is one person’s journey and gives some useful insights and ideas as well as looking at current research.

For people interested in this topic or someone they know who is autistic this will be an interesting read. To know that the author has succeeded with a PhD is a triumph in itself. Grandin is realistic in the book saying that many will not attain this level of success and it is important to recognise each individual’s level of success in itself.

The book is written in the first person and has a mix of personal life stories, research and experiences of other autistic people. The subtitle of the book “Exploring the strength of a different kind of mind” sums up the direction the book takes.

The author sees autism as a strength to be utilised rather than a mental disorder. The prologue sets the tone for this when Grandin say “do not allow a child or adult to become defined by a DSM label” (Diagnostic and Statistical Manual of Mental Disorders). The need to focus on the positive attributes of this unique brain is the key feature of the book.

Part 1 consists of 4 chapters focusing on the autistic brain. In Chapter 1 Grandin looks at the meanings of autism and outlines both the history of autism and the struggles in history to define it. Within this she utilises a series of personal experiences. Chapters 2 and 3 look at neuroimaging (MRI) along with genetics respectively and how research has helped gain an understanding of autism. In Chapter 4 Grandin stresses the importance of understanding the role of the senses and how proper management can help the individual.
A criticism that the author makes is that much of autism research has been from the outside looking in rather than from the autistic persons perspective themselves.

In Part 2 “Rethinking the autistic brain” the focus is on practical ideas for both the autistic person and those living with or looking after them. In Chapter 5 it is noted that family and the medical profession often work using labels to identify the condition. Note is made that it is about looking past the labels particularly those set by the DSM. In Chapter 6 “knowing your own strengths” she identifies that a strength of autistics is their ability to pay greater attention to details, then develops this further as she moves into Chapter 7 where is argues that autistics think in pictures.

Once again these chapters are a mix of Grandin’s experience, those of other autistic people and what current research is finding. The final chapter offers advice for the autistic person who wants to move “from the margins to the mainstream”.

This is an excellent read for anyone. It offers insight and hope for those who are themselves autistic, involved with someone autistic or raising an autistic child. By focussing on their unique contributions rather than their weaknesses autism can be turned into a gift, not a disability.

Liane Dixon
Clinical Studies Research Nurse
Academic Department of Surgery
University of Otago, Christchurch, New Zealand
The Women’s Health Book. A complete guide to health and wellbeing for women of all ages


The Royal Women’s Hospital in Melbourne, Victoria is the largest health facility in Australia specialising in women’s health. The staff at “The Women’s”—many of whom have international reputations for their contribution to research and clinical practice—has put together a comprehensive volume of health advice for women.

This guide is very accessible and well referenced and covers women’s health issues through all life stages. The aim of the writing group is to encourage women to share knowledge about health, educate others about important issues such as sexual violence and to advocate for gender equality in health.

The book is separated into four main areas dealing with adolescent health, young women, the midlife years and later years. There is a strong focus on healthy lifestyle and taking control of your health and, throughout the book, bullet points highlight the key issues. It discusses health screening and provides a clear table of what should be done and when. In doing so, it follows governmental screening advice and manages to steer clear of any controversy, such as that currently surrounding mammography.

A range of topical issues are discussed including body image, mental health, sexuality, contraception, pregnancy, abortion, menopause, use of complementary therapies, gynaecological cancer and being a cancer carer. The book has a chapter on “Getting the most out of your health consultation” which provides clear information and advice to the woman on the sort of checklist they should use before a consultation. It empowers women to ask the right questions and become a partner in their healthcare decisions.

There are some excellent diagrams illustrating the basics of the menstrual cycle and female genitalia. The “Resources” section at the end of the book is a highlight with its comprehensive listing of medical, community and educational services.

Many of these services have an online presence, which allows doctors and women outside Australia to access the information. Several of the websites, such as the
Melbourne-based Jean Hailes organisation and the Australasian Menopause Society, provide excellent resources for women and their doctors.

The book is very much focused on the Australian environment but the information is universal. With the increasing impact of the Internet and self-proclaimed gurus on our patients’ lives and the variable quality of the information they provide, this book provides a much more accurate, evidence-based approach.

The level of the text would suit someone with a good knowledge of English. It is the kind of book that women should have as their healthcare guide and I would encourage GPs, women’s health specialists and healthcare providers with an interest in women’s health to use this book as a clear and concise source of information.

Anna J Fenton
Endocrinologist

Christchurch Women’s Hospital
Christchurch, New Zealand

Women’s Health
Level 2, 21 Caledonian Rd, Edgeware
Christchurch, New Zealand
Migration, Ethnicity, Race and Health in Multicultural Societies (2nd edition)


Bhopal is a Professor of Public Health at the University of Edinburgh and Honorary Consultant of Public Health at NHS Lothian. This is the second edition of this book, with the first being published in 2007. How this edition differs from the first in that the author now adds emphasis on migration and health, as opposed to his previous stance on the concept of ethnicity.

Bhopal has built on international examples that were in the first edition in relation to policy, including World Health Organization initiatives of 2008 and 2010. Bhopal has also generally updated concepts in the first edition, has simplified the language, shortened reference lists and added new material specifically on migration, various groups of migrants, and information of ‘special’ minority groups, i.e. indigenous and Roma (gypsy) populations.

This edition has 10 chapters detailing the concepts of race, ethnicity and migration in the context of health care.

Specific chapters detail health and international migration, terminology and classifications: census and population registers, collecting and interpreting data, historical development of health and services, assessing health needs using quantitative and qualitative data, inequalities, inequities and disparities, priority setting, policy and strategy to improve health, research policy and researching and finishes with theoretical, ethical and future-orientated perspectives on health, migration status, race and ethnicity.

Each chapter has learning objectives, with exercises relating to key concepts and the application of these concepts. Bhopal describes the historical notions of race and ethnicity and how these have shaped current views, specifically in relation to health. He talks about the gathering of ethnic health data, and a framework for the use of this data for improving the health of minority populations. He describes the impact of migration and how important this factor is in considering the provision of health services and public health campaigns.

Bhopal even describes the touchy subject of racism and its continued prevalence in society worldwide. There are general and more specific examples of policy, lessons, and descriptions that relate to each concept in each chapter from different societies and countries. Most of these examples are UK-based, however do include examples from Europe, Africa, the USA and further afield.
Bhopal’s concepts and points of view are well thought out and researched. The concepts and frameworks are well described and easily applied to different settings in health.

This book would be useful for any health professional involved in public health or those wanting a greater understanding of the impact of migration, race and ethnicity in the ever-changing context of health care. It would also be a valuable addition to any health school library or a valuable tool for anyone wishing to gain more understanding on the implication of migration ethnicity and race on health service delivery.

Rebecca Pascoe
Clinical Studies Research Nurse
Academic Department of Surgery
University of Otago Christchurch
Christchurch, New Zealand
The Rise and Fall of National Women’s Hospital. A history


New Zealand’s National Women’s Hospital recently held its 50th anniversary and this therefore is a timely release of Linda Bryder’s latest book. This is not another book about the Cartwright Inquiry nor a simple documentation of personnel and events in the history of National Women’s Hospital.

Childbirth and hence women’s health and care of the newborn is a core aspect of what it is to be human. The social history of our attitudes and behaviour in the care of women is therefore of wide interest. Bryder captures this well in her description of the history of National Women’s Hospital.

In some ways she narrates the transition of the perspective of New Zealand society from well-meaning paternalistic and anglophile to the modern political environment.

Her history, while not exhaustive, is well written and a very interesting read. It appears well researched and in the absence of knowledge to the contrary I am happy to accept the reported facts at face value.

There are a number of aspects of the history that are perhaps under-reported and a number of significant contributions will not be mentioned. Her interpretation of events is unlikely to please all readers and like her previous book on the National Women’s Inquiry will create some debate. The fact that women’s health care is very much alive and well in Auckland is perhaps not reflected in the title and may also cause some concern.

It is a reminder of the enormous goodwill and hard work from New Zealanders and their health providers that have helped create one of the best public health services in the world. Indeed, New Zealand women and the wider medical and political community should look beyond the details, absorb the important lessons of the past, and work together to provide high quality health care for all New Zealand women and their newborn children, while maintaining an international contribution to advancements in medical science. In conclusion an interesting read which I believe many readers beyond those with a direct interest in National Women’s Hospital will find both enjoyable and thought-provoking.

Peter Sykes
Associate Professor and Head of Department
Obstetrics & Gynaecology, University of Otago
Christchurch, New Zealand