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This Issue in the Journal

Using bread as a vehicle to improve the iodine status of New Zealand children
Meredith Rose, Rosie Gordon, Sheila Skeaff

New Zealand children are mildly iodine deficient and this may adversely affect brain development. In order to improve iodine status, the mandatory use of iodised salt in most bread in New Zealand will begin in September 2009. This legislation should improve the iodine status of children who consume these bread products. However, a reduction in the use of salt in bread may jeopardise this initiative and bread producers are encouraged to use iodised salt in as many bread products as possible to maximise improvement to the iodine status of New Zealand children.

Rates of common communicable illnesses in non-anaemic 12–24 month old South Island, New Zealand children
Andrea J Cross, Anne-Louise M Heath, Elaine L Ferguson, Andrew R Gray, Ewa A Szymlek-Gay

This study looked at the rates of communicable illness in 1-year-old South Island children and found respiratory illness to be most common. In addition we found that attending childcare and having preschool-age children was associated with increased rates of illness in this age group. In addition the study showed that teething was associated with increased rates of illness, although the exact mechanism behind this is unknown.

Comparison of the content of the New Zealand influenza pandemic plan with European pandemic plans
Nick Wilson, Michael G Baker

We aimed to critically review version 16 of the New Zealand (NZ) influenza pandemic plan in relation to the content of 29 European pandemic plans. We found that in terms of plan content on border control aspects, the NZ plan scored higher than the average European plan and similarly for the antiviral aspects. However, it scored slightly lower for the vaccine aspects. This comparison process also identified some gaps which could be worth addressing in the planned 2009 version of the NZ plan. In summary, the NZ influenza pandemic plan compared favourably with the average European plan in many aspects but not all.
The epidemiology of cryptosporidiosis in New Zealand, 1997–2006
Saskia J Snel, Michael G Baker, Kamalesh Venugopal

Cryptosporidiosis is a gut infection caused by swallowing a single-celled organism (the protozoan Cryptosporidium parvum or hominis). It is the fourth most commonly notified infectious disease in New Zealand. This study gives a comprehensive description of 10-years of notified and hospitalised cases. The incidence of infection in New Zealand was markedly higher than that reported by other developed countries. The pattern of infection suggests most cases are coming from animals (i.e. zoonoses) which might explain New Zealand’s high rates. Prevention should focus on reducing transmission in rural areas, particularly to children during the calving and lambing season in spring.

The epidemiology of giardiasis in New Zealand, 1997–2006
Saskia J Snel, Michael G Baker, Kamalesh Venugopal

Giardiasis is a gut infection caused by swallowing a single-celled organism (the protozoan Giardia duodenalis). It is the second most commonly notified infectious disease in New Zealand after campylobacteriosis. This study gives a comprehensive description of 10 years of notified and hospitalised cases. The incidence of infection in New Zealand was markedly higher than rates reported for other developed countries. There is no obvious reason for New Zealand’s high rate as the pattern of infection suggests that most cases are coming from human sources. Prevention measures include careful hand washing, particularly after nappy handling, and efforts to avoid contaminated food and drink when travelling.

Has smoking prevalence markedly decreased despite more cigarettes released for sale?
Murray Laugesen

Just before the election, the National Health Survey said smoking prevalence was falling fast at last, and that only 18% of adults smoked daily. However cigarettes released to market actually increased, by 7%. How could this be? The main reason seems to be that smokers are under-reporting their smoking, probably due to the increased social pressures against smoking. But if smoking is only falling slowly, why not faster? Roll-your-own (RYO) smoking is cheap—a cup of coffee is equal in price to 12 RYO cigarettes. This discourages quitting. Also, government is losing $300 million annually by not taxing RYO cigarettes the same per cigarette as factory-made cigarettes.
School is back in New Zealand—and so is the junk food

Jennifer Utter, Robert Scragg, Teuila Percival, Robert Beaglehole

With the Government’s recent decision to remove the healthy food policy for schools, schools are now back to (the junk food) business as usual. The sale of junk food by school canteens is a message to children that it is okay to eat junk food. It will also contribute to the current generation of young people facing a lifetime burden of obesity, diabetes and other chronic diseases because of poor nutrition. This will have huge negative impacts on both the health system and the national economy.

The 2008 clause in the National Administration Guidelines (NAG) requiring schools to make only healthy foods and beverages available at school was a positive step towards ensuring that schools were safe and healthy for children. The healthy food clause meant that New Zealand was leading the way on this issue by providing clear rules on what foods were acceptable and appropriate for children and young people to eat at school.

The healthy food policy had the potential to directly benefit the 56% of children and 62% of adolescents who buy food from school canteens. Our experience of working with schools on obesity prevention over the last 5 years has been that schools only started making major changes towards improving the foods in their school canteens following the enactment of the healthy food clause.

The Minister of Education points out that a remaining clause in the NAG requires schools to promote healthy food and drink to students. Unfortunately this clause is unmeasurable and unenforceable, and unlikely to have any meaningful benefit.

The removal of the healthy food clause means that school food services in New Zealand will continue to contradict nutritional advice and recommendations, just as they did before the clause was introduced.

Surveys of school canteens prior to the 2008 clause found that pies, sausage rolls, chips and crisps, cakes and donuts, and sweet drinks—all energy-dense and nutrient-poor—were common in school canteens. Fewer than half of all schools had fruit on the menu and if healthier options were available, they were more expensive. Given these limited choices, children and adolescents who buy food from school canteens are prevented from making healthy choices.

While it is recognised that schools are under pressure, under-resourced, and under-appreciated for ensuring the education and wellbeing of our children, lowering standards for school foods is not the way to deal with these issues. Food and nutrition impacts heavily on the wellbeing of children and young people and what they eat at school contributes significantly to their overall diet.

Approximately one-third of children and adolescents in New Zealand are overweight or obese. Childhood obesity tracks into adulthood and excess weight is a cause of cardiovascular disease, diabetes, and cancer. It is therefore ironic that shortly after the healthy food clause was repealed, the Minister of Health expressed concern about...
the “tsunami of people coming at us with diabetes” at the opening of a new facility for treating patients with kidney failure.9

When the healthy food clause was announced in 2007, media attention focussed on the few critics of the new clause. Views on individual freedom and personal responsibility contributed to its repeal. We believe that there is no justification for or evidence to support the repeal of the healthy food clause.

**Good nutrition has everything to do with teaching and learning**

According to the former President of the New Zealand Principals’ Federation, Judy Hanna, the healthy food clause was “a compliance issue that has nothing to do with teaching and learning.”10 This opinion is not supported by research evidence. Adequate nutrition is essential for healthy and normal child development and the availability, accessibility, and low-cost of unhealthy processed foods common to New Zealand schools does not support healthy food choices by young people.

Consumption of food additives found in many processed foods marketed to children (such as sodium benzoate and artificial food colours) results in increased hyperactivity in children11,12 and New Zealand research has shown that hyperactivity negatively impacts on student learning.13 Children and young people who experience overweight and obesity are more likely to experience stigmatisation, discrimination, peer-exclusion, and other psychosocial consequences,8 all of which are likely to inhibit their ability to do well at school.

**Good nutrition should have nothing to do with fundraising**

A criticism of the healthy-food policy was that schools could no longer hold sausage sizzles or cake stalls for fundraising.10 Indeed, a 2002 survey of New Zealand schools found that most schools engaged in fundraising activities that often relied on the sale of foods high in fat or sugar.14

Schools may also rely on revenue generated through the school canteen, but requiring a canteen to sell healthy foods does not necessarily mean financial loss for the school. Studies have demonstrated that price reductions on healthy foods in schools increase their sales without any impact on total sales revenue.15,16 Likewise, schools can profit by selling healthy foods at the canteen.17

Funding for schools should not be at the expense of children’s health, just as schools would never be permitted to sell cigarettes or alcohol to increase revenue.

**Healthy food in schools helps students make healthy choices**

The healthy food policy meant that parents could be sure that their children were able to make healthy food choices at school. Children do not always have the maturity and cognitive development to make the healthiest food choices, particularly in a society where they are heavily targeted by the food industry. We cannot expect children and young people to make the healthiest food choices unless we make the healthy choices accessible and cheaper.

By creating school food environments where only healthy foods are available, students have the freedom to make their own personal choices within a range of healthy options. The healthy food clause did not prevent students from bringing junk
food to school from home or buying food on the way home from school; they could exercise their own choices outside of school.

**Conclusion**

The 2008 NAG clause encouraging healthy eating in schools was a positive step towards improving child and adolescent nutrition in New Zealand. Apart from ensuring that schools were becoming healthier places for children and adolescents, the healthy food clause had the potential to greatly benefit vulnerable children who have poorer food intake patterns and more obesity.\(^3,4\)

Unfortunately, the healthy food clause has been removed before its full impact could be realised. It is not too late to reinstate the clause as one part of a comprehensive policy to prevent obesity and diabetes; this would be good value for money and reflect the Government’s concern for the health of New Zealand’s children and young people.

**Competing interests:** None known.

**Note:** Statements or opinions expressed in this editorial reflect the views of the authors and do not necessarily reflect official policy of the New Zealand Medical Association (the Journal's owner).

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What proportion of cancer is due to obesity?

Tony Blakely, Diana Sarfati, Caroline Shaw

The evidence linking overweight and obesity to the incidence of a range of cancers has strengthened over the last couple of years. A recent review of 221 datasets published in The Lancet found that there were associations between increasing body mass index (BMI) and cancers of oesophagus, kidney, thyroid, and colon, as well as leukaemia, multiple myeloma, and non-Hodgkin’s lymphoma. In addition, increasing BMI was associated with rectal cancer and malignant melanoma among men, and cancer of the endometrium, postmenopausal breast, and gall bladder among women. These findings are consistent with, and extend, another recent major review.

Rates of obesity and overweight increased in New Zealand during the 1980s and 1990s, but may have now reached a plateau. The distribution and pattern of BMI varies between population groups (Table 1).

Table 1. Proportionate BMI category distribution, within sex by ethnic groups

<table>
<thead>
<tr>
<th>BMI (kg/m²)</th>
<th>Total</th>
<th>Māori</th>
<th>Pacific</th>
<th>Total</th>
<th>Māori</th>
<th>Pacific</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-overweight*</td>
<td>38.0%</td>
<td>35.8%</td>
<td>18.1%</td>
<td>50.2%</td>
<td>42.2%</td>
<td>17.3%</td>
</tr>
<tr>
<td>Overweight †</td>
<td>42.1%</td>
<td>37.2%</td>
<td>43.9%</td>
<td>27.7%</td>
<td>31.3%</td>
<td>34.8%</td>
</tr>
<tr>
<td>Obese ‡</td>
<td>19.9%</td>
<td>27.0%</td>
<td>38.0%</td>
<td>22.1%</td>
<td>26.5%</td>
<td>47.8%</td>
</tr>
<tr>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Source: 2002/03 Health survey estimates from Tracking the Obesity Epidemic Report.

* BMI <25.0 for European/Other, <26.0 for Māori and Pacific.
† BMI 25.0 to 29.9 for European/Other, 26.0 to 31.9 for Māori and Pacific.
‡ BMI ≥30.0 for European/Other, ≥32.0 for Māori and Pacific.

Men are more likely to be overweight than women, but women are more likely to be obese. Māori and Pacific are more likely to be obese than European/Other, and there is an evolving socioeconomic gradient in BMI distribution (those in lower socioeconomic groups more likely to be overweight or obese) which is stronger for women, but increasingly evident for men.

Similarly, the burden of cancer is not evenly distributed in New Zealand. Compared with non-Māori, Māori are both more likely to be diagnosed with cancer and to die from it. This is true both for all cancers combined, and for many specific cancer sites.

Inequalities in cancer mortality rates between Māori and non-Māori have increased throughout the 1980s and 90s. Pacific people also have higher incidence and mortality, and there is a gradient of increasing incidence of cancer with increasing deprivation. Those in the most deprived quintile have at least a 25% higher rate of cancer incidence than those in the least deprived group.
These three pieces of evidence combine to suggest that increasing BMI currently has an impact on cancer incidence in New Zealand, that this impact will increase over time, and (in addition) it will have an increasing role in driving disparities in cancer incidence between ethnic and socioeconomic groups in the next generations.

In this editorial we synthesise the New Zealand data on obesity and cancer rates with international estimates of the relative risk of cancer by BMI or overweight/obesity category. Our objective is to estimate the contribution of obesity to cancer for the total population, and provide indicative estimates by ethnic group. It must be emphasised that our estimates are limited by the quality of data we have available and the assumptions we are required to make. For example, the relative risk estimates are from overseas studies conducted in the past— it is not guaranteed that they will apply to ethnic groups in New Zealand into the future.

The source reference for these relative risks\(^1\) attempted to rule out confounding by important factors such as smoking, but there may be residual confounding by other lifestyle risk factors that were variously modelled (e.g. physical activity, hormone replacement therapy).

We use the population attributable risk percent (PAR%, also known as population attributable fraction) as an indicative estimate. Our calculations assume complete reversibility of risk, and that everyone moves to a BMI of less than 25 (or less than 26 for Māori or Pacific)—obviously not a realistic policy outcome in the foreseeable future. The PAR% is, therefore, the percentage reduction in the cancer incidence under such a scenario.

Finally, we have used overall population statistic inputs and rates—had we been able to use all of relative risks, percentage obesity, and cancer rates by narrow age groups, and a linear modelling of BMI rather than categorical, the results might differ modestly. These caveats issued, we still believe it is useful to gain an indicative understanding of the possible impact of obesity on cancer to inform policy decisions.

The first column of Table 2 shows the relative risk estimates by cancer site for a 5-unit increase in BMI from Renehan et al.\(^1\) The PAR% are shown by sex, for the total population, and for Māori and Pacific people.

Obesity appears to have a substantial contribution to the incidence of oesophageal, colon (male), gallbladder (female), endometrial, renal, and thyroid cancer with PAR% of 15% or more. This means that if the total population moved to a BMI of less than 25 (or less than 26 for Māori or Pacific) , we might expect the incidence of these cancers to reduce by at least 15%.

For the total population we estimated the PAR% for all cancers combined as 5% for males and 4% for females. For Māori, the PAR% was a little less at 4% for Māori males, due to Māori male cancers being more likely to be cancers that are not weight related, and a little more for Māori females (5%). Any contribution of overweight and obesity to total cancer inequalities between Māori and non-Māori in the future will therefore depend on both overweight and obesity distributions by ethnic group, and the changing ‘background’ incidence and relative shares of cancers that are less related to weight—especially lung cancer.
Table 2. Relative risk of developing cancer for each 5 unit increase in BMI, and PAR% of each cancer due to having a BMI ≥ 25 for European/other or ≥ 26 for Māori and Pacific

<table>
<thead>
<tr>
<th>Cancer type</th>
<th>Male RR *</th>
<th>Male Total PAR%</th>
<th>Māori PAR%</th>
<th>Pacific PAR%</th>
<th>Female RR *</th>
<th>Female Total PAR%</th>
<th>Māori PAR%</th>
<th>Pacific PAR%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oesophageal adenocarcinoma</td>
<td>1.52</td>
<td>32%</td>
<td>35%</td>
<td>42%</td>
<td>1.51</td>
<td>30%</td>
<td>33%</td>
<td>44%</td>
</tr>
<tr>
<td>Colon</td>
<td>1.24</td>
<td>17%</td>
<td>19%</td>
<td>24%</td>
<td>1.09</td>
<td>6%</td>
<td>7%</td>
<td>11%</td>
</tr>
<tr>
<td>Rectal</td>
<td>1.09</td>
<td>7%</td>
<td>8%</td>
<td>10%</td>
<td>1.02</td>
<td>1%</td>
<td>2%</td>
<td>3%</td>
</tr>
<tr>
<td>Gall bladder</td>
<td>1.09</td>
<td>7%</td>
<td>8%</td>
<td>10%</td>
<td>1.59</td>
<td>33%</td>
<td>37%</td>
<td>48%</td>
</tr>
<tr>
<td>Pancreas</td>
<td>1.07</td>
<td>6%</td>
<td>6%</td>
<td>8%</td>
<td>1.12</td>
<td>8%</td>
<td>10%</td>
<td>9%</td>
</tr>
<tr>
<td>Melanoma</td>
<td>1.17</td>
<td>13%</td>
<td>14%</td>
<td>18%</td>
<td>0.96</td>
<td>-3%</td>
<td>-3%</td>
<td>-6%</td>
</tr>
<tr>
<td>Postmenopausal breast</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.12</td>
<td>8%</td>
<td>10%</td>
<td>14%</td>
</tr>
<tr>
<td>Endometrial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.59</td>
<td>33%</td>
<td>37%</td>
<td>48%</td>
</tr>
<tr>
<td>Renal</td>
<td>1.24</td>
<td>17%</td>
<td>19%</td>
<td>24%</td>
<td>1.34</td>
<td>21%</td>
<td>24%</td>
<td>33%</td>
</tr>
<tr>
<td>Thyroid</td>
<td>1.33</td>
<td>23%</td>
<td>25%</td>
<td>30%</td>
<td>1.14</td>
<td>10%</td>
<td>11%</td>
<td>16%</td>
</tr>
<tr>
<td>Multiple myeloma</td>
<td>1.11</td>
<td>8%</td>
<td>9%</td>
<td>12%</td>
<td>1.11</td>
<td>8%</td>
<td>9%</td>
<td>13%</td>
</tr>
<tr>
<td>Non-Hodgkin’s lymphoma</td>
<td>1.06</td>
<td>5%</td>
<td>5%</td>
<td>7%</td>
<td>1.07</td>
<td>5%</td>
<td>6%</td>
<td>9%</td>
</tr>
<tr>
<td>Leukaemia</td>
<td>1.08</td>
<td>6%</td>
<td>7%</td>
<td>9%</td>
<td>1.17</td>
<td>11%</td>
<td>13%</td>
<td>19%</td>
</tr>
<tr>
<td>Weighted ‡ PAR% across cancers</td>
<td></td>
<td>5%</td>
<td>4%</td>
<td>-</td>
<td>4%</td>
<td>5%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease and stroke</td>
<td>1.29</td>
<td>20%</td>
<td>22%</td>
<td>28%</td>
<td>1.29</td>
<td>19%</td>
<td>21%</td>
<td>30%</td>
</tr>
</tbody>
</table>

† Population attributable risk percents, assuming: full risk reversibility as given by the RRs; a counterfactual scenario where everyone had a BMI less than 25 for European/Other or less than 26.0 for Māori and Pacific; that the difference in average BMI between each of the three categories is equal to 5.0, allowing a simple PAR% calculation with the given RRs. This latter assumption is relatively robust, based on distributional data shown in the Tracking the Obesity Epidemic Report (workings available from authors on request).
‡ We used the incidence rates as given in the Unequal Impact Report, Tables 6.2 and 6.3, for each cancer site divided by the total cancer incidence.
# Using a common relative risk estimate of 0.95 for each unit decrease in BMI from the GBD Comparative Risk Assessment project, which translates into a 1.29 relative risk for a five-unit increase in BMI.

We might anticipate that if the distribution of cancers affecting Māori and Pacific proportionately shift to more obesity-related cancers (due to reductions in lung, cervical, stomach, hepatocellular, and other cancers) then the PAR% will increase.

By way of comparison, we also estimated the PAR% of being overweight or obese (compared to a non-overweight BMI) for ischaemic heart disease and ischaemic stroke, using relative risk estimates from the WHO comparative risk assessment project.

The PAR% were 20% and 19% for total males and females, and higher for Māori and Pacific. It must be noted, however, that it is difficult to disentangle BMI from physical activity and dietary factors such as saturated fat, or fruit and vegetables, in the diet as a causal and unconfounded risk factor for cardiovascular disease (CVD). Moreover any progress to the counterfactual (total population with BMI less than 25
or 26) would be likely to involve simultaneous changes in these other associated risk factors.

Such arguments would also apply to cancers, but not to the same extent as for CVD. So while this PAR% for CVD should also be treated as indicative, it does suggest that the impact of overweight or obesity is considerably larger on the incidence of cardiovascular disease than on cancer incidence.

It is biologically plausible that being overweight or obese is a causal factor for some cancers, independent of other risk factors. There are a number of possible mechanisms. First, insulin-like growth factors increase the risk of some cancers. Second, levels of sex steroids that increase cancer risk (e.g. oestradiol) vary with weight. This is likely to be at least part of the explanation for increased risk of breast cancer among obese (postmenopausal) women. Third, adiponectin secreted from visceral fat adipocytes, with blood levels inversely proportional to BMI, are probably antiangiogenic and anti-inflammatory. Fourth, there may be specific mechanical risks associated with weight, for example reflux is a risk factor for oesophageal adenocarcinoma.

It is important not to feed a climate of fear and hysteria about obesity, but equally we need to consider its likely impact on health and disease. It is also imperative to consider the obesogenicity of our environments and culture, not just victim blame.

It seems likely that overweight and obesity will be increasingly responsible in the future for both disease burden in New Zealand, and also for an increasing contribution to ethnic and socioeconomic inequalities in health. Cancer is involved in that mix, along with CVD and diabetes.

Competing interests: None known.

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Using bread as a vehicle to improve the iodine status of New Zealand children

Meredith Rose, Rosie Gordon, Sheila Skeaff

Abstract

Aim To determine the iodine status of a sample of Dunedin school children, and to estimate how the addition of iodised salt to bread will improve their iodine status.

Method Between October and November 2007, iodine status in a sample of 93 Dunedin school children was assessed by urinary iodine concentration (UIC), serum thyroglobulin (Tg), and dietary iodine intake, estimated using an iodine-specific food frequency questionnaire. Data from the 2002 National Children’s Nutrition Survey and the New Zealand Food Composition Database were used to calculate the increase in total dietary iodine intake if bread is made with iodised salt, and subsequently, the predicted increase in UIC.

Results Both the median UIC of 63 mcg/L (interquartile range (IQR) 44–78 mcg/L) and the median serum Tg concentration of 14 mcg/L (IQR 10–23 mcg/L) classify this sample of children as mildly iodine deficient. The estimated dietary iodine intake was 54 mcg/day (IQR 41–65 mcg/L), which is well below the estimated average requirement (EAR) of 75 mcg/day; 83% of children in this study were found to have iodine intakes below the EAR. The addition of iodised salt to bread would increase the average iodine intake of these children to 75–104 mcg/day, thus decreasing the number of children who have an iodine intake less than the EAR to 4–46%. Consequently, the median UIC of these children would increase to 95–151 mcg/L.

Conclusion The introduction of iodised salt x bread, which is currently scheduled to become mandatory in September 2009, should improve the iodine status of New Zealand children. The use of iodised salt in other bakery products is encouraged to maximise this improvement.

Iodine is an essential nutrient because it is needed for the formation of thyroid hormones. Due to glaciation, which causes low soil iodine levels, New Zealand has a history of iodine deficiency.

A study carried out by Hercus in the 1920s reported endemic goitre throughout New Zealand, but with the introduction of iodised salt in 1938 at the level of 40–80ppm, the goitre rate fell to 0.1% by 1958.

Recently, however, there has been a re-emergence of iodine deficiency in New Zealand, with several studies reporting mild iodine deficiency in New Zealand children. The decline in iodine status has been attributed to a decreased concentration of iodine in milk resulting from the reduced use of iodophors as sanitisers in the dairy industry, an increase in processed foods in the diet which are made with non-iodised salt, an increase in the consumption of more fashionable salt products (such as non-iodised rock or sea salt), and decreased consumption of...
discretionary (i.e. table and cooking) iodised salt. A recent study in the USA reported that the iodine content of discretionary salt may not be consistent, both between brands and within an individual container of salt.

The effects of severe to moderate iodine deficiency on health are unequivocal and well documented, and include cretinism, hypothyroidism, and complications during pregnancy. There is a growing body of evidence that mild iodine deficiency also has adverse health consequences, with studies suggesting a link between mild iodine deficiency and decreased cognitive functioning, growth, and an increase in the prevalence of attention deficit hyperactivity disorder (ADHD).

A study carried out in Spain (n=1221) in a mildly iodine-deficient population reported that children with urinary iodine concentrations (UIC) below 100 mcg/L, the World Health Organization (WHO) cut-off for adequate iodine status, had significantly lower intelligent quotients (IQs) than children with UICs above this level.

Aghini Lombardi et al (n=270) found that children living in an area of mild iodine deficiency, and children born before iodine prophylaxis was introduced to an area that was now considered iodine sufficient, had slower reaction times than children who had always lived in an area of iodine sufficiency. Riaño Galán et al’s study (n=61) observed that in an area of mild iodine deficiency, children born to mothers with UIC below 200 mcg/L had poorer verbal and cognitive development than children born to mothers with UICs above 200 mcg/L.

A study by Zimmermann et al (n=310) reported that when a mildly iodine-deficient, slightly growth-retarded group of children in South Africa were given iodine supplementation, insulin-like growth factor-1 concentrations subsequently increased, thus suggesting a relationship between mild iodine deficiency and growth. One small study (n=16) carried out in Italy reported higher ADHD prevalence in children born to mothers living in a mildly iodine-deficient area compared to children born to mothers living in an iodine-sufficient area.

Other studies also suggest links between ADHD and resistance to thyroid hormone, and ADHD symptoms and thyroid stimulating hormone (TSH) levels that are elevated but still within the normal range. Together, these studies suggest that mild iodine deficiency may present risks to the cognitive abilities, growth, and behaviour of mildly iodine-deficient New Zealand children.

The decrease in iodine status in New Zealand, and its potential risks, has led to the recent amendment of Standard 2.1.1 – Cereals and Cereal Products in the Australia New Zealand Food Standards Code, which mandates the use of iodised salt in the production of bread (i.e. all yeast containing breads and flat breads that are baked, including sliced bread, bread rolls, pita breads, focaccia, bagels, English muffins, sweet buns, and fruit bread) in New Zealand from September 2009.

The aim of this study was to determine the iodine status of a sample of Dunedin school children, and to estimate how the addition of iodised salt to bread will improve their iodine status.

**Method**

**Participants and recruitment**—A convenience sample of children aged 10–12 years from around Dunedin was obtained by approaching four intermediate schools and two high schools; two
intermediate schools agreed to participate. Children were recruited through presentations in their classroom. Advertisements for participants were also placed in the local community newspaper allowing children from other schools the opportunity to participate. Interested parents and children provided a home address and were sent an information sheet, brochure and consent form in the mail. A total of 93 children aged 10 to 13 years participated in this study. Seventy children were recruited through the two intermediate schools, and 23 were recruited through newspaper advertising.

**Data collection**—For children recruited through their school, data collection was carried out at school, during the school day. Children who were recruited by newspaper advertising visited the Department of Human Nutrition after school or on a Saturday. Prior to sample collection the parent/guardian of each child completed a questionnaire that included contact details, child’s age, and ethnicity (European/Pakeha, Māori, Pacific [Islander], and Other), family doctor, household income, child’s use of supplements, medical conditions, allergies, medication use, and the perceived health status of the child.

The questionnaire also included an iodine-specific food frequency questionnaire (FFQ), which asked parents to describe the frequency of consumption by the child of foods that we considered the main source of iodine for New Zealand children (i.e. dairy products, milk, red meat, poultry, fish, shellfish, pulses and legumes, fruit and eggs).

Frequency categories included ‘never’, ‘less than once a week’, ‘1–3 times per month’, ‘once a week’, ‘2–4 times a week’, ‘5–6 times per week’, ‘once per day’, or ‘2 or more times a day’. All data was collected between October and November 2007.

Upon arrival at their appointment children had their height and weight measured using standard anthropometric techniques. For measurements children removed their shoes, emptied their pockets and, in most cases, wore their school uniform. A causal urine sample was obtained from each child. Children were asked to urinate in a bowl and then one of the investigators transferred approximately 5 mL of urine into a clean plastic specimen container (Labserv Technologies, Canada). Urine was frozen at -20°C until analysis.

Approximately 1 mL of non-fasting, whole capillary blood was collected by a finger prick blood sample from each child using a Tenderlett blood collection device (International Technidyne Corporation, New Jersey, USA). Blood was left to clot at room temperature for 60 minutes before being spun by centrifuge for 10 minutes. Serum was stored at -20°C until analysis.

**Data analysis**—Raw anthropometric data was entered into EpiInfo (CDC, Atlanta, GA, USA) and z-scores for height-for-age (HAZ) and weight-for-age (WAZ) were calculated using the National Centre for Health Statistics (NCHS)/WHO 1979 growth reference data. (This set of growth reference data was chosen because it better represents the growth patterns of New Zealand children in this age group and was formulated using standardised methodologies.) Calculations of descriptive statistics was carried out using Microsoft Excel 2008 software.

UIC was measured using method A as recommended by the International Council for the Control of Iodine Deficiency Disorders (ICCIDD). A certified reference material (Seronorm Trace Elements Urine, Sero AS, Asker, Norway) was used with each batch of samples and gave a mean iodine concentration of 132 mcg/L, within the expected range of 132–150 mcg/L, and a coefficient of variation (CV) of 4.4% (n=40). An internal standard (i.e. pooled urine sample) was also analysed with each batch of samples and gave a CV of 2.9% (n=20).

Tg concentration was measured from serum samples by Endolab, Christchurch Hospital, Christchurch. Samples were tested for the presence of auto-antibodies to Tg (TgAb) as these antibodies can interfere with Tg determination. Five fasting tested positive for TgAb and the results from these subjects were not included in Tg data analysis. Both Tg and TgAb were measured using immunoenzymatic assays with chemiluminescence detection. The Tg assay had an analytical detection limit of 0.1 µg/L and accuracy checked using the CRM 457 Tg standard (European Community Bureau of Reference). The inter-assay CV was 25% at 0.2 mcgTg/L, 8% at 40.4 mcgTg/L, and 5% at 333 mcgTg/L. Intra-assay CV’s were 5% at 0.2 mcgTg/L, 2% at 40.4 mcgTg/L, and 2% at 333 mcgTg/L.

UIC and serum Tg were skewed and are presented as medians and interquartile ranges.

**Dietary iodine intake**—Using the data obtained from the FFQ, the average total iodine intake was calculated using data (i.e. iodine content and serving sizes) from the New Zealand Food Composition Database (FOODfiles, 2006). This information was then combined with data from the 2002 National...
Children’s Nutrition Survey (CNS02) on children’s bread consumption to estimate the effect the introduction of iodised salt to bread will have on the average dietary iodine intake of these children. The formula of the US Institute of Medicine\textsuperscript{20} to estimate average dietary iodine intake from UIC (Daily intake = UIC/0.92 × [0.0009L/hr/kg × 24hr/day × weight]) was used to calculate the average change in UIC from the predicted change in dietary iodine levels.

**Ethical approval**—Ethical approval for this study was obtained from the University of Otago Ethics Committee. All children and their parents provided written informed consent before participating in the study.

**Results**

The study population (n=93) included 43 females and 50 males aged 10–13 years. The sociodemographic characteristics of the study population are summarised in Table 1. The median height of the study population was 152.4 cm, and the HAZ was 0.26 standard deviations greater than the reference population. The median weight was 42 kg, and the WAZ was 0.27 standard deviations greater than the reference population.

The iodine status of the population is summarised in Table 2. Both the median UIC and the median Tg concentration are indicative of mild iodine deficiency according to WHO/United Nations Children’s Fund (UNICEF)/ICCIDD.\textsuperscript{18} The estimated dietary iodine intake of 54 mcg/day is well below the EAR of 75 mcg/day for children of this age group.\textsuperscript{21}

One participant had a much higher daily iodine intake (179 mcg/day) compared to the other participants based on the FFQ results; this participant reportedly ate fish daily and other seafood 2–3 times a week. Sixty percent of parents reported that their child used iodised salt at the table, and 70% of parents reported using iodised salt during cooking. However, only 30% of children reported they used iodised salt regularly (i.e. more than once a day).

From the New Zealand Food Composition Database (FOODfiles, 2006)\textsuperscript{19} bread contains anywhere from trace amounts to 8 mcg of iodine per 100 gm; the median iodine content across all breads listed is 1 mcg iodine per 100mg. Bread also contains from 330 to 750 mg sodium per 100 gm. If we assume that 90% of sodium in bread comes from salt, then the sodium from salt content of bread can be calculated to be 300 to 675 mg per 100 gm.

After September 2009, this salt could contain from 25 to 65 ppm iodine; however, in reality, salt manufactures will aim for the mid point of 45 ppm to ensure they are within this range. Using the formula current iodine concentration in bread + (salt content of bread × concentration of iodine in salt), the new iodine content of bread was calculated to range from 15 to 31 mcg per 100 mg of bread (see Table 3).

Approximately 10% of iodine is lost during the baking process, decreasing the amount of available iodine in 100 gm of bread to from 14 to 28 mcg (see Table 3).
Table 1. Sociodemographic characteristics of study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of participants</th>
<th>Percentage</th>
<th>National data*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>43</td>
<td>46%</td>
<td>49%</td>
</tr>
<tr>
<td>Male</td>
<td>50</td>
<td>54%</td>
<td>51%</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td>na†</td>
</tr>
<tr>
<td>10–10.99 years</td>
<td>7</td>
<td>8%</td>
<td>49%</td>
</tr>
<tr>
<td>11–11.99 years</td>
<td>41</td>
<td>44%</td>
<td>47%</td>
</tr>
<tr>
<td>12–12.99 years</td>
<td>44</td>
<td>47%</td>
<td>51%</td>
</tr>
<tr>
<td>13–13.99 years</td>
<td>1</td>
<td>1%</td>
<td>49%</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European/Pakeha</td>
<td>73</td>
<td>79%</td>
<td>67%</td>
</tr>
<tr>
<td>Māori</td>
<td>13</td>
<td>14%</td>
<td>14%</td>
</tr>
<tr>
<td>Pacific‡</td>
<td>4</td>
<td>4%</td>
<td>7%</td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
<td>3%</td>
<td>12%</td>
</tr>
<tr>
<td>Parental income</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$20,000</td>
<td>6</td>
<td>6%</td>
<td>9%</td>
</tr>
<tr>
<td>$20,000–$50,000</td>
<td>22</td>
<td>24%</td>
<td>32%</td>
</tr>
<tr>
<td>&gt;$50,000</td>
<td>44</td>
<td>47%</td>
<td>59%</td>
</tr>
<tr>
<td>Declined to answer</td>
<td>21</td>
<td>23%</td>
<td></td>
</tr>
<tr>
<td>Health status</td>
<td></td>
<td></td>
<td>na</td>
</tr>
<tr>
<td>Excellent</td>
<td>77</td>
<td>84%</td>
<td></td>
</tr>
<tr>
<td>Fair</td>
<td>14</td>
<td>16%</td>
<td></td>
</tr>
<tr>
<td>Poor</td>
<td>0</td>
<td>0%</td>
<td></td>
</tr>
</tbody>
</table>

*from NZ Census 2006 data‡; †na = not applicable; ‡mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Table 2. Iodine status of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median (IQR)*</th>
<th>Recommended value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary iodine (mcg/day)</td>
<td>54 (41–65)</td>
<td>75†</td>
</tr>
<tr>
<td>Percentage less than EAR</td>
<td>83%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>UIC (mcg/L)</td>
<td>63 (44–78)</td>
<td>100–199‡</td>
</tr>
<tr>
<td>Percentage &lt; 50 mcg/L</td>
<td>33%</td>
<td>&lt;20%</td>
</tr>
<tr>
<td>Percentage &lt;100 mcg/L</td>
<td>88%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>Tg (mcg/L)</td>
<td>14 (10–23)</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

*IQR = Interquartile range
†EAR for NZ Children aged 9–13 years
‡WHO/UNICEF/ICCIDD cut-offs for adequate iodine status

Results from CNS02 indicate that, on average, children consume from 147 to 180 gm of bread products per day, thereby the iodine consumed from bread products after the addition of iodised salt will range from 21 to 50 mcg/day. When this was applied to the study population’s current iodine intake, it raised the median intake from 54 mcg I/day to 75–104 mcg I/day. This would result in 4–46% of the children in this study having iodine intakes less than the EAR.
Table 3. Proposed Range of Iodine Concentration of Bread due to varying salt content, and varying levels of salt iodisation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Current iodine content of bread mcg/100 gm (0.00–8.00)</th>
<th>Salt content of bread mg/100 gm</th>
<th>Concentration of iodine in salt ppm</th>
<th>New iodine content of bread mcg/100g</th>
<th>Iodine available after baking mcg/100g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lowest level of salt in bread</td>
<td>1</td>
<td>300</td>
<td>45</td>
<td>15 (14–22)</td>
<td>14 (13–20)</td>
</tr>
<tr>
<td>Highest level of salt in bread</td>
<td>1</td>
<td>675</td>
<td>45</td>
<td>31 (30–38)</td>
<td>28 (27–34)</td>
</tr>
</tbody>
</table>

The predicted improvement in iodine status of the study population due to the introduction of iodised salt to bread at the expected level is presented in Table 4 and shows that the calculated amount of iodine that will be added to the diet would result in a median UIC of 95–151 mcg/L. This would lead to 2–6% of the children from this study having UICs less than 50 mcg/L.

Table 4. Predicted iodine status of the study population after iodised salt is added to bread

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median (IQR)</th>
<th>Recommended value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary iodine (mcg/day)</td>
<td>75–104</td>
<td>75 *</td>
</tr>
<tr>
<td>Percentage less than EAR</td>
<td>4–46%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>UIC (mcg/L)</td>
<td>95–151</td>
<td>100–199</td>
</tr>
<tr>
<td>Percentage &lt; 50 mcg/L</td>
<td>2–6%</td>
<td>&lt;20%</td>
</tr>
<tr>
<td>Percentage &lt;100 mcg/L</td>
<td>6–60%</td>
<td>&lt;50%</td>
</tr>
</tbody>
</table>

* EAR for NZ Children aged 9–13 years.

Discussion

Both the median UIC and the median serum Tg concentration indicate the presence of mild iodine deficiency in this population of Dunedin schoolchildren. The WHO/UNICEF/ICCIDD criterion for identifying mild iodine deficiency in a population is a median UIC from 50–99 mcg/L. The value of 63 mcg/L found in the current study clearly indicates the presence of mild iodine deficiency. Additionally, WHO/UNICEF/ICCIDD state that no more than 20% of the population should have UICs less than 50 mcg/L. In this sample of children, 33% of the children had a UIC below this level.

These results support those of Skeaff et al who reported a median UIC of 66 mcg/L in Dunedin and Wellington schoolchildren (n=300), and the results of the CNS02 (n=3275), which found a median UIC of 66 mcg/L in a nationally representative sample of children between the ages of 5 and 14 years.

The WHO/UNICEF/ICCIDD criterion for identifying mild iodine deficiency in a population using serum Tg is a median Tg concentration from 10–19.9 mcg/L. The value of 14.4 mcg/L found in the current study indicates the presence of mild iodine deficiency in these children, and supports the UIC results.
As stated previously, the results of a large cross-sectional study carried out in Spain indicate that mild iodine deficiency puts children at risk of impaired cognition, and a randomised controlled trial has found that when mildly iodine deficient children are given supplementation, concentrations of growth hormones increased, suggesting that iodine deficiency can impair growth.

The results of the current study indicate that New Zealand children are unlikely to be affected by any iodine deficiency induced growth retardation as both HAZ and WAZ of the study population are greater than those of the reference population. However, the potential risk to cognition is a cause for concern.

In the current study, iodised salt was used at the table by 60% of the children. This figure is slightly higher than that reported in other studies, although use of salt in cooking by 70% of children is similar to other studies carried out in New Zealand. Skeaff et al’s study of 8 to 10 year olds in Dunedin and Wellington found that 50% of children used salt at the table, and 69% of caregivers reported using salt in cooking. Data from the CNS02 reported that 13% of children usually added salt at the table, 36% added salt sometimes, and 52% never added salt to meals at the table.

While iodised salt was used by 70% of the children in the current study, only 30% used salt regularly (i.e. once a day or more), indicating that discretionary use of iodised salt is likely to be making only a minor contribution to the total dietary iodine intake in these children.

The mean daily iodine intake estimated with the FFQ in this sample of children was 54 mcg/day; 83% of children in this study had dietary iodine intakes less than the EAR of 75 mcg/day. This value is comparable to the estimated mean dietary iodine intake reported in the 2003/04 NZTDS, which for 11–14 year old males was 60 mcg/day, and for 11–14 year old females was 50 mcg/day.

In our study iodised salt use was not included when quantifying the daily intake of iodine from the FFQ, meaning that the iodine intake could have been underestimated and may account for the discrepancy between the estimated dietary iodine intake of 54 mcg/day and the median UIC of 63 mcg/L. However, the less than regular use of iodised salt by more than two thirds of the children makes it unlikely that including iodised salt use would have markedly increased the dietary iodine intake measured here. Nonetheless, the FFQ results need to be interpreted with an element of caution.

While FFQs are frequently used in studies due to their low respondent burden, they are not the most accurate method of dietary assessment. Additionally, the FFQ used in this study has not been validated for use in children. Despite these limitations, the fact that the pattern of foods contributing to dietary iodine intake is similar to that of the 2003/04 New Zealand Total Diet Survey, and that unusually high iodine intakes can be easily attributed to specific food groups suggests that that the FFQ used in this study gave a reasonable representation of dietary iodine intake.

From the FFQ an average serving of milk was calculated to contain 20 mcg of iodine, while an average serving of seafood was calculated to contain 152 mcg iodine. However, on average, servings of milk were consumed 1.24 times a day, meaning that milk contributed 43% of the dietary iodine intake, while servings of seafood were only consumed 0.03 times a day, making the contribution of seafood only 9% of the average dietary iodine intake.
The results of one participant who regularly consumed fish and other seafood indicate that frequent consumption of these foods can increase daily iodine intake to above the recommended dietary intake (RDI) of 120 mcg/day. Regardless, recommending an increase in seafood consumption would not be practical way of increasing iodine intake in this age group, as many children find seafood unpalatable, and fish and other seafood is expensive in New Zealand. It seems unlikely that individual dietary modification would be a practical way of increasing iodine intake to the recommended level in this age group.

The recent amendment of Standard 2.1.1 – Cereals and Cereal Products in the Australia New Zealand Food Standards Code\textsuperscript{16} will require the mandatory use of iodised salt in the production of most breads after September 2009. While we cannot ascertain exactly how much bread individual children of this age group will need to consume to improve their individual iodine status, we have estimated that the addition of iodised salt to bread will, on average, increase children’s iodine intakes from 21 to 50 mcg/day and that this increase in iodine intake would decrease the number of children who have an iodine intake less than the EAR to 4-46%, compared to the 83% observed in the current study.

Using the US Institute of Medicine formula\textsuperscript{20} we predicted that the median UIC would increase to 95–151 mcg/L, and that consumption of bread that contains the highest proportion of salt, would result in only 2% of the children having UICs less than 50 mcg/L and only 6% of children having UICs <100mcg/L. According to WHO/UNICEF/ICCIDD, this would mean that the children would have adequate iodine status.

However, with the current emphasis from the National Heart Foundation for the reduction of the sodium content of commercially made bread, the possibility that bread will be able to continue to deliver sufficient iodine to significantly improve iodine status is a concern. It seems prudent, therefore, to encourage bread manufacturers to use iodised salt in all the bakery products they produce to maximise the increase in dietary iodine.

Additionally, a comprehensive monitoring programme should be established to monitor the impact of adding iodised salt to bread, and to allow for the early identification of a reduction in the salt content in bread that might impact on the iodine status of the population. It may be that at some time in the future a vehicle other than salt will need to be identified to ensure adequate intake of iodine in the New Zealand population.

Concerns have been raised over the potential for toxic intakes of iodine with the implementation of mandatory fortification of bread with iodised salt, especially in younger children. However, calculations using the dietary information from the one child in this study with a current iodine intake of 179 mcg/L suggest that it is highly unlikely that a child aged 10–14 years with an unusually high iodine intake would reach the current upper level of intake (UL) of 600 mcg/L.

If this child consumed the higher amount of bread recorded in the CNS (180 g), which contained the highest amount of salt (660 mg/100 gm), with the highest level of salt iodisation (65 ppm), this child would only be consuming 256 mcg iodine/day which is less than half of the UL. Even if this child ate 350 g of bread a day, which equates to
between 6 and 8 slices of bread, their daily iodine intake would be 329 mcg/day, which is still well below the UL.

The findings of this study indicate that using bread as a vehicle for introducing more iodine to the diet can improve the iodine status of New Zealand children, and could reduce the risk of complications associated with mild iodine deficiency. However, decreasing the salt content of bread has the potential to limit the effectiveness of this intervention, and bread producers should be encouraged to use iodised salt in as many bread products as possible to maximise improvement to the iodine status of New Zealand children.

**Competing interests:** None known.

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**References:**


Rates of common communicable illnesses in non-anaemic 12–24 month old South Island, New Zealand children
Andrea J Cross, Anne-Louise M Heath, Elaine L Ferguson, Andrew R Gray, Ewa A Szymlek-Gay

Abstract

Aims To describe the incidence of parentally reported illness in otherwise healthy South Island toddlers; characterise the predictors of illness; and determine whether there was a relationship between teething and illness in this population.

Methods A 20-week randomised controlled trial was conducted on 1-year-old children (n=225) from Otago and Southland between February 2004 and December 2005. Information on symptoms of morbidity, occurrence of teething, and childcare attendance were recorded daily throughout the intervention period. Morbidity symptoms were categorised into respiratory illness (RI), gastrointestinal illness (GII), ear infection, and total illness, and the number and duration of events were determined.

Results The mean (SD) number of total illnesses was 3.4 (2.3) per 20 weeks, with an average duration of 4.5 days. Episodes of RI were most common (50% of total illness events), and tended to be the longest in duration (mean of 3.7 days). Having siblings aged less than 5 years (23% increase, 95%CI 6%–42%, p=0.007) and attending childcare (72% increase, 95%CI 38%–113%, p<0.001), were positively associated with the number of total illness events but not duration. In addition, teething was positively associated with total events (OR 1.94, 95%CI 1.45–2.60, p<0.001), RI events (OR 2.03, 95%CI 1.41–2.93, p<0.001) and GII events (OR 1.90, 95%CI 1.36–2.67, p<0.001).

Conclusion This study has shown that illness (particularly RI) is common in the second year of life. It has also confirmed that attending childcare and having siblings aged under 5 years increases the number of illness events. An association between teething and the occurrence of illness was also seen but the exact nature of this relationship requires verification.

In New Zealand there are few quantitative data describing the illnesses experienced by otherwise healthy children. In fact, the existing data are only for notifiable disease or hospitalisation rates, with information on common childhood illnesses based primarily on experiential data from primary care practitioners. Furthermore, the figures reported in the literature are commonly combined for the one to four year-old age group. This may be masking important information at specific ages, particularly given that rates of illness are reported to be higher during the second year of life than at any other point in childhood.

International research has repeatedly shown that low socioeconomic status (SES) and attending childcare are associated with higher rates of illness in preschool children.
Research has shown that while people who are of low SES visit the doctor more often they are also more likely to have unmet health needs.\textsuperscript{14}

New Zealand preschool children are eligible for subsidised primary care. However, there are other issues such as access to transport and prescription costs that may act as barriers to health service utilisation and result in associations between illness and SES.\textsuperscript{14} Further, with the increasing number of working parents and the corresponding growth in childcare attendance,\textsuperscript{15} increased illness in those who attend childcare would have important implications for primary care planning, practice, and funding.

An additional factor requiring further research is the discordance between parental beliefs about teething and illness and the view of health professionals. Specifically, surveys indicate that many parents\textsuperscript{16–18} and health professionals\textsuperscript{19,20} believe that teething causes, or predisposes a child to, certain illnesses, while WellChild providers state that teething is not in itself a cause of illness.\textsuperscript{21} Thus, a comprehensive exploration of the relationship between teething and robustly defined illness is of interest.

Therefore, the objectives of this study were primarily to describe the incidence of parentally reported illness in otherwise healthy South Island toddlers and characterise the predictors of illness in this sample. A secondary objective was to determine whether there was a relationship between teething and illness in this population.

\textbf{Methods}

\textbf{Study design}—This study involved secondary analysis of data collected during the Toddler Food Study (TFS). The TFS was a randomised, partial-blind, placebo-controlled, 20-week intervention trial conducted between February 2004 and December 2005, and was designed to evaluate the efficacy of food-based recommendations for improving iron and zinc status.

To be eligible for participation toddlers needed to be:

- Aged between 12 and 20 months;
- Apparently healthy;
- Non-anaemic (two cutoffs applied: haemoglobin <105 g/L and haemoglobin <110 g/L, and serum ferritin <12 µg/L);
- Willing to comply with the food-based intervention; and
- Not known to suffer from medical conditions, or be taking medications, known to affect iron absorption (including iron supplements and iron-fortified toddler milk).

The trial involved a convenience sample of 225 1-year-old children from Dunedin, Balclutha, Oamaru, Milton, and Invercargill. Full details of the study design and research methodologies can be found elsewhere.\textsuperscript{22}

Ethical approval was granted by the Human Ethics Committee of the University of Otago and written informed consent was obtained from a guardian of each toddler participating in the study.

\textbf{Data collection}—Parents completed a pre-tested questionnaire which included questions to determine sociodemographic characteristics of the child and their family and the child’s health status at baseline. Ethnicity was self-determined with parents able to tick all ethnic groups that applied.

For the child, the presence of illness, teething, and childcare attendance were recorded by parents daily throughout the intervention. Data collected included whether the child had been unwell (yes/no); had any of the following illness symptoms (yes/no): runny or blocked nose, cough, sore throat, wheezing or difficulty breathing, cold or flu, fever, ear infection, diarrhoea (defined as: three or more loose, watery stools in 24 hours; and vomiting); had been teething (yes/no, parentally defined); or had attended childcare (yes/no).
Childcare attendance was defined as home- or centre-based care with two or more children not including siblings. Parents were also instructed to indicate each day whether any other illnesses had occurred. Every 2 weeks a member of the study team visited the families to collect the illness records. At these visits records were checked for errors and inconsistencies with immediate resolution where possible. Parents were also reminded of the importance of complete records.

Length (accurate to ±0.1 cm) and weight (accurate to ±0.1 kg) were measured at baseline using standardised methods. These were used to calculate body-mass-index (BMI) and to compute percentiles using the WHO growth standards.

Non-fasting blood samples were obtained by venipuncture and analysed for serum transferrin receptor (STfR) in the Human Nutrition laboratory at the University of Otago by enzyme immunoassay using a commercial kit (Ramco Laboratories Inc, Stafford, TX). Only STfR was reported in the current study because unlike the other measures of iron status measured in the TFS it does not change with infection.

Data analysis and definitions—Morbidity data for the entire 20-week period were categorised into four broad categories because of the relatively low frequency of illness; namely:

- **Respiratory infection** (RI)—defined as any of:
  - Cold or flu;
  - At least three of runny or blocked nose, cough, sore throat, or wheezing or difficulty breathing (in non-asthmatics);
  - “Other” respiratory infection (e.g. croup);
- **Ear infection**;
- **Gastrointestinal infection** (GII)—defined as any of diarrhoea, vomiting, or “other” symptom of gastrointestinal infection (e.g. stomach pain); and
- **Total illnesses** (any of RI, ear infection, GII, or “other” illness not classified into the previous categories [e.g. chicken pox]).

A **morbidity episode** for a specific illness category was defined as an event of morbidity symptoms for which there were at least three illness-free days on either side of it.

The **episodic duration** was defined as the number of days the child experienced morbidity symptoms during a morbidity episode.

**Unwell events** and **unwell duration** were defined by applying the same criteria as was used for morbidity episode and episodic duration to the days when parents reported their child was Unwell (yes/no responses to “Was your child unwell today?”).

Total illness data were also grouped by month to explore seasonal variation in the incidence of illness. This was done by assigning each event to a month based on the date of the first day of illness. Illnesses that crossed over months were classified into the month the first day of illness fell into.

Statistical analysis—Intercooled Stata v9.0 software (StataCorp, College Station, TX, USA) was used to generate the morbidity variables and for all statistical analyses. A p-value of <0.05 was considered to indicate statistical significance.

The statistics used to describe the illness data (Table 2) were: total number of events, the number (%) of children having one or more episode, the mean number of episodes during the 20-week study, the mean number of episodes per year, the mean duration of episodes, and incidence rate. These were calculated for total illness, each of the individual illness categories and unwell.
Table 1. Baseline characteristics of study population (all values No. (%) unless otherwise indicated)

<table>
<thead>
<tr>
<th>Variable</th>
<th>n=225</th>
<th>Variable</th>
<th>n=225</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio-demographic and family characteristics</strong></td>
<td></td>
<td><strong>Characteristics related to health status</strong></td>
<td></td>
</tr>
<tr>
<td>Age (months) (^a)</td>
<td>17.1 (2.8)</td>
<td>Born premature (&lt;37 weeks gestation) (^b)</td>
<td>22 (9.8)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td>Low birth weight (&lt;2500g) (^c)</td>
<td>14 (6.4)</td>
</tr>
<tr>
<td>NZ European</td>
<td>178 (80)</td>
<td>Breastfed for &gt;6 months (^d)</td>
<td>100 (51.3)</td>
</tr>
<tr>
<td>NZ Māori</td>
<td>25 (11)</td>
<td>Asthma</td>
<td>8 (4)</td>
</tr>
<tr>
<td>Pacific Peoples</td>
<td>5 (2)</td>
<td>Other health condition</td>
<td>21 (9)</td>
</tr>
<tr>
<td>Other</td>
<td>16 (7)</td>
<td>Regular medication use</td>
<td>18 (8)</td>
</tr>
<tr>
<td>Mother with a University education</td>
<td>82 (36)</td>
<td>&quot;Excellent&quot; health reported by parent</td>
<td>112 (50)</td>
</tr>
<tr>
<td>Father with a University education</td>
<td>51 (24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Household income (NZD)</td>
<td></td>
<td><strong>Anthropometric and biochemical iron status</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;$30,000</td>
<td>27 (12)</td>
<td>Anthropometry (^e)</td>
<td></td>
</tr>
<tr>
<td>$30,000 - $70,000</td>
<td>123 (55)</td>
<td>Length (cm)</td>
<td>51.0 (4.5)</td>
</tr>
<tr>
<td>&gt;$70,000</td>
<td>40 (18)</td>
<td>Weight (kg)</td>
<td>11.3 (1.8)</td>
</tr>
<tr>
<td>Not answered</td>
<td>16 (7)</td>
<td>Weight-for-age percentile</td>
<td>67.4 (20.7)</td>
</tr>
<tr>
<td>Household occupants</td>
<td></td>
<td>BMI-for-age percentile</td>
<td>71.8 (25.1)</td>
</tr>
<tr>
<td>Number of total people in household (^f)</td>
<td>3.0 (1.1)</td>
<td>Serum Transferrin Receptor (^g)</td>
<td>8.8 (6.6, 7.0)</td>
</tr>
<tr>
<td>Number of siblings aged &lt;6 years (^d)</td>
<td>0.4 (0.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child never in a smoking household</td>
<td>208 (93)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)=Mean (SD); \(^b\)="Unsure or missing" n=2; \(^c\)="Unsure or missing" n=6; \(^d\)="Unsure or missing" n=62; \(^e\)=Other categories: "Good", "Fair", "Poor"; \(^f\)=Percentiles determined by comparison with the World Health Organization child growth standards; \(^g\)=Geometric mean (95% confidence interval) n=214.
Table 2. Parentally-reported illness rates over a 20-week period (All values Mean (SD) unless otherwise indicated)

<table>
<thead>
<tr>
<th>Variable</th>
<th>n=220</th>
<th>Gastrointestinal Illness</th>
<th>n=220</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total illnesses a</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of events</td>
<td>757</td>
<td>Total number of events</td>
<td>238</td>
</tr>
<tr>
<td>Number (% with ≥ 1 episode</td>
<td>205 (93)</td>
<td>Number (% with ≥ 1 episode</td>
<td>160 (73)</td>
</tr>
<tr>
<td>Episodes per 20 week period</td>
<td>3.4 (2.3)</td>
<td>Episodes per 20 week period</td>
<td>1.5 (1.6)</td>
</tr>
<tr>
<td>Mean episodes per year</td>
<td>8.9</td>
<td>Mean episodes per year</td>
<td>3.1</td>
</tr>
<tr>
<td>Episodic duration (days)</td>
<td>4.5 (4.1)</td>
<td>Episodic duration (days)</td>
<td>2.1 (2.0)</td>
</tr>
<tr>
<td>Incidence rate (per month)</td>
<td>0.3</td>
<td>Incidence rate (per month)</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory Illness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of events</td>
<td>375</td>
<td>Total number of events</td>
<td>115</td>
</tr>
<tr>
<td>Number (% with ≥ 1 episode</td>
<td>161 (73)</td>
<td>Number (% with ≥ 1 episode</td>
<td>55 (25)</td>
</tr>
<tr>
<td>Episodes per 20 week period</td>
<td>1.7 (1.6)</td>
<td>Episodes per 20 week period</td>
<td>1.0 (1.3)</td>
</tr>
<tr>
<td>Mean episodes per year</td>
<td>4.4</td>
<td>Mean episodes per year</td>
<td>2.5</td>
</tr>
<tr>
<td>Episodic duration (days)</td>
<td>3.7 (4.4)</td>
<td>Episodic duration (days)</td>
<td>1.8 (3.1)</td>
</tr>
<tr>
<td>Incidence rate (per month)</td>
<td>0.4</td>
<td>Incidence rate (per month)</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhoea b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of events</td>
<td></td>
<td>Total number of events</td>
<td>1115</td>
</tr>
<tr>
<td>Number (% with ≥ 1 episode</td>
<td></td>
<td>Number (% with ≥ 1 episode</td>
<td>219 (96)</td>
</tr>
<tr>
<td>Episodes per 20 week period</td>
<td></td>
<td>Episodes per 20 week period</td>
<td>5.1 (2.8)</td>
</tr>
<tr>
<td>Mean episodes per year</td>
<td></td>
<td>Mean episodes per year</td>
<td>13.2</td>
</tr>
<tr>
<td>Episodic duration (days)</td>
<td></td>
<td>Episodic duration (days)</td>
<td>5.3 (3.6)</td>
</tr>
<tr>
<td>Incidence rate (per month)</td>
<td></td>
<td>Incidence rate (per month)</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ear infections</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of events</td>
<td>176</td>
<td>Total number of events</td>
<td>1115</td>
</tr>
<tr>
<td>Number (% with ≥ 1 episode</td>
<td>88 (40)</td>
<td>Number (% with ≥ 1 episode</td>
<td>219 (96)</td>
</tr>
<tr>
<td>Episodes per 20 week period</td>
<td>0.8 (1.3)</td>
<td>Episodes per 20 week period</td>
<td>5.1 (2.8)</td>
</tr>
<tr>
<td>Mean episodes per year</td>
<td>2.1</td>
<td>Mean episodes per year</td>
<td>13.2</td>
</tr>
<tr>
<td>Episodic duration (days)</td>
<td>2.3 (2.5)</td>
<td>Episodic duration (days)</td>
<td>5.3 (3.6)</td>
</tr>
<tr>
<td>Incidence rate (per month)</td>
<td>0.2</td>
<td>Incidence rate (per month)</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unwell c</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of events</td>
<td></td>
<td>Total number of events</td>
<td>1115</td>
</tr>
<tr>
<td>Number (% with ≥ 1 episode</td>
<td></td>
<td>Number (% with ≥ 1 episode</td>
<td>219 (96)</td>
</tr>
<tr>
<td>Episodes per 20 week period</td>
<td></td>
<td>Episodes per 20 week period</td>
<td>5.1 (2.8)</td>
</tr>
<tr>
<td>Mean episodes per year</td>
<td></td>
<td>Mean episodes per year</td>
<td>13.2</td>
</tr>
<tr>
<td>Episodic duration (days)</td>
<td></td>
<td>Episodic duration (days)</td>
<td>5.3 (3.6)</td>
</tr>
<tr>
<td>Incidence rate (per month)</td>
<td></td>
<td>Incidence rate (per month)</td>
<td>1.4</td>
</tr>
<tr>
<td>Morbidity episode = An event of morbidity symptoms with three illness-free days on either side.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Episodic duration = The number of days the child experienced morbidity symptoms during each event.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Defined as any of: RI, ear infection, GII, or “other” illness not classified into the previous categories (e.g. chicken pox).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b A subcategory of GII.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c Defined as parent indicating child was “Unwell”.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The mean number of episodes per year was extrapolated from the mean number of episodes per 20 weeks. Incidence rates were calculated by dividing the mean number of events during the study by the total number of days at risk. All descriptive data are unadjusted. Unadjusted statistical comparisons (Figure 1) were made using two sample t-tests.

To determine whether certain sociodemographic, family, and illness-related characteristics were predictors of total illnesses, we developed models which looked at the predictive power of these variables using fractional polynomials for continuous predictors. Specifically, we used Poisson regression to determine their relationship with the number of morbidity episodes, and linear regression to determine the relationship with episodic duration (Table 3).

Variables considered in the final model were: age, sex, age×sex, serum transferrin receptor (a measure of iron status not confounded by infection), education (highest maternal qualification: 3-year degree or higher, not a 3-year degree or higher, not answered), household income (in NZD: less than $30,000, $30,000-$70,000, more than $70,000, not answered), prioritised ethnicity (in order of priority: NZ Māori, Pacific Peoples, Other, NZ European), proportion of days in childcare, proportion of days the child was in the study in the peak illness months (May to September (see Figure 1)) and TFS treatment group.
To determine whether teething on a given day was temporally associated with illness on that day we modelled teething and the various morbidity categories using logistic regression with robust standard errors and Huber-White sandwich estimator to control for repeated measures. This was done for total illness, RI, ear infection, GII, diarrhoea, and “other” illness.

Log transformations were used where residuals were skewed or exhibited non-constant variance (Episodic duration [Table 3]). Both marginal and conditional standardised residual plots were examined for the purposes of model checking.

**Results**

The baseline sociodemographic and biochemical characteristics of the toddlers are shown in Table 1. Overall, those who participated in the TFS had a mean age of 17.1 months, were more likely to be male (56%) and were more likely to be NZ European (80%). The baseline characteristics of participants were similar between sexes, except that boys were significantly taller and heavier and had a higher BMI.

Of the 486 families expressing an interest in the study, 225 toddlers were enrolled, from which 10 were lost to follow-up. Compared with those who completed the study, those lost to follow up were more likely to be female and a lower percentage of their mothers had a university level education.
Table 3. Predictors of parentally-reported total illnesses

<table>
<thead>
<tr>
<th>Variables</th>
<th>Illness events</th>
<th>P-value</th>
<th>Duration of illness episode</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.01 (0.97, 1.05)</td>
<td>0.513</td>
<td>0.07 (0.93, 1.02)</td>
<td>0.228</td>
</tr>
<tr>
<td>Sex</td>
<td>1.78 (0.66, 5.63)</td>
<td>0.336</td>
<td>0.42 (0.11, 1.58)</td>
<td>0.198</td>
</tr>
<tr>
<td>Ethnicity (NZ European vs. NZ Māori)</td>
<td>0.91 (0.69, 1.22)</td>
<td>0.507</td>
<td>1.14 (0.92, 1.40)</td>
<td>0.437</td>
</tr>
<tr>
<td>Household income (NZ$; $30,000 – $70,000 or &lt; $30,000)</td>
<td>1.20 (0.97, 1.51)</td>
<td>0.086</td>
<td>1.04 (0.75, 1.45)</td>
<td>0.884</td>
</tr>
<tr>
<td>Maternal education (University vs. Non-university qualification)</td>
<td>1.08 (0.83, 1.39)</td>
<td>0.444</td>
<td>0.95 (0.77, 1.19)</td>
<td>0.710</td>
</tr>
<tr>
<td>Siblings aged under 5 years</td>
<td>1.23 (1.06, 1.42)</td>
<td>0.027</td>
<td>1.11 (0.84, 1.49)</td>
<td>0.499</td>
</tr>
<tr>
<td>Proportion of days in childcare</td>
<td>1.72 (1.38, 2.13)</td>
<td>0.001</td>
<td>0.95 (0.86, 1.05)</td>
<td>0.221</td>
</tr>
<tr>
<td>Proportion of days in peak illness months</td>
<td>1.58 (1.19, 2.11)</td>
<td>0.020</td>
<td>1.43 (1.01, 1.99)</td>
<td>0.033</td>
</tr>
<tr>
<td>Serum Transformin receptor (non-status)</td>
<td>0.94 (0.88, 1.01)</td>
<td>0.078</td>
<td>0.95 (0.84, 1.04)</td>
<td>0.338</td>
</tr>
</tbody>
</table>

a The multivariate analysis adjusted for variables listed in the table and age*sex interaction term and Toddler Food Study group.

b Values are Incidence rate ratio (95% CI).

c Values are Ratio of the mean difference (95% CI).

d Reference category: no siblings.

e Months from May to September.

Rates of illness are shown in Table 2. Over the 20-week intervention there was a mean of 3.4 total illness events, which lasted an average of 4.5 days, at an incidence rate of 0.9 per month or approximately nine events per child per year. RI episodes were most common (375 total events), followed by GII events (328 total events). In addition, RI episodes tended to be of longer duration (mean of 3.7 days).

The mean (SD) number of days spent in childcare per month was 5.6 (6.3) for all children (i.e. attendees and non-attendees) and 8.4 (6.0) days per month after excluding those who never attended childcare (n=74). The mean (SD) number of days a parent reported a child was teething was 2.3 (3.4) per month in all children and 3.2 (3.7) days per month after excluding children who did not teethe (n=61).

Figure 1 shows the mean number of total illness episodes by month. This shows that the incidence of illness was highest between the months of May and September. Further, the mean number of illness events in June, August, and September were significantly higher than in January, February, March, April and October, and the mean number of illnesses in June and August were significantly higher than in November and December (P<0.05).

Table 3 shows the relationship between those variables analysed as predictors of illness and both the number and duration of total illness episodes. These data show that the number of siblings aged less than 5 years (23%, 95%CI 6%–42%), the proportion of days spent in childcare (72%, 95%CI 38%–113%), and the proportion of days in the peak illness season (59%, 95%CI 19%–111%) were significantly associated with increased number of total illness events, but only the latter was associated with increased duration of total illness (43%, 95%CI 3%–93%). Further analysis showed that the total number of household occupants was not a predictor of illness when included in the final model (incidence rate ratio: 1.01, 95%CI 0.94–1.10, p=0.658).
Table 4 shows the relationship between teething and various illness categories. These data show that teething was associated with a 94% increase in total events (95%CI 45%–160%), 103% more RI events (95%CI 41%–193%), and 90% more GII events (95%CI 36%–167%). Further analysis of GII events showed that there was also a 122% increase in diarrheal events associated with teething (95%CI 53%–220%). Sub-group analysis of the number and duration of all illness events between those who never teether during the study and those who did, found that non-teethers experienced an average of 0.68 fewer events (95%CI 0.02, 1.34; p=0.045) but there was no difference in duration (data not shown).

Table 4. Relationship between teething and illness (values are odds ratio [95% CI])

<table>
<thead>
<tr>
<th>Variables</th>
<th>Illness events</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Illnesses</td>
<td>1.54 (1.45, 2.60)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Respiratory Illness</td>
<td>2.05 (1.41, 2.93)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ear Infection</td>
<td>1.56 (0.98, 2.49)</td>
<td>0.050</td>
</tr>
<tr>
<td>Gastrointestinal Illness</td>
<td>1.90 (1.30, 2.57)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>2.22 (1.53, 3.23)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Other Illness</td>
<td>0.53 (0.49, 1.78)</td>
<td>0.83</td>
</tr>
</tbody>
</table>

Discussion

This prospective 20-week analysis of illness in 225 non-anaemic South Island children aged between 12 and 20 months found that, of the three specific illness categories examined, RI was both most frequently observed and of the longest duration. These data also showed that having pre-school aged siblings and being in childcare increases the number of total illnesses but has no effect on the duration of illness. Teething was also significantly associated with total illness, RI, GII, and diarrhoea.

In our study there were approximately 9 total illness and 13 unwell events per child per year. This is in agreement with the New Zealand Paediatric Society which estimates that young children average around 12 infections per year. This study has also confirmed previous research showing RI to be the most common illness experienced by children in their second year of life where it is estimated that RI comprises around 50% of general practitioner consultations in young children.

The association between childcare and illness in pre-school children is well established, although we believe that this is the first time it has been shown in a New Zealand population. Past research has consistently shown childcare to be associated with higher rates of GI, ear infection, and RI where it has been estimated that up to 30% of RI is associated with childcare attendance. The
mechanism for this is probably increased exposure to infectious agents. This mechanism may also account for the association between illness and having siblings aged less than 5 years found in this study. This finding is also consistent with previous studies which have demonstrated an increased incidence of RI in infants with older siblings.\textsuperscript{10,29}

In contrast to studies conducted in other countries,\textsuperscript{9,30} indicators of SES (i.e. maternal education and household income) were not found to be predictors of illness in this population. This is likely due to clustering of socioeconomic indicators in this study, such that nearly 60\% of families had an annual household income between $30,000 and $70,000. Thus, it is likely that there were insufficient families in the high and low income categories to allow differences in rates of illness to be seen, and as such we cannot rule out SES as a predictor of illness in New Zealand children. Furthermore, it is also possible that there was an interaction between SES and attendance at childcare in this study.

While previous New Zealand research\textsuperscript{31} has shown a relationship between household crowding and illness in children, a similar relationship was not seen in this sample. One possible reason for this discrepancy is that “total number of people in the household” was not a sufficiently robust measure of crowding to allow a relationship to be detected because it does not take into account household size. However, because we did not collect information about the number of bedrooms in the house, a more widely accepted index such as the Canadian National Occupancy Standard could not be calculated. An additional explanation is that overcrowding was uncommon in our study population; which is consistent with data showing that Dunedin (the main population base for our study) has the lowest levels of crowding of any city in New Zealand.\textsuperscript{32} Moreover, as crowding is likely to be related to SES, the socioeconomic variables in our models may have masking any relationship between crowding and illness.

The positive associations between teething and total illness, RI, GII, and diarrhoea found in this study were surprising, and in contrast to information given to parents.\textsuperscript{21} However, it is difficult to interpret these findings for a number of reasons. Several surveys have shown that parents believe that teething is a cause of illness,\textsuperscript{16,17,20} while prospective studies have shown that only dribbling, disturbed sleeping and reddened cheeks, but not diarrhoea or symptoms of RI are associated with teething.\textsuperscript{33,34} There is also evidence to suggest that some parents may characterise teething by the presence of what they consider to be “teething symptoms” rather than by the eruption of a tooth.\textsuperscript{18} If this was done by parents in this study, then rates of illness occurring with teething may be inflated. However, 52\% of teething reports were not associated with illness symptoms so this cannot be the sole explanation. Furthermore, many parents (n=61) did not record teething throughout the 20-week study and as it is likely that the child would have had teeth erupting during this time, this suggests that some parents believed that tooth eruption could occur in the absence of “teething”. The exact mechanism behind these observations is difficult to establish from these data and therefore requires further exploration in a study that objectively measures teething status and symptoms of illness in this age group.

The major limitation of this study is that data were based on parental reports which were not generally confirmed by an objective source, such as diagnosis by a health
professional. Nonetheless, this is likely to have a minimal effect on the results presented here as parental reports would be more likely to result in incorrect classification of an illness rather than fabrication of illnesses. Furthermore, at least for RI, any effect of over-reporting of individual symptoms was minimised by requiring participants to have at least three of runny or blocked nose, cough, sore throat, or wheezing or difficulty breathing. Similarly the classification of diarrhoea as “three or more loose, watery stools per day” means that it is unlikely that a single or occasional loose stool was incorrectly classified as diarrhoea.

The major strengths of this study are the comprehensive daily collection of data for 20 weeks (i.e. 140 days of collection per child), the rigorous definitions for RI and diarrhoea despite being based on parental reports, and the way in which Morbidity Events and Episodic Duration were defined. These quantitative data on common communicable illnesses in New Zealand children in their second year of life may be of use to general practitioners and WellChild providers when advising parents of how much illness is usual in this age group. Furthermore, the confirmation of a positive relationship between illness and both childcare and number of siblings aged less than five in a New Zealand setting provide some indications for further research.

In particular it may be important to determine whether those in childcare utilise primary care services more often than non-attendees, a factor which could have important implications for funding and future service provision. This may be particularly pertinent given the recent Government introduction of 20 hours of free early childhood education for three and 4 year olds and free general practitioner visits for those aged under 6 years.

In conclusion this study has characterised the rates of common communicable illness in South Island children in their second year of life. Specifically it has shown RI to be the most commonly observed illness, and also the illness of longest mean duration. In addition, this study found that attending childcare and having siblings aged under 5 years increased the number of illness events. An association between teething and the occurrence of illness was also seen but must be confirmed in a study that objectively monitors tooth eruption, as well as morbidity.

**Competing interests:** None known.

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References:

Comparison of the content of the New Zealand influenza pandemic plan with European pandemic plans

Nick Wilson, Michael G Baker

Abstract

Aim To critically review version 16 of the New Zealand (NZ) influenza pandemic plan in relation to the content of European pandemic plans.

Methods We used a published framework that had been developed for describing 29 European pandemic plans (all of which were available in 2006). This framework was used to rate the content of the NZ plan compared with the combined results for European plans.

Results In terms of plan content on border control aspects, the NZ plan scored higher than the average European plan (8.0 vs 4.9 out of 10.0 respectively) and similarly for the antiviral aspects (13.5 vs 10.6 out of 17.0). However, it scored slightly lower for the vaccine aspects (4.5 vs 5.3 out of 11.0). An alternate (more stringent) scoring system suggested that the relative quality of the NZ plan was poorer for antiviral aspects and fairly similar for vaccine aspects (to the average European plan). Even so, this framework had various limitations and probably favoured European countries which often have their own vaccine/antiviral production capacity. The NZ plan may also have scored more highly if the framework used considered other control measures (e.g. social distancing interventions). This comparison also identified some gaps which could be worth addressing in the planned 2009 version of the NZ plan (e.g. improved detail around priority groups for antivirals and pandemic vaccine and consideration of pneumococcal vaccine).

Conclusions The NZ influenza pandemic plan compared favourably with the average European plan in many aspects but not all. There is scope for further improvements and additions to be made in the next (2009) version of the NZ plan.

The threat of pandemic influenza remains a substantial concern globally. Recently the head of the World Health Organization (WHO) has commented on this threat:

Turning to the threat of pandemic influenza, [Margaret] Chan cautioned that ‘it has by no means receded, and we would be very unwise to let our guard down or slacken our preparedness measures.’ Countries with solid health infrastructures and efficient mechanisms for reaching vulnerable populations will be in the best position to cope, she said.¹

As well as being a concern for governments, survey data from the United States indicates that “avian flu” is one of the three biggest perceived health threats by the public (along with cancer and HIV/AIDS).²

New Zealand (NZ) has responded to the pandemic influenza threat by funding research,³ developing and revising very detailed national level pandemic plans,⁴ and running simulation exercises to test and refine the planning tools.⁵ There has also been active planning at the district health board level.⁶ An earlier analysis of plans in the
Asia/Pacific region found the NZ national-level plan (version 14 in 2005) “compared favourably with the best European plans”.7

The current (version 16) NZ plan,4 has been compared with other plans in terms of its border control aspects and found to be comparatively well developed.8 But there have been no other published studies on this plan, despite the public health importance of such work and the ongoing process of plan revision being undertaken in this country.

Methods

Framework—We used a published framework that had been developed for describing the content of pandemic plans.9 This work was also the data source for the content information on the 29 European plans considered. Items in the framework that were excluded are in Table 1. All these European plans were eligible for inclusion if they were published before 30 September 2006 (which was the same year that the current version 16 NZ plan was published).

Table 1. Items included in the published framework9 but which were excluded from this analysis for specific reasons

<table>
<thead>
<tr>
<th>Item</th>
<th>Reason for exclusion from this analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restrictions anticipated on importing goods from affected countries considered</td>
<td>Most European countries referred to restrictions on poultry imports with regard to this item. However this specific issue is not relevant to NZ as live poultry imports are already illegal.</td>
</tr>
<tr>
<td>Plans to secure pre-purchase agreement with vaccine companies for the supply of pandemic strain vaccine</td>
<td>This item was considered less definite than the subsequent framework item which was focused on instead (i.e. if a secured pre-purchase agreement had been made).</td>
</tr>
<tr>
<td>Provisions to package active pharmaceutical ingredient in capsule described</td>
<td>This item was not considered relevant for NZ since the antiviral stockpile is of fully made-up capsules.</td>
</tr>
<tr>
<td>Country planning to stockpile antivirals</td>
<td>This item was not considered relevant since NZ already has a stockpile (instead the issue of the presence of a stockpile and its documentation was focused on in the framework used).</td>
</tr>
<tr>
<td>Antivirals planned for pre-exposure prophylaxis</td>
<td>This item was considered too similar to the preceding framework item which was focused on instead (i.e. “Antivirals planned for pre-exposure prophylaxis during a pandemic”).</td>
</tr>
</tbody>
</table>

Data abstraction and scoring—The data extraction tool developed and used by the authors of the European plan analysis9 could not be obtained from them. Therefore for the 29 European countries the combined data were abstracted from the three bar graphs in the published article.9 When compared with the numbers quoted in the text of this article, this abstraction process appeared to be 100% accurate (i.e. the scale of the bar graph was easily and accurately interpretable). The totals for all the countries were averaged within each domain (see the bottom row of Tables 2 to 4).

Under each of the categories in the framework, the NZ plan (including its associated appendices) was checked. This process involved a full reading of the plan and then systematic word searches of all the files that covered the full electronic version. A “yes” for content inclusion was scored as a “1.0”. Where the NZ plan was not entirely explicit in its coverage of a content area, this item was graded as “partly” and was scored as “0.5”. In a more stringent alternative approach the “partly” assessments were given a zero score.

Results

The NZ plan followed the general pattern of such plans having elements of both a strategic plan and an operational plan (i.e. 25/29 of the European plans had this pattern). In terms of page length, the NZ plan at 198 pages (with appendices) was the
fourth longest. Longer plans were those from Finland (202 pages), the Netherlands (59 pages plus 246 pages of appendices) and Switzerland (249 pages in three parts).

In terms of plan content on border control aspects, the NZ plan scored higher than the average European plan (8.0 vs 4.9 out of 10.0 respectively) (Table 2). Similarly, for the antiviral aspects (13.5 vs 10.6 out of 17.0) (Table 4). However, the NZ plan scored slightly lower for the vaccine aspects (4.5 vs 5.3 out of 11.0) (Table 3).

Table 2. Coverage of border control measures mentioned in the national pandemic preparedness plans (29 European nations and NZ)

<table>
<thead>
<tr>
<th>Measures detailed in the plan</th>
<th>For 29 European countries (N)</th>
<th>For NZ*</th>
<th>Comment with regard to the NZ plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1) Absolute ban on the entry of people arriving from affected areas.**</td>
<td>5</td>
<td>Yes (p139)</td>
<td>Border closure from affected areas is detailed in the NZ plan.</td>
</tr>
<tr>
<td>1.2) Selective restrictions on the entry of people arriving from affected areas.**</td>
<td>16</td>
<td>Yes (p139)</td>
<td>This option exists in the plan and may be combined with requiring time in quarantine for people from certain pandemic-affected areas.</td>
</tr>
<tr>
<td>1.3) Mentions following WHO recommendations on travel</td>
<td>16</td>
<td>No</td>
<td>The plan frequently refers to WHO but not specifically to following WHO travel recommendations. Nevertheless, the plan does detail the provision of advice to travellers (e.g. p26).</td>
</tr>
<tr>
<td>1.4) Information for travellers</td>
<td>22</td>
<td>Yes (p26)</td>
<td>The plan provides examples of travel advice health educational materials (p197).</td>
</tr>
<tr>
<td>1.5) Measures at borders for international travellers coming from or going to affected areas</td>
<td>21</td>
<td>Yes</td>
<td>See below on exit screening and entry screening.</td>
</tr>
<tr>
<td>1.6) Entry screening anticipated.**</td>
<td>17</td>
<td>Yes (p44)</td>
<td>The plan mentions “intense surveillance” at the border and “enhanced screening” by the Customs Service (p129). Nevertheless, there is no specific mention of “health declarations” (for entry screening).</td>
</tr>
<tr>
<td>1.7) Exit screening anticipated</td>
<td>10</td>
<td>Yes (p141)</td>
<td>The plan is quite detailed on exit screening (i.e. health declarations and temperature measurements).</td>
</tr>
<tr>
<td>1.8) Quarantine of passengers coming from suspected areas anticipated.**</td>
<td>11</td>
<td>Yes (p134)</td>
<td>This issue is detailed in the plan. (Furthermore, local level planning for quarantine around international airports has also been undertaken).</td>
</tr>
<tr>
<td>1.9) Measures for travellers on board international conveyances from affected areas</td>
<td>9</td>
<td>No</td>
<td>There is no mention in the plan of separation measures or provision of masks to passengers/crew. But the plan does have detailed information about mask use in other settings.</td>
</tr>
<tr>
<td>1.10) International cooperation with neighbouring countries explicit</td>
<td>14</td>
<td>Yes (p26)</td>
<td>The plan specifically states such cooperation (especially with Australia) in many places.</td>
</tr>
</tbody>
</table>

| Total score (out of 10 categories) | 4.9/10.0 (average) | 8.0/10.0 |

* Example pages for the NZ plan (i.e. in many cases there is additional detail in other parts of the plan).

** This measure is not actually in WHO guidelines and may even be advised against by WHO. Nevertheless, it was retained in the framework as it might actually be appropriate in some circumstances and for island nations which are likely to have better scope for successful border control (see also the Discussion).

In a more stringent alternative approach (where the “partly” assessments were given a zero score), the scores for vaccine aspects dropped further behind the average for the European plans (i.e. to 3.0 out of 11.0) (Table 3). For antiviral aspects the NZ plan score dropped to only just above the average for the European plans (i.e. to 11.0 out of 17.0) (Table 4).
Table 3. Coverage of vaccine strategy measures mentioned in national preparedness plans (29 European nations and NZ)

<table>
<thead>
<tr>
<th>Measures detailed in the plan</th>
<th>For 29 European countries (N)</th>
<th>For NZ</th>
<th>Comment with regard to the NZ plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1) A strategic plan for pandemic vaccination</td>
<td>28 Yes (p99)</td>
<td>The plan refers to vaccination in many places and states the pandemic vaccine will be ordered at the appropriate time (from an Australian manufacturer).</td>
<td></td>
</tr>
<tr>
<td>2.2) A strategic plan for pneumococcal vaccination in pandemic phase</td>
<td>11 No</td>
<td>There is no mention of pneumococcal vaccination in the plan.</td>
<td></td>
</tr>
<tr>
<td>2.3) A strategic plan to vaccinate the whole population</td>
<td>18 Partly (p169)</td>
<td>This is not entirely clear in the plan but it is probably implied by the statement: “until the population at large is protected by vaccination”.</td>
<td></td>
</tr>
<tr>
<td>2.4) Defined priority groups for influenza vaccination</td>
<td>26 Partly</td>
<td>The plan cross-refers to an associated online document on ethical issues, which gives some statements that support prioritisation towards health professionals and towards patients who would meet clinical criteria for treatment (e.g. “influenza vaccination”) during normal times. Appendix D of the plan also discusses issues around “reciprocity” and “fairness” which may inform the prioritisation issue. See the additional text in the Discussion section of this article.</td>
<td></td>
</tr>
<tr>
<td>2.5) Sizes of priority groups given or referenced</td>
<td>16 No</td>
<td>This is not detailed, even though some data are fairly readily available (i.e. the size of the health care workforce, the essential services workforce and numbers of people with chronic conditions such as diabetes—as per the latest NZ Health Survey11).</td>
<td></td>
</tr>
<tr>
<td>2.6) Provision of storage for vaccines described</td>
<td>12 No</td>
<td>The plan was written before a supply of pre-pandemic vaccine was subsequently imported into NZ.</td>
<td></td>
</tr>
<tr>
<td>2.7) Operational plan for the distribution of vaccines</td>
<td>14 No</td>
<td>The plan says this is still being developed: “The Ministry of Health is working on the logistics of mounting a mass immunisation campaign. This will be published as guidance for DHBs who will be tasked with operationalising such a campaign, if required” (p99).</td>
<td></td>
</tr>
<tr>
<td>2.8) Specifies which healthcare workers will administer vaccine</td>
<td>11 No</td>
<td>The role of district health boards (DHBs) is specified (p33), but not to the type of health worker level of detail.</td>
<td></td>
</tr>
<tr>
<td>2.9) Provisions of medical equipment (needles, syringes) to support vaccine administration</td>
<td>9 Yes (p99)</td>
<td>The plan states: “NZ has in store sufficient needles/syringes, sharps containers and other vaccination equipment and supplies to mount a mass vaccination campaign.”</td>
<td></td>
</tr>
<tr>
<td>2.10) Tender for H5N1 vaccine procurement</td>
<td>5 Partly (p99)</td>
<td>The plan states: “The purchase of a small quantity of vaccine effective against a clade of the H5N1 influenza virus is being considered. Such vaccines are still in development” (p99). (Since this time a stockpile of such vaccine has been purchased12).</td>
<td></td>
</tr>
<tr>
<td>2.11) Secured pre-purchase agreement with vaccine companies for the supply of pandemic strain vaccine</td>
<td>4 Yes (p99)</td>
<td>The plan refers to a pre-existing arrangement with an Australian manufacturer.</td>
<td></td>
</tr>
<tr>
<td>Total (out of 11 categories)</td>
<td>5.3/11.0 (average)</td>
<td>4.5/11.0</td>
<td>(Or 3.0/11.0 for the NZ plan using the alternative more stringent scoring approach).</td>
</tr>
</tbody>
</table>
Table 4. Coverage of antiviral drug strategy measures mentioned in national preparedness plans (29 European nations and NZ)

<table>
<thead>
<tr>
<th>Measures detailed in the plan</th>
<th>For 29 European countries (N)</th>
<th>For NZ</th>
<th>Comment with regard to the NZ plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1) Strategic plan for the use of antivirals</td>
<td>28 Yes (p172)</td>
<td></td>
<td>The plan refers to the use of antivirals as part of border control, for cluster control and for reducing morbidity and mortality from pandemic influenza (see Appendix I in the plan).</td>
</tr>
<tr>
<td>3.2) Specifies which antivirals will be used</td>
<td>22 Yes (p99)</td>
<td></td>
<td>Oseltamivir (Tamiflu) is the antiviral referred to. However, there is no mention of amantadine or zanamivir (Relenza).</td>
</tr>
<tr>
<td>3.3) Doses and duration of treatment recommended in the plan</td>
<td>20 Yes (p173)</td>
<td></td>
<td>See Appendix I in the plan.</td>
</tr>
<tr>
<td>3.4) Strategy for antiviral use in early containment</td>
<td>18 Yes (p47)</td>
<td></td>
<td>As part of intensive cluster control operations antivirals will be offered to close contacts (unless surveillance indicates clusters are already too widespread to attempt control).</td>
</tr>
<tr>
<td>3.5) Plans to use antivirals in people working with animals/birds during animal outbreak</td>
<td>20 Partly (p28)</td>
<td></td>
<td>This is probably implicit in the statement: “Ensure appropriate protection and training for animal workers and exposed humans (poultry and pigs most likely) to reflect WHO guidelines and NZ guidelines and legislation.”</td>
</tr>
<tr>
<td>3.6) Antivirals planned for treatment</td>
<td>28 Yes (p94)</td>
<td></td>
<td>The plan states that: “If and when the pandemic becomes more widespread within NZ, it is anticipated that antivirals will be reserved for the treatment of cases.” Elsewhere an antiviral distribution role is suggested for community-based assessment centres (CBACs)—“to those meeting agreed clinical criteria” (p97).</td>
</tr>
<tr>
<td>3.7) Antivirals planned for pre-exposure prophylaxis during a pandemic</td>
<td>22 Yes</td>
<td></td>
<td>See item 3.1 above on use in cluster control. In such settings antivirals can be used for people defined as contacts and for those living in a defined area where cases have occurred (and hence the antivirals will function as pre-exposure prophylaxis).</td>
</tr>
<tr>
<td>3.8) Antiviral use for treatment explicitly prioritized over that for prophylaxis</td>
<td>15 Yes (p170)</td>
<td></td>
<td>The plan states that: “in general, it is anticipated that National Reserve medication will be used for the early treatment of people who become ill, rather than for prophylaxis.” Also: “The NZ Pandemic Influenza Technical Advisory Group concurs with the WHO opinion that pre-exposure prophylaxis for the population at large, or for the workforce in general, is neither practical nor possible because of the very large volume of medication required. The use of National Reserve stocks in this way would unreasonably deprive many people of any chance of treatment should they become ill.”</td>
</tr>
<tr>
<td>3.9) Priority groups for treatment defined</td>
<td>19 Partly (p172)</td>
<td></td>
<td>The plan argues that it is not justified to be too specific at this point in time concerning prioritisation policies and criteria (p172). But it also states that “Should prioritisation become necessary, medication will be prioritised towards population groups that are suffering poorer outcomes in terms of morbidity and/or mortality AND who appear to be able to benefit most from antiviral medication, and to people who provide certain identified services and functions essential for effective direct pandemic responses.” (See the Discussion below for further comment).</td>
</tr>
<tr>
<td>3.10) Priority groups for prophylaxis defined</td>
<td>22 Yes</td>
<td></td>
<td>See the response to item 3.1 above (regarding border control and cluster control). Furthermore, the plan states that those on home quarantine may be on antiviral prophylaxis (p41).</td>
</tr>
<tr>
<td>3.11) Sizes of priority groups given or referenced</td>
<td>16 No</td>
<td></td>
<td>See responses to item 2.5 above.</td>
</tr>
<tr>
<td>3.12) Provisions for storage described</td>
<td>15 Yes (p168)</td>
<td></td>
<td>The plan states that: “supplies are stored in several locations in NZ, with ready-use supplies available to support a rapid response anywhere in the country within a few hours.”</td>
</tr>
</tbody>
</table>
Measures detailed in the plan | For 29 European countries (N) | For NZ | Comment with regard to the NZ plan
---|---|---|---
3.13) Operational distribution plan for antivirals described | 18 | Partly | It is implied that such details exist but they are not spelled out in great detail: “Prepare for the release of National Reserve volumes of antivirals, and consider pre-positioning bulk supplies” (p39). Also: “Release antivirals for use according to policy in border management operations” (p44).
3.14) Named centres for local distribution | 13 | Partly | See item 3.6 above regarding CBACs.
3.15) Requirement for prescription for antivirals | 11 | Yes (p173) | There is no mention of any plans to change this status in a pandemic setting.
3.16) Country documents existing stockpile of antivirals | 14 | Yes (p168) | The precise size of the national reserve is not stated in the plan but this has been made publicly available by the Ministry (i.e. 855,000 courses).
3.17) There is antiviral stockpile reserved specifically for early containment | 6 | Partly | It is planned to use some of the stockpile for cluster control (see item 3.4 above). However a set proportion of the stockpile has not been designated for this specific use (which might be appropriate given the advantages of a flexible response).
Total (out of 17 categories) | 10.6/17.0 (average) | 13.5/17.0 | (Or 11.0/17.0 for the NZ plan using the alternative more stringent scoring approach).

In addition to the scoring process, the plan comparison resulted in various gaps in the NZ plan being identified. These gaps are briefly outlined in the tables with the major ones being around priority groups (Tables 3 and 4) and a strategic plan for pneumococcal vaccination (Table 3).

**Discussion**

**Main findings and interpretation**—The use of this framework suggests that the version 16 (current) NZ pandemic plan is generally more detailed relative to the average European plan (for those that were also available in 2006). This finding is reassuring, especially considering that the framework used had somewhat of a European focus. That is it included countries that are generally both larger and wealthier per capita than NZ and which sometimes even had their own influenza vaccine production facilities (8/29) or actual plans to develop influenza vaccine production capacity (4/29).

Some also have their own capacity to produce antivirals and all at least have near neighbours with vaccine/pharmaceutical industrial capacity. Furthermore, the framework ignored various social distancing interventions that might have allowed the NZ plan to score relatively more highly (see the limitations subsection below). Another possible disadvantage for NZ was that some of its planning work was in supporting documents that were not considered in this analysis of plans (e.g. work on ethical issues and evaluations of the simulation exercises that have been conducted).

The relatively favourable scores for aspects of the NZ plan are perhaps not surprising given the number of revisions the plan has undergone (currently at version 16), and the fact that the plan has benefited from fairly rigorous testing over several years. New Zealand society may also be relatively good at such planning given its small size, the relatively simple system of government (no state/federal system), and the fact that it needs to plan for a relatively high occurrence of other natural disasters (particularly floods, earthquakes, and volcanic events).
Nevertheless, the NZ plan is not very explicit on the issues of priority groups for vaccination and antivirals (e.g. items 2.4 and 3.9). Even so, the work from the National Ethics Advisory Committee (NEAC)\(^\text{10}\) would appear to justify being more explicit around frontline health workers being a priority group for both vaccines and antivirals. The NEAC noted that: “Opinion polls in NZ have suggested strong public support for the idea that frontline health workers should receive priority access to antiviral medication. This suggests reciprocity is a shared public value…”. (The opinion poll was reported in 2006\(^\text{13}\)). Similarly, other NEAC statements around patients who meet clinical criteria for care in normal times can be interpreted as also favouring the use of vaccination and antivirals for those who have greatest need.

These population groups include those already eligible for fully-subsidised seasonal influenza vaccine on the grounds of increased risk of adverse health outcomes from influenza infection. Being more explicit at a planning stage on prioritisation issues may give additional reassurance to key workers and reduce absenteeism in times of crisis (e.g. as was seen among some healthcare workers during the SARS epidemic). While the flexibility to change approach in light of the particular characteristics of a new pandemic strain is desirable, the scientific community probably knows enough about pandemic and seasonal influenza to still make reasonably detailed plans in the pre-pandemic period.

Indeed, 21 out of 22 of the European plans that covered pre-exposure antiviral prophylaxis suggested that health care workers be recipients of these.\(^{9}\) Another study of 31 countries reported that 84% had identified at least one priority group (and that health care workers were identified in all of these).\(^{14}\) As well as most other countries with plans having identified priority groups for scarce resources such as vaccines and antivirals,\(^{15}\) a number of these groups are specifically listed in WHO guidelines (tabulated elsewhere\(^\text{15}\)). The most sophisticated prioritisation work to date may also help guide prioritisation efforts around different age groups based on years of life lost\(^{16}\) (albeit with potential adjustment as information on the new pandemic strain emerges). A recent major US report also highlights the need for an ethical framework to allow for prioritisation of antivirals.\(^\text{17}\)

In line with these arguments, the Ministry of Health appears to be considering some prioritisation for its use of \textit{pre-pandemic} vaccine. As stated on its website: “key front-line health workers and other front-line pandemic response personnel” are prioritised for this vaccine in its draft considerations.\(^\text{12}\) Prioritisation of antivirals is however far more complex given the trade-offs between potential use for the treatment of sick individuals at high risk of death and pre-exposure prophylaxis of front-line healthcare workers. The WHO recommends saving antiviral supplies for the former while Norway has explicitly prioritised antiviral prophylaxis for continuously exposed health care workers over treatment of sick patients so as to maintain a functioning health service.\(^9\)

A further possible weakness of the NZ plan is the lack of specific attention to pandemic planning around avoiding the further exacerbation of health inequalities for already disadvantaged populations (as raised in the international literature\(^\text{18}\)). The 1918 influenza pandemic had a disproportionately severe impact on Māori mortality\(^\text{19,20}\) and recent NZ research also indicates higher seasonal influenza hospitalisation rates among those in crowded housing and with young children.\(^{21}\)
Another area in the NZ plan that could be strengthened is including discussion of pneumococcal vaccination (item 2.2). There is some new evidence for the impact of pneumococcal vaccine on reducing invasive disease in those aged 65 years and over. An earlier Cochrane systematic review also identified a benefit for preventing invasive pneumococcal disease (but not the incidence of pneumonia or death in adults with or without chronic illness). Various authors have recently argued for considering this vaccine in pandemic planning.

Although NZ has taken an alternative path of stockpiling antibiotics, it could be that use of pre-pandemic pneumococcal vaccination is also worth considering and might provide additional reassurance (since it is less dependent on continued health service functioning). At least there could be evidence in the NZ plan that these issues have been given appropriate consideration by relevant experts.

Limitations of this analysis—This brief analysis has a number of limitations. A major one is that the framework used only focused on the domains of border control, vaccines, and antivirals. In particular the importance of quarantine appears to be under-rated in this framework and plans for various other non-pharmaceutical interventions are not considered at all: education strategies to reduce incoming traveller numbers; community restrictions such as school and workplace closures; media campaigns to promote hand hygiene and cough etiquette; strengthening surveillance systems; improving access to rapid diagnostic tests; promoting systems that allow remote diagnosis (e.g. via video-links over the Internet); contract tracing systems; and the promotion of voluntary sheltering (i.e. voluntary sequestration of healthy people to avoid exposure).

Furthermore, plans can also potentially have country-specific research agendas for pandemic preparedness and can demonstrate a “whole-of-government” approach to planning and these items are not in the framework used. If these issues had been considered in the framework, then the NZ plan may well have scored even higher relative to the average European plan since it does give consideration to many of these issues.

Another limitation of the framework used was that it does not weight variables according the country-specific characteristics (e.g. available resources such as vaccine production capacity mentioned above, and geographical isolation). For example, item 1.3 (mentioning following WHO recommendations on travel) was an item where the NZ plan scored “zero”. Yet this criterion might not be that appropriate for an island nation such as NZ which may wish to take a particularly rigorous approach to border control. Furthermore, WHO guidelines are fairly general and there is relatively little consideration of the special issues facing remote island nations in any WHO documents on pandemic influenza published to date.

Possibly the ideal is for there to be WHO pandemic plan assessment frameworks and guidelines that are more tailored to different types of countries and especially for island nations (given that islands may have more scope for border control and within-country control). Such frameworks could come with detailed scoring systems to allow more sophisticated approaches to quantifiable plan comparisons. This refinement would prevent the fairly crude approach to scoring undertaken in this article (i.e. where we could no obtain the data extraction tool used in the European analysis).
Earlier work by Mounier-Jack and Coker\textsuperscript{25} has been somewhat criticised for not including any site visits.\textsuperscript{26} These critics state that “the experience of the national assessments during country visits has demonstrated that looking at plans alone often gives an incomplete and sometimes misleading picture of a country’s state of preparedness.” These critics also noted that the pandemic plans in many of these European countries were rapidly superseded as a result of ongoing planning developments. These issues may also apply to some extent to this 2007 publication of European plans by Mounier-Jack et al that was used in this analysis. Nevertheless, this problem is largely unavoidable without conducting very much more expensive studies (with site visits) that are published quickly outside of the journal literature (i.e. without lengthy peer review and publishing processes).

Finally, the data extraction from the NZ plan for comparison purposes was done by only one person (NW) with no assessment for inter-observer reliability. Nevertheless, both authors do have a high level of familiarity with the content of this NZ plan as a result of ongoing research work around influenza control in recent years for different agencies.

**Possible implications for future NZ planning**—Based on the results and discussion above, the following specific issues could be addressed in the next (probably to appear in 2009) version of New Zealand’s pandemic plan. Some of these points may also be relevant for DHB level plans:

- Consider the addition of more explicit details on the priority groups for vaccination, antivirals (for both treatment and prophylaxis) and other potentially limited resources (e.g. personal protective equipment). These details could extend to the different types of health workers (e.g. hospital doctors and nurses, primary care health workers, ambulance staff) and the different types of essential workers (e.g. police, fire-fighters, border control workers, defence personnel). In addition it may be desirable to specifically mention those who are currently eligible for fully-subsidised influenza vaccine due to increased risk of adverse sequelae (e.g. those over 65 years, and those with chronic heart disease, chronic respiratory disease, asthma treated with inhaled steroids).

  Additional pandemic control provisions for the most needy New Zealanders could also be considered (e.g. those living in deprived areas, in crowded houses, and with large numbers of children in the house).

- Consider adding discussion of pneumococcal vaccination in the NZ plan (item 2.2 and discussed above).

- Consider various additions that are covered in the framework for border control (item 1.9) and with regard to vaccines (items 2.3, 2.5–2.8).

Furthermore, the NZ health sector could consider further studies of the pandemic plans of other countries (to see if additional lessons can be learnt), and could further expand its own pandemic research agenda.\textsuperscript{27,28} Possible priority plans are those from countries which have recently tested their plans in simulation exercises (e.g. Singapore, UK,\textsuperscript{29} France,\textsuperscript{30} and Australia\textsuperscript{31,32}).
Other reviews of pandemic plans from multiple countries\(^7,15\) and of sub-national plans (e.g. of US states\(^33\)) may also be worthy of further examination.

**Competing interests:** Both authors have undertaken past contract work on pandemic influenza epidemiology for the Ministry of Health, but have not participated in the drafting of the pandemic plan examined in this article. They have no other competing interests.

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**References:**

The epidemiology of cryptosporidiosis in New Zealand, 1997–2006

Saskia J Snel, Michael G Baker, Kamalesh Venugopal

Abstract

Aims New Zealand has a high incidence of cryptosporidiosis compared to other developed countries. This study aimed to describe the epidemiology of this disease in detail and to identify potential risk factors.

Methods We analysed anonymous cryptosporidiosis notification (1997–2006) and hospitalisation data (1996–2006). Cases were designated as “urban” or “rural” and assigned a deprivation level based on their home address. Association between disease rates and animal density was studied using a simple linear regression model, at the territorial authority level.

Results Over the 10-year period 1997–2006, the average annual rate of notified cryptosporidiosis was 22.0 cases per 100,000 population. The number of hospitalisations was equivalent to 3.6% of the notified cases. There was only 1 reported fatality. The annual incidence of infection appeared fairly stable, but showed marked seasonality with a peak rate in spring (September–November in New Zealand). The highest rates were among Europeans, children 0–9 years of age, and those living in low deprivation areas. Notification rates showed large geographic variations, with rates in rural areas 2.8 times higher than in urban areas, and with rural areas also experiencing the most pronounced spring peak. At the territorial authority (TA) level, rates were also correlated with farm animal density.

Conclusions Most transmission of Cryptosporidium in New Zealand appears to be zoonotic: from farm animals to humans. Prevention should focus on reducing transmission in rural setting, though more research is needed to identify which strategies are likely to be most effective in that environment.

Cryptosporidium is one of the most common causes of protozoan diarrhoea worldwide, and leads to significant morbidity and mortality in both the developing and developed world. Transmission is through the faecal-oral route following direct or indirect contact with the transmissive stages of the organism, including person-to-person, zoonotic, waterborne, foodborne, and airborne transmission.1

Cryptosporidium parvum and Cryptosporidium hominis (previously known as C. parvum genotype 1 or the human genotype) are the most commonly reported causes of human cryptosporidiosis. C. hominis appears to be strictly a human pathogen. The main reservoir of C. parvum is animals, especially cattle.2

The New Zealand environment contains large numbers of farm animals and widespread use of surface water as a drinking water source.3 Surface water is difficult to protect from contamination by animal faeces. It is therefore likely that zoonotic transmission of Cryptosporidium is an important source of human infection in
New Zealand, and a large contributor to the high incidence seen in this country. However, there is as yet no published evidence on the importance of this source.

This study aimed to describe the incidence and impact of cryptosporidiosis in New Zealand, and to identify potential risk factors and environmental exposures that might be contributing to the high rates. Considering the large number of farm animals in New Zealand, a special focus was on estimating the contribution of zoonotic transmission.

**Methods**

Data from the national notifiable disease surveillance system were analysed for the 10-year period 1997 to 2006. Cryptosporidiosis had become legally notifiable by diagnosing medical practitioners in mid-1996. The case definition requires a clinically compatible illness and detection of *Cryptosporidium parvum* oocysts in a faecal specimen. The Institute of Environmental Science and Research Ltd (ESR) collects national notification data under contract to the Ministry of Health.

In addition, data on cryptosporidiosis hospitalisations (principal diagnosis) for 1996 to 2006 were obtained from the New Zealand Health Information Service (NZHIS), which is part of the Ministry of Health. This condition has been separately coded as a cause of hospital admission from 1995 (ICD9CM code 136.8 from 1995 and ICD10AM code A07.2 from 1999 onwards). We also reviewed published annual summaries of outbreaks to obtain data on the number attributed to cryptosporidiosis.

To examine the potential role of environmental sources, notified and hospitalised cases were designated as urban or rural based on the Statistics New Zealand classification of the area unit in which they resided. Statistics NZ defines a seven-level urban/rural profile based on the size of the populations and their employment status. Three of the categories are urban (main urban areas, satellite urban areas, independent urban areas) and four are rural (rural areas with high urban influence, rural areas with moderate urban influence, rural areas with low urban influence, and highly rural/remote areas). The 2001 Census classified 85.7% of the New Zealand population as urban, and 14.2% as rural.

To examine potential zoonotic transmission, farm animal density (sheep, cattle, horses, and deer per hectare grassland) was determined at territorial authority (TA) level and compared to the rate of disease. These data came from Statistics New Zealand, which carries out an Agriculture Production Census every 5 years, with the most recent in 2002. The TA are the smallest area unit in which these agricultural data are provided.

The analyses were carried out in EpiInfo (version 3.4), Microsoft Excel, and SPSS software. Rates were calculated using population data from the 2001 census (the mid-point for the 10-year period covered by the analysis). Rates for ethnic groups (based on prioritised ethnicity), sex, and rural/urban areas were directly age standardised to the age distribution of the New Zealand population in 2001. Rate ratios (RR) and 95% confidence intervals (95% CI) were calculated. Trends in notification and hospitalisation rates over time and across geographic areas (TA) were compared.

**Results**

**Cryptosporidiosis notifications, hospitalisations, fatalities, and outbreaks**—Table 1 shows the number of notifications, hospitalisations, fatalities, and outbreaks of cryptosporidiosis in New Zealand for the 10-year period 1997–2006. Less than 3.6% of notified cases were hospitalised and only 1 fatality was reported over this period.
Table 1. Incidence of cryptosporidiosis notifications, hospitalisations, fatalities and outbreaks, 1997–2006

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number of cases</th>
<th>Incidence rate and proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notifications</td>
<td>8212 cases in 10 years</td>
<td>Annual rate of 22.0 per 100,000 population</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>293 cases in 10 years</td>
<td>Annual rate of 0.78 per 100,000 population</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Equivalent to 3.6% of notifications*</td>
</tr>
<tr>
<td>Fatalities</td>
<td>1 case in 10 years</td>
<td>0.01% case fatality</td>
</tr>
<tr>
<td>Outbreaks</td>
<td>130 outbreaks and 955 cases in 10 years</td>
<td>Equivalent to 11.6% of total notifications*</td>
</tr>
</tbody>
</table>

*Assumes all hospitalised and outbreak cases were also notified, so true value will be less.

Cryptosporidiosis incidence by year and month—Cryptosporidiosis became notifiable in July 1996, with 1997 providing the first full year of notification data. Notifications rose to a peak of 1208 cases in 2001 followed by a decline in incidence over the following 5 years (Figure 1). The incidence of hospitalised cases generally followed a similar trend to notified cases, again with a peak incidence in 2001, followed by a small decline. However, a consistent trend was undetectable. At the district health board level, cryptosporidiosis hospitalisation and notification numbers showed a moderately high correlation for the 10-year period with $R^2=0.67$ (data not shown).

Figure 1. Number of cryptosporidiosis notifications (1997–2006) and hospitalisations (1996–2006), by year
Figure 2 shows the number of notified cases by month from January 1997 to December 2006. There was a consistent annual peak in incidence in spring (September–November). This seasonality can also be seen in Appendix 1, which shows the rates and rate ratios by season. The risk for infection with *Cryptosporidium* was 4.92 times higher in spring compared with the risk in summer. A second peak in late summer/early autumn (February–April) was also present in some years, being particularly prominent in 1998, 1999, and 2001.

**Figure 2. Number of cryptosporidiosis notifications by month, January 1997 to December 2006**

![Graph showing the number of cryptosporidiosis notifications by month from January 1997 to December 2006.](image)

**Figure 3. Rate ratios of cryptosporidiosis notifications by grade of rurality (main urban area = reference value), average for 1996–2007**

![Graph showing rate ratios of cryptosporidiosis notifications by grade of rurality.](image)

**Cryptosporidiosis in urban and rural areas**—The rate of cryptosporidiosis was almost three times higher in rural areas (50.68/100,000) than in urban areas (17.22/100,000) (Appendix 1). The incidence of cryptosporidiosis showed a dose-
response relationship with increasing levels of rurality (Figure 3), for both notifications and hospitalisations (Appendix 1).

Over time, the rates in rural areas were consistently higher than in urban areas (Figure 4). Trends showed a similar pattern to that seen for total national data (Figure 1).

Figure 4. Cryptosporidiosis notification rate per 100,000, comparing urban and rural areas, by year, 1997–2006

Figure 5 shows the rate of notified cryptosporidiosis in rural and urban areas by month (annualised rate per 100,000 population). An annual peak in spring (September–November) is clearly visible in rural areas. Urban areas showed a small peak in spring as well. By contrast, the additional summer/autumn (February–April) peaks present in 1998, 1999, and 2001 were largely confined to urban areas.

Cryptosporidiosis incidence by territorial authority—Figure 6 shows rates of notified cryptosporidiosis by TA (divided into quintiles based on average annual rate per 100,000 population). The two highest quintiles (with darkest shading) were above the average national incidence rate (22.0 cases per 100,000 population). Particularly high rates were seen in Mackenzie District (115.68/100,000), Waimate District (98.62/100,000), and Clutha District (91.42/100,000), which are all situated in the South Island (data not shown).
Characteristics of people affected with cryptosporidiosis—Both notification and hospitalisation data showed highest cryptosporidiosis incidence in infants and young children (aged 0–4 years). The notification rate for children aged 0–4 years was 8.3 times higher than the reference group (those 20–29 years of age). Children 5–9 years also had a significant higher risk for cryptosporidiosis. There was no difference in notification rates between males and females, though hospitalisation rates were significantly higher for females (Figure 7 and Appendix 1).

The rate of cryptosporidiosis was highest among Europeans (21.94/100,000) compared with other ethnic groups. The rate of cryptosporidiosis was also highest in the lowest deprivation areas (35.16/100,000 for NZDep level 1-2) and decreased with increasing deprivation levels. This area-based index assigns a deprivation level on a decile scale, based on census derived measures. Deprivation index number 1 represents the least deprived population with index level 10 representing the most deprived population (Figure 7 and Appendix 1).

Cryptosporidiosis by self-reported risk factors—Table 2 shows the number and proportion of cases reporting specific exposures. Contact with farm animals was the most commonly reported risk factor (59.4%) followed by attending school or childcare (43.4%) and drinking or using untreated drinking water (38.7%). Overseas travel during the incubation period was relatively uncommon (5.7%).

Figure 5. Cryptosporidiosis notification rate per 100,000, comparing urban and rural areas, by month, January 1997–December 2006
Figure 6. Cryptosporidiosis notification rates by New Zealand territorial authority, average annual rate per 100,000 for 1997–2006
Table 2. Number and proportion of cryptosporidiosis cases reporting specific exposures during their incubation period, 1997–2006

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Number reporting information</th>
<th>Percentage reporting positive exposure (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Farm animals</td>
<td>4479</td>
<td>59.4</td>
</tr>
<tr>
<td>School, childcare</td>
<td>6430</td>
<td>43.4</td>
</tr>
<tr>
<td>Untreated drinking water</td>
<td>3697</td>
<td>38.7</td>
</tr>
<tr>
<td>Recreational water</td>
<td>4471</td>
<td>32.7</td>
</tr>
<tr>
<td>Faecal matter, vomit</td>
<td>4064</td>
<td>30.2</td>
</tr>
<tr>
<td>Other symptomatic case</td>
<td>4103</td>
<td>26.9</td>
</tr>
<tr>
<td>Food premise</td>
<td>2793</td>
<td>26.0</td>
</tr>
<tr>
<td>Sick animals</td>
<td>3519</td>
<td>25.0</td>
</tr>
<tr>
<td>Confirmed case</td>
<td>4925</td>
<td>12.4</td>
</tr>
<tr>
<td>Overseas</td>
<td>6273</td>
<td>5.7</td>
</tr>
</tbody>
</table>

Figure 7. Cryptosporidiosis notification rates by case characteristics, average annual rates per 100,000 for 1997–2006

Cryptosporidiosis related to farm animal density in territorial authorities—We regressed the average annual rate of cryptosporidiosis per 100,000 population in each TA in New Zealand with farm animal density in each TA. Farm animal density was expressed as number of farm animals (sheep, cattle, deer, horses) per hectare of grassland.

Farm animal data were obtained from the Statistics New Zealand’s Agricultural Production Census. We used the 2002 Census as this was closest to the mid-point of
the 10-year period of cryptosporidiosis data. Cryptosporidiosis showed a small positive correlation ($R^2=0.1965$) with farm animal density (Figure 8).

**Figure 8:** Cryptosporidiosis notification rate (average annual cases per 100,000 for 1997–2006) correlated with density of farm animals (beef, sheep, horses, and deer per hectare in 2002), by territorial authority

**Discussion**

This detailed analysis of the descriptive epidemiology of cryptosporidiosis, based on the first ten full years of notification data, found that this disease has a relatively high rate in New Zealand (22.0 cases per 100,000). The disease showed a marked dose-response relationship with rurality, a striking association with spring season in rural areas, and a small correlation with farm animal density. These observations are consistent with farm animal reservoirs acting as an important source of human infection.

The number of fatalities and hospitalisations due to cryptosporidiosis is not particularly high with only one death recorded since 1997 and less than 3.6% of cases hospitalised. However, the notification rate is higher than comparable countries, including Australia (15.8 per 100,000 in 2005), United Kingdom (8.5 per 100,000 in 2005), other European countries such as Germany (1.6 per 100,000 in 2005), and the United States (3.0 per 100,000 in 2005). The true population rate will be even higher than presented, because only a small proportion of cases will seek medical attention and provide specimens for laboratory testing.

Notification and hospitalisation data showed a consistent pattern over time. Higher awareness of the disease and improved laboratory testing procedures could provide an...
explanation for the rise up to 2001. The subsequent small decline could indicate reduced transmission, though it is too soon to draw any conclusions.

Previous studies reported that the most important risk factors for cryptosporidiosis worldwide are in order of importance: travelling, contact with persons with diarrhoea, contact with farm animals, swimming, toileting children, and drinking unboiled water. Notification data, hospitalisation data and self-reported risk factors in this study suggest living in rural areas and contact with farm animals are the most important risk factors in New Zealand, with only a small additional contribution from recent overseas travel.

High rates are found among infants and young children. This age distribution is common in developed countries. It is known that children are more susceptible to parasitic infections and that childcare is an important source of infection for cryptosporidiosis. Young children also have more regular visits to a doctor, which increases the chance of notification. It has also been suggested that asymptomatic carriage of C. hominis may be common in young children who then act as an important reservoir of infection.

The relatively high rates of cryptosporidiosis among Europeans are surprising, as Maori and Pacific people generally experience higher rates of infectious diseases in New Zealand. Notification data also showed an inverse relationship between deprivation and cryptosporidiosis rates. This is an unusual outcome, because high deprivation is a risk factor for several infectious diseases. These findings suggest that the differences across ethnic and socioeconomic groups are likely to be strongly affected by patterns of accessing medical care for gastroenteritis. These apparent differences in rates may therefore be a surveillance ‘artefact’.

Possibly the most distinctive feature of cryptosporidiosis epidemiology in New Zealand is the remarkable high rate in rural areas, with a strong and consistent dose-response relationship with increasing levels of rurality. A positive correlation with farm animal density suggests that these animals may be reservoirs responsible for the high rates of disease in rural areas. Cryptosporidiosis can therefore be considered an important zoonotic disease in New Zealand. Previous studies in New Zealand support this hypothesis.

A descriptive study of this type cannot identify the exact transmission mode of Cryptosporidium in rural areas, where it may come from direct contact with farm animals or contaminated environments, or indirectly through contamination of surface (drinking) water with animal faeces. Direct transmission of cryptosporidium from animals to humans has been demonstrated in New Zealand. An association between poor water quality and human cryptosporidiosis has also been found. The risk of Cryptosporidium ingress into drinking-water supplies is not associated with rainfall and river flow in New Zealand. However, it is still possible that drinking water is an important source of infection.

Our analysis cannot identify which farm animals are the most important reservoirs. The regions with the highest rates in the North Island contained high concentrations of cattle (both dairy and beef) which suggests that cattle are important reservoirs. By contrast, the highest rates were found in the South Island, which has significant rural
areas dominated by sheep farming suggesting these animals may also act as an important source of infection.

Human cryptosporidiosis in New Zealand showed a marked seasonal pattern, with the largest number of notifications occurring in spring. This timing coincides with the main calving season in early spring. Spring is also the most important lambing season in New Zealand. Young livestock appear to excrete large numbers of *Cryptosporidium* oocysts. Only calves less than 2 months of age are hosts for *C. parvum*. Many older cattle are infected by the non-zoonotic *C. bovis* rather than *C. parvum*. These observations support the role of young animals as important reservoirs for human infection.

In the early part of the observation period (particularly 1998, 1999, and 2001), higher cryptosporidium rates were also seen during late summer/early autumn in urban areas. This spatio-temporal pattern would be consistent with anthroponotic transmission of *C. hominis* through contaminated swimming pools during the swimming season, as shown in previous studies.

The publicity following a large cryptosporidiosis outbreak in 1998 may have contributed to improved regulations and filtration systems in public swimming pools resulting in the subsequent disappearance of this ‘swimming pool peak’ after 2001.

New Zealand’s relatively high rate of cryptosporidiosis suggests that this public health problem deserves further attention. The symptoms of cryptosporidiosis (diarrhoea, abdominal pain) cause affected people considerable discomfort. The disease also results in high economic costs. Infectious intestinal diseases are estimated to cause up to 823,000 cases of illness per year in New Zealand. The economic cost of these illnesses was estimated in 2000 as approximately NZ$216 million.

Based on an estimated cost per case of $599 (updated average cost of a case of intestinal infectious disease from $462 in May 1999) and 821 cases per year, cryptosporidiosis results in economic costs of approximately NZ$492,000 per year (the true economic cost would be many times higher than this if non-notified cases were also included).

Prevention of cryptosporidiosis should focus on reducing transmission in the rural environment, particularly from farm animals to humans during spring. Such measures could include general advice about hand washing after contact with farm animals and contaminated environments.

Children should receive special attention, because disease risk is concentrated in this group (over half of notifications and hospitalisations are <10 years of age). Caregivers of children living in rural areas could be targeted during the spring period when disease risk is highest.

This analysis has the limitations associated with use of routinely collected surveillance data. In general, the disease estimates in this paper are highly conservative. It is well recognised that notified cases of enteric disease are only a small proportion of total disease occurring in the community. A recent acute gastrointestinal illnesses (AGI) study in New Zealand estimated that only 0.5% of community cases of AGI, and approximately 2.0% of cases that visited a GP, were eventually notified. Our analysis of hospitalisation numbers only included cases...
where cryptosporidiosis was recorded as the principal diagnosis, which would have significantly underestimated numbers.

Descriptive data on their own have limited ability to identify sources of infection. There is considerable potential to extend the simple univariate analyses presented here. A multivariate model could, for example, help to identify the separate contribution of ethnicity and deprivation to the observed cryptosporidiosis rates. Spatial analysis could also investigate how this disease was related to animal density, drinking water quality and other exposures of interest at a much finer level of spatial resolution, such as the census area unit.

The higher rate of cryptosporidiosis in rural areas deserves further investigation. Such research could focus on identifying the modes of transmission in this setting, particularly the relative importance of direct contact with farm animals and contaminated environments, and the role of contaminated drinking water. This research could also assess the contribution of both cattle and sheep as sources of disease transmission. The results of these studies could identify measures to reduce the high rates of cryptosporidiosis in New Zealand.

**Competing interests:** None known.

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**References:**


Appendix 1. Cryptosporidiosis notification and hospitalisation numbers and rates (average annual rate per 100,000 population), by season, rural-urban domicile, age group, sex, ethnicity, and deprivation level, 1997–2006, New Zealand

<table>
<thead>
<tr>
<th>Category</th>
<th>Notified</th>
<th>Rate</th>
<th>RR (95% CI)</th>
<th>Hospitalised</th>
<th>Rate</th>
<th>RR (95% CI)</th>
<th>Hospital proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Season</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Summer (Dec-Feb)</td>
<td>92</td>
<td>9.86</td>
<td><strong>1.00</strong></td>
<td>3</td>
<td>0.35</td>
<td><strong>1.00</strong></td>
<td>3.3%</td>
</tr>
<tr>
<td>Autumn (Mar-May)</td>
<td>165</td>
<td>17.62</td>
<td>1.79 (1.648 – 1.936)</td>
<td>3</td>
<td>0.33</td>
<td>0.94 (0.575 – 1.534)</td>
<td>1.8%</td>
</tr>
<tr>
<td>Winter (Jun-Aug)</td>
<td>110</td>
<td>11.74</td>
<td>1.19 (1.091 – 1.300)</td>
<td>4</td>
<td>0.46</td>
<td>1.30 (0.828 – 2.051)</td>
<td>3.6%</td>
</tr>
<tr>
<td>Spring (Sep-Nov)</td>
<td>455</td>
<td>48.68</td>
<td>4.92 (4.583 – 5.280)</td>
<td>19</td>
<td>1.99</td>
<td>5.64 (3.892 – 8.160)</td>
<td>4.2%</td>
</tr>
<tr>
<td><strong>Urban-rural</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban Total</td>
<td>552</td>
<td>17.22</td>
<td><strong>1.00</strong></td>
<td>20</td>
<td>0.62</td>
<td><strong>1.00</strong></td>
<td>3.8%</td>
</tr>
<tr>
<td>Rural Total</td>
<td>270</td>
<td>50.68</td>
<td>2.84 (2.658 – 3.029)</td>
<td>9</td>
<td>1.67</td>
<td>2.68 (2.088 – 3.440)</td>
<td>3.0%</td>
</tr>
<tr>
<td>Main urban</td>
<td>424</td>
<td>15.98</td>
<td><strong>1.00</strong></td>
<td>10</td>
<td>0.37</td>
<td><strong>1.00</strong></td>
<td>2.5%</td>
</tr>
<tr>
<td>Satellite urban</td>
<td>22</td>
<td>19.63</td>
<td>1.20 (1.099 – 1.302)</td>
<td>0</td>
<td>0.36</td>
<td>0.97 (0.356 – 2.625)</td>
<td>0%</td>
</tr>
<tr>
<td>Independent urban</td>
<td>106</td>
<td>24.13</td>
<td>1.58 (1.401 – 1.775)</td>
<td>10</td>
<td>2.22</td>
<td>5.94 (4.491 – 7.862)</td>
<td>10.9%</td>
</tr>
<tr>
<td>Rural, high rural</td>
<td>34</td>
<td>35.39</td>
<td>2.18 (2.094 – 2.276)</td>
<td>1</td>
<td>0.73</td>
<td>1.96 (0.910 – 4.217)</td>
<td>3.0%</td>
</tr>
<tr>
<td>Rural, moderate</td>
<td>63</td>
<td>46.56</td>
<td>2.88 (2.776 – 2.998)</td>
<td>2</td>
<td>1.18</td>
<td>3.17 (1.870 – 5.377)</td>
<td>2.9%</td>
</tr>
<tr>
<td>Rural, low urban</td>
<td>125</td>
<td>55.53</td>
<td>3.32 (3.189 – 3.462)</td>
<td>5</td>
<td>2.36</td>
<td>6.33 (4.537 – 8.840)</td>
<td>3.3%</td>
</tr>
<tr>
<td>Highly rural/remote</td>
<td>48</td>
<td>62.92</td>
<td>3.67 (3.594 – 3.755)</td>
<td>1</td>
<td>1.70</td>
<td>4.56 (2.558 – 8.128)</td>
<td>2.4%</td>
</tr>
<tr>
<td><strong>Age group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–4</td>
<td>369</td>
<td>136.15</td>
<td>8.30 (7.686 – 8.961)</td>
<td>10</td>
<td>3.66</td>
<td>5.93 (3.941 – 8.920)</td>
<td>2.5%</td>
</tr>
<tr>
<td>5–9</td>
<td>124</td>
<td>43.47</td>
<td>2.67 (2.440 – 2.923)</td>
<td>6</td>
<td>2.10</td>
<td>3.40 (2.194 – 5.271)</td>
<td>4.8%</td>
</tr>
<tr>
<td>10–14</td>
<td>52</td>
<td>17.89</td>
<td>1.10 (0.988 – 1.232)</td>
<td>2</td>
<td>0.72</td>
<td>1.17 (0.671 – 2.047)</td>
<td>3.8%</td>
</tr>
<tr>
<td>15–19</td>
<td>36</td>
<td>13.50</td>
<td>0.83 (0.735 – 0.943)</td>
<td>1</td>
<td>0.34</td>
<td>0.55 (0.261 – 1.159)</td>
<td>2.8%</td>
</tr>
<tr>
<td>20–29</td>
<td>79</td>
<td>16.21</td>
<td><strong>1.00</strong></td>
<td>3</td>
<td>0.62</td>
<td><strong>1.00</strong></td>
<td>3.8%</td>
</tr>
<tr>
<td>30–39</td>
<td>84</td>
<td>14.55</td>
<td>0.90 (0.814 – 0.989)</td>
<td>3</td>
<td>0.45</td>
<td>0.73 (0.433 – 1.236)</td>
<td>3.6%</td>
</tr>
<tr>
<td>40–49</td>
<td>40</td>
<td>7.48</td>
<td>0.46 (0.410 – 0.521)</td>
<td>2</td>
<td>0.30</td>
<td>0.48 (0.263 – 0.886)</td>
<td>5.0%</td>
</tr>
<tr>
<td>50–59</td>
<td>19</td>
<td>4.56</td>
<td>0.28 (0.241 – 0.330)</td>
<td>2</td>
<td>0.36</td>
<td>0.58 (0.313 – 1.081)</td>
<td>10.5%</td>
</tr>
<tr>
<td>60–69</td>
<td>12</td>
<td>4.07</td>
<td>0.25 (0.207 – 0.306)</td>
<td>1</td>
<td>0.32</td>
<td>0.52 (0.245 – 1.089)</td>
<td>8.3%</td>
</tr>
<tr>
<td>70+</td>
<td>7</td>
<td>2.08</td>
<td>0.13 (0.100 – 0.165)</td>
<td>1</td>
<td>0.25</td>
<td>0.40 (0.184 – 0.878)</td>
<td>14.3%</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Male</td>
<td>403</td>
<td>22.08</td>
<td><strong>1.00</strong></td>
<td>12</td>
<td>0.65</td>
<td><strong>1.00</strong></td>
<td>3.0%</td>
</tr>
<tr>
<td>Female</td>
<td>410</td>
<td>21.43</td>
<td>1.04 (0.858 – 1.250)</td>
<td>17</td>
<td>0.91</td>
<td>1.39 (1.103 – 1.758)</td>
<td>4.1%</td>
</tr>
<tr>
<td>Unknown</td>
<td>8</td>
<td>na</td>
<td>na</td>
<td>0</td>
<td>na</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>629</td>
<td>21.94</td>
<td><strong>1.00</strong></td>
<td>24</td>
<td>0.83</td>
<td><strong>1.00</strong></td>
<td>3.8%</td>
</tr>
<tr>
<td>Maori</td>
<td>56</td>
<td>10.62</td>
<td>0.27 (0.205 – 0.347)</td>
<td>4</td>
<td>0.68</td>
<td>0.51 (0.19 – 1.00)</td>
<td>7.1%</td>
</tr>
<tr>
<td>Pacific</td>
<td>8</td>
<td>3.54</td>
<td>0.11 (0.080 – 0.157)</td>
<td>0</td>
<td>0.13</td>
<td>0.09 (0.0 – 1.00)</td>
<td>0%</td>
</tr>
<tr>
<td>Other</td>
<td>22</td>
<td>8.46</td>
<td>0.27 (0.225 – 0.336)</td>
<td>2</td>
<td>0.61</td>
<td>0.54 (0.0 – 1.00)</td>
<td>9.1%</td>
</tr>
<tr>
<td>Unknown</td>
<td>106</td>
<td>na</td>
<td>na</td>
<td>0</td>
<td>na</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td><strong>Deprivation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–2</td>
<td>2,363</td>
<td>35.16</td>
<td><strong>1.00</strong></td>
<td>4</td>
<td>0.61</td>
<td><strong>1.00</strong></td>
<td>1.9%</td>
</tr>
<tr>
<td>3–4</td>
<td>2,112</td>
<td>30.05</td>
<td>0.85 (0.806 – 0.906)</td>
<td>6</td>
<td>0.91</td>
<td>1.49 (1.008 – 2.209)</td>
<td>2.6%</td>
</tr>
<tr>
<td>5–6</td>
<td>1,496</td>
<td>20.27</td>
<td>0.58 (0.541 – 0.616)</td>
<td>5</td>
<td>0.68</td>
<td>1.11 (0.735 – 1.678)</td>
<td>3.6%</td>
</tr>
<tr>
<td>7–8</td>
<td>1,251</td>
<td>15.66</td>
<td>0.45 (0.417 – 0.478)</td>
<td>8</td>
<td>0.95</td>
<td>1.56 (1.067 – 2.280)</td>
<td>6.5%</td>
</tr>
<tr>
<td>9–10</td>
<td>984</td>
<td>12.78</td>
<td>0.36 (0.338 – 0.392)</td>
<td>6</td>
<td>0.74</td>
<td>1.21 (0.812 – 1.812)</td>
<td>5.0%</td>
</tr>
<tr>
<td>Total</td>
<td>821</td>
<td>22.0</td>
<td>na</td>
<td>29</td>
<td>0.78</td>
<td>na</td>
<td>3.6%</td>
</tr>
</tbody>
</table>

1. Number is the average annual number rounded to the nearest integer.
2. Rate is the average annual rate per 100,000 population.
3. RR = Rate ratio calculated in relation to reference value in bold, 95% CI = 95% confidence interval calculated based on 10-year period.
4. Proportion hospitalised is based on recorded hospitalisations expressed as a percentage of number notified.
5. Notification rates for ethnic groups, sex and urban-rural distribution were directly age standardised to the age distribution of the New Zealand population at the 2001 Census with confidence intervals calculated according to the methods used for age standardised data. Hospitalisation numbers were too low for age standardising, which led to wide confidence intervals. Only the age-standardised RRs for ethnicity were significantly different compared to the crude RR, and therefore we used the crude RR for sex and urban-rural distribution and age-standardised RR only for ethnicity.
The epidemiology of giardiasis in New Zealand, 1997–2006

Saskia J Snel, Michael G Baker, Kamalesh Venugopal

Abstract

Aims New Zealand has a higher incidence rate of giardiasis than other developed countries. This study aimed to describe the epidemiology of this disease in detail and to identify potential risk factors.

Methods We analysed anonymous giardiasis notification (1997–2006) and hospitalisation data (1990–2006). Cases were designated as urban or rural and assigned a deprivation level based on their home address. Association between disease rates and animal density was studied using a simple linear regression model, at the territorial authority (TA) level.

Results Over the 10-year period 1997–2006 the average annual rate of notified giardiasis was 44.1 cases per 100,000 population. The number of hospitalisations was equivalent to 1.7% of the notified cases. There were 2 reported fatalities. The annual incidence of notified cases declined over this period whereas hospitalisations remained fairly constant. Giardiasis showed little seasonality. The highest rates were among children 0–9 years old, those 30–39 years old, Europeans, and those living in low deprivation areas. Notification rates were slightly higher in rural areas. The correlation between giardiasis and farm animal density was not significant at the TA level.

Conclusions The public health importance of giardiasis to New Zealand mainly comes from its relatively high rates in this country. The distribution of cases is consistent with largely anthroponotic (human) reservoirs, with a relatively small contribution from zoonotic sources in rural environments and a modest contribution from overseas travel. Prevention efforts could include continuing efforts to improve hand washing, nappy handling, and other hygiene measures and travel health advice relating to enteric infections.

*Giardia* is one of the most common causes of protozoan diarrhoea worldwide, and leads to significant morbidity and mortality in both the developing and developed world. Transmission is through the faecal-oral route following direct or indirect contact with the transmissive stages of the organism, including person-to-person, zoonotic, waterborne, foodborne, and airborne transmission.\(^1\)

*Giardia duodenalis* (synonyms: *Giardia lamblia, Giardia intestinalis*) is the only species found in humans and the majority of domestic and wild mammals.\(^2\) There is extensive genetic variability within *G. duodenalis*. Genotype A and B, now widely referred as Assemblage A and B, are the only genotypes which include humans in their host range.\(^3\)

The New Zealand environment contains large numbers of farm animals and widespread use of surface water as a drinking water source.\(^4\) Surface water is difficult to protect from contamination by animal faeces. Zoonotic transmission from farm
animals would be a likely source of infection and may be contributing to New Zealand’s high rates of disease. However, the importance of such reservoirs is not known.

This study aimed to describe the incidence and impact of giardiasis in New Zealand, and to identify potential risk factors and environmental exposures that might be contributing to the high rates. Considering the large numbers of farm animals in New Zealand, a special focus was on estimating the contribution of zoonotic transmission.

Methods

Data from the national notifiable disease surveillance system were analysed for the period 1997 to 2006. Giardiasis became legally notifiable by diagnosing medical practitioners in mid-1996. The case definition requires a clinically compatible illness and detection of *Giardia* cysts, trophozoites, or antigen in faeces. The Institute of Environmental Science and Research Ltd (ESR) collects national notification data under contract to the Ministry of Health.

In addition, data on giardiasis hospitalisations (principal diagnosis) from 1990 to 2005 were obtained from the New Zealand Health Information Service (NZHIS), which is part of the Ministry of Health. This condition is coded as a cause of hospital admission (ICD9CM code 007.1 and ICD10AM code A07.1 from 1999 onwards). We also reviewed published annual summaries of outbreaks to obtain data on the number attributed to giardiasis.

To examine the potential role of environmental sources, notified and hospitalised cases were designated as urban or rural (based on the Statistics NZ classification of the area unit in which they resided). Statistics NZ defines a seven-level urban/rural profile based on the size of the populations and their employment status. Three categories are urban and four are rural. The 2001 Census classified 85.7% of the population as urban, and 14.2% as rural.

To examine potential zoonotic transmission, farm animal density (sheep, cattle, horses, and deer per hectare grassland) was calculated for each territorial authority (TA) and compared to the rate of disease. These data came from Statistics New Zealand, which carries out an Agriculture Production Census every 5 years, with the most recent in 2002. The TA level is the smallest area unit in which these agricultural data are provided.

The analyses were carried out in Epi Info (version 3.4), Microsoft Excel, and SPSS software. Rates were calculated using population data from the 2001 census (the mid-point for the 10-year period covered by the analysis). Rates for ethnic groups (based on prioritised ethnicity) and sex were directly age standardised to the age distribution of the New Zealand population in 2001. Rate ratios (RR) and 95% confidence intervals (95% CI) were calculated. Trends in notification and hospitalisation rates over time and across geographic areas (TA) were compared.

Results

Giardiasis notifications, hospitalisations, fatalities, and outbreaks—Table 1 shows the number of notifications, hospitalisations, fatalities and outbreaks of giardiasis in New Zealand for the 10-year period 1997-2006. Less than 1.7% of notified cases were hospitalised and only 2 fatalities were reported over this period.
Table 1. Incidence of giardiasis notifications, hospitalisation, fatalities, and outbreaks, 1997–2006

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number of cases</th>
<th>Rate and proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notifications</td>
<td>16,471 cases in 10 years</td>
<td>Annual rate of 44.1 per 100,000 population</td>
</tr>
<tr>
<td>Hospitalisations</td>
<td>278 cases in 10 years</td>
<td>Annual rate of 0.74 per 100,000 population Equivalent to 1.7% of notifications*</td>
</tr>
<tr>
<td>Fatalities</td>
<td>2 cases in 10 years</td>
<td>0.01% case fatality</td>
</tr>
<tr>
<td>Outbreaks</td>
<td>234 outbreaks and 1037 cases in 10 years</td>
<td>Equivalent to 6.3% of total notifications*</td>
</tr>
</tbody>
</table>

*Assumes all hospitalised and outbreak cases were also notified, so true value will be less

Giardiasis incidence by year and month— Giardiasis became notifiable in July 1996, with 1997 providing the first full year of notification data. Notification declined significantly over the 10 years from 1997 to 2006 (Chi-squared test for trend, p<0.001) (Figure 1). By contrast, hospitalisation numbers were relatively constant over the 17-year period 1990 to 2006. At the district health board level, giardiasis hospitalisation and notification number showed no correlation with each other (R²=0.06, data not shown).

Notified cases by month (Figure 2) showed a moderate peak every year in late summer/early autumn (January – April) from 1997 until 1999, but this pattern largely disappeared in subsequent years.

Figure 1. Number of giardiasis notifications (1997–2006) and hospitalisations (1990–2006), by year
Giardiasis in rural and urban areas—The rate of giardiasis was 23% higher in rural areas (53.20/100,000) than in urban areas (42.58/100,000) (Appendix 1). However, there was not a consistent dose-response relationship with increasing levels of rurality (Figure 3).
Over time, the rates in rural areas were consistently higher than in urban areas (Figure 4). Both urban and rural rates showed a similar downward trend in incidence (Figure 1).

Figure 4. Giardiasis notification rate per 100,000, comparing urban and rural areas, by year 1997–2006

Figure 5 shows the rate of notified giardiasis in rural and urban areas by month (annualised rate per 100,000 population). This analysis suggests that the small late summer/early autumn (January–April) peak evident from 1997 until 1999 had contributions from both urban and rural sources.

Giardiasis incidence by territorial authority—Figure 6 shows rates of notified giardiasis by TA (divided into quintiles based on average annual notification rate per 100,000 population). The two highest quintiles (with darkest shading) were above the average national incidence rate (44.1 per 100,000 population).

In the North Island, the highest rates were seen in the Waikato and Matamata-Piako District (79.41/100,000), Hastings District (67.63/100,000), and around Wellington (71.54/100,000) (data not shown). In the South Island, high rates were in the Buller District (60.27/100,000), Grey District (65.94/100,000), and in Queenstown-Lakes District (61.61/100,000) (data not shown).
Characteristics of people affected with giardiasis—The incidence of giardiasis was highest in infants and children aged 0–4 years (147.01/100,000) and 5–9 years (47.66/100,000) with another peak in the 30–39 year age group (69.63/100,000). The notification rate was slightly higher in males than in females (Figure 7 and Appendix 1).

The rate of giardiasis was higher among Europeans (39.30/100,000) and Other ethnicities (37.65/100,000) compared with Maori and Pacific rates. The rate of giardiasis was also highest in the lowest deprivation areas (65.57/100,000 for NZDep level 1-2) and decreased with increasing deprivation levels. This area-based index assigns a deprivation level on a decile scale, based on census derived measures. Deprivation index number 1 represents the least deprived population with index level 10 representing the most deprived population (Figure 7 and Appendix 1).7

Giardiasis by self-reported risk factors—Table 2 shows the number and proportion of case reporting specific exposures. Faecal matter and vomit (40.0%), drinking untreated drinking water (35.3%), and contact with other symptomatic cases (34.9%) were the most common self-reported positive exposures. Overseas travel during the incubation period was reported far more commonly (19.1%) than would be expected.

Giardiasis related to farm animal density in territorial authorities—We regressed average annual rate of giardiasis per 100,000 population in each TA in New Zealand with farm animal density in each TA. Farm animal data were obtained from Statistics New Zealand’s Agricultural Production Census. We used the 2002 Census as this was closest to the mid-point of the 10-year period of giardiasis data. There was no correlation between the number of farm animals per hectare of grassland and the rate of giardiasis per 100,000 population in each TA (R^2=0.0643, data not shown)
Figure 6. Giardiasis notification rates by New Zealand territorial authorities, average annual rate per 100,000 for 1997–2006
Table 2. Number and proportion of giardiasis cases reporting specific exposures during the incubation period, 1997–2006

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Number reporting information</th>
<th>Percentage reporting positive exposure (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faecal matter, vomit</td>
<td>3883</td>
<td>40.0</td>
</tr>
<tr>
<td>Untreated drinking water</td>
<td>3548</td>
<td>35.3</td>
</tr>
<tr>
<td>Other symptomatic case</td>
<td>3996</td>
<td>34.9</td>
</tr>
<tr>
<td>Recreational water</td>
<td>4679</td>
<td>32.8</td>
</tr>
<tr>
<td>Confirmed case</td>
<td>6856</td>
<td>29.3</td>
</tr>
<tr>
<td>School, childcare</td>
<td>8709</td>
<td>27.9</td>
</tr>
<tr>
<td>Food premise</td>
<td>3070</td>
<td>26.4</td>
</tr>
<tr>
<td>Farm animals</td>
<td>4138</td>
<td>26.5</td>
</tr>
<tr>
<td>Overseas</td>
<td>9200</td>
<td>19.1</td>
</tr>
</tbody>
</table>

Figure 7. Giardiasis notification rates by case characteristic, average annual rate per 100,000 for 1996–2006

Discussion

This detailed analysis of the descriptive epidemiology of giardiasis, based on the first ten full years of notification data, showed that this disease has a relatively high rate in New Zealand (44.1 cases per 100,000). The data are consistent with largely anthropogenic transmission, with a relatively small contribution from zoonotic reservoirs in rural environments and a modest contribution from overseas travel.
There were only 2 fatalities recorded as a result of giardiasis in 10 years, and hospitalisations were less than 1.7% of the notified cases. However, the rate of disease in New Zealand is higher than comparable countries: United Kingdom (5.5 per 100,000 in 2005), Germany (5.5 per 100,000 in 2005), and the United States (7.1 per 100,000 in 2005). The true population rate is likely to be even higher than notified cases suggest, because only a small proportion of cases will seek medical attention and provide specimens for laboratory testing.

Giardiasis notifications declined significantly from 1997 to 2006, whereas hospitalisations remained relatively constant. Because these trends are not consistent it is difficult to draw conclusions about changes in risk over time.

The most important risk factors for giardiasis described in the literature are in order of importance: travelling abroad, swallowing water while swimming, being European or Asian, exposure to surface water or fresh recreational water, exposure to human waste, eating lettuce, and being a housewife or a nursing mother. Most of these risk factors are related to anthropotic transmission.

Notable self-reported exposures in our analysis were mainly anthropotic: contact with faecal matter or vomit (40.0%), use of untreated drinking water (35.3%) and contact with other symptomatic cases (34.9%). Almost 20% of cases reported overseas travel during the incubation period confirming the importance of this source of exposure.

The descriptive epidemiology did not suggest a large contribution from zoonotic sources. It is known that G. duodenalis is carried by adult animals (domestic and farm animals) all year round, which may explain the absence of seasonality in human giardiasis. However, only 4.2% of cases reported contact with sick animals during their incubation period. Rates of giardiasis were only slightly elevated in rural areas. In addition, giardiasis rates were not correlated with animal density. Other research has also concluded that zoonotic transmission from farm animals to humans is not particularly important.

The high rates among infants and young children (0–4 years) were not surprising. This age distribution is common to other developed countries. It is known that infants and young children are more susceptible for parasitic infections. Young children also have regular visits to a general practitioner, which results in a higher chance of notification. The peak in young adults (30–39 year) has also been reported in the literature. This age group is more likely to have contact with young children, as parents and/or caregivers, and is for this reason more often involved in nappy handling. Contact with young children and nappies are known risk factors for giardiasis.

The relatively high rates among Europeans are more surprising, as Maori and Pacific people generally experience higher rates of infectious diseases in New Zealand. Additionally, notification data showed an inverse relationship between deprivation and giardiasis rates. This is also an unusual outcome, because high deprivation is a risk factor for most infectious diseases. These two findings suggest that the notification rates across ethnic and socioeconomic groups are likely to be strongly affected by patterns of accessing medical care for gastroenteritis. Apparent differences in rates may therefore be a surveillance ‘artefact’.
Previous studies of giardiasis in New Zealand reported seasonal patterns, with a late summer/early autumn peak (March/April).\textsuperscript{13,15,25,37} We only found a slightly higher risk during autumn (RR 1.17). This pattern would be consistent with transmission via swimming pool outbreaks, as has been demonstrated for cryptosporidiosis in New Zealand.\textsuperscript{38}

New Zealand’s relatively high rates of giardiasis suggest that this public health problem deserves further attention. The symptoms of giardiasis (diarrhoea, abdominal pain) cause people considerable discomfort. The disease also results in high annual economic costs. Infectious intestinal disease has been estimated to cause up to 823,000 cases of illness per year in New Zealand.\textsuperscript{39} The economic cost of these illnesses was estimated in 2000 as approximately NZ$216 million.\textsuperscript{40}

Based on an estimated cost per case of $599 (updated average cost of a case of intestinal infectious disease from $462 in May 1999\textsuperscript{40}) and 1647 cases per year, this disease results in economic costs of approximately NZ$987,000 per year. The true economic cost would be many times higher than this if non-notified cases were also included.

Prevention of giardiasis should focus on measures to reduce person-to-person transmission. Based on existing evidence, such measures could include continuing efforts to improve hand washing, nappy handling, and other hygiene measures. Travel health advice relating to enteric infections may also be useful. Continuing Ministry of Health efforts to improve the quality of drinking water may also reduce rates of giardiasis in New Zealand.\textsuperscript{41,42}

This analysis has the limitations associated with use of routinely collected surveillance data. It is well recognised that notified cases of enteric disease are only a small proportion of total disease occurring in the community. A recent acute gastrointestinal Illnesses (AGI) study in New Zealand estimated that only 0.5% of community cases of AGI, and approximately 2.0% of cases that visited a GP, were eventually notified.\textsuperscript{43} Our analysis of hospitalisation numbers only included cases where giardiasis was recorded as the principal diagnosis, which would have significantly underestimated numbers.

Descriptive data on their own have limited ability to identify the likely sources of infection. There is considerable potential to extend the simple univariate analyses presented here. A multivariate model could, for example, help to identify the separate contribution of ethnicity and deprivation to the observed giardiasis rates. Spatial analysis could investigate associations with other exposures (such as drinking water quality) at a much finer level of spatial resolution.

This analysis has identified several important research questions. The lack of evidence for a large contribution from animal reservoirs means that the high rates of disease in New Zealand remain unexplained. Potential interventions to reduced person-to-person transmission and travel-related enteric infection deserve further investigation.

**Competing interests:** None known.

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Acknowledgements: The Institute of Environmental Science and Research Ltd (ESR) supplied the notification data and the New Zealand Health Information Service supplied the hospitalisation data. Simon Hales helped construct the maps in ArcGIS.

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References:


Appendix 1. Giardiasis notification and hospitalisation numbers and rates (average annual rate per 100,000), by season, urban-rural domicile, age group, sex, ethnicity, and deprivation level, 1997–2006, New Zealand

(\textit{na}=not applicable)

<table>
<thead>
<tr>
<th>Category</th>
<th>Notifications</th>
<th>Hospitalisations</th>
<th>Hospital proportion$^d$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.$^1$ Rate$^e$ RR (95%CI)$^f$</td>
<td>No.$^1$ Rate$^e$ RR (95%CI)$^f$</td>
<td>%</td>
</tr>
<tr>
<td><strong>Season</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Summer (Dec-Feb)</td>
<td>403 43.14</td>
<td>6 0.62</td>
<td>1.00</td>
</tr>
<tr>
<td>Autumn (Mar-May)</td>
<td>472 50.48 1.17 (1.121 – 1.220)</td>
<td>7 0.77</td>
<td>1.24 (0.878 – 1.754)</td>
</tr>
<tr>
<td>Winter (Jun-Aug)</td>
<td>404 43.25 1.00 (0.960 – 1.047)</td>
<td>8 0.81</td>
<td>1.31 (0.931 – 1.844)</td>
</tr>
<tr>
<td>Spring (Sep-Nov)</td>
<td>368 39.42 0.91 (0.874 – 0.956)</td>
<td>7 0.77</td>
<td>1.24 (0.878 – 1.754)</td>
</tr>
<tr>
<td><strong>Urban-rural$^g$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban Total</td>
<td>1364 42.58</td>
<td>22 0.68</td>
<td>1.00</td>
</tr>
<tr>
<td>Rural Total</td>
<td>283 53.20 1.23 (1.187 – 1.282)</td>
<td>5 1.02</td>
<td>1.49 (1.107 – 2.009)</td>
</tr>
<tr>
<td>Main urban</td>
<td>1185 44.62</td>
<td>16 14.23</td>
<td>1.00</td>
</tr>
<tr>
<td>Satellite urban</td>
<td>44 39.63 0.90 (0.882 – 0.925)</td>
<td>1 0.30</td>
<td>1.97 (1.118 – 3.463)</td>
</tr>
<tr>
<td>Independent urban</td>
<td>136 30.96 0.73 (0.688 – 0.765)</td>
<td>5 4.91</td>
<td>1.80 (1.303 – 2.499)</td>
</tr>
<tr>
<td>Rural, high urban</td>
<td>54 56.58 1.25 (1.231 – 1.271)</td>
<td>1 0.67</td>
<td>1.58 (0.806 – 3.090)</td>
</tr>
<tr>
<td>Rural, intermediate</td>
<td>76 56.39 1.28 (1.254 – 1.301)</td>
<td>1 0.58</td>
<td>1.61 (0.917 – 2.842)</td>
</tr>
<tr>
<td>Rural, low urban</td>
<td>121 53.97 1.20 (1.175 – 1.235)</td>
<td>3 3.66</td>
<td>2.10 (1.403 – 3.134)</td>
</tr>
<tr>
<td>Highly rural</td>
<td>31 41.07 0.89 (0.869 – 0.908)</td>
<td>3 0.02</td>
<td>0.88 (0.326 – 2.371)</td>
</tr>
<tr>
<td><strong>Age group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-4</td>
<td>398 147.01 3.67 (3.474 – 3.872)</td>
<td>6 2.29</td>
<td>2.59 (1.756 – 3.823)</td>
</tr>
<tr>
<td>5-9</td>
<td>136 47.66 1.20 (1.121 – 1.287)</td>
<td>1 0.45</td>
<td>0.51 (0.276 – 0.956)</td>
</tr>
<tr>
<td>10-14</td>
<td>43 14.69 0.37 (0.334 – 0.412)</td>
<td>1 0.45</td>
<td>0.51 (0.272 – 0.941)</td>
</tr>
<tr>
<td>15-19</td>
<td>31 11.54 0.29 (0.259 – 0.329)</td>
<td>0 0.15</td>
<td>0.17 (0.066 – 0.467)</td>
</tr>
<tr>
<td>20-29</td>
<td>193 39.66</td>
<td>4 0.88</td>
<td>1.00</td>
</tr>
<tr>
<td>30-39</td>
<td>402 69.63 1.75 (1.658 – 1.848)</td>
<td>4 0.69</td>
<td>0.78 (0.510 – 1.207)</td>
</tr>
<tr>
<td>40-49</td>
<td>200 37.20 0.94 (0.881 – 0.999)</td>
<td>3 0.58</td>
<td>0.65 (0.411 – 1.036)</td>
</tr>
<tr>
<td>50-59</td>
<td>129 30.76 0.78 (0.724 – 0.833)</td>
<td>3 0.69</td>
<td>0.78 (0.490 – 1.256)</td>
</tr>
<tr>
<td>60-69</td>
<td>76 26.83 0.68 (0.625 – 0.737)</td>
<td>2 0.81</td>
<td>0.92 (0.555 – 1.529)</td>
</tr>
<tr>
<td>70+</td>
<td>40 12.37 0.31 (0.281 – 0.348)</td>
<td>2 0.62</td>
<td>0.70 (0.413 – 1.193)</td>
</tr>
<tr>
<td><strong>Sex$^h$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>833 45.68</td>
<td>14 0.75</td>
<td>1.00</td>
</tr>
<tr>
<td>Female</td>
<td>786 41.04 0.92 (0.858 – 0.981)</td>
<td>14 0.74</td>
<td>0.98 (0.775 – 1.240)</td>
</tr>
<tr>
<td>Unknown</td>
<td>29 na</td>
<td>0 na</td>
<td>na</td>
</tr>
<tr>
<td><strong>Ethnicity$^i$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>1127 39.30</td>
<td>20 0.70</td>
<td>1.00</td>
</tr>
<tr>
<td>Maori</td>
<td>87 16.49 0.33 (0.305 – 0.367)</td>
<td>4 0.80</td>
<td>1.15 (0.760 – 1.760)</td>
</tr>
<tr>
<td>Pacific</td>
<td>14 6.17 0.14 (0.118 – 0.155)</td>
<td>1 0.30</td>
<td>0.40 (0.240 – 0.640)</td>
</tr>
<tr>
<td>Other</td>
<td>99 37.65 0.76 (0.733 – 0.792)</td>
<td>3 1.03</td>
<td>1.54 (0.782 – 2.330)</td>
</tr>
<tr>
<td>Unknown</td>
<td>320 na</td>
<td>0 na</td>
<td>na</td>
</tr>
<tr>
<td><strong>Deprivation$^j$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2</td>
<td>441 65.57</td>
<td>3 0.49</td>
<td>1.00</td>
</tr>
<tr>
<td>3-4</td>
<td>371 52.82 0.81 (0.772 – 0.843)</td>
<td>6 0.80</td>
<td>1.62 (1.055 – 2.494)</td>
</tr>
<tr>
<td>5-6</td>
<td>301 40.75 0.62 (0.595 – 0.653)</td>
<td>4 0.51</td>
<td>1.05 (0.658 – 1.671)</td>
</tr>
<tr>
<td>7-8</td>
<td>278 34.84 0.53 (0.508 – 0.559)</td>
<td>6 0.74</td>
<td>1.50 (0.982 – 2.303)</td>
</tr>
<tr>
<td>9-10</td>
<td>248 32.19 0.49 (0.469 – 0.517)</td>
<td>9 1.10</td>
<td>2.25 (1.504 – 3.360)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1647 44.07</td>
<td>28 0.67</td>
<td>na</td>
</tr>
</tbody>
</table>

1. Number is the average annual number rounded to the nearest integer.
2. Rate is the average annual rate per 100,000 population.
3. RR = Rate ratio calculated in relation to reference value in bold, 95%CI = 95% confidence interval calculated based on 10-year period.
4. Proportion hospitalised is based on recorded hospitalisations expressed as a percentage of number notified.
5. Notification rates for urban/rural distribution, sex and ethnic groups were directly age standardised to the age distribution of the New Zealand population at the 2001 Census with confidence intervals calculated according to the methods used for age standardised data.$^{44,45}$ Hospitalisation numbers are too low for age standardising, which led to wide confidence intervals. Only the age-standardised RRs for ethnicity were significantly different compared to the crude RR, and therefore we used the crude RR for sex and urban-rural distribution and age-standardised RR only for ethnicity.
Has smoking prevalence markedly decreased in New Zealand despite more cigarettes released for sale?

Murray Laugesen

Abstract

Aims To assess whether smoking declined markedly since 2003, as reported by the New Zealand Health Survey (NZHS) of 2007.

Method Comparison of daily smoking prevalence from the NZHS, Census, and annual ACNielsen Ltd smoking prevalence survey against tobacco and cigarette volumes released to the domestic market, 1996-2007.

Results From 2003 to 2007, NZHS-reported daily cigarette smoking prevalence decreased from 22.8% to 18.1%, implying 125,000 (-17%) fewer smokers, whereas cigarettes annually released for sale increased 7.5% from 3957 to 4253 million sticks. In contrast, the Census and the ACNielsen commercial survey estimated 1.0 and 1.5 percentage point decreases respectively in numbers smoking. Identifiable factors explained up to 34% of the decrease in numbers smoking. Anti-smoking sentiment was greater in 2007.

Conclusion It is highly doubtful if adult daily smoking prevalence has yet decreased below 20%. Smokers responding to the 2007 NZHS, more than in previous health surveys, tended to underreport their smoking. They may have opted out of responding altogether, or otherwise not reported they smoked. Future health surveys should include biochemical validation of smoking status.

In its report to the incoming government in November 2008, the Ministry of Health noted that “Since 2003 there has been a 5% fall in the total number of people who smoke every day.”¹ This refers to the National Health Survey result showing daily adult smoking prevalence had fallen 4.8 percentage points between 2003 and 2007, from 22.9% to 18.1%.² As smoking prevalence is now the bottom line for measuring success in tobacco control, success depends on health surveys to call the results correctly.

Despite every statistical precaution being taken, surveys still depend on eliciting truthful answers. For illegal drugs, this requires urine testing. For tobacco smoking we rely on self-report. However, public opinion against smoking is now severe. In a telephone poll in 2006, 52% of adults supported a ban on the sale of all tobacco products,³ while in 2007, 76% said smoking is ‘not at all acceptable’ at outdoor children’s playgrounds.⁴

Surveys tend to under-report community levels of smoking, but (for reasons of cost) few surveys report biochemically-validated results, which would detect those responders who report their smoking incorrectly. For example, testing for cotinine (a by-product of nicotine in saliva) has shown national surveys underestimated smoking
prevalence by 0.6, 2.8, and 4.4 percentage points in the United States, England, and Poland respectively.

Cotinine concentrations in those misclassified as non-smokers were indicative of high levels of smoke intake. In 1997, in Christchurch, pregnant women tested for serum cotinine showed they under-reported smoking prevalence by 5.5 percentage points, meaning that 22% of the smokers would otherwise have been unidentified as such by their midwife or doctor.

In particular, health surveys encourage healthier responders and/or responses. New Zealand-wide health surveys to date—in 1990, 1993, 1997, 2003, and 2007—have reported cigarette smoking prevalences respectively 3.0, 4.0, 0.7, 2.1, and 4.9 percentage points below that of ACNielsen’s commercial survey in that year. Census smoking prevalence was intermediate between NZHS and ACNielsen’s results (Table 1).

As a reality check, trends in numbers smoking were compared against cigarettes and tobacco volumes released, as given by Customs excise data. If smoking prevalence (the percentage of adults who smoke) decreases, then fewer smoke, and the volume of demand for cigarettes decreases as expected. If, however, the cigarette supply holds up, remaining smokers must be buying and smoking more on average.

Method
Focusing on ages 15 years and over from 1996 to 2007, tobacco and cigarettes released for sale from Statistics New Zealand were compared against daily smoking prevalence reported from the periodic New Zealand health surveys during these years, the Census smoking questions in 1996 and 2006, and by the annual ACNielsen commercial survey of cigarette smoking.

Statistics New Zealand annually publish Customs data on tobacco and cigarette volumes released for sale, and on resident population aged 15 and over. Tobacco for hand-rolling was estimated to produce 2 million roll-your-own (RYO) cigarettes per tonne, based on an estimated national average tobacco weight of 0.5 g per RYO cigarette in 2006. All smoking prevalence surveys involved visits to homes. To enable cross-survey comparison only daily cigarette smoking prevalence was considered, defined to include both factory-made and RYO cigarette smoking. Results were not standardised for differences in age structure of the population; instead crude prevalence data were used to estimate actual numbers of smokers in each year, for appropriate comparison with the numbers of cigarettes released for sale.

The ACNielsen cigarette survey purchased by the Ministry of Health from 1982 to 2007 was based on an annual omnibus survey asking about various consumer items (cars, whiskey), and used a show card of various tobacco products to ask about regular use. Smoking is thus accepted as normal consumer behaviour in a way not possible in a health survey.

The NZHS of 1996/7 was mainly fielded in 1997. The 2002-3 NZHS, based on computer-assisted personal interviews, was mainly in the field in 2003. The Census asked a question on cigarette smoking in March 1996 and March 2006. The Census, being filled in by parents, tends to under-report smoking at age 15 to 19 years, compared with the ACNielsen survey, in which the teenager is interviewed individually. Of adults age 15 years and over, 8.6% gave an unusable answer to the Census smoking question, and daily smoking prevalence was estimated from the 91% giving usable answers.

The 2006-7 NZHS was in the field from October 2006 to November 2007, and thus mainly reports on the 2007 year. The 2006 and 2008 Tobacco Use surveys and the 2007 Alcohol and Drug Use surveys only included smokers aged 15 to 64 years and are not reported here, but four government surveys in 2006-7 were available for age-standardised comparisons for ages 15 to 64 years.

The Health Sponsorship Council’s Monitor is a telephone survey of 1500 to 2000 people age 15 years and over annually from 2003 to 2007, excepting 2006.
Results

All tobacco sold is smoking tobacco, and 99% is smoked as cigarettes, as analysed from tobacco manufacturers’ annual returns to the Ministry of Health. Factory-made cigarettes accounted for 69% and RYO tobacco for 30% of the dry weight of all tobacco used.

Cigarettes released for sale—In contrast, total cigarettes released, including factory-made and RYOs, rose by 7.5%, from 3957 million in 2003 to 4253 million in 2007 (Table 1).

Numbers of smokers—NZHS reported a 17% decrease in numbers smoking in 2007; that is, 125,000 fewer smokers (Table 1). The ACNielsen survey, however, showed only a 1.5% decrease in smoking population from 2003 to 2007, and the Census showed a 1% decrease from 1996 to 2006.

Smoking prevalence (Table 1)—The NZHS reported a sharp decrease in daily smoking prevalence from 22.8 in 2002-3 to 18.1% in 2006-7. (The value for 2006-7 age-standardised to the 2006 census value was 17.8 %.) ACNielsen reported a gradual decline in smoking prevalence: 26% in 1996-7, 23.6% in 2006, and 23% in 2007—a decline of only 3 percentage points in 10 years (Table 1), but showed no decline between 2004 and 2007 (Table 1).

Table 1. Changes in proportions and numbers smoking, versus cigarettes released for sale, 1996–2007

<table>
<thead>
<tr>
<th>Year</th>
<th>Cigarettes released counting RYOs as 0.5 g each Millions</th>
<th>Resident population age 15 and over Millions</th>
<th>Smoking prevalence as percentage of adults age 15 and over, and smoking population in thousands, not age-standardised</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cigarettes released counting RYOs as 0.5 g each Millions</td>
<td>Resident population age 15 and over Millions</td>
<td>ACNielsen % ‘000</td>
</tr>
<tr>
<td>1996</td>
<td>4976</td>
<td>2.8958</td>
<td>26</td>
</tr>
<tr>
<td>1997</td>
<td>4913</td>
<td>2.9282</td>
<td>26</td>
</tr>
<tr>
<td>2003</td>
<td>3957</td>
<td>3.1259</td>
<td>25</td>
</tr>
<tr>
<td>2004</td>
<td>4002</td>
<td>3.1780</td>
<td>23</td>
</tr>
<tr>
<td>2005</td>
<td>4214</td>
<td>3.2210</td>
<td>23.6</td>
</tr>
<tr>
<td>2006</td>
<td>4197</td>
<td>3.2665</td>
<td>23.6</td>
</tr>
<tr>
<td>2007</td>
<td>4253</td>
<td>3.3424</td>
<td>23</td>
</tr>
<tr>
<td>2003–7</td>
<td>296</td>
<td>0.2165</td>
<td>-2.0</td>
</tr>
<tr>
<td>Difference</td>
<td>7.5%</td>
<td>6.9%</td>
<td>-8%</td>
</tr>
</tbody>
</table>

Source: Cigarettes,10,17 Resident population,11 AC Nielsen,1 New Zealand Health Surveys,1,7,8 Census.14 Numbers smoking estimated from survey prevalence fraction × resident population.

The Census reported a 3-percentage points decrease in smoking prevalence over 10 years, from 23.7% in 1996 to 20.7% of adults in 2006. This was paralleled by a similar increase in the percentage of never-smokers, while the percentage of ex-smokers remained at 21% in both Censuses, indicating that the proportion quitting was matched by the proportion resuming smoking.
Comparisons, based on daily smoking at ages 15 to 64 years, and age-standardised against the 2006 Census, showed that the lowest smoking prevalence was given by the 2007 New Zealand Health Survey, though the three 2007 surveys (New Zealand Health Survey, Alcohol and Drug Survey, and the 2008 Tobacco Use Survey) had overlapping confidence limits. The narrow confidence intervals of the Census did not overlap the confidence limits of the 2007 or 2008 surveys. Thus three Ministry of Health surveys in 2007–8 reported lower smoking prevalence in 2007 than the Census of March 2006.

Action on Smoking and Health (ASH) national surveys show smoking prevalence at 14–15 years of age decreased steadily from 1999 to 2007. By 2007, these students populated the 15–24 year age group. The New Zealand Health Surveys found smoking prevalence for 15-24 year-olds decreased 3.3 percentage points, from 27% in 2003 to 23.7% in 2007, equal to 9,400 fewer youth smoking.

After smoking was banned in all workplaces and hospitality venues from December 2004, the proportion of adults reporting it was “not at all acceptable” to smoke at outdoor sports fields or courts, increased from 35–37% in 2003–05, to 51% in 2007.

Discussion

As Table 1 shows, smoking prevalence values in 2006, 2007, and 2008 were lower than in 2003. Also, smoking prevalence was recorded as lower in 2007–8 than in (the Census of) 2006. The question is, how credible are the lower smoking prevalences found in 2007–8, and how to interpret them?

NZHS results from 2003 and 2007, equate to 125,000 fewer smokers, a 17% decrease in numbers of adults smoking in 4 years. (Table 1) The numbers of cigarettes released to the market increased 7.5%, however, during these 4 years, from 2003 to 2007—an increase of 296 million in cigarettes released annually (Table 1).

These two trends are incompatible. For the NZHS result to be compatible with cigarette volumes released, remaining smokers would have to buy 30% more cigarettes per day [100*1.075/(1-0.17) =130], those previously smoking 20 a day and still smoking would need to buy 26 cigarettes a day.

The 2006 Census recorded smoking prevalence to be three percentage points below the value from the 1996 Census. If this decrease was due to quitting, the proportion of former smokers (21% in 1996, 21% in 2006) should have increased. Instead, the proportion which had never smoked increased 3 percentage points, suggesting no change attributable to smoking cessation between 1996 and 2006.

What factors might explain the 125,000 decrease in numbers smoking reported by the New Zealand Health Surveys from 2003 to 2007?

Firstly, how much is explained by survey methods and demographics? For example, how much of the NZHS decrease is confirmed by other surveys? As Table 1 shows, 12,000 fewer smokers or 1.5% of 125,000 can be explained by the AC Nielsen survey 2003 to 2007. Again, how much can be explained by changing age structure of the population?

Age-standardisation of the health survey data to the 2006 Census population (Ministry of Health data, unpublished 2008) would narrow the decrease in prevalence from 4.8
percentage points in Table 1, to 4.5 percentage points, equivalent to 6% of the decrease in numbers of smokers. The 95% upper confidence limit for the NZHS in 2007 was 19.0%, which reduces the decrease since 2003 by 19%. Finally, 9400 fewer youth taking up smoking would account for 7.5% of the 2003–7 decrease in smoking numbers at age 15 years and above. Thus in total, these factors account at best for 34% of the NZHS decrease in numbers smoking from 2003 to 2007.

Secondly, was this a smokefree law effect? The evidence suggests not. AC Nielsen’s survey showed 25% smoked in 2003, 23% in 2004, and 23.5% in 2005. The Smokefree Environments Amendment Bill banning smoking in bars and remaining workplaces was enacted in December 2003, and took effect from December 2004. Any effect of this legislation on smoking prevalence was in place by 2004, and does not explain a decrease in smoking prevalence between the Census of 2006 and the New Zealand Health Survey result of 2007.

Thirdly, did smoking decrease due to graphic cigarette packet warnings? No. Regulations required manufacturers to put new warnings on sale between February and August 2008. The Health Survey, however, was in the field in 2006–7 before these regulations took effect, so smokers responding to NZHS had not yet seen graphic warnings on their cigarette packets.

Fourthly, was this a price effect? No. Price increases tend to lower sales. Cigarette excise and prices, however, remained the same in real terms from 2001 to 2007 inclusive.

Lastly, is the 2007 New Zealand Health Survey defective in a unique way? No; the 2007 Alcohol and Drug Survey gave a similar low result, and the confidence limits overlap. The New Zealand Health Survey result is not an outlier on its own. Rather these two health surveys may both have been prone to a health bias in 2007 favouring a lower reported smoking prevalence.

The decline in smoking prevalence from 2003 to 2007 reported by the New Zealand Health Surveys is implausible and incompatible with the increased volumes of cigarettes released for sale. This was not due to commercial fluctuations in volumes released for sale, as tobacco volumes used in manufacture show similar annual trends.

The one possible cause of the apparent 2007 decrease in smoking prevalence (which is not seen in the 2006 surveys) is the increased unacceptability of smoking that was detected in the Health Sponsorship Council monitor of 2007. Social undesirability of smoking may have influenced some smokers to either opt out of responding to the survey as a whole or to the smoking questions in particular, or disown their smoking when responding.

Admitting to smoking is embarrassing for many smokers. There is no biochemical proof that this was the case in the New Zealand Health Survey, but for future health surveys it would be advisable to validate reported smoking status, by testing salivary cotinine or exhaled carbon monoxide. Although cotinine tests are expensive, such costs are only a small fraction of the total cost of a national smoking survey, and essential to its correct interpretation. There is no other way to measure for changes in the tendency for smokers to under-report their smoking.
Until future surveys can be validated, the rate of recent decline in smoking is best judged by the Census, namely 3 percentage points in 10 years, at which rate it would take 70 years to reach near-zero smoking.

If smoking prevalence is falling no faster than indicated by the 1996–2006 Censuses, ending the cigarette deaths epidemic (4500 deaths a year) requires intervention from Government, and not just the health sector. Government can induce marked decreases in smoking prevalence, as in 1987 to 1991, when adult smoking prevalence declined from 30% to 26%; successive cash-strapped governments repeatedly increased tobacco excise above the level of inflation.

Government last increased the real tobacco excise rate in 2000. Annual excise adjustments for inflation have since kept the price of smoking of factory-made cigarettes high. However nearly half of all smokers now smoke RYOs, for 4 or 5 dollars a day. The price of a cup of coffee buys enough tobacco for 12 RYO cigarettes.

Unsurprisingly, smoking prevalence overall is reducing extremely slowly, despite greater government funding for a wider range of stop smoking programmes and products since 2000. The tobacco excise rate on factory-made cigarettes, when adjusted for incomes, is one of the highest among industrialized nations, so that any government seeking extra revenue may hesitate to raise it much. Moreover, if the excise rate is simultaneously increased on loose RYO tobacco by the same percentage, the effect on smoking prevalence would be blunted by more smokers shifting to the cheaper RYOs instead of quitting smoking.

As in 2000, increasing the tobacco excise rates evenly on all tobacco products is unlikely to gain revenue or reduce smoking prevalence.

Action is now required to focus on the real problem—RYO smoking is cheap and available at half the cost of smoking factory-made cigarettes. The lower tax on RYOs deprives government of over $300 million annually, and dissuades smokers from quitting. It is necessary to raise the excise on RYO cigarettes to the same level per cigarette as for factory-made cigarettes. This would require a doubling of the current excise per gram on loose tobacco, probably phased in over several steps.

Competing interests: None (Murray Laugesen is a public health physician and independent contract researcher). No funding was received for this study.

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References:


New Zealand’s venomous creatures
Robin J Slaughter, D Michael G Beasley, Bruce S Lambie, Leo J Schep

Abstract

Aim New Zealand is home to a small number of venomous creatures. The purpose of this review is to educate and update healthcare professionals on the management of envenoming from these creatures.

Methods An extensive literature review was performed by systematically searching OVID MEDLINE and ISI Web of Science. In addition, further information was obtained from book chapters, relevant news reports, and web material.

Results The signs and symptoms resulting from envenoming of clinically significant venomous creatures found in New Zealand are discussed. Definitive medical treatment recommendations are made.

Conclusion Encounters with New Zealand’s few venomous creatures, while rarely fatal, can cause significant morbidity. Effective management can be achieved by informed health professionals having regard to the principles outlined in this review.

New Zealand is host to a small but varied group of terrestrial and marine venomous creatures. They range from species which cause only minor injuries, such as bluebottle jellyfish (*Physalia utriculus*), to creatures which can cause significant systemic envenoming, such as the yellow bellied sea snake (*Pelamis platurus*). Although envenoming caused by these animals is relatively rare, there are still a sufficient number of cases each year in New Zealand to warrant a broad review and update of current recommendations for management.

In this article we will examine New Zealand’s venomous creatures, including their habitats and distribution, the clinical effects of envenoming, and appropriate first aid and definitive medical treatment.

Katipo and Redback spiders

Description—There are five medically significant genera of spider in the world (*Atrax*, *Hadronyche*, *Latrodectus*, *Loxosceles*, and *Phoneutria*). New Zealand has two venomous spiders from the genus *Latrodectus*: the endangered native katipo (*Latrodectus katipo*) and an Australian import, the redback spider (*Latrodectus hasseltii*). Another spider, the “black katipo”, was once thought to be a separate species (*Latrodectus atritus*) but has been found to be a junior synonym of *L. katipo*.\(^1\) Katipo is a Māori name and means “night stinger”; it is derived from two words, *kakati* (to sting), and *po* (the night).\(^2\)

*L. katipo* is found throughout coastal regions of New Zealand, including most of the North Island, and south to Greymouth on the west coast and Dunedin on the east coast of the South Island.\(^3,4\) Katipo spiders have a highly specialised habitat and are only
found near the shoreline amongst sand dunes. Redback spiders were first found in the early 1980s in Wanaka and have spread further afield since then.

Figure 1. *Latrodectus katipo* – Katipo spider

Figure 2. *Latrodectus hasseltii* – Redback spider

The katipo and redback are similar in appearance, both being dark brown to black in colour, with an orange to red jagged stripe running down the length of the dorsal abdomen (Figure 1). Katipo spiders are slightly smaller than redback spiders (~10 mm body length) (Figure 2).

Both spiders have a red hourglass marking on the lower surface, a common trait in many *Latrodectus* spiders. Female katipo found north of 39°15’S (*L. katipo var. atritus*) almost always lack the red stripe on the dorsal abdomen.

There are very few reported cases of katipo or redback bites in New Zealand. Historically, there is some evidence of occasional significant symptoms from katipo bites. Black katipo bites have not been considered particularly dangerous, although a suspected case from Te Kopuru with mild to moderate symptoms has been reported.

Bites from these spiders are not common, as by nature they are shy, non-aggressive animals. Additionally, the narrow habitat of the katipo and diminishing population means interaction between humans and the spider is typically minimal. Accidentally disturbing a web or antagonising a spider will likely result in the spider biting in self defence. If the female spider is protecting an egg sac it may act more aggressively.

**Signs and symptoms**—Bites produce a syndrome known as latrodectism, which is characterised principally by pain. There may initially be minimal effects associated with the instant of the bite before local redness, diaphoresis, and pain develop. Pain often increases in severity and spreads proximally.

Abdominal, back, or chest pain can occur. Non-specific systemic symptoms may develop, including nausea, vomiting, generalised diaphoresis, headache, lethargy, malaise and hypertension; less commonly other neurologic and autonomic symptoms may occur.
Treatment—To help minimise systemic venom spread, victims should be reassured and persuaded to remain still. The application of a cold pack may help relieve local pain.

All bites should be medically observed in a hospital for at least 6 hours to detect any onset of symptoms. Patients can initially be treated with opioid analgesia and benzodiazepines. If pain is refractory to initial treatment, or if significant signs and symptoms of latrodectism occur, redback antivenom can be considered.

Current limited evidence on safety and efficacy would support antivenom use in cases of latrodectism causing either systemic effects or severe or persistent pain. It is likely to be effective for both redback and katipo spider bites. Adverse reactions to this antivenom are relatively uncommon; early anaphylactoid reactions have been reported as occurring in 0.5 to 0.8% of patients, while less than 5% develop serum sickness. Severe anaphylaxis or death has not been reported.

White tailed spider

Description—The white tailed spider (Lampona cylindrata and L. murina) is an Australian native which arrived in New Zealand in the late 19th Century. Often found both inside and outside the home, it hides during the day in small crevices in walls, crawl spaces, shoes, bed linen, and under clothes or items left on the floor. This spider is distinctive with a dark grey to black cylindrical body, and is readily identifiable by a small white patch at the end of the abdomen, above the spinnerets. It is between 12 and 17 mm long (Figure 3).

Figure 3. Lampona cylindrata – White tailed spider

In New Zealand the white tailed spider has only become a medical concern in the last 15 to 20 years where it has been linked to necrotising arachnidism. This concern has been due, in part, to media attention and medical reports in both New Zealand and Australia, suggesting bites may cause necrotic ulcers.

However, a recent study has demonstrated that there is no link between white tailed spider bites and tissue necrosis. A prospective cohort study of 130 cases with confirmed bites was conducted. Patients were included only if there was a clear history of a bite, and the spider was caught at the time of the bite and later identified by an expert arachnologist. None of the 130 cases developed necrotic ulcers. Indeed, the only spiders proven to cause necrotising arachnidism belong to the genus *Loxosceles*, such as the North American brown recluse spider, and are not found in New Zealand.
**Signs and symptoms**—The bite from a white tailed spider may cause local symptoms of pain, redness, swelling, and pruritus.¹⁸ Less commonly minor systemic symptoms of nausea, vomiting, malaise, and headache can also be seen. All these symptoms are generally mild and self-limiting.¹⁸ As with any puncture wound, there is a risk of secondary infection.

**Treatment**—First aid consists of ensuring the wound is cleaned, along with application of an ice pack to reduce pain and swelling.

Further treatment is seldom required but may include simple analgesia and/or antihistamines for symptomatic relief. Patients presenting with a lesion thought to be associated with a spider should be thoroughly investigated for another cause.²⁰ A good history and physical examination should be undertaken.

For severe or persistent necrotic lesions, microbiological investigation should be performed, including cultures for organisms such as fungi and unusual bacteria.²¹ Alternative diagnoses to spider bite should be considered, as a wide array of conditions have been misdiagnosed as necrotic arachnidism; these include sporotrichosis,²² Pyoderma gangrenosum,²³ Mycobacterium ulcerans,²⁴ and even chemical burns.²⁵

**Jellyfish**

**Description**—Of the numerous jellyfish within New Zealand’s coastal waters, only two species (within the same genus) are considered medically important: the Bluebottle jellyfish (*Physalia utriculus*) and the Pacific or Portuguese Man-of-War jellyfish (*Physalia physalis*). The two jellyfish species most dangerous to humans,²⁶ the box jellyfish (*Chironex fleckeri*) and irukandji jellyfish (*Carukia barnesi*), are not found in New Zealand.

Bluebottles have a distinctive bright blue floating bladder, measuring about 2–15 cm long, and one main deep blue fishing tentacle which may be up to 10 m in length.²⁷ (Figure 4)

The Pacific or Portuguese Man-of-War is a larger form of the bluebottle; its floating bladder may be up to 25 cm long, and it has up to five main tentacles.²⁸ (Figure 5)
All jellyfish possess microscopic stinging cells called nematocysts. These structures are numerous on the tentacles or body of the animal, and are used to capture prey. A small dose of venom contained within each nematocyst is discharged in response to chemical or mechanical stimulation.29 Nematocysts from many jellyfish do not penetrate human skin and/or their venom is not toxic to humans; encounters with these therefore do not produce a significant reaction. However, Physalia nematocysts do penetrate human skin, and envenoming may lead to systemic effects.

**Signs and symptoms**—Most victims of Physalia envenoming will display no signs and symptoms other than localised pain and pruritus. Characteristically, stings cause a linear collection of elliptical blanched weals, with a surrounding red flare (resembling a "string of beads").30 Extensive stinging (more likely from larger specimens) may lead to systemic symptoms including nausea, vomiting, headache, chills, drowsiness, breathing difficulties, cardiovascular collapse, or death;31,32 however, systemic symptoms are rare.33 Mild localised hypersensitivity reactions can occur (e.g. rash, urticaria, itching), but anaphylaxis is uncommon.34

**Treatment**—Initially the victim should be prevented from rubbing the area or performing vigorous muscular activity, as this will lead to greater discharge of attached nematocysts and venom movement into the general circulation.35 On-site first aid consists of flushing the affected area with sea water to help remove any adherent tentacles,28 careful removal of tentacles with forceps may be required.
Vinegar, which is used successfully to treat box jellyfish stings in north Australia,\textsuperscript{28} is contraindicated in New Zealand as it causes additional discharge of Physalia nematocysts, leading to a greater envenoming.\textsuperscript{36} Fresh water may also cause a discharge of nematocysts to a lesser extent, but it is acceptable to initially flush the area with fresh water if sea water is not available.

Traditionally, ice or cold packs were recommended for pain relief following Physalia stings;\textsuperscript{37} however, a recent randomised controlled trial has shown significant benefit of hot water over cold packs.\textsuperscript{33} Hot water immersion or showers should now be considered the treatment of choice for Physalia envenoming. The technique as described (below) for fish stings should be followed, or alternatively a hot shower may be all that is required to alleviate pain.

Further treatment consists of ensuring adequate pain relief with topical anaesthesia and simple analgesics (e.g. paracetamol), or parenteral administration of an opioid in severe cases. Infection, hypersensitivity reactions, or systemic symptoms are rare complications; monitoring and supportive care should be undertaken if required. There is no antivenom available for Physalia stings.

Stingrays

**Description**—Stingrays are found throughout New Zealand’s coastal waters, generally in shallow intertidal areas such as sheltered bays, river mouths, and other sandy regions.\textsuperscript{38} They are cartilaginous fish with a characteristic round, flattened body, and a thin tail (Figure 6). The tail contains at least one serrated spine on the dorsal surface; each spine has two ventrolateral glandular grooves containing the venom glands surrounded by the epidermis.\textsuperscript{38,39}

Stingrays are not belligerent and do not attack humans if unprovoked. The majority of stingray injuries are to the lower limbs and usually occur when swimmers or divers accidentally step on them. Injuries can also occur when fishermen find stingrays in their nets or on their lines.\textsuperscript{40}

**Figure 6. Dasyatis brevicaudata** – Short-tail stingray

![Dasyatis brevicaudata](Image)

**Signs and symptoms**—The most significant concerns following a stingray strike are the risk of traumatic injury, envenoming, and bacterial wound contamination.\textsuperscript{41,42} Such strikes may cause lacerations or puncture wounds, which may involve direct
injury to tendons, muscles, nerves, blood vessels or internal organs. Associated envenoming may cause intense local pain, oedema, and muscle cramps; local blistering or necrosis can also occur and may be extensive. Serious injury or death, though rare, has occurred due to exsanguination or direct trauma to vital organs (venom may contribute to the internal organ damage from such injuries), and from complications such as septicaemia or tetanus infection. Systemic symptoms, though uncommon, may include nausea, vomiting, diarrhoea, hypotension, syncope, salivation, tremor—and in rare cases convulsions, arrhythmias, or circulatory collapse.

Treatment—First aid consists of flushing the affected area with fresh water (sea water will suffice if fresh water is unavailable). Flushing assists in the removal of venom and barb fragments. Any haemorrhage must be controlled with local pressure. Stingray venom is heat labile, and much pain relief can therefore be achieved through the application of heat to the wound. Hot water immersion can be of considerable benefit and should be trialled at an optimum temperature of around 45°C for 15 to 20 minutes, taking care not to cause a thermal burn. The water temperature should be checked before immersion to ensure water temperature is bearable without injury.

If pain subsides, immersion should continue for up to 2 hours. Ensure the water remains at around 45°C. However, if pain relief is not achieved in the first 15 to 20 minutes, the procedure should be abandoned.

Early exploration and debridement of the wound is essential in all cases. All patients should be transported to a hospital as soon as practical for definitive wound management. Secondary infection is a significant cause of morbidity.

All foreign matter, including retained sting fragments and any non-viable tissues, must be removed. Local anaesthesia or a regional nerve block should be used, but avoid adrenaline as this delays microvascular clearance of venom, thereby aggravating necrosis. Deep wounds may require debridement under general anaesthesia. Spines are usually radiopaque, and X-ray examination should be used to ensure removal of all fragments. Ultrasound may be required if there are still doubts regarding retained spines. Following debridement, the area should be thoroughly cleaned and left open to granulate and heal by secondary intention.

Infiltration of the wound area with local anaesthetic may be required in the event of severe pain. Regional nerve block and/or parenteral opioids may also be required. There is no specific antivenom available. Supportive care should be undertaken for any systemic symptoms.

Stingray strikes may introduce a range of marine bacteria into a wound, or the spine may break and contaminate the site. Sequelae to these events include ulceration, infection, necrotizing fasciitis, and osteomyelitis. Broad-spectrum prophylactic antibiotics are not necessary for all wounds. However, they are indicated where there is considerable foreign material present, if there is a delay of 6 hours or more in wound debridement, or if the wound is deep.

If infection is evident, then a broad spectrum parenteral antibiotic regimen is advised. Subsequent culture results should be used to determine the best antibiotic for continued management (specify seawater involvement when submitting a swab or...
specimens for microbiological analysis). Ensure tetanus prophylaxis is up to date following any stingray wound. All patients require medical review within 1 to 2 days, to detect any early evidence of wound necrosis or infection.

Other venomous marine punctures

Description—New Zealand’s coastal waters are host to a range of venomous fish and sea urchins. Most of these fish are classified within the Scorpaenidae family (scorpion fish) (Figure 7) and come in a wide variety of sizes, shapes, and colours.

Apart from stingrays, further common venomous fish in New Zealand include spiny dogfish (*Squalus acanthias*) (Figure 8) and elephant fish (*Callorhinchus milii*) (Figure 9), plus the brown bullhead catfish (*Ameiurus nebulosus*)—an introduced freshwater catfish (Figure 10).

These venomous fish have external spines, which, depending on the species, may be located on a variety of positions on the fish including the dorsal (common), pectoral, shoulder, pelvic, opercular, anal, and caudal regions.

The different fish venoms have not been studied extensively, but are considered to be heat labile and all produce a similar toxic course.

Puncture wounds can also be caused by a variety of sea urchins, including kina (*Evechinus chloroticus*) (Figure 11) which are grouped within the Echinodermata phylum. Sea urchins have two venom apparatus: external spines and pedicellariae.
(small grasping organ that is covered by venom-producing glandular tissue). Injuries typically occur to the feet or hands after victims step on or handle fish or sea urchins.

**Figure 11. Evechinus chloroticus – Kina**

Signs and symptoms—Symptoms are generally restricted to severe local pain, which can spread to the whole of the affected limb. Mild, non-specific systemic effects can also occur (including nausea, diaphoresis, and hypotension). Systemic symptoms are thought to be the result of circulating venom. In rare instances, sea urchin stings induce a delayed type hypersensitivity reaction. Clinical effects include local pruritus and erythema—along with vesicular eruptions, paraesthesia, malaise, and myalgia.

Treatment—Initial first aid consists of flushing the affected area with fresh water, control of any haemorrhage, and hot water immersion (as outlined for stingray stings). While it is unusual for fish spines to break off, spines of sea urchins can commonly fracture and remain lodged in the wound. Protruding sea urchin spines can be removed using forceps. Fish and sea urchin spines are typically radiopaque and an X-ray or ultrasound can help identify any difficult-to-remove foreign material. If there is evidence of retained fragments, surgical exploration and debridement must be undertaken.

It is especially important for sea urchin stings that all foreign material is removed, as long-term lesions—such as chronic granulomas of various aetiologies and histologies (including sarcoideal)—can occur. These granulomas are sometimes accompanied by fibrosis, necrosis, or microabscesses. Some represent non-specific foreign body inflammatory reactions, and others are considered delayed type hypersensitivity reactions to an unknown antigen.

Further treatment of fish and sea urchin injuries includes adequate pain relief, control of infection, tetanus prophylaxis, and (if required) supportive care for any systemic symptoms.
Sea Snake

Description—Sea snakes are a diverse group of front-fanged venomous snakes that belong to the Elapidae family. They vary in both size and colour, with most species growing to 1.2 to 1.5 m in length (Figure 12). Sea snakes are distributed mainly in warm tropical waters of the Indian and Pacific Oceans, with the majority found either close to the shore or around coral reefs.

Figure 12. *Laticauda colubrina* – Banded sea krait

![Banded sea krait](image)

One pelagic species, the yellow bellied sea snake (*Pelamis platurus*), is widely distributed from the east coast of Africa across the Indian and Pacific Oceans, south to New Zealand and across to the western coast of the Americas. Another species, the banded or yellow-lipped sea krait (*Laticauda colubrina*), has a wide distribution throughout the Pacific.

These two snakes are not common in New Zealand’s coastal waters, but they may occasionally beach themselves around the northern coast of New Zealand. It is estimated that only about a handful of these snakes wash up each year. Worldwide sea snake bites are encountered very infrequently and, to our knowledge, no cases of envenoming have been reported in New Zealand. Even so, there is a risk that envenoming could occur if a beached specimen is handled.

Signs and symptoms—All species of sea snake possess similar venom and therefore cause similar signs and symptoms; several attributable to myotoxins. Although it is estimated that 80% of bites result in no or trivial envenoming, there is the risk of severe and life-threatening effects occurring following bites. If envenoming does occur, there is often an asymptomatic time interval, ranging from one to several hours, before the onset of systemic symptoms.

Systemic toxicity is usually heralded by myolysis; patients typically develop significant muscle aches and pain, sometimes in association with weakness. Associated myoglobinuria may lead to renal effects including acute renal failure. Patients can also develop a flaccid paralysis with ptosis, ophthalmoplegia, and depressed or absent deep tendon reflexes. This may progress to more severe paralysis leading to full respiratory arrest.
Treatment—Following envenoming, appropriate first aid consists of applying a pressure immobilisation bandage as early as possible to retard venom transport via the lymphatic system.\(^\text{71}\)

Pressure immobilisation, particularly the immobilisation, appears to be the most effective method to minimise the systemic spread of snake venom.\(^\text{72}\) It consists of applying a broad compression bandage over the bitten area (as firmly as that used for a sprained ankle) followed by a second firm bandage applied from the tip of the limb heading proximally toward the body to cover as much of the limb as possible. A splint or sling should then be used to immobilise the affected region. The patient should remain completely immobilised for transport to hospital.\(^\text{73}\)

If there is evidence of systemic envenoming, definitive treatment consists of administering antivenom. CSL Polyvalent Snake Antivenom is suitable and this resource is available from the Auckland Hospital Pharmacy. Supportive care of an envenomed patient is probably necessary; this can include management of myolysis, paralysis, and their complications.

IV fluids to help maintain urine output are required should myolysis become evident, and urinary alkalinisation may also be helpful. However, any developing renal failure or hyperkalemia may mandate haemodialysis. Atropine and neostigmine may provide short-term reversal of respiratory paralysis.\(^\text{74}\) In the event of respiratory compromise, early consideration for intubation and artificial ventilation is recommended. Following appropriate treatment the prognosis is good.

Summary

New Zealand has a very small number of venomous creatures, and mortality from envenoming is low. In some circumstances, however, there is the potential for prolonged morbidity if appropriate treatment is not initiated. Because these cases of envenoming are uncommon, this review has been written to assist the healthcare provider to identify the species in cases of likely envenoming, and provide the appropriate first aid and definitive treatment to ensure a satisfactory outcome for the patient.

Glossary

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Antivenom</td>
<td>Serum antibodies that specifically bind to and thereby neutralise venom</td>
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<tr>
<td>Arachnid</td>
<td>Species within the subphylum Chelicerata (joint-legged invertebrate animals) that include spiders and scorpions</td>
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<tr>
<td>Arachnidism</td>
<td>Poisoning resulting from the bite or sting of an arachnid</td>
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<tr>
<td>Envenoming</td>
<td>The injection of sufficient venom into animals, including humans, to cause clinical symptoms</td>
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<tr>
<td>Latrodectism</td>
<td>The clinical syndrome caused by the venom of <em>Latrodectus</em> spp. spiders.</td>
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<tr>
<td>Nematocysts</td>
<td>Specialised stinging cells found on animals in the phylum Cnidaria</td>
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<tr>
<td>Pedicellariae</td>
<td>A sea urchin’s small grasping organ that is covered by venom producing glandular tissue</td>
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Competing interests: None known.

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Putting population health into practice through primary health care

Pat Neuwelt, Don Matheson, Bruce Arroll, Anthony Dowell, Doone Winnard, Peter Crampton, Nicolette Fay Sheridan, Jacqueline Cumming

Abstract

The introduction of the Primary Health Care Strategy has offered opportunities to take a population health approach to the planning and delivery of primary health care. The lack of a common understanding of population health between primary care and public health has been the prompt for a group of academics and practitioners to join forces and produce this statement on a population health approach to primary care, through primary health care. This paper takes the position that the features of a population health approach (such as a concern for equity, community participation, teamwork and attention to the determinants of health) enhance general practice care rather than undermine it. We conclude that the contribution of the health sector towards population health goals can be achieved through collaboration between GPs, nurses, other primary health care workers, and communities, together with health promotion and public health practitioners. Finding common language and understanding is an important step towards improving that collaboration.

Our patients can reasonably expect to have their immediate medical needs attended to, along with an examination of broader issues which may have led to their presentation. It follows logically that family physicians must have a role in looking beyond illness, and trying to shape behavioural, societal and environmental influences on ill-health.1, p.84.

The population perspective is also an attitude of mind, a looking beyond the individual patient with head injury, lead poisoning, or salmonella infection to other people at risk from the same health hazards.2, p.1805.

The opening quotes from two professors of general practice highlight well the balance that primary care strives for in practising clinical medicine while also aiming to have a positive impact on the social and environmental factors that influence people’s experience of health and illness.

The 2001 New Zealand Primary Health Care Strategy aspired to improve the health of New Zealanders and to reduce health inequalities, through the establishment of primary health organisations (PHOs) responsible for adopting a population health approach to the planning and delivery of primary care services.3 Seven years later, there is still not a common understanding across the sector of what a population health approach is in the primary care context.

This paper aims to address that question, for both primary care and public health audiences in Aotearoa New Zealand. It argues that it is a population health approach, along with an emphasis on population health outcomes, which will have the most positive impact on improving health and on the reduction of health inequities.

A population health approach to primary care delivers both high quality individual care and places an emphasis on equity, community participation, and social
determinants of health. This paper also endorses the significant changes made by many in primary health care and general practice in the last seven years, and celebrates the ways in which a practical population health approach is already being achieved.

There has been wide debate internationally for some time on the interface between population health and primary care, and it continues to be reviewed in light of the ongoing barriers in access to basic health care and to health determinants for many people in many nations.4–6

This paper will identify that while primary care and public health are separate areas of practice, the former emphasising personal health and the latter population health, both can adopt a population health approach, as well as contribute to the achievement of population health outcomes. Further, the paper will attempt to clarify the distinctions between population health, primary care, and related concepts for the primary care sector in Aotearoa New Zealand (see Table 1).

Table 1. Characteristics of population health, primary health care, and related terms

<table>
<thead>
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<th>EMPHASIS ON:</th>
<th>Population Health</th>
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* General practice is involved in individual-level disease prevention, for example screening, immunisation and smoking cessation, which contributes to population health outcomes; +++ Strong correlation; + Weak correlation; – Little or no correlation.

We begin by offering internationally-accepted definitions of the basic terms used.

**Defining primary care, primary health care and public health**

Primary care is that level of a health service system that provides entry into the system for all new needs and problems, provides person-focused (not disease-oriented) care over time, provides care for all but very uncommon or unusual conditions, and coordinates or integrates care provided elsewhere by others.7, pp 8–9.

A key component of primary care in New Zealand is the care delivered by general practice teams (usually GPs, practice nurses and their support staff) largely, but not wholly, through GP-owned small businesses. Within recent history, general practice
has emerged as a medical specialty with its own college. In New Zealand, a general medical practitioner (GP) is defined as follows:

A general practitioner is an appropriately qualified medical graduate who has particular knowledge and skills to provide personal, family, whanau, and community-oriented, comprehensive primary care. A GP’s care continues over time, is anticipatory as well as responsive, and is not limited by the age, sex, race, religion, or social circumstances of patients, nor by their physical or mental states.\(^8\)

The World Health Organization coined a broader term, that of ‘primary health care’, at the time of the Alma-Ata Declaration on Primary Health Care in 1978.\(^9\) The addition of the term ‘health’ added further meaning to the term ‘primary care’. Primary health care incorporates not only the first level of contact with the health system, but also care based on the following:

- A concern for equitable access to health services;
- The involvement of individuals and communities in developing strategies to improve their health; and
- A concern for addressing the social and environmental determinants of people’s ill-health.

Primary health care incorporates some of the key principles of a population health approach: those of equity, partnership with communities and a concern for social, economic, and environmental determinants of health. The orientation towards these principles is stronger in primary health care and health promotion than in primary medical care or traditional public health (see Table 1).

The New Zealand Primary Health Care Strategy (the Strategy) adopted this broad notion of primary health care, with emphasis on improving the key determinants of health. It called for an improvement in access to health care for disadvantaged groups and for community involvement in PHO governing and decision-making processes. It also highlighted the importance of engaging a wider range of health professionals—in particular, nurses—in the delivery of primary care, the development of a wider ‘primary health care team’ within a PHO (e.g. the inclusion of health promoters) and the value in engaging with organisations beyond the health sector.

The Strategy, therefore, requires primary care services, which deliver the majority of personal healthcare in community-based settings, to become part of primary health care services, which continue to offer that personal healthcare, while adopting a population health approach to achieving equitable population health outcomes.

Primary health care has been stated to sit at the interface of primary care and public health.\(^10\) Public health is “about promoting wellbeing and preventing ill health. It is focused on promoting the health of populations rather than treating diseases, disorders and disabilities in individuals”.\(^11\)

**Defining population health in primary health care**

If we are aiming for improved primary health care services through the Primary Health Care Strategy, what, then, is a population health approach? In the present primary care sector, one might get a different answer from different stakeholders. Public health policymakers, GP organisations, PHOs, and health promoters might...
each define it differently, and there could even be a lack of consensus within these stakeholder groups. The possible opinions about a population health approach in primary care might include the following:

- Not a ‘proper’ public health concept, but a useful way of engaging the primary care sector and GPs in public health issues;
- A term coming out of the bureaucracy (which, as such, will have limited life and impact, and can safely be ignored) that is useful for discussion with government, so they get a better understanding of how general practice does population health already;
- A useful term to improve the focus of the GP sector on inequalities and meeting targets for a population.

Similarly, the international health literature contains no universally accepted working definition of population health (see, for example, \(^{12, 13}\)). It is a broader concept than that of ‘public health’, encompassing both clinical activities in health services and broader actions that address the social and economic determinants of health. Poore, who coordinated the writing of an insightful New Zealand document on the contributions that both personal health and public health can make to population health,\(^{14}\) has identified the following features of a population health approach:

- A culture across the organisation (such as PHO or DHB) that places the same emphasis on promoting health and preventing disease as on treating illness;
- Investment in activities that influence the determinants of health;
- Operational commitment to reducing inequalities;
- Intersectoral and intrasectoral collaboration on local initiatives so that there are working partnerships and alliances with a range of community groups;
- Genuine community participation;
- Support for sustainable community development;
- Data collection that is comprehensive and considers ethnicity, deprivation and outcomes;
- Workforce development to support this wider population health approach.\(^{15}\)

As described in the Alma-Ata Declaration, primary health care has a goal of achieving health equity, through both community participation and working across sectors on the determinants of health. At a population level, these tasks are most readily achieved through PHOs, but they require the involvement of general practices.

**Population health in practice in primary health care**

What does a population health approach mean, in reality, for the daily work of general practitioners and primary care nurses? There are many activities of general practice that already contribute to population health outcomes, such as immunisation, antenatal care, health education, and screening.\(^5\) In carrying out such activities, it is evident that the population is ‘the client of care’, alongside individuals and families.\(^{16}\)
In caring for the population as ‘client’, a population health approach emphasises equity, community participation, and social determinants. Equity can be defined in clinical terms as ‘triage’; those who need services most are prioritised over those who need them least. Equity has implications for population-level funding of care nationally, regionally and within PHOs, but also for how services are delivered on a day-to-day basis.

For example, given the strong evidence for the poor access to primary care by Māori, and of their 7-year lower life expectancy than non-Māori in New Zealand, it would be equitable (in the sense of aiming to bring Māori health status up the level of other New Zealanders) to prioritise the needs of Māori patients in day-to-day clinical practice, and in the way the nation’s practices are distributed and funded. (For a useful resource on a Treaty-based approach to health programmes, see TUHA-NZ17).

In some areas, PHOs have utilised ‘Services to Improve Access’ funding to support Māori and Pacific patients, and those in the poorest quintile areas, to access cardiovascular screening and mental health assessments.

Along with a commitment to equity, a population health approach in primary care means a commitment to the involvement of communities (particularly disadvantaged communities) in determining strategies to promote health and to diagnose and treat illness within PHOs. If they were consulted, it is likely that members of disadvantaged groups, such as Māori communities or underserved populations (e.g. youth), would recommend many ways that primary care services could improve access for their population groups. For example, the employment of general practice staff that reflect patients’ ethnicities could be a recommendation.

The development of outreach clinics in schools or on marae could be another recommendation. Neuwelt18 has recently produced a toolkit for PHOs which outlines ways in which meaningful community participation in PHOs can be a tool for promoting equity.

Finally, a population health approach incorporates a concern for the determinants of health. The minimisation of the impacts of socioeconomic disadvantage on health is well within the scope of general practice. General practitioners and nurses have coined the term ‘patient-centred care’, which highlights the importance of context (social and environmental) to a person’s risk and experience of illness.19

Determinants of health are the context of people’s lives, including individual characteristics (e.g. gender, education, income, ethnicity) and the characteristics of society (e.g. income inequality, racism, the political system, population-level access to housing, and education). General practitioners and nurses serving high-needs populations know well the frustration of repeatedly offering treatment to an asthmatic child, for example, when the underlying determinant is damp housing.

Committed primary care practitioners are supported by the Primary Health Care Strategy to advocate for patients with, for example, housing (HNZ) or income (WINZ) agencies, and to link patients to community and specialist resources. Opportunities for population-level advocacy are equally important. Practitioners within general practices and PHOs are well placed to engage in advocacy for such determinants of health as access to adequate housing for the populations they serve.
Many individual practitioners and PHOs are undertaking innovative approaches to improving the health of the populations they serve. For example, a number of PHOs are involved in projects to improve the insulation of houses for families with health problems. The GP role in these is to refer patients to the programme, so it is a joint PHO-GP approach to prevention through addressing health determinants. Further exploration of the population health approaches being applied in the primary health care setting through PHOs is worthy of another paper.

**Conclusion**

The introduction of the Primary Health Care Strategy has offered opportunities to build and enhance the already changing roles of general practice and the primary care team. While clinical care is the essence of general practice, population health goals enhance general practice care, as identified by Weller and McWhinney in the opening quotes. The population health goals of primary health care are improved health equity through community participation and action on the determinants of health. These can be achieved through teamwork and collaboration.

The further development of a population health approach in primary health care requires the strengthening of existing teams including doctors, nurses, cultural brokers, health promoters, and community workers within primary health care. It also requires the building of relationships between primary care and public health, as well as between primary care and local communities. It is an expansive primary health care workforce, when seen in that light.

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Carbon pricing in New Zealand: implications for public health

Divya Dhar, Alexandra Macmillan, Graeme Lindsay, Alistair Woodward

Abstract

The likely health effects of climate change make it one of the most pressing global public health issues of our time. Effects range from more intense and frequent cyclones, flooding, and heat waves through to changing infectious disease patterns, food and water insecurity, sea-level rise, and economic and social disruption. The governments of almost all developed nations are now focusing their attention on national policy responses to the threat of climate change. In New Zealand, it is currently unclear what path our current government will take to contribute to the global response and fulfil our Kyoto obligations.

In this paper we discuss the main carbon pricing options currently under consideration, and their implications for health and health inequities in New Zealand. We summarise the literature about the likely health and equity implications of different kinds of carbon pricing policy. A health sector voice in these significant policy decisions is vital to ensuring a policy that both addresses the threats to wellbeing of climate change, and maximises the potential health and equity win-wins of an adequate and well-designed response.

There is wide scientific agreement that the Earth’s climate is changing and that these changes are very likely to be consequences of human activity. There is also growing agreement that the health effects of climate change make it one of the most pressing global public health issues of our time. Evidence suggests that the effects on health and wellbeing will be widespread and diverse.

Extreme weather events such as cyclones, flooding, and heat waves are predicted to increase in both intensity and frequency. As well as changes in infectious disease patterns and increasing foodborne illness, we need to consider the more widespread implications for health and wellbeing of food and water insecurity, sea-level rise, biodiversity loss, economic and social disruption. Variations in effects and abilities to adapt will exacerbate inequities both between and within countries. Our contribution to prevention and our ability to adapt will both be important for future quality of life in New Zealand.

Several approaches are available to governments for mitigating and adapting to climate change. It is unclear at present what path New Zealand will take: the Emissions Trading Scheme proposed by the previous [Labour] Government is now under review. However, it is highly likely that this country will introduce carbon pricing, in some form, as a component of climate change policy.

We argue here that the complexity and scope of the effects on health, resulting from both climate change and societal responses to climate change, mean that public health should be central to the policy debate.
In this paper we consider the potential effects on health and wellbeing of one particular response to climate change—carbon pricing, by which we mean an environmental surcharge on the cost of carbon fuels.

We propose ways of thinking about the balance between short- and long-term risks, including the potential impacts of climate change; and explore ways of implementing carbon pricing to maximise the “win-wins” for health and environment.

**Policy response options**

This year is significant for New Zealand and the world in terms of climate change policy. In addition to New Zealand reviewing its Emissions Trading Scheme, the most important meeting of the parties of the UN Framework Convention on Climate Change (UNFCCC) since the Kyoto Protocol occurs in December 2009 in Copenhagen. At this meeting, states will agree on a replacement for the Kyoto protocol. It is unclear at this stage what emission reduction targets will result from the Copenhagen talks.

The National-led New Zealand Government have called for a 50% reduction in emissions compared with 1990 levels, by 2050. However, this may not be enough to avoid serious damage. For instance, it is estimated that a 50% reduction would mean there was still a roughly 1 in 2 chance of a billion people being short of water in 2050, with that number doubling by the end of the century as temperatures worldwide continued to rise due to committed global warming. The UK recently called for an 80% reduction by 2050 compared to 1990 levels. This reflects recent updates in climate science reporting that greater reductions in emissions are now indicated.

As we delay the implementation of global and domestic responses, the levels and timeframes for emissions reductions become more drastic in order to be tolerably certain that there will not be unacceptable social and ecological impacts.

How should a reduction of this order of magnitude be achieved, in an equitable fashion? The Global Commons Institute has proposed an approach known as Contraction and Convergence. This involves a reduction in total emissions (contraction) in which the heaviest polluters make the greatest changes, and the outcome by 2100 is the same level of emissions per capita in all countries (convergence).

For contraction and convergence to be successful, a globally agreed, unified approach is necessary, with all countries paying heed to per capita as well as total emissions. The Organisation for Economic Co-operation and Development (OECD) countries (including New Zealand) contribute 40% of the world’s emissions.

Although New Zealand is a small contributor to overall emissions because of a small population, our per unit of GDP and per capita emissions are better indicators of performance on a global level. Of the 30 OECD countries, New Zealand produces the fourth highest greenhouse gas emissions per unit of GDP and the fifth highest emissions per capita.

As the pressure to act on climate change increases, governments around the world are weighing up strategies that will reduce carbon emissions with the minimum negative economic effect (Table 1).
Price instruments are likely to change behaviour more rapidly than information and education, whether this applies to household waste generation, energy, water, or transport use, and their effectiveness depends on the sensitivity of firms and consumers to changes in the cost of emissions.

Of the price-based mechanisms, carbon taxes have the broadest range, applying to all fossil fuels and all sectors, therefore supporting almost all forms of energy conservation and providing, potentially, a wide range of additional benefits. This is in contrast to tradable permits, which affect more directly large industrial emitters, and reach consumers by “trickle-down” price rises.

A combination of responses is most common internationally, but it is useful to bear in mind there are a number of important differences between taxing and trading carbon.

**Emissions trading**—The quantity of emissions is fixed (a “cap”) and the right to emit becomes a commodity to be traded on the domestic and international market. The cap is broken up into units or permits, and these are allocated (either sold, or assigned) to participating industries. To comply without cost, industries must emit less than the number of permits they hold. However, further permits or units can be bought. In theory, this means reductions in carbon emissions occur globally where the cuts are least costly. Depending on how the trading scheme is organised, costs and revenues may be largely contained in the private sector.

**Carbon taxation**—The marginal cost of carbon emissions is fixed, and this cost is paid at the point of consumption. This means the external costs of greenhouse gas emissions are paid directly. The cost of carbon is set by government and revenues return to central government as with any other tax.

Depending on the number of permits in circulation, an emissions trading scheme ties the market closely to the environmental target. In addition the cost of emissions adjusts automatically to the international economic climate, avoiding the “stickiness” of a tax, which takes time to review in the light of fluctuating economic markets. However, carbon taxes tend to be broader in scope, and they can more easily reach individual consumers as well as industries. This can result in a greater range of wellbeing co-benefits. In addition, it may be easier to moderate inequitable effects of carbon taxes: revenue flows directly to central government, and may (if government wishes) be utilised to reduce adverse impacts on vulnerable groups such as low-income households.

The previous New Zealand Government led by Helen Clark proposed tradable permits in the form of an Emissions Trading Scheme (ETS), as part of a suite of policies including direct regulation, improved education, and research. The scheme would...
require permits for any greenhouse gas emissions, which would be compulsorily relinquished to central government. The government would use some permits to fulfil its international obligations under the Kyoto Protocol, and the rest could be used in a number of different ways. Permits might be given back to industries that were unable to make rapid changes in their processes, or could not pass on costs to consumers.

Other permits could be auctioned to domestic industry or sold on the international market. The money raised in this way could be invested in low carbon technologies, adaptation to climate change, or whatever is required to ameliorate the social effects of the scheme. Australia is considering a similar kind of emissions trading scheme as recommended by the Garnaut Climate Change Review\textsuperscript{16} and discussed in a recent Commonwealth Green Paper.\textsuperscript{17}

The present New Zealand Government, led by John Key, has announced that it will review the ETS, as well as other carbon-reduction strategies such as taxes on fuel, congestion, and agricultural methane. Such taxes most directly affect the energy, transport, and agriculture sectors, with further flow-on effects for the cost of food and commodities.

Whatever combination of policies is chosen, there will be far-reaching consequences. In the following discussion we focus on the ways in which health and health inequities may be affected by moving to a system in which the full environmental costs are reflected more accurately in the price of carbon.

**Implications for human wellbeing**

The strategies outlined in Table 1 are currently being evaluated with a focus on economic effects. Measuring economic impacts captures some of the upstream "drivers" effects on health, but a more explicit examination of the health effects of these options is warranted. As part of the policy machinery for tackling climate change, a relevant model of health that reflects the links between economy, environment and wellbeing is crucial.

Trevor Hancock's model of human development\textsuperscript{18} demonstrates the inter-relationship between health (and social factors), environment, and the economy, and implies that economic activity serves not only to preserve the environment but also improve wellbeing (see Figure 1).

**Figure 1. Model of health and human development\textsuperscript{18}**
The Intergovernmental Panel on Climate Change (IPCC) Summary for Policy Makers recommends evaluating policies using a framework that mirrors this model (equity, environmental effectiveness, cost effectiveness) and adds institutional feasibility. Considering these aspects in relation to climate change mitigation, our first priority is to viability—thus ensuring a long-term level of carbon in the atmosphere that is consistent with human and ecosystem survival. Environmental and economic sustainability are closely intertwined.

The IPCC suggests that responding to climate change will have a net benefit to GDP for many countries, particularly those who develop and rapidly adopt new technology, creating vibrant low carbon economies, while avoiding the costs of negative climate change impacts. In addition a well-functioning Emissions Trading Scheme will reduce the economic impact of meeting Kyoto obligations. This leaves us with two more elements in Figure 1—health and equity.

**Effects of mitigation on health and equity (co-benefits and regressiveness)**

A well-designed carbon taxation scheme—or a combination of carbon taxation and emissions trading scheme—could achieve health co-benefits or “win-wins” for health. For example, a reduction in the burning of carbon will improve air quality. It has been estimated that air pollution from road vehicles and homes heating result in almost 1000 adult deaths per year in New Zealand urban settings.

Other positive health effects will depend on the availability of alternatives to fossil fuels, and the availability of affordable improvements to energy efficiency (influencing the sensitivity of consumer choices to price). For instance, where public transport, walking and cycling are convenient, safe, and affordable, transition to these modes as a result of increasing motor vehicle fuel prices will increase levels of physical activity, reduce road traffic injury, and improve opportunities for social connections.

Improvements in household energy efficiency, and clean heating technologies, could prevent excess winter mortality, and respiratory hospitalisations.

Depending on how it is implemented, carbon pricing may be regressive (i.e. a greater burden on the poor than wealthy people) or progressive in nature (a lesser burden on lower income people). For instance, there will be heavier economic demands on households as the increased cost of carbon is passed on through rising prices of products and services. This is regressive if costs bear most heavily on those with low incomes, and in these circumstances inequalities in health and wealth would be made worse.

In New Zealand, low-income households already spend a higher proportion of their income than high-income households on non-discretionary carbon-related expenses such as household fuel and power. Households in the lowest income quintile now spend 9.7% of their income on household fuel and power compared to 7.1% in 2004.
Even without an Emissions Trading Scheme or carbon taxation, the pressures on low-income households will continue if the price of energy rises. Fuel poverty—defined as households spending more than 10% of their income on fuel use to heat the home to an adequate standard of warmth—already affects between 10 and 15% of all households in New Zealand.50

Of all fossil fuels, the price of petrol is likely to increase most sharply as a result of a carbon charge.31 If people are unable to switch to more carbon-efficient modes of private or public transport due to lack of infrastructure, access, or affordability, then their ability to access employment, health facilities, and social and recreational activities is sharply impaired.

 Those on low incomes are more vulnerable for many reasons. For instance, in some areas there is an inverse relationship between access to public transport and neighbourhood deprivation in New Zealand.32 To compensate, people on low incomes may respond to the rising costs of travel by reducing spending on essentials such as nutritious food, household heating, electricity, and water—with predictable adverse consequences for health.

There are already significant pressures on food supplies in many parts of the world; for example, the world food price index rose by 40% in 2007 (compared with an increase of 7% the year before).33 Climate-related increases in fuel and agricultural costs, conversion of agriculture land to biofuel generation, and extreme weather events will exacerbate threats to food security.

In New Zealand, low-income households spend a greater proportion of their household expenditure on food than those on higher incomes.19 If carbon prices rose steeply, the flow-on effects on food costs would therefore cause a disproportionate burden on those already most vulnerable. The effect of a systemic carbon price rise will also increase inflation In New Zealand, exacerbating fuel and food inequities through a “welfare effect”.34

Modelling conducted by Suzi Kerr and Brian Easton on the proposed Emissions Trading Scheme indicates that a carbon price of $50 per tonne would contribute to job losses, but these would be less drastic than those associated with the market shocks of the 1980s and 1990s; and price rises would be less than the increases in oil and electricity prices in 2006–2008.35 These analyses were limited to examining average effects across society and did not focus on any potential differential impacts. Whether the carbon price will go beyond $50 per tonne is the major uncertainty—according to the IPCC, a price of up to $100 per tonne may be needed to reduce global emissions to the levels required to avoid damaging climate change. Social effects may depend also on the rate of change: rapid increases in prices (which of course are more likely the longer interventions are delayed) are tolerated less well than gradual changes. Research to date has not examined the possible effects on lower-income households of rapid adjustments of the price of carbon.

In summary, carbon pricing could bring important co-benefits, especially if alternative strategies to fossil fuel use for household energy and transport are convenient and affordable. However, health inequities could be made worse through the regressive nature of the scheme. Enhancing potential co-benefits and reducing the costs for low-income households should be central to the design and implementation of the ETS or
any other carbon pricing strategy. Experience with carbon pricing interventions elsewhere indicates these goals are achievable.

Evidence for policy implementation to maximise progressiveness

Internationally the best evidence on the effects of carbon taxation policies and emissions trading schemes comes from the European Union (EU). In the EU, carbon pricing has had regressive social effects, but there are also signs that the negative impacts can be softened, avoided altogether, or even reversed by revenue recycling. For instance, schemes that directed additional revenues specifically to improving energy efficiency and subsidising fossil fuel alternatives had a more progressive effect than those in which carbon revenues are used to reduce other taxes (such as income tax).36-44

The European experience shows that progressive allocations are possible, but it is not certain that additional revenue gained from a carbon tax would be spent in this way. Tax hypothecation, on the other hand, might be a way of earmarking new money for schemes that will reduce inequalities. For instance, revenue generated by carbon-charging might be legislatively directed to public transport and housing insulation for those in higher deprivation levels.

The potential for progressive outcomes across the income spectrum has been demonstrated in Italy.44 The introduction of a carbon tax in Italy resulted in significant rises in transport fuel costs, as well as rising household energy costs. However, the revenue from carbon taxation has been explicitly targeted to reductions in social security contributions; reducing taxation on heating fuels for the poorest and coldest areas; and improving the environmental efficiency of energy use.

International experience also indicates that regressive outcomes depend on balance of taxation between transport fuels and household energy, and the availability of alternatives. In New Zealand, demand for transport fuel is relatively inelastic due to the lack of convenient alternatives and therefore adding to the cost of carbon will be particularly regressive without some form of revenue recycling into (for example) better public transport in low-income areas.

Similarly, low-income households are already finding it difficult to respond to rising electricity costs, particularly in cold weather, due to poorly insulated houses and a lack of affordable, healthy heating alternatives. In response, revenues might be tied to subsidised insulation retrofits and other measures that improve household energy efficiency. This was the purpose of the Household Energy Fund proposed in October 2008, and now under review.

A version of tax hypothecation directed towards reducing inequalities has been recently accepted in New Zealand as part of the Auckland Regional Fuel Tax.45 The cost of fuel in the Auckland region will progressively increase by up to 5 cents per litre and the revenue will be reinvested into public transport projects. Prioritisation of low-income areas for improvements in public transport initiatives has been included in the scheme. Such direction has the potential to reduce economic inequalities by preferentially benefitting households in poverty.
Conclusions

In conclusion, if climate change is not controlled through timely central government means then health losses will occur worldwide. However, worldwide there is also the potential for a “triple dividend” for health if we get our policy response right.

An emissions trading scheme that establishes the right market price for carbon to achieve internationally agreed carbon targets could prevent the negative effects of climate change on environmental, economic, and physical wellbeing. However, a carbon taxation scheme that hypothecates revenues could also have significant wellbeing co-benefits and reduce socioeconomic inequalities—by improving the affordability and convenience of fossil fuel alternatives for low-income groups. Revenue recycling in this form also has the potential to reduce wellbeing inequalities.

Finally, by creating convenient, affordable fossil fuel alternatives in the transport and housing sectors, we make behaviour change towards healthier homes and transport easier to achieve long term. A carbon pricing scheme that reflects the key tax-shifting principles of broad coverage, predictable implementation, revenue neutrality, and protection of low-income households could create a favourable environment for addressing our climate change obligations while improving wellbeing and equity.

However, there is a risk that New Zealand's response to climate change will fall between two stools—making an insufficient dent in our obligations to mitigate the climate change impacts on wellbeing, while introducing a regressive policy that in itself is likely to increase health inequities.

The health sector remains one of the most respected sections of society. Health professionals must play their role in advocating for the “healthiest” kind of policy response to climate change—a policy that is predictable, structured to assist low-income communities, with revenues returned in ways that further benefit low-income households. As the global climate change rhetoric becomes more urgent, the organised actions of society to protect public health become more crucial.

Examples of activities for health professionals include: education and discussion with peer networks; encouraging health agencies to contribute to the public debate; and stressing that protection and improvement to human wellbeing is a strong justification for taking appropriate action on climate change.

In particular, submissions can be made by individuals and groups to the review of the Emissions Trading Scheme by Friday 27 February 2009 (late submissions are likely to be accepted by arrangement). Information about making a submission to the Parliamentary Review Committee can be found at http://www.parliament.nz/en-NZ/SC/SubmCalled/4/8/d/49SCETSreviewets200902131-Review-of-the-Emissions-Trading-Scheme-and.htm

There are opportunities to join in action with other health professionals globally in preparation for the next United Nations climate change conference in Copenhagen in December 2009. Contact the authors for more information.
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Cannabinoid hyperemesis presenting to a New Zealand hospital

Martin Watts

Abstract
Cannabis use is common among the general population in many areas of the world and cannabis is readily available in much of New Zealand. We report an unusual complication of chronic cannabis use which has recently been described overseas. Cannabinoid hyperemesis with abnormal bathing behaviour is a syndrome of episodic cyclic vomiting, often associated with relief being obtained by hot water bathing or showering. Underlying the disorder is the chronic use of cannabis. The proposed mechanism of the syndrome is accumulation of cannabis metabolites in the brain. Abstaining from cannabis use is likely to prevent ongoing recurrences. Although there have been limited reports in the medical literature, it is likely that the syndrome is commoner than previously recognised and the presentation could easily be confused with psychiatric illness or cyclic vomiting syndromes.

Case report
A 32-year-old man presented via ambulance to the emergency department (ED) with abdominal pain and severe vomiting. On arrival the patient was retching and vomiting and was distressed by episodic crampy abdominal pain. His illness had begun on the morning of admission following a 4-day prodrome of anorexia and nausea. His initial vital signs were noted to be normal, abdominal examination was unremarkable, and general examination revealed the patient to be uncomfortable with some distress, diaphoretic, and taking frequent sips of bottled water.

An intravenous line was established, bloods sent to the laboratory and he was commenced on intravenous saline and antiemetics. While in the ED the patient repetitively requested access to the shower facilities, having previously noted that having a shower provided relief of his symptoms.

Review of the patient's medical file revealed a history of a brief drug induced psychotic episode when he was aged 19 years old. At this time the patient was persistently positive on urine testing for cannabis use. Additionally in the 3-year period prior to the index presentation he had presented to the hospital 16 times with abdominal pain and vomiting, these admissions usually occurring two or three times over the course of several days, interspersed with several months of no presentations. The patient had been admitted as an inpatient on four occasions.

Investigations had included ultrasound scanning and upper gastrointestinal endoscopy, none of which had demonstrated a cause for his symptoms. Review of the notes showed that on at least five of the previous presentations the need for frequent showering had been documented in the nursing or medical notes. This had raised
concerns about the patient's behaviour in the ED, especially as he had become aggressive and agitated when denied access to showering facilities.

Laboratory studies showed no significant abnormalities. The patient's symptoms settled with analgesia, antiemetics and intravenous fluids over the following 12-hour period. He was allowed regular access to the ED showering facilities. While being treated, further questioning elicited a history of daily cannabis use by the patient since the age of 16 years. A literature search was also performed looking particularly for cyclical vomiting associated with cannabis use. Information regarding cannabinoid hyperemesis was accessed and the link with abnormal bathing behaviour noted.

Following resolution of symptoms the patient was able to be discharged home with information regarding the syndrome. He was offered counselling and community assistance with cannabis cessation, however he continued to smoke cannabis and subsequently has continued to present intermittently to the ED with recurrent symptoms.

Discussion

The syndrome of cyclical cannabinoid hyperemesis and abnormal bathing behaviour associated with chronic cannabis use was first described in the medical literature in Australia in 2004. Following this initial series, isolated case reports have appeared from other areas of the world including Europe and more recently North America.

The syndrome is still not well classified or understood and it is likely that many cases are not yet recognised. As shown in this case, the syndrome consists of cyclical vomiting associated with chronic use of cannabis. Associated symptoms (which were displayed by this patient) include sweating, polydypsia, and the need for frequent showering to relieve symptoms.

Cannabis has effects on many areas of the gastrointestinal tract and brain including areas affecting appetite and vomiting, and indeed cannabinoids have previously been studied for their antiemetic properties. Current theories relating to the cyclic syndrome suggest the cause may be accumulation of long-acting cannabis metabolites in susceptible individuals. There may be a genetic component and the quality of the cannabis and its method of cultivation may also be factors.

The bathing behaviour has been suggested to be due to its effect on thermoregulation and body temperature changes reducing the vomiting. Because of the components of chronic drug-use and the obsessive/compulsive need to bath or shower in an agitated patient, the syndrome could easily be confused with psychiatric illness.

The management of cannabinoid hyperemesis with abnormal bathing behaviour includes recognition of the syndrome and exclusion of other potential medical and psychiatric causes of recurrent abdominal pain and vomiting. The patient described had been investigated on previous occasions, including endoscopy, and no other cause for his symptoms were found.

Specific management has not been defined, but supportive care with the use of appropriate intravenous fluid rehydration, analgesia, and antiemetics would appear appropriate.
There is no reason to deny the patient access to showering or bathing facilities as they effectively reduce the distressing symptoms. Long-term management should look to address the cause of the syndrome: chronic cannabis use. Abstinence from cannabis will result in cessation of the syndrome.

Patients with cyclical vomiting syndromes presumed due to other pathologies should be questioned about cannabis use and urine testing for cannabis metabolites considered. Abnormal bathing behaviour, if noted, should increase the suspicion of the syndrome.

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Bilateral inferior visual field defects from a childhood near-drowning

Sohrabh Memon, Graham A Wilson

Near-drowning in children is a common problem and has been associated with a number of long-term injuries. Neurological damage following near-drowning can take a broad spectrum of outcomes ranging from death or a persistent vegetative state to complete recovery. More subtle cognitive difficulties have been shown in many patients with reported complete recovery in long-term follow-up.¹ ²

We report a case of a 31-year-old male patient in New Zealand experiencing a cold water near-drowning at the age of 18 months, presenting later with bilateral inferior visual field defects.

Case report

A 31-year-old male was referred from his general practitioner (GP) with a visual field defect after presenting for a routine examination for a heavy vehicle driving license. At the age of 18 months the patient suffered a near-drowning episode. His mother found him face-down in a motel pool. It was winter, snow was on the ground, and the pool very cold. It was estimated he had been in the pool for at least 10 minutes.

His mother described him as being “blue”, “frozen stiff”, and “swollen with water” but managed to get him breathing again herself using chest and abdominal compressions to release the water. The resuscitation was completed by a GP by placing him in a cold bath which was slowly warmed to 37°C over 3 hours. His mother recalls that later that day the little boy could see the airplanes on the motel wallpaper.

At the age of 24 months he was referred to an ophthalmologist due to parental concern that he was ‘always bumping in to things’. On examination at the time, pupil movements, visual acuity, and visual fields to confrontation were assessed as normal, and the child’s parents were reassured.

As a teenager he did well at school and played sport well, though there were ongoing concerns about clumsiness. There were no seizures or suggestion of neurological deficits, with normal speech, motor skills, and memory.

On examination at age 31 years, the visual acuity was 6/6 bilaterally. The eye and anterior visual pathway examination was normal with no optic disk pallor. Ishihara colour testing was full in each eye. There was a significant bilateral inferior visual field defect (Figure 1).

MRI scan showed normal optic nerves, chiasm, optic tracts and occipital cortex. There were no focal lesions. A diagnosis of bilateral occipital lobe ischaemia secondary to near-drowning was made.
Discussion

This case demonstrates the specificity possible with cerebral ischaemic injury secondary to cold water near drowning in a child. The association of specific visual field defects with near drowning is novel to the reported literature to our knowledge. It highlights the need for visual field testing by GPs and optometrists when there is a history of a past neurological event.

Near-drowning has been associated with a large range of possible neurological sequelae, ranging from having no functional deficits to brain death. There is little reporting of long-term follow-up in such patients with apparently initially normal outcomes as in this case. One case report has shown broad social and cognitive difficulties 6 years following a near-drowning, despite grossly normal neurological status.1

Retrospective reviews of cases of paediatric near-drowning have shown prognosis to be closely related with vital signs on presentation to hospital. In one study, all patients presenting with detectable pulse and blood pressure showed no ongoing neurological deficits at 2-year follow-up.2 In another, all children conscious on ED admission showed full neurological recovery.3

In cases of paediatric near drowning where the patient has no obvious neurological deficits on initial presentation it is unlikely that further neurological assessment would be extended to include full visual field testing. It is therefore possible for deficits such as that sustained in this case to have gone unnoticed.

Although no detectable changes within the occipital cortex were identified on MRI, bilateral occipital lobe ischaemia secondary to anoxia and systemic hypotension remains the most likely scenario in this case. There are many common variations of the calcarine artery’s vascular supply to the calcarine sulcus and in 20% of cases two branches (superior and inferior) have been demonstrated to exclusively supply the areas above and below the calcarine fissure.4 Therefore ischaemia to the superior wall of the calcarine sulcus is indeed plausible, although the predilection for superior as opposed to inferior is less explainable.

Watershed infarction is also a possibility as this has been demonstrated in the occipital lobe in experimental studies. Case reports of complete hemianopia resulting from occipital ischaemia in adults certainly do exist.5–7
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References:

Palatal perforation
Savino Sciascia, Anna Kuzenko, Maria Tiziana Bertero

Clinical
A 49-year-old woman presented to our centre with fever, chronic nasal obstruction with ulcerations, and bloody nasal discharge. Laboratory studies showed leucocytosis (WBC count of $13.8 \times 10^9$ cells/L).

Physical examination revealed a perforation of the midline palate and local tumefaction of the nasal pyramid with no other signs of systemic disease (Figure 1). A computed tomographic (CT) scan of the head-neck region showed an increased density in the maxillary and ethmoid sinuses and palatal destruction (Figure 2). High-resolution CT of the lung was normal.

Biopsy revealed chronic inflammatory process with giant cells, necrosis, and ulceration on epithelial cells.

What is the diagnosis?
Answer

Biopsy was consistent with *Wegener granulomatosis (WG)*. Further laboratory studies showed elevated cytoplasmic-pattern antineutrophil cytoplasmic antibody (c-ANCA). A diagnosis of *limited WG* was made.

Surgical correction (including necrectomy and immunosuppressive therapy with cyclophosphamide associated with steroids) was performed with temporary benefit.

Discussion

Local complications of WG include chronic rhinitis, sinusitis, epistaxis, and in rare cases, palatal perforation. Other causes of palatal perforation include infections (syphilis, tuberculosis, or fungal infection), cocaine abuse, sarcoidosis, neoplasms (salivary or squamous cell), and midline lethal granuloma, a type of T-cell lymphoma.

Localised forms of WG are characterised by limited disease that involves only the upper airway. Diagnosis in limited forms is complex because histology is diagnostic in only 50% of cases, and only 60% of patients have a positive c-ANCA level.

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Severe headache when standing

Osman Temizoz, Hakan Genschellac

Clinical

A 47-year-old previously healthy man presented with a 3-year history of severe headache increased in the erect position and relieved in the supine position. He also had dizziness, nausea, and tinnitus. The pain was not relieved with common analgesics. He had no history of trauma, lumbar puncture, or surgery of any kind.

Neurological examination and routine blood laboratory analyses were within the normal ranges. Cranial magnetic resonance (MR) examination was performed to investigate the severe headache (Figures 1A and B).

Figure 1A

Figure 1B

What is the diagnosis?
The T1-weighted MR images show diffuse thickening and enhancement of the dura (arrows). The clinical and MR imaging findings suggest a diagnosis of intracranial hypotension (IH).

Discussion

IH was first described by Schaltenbrand in 1938. It typically presents with postural headache, often associated with one or more of the following symptoms: nausea, vomiting, dizziness, vertigo, deafness, neck stiffness, and blurred vision.

The IH is the result of low cerebrospinal fluid (CSF) volume caused by either spontaneous or post-traumatic dural laceration. Also, it can result from a disc herniation, a minor head trauma, vigorous exercise, sexual activity, or a violent bout of coughing or sneezing. When, after thorough investigation, no cause of CSF leak is found, the condition is characterised as idiopathic or spontaneous hypotension.

Cranial MR imaging (MRI) is an important component of the current diagnostic criteria. MRI reveals diffuse dural thickening and enhancement, subdural fluid collections or haematomas, downward displacement of the cranial contents, and pituitary hyperaemia.

Conservative procedures such as bed rest and hydration are utilised to treat IH. If these are ineffective, then continuous epidural saline infusion, epidural fibrin glue injection, or epidural blood patch are second-order treatment alternatives.

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Editorial: meeting in Napier

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THE annual meeting of the Branch held in Napier was undoubtedly one of the most successful meetings of recent years. The papers read were not perhaps of much scientific importance, but they were interesting and valuable for many reasons. The chief value of the meeting, however, consisted in the proof that it gave of the unanimity of the Profession on all essential points, and of the high standard of Professional honour which it was determined at all costs to maintain.

There were many matters of great importance to the Profession, which were fully discussed by the 40 or more representative men who were there from all parts; and this discussion, and the clearing of the air which resulted from the mutual interchange of views, cannot fail to prove of the utmost advantage.

The intricate and thorny question of the Friendly Societies was discussed fully in all its bearings, and measures were taken which are certain to lead eventually to a readjustment of the prevailing conditions. Concerning our own Journal, some valuable indication of the wishes of the majority were obtained, and the discussion made it evident that the reading of papers at Divisional meetings before sending them to the Journal was to be encouraged as far as possible.

A serious attempt was made, at the suggestion of the Vice-President of the New Zealand Federation of Chemists, to devise a means of checking the repetition of doctors’ prescriptions containing morphia or other drugs, the too frequent use of which might become a serious danger to the patient. After many schemes had been suggested, it was ultimately decided to form a committee to go fully into the question.

The evil is a very real one, but the solution is difficult. It was thought, however, that some check would be put to the practice if doctors were to mark on their prescriptions how many times it might be repeated, and each chemist making it up were to mark it each time he repeated it.

A good idea was that of making the Hon. Mr. Buddo, the Minister of Public Health, an honorary member of the British Medical Association; it was found that the rules permitted a general meeting to confer that title in exceptional cases. The Minister was present during the discussion on the proposed amendments to the Medical Practitioners’ Act, and expressed himself as entirely in favour of something being done.

Another point it was decided to urge on the Government was that sera and vaccines should be admitted duty free. The Council decided unanimously that there was no more reason why racing clubs should have honorary surgeons than honorary solicitors; that the post was generally accepted thoughtlessly, and merely because the doctor was asked to allow himself to be nominated, but that in some instances it was used as a means of advertising; that on all grounds the practice was a bad one and must he stopped, and that the doctors’ names must be removed from the race cards.
With regard to life insurance examination fees, the acceptance of any fee less than one guinea was vetoed. It was clearly pointed out that the doctor’s reputation was pledged by the report filled up, and that whether the questions asked were many or few mattered very little, the responsibility being the same in every case. It was decided to send a copy of the resolution to the insurance companies, and we feel no doubt whatever that they will see the fairness and justice of the claim and will readily assent to it.

The social side of the meeting was well catered for. The annual dinner was eaten at the Masonic Hotel, and the usual toasts were duly honoured. The Mayor of Napier contributed largely to the success of the meeting, and his genial hospitality was much appreciated. The President, Drs. Bernau and De Lisle were also prominent entertainers; and Dr. Henley was a most efficient secretary. The trips to Grasmere and Frimley gave the greatest possible pleasure to every one.
Screening for colorectal cancer (CRC)

Although colonoscopy is untested in randomised trials, many lay organisations and specialty societies advocate it as the preferred screening method. In this paper from Toronto the authors attempt to evaluate the association between colonoscopy and CRC deaths. Their population-based case control study involved over 10,000 cases and over 50,000 controls. And their conclusion was that colonoscopy is associated with fewer deaths from CRC. This association is primarily limited to deaths from cancer developing in the left side of the colon. They and an editorial commentator observe that a case control study is less useful than a prospective randomised trial.

The conclusion of the study (and the commentator) was that it appears that the procedure reduces mortality by 60–70%. Slightly disappointing but as the editorial points out “it would be remarkably high compared with screening for other types of cancer such as breast (a 25% cancer mortality reduction at best) or prostate cancer (no proven cancer mortality reduction).”


Second-generation or first-generation antipsychotic drugs in the management of schizophrenia

In this meta-analysis the authors compared nine second-generation antipsychotic drugs with first-generation drugs for overall efficacy (main outcome), positive, negative and depressive symptoms, relapse, quality of life, extrapyramidal side-effects, weight gain, and sedation. The second generation drugs were: amisulpride, aripiprazole, clozapine, olanzapine, quetiapine, risperidone, sertindole, ziprasidone, and zotepine. In 95 of the 150 trials analysed the second-generation antipsychotic was compared with the high-potency first-generation antipsychotic haloperidol.

Four of the second generation drugs (amisulpride, clozapine, olanzapine, and risperidone) were better in overall efficacy. All induced fewer extrapyramidal adverse effects than haloperidol. There seems to be a clear message here. However, an editorial commentary notes that the use of haloperidol as the first-generation antipsychotic in these trials means that they were biased in favour of the second-generation drugs. This being so because haloperidol frequently causes extrapyramidal features, particularly in the higher dose range used in earlier trials. Maybe not so clear-cut after all.

Dexamethasone to prevent nausea and vomiting after tonsillectomy in children—probably not

Common complications of tonsillectomy are postoperative nausea and vomiting, pain, and bleeding. Nonsteroidal anti-inflammatory drugs (NSAIDs) have been widely used in this setting for their pronounced analgesic efficacy and a lack of the emetogenic effects. However, their antiplatelet activity is of concern. So what about dexamethasone which has good antiemetic properties?

In this Swiss trial, children were randomised to dexamethasone in one of three doses (0.05, 0.15, or 0.5 mg/kg) or placebo intravenously. The two higher doses of dexamethasone were effective in preventing nausea and vomiting. However, postoperative bleeding was much more common in the dexamethasone treated subjects, particularly in those receiving the 0.5 mg/kg dose. The trial was stopped for safety reasons because of the increased rate of postoperative haemorrhage related to dexamethasone.


Cognitive decline in ageing doctors

Knowing when to give up practice is an important decision for most doctors and a critically difficult decision for some. It is also an important matter for their patients. In this paper two members of the Medical Practitioners Board of Victoria examine various aspects of the problem. They note that there are no agreed guidelines to help Medical Boards decide what level of cognitive impairment in a doctor may put the public at risk. They have conducted a MEDLINE search of the literature with a variety of keywords and found that this provided little help. They make the point that practitioners with early dementia often lack the insight to accept that they are no longer able to practise safely.

So do they have any solutions? Obviously continuing professional development programmes and re-certification offer some help. The question arises—should there be a more proactive approach? A difficult problem.


Azathioprine or methotrexate—which drug is the safest as a maintenance immunosuppressive?

These agents are commonly used as immunosuppressants in a wide range of diseases—rheumatoid arthritis, psoriatic arthropathy, polymyalgia rheumatica, Wegener’s granulomatosis, and microscopic polyangiitis. In this paper a prospective comparison of these drugs has been made in the management of ANCA-associated vasculitis. Remission maintenance was equally good, about 80%, for both drugs and their adverse effects profile were not significantly different. The authors conclude that both drugs are reasonable alternatives in ANCA-associated vasculitis and we presume that this would also be true for the other diseases.

Removal of the requirement for schools to only sell healthy food a giant leap backwards

We find the decision by the Minister of Education to revoke the school National Administration Guidelines clause requiring only healthy foods to be sold in schools incomprehensible. The decision has reversed what was an extremely positive change for the health of New Zealand’s children.

Our reasons for opposing removal of the clause follow.

• The Minister, Hon Anne Tolley, states that the core business of schools is to provide students with a “quality…learning environment”. Students cannot be expected to benefit fully from such an environment if they are inadequately nourished or experiencing poor health because of poor nutrition. Research suggests that good nutrition is associated with better overall school and academic performance, intelligence, and psychosocial functioning. Thus, a healthy school food environment seems imperative for a quality learning environment.

• The Minister states that boards of trustees can “make their own decisions about appropriate food and drink options”. However, the reason the clause was introduced was that school food under boards of trustees was not sufficiently nutritious. A study in 2007 by Utter et al showed an association between use of school canteens in New Zealand and more frequent consumption of high fat and high sugar foods. An earlier study by Carter et al found that the most commonly available foods in primary schools were pies (79%), juice (57%), and sausage rolls (55%). There were over five times more unhealthy meals on offer than healthy meals, and filled rolls (a healthy option) were the most expensive item.

• It is unrealistic to expect boards to have the detailed nutrition knowledge necessary to ensure an appropriate and healthy food service, especially with their already heavy workload. The Minister’s decision means that whilst schools are still required to promote healthy foods, they must determine what foods are healthy and, of greatest concern, can continue to sell unhealthy food. This is contradictory and suggests ‘do as I say, not as I do’ to children.

• Relying on education alone has not worked for other public health issues such as smoking, seat belts, cycle helmets, and drink driving, which ultimately required regulation to bring about positive changes in behaviour. There is no reason to think education will work any better for tackling our obesity problem. If we expect people to eat healthily, the environment must be changed to “make healthy choices the easy choices”.

• Relying on nutrition education alone also assumes there is free choice in school canteens. However, food choices are constrained by the environment. If all that is available is food that is unhealthy and heavily promoted in the media, or if healthy foods are more expensive, then students don’t have a true
choice. Furthermore, younger children can not be expected to make rational choices about food based on health.

- Development and implementation of the Ministry of Education's guidelines on ‘Food and Nutrition for Healthy, Confident Kids’ was the result of a prolonged and extensive amount of work on the part of government, schools, health sector, and the food industry. That expense and resource was well justified given the potential long-term gains, but has now been completely wasted, without even evaluating its potential success.

- The Minister has also stated that there is confusion about the guidelines amongst schools. However, this could be resolved given sufficient time to work with schools. ERO report that since the clause came into effect in June 2008, 95% of schools had already implemented the guidelines.

- The fact that children bring unhealthy food into school or buy it out of school is not, as has been proposed, a reason to allow unhealthy food to be sold at school. The same rationale is not accepted in other circumstances. For example, it could never be acceptable to allow cigarettes to be sold at school because students could buy them at a local dairy.

- Whereas at one time New Zealand was seen as a global leader in our efforts to tackle obesity, this latest move by the National government puts New Zealand seriously out of step with other countries. The UK Government, for example, is making laudable efforts to tackle childhood obesity, including implementing regulations around school food, and embarking on plans to broaden the reach of such regulations to include other key environments such as restaurants and workplaces.

The future cost to the country of obesity and nutrition-related disease is immense. It is of the utmost importance that we support young people to develop healthy eating habits as they grow. This requires environments that support healthy food choices.

The current rates of childhood overweight (21%) and obesity (8%) show that existing environments are not conducive to healthier choices. Government intervention is justified to protect children and to prevent the high societal costs of nutrition-related disease. This does not make New Zealand a ‘nanny state’, but simply a caring state.

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Carbohydrate withdrawal: is recognition the first step to recovery?

In October 2008 we submitted a paper to a little-known medical journal proposing that high glycaemic index (GI) carbohydrates may be more ‘rewarding’ than other foods and that this may be responsible for the global rise in obesity observed globally over the last 30 years.¹ Our paper attracted little attention, until a British tabloid published a story based on our article on 4 January 2009.² Several television and radio interviews followed.

After the publicity we received a number of emails from persons who identified with the article. Some were relieved that the medical community had begun to consider obesity as an addiction rather than primarily a metabolic problem associated with imprudent food choices.

In the original article, I claimed that obese persons may experience a withdrawal syndrome (after abstinence from high GI foods) with symptoms such as craving and low mood, although I had little support for these claims in the medical literature.

Symptoms of carbohydrate withdrawal were thought to be similar to those associated with other drug dependencies. The only description we had found of food/carbohydrate withdrawal was reported by Atkins³ of an obese individual who had made repeated unsuccessful attempts to reduce his weight and experienced restlessness and tremors after short term abstinence from sugar. Sugar withdrawal has also been induced in rodents.⁴

Email correspondence extracts from a 38-year-old woman from Wisconsin, USA, received initially on the 1 of February 2009 (consent obtained for reproduction) follow:

…For the first 3 weeks I cut all processed sugar and flour from my diet and suffered mood swings with extreme tension and depression, even a sense of hopelessness at times, I had horrible stomach pains, all my joints and muscles throbbed, and I had the shakes constantly. I don't even know how to describe the horrible headaches that went along with all this. People who knew me started thinking I was hiding a drug problem. The worst physical symptoms have been gone for about 2 weeks now, and the cravings are finally starting to subside…I look at birthday cake today and all I see is myself curled up in the foetal position crying in bed. …The worst part of the addiction lasted 3 weeks. The first 3 days were normal, but then on the fourth day the worst cravings began. All I could think about was ice cream, chocolate, and cheesecake. The cravings started to subside after the third week, but once I started feeling better I [thought] about food less. The shakes and the headaches really were the worst part!

Before her diet changed, she reported a weight of 124 kg (BMI 41.0 kg/m²), that lowered to 114 kg (BMI 37.7 kg/m²) 6 weeks later. Similarly, her fasting venous glucose dropped from 7 to 6 mmol/L and her total cholesterol changed from 5.7 mmol/L to 4.6 mmol/L over the same period.

Although this case does not prove our hypothesis, it may explain why obese people find it difficult to adhere to advice to reduce intake of refined carbohydrates. Her description is similar to an opiate withdrawal syndrome (craving, aches and pains and
muscular spasm or twitching). The time course—worst in the first weeks and resolving with continued abstinence within 4 weeks—again concurs with a withdrawal syndrome.

Further work may indicate if these symptoms can be reliably measured and mapped over time in obese subjects that limit their intake of high GI food. The magnitude of health resource devoted to the treatment of obesity and its consequences argues that such work be prioritised.

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Barriers to a better management for acute coronary syndrome—insights from the Otago-Southland ACS Registry, 2000–2002

In 2004, after auditing the management of acute coronary syndrome (ACS) in Dunedin Hospital and a review showing declining in-hospital mortality rates over a 3-decade period, we thought that an overview of performance from two related hospitals in the Otago-Southland region might provide not only local but also real-world data on ACS (without the recruitment bias intrinsic to all randomised trials).

In its conception, acknowledging the retrospective nature of the review, we believed that patients’ characteristics should be fully captured and summarised as an “ACS risk score”. The score would then serve as a language for scientific communication as well as enhance statistical power in analysing the impact of therapies including both evidence-based medications and revascularisation.

Major findings of the 2000–2002 Otago Southland Registry—The Registry contains 1143 patients with 1057 hospital survivors. Among these survivors of ACS followed for 2 to 5 years for survival status, the Otago-Southland ACS Registry confirmed the prognostic benefit of evidence-based therapies (EBT) including statins and revascularisation during the initial hospitalisation. It also established the validity of GRACE score in predicting outcome through 4 years, and found that the score could potentially be improved by adding the simple parameter of blood pressure recorded before hospital discharge.

The central part in interpreting the registry findings is how to enhance practice of EBT given the geographic constraint with a relatively small population spread over a big area. A disparity of outcome was noted between the Dunedin and Invercargill Hospitals. Of note, there was a 2½-fold lower utilisation of angiography and revascularisation among patients first admitted to Invercargill Hospital—a finding echoing with those from a similar Waikato-Taranaki study on non-ST elevation ACS patients in 1999 and a 2-week national audit (NZACS) in 2002. These data all illustrated a disparity of health care where patients admitted to peripheral hospitals less often received invasive therapies.

Steps in enhancing EBT for ACS—insights gleaned from the Otago-Southland Registry:

1. To have the patients in the reach of the treatment facilities
   
   In the country areas, a special algorithm to channel ACS patients is much needed. In a separate prospective study, we found that a major factor for delayed presentation of ACS was that patients sought help from their general practitioners. Patients may not have perceived their symptoms as sufficiently serious to call an ambulance, or may have the false reassurance from having arranged a clinic appointment.
A recent article in the *Journal* has called for a national audit for the management in the pre-hospital phase of acute myocardial infarction, emphasising also the benefit from defibrillation to enhance survival in many patients. Perhaps another target is to get collaboration from general practitioners so that some form of triage algorithms is put into place, so that those with ischaemic symptoms are either seen immediately or diverted to ambulance transfer to nearby facilities.

2. **To establish standards in ACS management**

While National guidelines are constantly updated to enhance the appropriate use of EBMs, interventions can only be performed in the referral centers where higher procedural volume helps guarantee a good standard. In the Otago-Southland Registry, 19 patients died in-hospital with ACS despite having percutaneous or surgical interventions (14 with PCI alone, 2 with CABG alone, and 3 with both; unpublished data). This compared to 481 hospital survivors with interventions performed (358 with PCI alone, 104 with CABG alone, and 19 with both) and 67 hospital non-survivors who did not have interventions.

Among the 1057 hospital survivors, the benefit of interventions is clear over a 2 to 5 years follow-up period with an unadjusted hazard ratio for mortality of 0.29 (95% confidence interval 0.20 to 0.42). After adjusting for baseline differences and the use of statins, the hazard ratio was 0.39 (95% confidence interval 0.27 to 0.58). The benefit appeared most prominent among the NSTEMI patients.

3. **To enhance the collaboration between referring and referral hospitals**

Notwithstanding potential limitations of retrospective data collection, there was indeed evidence for a selection that younger and fitter (lower creatinine level and Killip class) patients were transferred for angiography, as shown in Table 3 of the article comparing 33 transfer cases with 97 non-transfer cases with NSTEACS. It is also interesting to note that prescription of statins was much higher in Dunedin Hospital than in Invercargill Hospital—an observation observed neither in NZACS nor Waikato-Taranaki study. This is possibly a reflection of tertiary center specialists advancing practice ahead of national guidelines.

While in-hospital mortality differed by only 3.3% (10.7% vs 6.4%), the sobering finding is that mortality difference widened to ~10% at both 6 months (19.1% vs 9.6%) and 12 months (22.1% vs 12.1%). Importantly, adjustment for use of EBM or angiography during the index admission only increased the hazard ratio reflecting an even higher mortality risk of the Invercargill patients. When the follow-up period was extended to 5 years (mean follow-up time of 43 months), the mortality difference was no longer significant. Since in this database the predictors of survival after initial discharge included statins use and revascularisation, the logical explanation for the worse 1-year outcome of the Invercargill Hospital is that sicker patients were actually “under-served” with respect to appropriate EBT including both statins and judicious revascularisation.
The potential under-treatment for sicker patients is a complex yet very likely a “generic” issue for many peripheral hospitals in New Zealand. Transferral between the Invercargill and Dunedin Hospitals is a 3-hour ambulance journey and for more serious cases retrieved by helicopters a 1-hour journey. Apart from cost and logistic constraints in arranging transfers, death during transfer is always a dreaded possibility with potential medicolegal implications, although this was not separately recorded in our database.

While decision on transfers should always be regarded as a mutual one between staff of the two hospitals, facing the “eye-catching” mortality differences required courage and dedication.

When these results were first known, my co-primary researcher (Dr EW Tang) met with all Invercargill consultants in December 2005. I extend my sincere thanks and deep respect to all of them for acknowledging the results and allowing its publication as well as progression to subsequent analyses, with an open mind dedicated to improving health care and assisting academic research.

Practice in Dunedin has changed in recent years, in that over two-thirds of cases undergoing coronary angiography are acute cases including many transferred from peripheral hospitals.

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References:


Discussing prostate cancer testing with patients

We note with interest Dr Robin Smart’s review article of 12 December 2008 (http://www.nzmj.com/journal/121-1287/3425), which discusses the recent information provided to assist primary health care practitioners discussing prostate cancer testing with patients. In response to Dr Smart’s comments, the New Zealand Guidelines Group (NZGG) would like to clarify the current situation with regard to the provision of this information.

In 2007, NZGG was contracted by the Ministry of Health to adapt (for the New Zealand environment) Australian consumer and practitioner materials regarding PSA testing. This work did not involve a systematic review of the literature regarding the effectiveness of prostate cancer testing in asymptomatic men. (The last systematic review of this literature done in New Zealand was in 2002.) The work was not intended to alter existing recommendations on population screening or act as an evidence-based guideline regarding screening for prostate cancer in asymptomatic men. Rather the resources were intended to provide clear information about prostate cancer and help inform the discussions between New Zealand men and their health practitioner when considering prostate cancer testing.

Dr Smart’s article cites a number of references supporting the testing for prostate cancer in asymptomatic men. At this time it is inappropriate to enter a debate regarding this data, given the recent Ministry of Health request for proposal for an update of the evidence. NZGG would, however, like to clarify the role of the evidenced-based guideline and emphasise the importance of a systematic review of evidence when attempting to objectively assess a complex and varied body of evidence in any clinical area.

NZGG’s primary function is the production of “evidence based guidelines”. These are guidelines in which recommendations are clearly derived from the outcomes of a systematic search of the existing evidence followed by the formal critical appraisal of this literature. These processes that are undertaken by independent experts (i.e. individuals with no professional connection to the field of research) who are specifically trained in the appraisal of research methods and study quality. This independent appraisal is critical—as evidence on any particular topic is of variable methodological quality, it is important that people skilled in the assessment of research methods conduct guideline literature reviews. This increases the likelihood of recommendations being based on valid and appropriate information.

NZGG strongly encourages continued interest and debate in this important topic, however we urge the health sector to keep in mind that, at this time, there is no up-to-date New Zealand independent systematic review of the research evidence (or related recommendations). Without such it is difficult to critique the pros and cons of the various approaches proposed. NZGG looks forward to working with the sector to progress such work.
References:


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For further information please consult the Deans of the Schools of Medicine or write to Professor A.D. Campbell, Honorary Secretary, Managing Trustees, Graham Aitken Nuffield Trust, C/- Department of Chemistry, University of Otago, P.O. Box 56, Dunedin.

Applications must be submitted to Professor Campbell by 31 March 2009
Drug Hypersensitivity


This is one of the first texts on the subject of drug hypersensitivity that covers such an extensive range of topics. Other textbooks on the subject focus on a single or limited number of areas or are specific to a particular organ of involvement or drug class. The book therefore has broad appeal with interest to those practising and/or researching in allergy, clinical immunology, dermatology, pharmacology, HIV medicine, microbiology, haematology, anaesthetics, nephrology, or hepatology.

The book is organised into 5 sections comprised of 33 consistently structured chapters that contain useful tables, figures, coloured photos, and up-to-date references.

The first section (2 chapters) reviews the epidemiology of drug allergy and outlines drug and host-related risk factors for its development. The second chapter specifically focuses on severe cutaneous adverse reactions (SCAR) such as Stevens-Johnson syndrome (SJS) and drug-induced hypersensitivity syndrome with emphasis on high risk drugs and observed latency periods between initial ingestion of the drug and onset of symptoms.

The second section (12 chapters) examines the pathomechanisms, genetics and animal models of drug hypersensitivity. The initial 5 chapters focus on immunopathogenesis. Both well established mechanisms such as haptenisation and novel theories such as the “p-i or pharmacological interaction with immune receptors” are discussed. A chapter is devoted to the exciting area of pharmacogenetics with emphasis on the strong influence of genetic susceptibility to SJS in Han Chinese in relation to allopurinol and carbamazepine use. One chapter deals with the adverse side effects to biological agents, which are being exponentially developed and increasingly used in clinical practice. There are three chapters that each describes hypersensitivity to an antiretroviral drug, which may have been better condensed to a single chapter. Another chapter advocates for better animal models in drug development that predict for and thereby reduces the risk of SCAR.

The third section (13 chapters) dedicates chapters to various generalised and organ-specific hypersensitivity diseases with an informative initial chapter on the current classification, causes and manifestations of various drug hypersensitivity reactions. A proportion of chapters are drug-specific (beta-lactam antibiotics, non-beta-lactam antibiotics, contrast media, and aspirin/NSAIDS). Other more specific topics include perioperative anaphylaxis and paediatric drug and vaccine allergy. These clinical chapters are detailed and comprehensive and do not provide information in a bulleted or boxed format for clinicians seeking swift and succinct direction.

The fourth section (2 chapters) is dedicated to in vivo and in vitro diagnosis and the fifth section (2 chapters) on desensitisation to antibiotics, chemotherapy agents and monoclonal antibodies. These sections do provide concise advice and protocols for...
physicians. However, the section on desensitisation should have been broadened to include protocols on cephalosporins, sulphonamides, aspirin and insulin.

I can recommend this textbook as a very useful reference for the clinician, pharmacologist, epidemiologist and researcher. The editor and authors have successfully negotiated the challenging task of describing and crystallising the protean and nebulous aspects entailed in the dynamic field of drug hypersensitivity.

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