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This Issue in the Journal

Classification of ‘healthier’ and ‘less healthy’ supermarket foods by two Australasian nutrient profiling models
Helen Eyles, Delvina Gorton, Cliona Ni Mhurchu

Nutrient profiling is the process of categorising or ranking foods relative to one another according to their nutrient profile/contents. The aim of this paper was to determine whether a modified version of the Heart Foundation Tick nutrient profiling model appropriately classifies supermarket foods as ‘healthier’. ‘Healthier’ products were used for promotion in a supermarket study. The modified Heart Foundation Tick nutrient profiling model was found to categorise supermarket products in a similar way to another widely used (modified) nutrient profiling model. Both nutrient profiling models were found to appropriately distinguish between ‘healthier’ and ‘less healthy’ foods. Therefore, the modified Heart Foundation Tick model appropriately identified ‘healthier’ foods for use in the supermarket study. In addition, both nutrient profiling models have the ability to help people make better food choices.

Human myiasis in New Zealand: imported and indigenously-acquired cases: the species of concern and clinical aspects
José G B Derraik, Allen C G Heath, Marius Rademaker

Myiasis is the infestation of live human and vertebrate animals with fly larvae (maggots). Reports of myiasis in humans in New Zealand are somewhat rare, and little attention has been paid to this issue in the local medical literature. More than 20 fly species that are present in New Zealand have been associated with cases of human myiasis overseas. Here we review the relatively few cases of myiasis either acquired in New Zealand or imported into this country on returning travellers. As many medical practitioners are unaware of myiasis or encounter it rarely, we provide a brief discussion of the clinical features and treatment.

An outbreak of Salmonella typhimurium phage type 1 associated with watermelon in Gisborne, January 2009
Lisa McCallum, Michelle Torok, Muriel Dufour, Alan Hall, Geoff Cramp

This paper describes a case control study of an outbreak of Salmonella typhimurium phage type 1 that was associated with watermelon in Gisborne in January 2009. Epidemiological and physical evidence suggested it may have been due to contaminated waste from birds, animals and/or humans; lack of temperature control may have also contributed. The authors suggest that melon growers keep fruit free from potential Salmonella-contaminated wastes and to chlorine wash watermelon prior to sale. Furthermore, cut watermelon should be kept chilled prior to sale.
Prevalence of sexually transmitted infections in men who have sex with men presenting to Auckland Sexual Health Service
Sunita Azariah, Nicky Perkins

HIV and infectious syphilis incidence has steadily been increasing in New Zealand in recent years and research has indicated that men who have sex with men (MSM) are at most risk of acquiring these sexually transmitted infections (STIs). This study is the most recent published research comparing diagnosis of STIs and possible risk factors in men who have sex with men attending a public sexual health service in NZ. STIs including HIV were diagnosed in men who reported high levels of consistent condom use confirming that other modes of transmission other than anal sex are implicated and that STI screening should be offered to all sexually active MSM even if they report consistent condom use.

Although most men reported reasonable levels of condom use, a significant minority reported rarely or never using condoms for anal sex and many men were engaging in concurrent relationships with multiple sexual partners and these factors are known to enhance STI transmission. 65% of the sample had used the internet for meeting sex partners and this offers potential in novel health promotion interventions at reducing STI and HIV acquisition in MSM. Better STI surveillance in New Zealand is required in order to improve our knowledge of STI trends in particular risk populations.
Driving disease emergence: will land-use changes beat climate change to the punch?

David Slaney, José G B Derraik, Philip Weinstein

In this issue of the *New Zealand Medical Journal*, Winkworth in her article “Land-use change and emerging public health risks in New Zealand: assessing *Giardia* risks” highlights two important points on which we would like to elaborate.

First, a global problem that often takes a back seat to issues such as climate change is the continuous modification of the natural environment due to land-use changes. These can have direct and indirect impacts on ecosystems, with downstream effects not only on the environment and the economy, but also on human health. Human activities can profoundly alter ecosystem functioning, on which we depend for the provision of basic services underpinning human well-being. Winkworth and a number of other researchers argue that land-use changes have contributed to the recent emergence or re-emergence of many infectious diseases.1-3

The second point is that investigations of health problems can often be assisted by taking a multidisciplinary approach. We would go further and state that a multidisciplinary (and multisector) approach is fundamental to adequately address wider human health issues. Such an approach makes it possible to identify interventions further up the causal chain, which ultimately may provide more cost-effective public health strategies than those treating solely the symptoms of the problem.

New threats to human health associated with components of global environmental change are not a recent phenomenon. Humans have been modifying the natural landscape for millennia, and it is argued that previous societies may have brought about their own demise, mainly via population growth and deforestation leading to degraded water supplies and increased incidence of water- and vector-borne diseases.4 Similarly, one of the primary forces altering the environment in the 21st Century is land-use change, particularly through accelerated urbanisation.5

Changes in land-use not only alter the basic physical properties of the environment (e.g. hydrology, soil structure, and topography), but also the biological make-up of an ecosystem, including the pathogenic and parasitic biota. These changes in turn lead to a disruption of the ecosystem and may increase human exposure to vectors and pathogens.

However, urbanisation is not the only form of land-use change altering human disease risk. Human activities such as agriculture, industry and mining, and their associated inputs (run-off or byproducts) into freshwater ecosystems can directly affect human health. Such activities are associated with increased incidence of water-borne diseases, including more frequent *Cryptosporidium* outbreaks, increased *Giardia* prevalence, and possible water-borne transmission of *Campylobacter*.6 *Giardia* in
particular, is a common protozoan agent distributed worldwide that is associated with a high disease burden.

The prevalence of infection for *Giardia* ranges from 1% to 30% in different parts of the world, with the highest levels occurring in countries with poor sanitation. The parasite can be acquired via drinking or swallowing contaminated water, eating uncooked contaminated food, or via contact with an infected person. In New Zealand, giardiasis is one of the most commonly notified enteric diseases and rates are high compared to other developed countries. Surveys of New Zealand’s freshwaters indicate that the pathogen is widespread in the environment.

It has long been recognised that water-borne pathogens in New Zealand are intricately linked to local ecological dynamics. *Campylobacter* for instance, exhibits complex spatial and temporal patterns of environmental prevalence and infection in humans. For this organism, land-use changes are considered a more significant driver of disease emergence than climate change. In the case of *Giardia*, Winkworth argues that environmental modification associated with land-use changes may be increasingly driving water-borne exposure to this pathogen. At worst therefore, climate change may compound the already significant burden of disease.

Also, as pointed out by Winkworth, a co-ordinated approach using different disciplines would likely make a more significant contribution to the investigation of emerging human health issues. For example, to devise the optimal response (or set of responses) to any particular ecosystem disruption, one requires a detailed understanding of that ecosystem. It is therefore advisable that multi-disciplinary investigations are carried out when considering the potential human health impacts associated with significant land-use changes.

In such scenarios, ecological studies are valuable for examining the distribution and abundance of a pathogen in a particular ecosystem, as well as its interactions with other species and the abiotic environment, thus providing a better understanding of the dynamics of a potential infectious disease. Therefore, in devising medical and public health responses, it would be useful to follow true and tried ecological principles.

The adoption of multidisciplinary (and multisector) approaches is consequently a fundamental tool to tackle wider human health issues. Solutions to health problems would likely flourish with a cross-disciplinary approach incorporating fields such as Medical and Veterinary Sciences, Environmental Health, Ecology, Geography, Social Sciences and Commerce, and it is good to note that there are examples of this happening in New Zealand.

The importance of such a holistic approach has recently received wider recognition, leading to a number of global initiatives to further this cause (e.g. [www.oneworldonehealth.org](http://www.oneworldonehealth.org); [www.onehealthinitiative.com](http://www.onehealthinitiative.com)). Unfortunately, it seems that currently ecosystem health is still lagging behind the interests of animal and human health, and it needs to be placed more firmly on the agenda.

Without healthy ecosystems, human societies will not be able to attain the necessary services to safeguard human well-being. Consequently, as exemplified from Winkworth’s article, land-use change may be a bigger elephant in the room than is
climate change and multi-disciplinary approaches to address emerging infectious diseases are more urgently required than ever.

**Competing interests:** None.

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**References**

Classification of ‘healthier’ and ‘less healthy’ supermarket foods by two Australasian nutrient profiling models

Helen Eyles, Delvina Gorton, Cliona Ni Mhurchu

Abstract

Aim To determine whether a modified version of the Heart Foundation Tick (MHFT) nutrient profiling model appropriately classifies supermarket foods to endorse its use for identifying ‘healthier’ products eligible for promotion in a supermarket intervention trial.

Methods Top-selling products (n=550) were selected from an existing supermarket nutrient composition database. Percentage of products classified as ‘healthier’ by the MHFT and a modified comparator model (Food Standards Australia New Zealand; MFSANZ) were calculated. Percentage agreement, consistency (kappa statistic), and average nutrient values were assessed overall, and across seven food groups.

Results The MHFT model categorised 16% fewer products as ‘healthier’ than the MFSANZ model. Agreement and consistency between models were 72% and kappa=0.46 (P=0.00), respectively. For both models, ‘healthier’ products were on average lower in energy, protein, saturated fat, sugar, and sodium than their ‘less healthy’ counterparts.

Conclusion The MHFT nutrient profiling model categorised regularly purchased supermarket foods similarly to the MFSANZ model, and both appear to distinguish appropriately between ‘healthier’ and ‘less healthy’ options. Therefore, both models have the potential to appropriately identify ‘healthier’ foods for promotion and positively influence food choices.

The term nutrient profile refers to the nutritional composition of a food or beverage. Nutrient profiling is the process of categorising or ranking foods relative to one another according to their nutrient profile. Several nutrient profiling models exist globally and are used for a variety of purposes, including front-of-pack labelling, food service policy, nutrition education, restriction of advertising of foods to children, and determination of the eligibility of foods to make health claims.

In New Zealand, the best-known and longest established nutrient profiling model is the Heart Foundation (HF) Tick, a voluntary self-funded signposting scheme that has been used by manufacturers in New Zealand and Australia since 1990 to promote foods that are healthier choices within their food category.

In 2007, a modified version of the HF Tick model was used to determine ‘healthier’ foods eligible for promotion in the Supermarket Healthy Options trial (SHOP), a large (n=1104), randomised controlled trial of the effectiveness of price discounts and tailored nutrition education for improving supermarket food purchases.

SHOP was undertaken in eight supermarkets in the Wellington region of New Zealand, from 2007 to 2009. The study protocol for the SHOP trial was approved by
the University of Auckland Human Ethics Committee (reference 2006/462) and all participants provided written, informed consent. (SHOP Trial Registration: Current Controlled Trials ACTRN1260700007437.) Further information regarding methods and findings is available elsewhere.8,13–15

At the time of trial development, the HF Tick was the only nutrient profiling model available in New Zealand specifically developed to categorise supermarket products.12 Underpinning the HF programme is a set of category-based nutrient criteria that promotes reduced sodium, trans fat, and saturated fat (negative nutrients), increased fibre and calcium (positive nutrients), and sets a limit on energy.12

The nutrient criteria for the HF Tick programme are developed and periodically reviewed by a Food Programme Criteria Working Group with representatives from New Zealand and Australia, and are based on the nutrient profile of food currently in the marketplace. Revisions to criteria are made according to HF nutrition policy, government strategies, nutrition science, and public health priorities.

Foods or beverages failing to meet any one nutrient criterion for the appropriate food category are deemed ineligible to carry the HF Tick (i.e. ‘less healthy’). The HF Tick programme encourages industry reformulation,16 and the Tick logo is currently present on over 2000 foods in New Zealand and Australian supermarkets.12,17

Current HF Tick criteria are available on the National Heart Foundation of Australia website for a select number of product categories.18 The September 2006 version of the HF Tick criteria was used for the SHOP trial.

Since the design of the SHOP trial, Food Standards Australia New Zealand (FSANZ) has developed the Health Claims Nutrient Profiling Calculator,11 a proposed nutrient profiling model to determine whether New Zealand and Australian supermarket foods are eligible to carry health claims.

The proposed FSANZ model is a modified version of the UK Food Standards Agency scheme developed to tighten controls around the advertising of food to children,19 and has been promoted as the future nutrient profiling model for use in the New Zealand marketplace. The UK model was rigorously developed and tested,1 and although still under development (current version=4),20 the FSANZ version has been tested on 10,000 New Zealand and Australian food products.11

In contrast to the category-based nature of the HF Tick model, the FSANZ Health Claims Nutrient Profiling Calculator is an across-the-board model where nutrient criteria are essentially the same for all foods and beverages. However, the FSANZ model specifies three types: (1) beverages and milk; (2) foods not included in categories one or three, and (3) most cheeses, edible oil, edible oil spreads, margarine, and butter.

The FSANZ model calculates baseline points on ‘negative’ nutrients such as saturated fat and sodium, and removes points based on ‘positive’ nutrients such as protein and fibre. Providing they meet additional qualifying criteria including scientific substantiation, foods scoring <1, <4, and <28 points for categories (1), (2), and (3), respectively are classified as eligible to carry a health claim (i.e. ‘healthier’).11

An effective nutrient profiling model is one that appropriately classifies foods in their chosen context consistent with dietary guidelines.11,21,22 Such models generally
consider nutrients that have a positive effect on health, such as fibre, as well as those that have a negative effect, such as sodium and saturated fat. Both the FSANZ and HF Tick models do this. Nevertheless, the HF Tick model has not been formally tested, and with regards to the internal validity of the SHOP trial, it was essential that the MHFT model was assessed to confirm that it appropriately distinguished between ‘healthier’ and ‘less healthy’ foods and beverages.

Furthermore, with the impending implementation of the FSANZ scheme, these two nutrient profiling models could co-exist in the Australia-New Zealand market-place, and thus it is important to know how these two models compare.

Table 1. Key criteria included in the modified Heart Foundation Tick and Food Standards Australia New Zealand Health Claims Nutrient Profiling Calculator models

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Modified Heart Foundation Tick Model (category specific)*</th>
<th>Modified Food Standards Australia New Zealand Health Claims Nutrient Profiling Calculator (per 100g/mL base†)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative nutrients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Saturated fat</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Sodium</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Sugar</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Positive nutrients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibre</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Protein</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Fruit content</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Vegetable content</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Dairy or soy content</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Nuts and/or seeds content</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Legume content</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Potato or kumara content</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Seafood content</td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Other criteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serving size</td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Nothing added (i.e. product sold in natural form)</td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Cooking methods (e.g. no frying methods)</td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Presence of skin, external and internal fat</td>
<td>√</td>
<td></td>
</tr>
</tbody>
</table>

* Criteria presented in this table are consistent with the modified version of the September 2006 HF Tick criteria, as used for the SHOP trial and this research. Not all nutrients or bases are relevant to all food categories. Note: The HF Tick criteria have evolved over time. Sugar is no longer a criterion, and a greater focus has been placed on energy and specific types of fat (saturated and trans fat) in many categories. Current criteria for some food categories are available at: http://www.heartfoundation.org.au/Professional_Information/Tick/Health_Professionals/Pages/TickCriteria.aspx

† Criteria presented in this table are consistent with the modified FSANZ (Version 2) nutrient profiling model. Full details of the criteria are available on-line: http://www.foodstandards.gov.au/foodmatters/healthnutritionandrelatedclaims/nutrientprofilingcal3499.cfm

There is no gold standard to validate nutrient profiling models. However, their performance and effectiveness are commonly tested by comparing the classification of key foods with expert opinion or by correlating food scores/classifications with
those of existing, robust models.\textsuperscript{25,26} This research aimed to compare the classification of a range of regularly purchased supermarket food products (n=550), based on their nutrient composition, using modified versions of the HF Tick model and the FSANZ Health Claims Nutrient Profiling Calculator.\textsuperscript{11,18}

The purpose was to determine whether the MHFT nutrient profiling model appropriately identified ‘healthier’ products eligible for promotion in the SHOP trial.\textsuperscript{8} Key criteria considered within the MHFT and MFSANZ health claims nutrient profiling calculator models are presented in Table 1.

\textbf{Method}

\textbf{Selection of food products}

550 frequently purchased food and beverages were selected from a food composition database of 3000 top-selling supermarket products, developed specifically for the SHOP trial.\textsuperscript{8} The top-selling 100 products from each of the following four core food groups were selected from the database: Fruit and vegetables; Cereal and cereal products; Meat and alternatives; and Milk and milk products. Fifty top-selling products were also selected from each of three non-core food groups: Fats and oils; Occasional (treat) foods (e.g. pies, pastries and pizzas); and Other miscellaneous foods (cooking sauces, dips and relishes, salad dressings and mayonnaise, stocks and soups, and general ready-made meals).

Products that required draining or reconstituting prior to consumption were excluded. Further information regarding development of the SHOP nutrient composition database has been published elsewhere and a list of excluded products is available on request.\textsuperscript{27,28}

\textbf{Applying the nutrient profiling models to selected food products}

\textbf{Modification and application of the HF Tick model—}Due to the selective nature of the SHOP database (n=3000 top-selling products), modifications were made to the HF Tick criteria to make them suitable for use in the SHOP trial. Modifications primarily focused on relaxation of selected nutrient cut-offs to allow more products to meet the ‘healthier’ criteria, for example, the sodium criterion for ‘healthier’ bread was relaxed by 50mg/100g.

In addition, all trans fat criteria were removed because these data were not available in the database. All modifications were made following discussion with Tick Programme team and were reflective of the New Zealand Food and Nutrition Guidelines for Healthy Adults.\textsuperscript{29}

\textbf{Modification and application of the FSANZ health claims nutrient profiling calculator model—} Version two of the FSANZ health claims nutrient profiling model was used for this research.\textsuperscript{11} Modifications were made to the FSANZ model due to its requirement to define percentage of fruit, vegetables, nuts and legumes (v points) for scoring.\textsuperscript{11} A list of standard v points was developed. For example: all plain fruits and vegetables were automatically awarded the maximum 8 v points; and milk and milk products were awarded the minimum 0 v points. For mixed foods such as pizzas and cereal bars it was not possible to allocate v points. Missing fibre values in the SHOP food composition database were obtained using the closest product match from the New Zealand Food Composition Database (NZFCD).\textsuperscript{30}

Products were grouped into their appropriate MFSANZ food type (1, 2, or 3). One of three algorithms (one for each food type) was applied to the nutrient composition information for each product, automatically classifying it as ‘healthier’ or ‘less healthy’ according to MFSANZ criteria.

\textbf{Statistical analysis}

The final list of 550 products was uploaded to SPSS v16 (2007) software for analysis. Descriptive analyses were undertaken to describe the number of products included from each food group and food category and the proportion of products classified as ‘healthier’ and ‘less healthy’ by the two nutrient profiling systems. Percentage agreement between the two systems was also assessed.
An inter-rater reliability analysis using the kappa statistic was performed to determine consistency across the two food profiling systems. Level of significance was set at 5%. The kappa statistic is a ratio statistic for categorical data that describes the proportion of times two raters agree, adjusting for the proportion of times that they are expected to agree by chance alone (kappa = (observed agreement – expected agreement)/(1 – expected agreement)). \(^{31}\) Values range from -1.0 to 1.0.

Kappa is considered a more conservative estimate of inter-rater agreement than percentage agreement because it takes into account that some of the agreement between raters was likely to have occurred by chance alone. \(^{31}\) A kappa of one indicates perfect agreement whereas a kappa of zero indicates agreement equivalent to chance. \(^{31}\) Kappa may be weighted to account for the degree of disagreement between raters and thus a weighted kappa is often used for ordered categories. \(^{31}\) Standard weights are automatically applied in SPSS and were used for these analyses. Descriptive analyses were also undertaken to determine the average nutrient values for ‘healthier’ and ‘less healthy’ products under each of the models.

**Results**

550 products from 7 food groups and 51 food categories were assessed (Table 2). The MHFT model categorised 236/550 (43%) of all products as ‘healthier’. The MFSANZ model categorised 326/550 (59%) of all products as ‘healthier’. With the exception of the fats and oils food group, a greater number of products overall in each food group were classified as ‘healthier’ under the MFSANZ model than under the MHFT model (Table 2).

On average across all 550 products, percentage agreement between the two nutrient profiling systems was 72%. For individual food groups, percentage agreement ranged from 84% for milk and milk products to 52% for miscellaneous foods (Table 2). Overall consistency between the two food profiling models was ‘moderate’, \(^{32}\) (kappa=0.46 (SE, 0.04); P-value, 0.00). For individual food groups, consistency ranged from ‘good’ for milk and milk products (kappa=0.67 (SE, 0.08); P-value, <0.001) to ‘poor’ for miscellaneous foods (kappa=0.07 (SE, 0.13); P-value, 0.59), \(^{32}\) the latter being the only food group where statistically significant agreement between the two models was not reached.

The distributions of the nutrient values of the products were found to be highly skewed. Therefore, average nutrient values for ‘healthier’ and ‘less healthy’ products were presented as medians (IQRs; Table 3). Overall, ‘healthier’ products were lower in energy, protein, saturated fat, sugar, and sodium than their ‘less healthy’ counterparts (within both models; Table 3). Using the FSANZ model, the median amount of fibre was similar between ‘healthy’ and ‘less healthy’ products. However, using the MHFT model, median fibre was lower in ‘healthier’ products than in ‘less healthy’ products (Table 3). The magnitude of differences between ‘healthier’ and ‘less healthy’ products within the models was similar, except for saturated fat, where the median value was higher using the MFSANZ model (median (IQR) = 7.4 (2.9 to 13.0) g vs. 4.8 (0.8 to 9.6) g for MHFT) and for sodium, where the median value was lower using the MHFT model (median (IQR) = 54.0 (6.0 to 296.0) mg vs. 133.0 (11.6 to 390.0) mg for MFSANZ; Table 3).
Table 2. Comparison of the classification of 550 regularly purchased supermarket foods by two nutrient profiling models

<table>
<thead>
<tr>
<th>Food group</th>
<th>Food category</th>
<th>Frequency</th>
<th>‘Healthier’ under modified FSANZ model (n)</th>
<th>‘Healthier’ under modified HF Tick model (n)</th>
<th>Number of same products classified as ‘healthier’†</th>
<th>Number of same products classified as ‘less healthy’‡</th>
<th>Percentage agreement</th>
<th>Kappa (SE; p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fats and oils</strong></td>
<td>Edible oil spreads</td>
<td>33</td>
<td>16</td>
<td>16</td>
<td>12</td>
<td>13</td>
<td>76%</td>
<td>0.57 (0.12; 0.00)</td>
</tr>
<tr>
<td></td>
<td>Vegetable oils</td>
<td>17</td>
<td>15</td>
<td>17</td>
<td>15</td>
<td>0</td>
<td>88%</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>50</td>
<td>31</td>
<td>35</td>
<td>27</td>
<td>13</td>
<td>80%</td>
<td></td>
</tr>
<tr>
<td><strong>Meat and alternatives</strong></td>
<td>Canned legumes</td>
<td>7</td>
<td>7</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>57%</td>
<td>0.49 (0.08;0.00)</td>
</tr>
<tr>
<td></td>
<td>Canned seafood</td>
<td>17</td>
<td>13</td>
<td>10</td>
<td>9</td>
<td>0</td>
<td>71%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Eggs§</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>0</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nut and seed spreads</td>
<td>7</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>43%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nuts and seeds</td>
<td>7</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Plain meat</td>
<td>11</td>
<td>8</td>
<td>7</td>
<td>7</td>
<td>3</td>
<td>91%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Plain poultry</td>
<td>7</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>14%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Plain seafood</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>0</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pre-sliced luncheon meats</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed meats</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>11</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed meat alternatives (e.g. tofu)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed poultry</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>71%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed seafood</td>
<td>7</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>43%</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>100</td>
<td>70</td>
<td>53</td>
<td>49</td>
<td>26</td>
<td>75%</td>
<td></td>
</tr>
<tr>
<td>Food group</td>
<td>Food category</td>
<td>Frequency</td>
<td>'Healthier' under modified FSANZ model (n)</td>
<td>'Healthier' under modified HF Tick model (n)</td>
<td>Number of same products classified as 'healthier'†</td>
<td>Number of same products classified as 'less healthy'‡</td>
<td>Percentage agreement</td>
<td>Kappa (SE; p-value)</td>
</tr>
<tr>
<td>------------</td>
<td>---------------</td>
<td>-----------</td>
<td>------------------------------------------</td>
<td>------------------------------------------</td>
<td>---------------------------------------------</td>
<td>------------------------------------------------</td>
<td>-------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Milk and milk products</td>
<td>Aged and processed cheese (e.g. Colby, edam)</td>
<td>17</td>
<td>3</td>
<td>7</td>
<td>3</td>
<td>10</td>
<td>76%</td>
<td>0.67 (0.08; 0.00)</td>
</tr>
<tr>
<td></td>
<td>Cream and alternatives</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>11</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Frozen desserts (e.g. ice cream)</td>
<td>16</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Milk and alternatives</td>
<td>24</td>
<td>16</td>
<td>16</td>
<td>13</td>
<td>5</td>
<td>75%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Un-ripened cheese (e.g. cottage cheese, feta)</td>
<td>16</td>
<td>10</td>
<td>5</td>
<td>5</td>
<td>6</td>
<td>69%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yoghurt and dairy desserts</td>
<td>16</td>
<td>15</td>
<td>14</td>
<td>14</td>
<td>1</td>
<td>94%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>100</td>
<td>44</td>
<td>42</td>
<td>35</td>
<td>49</td>
<td>84%</td>
<td></td>
</tr>
<tr>
<td>Cereal and cereal products</td>
<td>Bread</td>
<td>27</td>
<td>25</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>19%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Breakfast cereals</td>
<td>9</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>6</td>
<td>89%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cakes, muffins and other baked products</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>9</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cereal baked bars</td>
<td>9</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>7</td>
<td>78%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flour</td>
<td>9</td>
<td>9</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>11%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Plain grains</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>0</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed grains</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed pasta and noodles</td>
<td>9</td>
<td>7</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>56%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Savoury biscuits</td>
<td>10</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>60%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sweet biscuits</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>100</td>
<td>54</td>
<td>19</td>
<td>16</td>
<td>43</td>
<td>59%</td>
<td></td>
</tr>
<tr>
<td>Food group</td>
<td>Food category</td>
<td>Frequency</td>
<td>'Healthier' under modified FSANZ model (n)</td>
<td>'Healthier' under modified HF Tick model (n)</td>
<td>Number of same products classified as 'healthier'†</td>
<td>Number of same products classified as 'less healthy'‡</td>
<td>Percentage agreement</td>
<td>Kappa (SE; p-value)</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----------------------------</td>
<td>-----------</td>
<td>------------------------------------------</td>
<td>--------------------------------------------</td>
<td>--------------------------------------------------</td>
<td>-------------------------------------------------</td>
<td>-------------------</td>
<td>------------------</td>
</tr>
<tr>
<td>Fruit and vegetables</td>
<td>Canned fruit</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>38%</td>
</tr>
<tr>
<td></td>
<td>Dried fruit</td>
<td>8</td>
<td>6</td>
<td>8</td>
<td>6</td>
<td>6</td>
<td>0</td>
<td>75%</td>
</tr>
<tr>
<td></td>
<td>Frozen potato products</td>
<td>8</td>
<td>8</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>13%</td>
</tr>
<tr>
<td></td>
<td>Fruit bars (e.g. fruit leathers)</td>
<td>5</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>60%</td>
</tr>
<tr>
<td></td>
<td>Fruit juice</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>0</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td>Fruit pies, tarts and crumbles</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td>Fruit smoothies</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td>Fruit spreads</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td>Plain fruit</td>
<td>11</td>
<td>11</td>
<td>11</td>
<td>11</td>
<td>0</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Plain vegetables</td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>0</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Processed (canned/frozen) vegetables</td>
<td>13</td>
<td>13</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td>38%</td>
</tr>
<tr>
<td></td>
<td>Vegetable juice</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>100</td>
<td>85</td>
<td>66</td>
<td>62</td>
<td>11</td>
<td>73%</td>
<td></td>
</tr>
<tr>
<td>Occasional foods</td>
<td>Savoury pies and pastries</td>
<td>50</td>
<td>14</td>
<td>5</td>
<td>5</td>
<td>36</td>
<td>82%</td>
<td>0.44 (0.14; 0.00)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>50</td>
<td>14</td>
<td>5</td>
<td>5</td>
<td>36</td>
<td>82%</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Cooking sauces (e.g. pasta sauce)</td>
<td>11</td>
<td>9</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>64%</td>
<td>0.07 (0.13; 0.59)</td>
</tr>
<tr>
<td></td>
<td>Dips, relishes and similar</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>67%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salad dressings and mayonnaise</td>
<td>6</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>33%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stocks</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>General ready-made meals</td>
<td>7</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>29%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tomato sauce</td>
<td>6</td>
<td>3</td>
<td>6</td>
<td>3</td>
<td>0</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Soups</td>
<td>12</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>5</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>50</td>
<td>28</td>
<td>18</td>
<td>11</td>
<td>15</td>
<td>52%</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>TOTAL</strong></td>
<td>550</td>
<td>326</td>
<td>236</td>
<td>205</td>
<td>193</td>
<td>72%</td>
<td>0.46 (0.04; 0.00)</td>
</tr>
</tbody>
</table>

∗ The September 2006 HF Tick criteria and the FSANZ criteria were modified for the purposes of the SHOP trial and this research (as described in the manuscript)
† Number of same (individual) products that were classified as ‘healthier’ by both nutrient profiling models
‡ Number of same (individual) products that were classified as ‘less healthy’ by both nutrient profiling models
§ The eggs food category is not an included HF Tick category in New Zealand (Australia only)
Table 3. Mean nutrient values for 550 foods classified as ‘healthier’ and ‘less healthy’ by two nutrient profiling models*  

<table>
<thead>
<tr>
<th>Nutrient (Median (IQR) per 100g)</th>
<th>Energy (kJ)</th>
<th>Protein (g)</th>
<th>Saturated fat (g)</th>
<th>Sugar (g)</th>
<th>Sodium (mg)</th>
<th>Fibre (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Modified Heart Foundation Tick</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Healthier’ (n=236)</td>
<td>499.0 (231.6–1340.0)</td>
<td>3.3 (0.8–12.0)</td>
<td>0.8 (0.1–3.1)</td>
<td>2.2 (0.3–6.8)</td>
<td>54.0 (6.0–296.0)</td>
<td>0.4 (0.0–2.0)</td>
</tr>
<tr>
<td>‘Less Healthy’ (n=314)</td>
<td>1026.0 (670.5–1550.0)</td>
<td>6.2 (2.9–10.9)</td>
<td>4.8 (0.8–9.6)</td>
<td>3.0 (0.5–11.3)</td>
<td>391.5 (80.3–536.5)</td>
<td>1.2 (0.0–3.0)</td>
</tr>
<tr>
<td><strong>Modified Food Standards Australia New Zealand Health Claims Nutrient Profiling Calculator</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Healthier’ (n=330)</td>
<td>556.5 (260.3–1041.5)</td>
<td>4.4 (1.1–11.0)</td>
<td>0.8 (0.1–3.0)</td>
<td>2.2 (0.4–5.5)</td>
<td>133.0 (11.6–390.0)</td>
<td>0.9 (0.0–3.0)</td>
</tr>
<tr>
<td>‘Less Healthy’ (n=220)</td>
<td>1242.0 (878.0–1779.3)</td>
<td>5.3 (2.6–10.8)</td>
<td>7.4 (2.9–13.0)</td>
<td>4.2 (1.0–24.1)</td>
<td>390.0 (55.3–620.0)</td>
<td>0.8 (0.0–2.1)</td>
</tr>
</tbody>
</table>

* The September 2006 HF Tick and FSANZ criteria were modified for the purposes of the SHOP trial and this research (as described in the manuscript)
† Mean nutrient value of products classified as ‘healthier’ minus mean nutrient value of products classified as ‘less healthy’

For saturated fat, these differences appeared to be largely driven by the cereal and cereal products and miscellaneous food groups. For sodium, the differences were evident across all food groups except milk and milk products (data not shown).

**Discussion**

This study found that a higher proportion of products were classified as ‘healthier’ under the MFSANZ model compared with the MHFT model (60% vs. 43%, respectively). However, overall agreement was good (72%) and consistency was moderate (kappa=0.46), suggesting that the two models classify foods similarly. Statistically significant agreement (p<0.05) was observed between models for all food groups except Miscellaneous foods.

With the exception of fibre for the MHFT model, differences in average (median) nutrient values between ‘healthier’ and ‘less healthy’ foods within the models suggest they both appropriately distinguish between ‘healthier’ and ‘less healthy’ options.

The strengths of this research are that it compares two of the best-known and most extensively tested nutrient profiling schemes in New Zealand and Australia. In addition, this research focuses on top-selling supermarket foods, meaning the findings are highly relevant to the food purchases of many New Zealanders.

However, the following limitations should be considered: both nutrient profiling models were modified for the purposes of this research.

These modifications were likely to result in less-stringent versions of the models and as such the level of agreement might have been higher or lower than that which would be observed normally. With respect to the MFSANZ model, additional evidence required for higher level claims may have further affected results. Nevertheless, the current results were similar to those of another (unpublished) study comparing the HF Tick and FSANZ nutrient profiling models. A further possible limitation was the use of a selective food and nutrient database of 3,000 products; this meant some food categories contained fewer products than others within particular food groups.
Consequently, the number of ‘healthier’ and ‘less healthy’ products within some subgroups was small (Table 2). This may have skewed the nutrient values observed for some food categories and food groups, although would have had a lesser effect on overall averaged nutrient values. Further, the exclusion of drained and reconstituted foods may have affected the agreement observed overall and for the miscellaneous food category. Lastly, comparing the way two nutrient profiling models categorise the same foods is not a true measure of validity.

A better measure may be to compare the way each nutrient profiling model categorises a set of key ‘indicator’ foods that are both positively and negatively associated with healthier diets (i.e. whether the systems classify foods positively associated with health as ‘healthier’, and negatively associated with health as ‘less healthy’). Nevertheless, as nutrient profiling is a relatively new tool in public health, there is currently no accepted gold standard for testing the validity of food profiling schemes.

Only one other study to date has compared these two particular nutrient profiling models: Truong compared the classification of 281 commonly-consumed New Zealand foods and beverages across five nutrient profiling schemes, including the HF Tick and the FSANZ nutrient profiling calculator (standard versions). Truong used the first version of the FSANZ model of which the major difference compared with version used for this research (Version 2) was the moving of milk from Category 2 (Other foods) to Category 1 (Beverages). Similar to our finding of 72% overall agreement, Truong reported 76% agreement between the HF and FSANZ systems. However, the authors excluded fresh products such as fruits and vegetables (due to absence of a nutrition information panel) and butter (as deemed to be classified as ‘less healthier’ by most systems), which may partly explain the small difference in agreement observed between this and the current study.

Also similar to our results, Truong found a range of agreement across food groups and strong agreement for the classification of dairy products (mean agreement across the two food profiling systems ranged from 85% for desserts and snacks to 42% for vegetable oil spreads. Mean agreement for dairy products was 76%). The current study extends this research by including statistical comparisons and assessment of the average nutrient values of classified foods.

In the current study, inconsistencies observed in the classification of products across food categories and food groups were likely due to the differing frameworks employed by the two models (i.e. categorical for the MHFT model and across-the-board for the MFSANZ model). For example, a product high in fibre and sodium (such as a specific brand of breakfast cereal) would be classified as ‘less healthy’ under the MHFT model. However, this same product might be classified as ‘healthier’ under the FSANZ model if it contained enough fibre to offset its high sodium content.

Use of different nutrient cut-offs may have also contributed to the inconsistent categorisations observed between models, as could the incorporation of a number of food and nutrient components in the MHFT model that were not components of the FSANZ model (Table 1).

In summary, despite the fact that the nutrient profiling models compared in this research have differing frameworks and objectives, the MHFT nutrient profiling
model categorised regularly purchased supermarket foods similarly to the MFSANZ model and both appear to distinguish appropriately between ‘healthier’ and ‘less healthy’ options. Therefore, both models have the potential to appropriately identify foods eligible for promotion and positively influence food choices.

Good agreement was observed between the models for all food groups except Miscellaneous foods. This food group included condiments, stocks, ready-made meals, and soups.

If the HF and FSANZ nutrient profiling models should co-exist in the Australasian marketplace in the future, categorisation and labelling of such foods needs to be consistent between models to avoid consumer confusion. Future research examining the classification of key foods related to health outcomes (using original versions of the nutrient profiling models) would be useful for further validation of both profiling models.

**Competing interests:** None.

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Human myiasis in New Zealand: imported and indigenously-acquired cases; the species of concern and clinical aspects
José G B Derraik, Allen C G Heath, Marius Rademaker

Abstract
Reports of myiasis in humans in New Zealand are somewhat rare, and little attention has been paid to this issue in the local medical literature. A number of Diptera (fly) families present in New Zealand have been associated with cases of human myiasis: Calliphoridae (7 species), Fanniidae (2 species), Muscidae (3 species), Oestridae (4 species), Phoridae (3 species), Psychodidae (1 species), Sarcophagidae (2 species), Stratiomyidae (1 species) and Syrphidae (1 species). Despite these numbers, there have only been 6 published records and we obtained further 16 unpublished reports of myiasis acquired in New Zealand. Records of imported myiasis in humans are also rare, with only 2 published and 6 unpublished cases obtained. As many medical practitioners are unaware of myiasis or encounter it rarely, we provide a brief discussion of the clinical features and treatment.

Myiasis is defined as “the infestation of live human and vertebrate animals with dipterous larvae, which, at least for a certain period, feed on the host’s dead or living tissue, liquid body-substances, or ingested food”.¹ From a parasitological perspective myiases may be classified as obligatory, facultative or accidental.¹²³

Obligatory parasites—dependent on the host for a part of their life cycle.³ The larvae are deposited either directly on the skin or mucous membranes (e.g. Oestrus spp. and Rhinoestrus spp.), penetrate normal skin (e.g. Gasterophilus spp. and Hypoderma spp.), or become superimposed on pre-existing wounds (e.g. Chrysomya spp.).

Facultative parasites—normally free-living, with larvae developing in decaying organic matter,³ but which may occasionally contaminate living tissue, such as pre-existing wounds, ulcers and cavities (e.g. the genera Musca and Calliphora).

Accidental myiasis or pseudomyiasis—it occurs when the larvae of a normally free-living species are swallowed with contaminated food, passing through the alimentary canal where they may cause pathological reactions.¹

The literature on human myiasis in New Zealand is scarce, but recently two cases have been discussed in this journal, both of which were acquired overseas.⁴⁵ In this review, we provide a comprehensive account of human myiasis in New Zealand by: i) examining the Diptera species present in New Zealand that have been associated with human myiasis; ii) reviewing the published and unpublished records of human myiasis in New Zealand, which were either indigenously-acquired or imported; iii) briefly outlining the diagnostic and clinical features of human myiasis and its treatment, as few medical practitioners in New Zealand are acquainted with such a condition.
Clinical features of human myiasis

Myiasis in humans may lead to a number of clinical features. Cutaneous myiasis is characterised by infestation of the skin and subcutaneous tissue, and is mostly caused by the larvae of obligatory parasites, although a number of facultative parasites may be associated with wound myiasis. Cutaneous myiasis can be sub-divided into furuncular, creeping, wound and subcutaneous:

- **Furuncular myiasis**—boil-like lesions develop either as a consequence of larvae penetrating the skin directly (e.g. *Dermatobia* spp.), or by migrating from other parts of the body, most often the gastrointestinal tract. The lesions can be painful or tender, with patients often aware of a sensation of movement.

- **Creeping myiasis**—larva migrans or creeping eruption is commonly caused by the larvae of certain parasitic nematodes (*Ancylostoma* spp. and *Uncinaria* spp.), but cases of larva migrans from a number of Diptera species have been recorded, such as *Hypoderma* spp. and *Gasterophilus* spp. Lesions characteristically develop where the skin comes into contact with the ground, namely feet, buttocks and trunk. The larvae appear to penetrate through hair follicles and sweat gland orifices, and then ‘creep’ through the subcutaneous layer, forming a pruritic erythematous line.

- **Wound myiasis**—this tends to occur accidentally in neglected wounds, where larvae are deposited in suppurating wounds or on decomposing flesh. Species within the genera *Cochliomyia* and *Chrysomya* are the more common causative agents. The diagnosis is obvious when larvae are visible on the surface of the wound but more difficult when they have burrowed beneath the surface. It is worth noting that wound myiasis may be intentionally employed as a medical procedure (maggot debridement therapy - MDT), in which fly larvae reared artificially in sterile conditions are used to remove necrotic tissue. This treatment appears to have originated from observations of the beneficial effects of maggot infestations in the wounds of injured soldiers. The most widely used species for MDT is *Lucilia (=Phaenicia) sericata* (Figure 1) due to its preference for feeding on necrotic over healthy tissues.

- **Subcutaneous myiasis**—in this type of myiasis the larvae (e.g. *Hypoderma bovis* and *H. lineatum*) penetrate the subcutaneous tissue where they may remain for long periods, causing reddish, painful and oedematous masses that may develop into more classical furuncular myiasis. They can also induce a number of other dermatological eruptions including urticaria and erysipelas. More common sites are submaxillary, scapular and inguinal areas.

Myiasis may also affect body cavities such as the ears, eyes, nose, and genitals, as well as the gastrointestinal tract:

- **Ocular myiasis** (ophthalmomyiasis)—may be external involving the eyelid or conjunctiva, or it can involve deeper structures of the eye itself. It is most commonly caused by *Oestrus ovis*, but it may be associated with other genera such as *Hypoderma* spp. Patients present with conjunctivitis, tear formation and the sensation of a foreign body in the eye. Vision may be impaired or lost,
and more serious pathologies including death may result in the most severe cases.

- Nasal myiasis—also most commonly caused by *Oestrus ovis*. Symptoms include a burning sensation of the nasal mucosa, often accompanied by epistaxis. It may be complicated by sinusitis, pharyngitis and rarely, meningitis.

- Aural myiasis—it occurs mainly as a complication of chronic ear infections. Perforation of the tympanic membrane can lead to mastoiditis and rarely, meningitis. Symptoms include hearing loss, tinnitus, pain and haemorrhage.

- Urogenital myiasis—has been reported to be caused by a number of genera. Symptoms may include discharge, abdominal pain and secondary infections. Urinary tract myiasis is usually caused by migration of larvae from bladder to the urethra, with symptoms as those of cystitis and urethritis.

- Gastrointestinal myiasis—it is primarily pseudomyiasis, and is associated with the ingestion of larvae, leading to signs and symptoms similar to those associated with intestinal parasites.

**Figure 1. Lucilia sericata.** Although an agent of human myiasis, this species is medically employed in maggot debridement therapy (adult photo courtesy of John Carr; larvae photo believed to be in the public domain)

**Myiasis-causing flies established in New Zealand**

There are no native fly species in New Zealand that are known to have caused myiasis in humans. Although numerous introduced species of Diptera present in New Zealand cause myiasis, most are not commonly associated with human cases. Nevertheless, a number of these have been recorded to cause human myiasis overseas (Table 1) and, in some rare instances, in New Zealand as well (Table 2).

Most of the species listed in Table 1 are not obligatory but rather facultative parasites. One such species is the common house fly *Musca domestica* (Figure 2), which is associated in particular with wound myiasis (Table 1). Although this species is
extremely widespread and abundant throughout the world, human myiasis caused by *M. domestica* appear to be relatively rare.\(^9\)

A number of the species listed in Table 1 are obligate parasites of other mammals. For example, 15 years ago it was estimated that at least NZ$30–40 million in annual losses were accrued by sheep farmers in New Zealand\(^{111}\) due to myiasis associated with the blow flies (Calliphoridae) *Lucilia (=Phaenicia) cuprina*, *Lucilia (=Phaenicia) sericata*, *Calliphora stygia* and *Chrysomya rufifacies*.\(^{10-12}\) These species are also occasionally found on other livestock such as goats and cattle,\(^{11}\) but may cause myiasis in humans (Table 1).

*Lucilia sericata* in particular, seems to be associated with human wound myiasis in some countries,\(^{13}\) and less commonly in other forms of myiasis. Other species occasionally associated with ovine myiasis such as *Calliphora hilli* and *C. vicina* are also implicated in human myiasis (Table 1). In New Zealand, one case of aural myiasis caused by *Lucilia sericata* was recorded in the Waikato region (Table 3).

Recently two new introduced species of facultative parasites *Megaselia scalaris* and *M. spiracularis* (Phoridae) have been recorded in New Zealand.\(^{14}\) Larvae of *M. scalaris* have been associated with a number of cases of human myiasis, but human parasitism by *M. spiracularis* appears to be extremely rare (Table 1). Two other species of arguably lesser economic importance in New Zealand are *Oestrus ovis* and *Gasterophilus intestinalis* (Table 1). The only other member of the Oestridae in New Zealand, *Gasterophilus nasalis* is reported to be incapable of penetrating human skin,\(^1\) but has been associated with gastro-intestinal myiasis (Table 1).

**Figure 2. Musca domestica**, the common house fly (adult photo courtesy of Joseph Berger; larvae photo courtesy of Clemson University, USDA Cooperative Extension Slide Series).
Table 1. Diptera species present in New Zealand and records of human myiasis caused by them overseas. The list of Diptera was primarily based on available resources \cite{14,63,64} and on information from known authorities on this group.

<table>
<thead>
<tr>
<th>Family</th>
<th>Species</th>
<th>Type of myiasis</th>
<th>References</th>
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<tr>
<td>Calliphoridae</td>
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<td></td>
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<td>?Pollenia rudis</td>
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<td>Fannia canicularis</td>
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<td>Muscina stabulans</td>
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<td>Stomoxys calcitrans</td>
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<td></td>
<td>wound</td>
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<td>Gasterophilus intestinalis</td>
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<td>Gasterophilus nasalis</td>
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<td>Hydrotaea rostrata</td>
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<td>Oestrus ovis</td>
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<td>Megaselia scalaris</td>
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<td>nosocomial</td>
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<td></td>
<td></td>
<td>urogenital</td>
<td>95</td>
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</table>
Family | Species | Type of myiasis | References
--- | --- | --- | ---
Phoridae (cont.) | *Megaselia scalaris* (cont.) | wound | 1, 6
*Megaselia spiracularis* | gastro-intestinal | 1
 | pulmonary | 96
*Piophila casei* | gastro-intestinal | 1, 6, 12, 66, 97
 | nasal/oral | 6
 | urogenital | 98
Psychodidae | *Psychoda alternata* | gastro-intestinal | 6
 | ophthalmic | 99
Sarcophagidae | *Sarcophaga crassipalpis* | cutaneous | 13
 | ophthalmic | 100
 | wound | 6, 101
*Sarcophaga peregrina* | ? | 1, 102
Stratiomyidae | *Hermetia illucens* | cutaneous | 103
 | gastro-intestinal | 12, 104, 105
Syrphidae | *Eristalis tenax* | gastro-intestinal | 1, 12, 66, 72, 106
 | rectal | 107
 | urogenital | 12, 108, 109
† Identification uncertain.

Locally-acquired cases of myiasis

Despite the presence of the species listed in Table 1, cases of human myiasis acquired within New Zealand appear to be relatively rare (Tables 2 & 3). Seven cases were caused by the sheep botfly *Oestrus ovis*, and involved mainly ophthalmomyiasis externa (Tables 2 & 3).

*Oestrus ovis* is widespread in New Zealand sheep flocks, causing excessive mucus production and obstruction in the nasal passages, and occasionally pneumonia. There is some debate regarding the extent to which it leads to significant economic loss. More recently, it has been shown that light infestations may be well tolerated, but heavy infestations can cause losses in meat and wool.

*Oestrus ovis* (Figure 3) is an obligate parasite primarily of sheep and goats, but humans and other animals such as dogs may become accidental hosts. Unlike many fly species, *O. ovis* deposit live larvae (rather than eggs) that infest the host immediately. In their normal life cycle, the gravid female flies swarm around the heads of hosts, depositing larvae into the nostrils (and sometimes the eyes), and the larvae migrate into the nasal mucus membranes where they mature.

Interestingly *O. ovis* is capable of depositing larvae whilst still in flight, ejecting them onto the host. The stimuli for larviposition in *O. ovis* are not completely understood, but movement of a potential host is required, and perhaps light colouration, while the configuration of the human face has also been suggested as important.

The sheep botfly is regularly associated with human ophthalmic myiasis worldwide, but there are reports of nasal and pharyngeal myiasis as well (Table 1). Ophthalmic myiasis usually causes minor localised irritation, but it may lead to severe sequelae including disfigurement, blindness, and even death.
Table 2. Published records of imported and indigenously-acquired cases of human myiasis in New Zealand

<table>
<thead>
<tr>
<th>Origin of infestation</th>
<th>Species</th>
<th>Type of myiasis</th>
<th>References</th>
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</thead>
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<tr>
<td>Imported</td>
<td>Dermatobia hominis</td>
<td>cutaneous</td>
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<tr>
<td>New Zealand</td>
<td>Gasterophilus intestinalis</td>
<td>cutaneous</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>Oestrus ovis</td>
<td>ophthalmic</td>
<td>15, 16, 17, 18</td>
</tr>
<tr>
<td></td>
<td></td>
<td>nasal</td>
<td>15</td>
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</table>

Table 3. Unpublished accounts of imported and indigenously-acquired cases of human myiasis in New Zealand

<table>
<thead>
<tr>
<th>Origin of infestation</th>
<th>Species</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Imported              | ?Dermatobia hominis      | • ca. 1999. Female tourist arriving in New Zealand from Latin America. A single unidentified larva was removed from the patient’s skin in the occipital region. In view of the country of origin of the infestation, the species involved is presumed to be D. hominis (Joan Ingram, pers. comm. 2009).  
|                       |                          | • 2008. Cutaneous myiasis on tourist arriving from the Amazon region. Species presumed to be D. hominis (Kerry Read, pers. comm. 2009).                                                                     |
|                       | Dermatobia hominis       | • March 1999, Auckland Hospital. A 2nd-instar larva surgically removed from the right shoulder of a female tourist arriving in New Zealand from Bolivia (Trevor Crosby, pers. comm. 2010).  
|                       |                          | • April 1999, Auckland Hospital. A 3rd-instar larva removed from the leg of a female tourist arriving in New Zealand from Bolivia (Trevor Crosby, pers. comm. 2010).                                               |
|                       | Unidentified sp.         | • 2009. Waitakere Hospital. No details available, except that the patient was a New Zealander who had recently returned from a trip to South America (Fiona Larsen, pers. comm. 2010).              |
| New Zealand           | Eristalis tenax          | • Two cases of intestinal myiasis, the most recent of which was recorded in a patient from Blenheim in 2000. No further information is available (Graeme Paltridge, pers. comm. 2010). |
|                       | Lucilia sericata         | • April 1999, Waikato Hospital. Numerous larvae were removed from the left mastoid cavity of an 81-year-old male (Dallas Bishop, pers. comm. 2009).                                                        |
|                       | Oestrus ovis             | • January 1997, Hamilton. First instar larva removed from the eye of a human male (larva submitted to ACG Heath at the time by WG Elmsbury).                                                               
|                       |                          | • February 2005, Auckland Hospital. Three first instar larvae were removed from the eye (conjunctival sac) of a woman who lived in semi-rural Auckland (larvae provided to ACG Heath at the time by James Usher, LabPlus). |
|                       | Unidentified sp.         | • Waikato Hospital. Three cases of wound myiasis on leg ulcers on elderly patients, as a result of poor care (pers. obs.).  
|                       |                          | • Palmerston North. No date or details available, except that the patient had extensive myiasis on the leg (Scott Barker, pers. comm. 2010).                                      
|                       |                          | • 1995. Napier. Myiasis on leg ulcers of a male indigent, as a result of poor wound care (Ian McQuillan, pers. comm. 2010).                                                                                                                                 |
|                       |                          | • 2002. wound myiasis on a mentally ill woman from Canterbury as a result of poor wound care (Graeme Paltridge, pers. comm. 2010).                              |
Although the larvae of some flies may cause irreversible damage to orbital contents (e.g. Cochliomyia hominivorax), O. ovis ophthalmic myiasis is said to be self-limiting in humans, as the larvae generally do not develop beyond first stage in the human eye. As a result, the course of O. ovis myiasis is almost invariably benign conjunctival myiasis (ophthalmomyiasis externa) in healthy human hosts. Since O. ovis is widespread in New Zealand, and its clinical effects are relatively minor, it is likely that numerous cases of O. ovis ophthalmomyiasis externa go unreported.

One case of cutaneous myiasis in New Zealand due to Gasterophilus intestinalis (the horse botfly; Figure 4) has also been recorded. Horses are the primary hosts for this botfly, in which the larvae migrate through the animals’ alimentary canal to complete their life cycle. This does not occur in humans, and infestation appears to be limited to cutaneous myiasis. As with O. ovis, G. intestinalis appears incapable of developing beyond the first larval stage in human hosts.

Figure 4. Gasterophilus intestinalis, the horse botfly (adult photo courtesy of Robert Nash; larva photo courtesy of Kalumet)
There have been at least two recorded cases of intestinal myiasis caused by *Eristalis tenax* (Figure 5; Table 2), but no specific details have remained for any of the cases (Graeme Paltridge, pers. comm. 2010). *Eristalis tenax* (Syrphidae; commonly known as hover fly or drone fly) is a cosmopolitan species. There are occasional records of myiasis associated with it, particularly of accidental intestinal myiasis (Table 1) resulting from the ingestion of contaminated food. Clinical presentation is varied, and although it may be asymptomatic some patients may experience abdominal pain, nausea and vomiting.106

**Figure 5. Eristalis tenax**, the drone-fly or hover fly. Note the bee-like appearance of the adult and the characteristic ‘rat-tailed’ larva (adult photo courtesy of Fir0002/Flagstaffotos; larva photo courtesy of Jarmo Holopainen)

Despite the lack of published records, myiasis associated with infected wounds does occur in New Zealand (Table 3). Although we do not know the frequency of such occurrences or the species involved, these seem to be primarily opportunistic myiases associated with the elderly at home, as a result of poor wound care (pers. obs.; Graeme Paltridge, pers. comm. 2010).

Lastly, an article from Japan describes the case of a woman who apparently contracted cutaneous myiasis by the cattle warble fly, *Hypoderma bovis* (Oestridae), while travelling in New Zealand.33 This species has not established in the Southern Hemisphere,19 and it does not occur in New Zealand, although it has been introduced at least once on imported cattle from the UK (G. Adlam, pers. comm. 1977). Since *H. bovis* is established in Japan,32 the infestation most likely occurred in that country.

**Imported cases of myiasis**

Imported cases of human myiasis are a worldwide occurrence among travellers returning from the tropics.35–38 Although a few fly species may be involved, human cases appear to be caused primarily by *Dermatobia hominis* (Cuterebridae; Figure 6)33,35,37,39 and *Cordylobia anthropophaga* (Calliphoridae).35–38 Both species have a life cycle that alternates free-living and parasitic stages, causing primarily cutaneous myiasis.
Although these are particularly common overseas, cases of myiasis in travellers returning to New Zealand are rarely described. We have been able to ascertain the occurrence of only seven cases of imported myiasis in New Zealand, just two of which have been published in the medical literature (Tables 2 & 3). *Dermatobia hominis* was the likely culprit in six cases (certainly in two); a case of wound myiasis due to *Lucilia cuprina* imported from Fiji has also been recorded (Tables 2 & 3).

Surprisingly, there seems to be no recorded cases of *Cordylobia anthropophaga* myiasis imported into New Zealand (the larvae in Edwards’ was removed while overseas), but in view of its importance in cases worldwide we provide a more in depth discussion of this species and *D. hominis* as well.

**Dermatobia hominis**

The human botfly is widespread in tropical and subtropical Latin America, from the south of Mexico to the north of Argentina, and one report suggests that it is established in Saudi Arabia. The adult fly lays eggs on the body of anthropophilic insects which they catch, usually mosquitoes (Culicidae), but flies from six other Diptera families have also been implicated as vectors. Eggs remain attached to the vector and emerge upon contact with the skin of the host, eventually penetrating the skin and disappearing into the subcutaneous tissue. The range of hosts includes a large number of vertebrates such as humans, monkeys, most domestic animals, and birds. Although cases of *D. hominis* myiasis are primarily cutaneous, there are a number of records of ophthalmomyiasis. In some cases, the larvae may burrow into deeper tissues causing severe symptoms: deaths from larvae burrowing through the fibrous portion of the fontanelle of neonates have been reported.

The larvae are parasitic from the 1st to 3rd instars, taking 30 to 40 days for larval development to occur. Eventually larvae will abandon the host, falling onto the soil where they pupate, developing into adults some 30 to 60 days later. The incidence of *D. hominis* is directly related to suitable climatic conditions, preferring a high relative humidity and high mean temperatures (ca.20°C). Although *D. hominis* is present in subtropical South America, it seems unlikely that it would encounter...
suitable climatic conditions for establishment even in the warmest regions of New Zealand.

*Cordylobia anthropophaga*

The tumbu fly is widespread in sub-Saharan Africa, and it is a common cause of human myiasis. Cordylobia anthropophaga females lay egg batches directly on dry shaded ground, but these are also laid on laundry. As a result, cutaneous myiasis can occur from contact with infested clothing, leading to parasitism in normally unexposed areas of the body, such as the genitals.

Larvae hatch in 1–3 days but may survive for 9–15 days unnourished, until activated by the host’s body heat or movement. They are able to attach themselves and immediately burrow into the skin of an unsuspecting host, remaining at the site of entry, where they grow for 8–15 days in a furuncle-like lesion. Once the growth period is over, the third stage larva leaves the furuncle, falling to the ground to pupate. Apart from humans, *C. anthropophaga* is known to affect dogs and rats, but it is likely to also infest a range of other hosts.

Myiases due to *C. anthropophaga* are likely to be relatively benign as the larvae do not migrate into deeper tissues. Further, it seems that *C. anthropophaga* larvae secrete an antibacterial fluid, which may prevent secondary infection.

Prevention relies on ironing clothes prior to use, or drying them in full sunlight or under a mosquito net. Insect repellents are considered ineffective in the prevention of this myiasis. Although *C. anthropophaga* is a common cause of myiasis in travellers returning from endemic areas, the evidence that it has become established outside its African range and Saudi Arabia is poor and based solely on isolated case reports. These include cases in England, Netherlands, and Spain. A further report from Britain claims that the infestation was acquired in Portugal. However, although the patient herself had not visited any known endemic areas prior to her return to the UK, one cannot disregard the possibility that she had been in contact with contaminated clothing brought from Africa, as happened in cases acquired in England and Australia. One report from Japan describes successful emergence of a *C. anthropophaga* adult from a pupa at room temperature, but this is unlikely to have occurred outdoors, and even more unlikely to have successfully led to an established population. In view of its tropical distribution and the species’ lack of establishment in countries with warmer climates and greater frequency of imported cases, the risk of *C. anthropophaga* becoming established in New Zealand is considered to be very low.

**Diagnosis and treatment in New Zealand**

As is the case with other rare conditions, diagnosis of myiasis may be easily missed. However, since Diptera species able to cause myiasis in humans are present in New Zealand and the rate of international travel continues to increase, it is important that primary care physicians and nurses are aware of the clinical features of myiasis.
Patients often describe the sensation of movement under the skin in association with a lesion resembling a boil or furuncle. In the case of exotic species such as *D. hominis* and *C. anthropophaga*, such symptoms would be associated with recent travel history to the tropics, providing the attending medical practitioner with clues to reach an appropriate diagnosis. However, whilst the absence of recent travel to the tropics minimizes the likelihood of myiasis, it does not entirely exclude it, in view of the fly species present in New Zealand.

Numerous techniques have been employed to remove the larvae. In the case of furuncular lesions, occlusion of the skin pore for up to several hours to block the larva’s breathing orifice is a widely used method. This can be achieved with a variety of substances such as petrolatum, paraffin, beeswax, pork fat or chewing gum. These force the larva to protrude its posterior spiracle in search of air, consequently facilitating its removal. This is a useful technique as some botflies have a tapered shape with rows of spines and hooks which prevent simple extrusion through the central punctum (Figures 4 & 5).

When the larva surfaces for air, it can be manually extracted with the aid of forceps, with care not to puncture the larva. Alternatively, ethyl chloride sprays, liquid nitrogen, 15% chloroform in oil or 1% ivermectin cream have been used alone or in combination. Additionally, lidocaine can be injected into the base of the tissue cavity which the larva inhabits, thereby forcing the larva to the surface through hydrostatic pressure.

Pressure extraction by the application of slow, firm pressure to the sides of the lesion (similarly to squeezing an acne spot) is commonly used. A study in Ethiopia found that 87% of rural residents used this method to remove *C. anthropophaga* larvae. However, this can result in secondary infections, abscesses, severe inflammatory reactions and even fatal outcomes due to incomplete removal of the larvae. Therefore, it is important to extract the larvae in its entirety.

If the larva cannot be easily extracted, it may be necessary to enlarge the opening with a small incision under local anaesthetic. Alternatively, the whole lesion (and larva) can be primarily excised under local anaesthetic. It is important that the wound is thoroughly cleaned after removal of the larva.

For wound and cavitary myiasis, the cavity or wound can be irrigated with 15% chloroform in oil or soaked with 1% ether. Larvae can then be removed with forceps using an aseptic technique. Ivermectin has also been used for some cases of myiasis, particularly involving the eye and mouth.

**Conclusions**

It seems that the most important myiasis-causing species in New Zealand is the sheep nasal botfly *Oestrus ovis*. However, since such cases tend to be self-limiting, and the infestation is usually benign, it is likely that the majority of cases go unreported in the literature. As a result, the actual prevalence of myiasis in New Zealand is likely to be considerably greater than what is reported.
The increasing rate of international travel and the consequent greater number of travellers arriving from tropical regions is likely to lead to an increase in the number of cases being imported into New Zealand.

Fortunately, the main species involved are not likely to become established in New Zealand and are therefore unlikely to pose a biosecurity risk, although this risk assessment may change in the future under a climate change scenario. Nonetheless, it is important that medical practitioners are acquainted with the diagnosis and treatment of myiasis.

Also, in order to obtain an accurate estimate of myiasis incidence in the country, we encourage that such cases are appropriately recorded and/or published in the medical literature. For this purpose, assistance with larval identification can be obtained (ACG Heath; email: allen.heath@agresearch.co.nz).

Ideally, specimens should be preserved in a solution of 70% ethanol and 30% distilled water, but if necessary they may be also preserved in methylated spirits or spirituous liquors, which are likely to be available in most homes in New Zealand.

Competing interests: None.

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An outbreak of *Salmonella typhimurium* phage type 1 associated with watermelon in Gisborne, January 2009

Lisa McCallum, Michelle Torok, Muriel Dufour, Alan Hall, Geoff Cramp

**Abstract**

**Aim** To investigate an increase in *Salmonella typhimurium* phage type 1 (STM1) cases identified in the Gisborne region (eastern North Island, New Zealand) in January 2009.

**Methods** Initial investigations found that ham and watermelon were both consumed by a high proportion of cases. A case control study was conducted to determine if there was an association between cases of STM1 in Gisborne and consumption of ham or watermelon. Environmental investigations were conducted and included testing of ham and watermelon samples, as well as trace back of suppliers of these foods.

**Results** The case control study included 15 cases and 40 controls and found that cases were seven times more likely to have eaten watermelon compared with controls ($p=0.026$). Cases were one and a half times more likely to have eaten ham compared with controls ($p=0.620$). Pulsed field gel electrophoresis analysis determined that cases were caused by indistinguishable STM1 isolates. *Salmonella* was not recovered from any food samples. Trace back found watermelons were purchased from roadside stalls and came from one grower.

**Conclusions** This outbreak was associated with watermelon consumption from a grower in the Gisborne region. The outbreak was most likely controlled by the implementation of chlorine washing of watermelons at the grower’s pack house.

An increase in salmonellosis notifications was observed in the Gisborne region in late January 2009. Typing of isolates from Gisborne cases found that all notified cases were caused by *Salmonella Typhimurium* phage type 1 (STM1). An increase of STM1 cases was also seen in Auckland so further typing of STM1 isolates from Gisborne and Auckland was performed using pulsed field gel electrophoresis (PFGE). Pulsed field gel electrophoresis analysis demonstrated that STM1 cases from Gisborne were indistinguishable and were distinct from cases in Auckland and other regions. Initial case investigations found that nearly all cases had eaten watermelon and shaved ham.

A case control study was performed to examine the potential association between cases of STM1 with the Gisborne PFGE profile and consumption of watermelon or shaved ham.

**Methods**

**Case control study**—All cases were interviewed by a local Health Protection Officer, and samples of left over food were collected. Cases who agreed to participate in the case control study were subsequently re-interviewed by telephone. Trained interviewers used a standardized questionnaire.
which included questions about demographics, clinical symptoms, and exposure to potential risk factors for salmonellosis.

Risk factors included travel and fresh fruit and/or meat consumption. A case was defined as “a person who is infected with *Salmonella* Typhimurium phage type 1 with the ‘Gisborne’ PFGE profile, with onset of symptoms (diarrhoea and/or vomiting) after 01 January 2009, residing in New Zealand”.

Progressive digit dialling was used to recruit controls. The last digit of each case’s land line telephone number was increased by one and dialled. The person in the household to have the next birthday was asked to be interviewed. The process was repeated until three controls had been recruited for each case. This method gave a broad match on location. No other matching was performed. Each phone number was tried three times with at least two calls after 5pm. Controls were excluded from the study if they had been overseas during the last five days or if they had experienced symptoms of vomiting or diarrhoea at any time since 01 Jan 2009.

One case resided in Auckland, but was in Gisborne for their entire incubation period. The phone number used to recruit controls for this case was the phone number of the place the case stayed whilst in Gisborne. Where possible, the same interviewer conducted all interviews for a case and control set. Differences in the distribution of demographics between cases and controls were calculated using Pearson’s chi squared test. Crude odds ratios and 95% confidence intervals were estimated. P-values were calculated using Fisher’s exact test. Logistic regression was used to estimate odds ratios adjusted for age and sex. STATA version 9 was used for all analyses.

**Environmental investigation**—Trace back of the watermelon consumed by cases found that the majority of cases had purchased watermelon from roadside stalls belonging to a particular watermelon grower in the region. Health Protection staff visited the roadside stalls, as well as the watermelon growing patch and pack-house of the watermelon grower. Watermelons from the packhouse and fields were also collected for microbiological testing.

**Laboratory investigation**—Watermelons (both swabs of the outside surface of the melons and fruit pulp) from the grower in question, as well as leftover foods from cases, were tested for *Salmonella* at the Public Health Laboratory, Institute for Environmental Health and Research (ESR), Christchurch using standard methods. Stool samples from cases were tested at local community and hospital laboratories using standard methods. *Salmonella* isolates were then sent to ESR Enteric Reference laboratory for serotyping, phage typing and molecular typing (PFGE) using standard methods.

**Results**

**Case control study**—Nineteen cases of STM1 with the “Gisborne” PFGE profile were identified from 01 Jan 09 to 04 March 09. Of these, 18 met the case definition for the case control study. Two cases did not have landline phones and one case had moved overseas by the time the study was initiated so was not eligible for the study; one case was not interviewed, giving a response rate for eligible cases of 94%. The epidemic curve for this outbreak is shown in Figure 1.
Figure 1. Epidemic curve of the Gisborne *Salmonella typhimurium* phage type 1 outbreak (n=19)

Cases ranged in age from 5 to 79 years with a median age of 33. Sixty percent of cases were female (n=9). Most reported New Zealand European/European ethnicity (n=6, 40%), four reported Maori ethnicity (27%), one reported “other” ethnicity and the ethnicity of four cases was unknown. There were no statistically significant differences between cases and controls with regard to age group (p=0.10), sex (p=0.87) or ethnicity (p=0.61).

Table 1. Symptoms experienced by cases (n=14)

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>11</td>
<td>78.6</td>
</tr>
<tr>
<td>Fever</td>
<td>12</td>
<td>85.7</td>
</tr>
<tr>
<td>Chills</td>
<td>7</td>
<td>50.0</td>
</tr>
<tr>
<td>Vomiting</td>
<td>11</td>
<td>78.6</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>13</td>
<td>92.9</td>
</tr>
<tr>
<td>Headache</td>
<td>10</td>
<td>71.4</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>14</td>
<td>100.0</td>
</tr>
<tr>
<td>Muscle ache</td>
<td>11</td>
<td>78.6</td>
</tr>
<tr>
<td>Other*</td>
<td>4</td>
<td>28.6</td>
</tr>
</tbody>
</table>

*Other symptoms reported by cases: loss of appetite (n=2), low energy or weakness (n=2), low blood pressure (n=1) and sweaty (n=1).
Symptoms experienced by cases are shown in Table 1. Five cases (33%) were hospitalised, no cases died. The median duration of symptoms was 7 days (range 6 – 21).

Crude and adjusted odds ratios for exposures with significant p-values (p≤0.05) are shown in Table 2. Three cases reported close contact with symptomatic persons, one case reported social contact with symptomatic persons. After re-analysing the data excluding these cases the associations between illness and cutting up watermelon before washing and peeling, as well as illness and peach consumption, no longer reached statistical significance.

Cases had seven times the odds of eating watermelon compared with controls (adjusted OR 6.8; 95% CI 1.3-36.6; p=0.026). When we excluded cases with contact with symptomatic people from the analysis, cases had 10 times the odds of eating watermelon compared with controls (adjusted OR 9.9; 95% CI 1.5 -66.8; p=0.019). Although not statistically significant, cases had one and a half times the odds of eating ham compared with controls (adjusted OR 1.5; 95% CI 0.3- 6.4; p=0.620). Removing cases with contact with symptomatic people did not change the adjusted OR (adjusted OR 1.5; 95% CI 0.3 - 8.0; p=0.567). Nor did controlling for the consumption of watermelon (adjusted OR 1.5; 95% CI 0.3- 6.7; p=0.609).

After controlling for ham consumption, the adjusted OR for watermelon was 5.2 (95% CI 1.1- 24.2; p=0.034).

**Environmental investigation**—Health Protection staff found that the watermelons the grower kept at the roadside stalls were kept in the sun and were very warm. They also found the pack-house to be a tin lean-to with a bare floor. Rat faeces were found throughout the pack house floor and on a number of surfaces (tables, shelves etc) and birds were nesting in rafters above the packing table. The watermelon patch itself was located near a septic tank with the effluent disposal trenches possibly extending adjacent to or into the growing areas.

**Laboratory investigation**—All samples of watermelons and leftover foods from cases tested negative for *Salmonella*. PFGE analysis of isolates determined that cases were caused by indistinguishable STM1 isolates.

**Discussion**

The outbreak of *Salmonella typhimurium* phage type 1 in Gisborne was associated with the consumption of watermelon purchased from a road side stall in Gisborne. Although cases had increased odds of exposure to ham, this association was not statistically significant. Slicing the watermelon without washing and purchasing pre-cut watermelon were also associated with illness.

The epidemiological and environmental findings suggest that the watermelon surfaces may have been contaminated by bird, rodent and/or human waste, all of which have been implicated as sources of human salmonellosis. It is also possible that lack of temperature control may have contributed to the outbreak.

Although this is the first *Salmonella* outbreak associated with watermelon described in the literature in New Zealand, cut melon has been found to be associated with
salmonellosis elsewhere, and the US FDA have time and temperature requirements for the sale of cut melon.9

This study was relatively small, with only 15 cases and 40 controls, which limited our ability to detect significant associations. A sample size of 21 cases and 63 controls would be necessary to detect an odds ratio of 5.0 with 95% confidence and 80% power.

Recall bias may have been an issue in this study as the cases were re-interviewed for the case control study approximately 4 to 6 weeks post illness onset date. Cases were questioned about exposures in the five days prior to onset of symptoms (case exposure period). To minimise recall bias, we asked controls about the five days prior to interview rather than the corresponding case’s exposure period.

To determine the likelihood of controls having the same exposures during the corresponding case’s exposure period we asked controls “Are your responses to these questions likely to have been similar for the period <case exposure period>”. Half of the controls responded that it was likely to be the same; one fifth responded that it was likely to be different. The remaining (approximately 30%) were either not asked this question or did not know. For controls who responded that it was likely to be different, the reason most often given was that they were on holiday during the case exposure period so would have eaten out more often.

The large proportion of unknown responses was likely due to the fact that some interviewers were unaware of the case exposure period and did not ask this question. Of potential concern is that watermelon consumption is significantly affected by seasonality and changes in availability of watermelon. However, Gisborne public health staff indicated that watermelon was available in the area during the case control study.

Following preliminary investigations, the grower elected to chlorine wash the watermelons. Subsequent visits to the implicated grower found that watermelons and the pack house had been washed. It was reported that a strong smell of chlorine pervaded the area. There were no cases with onset dates after the chlorine wash. A media release also advised locals of the outbreak and recommended washing uncooked farm fresh produce before eating. Watermelon was not specifically mentioned in the media advice.

Although all watermelon samples tested negative for *Salmonella*, watermelon remains the most likely source of the outbreak. The epidemiological and physical evidence suggest the source of this outbreak was the watermelon from the implicated grower. The grower distributed watermelons outside the region but these were washed in chlorine wash before distribution. The only case of STM1 with the “Gisborne” PFGE profile residing outside the Gisborne region was visiting Gisborne during the five days prior to onset of symptoms.

It appears that only the watermelons purchased in the region from the roadside stall were a risk factor, as opposed to all watermelons from this grower. Prior to the outbreak, melons sent outside of this region were chlorinated whereas local melons sold on stalls were not. Chlorine washing of the watermelons prior to distribution is likely to have reduced contamination levels, or perhaps only some watermelons were contaminated, and these were only sold at the roadside stalls. This grower also
supplied other types of melon. Consumption of other melons was not associated with STM1 cases in this outbreak.

**Recommendations**—To prevent future outbreaks such as this, melon growers should be advised to keep fruit free from potential *Salmonella*-contaminated wastes and to chlorine wash watermelon prior to sale. Furthermore, cut watermelon should be kept chilled prior to sale.

**Competing interests:** None known.

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Prevalence of sexually transmitted infections in men who have sex with men presenting to Auckland Sexual Health Service

Sunita Azariah, Nicky Perkins

Abstract

Aim Reported incidence of HIV infection and infectious syphilis in New Zealand has been increasing in recent years in men who have sex with men (MSM) but there is no recent published data about prevalence of other sexually transmitted infections (STIs) in this group. Therefore we decided to collect data on prevalent STIs and possible risk factors in a sample of MSM presenting to Auckland Sexual Health Service (ASHS).

Method All MSM presenting to ASHS during the study period were eligible for enrolment. Data on demographics, sexual behaviour, use of recreational drugs and STI diagnoses was collected and entered into a data collection form after verbal consent was obtained.

Results Eighty-seven men participated in the survey. A third of the men were symptomatic (n=26) and 44% (n=38) were diagnosed with a new STI including 2 with infectious syphilis and 3 with HIV. The Internet (65%) was the most common method used for meeting sex partners (n=55) and 46% of men (n=37) reported use of recreational drugs within the previous 6 months. Sixty percent (n=52) of men reported more than 5 sexual partners within the previous 6 months and 52% were engaging in concurrent sexual partnerships with both casual and regular partners(n=45). Participants were more likely to use condoms for insertive and receptive anal sex with casual than with regular partners (p=0.0004, p=0.005), however a history of consistent condom use did not rule out diagnosis with a new STI or HIV.

Conclusion There was a high prevalence of STIs (44%) in this sample of MSM presenting to a sexual health clinic. Many were presenting with problems rather than attending for routine screening. Possible explanations for the high STI rate include high rates of concurrent sexual partnerships (52%), lower rates of consistent condom use within relationships and use of recreational drugs. Internet use was a common method for meeting sex partners and offers future potential for health promotion interventions for MSM. STI screening should be regularly offered to sexually active MSM regardless of whether they report consistent condom use.

New Zealand has shown similar trends to other developed countries with increasing incidence of HIV and infectious syphilis in men who have sex with men (MSM) since the late 1990’s. Since 2002 the majority of HIV infections in MSM have been locally acquired and in 2008, 70% of HIV infections notified to the AIDS Epidemiology Group were probably acquired in New Zealand.1

Infectious syphilis notifications to ESR have increased annually since 2004.2 Two NZ studies have shown MSM are also a major risk group for acquisition of infectious syphilis and that similar to HIV infection, the majority of infections were acquired
within New Zealand. These data confirm that MSM are at high risk for acquisition of HIV and syphilis, however there is no recent published data on prevalence of other sexually transmitted infections (STIs) in MSM in New Zealand. The aim of this study was to collect data on diagnoses of STI and HIV in a sample of MSM presenting to Auckland Sexual Health Service and to look at possible risk factors.

**Method**

**Study design**—MSM presenting to Auckland Sexual Health Service (ASHS) for routine STI screening or with symptoms of STI were eligible for enrolment. An MSM was considered to be any male presenting to the service that had ever had sex with a man. Individuals identifying as MSM during a standard sexual health consultation were asked for verbal consent to participate in the study. If consent was given the study questionnaire was then completed by the patient or the clinician. Data on demographics, sexual behaviour, use of recreational drugs and how sexual contacts were made was collected. The study questions were very similar but were slightly more detailed than those routinely asked in a sexual health history at ASHS. For comparison purposes, the definitions for regular and casual sex partners were the same as those used in the Gay Auckland Periodic Sex Survey (GAPSS). A regular sex partner was defined as someone the participant had had sex with 4 or more times in the previous 6 months and a casual sex partner was someone they had had sex with fewer than 4 times in the previous 6 months. Reported condom use was also categorised as in the GAPSS survey with participants being asked to estimate whether they used condoms always, almost always, about half, rarely or never for all their sexual encounters. A copy of the questionnaire was filed in the clinical notes to inform patient care. Data on incidence of laboratory or clinically confirmed STIs amongst participants was subsequently entered on the questionnaire form by the researchers after enrolment. Once results of all diagnostic tests were available the data was entered into an excel file for analysis.

**Statistical analysis**—In order to investigate risk factors for diagnosis of STI or HIV, binary logistic regression was used with two separate outcomes: STI or HIV diagnosed at ASHS in the previous 12 months to enrolment and new STI or new HIV infection diagnosed at ASHS at the enrolment visit. The explanatory variables were the demographic variables of age, ethnicity (European, Māori, Pacific or other) and country of birth (NZ or not). The behavioural variables were venues for meeting sex partners, numbers of reported male sex partners, proportion of known partners (100% or not), history of overseas sex or not and recreational drug use. The sexual practice variables were: reported anal sex practices with casual and regular partners and frequency of condom use with casual and regular partners. As there were a large number of explanatory variables compared to participants these three groups were looked at separately and then a combined analysis was run including any variables showing any indication of being related to STI/HIV diagnosis from each group. The explanatory variables of frequency of condom use during insertive or receptive anal sex with casual and regular partners were examined separately as only a subset of the participants used these practices. The relationship of having been tested for HIV within the past twelve months and STI diagnosis was also examined on its own, using a Chi square test as this information was only available on a subset of participants. The difference in condom use with casual and regular partners was examined using a paired Wilcoxon test, separately for insertive and receptive anal sex. The study was approved by the Northern X Regional Ethics Committee. Recruitment began in May 2008 and was completed in April 2009.

**Results**

Numbers in brackets refer to the number of participants that had responses recorded to these questions. Eighty-seven men who were approached agreed to fill in the survey and 2 declined giving a participation rate of 98%. The age range of participants was from 15 to 66 with a mean age of 34. The majority (70%) were born in New Zealand (n= 61) and were mainly of European ethnicity (70%). (Table 1) New Zealand born participants and those of Māori, Pacific and Asian ethnicity were under-represented when compared with data from the 2006 Auckland regional census data.
Forty-two percent of the participants gave “routine STI check” as their primary reason for attending the service. The remainder presented with anogenital symptoms (34%) or as STI contacts (7%) (Table 1).

### Table 1. Summary of demographic and behavioural data

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. (%)</th>
<th>Variable</th>
<th>No. (%)</th>
</tr>
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<tbody>
<tr>
<td>Ethnicity (n=87)</td>
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<td>Type of recreational drug* (n=81)</td>
<td></td>
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<tr>
<td>European</td>
<td>61 (70%)</td>
<td>Amyl Nitrate</td>
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</tr>
<tr>
<td>Māori</td>
<td>6 (7%)</td>
<td>Cannabis</td>
<td>19 (24%)</td>
</tr>
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<td>Pacific</td>
<td>5 (6%)</td>
<td>Viagra</td>
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<td>Other</td>
<td>15 (17%)</td>
<td>Ecstasy</td>
<td>11 (14%)</td>
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<td></td>
<td></td>
<td>Methamphetamine</td>
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<td></td>
<td></td>
<td>BZP</td>
<td>2 (3%)</td>
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<tr>
<td>Reason for attendance (n=86)</td>
<td></td>
<td>Method of meeting* partners (n=81)</td>
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<tr>
<td>STI check</td>
<td>37 (42%)</td>
<td>Internet</td>
<td>55 (65%)</td>
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<tr>
<td>Anorectal symptoms</td>
<td>8 (11%)</td>
<td>Sex on site</td>
<td>33 (40%)</td>
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<td>Urethritis</td>
<td>20 (23%)</td>
<td>Beat</td>
<td>5 (6%)</td>
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<tr>
<td>STI contact</td>
<td>6 (7%)</td>
<td>Bars</td>
<td>38 (46%)</td>
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<td>Other</td>
<td>15 (16%)</td>
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<td>22 (26%)</td>
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<td>Gender of partners (n=87)</td>
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<td>New STI diagnosis</td>
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<td>Male only</td>
<td>79 (91%)</td>
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<td>Male and female</td>
<td>8 (9%)</td>
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<td>No. of male partners in 6 months (n=87)</td>
<td></td>
<td>STI diagnosis (n=38)</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>2 (2%)</td>
<td>Chlamydia</td>
<td>8 (9%)</td>
</tr>
<tr>
<td>1</td>
<td>12 (13%)</td>
<td>Gonorrhoea</td>
<td>7 (8%)</td>
</tr>
<tr>
<td>2</td>
<td>7 (8%)</td>
<td>NSU</td>
<td>10 (11%)</td>
</tr>
<tr>
<td>3</td>
<td>9 (11%)</td>
<td>Syphilis</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>4</td>
<td>5 (6%)</td>
<td>HIV</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>5</td>
<td>10 (11%)</td>
<td>Herpes</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>6 to 10</td>
<td>19 (23%)</td>
<td>Warts</td>
<td>6 (7%)</td>
</tr>
<tr>
<td>11 to 20</td>
<td>12 (14%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21 to 49</td>
<td>8 (10%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50 or more</td>
<td>3 (3%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Multiple responses were possible for this question.

**Sexual behaviour (n=87)**—Most men (n=79, 91%) reported only male sex partners in the previous 6 months however 8 men (10%) reported female partners as well. One man reported more than 3 female partners in the previous 6 months. There was a wide range in the numbers of reported male sexual partners within the previous 6 months. Twenty-one percent reported only 1 or 2 sexual partners, 48% (n=42) reported more than 5 sexual partners (Table 1) and 3 reported more than 50 partners. Sixty-nine percent (n=60) of participants reported having had a regular sexual partner in the previous 6 months and 45 of these men also reported having casual sexual partners in the same time period. Therefore a large proportion of the sample (52%) was engaging in concurrent sexual partnerships.

**Known versus unknown partners (n=81)**—43% of men (n=35) reported they would be able to contact all of their sexual partners within the previous 6 months, however 16% (n=13) of the sample reported they had only had anonymous sex partners. In
those men who indicated that they would be able to contact 100% of their sexual partners, the reported numbers of partners ranged from 1 to more than 10.

**Method of meeting sex partners (n=81)**—The Internet was the most commonly cited method of meeting sex partners (65%, n=55) and 21% reported the Internet was their only means for meeting sex partners (n=18). Bars and sex on site venues (saunas) were the next most commonly cited method of meeting sex partners (Table 1).

**Use of recreational drugs (n=81)**—46% of men (n=37) reported use of recreational drugs within the previous 6 months and the majority of these had used them more than once (89%). Amyl nitrate was the most commonly reported drug (28%), followed by cannabis (24%), ecstasy (14%) and Viagra (10%). (Table 1)

**Oral sex**—84 men responded to this question and although 93% (n= 78) knew that oral sex was a risk for STI acquisition, the vast majority stated they never used condoms for oral sex with regular (88%) or casual partners (86%).

**Anal sex practices**—Quite a few of these questions had missing responses as not all men had had either casual or regular partners during the previous 6 months. Further not all men had engaged in anal sex and of those that did, some only practiced anal insertive and some only anal receptive sex.

Seventy-seven percent (n=67) of men responded that they engaged in insertive anal sex with regular sex partners and 74% (n=64) with casual sex partners. Sixty-six percent (n=44) of those said they always (A) or almost always used condoms (AA) for insertive anal sex with regular sex partners, 21% said rarely and 9% said never. More men (79%) said they always or almost always used condoms for insertive anal sex with casual sex partners than with regular partners (66%). (Table 2)

<table>
<thead>
<tr>
<th>Sexual practice regular partners</th>
<th>No. (%)</th>
<th>Sexual practice casual partners</th>
<th>No. (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insertive Anal</td>
<td>67 (77%)</td>
<td>Insertive Anal</td>
<td>64 (74%)</td>
<td>0.0004</td>
</tr>
<tr>
<td>Condom A or AA</td>
<td>44 (66%)</td>
<td>Condom A or AA</td>
<td>51 (79%)</td>
<td></td>
</tr>
<tr>
<td>Condom about half</td>
<td>3 (4%)</td>
<td>Condom About half</td>
<td>4 (6%)</td>
<td></td>
</tr>
<tr>
<td>Condom rarely</td>
<td>14 (21%)</td>
<td>Condom Rarely</td>
<td>5 (8%)</td>
<td></td>
</tr>
<tr>
<td>Condom never</td>
<td>6 (9%)</td>
<td>Condom Never</td>
<td>4 (6%)</td>
<td></td>
</tr>
<tr>
<td>Receptive Anal</td>
<td>60 (70%)</td>
<td>Receptive Anal</td>
<td>55 (63%)</td>
<td>0.005</td>
</tr>
<tr>
<td>Condom A or AA</td>
<td>41 (69%)</td>
<td>Condom A or AA</td>
<td>47 (85%)</td>
<td></td>
</tr>
<tr>
<td>Condom about half</td>
<td>2 (3%)</td>
<td>Condom About half</td>
<td>1 (2%)</td>
<td></td>
</tr>
<tr>
<td>Condom rarely</td>
<td>7 (12%)</td>
<td>Condom Rarely</td>
<td>2 (4%)</td>
<td></td>
</tr>
<tr>
<td>Condom never</td>
<td>10 (17%)</td>
<td>Condom Never</td>
<td>5 (9%)</td>
<td></td>
</tr>
</tbody>
</table>

Seventy percent (n=60) of men reported they engaged in receptive anal sex with regular sex partners and 63% (n=55) with casual sex partners. Sixty-nine percent of those said they always or almost always used condoms for receptive anal sex with regular partners and 29% said rarely or never. More men (85%) said they always or almost always used condoms for receptive anal sex with casual sex partners compared with regular sex partners (69%).
There was strong evidence of a difference in the reported use of condoms with regular and casual partners for both insertive and receptive anal sex (p=0.0004 and p=0.005 respectively).

**STI diagnoses (n=87)**—63% (n=55) of participants had previously had an HIV test at ASHS, 45% (n=25) within the previous 12 months. Fifty-one percent (n=45) of the sample had had previously been diagnosed with an STI at ASHS; 38% (n=17) of these within the previous 12 months. The most common previously diagnosed STIs were chlamydia (20%), non-specific urethritis (NSU, 16%) and gonorrhoea (13%).

Three men were HIV positive. Forty-four percent (n=38) of men were diagnosed with a new clinically or laboratory confirmed STI at the study enrolment visit, 9 of whom were asymptomatic (24%). The most common new STI diagnoses (Table 1) were NSU (11%), chlamydia (9%) and gonorrhoea (8%).

Two men were diagnosed with infectious syphilis and 3 were diagnosed with HIV. Four of the 7 men who were diagnosed with rectal chlamydia had reported using condoms always or nearly always with casual and regular sex partners. One man with rectal, urethral and pharyngeal gonorrhoea also reported similarly high rates of condom use with casual and regular sex partners. All 3 men diagnosed with HIV had had negative tests at ASHS within the previous 12 months and all had been previously diagnosed with genital ulcerative conditions (2 with syphilis and 1 with genital herpes). One of the men who were newly diagnosed with HIV had reported always using condoms for anal sex with casual and regular sex partners.

The only variable for which there was any evidence of being associated with a current infection from the 3 sets of variables initially investigated was ethnicity. Those of Māori or Pacific ethnicity were less likely to be found STI or HIV negative (OR 0.056, p=0.04, Table 3) at the study visit. The probabilities for all other variables were 0.2 or greater. An analysis was run including ethnicity and the 2 other variables with the strongest evidence – using sex on site and having sex overseas. In particular none of the four measures of reported condom use for anal sex practices could be shown to be related to currently diagnosed infection.

<table>
<thead>
<tr>
<th>Variable</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Māori or Pacific Ethnicity vs European or Other</td>
<td>0.04</td>
</tr>
<tr>
<td>Sex on Site venue vs other venue</td>
<td>0.55</td>
</tr>
<tr>
<td>History of Overseas Sex vs none</td>
<td>0.39</td>
</tr>
<tr>
<td>Insertive regular vs frequency of condom use</td>
<td>0.22</td>
</tr>
<tr>
<td>Insertive casual vs frequency of condom use</td>
<td>0.51</td>
</tr>
<tr>
<td>Receptive regular vs frequency of condom use</td>
<td>0.43</td>
</tr>
<tr>
<td>Receptive casual vs frequency of condom use</td>
<td>0.39</td>
</tr>
</tbody>
</table>

**Discussion**

The prevalence of STI was high (44%) in this sample of MSM presenting to a sexual health clinic which is not surprising as over half the men in the sample were attending either with symptoms or as STI contacts. This indicates that MSM are generally only
attending the clinic if they have specific issues or concerns rather than for routine sexual health screening.

Although this data cannot be generalised to the MSM population at large, such a high prevalence of STI is of real concern given the increasing numbers of HIV notifications in New Zealand in recent years, and the fact that there is an increased risk of transmission and acquisition of HIV associated with concurrent STI.6

We found that a history of consistent condom use for anal sex in the previous 6 months did not exclude new acquisition of STI or HIV infection. It is important to note that other sexual practices such as fingering, oral-anal sex (rimming) and use of sex toys are also implicated in STI transmission.7–9 Oral sex is a particularly significant risk factor for syphilis transmission in MSM.

In one study oral sex was the only sexual exposure reported by 22% of primary and secondary syphilis cases in MSM during the likely period of syphilis acquisition, compared with 7% of similar cases in heterosexual men and women.10 This data and the fact that STI are often asymptomatic implies that STI screening should be offered on a regular basis to all sexually active MSM regardless of a history of consistent condom use for anal sex.

Routine screening is also important when considering the fact that only 43% of this sample of MSM said they would be able to contact all their recent sex partners, making contact tracing difficult or impossible in many cases. It is important that we find ways in which to engage with MSM in the community to increase awareness of the importance of regular STI screening.

We were unfortunately unable to identify any statistically significant association between any investigated risk factors and STI/HIV acquisition in our sample, apart from ethnicity. (However this finding should be interpreted with caution as both Māori and Pacific were under-represented in this sample).

Possible reasons for this include the small sample size with not all participants reporting the variables of interest and the fact that this was a high-risk population anyway. We did however gain some useful insight into why syphilis and HIV notifications in New Zealand might be increasing. Firstly, a significant minority of men were engaging in high risk behaviour for acquisition of HIV by having unprotected receptive anal sex with both casual (15%) and regular sex partners (24%). A previous New Zealand study found that participants (both heterosexual and MSM) who practiced receptive anal sex were more likely to report a history of multiple STIs than those that did not.11

Secondly, concurrent sex partnerships were common in this sample, with 52% of participants reporting both regular and casual sex partners. Concurrency is known to accelerate the growth of an HIV epidemic, and enhances spread of other STI amongst the population.12,13 Thirdly, recreational drug use was very common with 45% (n=37) of participants reporting use of recreational drugs within the previous 6 months; ten percent had used Viagra and 4% had used methamphetamine.

International data indicates there is an increased risk of STI and HIV acquisition in MSM using recreational drugs, particularly with the combination of Viagra and methamphetamine. These drugs are often taken together because of the erectile
dysfunction associated with methamphetamine use, and are associated with increased length of sexual activity (“sexual marathons”) and unprotected anal intercourse. 14

Fourthly, the Internet was a common method cited for participants to meet sex partners with 63% of the sample having met a sexual partner via the Internet in the previous 6 months, and 21% using the Internet as their sole method of meeting sex partners. There is evidence both in New Zealand and overseas that Internet use in MSM is associated with higher rates of reporting unprotected anal sex, therefore increased Internet use may be an additional factor facilitating HIV transmission. 5,15

We compared our data to findings from the Gay Auckland Periodic Sex Survey (GAPSS) which is undertaken every 2 years by the New Zealand Aids Foundation and is the only other source of information on sexual behaviour in New Zealand MSM. 5 Our sample had similar behavioural characteristics to the GAPSS sample but there were more very highly sexually active men in the ASHS sample than in the GAPSS sample (47% compared to 32.4% having 6-50 sex partners in the preceding 6 months), and a very high rate of seeking partnerships on the Internet, with 65% of the ASHS sample meeting sex partners on the Internet compared to 38% of the GAPSS sample. (This difference in Internet use may be due to the fact that our study was more recent and may reflect general trends in use of the Internet).

Fifty-one percent of the ASHS sample had been previously diagnosed with an STI compared to 8% reported in the GAPSS survey5 and 37% in a national telephone survey,16 however the latter 2 studies differed from ours in that they were both community-based surveys so participants were more likely to be lower risk and STI data was self-reported and therefore more subject to bias.

An earlier study of STI diagnoses in sexual health clinic attendees found that syphilis and gonorrhoea were more likely to be diagnosed in MSM than heterosexuals, confirming that MSM attending sexual health clinics are a higher-risk population.17 Fewer participants in the ASHS sample reported use of recreational drugs compared with the GAPPS sample, however, this may be due to under-reporting, because the survey was frequently filled out with the help of the clinician, and participants may have been more reluctant to disclose their drug use.

Results from this study also has implications for the wider New Zealand population because 10% of the sample reported female sexual partners within the previous 6 months, similar to findings from other studies.18,19 “Bridging” from the MSM population to the heterosexual population in the context of increasing HIV incidence does represent a significant risk of HIV transmission to the heterosexual community.

In conclusion, in this sample of MSM from Auckland, STI prevalence was high even in those who reported consistent condom use. Possible explanations for this include high reported rates of concurrent sexual partnerships (52%), lower rates of consistent condom use within relationships and use of recreational drugs.

Internet use was a common method for meeting sex partners and offers potential in designing innovative health promotion interventions to reduce STI transmission and acquisition in MSM. STI screening should be regularly offered to sexually active MSM regardless of whether they report consistent condom use.
Additional data from MSM presenting to different health services would be useful to further contribute to our understanding of STI transmission and this is hampered by the fact that STI surveillance in New Zealand is not population-based and is not currently linked to sexual behaviour.

Competing interests: None known.

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References:


Land-use change and emerging public health risks in New Zealand: assessing *Giardia* risks

Cynthia L Winkworth

Abstract

Agriculture is key to New Zealand’s economy with land-use conversions in response to market forces occurring regularly, like that of recent dairy intensification throughout the country. However, land-use conversion can occasionally result in unexpected and significant consequences for public health that need to be accurately estimated and subsequently managed accordingly. For example, dairy cattle have high *Giardia* prevalence in New Zealand and identical strains from infected humans and cattle located in the same geographical region have recently been reported.

Thus, the high rates of human infections in New Zealand compared to similar socioeconomic countries caused by the waterborne pathogen *Giardia* are particularly concerning given the increasing dairy cattle populations on the landscape. However, the ability of traditional, evidence-based, epidemiological approaches to detect such causal relationships between land-use and *Giardia* infections is limited given the many possible indirect links between the two, in turn highlighting the need to develop appropriate risk assessment techniques. As such, the general requirements for and development of risk assessment frameworks to evaluate the likelihood of public health risks from waterborne pathogens are introduced and explored using *Giardia* in New Zealand as an example.

Specifically, the importance of recent advances in *Giardia*-based knowledge, the incorporation of such data into existing risk assessment frameworks and the influence of remaining research gaps are each discussed for expanding currently available risk assessment tools. Not surprisingly, the availability of appropriate risk assessment tools for agencies responsible for public health and environmental management would ensure the public health risks for *Giardia* resulting from land-use change could be quantified holistically and strategies subsequently developed through active agency communication to minimise such risks.

Throughout history, diseases have emerged and re-emerged in the human population as conditions came about that were favourable to pathogens. Not surprisingly, human-induced land-use changes have been important drivers of significant infectious disease outbreaks and of the more than 1,400 species of organisms known to cause human infections, roughly 60% are zoonotic and transmissible between non-human and human hosts. Thus, land-use changes that alter the type and number of animals across the landscape can result in novel opportunities for disease to occur in local human populations. The significant conversion and intensification of land to dairy farming in New Zealand may be one such example creating opportunities for infectious diseases to emerge and increase in humans.
The recent intensification of existing dairy farms and the conversion to dairy farming from lower density sheep and beef farming in New Zealand is causing concern, with reports of declines in water quality coincident with such land-use change. Not surprisingly, this shift has resulted in considerable physical alterations to the landscape, in addition to a range of significant direct and indirect environmental, social, economic and political consequences. However, an important, yet often overlooked, consequence of changing land use to dairy farming is an increased risk to public health, by the transport of potentially pathogenic microorganisms from animal hosts to humans via surface waters.

Resulting from both point (direct) and non-point (diffuse) sources, the contamination of waterways with microorganisms present in manure that can potentially cause human infections is particularly concerning given the shear volume of manure produced daily. For example, the average dairy cow produces twenty-five kilograms of faecal matter per day and preferentially defecates when crossing streams. Furthermore, it is reasonable to assume that faeces excreted onto farm fields by cattle could be transported via farm-field surface runoff to surrounding waterways.

Given the higher stocking densities typical of dairy farming (ranging upwards from 17.5 stock units per hectare) compared with those on beef and sheep farms in New Zealand (on average 10.5 stock units per hectare), microbial contamination can be increased substantially by dairy conversion. The possibility also exists for a concomitant increase in the risk of pathogen transmission to humans through the contact with, and consumption of, contaminated water. Consequently, potential public health risks need to be evaluated in areas experiencing rapid increases in cattle densities using risk assessment models.

The limitations of epidemiology

Epidemiology is an evidenced-based, scientific approach for studying disease-associated risk factors within populations and is the cornerstone of preventative medicine and the advancement of public health. Identifying direct links between animal sources of pathogens and subsequent human recipients using epidemiological approaches can be relatively straightforward to detect, such as the transmission of genetically identical organisms to humans that have handled sick animals. However, establishing causal relationships between sources of waterborne pathogenic organisms and infections in humans using epidemiology can often prove difficult owing to the frequently complex, and often multiple, transmission pathways linking the two. Nevertheless, there have been reports of indirect transmission in regions containing large livestock populations.

A comprehensive analysis of waterborne infectious disease outbreaks that occurred globally between 1990 and 2005 found the presence of livestock in the catchment, coupled with rainfall, was a substantial contributing factor in 19 of 61 outbreaks evaluated. Thus, while epidemiology can play an important role in determining the implications of land-use change for human disease, its power at unravelling cause and effect relationships that result in changes in disease frequency can be limited. Accordingly, the development of microbial disease risk assessment strategies to tease
apart potential public health risks as a result of significant land-use changes would be particularly useful.13

**Microbial risk assessment strategies**

When waterborne pathogens are presumed to be present in the environment it is necessary to assess the potential transmission risk of those organisms to humans, even in the absence of current disease outbreaks.19 However, while it is important to monitor municipal water supplies and recreational waterways for pathogens, several factors limit their ability to accurately detect such risks. These include poor correspondence between pathogen presence and microbial indicators20 or pathogens and turbidity (also used as an indicator21), time lags between sampling and pathogen identification,22 the occurrence of false negatives and positives20, the patchiness of pathogens in the tested substrate23 and the issue of appropriately-timed sampling schedules. Therefore, appropriate and accurate risk assessment strategies, in addition to traditional monitoring procedures, should be developed to minimise the likelihood of significant disease outbreaks.

Risk assessment strategies model the likelihood environmental sources of waterborne pathogens capable of causing disease in local human populations will do so given a particular set of variables.24 To accurately predict health risks, multiple models that link through a risk assessment framework are required to account for the complex transmission pathway of the organism from its source to human infections.23 For example, such a framework may comprise multiple models that represent five general steps of microbial transmission from farm animals to infected humans, as outlined in Figure 1.

**Figure 1. A generalised risk assessment framework for modelling the transmission of a waterborne pathogen from an environmental source through to subsequently infecting humans**

While several risk assessment model structures exist, with each requiring different types of input parameters,25 quantitative models are the ideal approach owing to the high level of accurate quantitative information employed. However, alternative models like semi-quantitative and qualitative models can also provide effective risk analyses when accurate quantitative information is limited.26 Nevertheless, regardless of the specific type of individual model utilised in the risk assessment framework, it is important the models are fully interchangeable, such that as new information becomes available it can be incorporated into the framework, improving the overall accuracy.27 For example, until recently microbial risk assessments were based on static models developed for chemical risk assessment, despite fundamental differences in the processes underlying microbial and chemical risks,23 including transmission mechanisms, exposure risks, incubation periods and immune status. Fortunately, an
increased awareness of disease risks from agricultural sources, as well as an improved understanding of the mechanisms surrounding microbial risks, has led to the development of dynamic models specifically for microbial risk assessment.

Significant information exists for the development of risk assessment frameworks to model the transmission risks of infectious organisms from their source to infected humans (Table 1). Not surprisingly though, the power of a risk assessment framework to accurately predict reality depends on the quality of the individual model components used to develop the overall framework, including their assumptions. Thus, when a number of key parameters remain unknown or unaccounted for in individual models, the overall accuracy of a risk assessment framework that incorporates those models is compromised. For example, in their model of the fate and transport of Cryptosporidium from dairy cattle to waterways in New York State, Walker & Stedinger (1999) assumed the pathogen moved in an identical manner to water. While the accuracy of the authors predictions was compromised by their disregard for surface entrapment and filtration processes, overall the dynamic model was an improvement on the static chemical-based models previously employed.

Table 1. Currently available quantitative information for developing a microbial risk assessment framework

<table>
<thead>
<tr>
<th>Framework step</th>
<th>Published data</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Source of pathogen (animals)</td>
<td>Microbial release from faeces</td>
</tr>
<tr>
<td>(b) Genetic characterisation</td>
<td>See Table 2</td>
</tr>
<tr>
<td>(c) Movement of pathogen in runoff</td>
<td>Soil erosion, Grassed waterways, Experimental columns, Transport state of pathogen, Transport mechanisms and fate, Overland flow—soil box, Georeferenced estimate of runoff</td>
</tr>
<tr>
<td>(d) Pathogen characteristics in water</td>
<td>Sedimentation of free and attached (oo)cysts</td>
</tr>
<tr>
<td>(e) Disease capabilities of pathogen</td>
<td>Human exposure to pathogens, Modes of transmission, Disease outbreak detection</td>
</tr>
</tbody>
</table>

Giardia in New Zealand

Giardia is a common protozoan parasite capable of causing infections in a wide range of mammalian hosts. Originally thought to show host specificity, substantial evidence suggests Giardia duodenalis (synonyms Giardia intestinalis and Giardia lamblia) is better considered a species complex, as multiple and genetically distinct assemblages have been characterised. Seven assemblages are currently recognised for G. duodenalis: A and B, the only assemblages capable of causing disease in humans but also identified from a wide range of other mammalian hosts including cattle, C and D isolated from dogs, E isolated from livestock (cattle, sheep and pigs), F isolated from felines, and G isolated from rats.

Worldwide G. duodenalis is one of the most frequently identified protozoan parasites causing gastro-intestinal disease (termed giardiasis) in humans. In New Zealand,
Giardiasis is the third most common notifiable human disease caused by a microbial agent and occurs at higher rates than in other socioeconomically similar countries. For example, during the past 5 years rates have ranged in New Zealand between 29 and 42 cases per 100,000 population compared to 15 and 8 per 100,000 for Canada and the USA, respectively. Yet in spite of the high rate of human giardiasis in New Zealand, few studies have investigated the likely causes.


<table>
<thead>
<tr>
<th>Area</th>
<th>Number of <em>Giardia</em> positive hosts in study</th>
<th>Prevalence rate in cattle</th>
<th>Assemblage in humans</th>
<th>Assemblage in calves</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand—North Island</td>
<td>15 calves</td>
<td>41%</td>
<td>A 73%</td>
<td>B 27%</td>
</tr>
<tr>
<td>Italy</td>
<td>30 humans</td>
<td>A 80%</td>
<td>B 20%</td>
<td></td>
</tr>
<tr>
<td>New Zealand—North Island</td>
<td>26 calves, 2 humans</td>
<td>10%</td>
<td>A 50%</td>
<td>B 50%</td>
</tr>
<tr>
<td>Australia</td>
<td>31 calves</td>
<td>89%</td>
<td></td>
<td>E 100%</td>
</tr>
<tr>
<td>Italy</td>
<td>24 calves, 37 humans</td>
<td>Not reported</td>
<td>A 45%</td>
<td>B 41%</td>
</tr>
<tr>
<td>USA—East Coast</td>
<td>237 calves</td>
<td>52%</td>
<td>A 13%</td>
<td>E 87%</td>
</tr>
<tr>
<td>USA—East Coast</td>
<td>204 calves</td>
<td>36%</td>
<td>A 9%</td>
<td>E 91%</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>59 humans</td>
<td></td>
<td>A 52%</td>
<td>B 22%</td>
</tr>
<tr>
<td>Portugal</td>
<td>14 calves</td>
<td>14%</td>
<td>A 14%</td>
<td>B 7%</td>
</tr>
<tr>
<td>New Zealand—South Island</td>
<td>40 calves, 30 humans</td>
<td>31%</td>
<td>A 77%</td>
<td>B 23%</td>
</tr>
</tbody>
</table>

Since the identification of *Giardia* from dairy cattle during the early 1980’s high prevalences have been described in newborn calves (ranging from 10-89% prevalence worldwide), while adults have shown low yet persistent levels. In New Zealand, recent studies focusing on dairy cattle have reported between 31% and 41% *Giardia* prevalence in calves and 5% prevalence in adults. No correlations between faecal morphology and the presence of *Giardia* in dairy cattle have been observed in New Zealand. However, despite an apparent lack of clinical symptoms, cattle excrete wide-ranging concentrations of *Giardia*, indicating active infections.
Until recently livestock were not considered a source of *G. duodenalis* strains capable of causing human disease, as historically they were reported to only harbour livestock-specific strains (Assemblage E).\(^5^0,5^8\) However, several studies have identified Assemblage A and B from cattle, the same *G. duodenalis* assemblages found to cause human disease (Table 2). Of particular concern in New Zealand is that dairy cattle appear to exclusively carry Assemblage A and B strains, with the livestock Assemblage E strain undetected to date.\(^4^6\)

### Linking land use and human giardiasis

While multiple transmission pathways are known for *Giardia*, transmission via water may be of particular significance in areas with large numbers of dairy cattle. This is because dairy cattle are typically maintained at high densities,\(^1^2\) they produce large quantities of faecal waste,\(^1^0,1^1\) exhibit high *Giardia* prevalence,\(^5^9\) and cysts have been detected from aquatic and terrestrial environments worldwide where large cattle populations reside.\(^6^1\) Furthermore, the cysts excreted in their faeces are environmentally resistant, immediately infectious to susceptible hosts\(^9\) and move passively across landscapes into waterways in surface runoff.\(^6^2\) Therefore, it is important to understand and estimate the contamination of waterways by *Giardia* from farm surface runoff, especially as the water may subsequently be used for recreational pursuits or human consumption.

In New Zealand a number of factors support a causative relationship between the use of land for dairy farming and the high levels of giardiasis infections in humans. These include the high prevalence of *Giardia* in dairy calves,\(^4^7,5^9\) the detection of *Giardia* in aquatic environments across the country,\(^6^1\) the intensive use of surface water for recreational purposes and municipal drinking water supplies,\(^6^3\) the high human rates of giardiasis compared to other nations\(^4^2\) and the isolation of identical *Giardia* genotypes from humans and calves located in the same geographic region.\(^4^6\) However, evidence of unambiguous links between farm animal density and giardiasis infections in humans in New Zealand using traditional epidemiological approaches have proved elusive.\(^6^4\) This is not to say causative relationships do not exist, only that the sensitivity of the authors’ methods may have been too coarse to detect any patterns present in the available data sets. Therefore, in light of the potential for *Giardia* transmission between dairy cattle and humans in New Zealand, a microbial risk assessment framework should be developed to help evaluate such risks.

While significant information exists for the development of a risk assessment framework to specifically understand the risk of environmental sources of *Giardia* to public health (Table 3), several important information gaps remain. For example, mechanisms responsible for the release and transport of *Giardia* cysts from bovine faeces remain poorly understood and are currently unknown for flow rates above 10ml per minute.\(^7^1\) Accordingly, recent runoff experiments employing saturated soil overland flow rates of 2 litres per minute were restricted to the use of *Giardia* present in liquid spikes, rather than cattle faeces.\(^6^2\) Although it remains to be determined, it is possible faecal particles may occupy sites where biocolloid straining\(^7^4\) would otherwise remove *Giardia*, with the pathogen consequently remaining in the runoff.\(^7^8\) Therefore, research to elucidate the mechanisms of release of *Giardia* from faecal matter, as well as the effect of faeces on *Giardia* movement through the soil, would further improve risk assessment simulations.
Table 3 Parameters currently known for assessing the risk of environmental sources of *Giardia* to public health

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Relevant studies</th>
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<tbody>
<tr>
<td>Cattle infection prevalence</td>
<td>Dairy cattle: calves(^{77,79}); adults(^{85})</td>
</tr>
<tr>
<td></td>
<td>Beef cattle(^{6})</td>
</tr>
<tr>
<td>Genotype present in cattle</td>
<td>Dairy cattle(^{5,51})</td>
</tr>
<tr>
<td>Rate of pathogen excretion</td>
<td>Average number of cysts excreted(^{60})</td>
</tr>
<tr>
<td></td>
<td>Volume of faeces: calves(^{67}); adults(^{50})</td>
</tr>
<tr>
<td>Pathogen viability</td>
<td>Water(^{68}); faeces(^{69}); soil(^{69})</td>
</tr>
<tr>
<td>Mechanism and release rate of <em>Giardia</em> from faeces</td>
<td>Limited to 10ml runoff per minute(^{0,71})</td>
</tr>
<tr>
<td>Transported state of <em>Giardia</em> (single cysts, clumps, etc.)</td>
<td>Attachment mechanisms(^{72,73})</td>
</tr>
<tr>
<td>Movement through experimental soil columns</td>
<td>(^{74})</td>
</tr>
<tr>
<td>Movement across landscape (single cysts)</td>
<td>Bare soil(^{62}); newly vegetated (^{62}); after one growing season(^{7})</td>
</tr>
<tr>
<td>Heterogeneity of pathogen</td>
<td>In water(^{61})</td>
</tr>
<tr>
<td>Sedimentation/settling rate</td>
<td>Sedimentation of free and attached cysts(^{98,76})</td>
</tr>
<tr>
<td>Contact time/exposure risk</td>
<td>For humans(^{58,77})</td>
</tr>
<tr>
<td>Dose ingested</td>
<td>For humans(^{91})</td>
</tr>
</tbody>
</table>

The effect of vegetation development along waterways at reducing *Giardia* in runoff reaching the waters edge has also been determined recently. However, despite a substantial reduction in *Giardia* reaching the waterway after one year of vegetation development (98% of that applied), approximately 3500 organisms per litre still reached the water’s edge.\(^{75}\) As the ingestion of as few as one to ten cysts has been reported to cause giardiasis in humans,\(^{41}\) the high levels of *Giardia* remaining are concerning. Although filtering capacities may improve as the vegetation develops further,\(^{79}\) with potentially greater rates of *Giardia* infiltration observed as more deeply-rooted species become established,\(^{75}\) this remains to be determined. Additionally, while further vegetation growth may result in lower *Giardia* numbers in runoff, the effect (if any) for the overall disease risk is unclear.

**Conclusion**

Significant challenges surround the conversion of land to dairy farming. This paper discussed an important consequence that is often overlooked; an increased risk to public health. As water resources become increasingly scarce and variable in quality due to escalating demand, it seems likely that the indirect impact of land-use conversion on human health may also escalate. Therefore, it is crucial good communication exists between agencies responsible for public health and those responsible for environmental management, such that management responses to land-use consequences are holistic. Not surprisingly, improving the techniques available for accurately evaluating potential risks is clearly beneficial to both public health and environmental managers for identifying, developing and implementing strategies to reduce the risks.
This paper has highlighted the public health challenges New Zealand faces in relation to managing land use conversions to dairy farming. In particular, it focussed on the waterborne pathogen *Giardia*, which causes significant disease in the New Zealand population compared with similar socioeconomic countries. By incorporating the recent advances in *Giardia* knowledge outlined in this paper into a risk assessment framework, an appropriate evaluation of the public health risk of *Giardia* as a result of land-use change to dairying in New Zealand can begin to take shape. For example, the framework previously developed for *Cryptosporidium* by Walker & Stedinger (1999) could be appropriately modified to assess *Giardia* risk by substituting *Giardia*-specific information where possible.

While use of the Walker & Stedinger model would significantly advance *Giardia* risk management assessments in New Zealand, important information gaps remain. Thus additional research is required to ensure further clarification and improvement to the overall accuracy of public health risk assessments of *Giardia* in New Zealand as a result of land-use change. While not inclusive, such gaps include a comprehensive understanding of the mechanisms responsible for the release of *Giardia* cysts from bovine faecal matter at different rainfall and surface runoff rates and in turn, the affect of dissolved faeces on *Giardia* movement through the soil, the *Giardia* cyst retention capabilities of riparian buffers older than one year and different surface runoff rates.

Clearly some uncertainty surrounding individual model components remains when assessing regional and site-specific *Giardia* risks given the practical limitations of testing all possible variations that may exist between different environments. However, while the available data may not be ideal for the particular situation being assessed, it should not preclude its inclusion in a risk assessment. Rather, the level of uncertainty inherent in the available data should be accounted for in the assessment simulations and discussed accordingly, ensuring risk evaluations are still performed and, consequently, prove informative for public health and environmental managers.

Competing interests: None.

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Anti-NMDA-receptor autoimmune encephalitis without neoplasm: a rare condition?

Mark Schmiedeskamp, Pietro Cariga, Annemarei Ranta

Anti-N-methyl-D-aspartic (NMDA)-receptor encephalitis is an acute and potentially lethal encephalitis. It was first described as a distinct disease by Dalmau et al in 2006, with subsequent case reports from around the world. Patients typically present with prominent psychiatric symptoms followed by progressive neurological dysfunctions and more than half of reported cases have occurred as a paraneoplastic phenomenon in association with tumours, especially ovarian teratoma.

Here we present two patients seen in a secondary care hospital that presented in quick succession and were not associated with a tumour. To the best of our knowledge these are the first non-paraneoplastic anti-NMDA-receptor encephalitides reported in New Zealand.

Case report 1

A previously healthy 17-year-old girl presented with an 8-day history of agitation, anxiety, poor oral intake, sleep deprivation, and decreased communication. Symptoms were attributed to a traumatic break up with her boyfriend. Apart from an elevated white blood count initial diagnostics were normal and she was admitted to psychiatry.

Over week 1 of hospitalisation symptoms continued to fluctuate and an electroencephalogram (EEG) on hospital day 5 suggested organic delirium. Brain magnetic resonance imaging (MRI) was normal, but cerebrospinal fluid (CSF) analysis supported CNS inflammation (see Table 1). IV aciclovir, antibiotics and a three-day course of methylprednisolone (1 gm/day) were started empirically without major benefit. Further extensive laboratory testing was unrevealing (see table), and a non-organic cause remained a consideration.

During week 2 she developed increasingly autonomic symptoms of intermittent tachycardia, hypertension, pupillary dilatation, and low grade fevers progressing to generalised tonic-clonic seizures, decreased level of consciousness and profound generalised dystonia requiring intubation on day 10.

At this point a diagnosis of autoimmune/paraneoplastic encephalitis was suspected, but a systemic tumour screen was negative. Despite the lack of identified tumour the patient was empirically restarted on IV steroids (dexamethasone 10mg/day) with the addition of IV immunoglobulins (0.4g/kg/day for 5 days) followed by plasmapheresis (five exchanges). Symptomatically her dysautonomia responded to clonidine, but her profound muscle rigidity was refractory to trials with benztprine, baclofen and benzodiazepines.
<table>
<thead>
<tr>
<th>TEST</th>
<th>CASE 1</th>
<th>CASE 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full blood count</td>
<td>WBC $11.3 \times 10^9/L$ (70% neutrophils)*</td>
<td>Normal</td>
</tr>
<tr>
<td>Renal function test, Ammonia</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Liver function test</td>
<td>Transient mild derangement</td>
<td>Transient mild</td>
</tr>
<tr>
<td>CRP</td>
<td>23 mg/L**</td>
<td>17 mg/L**</td>
</tr>
<tr>
<td>Copper/ceruloplasmin</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Beta-HCG, TSH, catecholamines</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>ANA</td>
<td>Negative</td>
<td>&gt;1:100, diffuse***</td>
</tr>
<tr>
<td>ENA</td>
<td>Positive SSA and RO52***</td>
<td>Negative</td>
</tr>
<tr>
<td>ds-DNA, ANCA, thyroglobulin, cardiolipin, microsome antibodies</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Porphyrins</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Anti-NMDA receptor antibodies</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Infective screen (bartonella, toxoplasma, herpes viruses, measles, mumps, tuberculosis, HIV)</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Tumour markers (CEA, Ca-125, 5HIAA)</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>White blood cells</td>
<td>$27 \times 10^9/L$ (100% monocytes)</td>
<td>$1 \times 10^9/L$</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>$19 \times 10^9/L$</td>
<td>$400 \times 10^9/L$</td>
</tr>
<tr>
<td>Protein, Glucose, Cytology</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Electrophoresis</td>
<td>Normal</td>
<td>Oligoclonal bands</td>
</tr>
<tr>
<td>Anti-NMDA receptor antibodies</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Cryptococcal antigen</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Viral PCR (herpes, CMV, EBV, enter-)</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Brain</td>
<td>Normal (CT, MRI, MRA, MRV)</td>
<td>Normal (CT, MRI)</td>
</tr>
<tr>
<td>EEG</td>
<td>Diffuse delta rhythm</td>
<td>Diffuse delta rhythm</td>
</tr>
<tr>
<td>Pelvis ultrasound</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Full body</td>
<td>Normal (CT)</td>
<td>Normal (PET/CT)</td>
</tr>
</tbody>
</table>

* Fluctuated between 9.4 and 33.2 x $10^9/L$ during the course of admission; ** Fluctuated and normalised during course of admission; *** Not clinically significant as isolated finding and low titre

During week 3 serum and CSF were sent for NMDA-receptor antibody titres and she demonstrated first signs of recovery early during week 4 with continued gradual improvement over the next weeks. She was discharged home on a gradual oral steroid taper after 7 weeks of hospitalisation.

Ten days after discharge positive results of NMDA receptor antibody titres became available confirming the diagnosis of anti-NMDA receptor encephalitis. Three weeks after discharge she had nearly returned to baseline and was off all medications.
At six-month follow-up she had resumed her normal life without any residual symptoms except for persisting amnesia of her illness. Follow-up pelvic ultrasound remained free from evidence of neoplasm.

**Case report 2**

A 23-year-old New Zealand woman presented to a British hospital with acute psychiatric symptoms. She had no history of medical, psychiatric or personality disorders. Five weeks before admission she became unduly anxious about a potential unwanted pregnancy (repeated negative tests), apathetic and ate poorly losing about 10Kg.

Admitted to a psychiatry unit she displayed mutism, dystonic postures, psychomotor retardation and agitation treated with haloperidol. Over week 1 of admission she developed fever, tachycardia, tremor and drowsiness, for which she was transferred to a medical unit. During week 2 she had two convulsive seizures requiring temporary intensive care, and received aciclovir and phenytoin.

At this stage brain imaging and serum laboratory investigations were unremarkable, including a non-specific low ANA titre (see table). Extensive CSF tests (see table) were also normal apart from oligoclonal bands (suggesting CSF inflammation, but not deemed specific for multiple sclerosis as not consistent with clinical picture and MRI findings). An EEG showed generalised rhythmic delta activity without epileptiform features.

During week 3 episodic dysautonomia (midriasis, fever, hyperhidrosis) was noted together with posturing, catatonic episodes, repetitive buccal movements and hyperreflexia. A working diagnosis of autoimmune encephalopathy was made and serum sample for anti-NMDA receptor antibodies sent. Pelvic MRI and full body PET/CT were unremarkable. She received plasma exchange over 5 days and IV methylprednisolone 0.5g daily for 5 more days followed by oral prednisone. Her psychiatric symptoms were managed with olanzepine and benzodiazepines.

From week 5 alertness, behaviour and appetite gradually improved; impaired cognition, mild tremor, stuttering, and hyperreflexia persisted. On day 40 NMDA receptor antibodies resulted positive. She then was transferred to Palmerston North Hospital and received intravenous immunoglobulins 0.4g/Kg/day for 5 days.

Upon arrival she was noted to exhibit disinhibited behaviour, anxiety, insomnia and amnesia but these gradually resolved over the following weeks. She was discharged on day 94 with residual mild impairment of strategic function and memory. At a 4-month outpatient follow-up all medications had been stopped and the only residual deficits were mild ongoing anterograde amnesia and subtotal amnesia to her medical illness. A 6-month pelvic ultrasound follow-up did not show any evidence of neoplasm.

**Discussion**

Anti-NMDA receptor encephalitis has only recently been described as a discrete entity that appears to be mediated by antibodies targeted mainly at the extracellular N-terminal domain of the NR1 subunit of NMDA receptors. These antibodies reversibly decrease the number of cell-surface NMDA receptors.1,2
The typically, but not exclusively, young female patients initially present with a characteristic neuropsychiatric syndrome with change of personality and behaviour, paranoia, and memory disturbances. This is accompanied or rapidly followed by multiple neurological deficits, predominantly dystonia, dyskinesia, seizures, autonomic instability, and decreased level of consciousness.\textsuperscript{1,2}

The diagnosis depends on recognising the characteristic clinical picture usually associated with CSF pleocytosis or other raised CSF inflammatory markers and abnormal EEG findings. MRI studies are often unrevealing although some patients will have increased FLAIR or T2 signal in the medial temporal lobes.\textsuperscript{2}

The majority of patients have evidence of a systemic tumour, most commonly ovarian teratoma. The definitive diagnosis is based on NMDA-receptor antibodies identified in serum or CSF. Treatment includes corticosteroids, plasma exchange, intravenous immunoglobulins, rituximab, cyclophosphamide, and azathioprine as well as tumour resection if detected.\textsuperscript{8}

Whilst the incidence of anti-NMDA-receptor encephalitis has not been established, publications on this condition are limited to case reports and case series and amongst these cases the majority were associated with systemic neoplasm suggesting that the non-paraneoplastic variety is a rather rare condition. However, the presentation of these two cases within only weeks of one another raises the question whether this could be a more common disorder than previously thought and may currently be underdiagnosed.

The two cases presented here were diagnosed and treated more rapidly than many other reported cases in the literature and enjoyed a very favourable outcome. The lack of identified tumour, even months after initial presentations in our patients, highlights the importance to initiate aggressive immune modulatory therapy even without evidence of a tumour and strongly argues against empiric ovarian resections which has been suggested by some. Early recognition and treatment appears to be critical to achieve a favourable outcome and cannot await positive NMDA receptor antibody results, which can take up to several weeks to be processed as is demonstrated in these two cases.

In summary, particularly young women initially presenting with prominent psychiatric symptoms followed by seizures, movement disorder, and dysautonomia should prompt this diagnostic consideration and if an alternative cause is not readily found treated aggressively even without evidence of systemic tumour or the reassurance of confirmatory antibody tests.

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References:


Atypical gastric ulcer with characteristic eschar appearance

Mohit Girotra, Sudhir K Dutta, H Jeffrey Schwartz

Abstract:
Cocaine is a known potent vasoconstrictor known to cause various complications ranging from nasal septum perforations to myocardial ischemia. Many gastrointestinal effects of cocaine are reported, including bowel ischemia and gangrene. The knowledge on endoscopic appearance of cocaine-induced gastric ulcers is limited, mainly due to presentation of patients with frank perforations. We report a case of 48 year old male, a non-smoker but chronic substance abuser, who presented with abdominal pain, mainly epigastric with radiation to the back. Abdominal CT scan was normal, and endoscopy showing a single chronic non-bleeding ulcer at the incisura. The ulcer due to large sized, round in shape with irregular borders and thick eschar appearance is characteristic of cocaine-induced ulcer. It is important for physicians to remain cognisant of gastrointestinal complications of cocaine, recognise these ulcers endoscopically and prevent perforations in these subsets of patients.

Case report
A 48-year-old African-American male, with no significant medical history but long-standing cocaine abuse, presented with abdominal pain. Pain was of 1-week duration, constant and sharp, localised to epigastrium, moderate in intensity and radiated to the back. The patient was also bothered by sour taste in his mouth and some nausea. He also had single episode of coffee ground emesis and dark coloured stools. He denied any alcohol or smoking but was increasingly using cocaine in the last 9 months. There was no previous history suggestive of peptic ulcer disease or non-steroidal anti-inflammatory medication intake.

Initial labwork including complete blood count, renal and liver function tests were unremarkable, except positive stool haemoccult. Electrocardiogram and troponins ruled out myocardial event and chest X-ray did not suggest any pulmonary process. Abdominal CT was normal. Upper endoscopy revealed a single chronic non-bleeding ulcer at the incisura, with an adherent clot which could not be washed away. The ulcer was large (2.5-cm diameter), round with irregular borders and thick black eschar rim (Figure 1). Biopsy from the ulcer returned as chronic active gastritis but negative for Helicobacter pylori. The patient, since not perforated or actively bleeding, was not considered a surgical candidate and was managed medically with proton pump inhibitors. His repeat endoscopy after 4 weeks showed improvement in ulcer.
Discussion

The image shows a large 2.5-cm ulcer, round and irregular borders, with thick black rim of eschar like tissue. This atypical appearance can be considered characteristic of cocaine-induced ulcer, especially in the background of absence of other ulcer forming risk factors.

Cocaine is a known potent vasoconstrictor and many gastrointestinal effects of cocaine are reported, including bowel ischemia and gangrene. There is impressive literature on cocaine-induced duodenal ulcers, described as large pre-pyloric ulcers, which present usually with perforation and undergo patch closure. The knowledge
on endoscopic appearance of cocaine-induced gastric ulcers is limited, mainly due to
presentation of patients with frank perforations. This is however, a unique description
of cocaine-induced ulcer in gastric region based on authors’ multiple experiences with
similar patients.

The black eschar is formed by tissue necrosis caused by vasoconstrictive effects of
cocaine. Hence, it is most advantageous for clinicians to remain cognisant of the
gastrointestinal complications of cocaine and identify them at ulcer stage with their
characteristic eschar appearance, to prevent further and more dreaded complications
like perforation and massive haemorrhage. Anti-ulcer and anti *Helicobacter pylori*
therapies do work, but abstinence from cocaine is the mainstay of therapy.

**Competing interests:** None known.

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632.
Kidney solitaire: pearls to share
Sanjay Bandyopadhyay, Manish Saha, Pranab Kumar Maity

A 25-year-old lady, with a history of recurrent second trimester pregnancy loss, was referred to us for intermittent pedal oedema, nausea and vomiting of 6 months’ duration. She also noticed slowly-progressive swelling of abdomen over the last 10 years. On examination, patient was grossly undernourished; abdomen was hugely distended and cystic in feel.

Figure 1. Figure showing the abdominal swelling

Routine investigations revealed anaemia (haemoglobin 8.4 gm%), raised serum urea (61 mg%) and creatinine (2.2 mg%), and microscopic haematuria.

Ultrasonography (USG) of abdomen showed a large septate cystic mass occupying the whole of the abdomen. Computed tomography (CT) scan of abdomen is shown.
What is the diagnosis?
Answer

CT scan showed absence of right kidney along with gross hydronephrosis of left kidney with thinned out renal cortex resulting from intrinsic obstruction at pelviureteric junction (PUJ). On laparotomy, obstruction could be easily negotiated through double J stent.

Postoperatively, patient had rapid profuse diuresis with near complete resolution of the swelling. Serial USG showed complete resolution of hydronephrosis and renogram revealed stable renal excretory function.

Discussion

In patients with solitary kidney, 40% have associated urologic anomalies in the collecting system and nearly 13% have stenosis at the PUJ. Congenital solitary kidney with hydronephrosis is a rare anomaly with mean age of diagnosis at 10 years. Majority are asymptomatic and detected on ultrasound examination. However, 15% have an irreversible lesion of variable severity.

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References:
Delayed puberty—an occult systemic cause

Rashmi S Manjunatha, George Varughese, Cordelia Phelan, Richard N Clayton

Case—A 17-year-old male presented with symptoms of absent secondary sexual characteristics, decreased appetite and infrequent non-specific abdominal pain. He had no headache, altered sense of smell, colour blindness or gynaecomastia. His other siblings had normal growth. He had a normal male body habitus, height below the 5th percentile, pre-pubertal body hair distribution with testes in the scrotum (12 ml bilaterally). Laboratory results revealed microcytic iron deficiency anaemia, hypogonadotropic hypogonadism with 46XY on chromosome analysis.

Barium meal follow-through study (Figure 1) demonstrated multiple strictures in the small bowel, which was not resectable raising the suspicion of Crohn’s disease.

Figure 1. Barium meal follow through showing multiple strictures in the small bowel
Histology (Figure 2) confirmed the diagnosis and azathioprine treatment was initiated alongside ferrous sulphate. Twelve months after starting treatment, his testosterone level came up and was within the reference range.

Interestingly, 2 years later his height was on the 23rd percentile on the growth chart, voice was deeper; he was getting erections and was sexually active with testicular volumes of 25ml bilaterally.

**Discussion**—Delayed puberty can be a complication of underlying inflammatory bowel disease in young patients. Although systemic diseases are well recognised to cause hypogonadotropic hypogonadism, this is often less commonly perceived. Delay in the diagnosis and management can delay the onset of puberty indefinitely with potentially disastrous consequences on the pubertal growth spurt with a reduction in final adult height. Testosterone levels often return to normal after recovery from underlying disease. Clinicians should have a high index of suspicion to diagnose this well described, but less commonly perceived cause of delayed puberty.1,2
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The Acid Sulphur Waters of Rotorua

First part of article by Dr Wohlmann published in 1911 February;9(37):36–45—and read before Annual Meeting of N.Z. Branch, B.M.A., March, 1911.

Mr. President, ladies and gentlemen,

In choosing the Acid Sulphur Waters of Rotorua as the subject of this paper, I have done so partly because this is a matter which I have had exceptional facilities for investigating, and to which for several years I have devoted special attention, and partly because I wish to bring more prominently under the notice of the profession a therapeutic agent of extraordinary value.

Certainly this water is now pretty widely known, but I am convinced that in the future its usefulness will be yet more appreciated, and I propose here not merely to give a description of the remedy, with which many of you are, of course, already familiar, but to endeavour to explain the manner in which it acts. If in doing this latter I make some statements which appear at first sight somewhat startling, I must plead in extenuation that in no case is any explanation advanced on theoretical grounds alone, but that every theory is based on data ascertained by numerous experiments extending over a period of years.

In all research work considerable allowance has to be made for the personal equation, the bias of the investigator, as well as for errors of technique. The enquiries now under consideration involved the making of a large number of blood counts, and those who have made the largest number of counts will be the first to acknowledge the abundant opportunities that exist for fallacies to creep in, and how easy it is by slight variations of technique to vary the blood count. For this reason I have omitted from the following tables the results of earlier work, and have confined myself to those obtained within the last two years. For this reason I was more than glad to obtain the assistance of two independent investigators, and I would like here to acknowledge my great indebtedness to Dr. Bertram, a former house-surgeon, and to Dr. Hay, the present house-surgeon at the Sanatorium, for the great and ungrudging help they have rendered me in making a large number of tedious blood counts.

For the sake of those not familiar with the thermal district, I will begin with a brief description of the mineral waters, their surroundings, and the manner in which they are generally used. Rotorua is a lake, roughly circular in shape, six or seven miles in diameter, surrounded by hills of volcanic origin rising from 600ft to 1,500ft above it. Between the hills and the lake is a more or less flat plain of pumice and sand. From terrace indications on the hillside the lake must evidently have extended some hundreds of feet up their slopes, so that the present pumice plain must have been the bed of a very much larger lake which swept the base of Ngongotaha are extended over Rotoiti and Rotoehu to Rotoma.

As a subsoil water, over at any rate part of this dried pumice lake-bed, the hot acid water exists, continuous at its margins with the lake water, and rising and falling with its varying levels. In places this water comes quietly to the surface as a spring—such,
for instance, as the “Priest” spring. At certain spot more especially at Whakarewarewa and Ohinemutu alkaline silicious waters issue with considerable violence, and sometimes with geyser action. These waters would appear to originate from considerable depths and to owe their great heat, in all cases at or about the boiling point, to passage over hot lava.

Such waters contain abundant $\text{H}_2\text{S}$, and from investigations which I have made but with which will not trouble you here, I am driven to the conclusion that they are the parent waters, and that the acid waters are, at any rate in part, formed from them by oxidation, the sulphides being converted into sulphates. The acid formation probably occur partly by direct oxidation of sulphides in the pumice bed and partly by the constant upward passage through the subsoil of gaseous $\text{S} \text{O}_2$.

The Springs.—There are three main acid spring, used for therapeutic purposes—the Postmaster, the Old Priest Spring, and the New Priest Spring—and each has its peculiar advantages and disadvantages, each is invaluable.
Short stature and coronary heart disease—are they related?

The first report on the inverse association between coronary heart disease (CHD) and height was published in 1951. Since then the subject has been controversial. This meta-analysis seeks to elucidate. It includes 52 observational studies involving more than 3 million individuals. Short is defined as below 160.5cm and tall as over 173.9cm. Their results—among the shortest height category the relative risks were 1.35 for all-cause mortality, 1.55 for all cardiovascular disease (CVD) mortality, 1.49 for CHD and 1.52 for myocardial infarction when compared with those within the highest height category.

So, overall being short confers an approximately 50% higher risk of CHD morbidity and mortality. An editorial muses on these findings – could it be because of the association of short stature and lower social-economic status? And what about the fact that women have less CHD than men and are generally shorter than men? And more importantly, what use is the knowledge—what special measures, if any, should the vertically challenged adopt?


Glucosamine in patients with chronic low back pain and degenerative lumbar osteoarthritis

Osteoarthritis is a very common age-related condition. Glucosamine is widely used by such patients either by self medication or on medical recommendation. Does it work? This double blind randomised trial was performed in Norway on 250 patients with chronic lumbar back pain for 6 months or longer. Half took 1500mg of oral glucosamine daily and the other half placebo. At 6 months and 1yr follow-up there was no difference in pain related disability in either group. Interestingly, mild adverse events were reported in about 30% in both arms of the trial.

JAMA 2010;304(1):45-52.

High-dose allopurinol for patients with chronic stable angina

Aspirin may not be the only old drug that can perform new tricks. Apparently, experimental evidence suggests that xanthine oxidase inhibitors can reduce myocardial oxygen consumption for a particular stroke volume. The authors of this paper suggest that it might well be useful in the treatment of patients with ischaemic heart disease. They have randomised 65 such patients in a double blind trial to receive either 600mg of allopurinol/day or placebo. After 6 weeks they report increased exercise time and less chest pain in those taking allopurinol. The ECG features also favoured allopurinol.

Consequently they conclude that allopurinol seems to be a useful, inexpensive, well tolerated, and safe anti-ischaemic drug for patients with angina. I note they excluded
patients with a left ventricular ejection fraction of <45% and patients whose
glomerular filtration rate was <45 mL/minute. Those with severe hepatic disease or
taking warfarin, azathioprine or 6-mercaptopurine were also excluded.


How useful are the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) in diagnosing septic arthritis?

This study involves a retrospective chart review of 163 patients who presented with a sore joint and had ESR, CRP and joint fluid aspiration analysis. The diagnosis proved to be inflammation in 72, sepsis in 44 and 47 were proven to be normal. The mean CRP for septic joints was 13, 8.5 for inflammatory joints and 6 for normal. The mean ESR for septic joints was 57, 48 for inflammatory joints and 43 for normal joints. So they conclude that the CRP is helpful and the ESR is not. I note that the white cell count was significantly higher in the septic joint cohort when compared with the normal and inflammatory groups.


Glycated haemoglobin A\textsubscript{1c} and the diagnosis of diabetes

The fasting plasma glucose value and the oral glucose tolerance test are most commonly used for the screening and diagnosis of diabetes. However, the need to fast for 8hrs in both and the multiple blood tests in the latter limit their value. On the other hand HbA\textsubscript{1c} values reflect the mean blood concentration of glucose over the preceding 2-3 months and involve a single blood test and no fasting. Consequently in 2009 an international expert committee published a report recommending the use of an HbA\textsubscript{1c} value of 6.5% or more as a diagnostic criterion for diabetes. It was appreciated that ethnic differences might occur and this paper and an editorial illustrate this point – viz in China the cut-off value appears to be 6.3%. This value proved to be more sensitive in predicting diabetes than a threshold of 6.5% or a fasting blood glucose of 7.0mmol/L or more in the Chinese population.

Defendants of the Cartwright Inquiry are unable to provide a description of ‘adequate care’ for cervical carcinoma in situ

Judging the adequacy of any treatment in health care should result from comparing treatments actually given or withheld with a generally accepted treatment standard—‘conventional treatment’. Although ‘conventional treatments’ should be based on empirical evidence that they are more likely to help than to harm patients, patients have quite often suffered because they have not been.

For more than 20 years Professor Charlotte Paul has claimed that ‘conventional treatment’ for cervical carcinoma in situ was withheld from women treated at the National Women’s Hospital in Auckland—most recently in an article co-authored with Professor Linda Holloway—and the claim has recently been reiterated by Professor Jo Manning.

I have challenged Professors Paul and Manning to justify their use of the term ‘conventional treatment’ by documenting the implied international agreement on an accepted treatment standard, and referring to the reliable empirical evidence on which it was based. Neither of them has taken advantage of several opportunities to respond to this challenge, so I have now assumed that they are unable to do so.

In her recent letter published in the NZMJ Professor Manning suggests that the definition of ‘conventional treatment’ is contained in a section of the Cartwright Report entitled ‘Adequate management of CIS’. It is not to be found there, or anywhere else in the Report.

Other defendants of the Cartwright Inquiry have tried to deflect the challenge to define ‘conventional treatment’, for example, by suggesting (wrongly) that I had blamed Professor Paul for “not providing the Inquiry (my emphasis) with a definition of conventional treatment”, and with interpretations of the views and alleged proposals of Professor Archie Cochrane on cervical screening. None of these attempted diversions can alter the fact that ‘conventional treatment’ for cervical carcinoma in situ—the standard against which the adequacy of its treatment should be judged—remains undefined by these defenders of the Cartwright Inquiry.

Some people reading the NZMJ in the hot-house atmosphere of New Zealand name-calling may wonder why an ‘outsider’ like me should spend so much time investigating and commenting on this affair.

My interest is longstanding. Twenty years ago, in a letter published in the BMJ, I noted that two gynaecologists at the National Women’s Hospital had been charged by the New Zealand Medical Council with “disgraceful misconduct” because treatment had been withheld from “women with carcinoma of the cervix after convincing evidence had emerged that such treatment could be expected to do more good than harm”.

I went on to observe that it was a sad irony that “it was at the National Women’s Hospital that evidence was first produced showing that giving corticosteroids to women who were expected to give birth before term reduced the chances that their babies would die… yet many obstetricians continued to withhold this life saving form of care.” I asked what fair-minded people were supposed to make of these apparent double standards.

In retrospect, I was wrong to take on trust the assertion that treatment that could be expected to do more good than harm had been withheld from women with “carcinoma of the cervix (sic)”. First, I had understood, incorrectly, that the focus of the Cartwright Inquiry had been the treatment of cervical cancer, when in fact the focus was the management of cervical carcinoma in situ. Second, I had assumed, also incorrectly, that the findings of the Cartwright Inquiry were trustworthy.

The factors that have recently led me to judge the Inquiry to be untrustworthy are (i) the findings of Professor Bryder’s research; (ii) correspondence with Professor Charlotte Paul; (iii) my search in vain for any definition of adequate treatment of cervical carcinoma in situ in the Cartwright Report itself; and (iv) reading Göran Larsson’s 1983 report demonstrating dramatically wide international variations in the ways cervical carcinoma in situ was being treated during the era concerned.

Some New Zealanders may assume that the reliability of the conclusions of the Cartwright Inquiry are of only parochial concern. This would be a mistake. The international prominence given to assertions that effective treatment had been withheld from women with cancer at the National Women’s Hospital in Auckland has led the so-called ‘unfortunate experiment’ there to be referred to in the same breath as the scandalous long term study of untreated syphilis in poor black sharecroppers in Tuskegee, Alabama. It is clear that this is a totally unjustified slur on the treatment of patients in Auckland.

People with substantial vested interests in believing that the findings of the Cartwright Inquiry are unchallengeable have not addressed the fundamental and irresponsible deficiency in their positions. The implication of their inability to define the components of the ‘conventional treatment’ that they allege was withheld from women treated at the National Women’s Hospital is that the Inquiry’s judgements about the adequacy of treatment remain unfounded in reliable evidence.

Challenges to define the evidential basis for judging treatment of cervical carcinoma in situ at the National Women’s Hospital to have been inadequate will not be deflected by bluster about ‘the judicial process’, references to ‘expert advice from world leaders’, attempts to divert attention from the challenge by irrelevant displacement activity, and ad personam attacks on those people impertinent enough to have raised important questions about the validity of the conclusions of the Cartwright Report.

The Inquiry should have based its judgements about the adequacy of treatment for cervical carcinoma in situ using comparisons of treatments actually given or withheld at the National Women’s Hospital with explicit, generally accepted treatment standards, founded on reliable empirical evidence. It is clear that the Inquiry’s judgements were not based on such evidence.
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Clare Matheson protests too much

Former patient Clare Matheson remains bitter over the treatment she received at National Women's Hospital (NWH). In raising this matter, both in her recent letter to the NZMJ\(^1\) and in her earlier statements to the New Zealand Herald\(^2\) it appears that Matheson has forgotten, or chosen to ignore, the fact that in May 1992 she and 18 other patients of the 3037 treated at NWH in the years 1955–86 for carcinoma \textit{in situ} of the cervix relinquished all complaints about their treatment by accepting an out-of-court settlement of their claim for $25M in compensation.

This settlement was drawn up by lawyers acting for Dr Green, Professor Bonham, the University of Auckland and the Auckland Area Health Board and agreed to by the plaintiffs’ lawyer, Rodney Harrison. Under the terms of this settlement the plaintiffs accepted the sum of just $1.02M in full and final settlement of all claims for all damages and costs.

For their part, the defendants made no admission of fault or liability. While their lawyers were confident of winning the case, the estimated cost of doing so (it was set down for 15 weeks) was a major factor in their decision not to proceed. A press release also revealed that “A further factor which influenced settlement was the health of Dr Green and Professor Bonham, and the defendants’ belief that it was in society’s wider interest to lay the National Women’s saga to rest.”

Clearly unhappy with the terms of the settlement, Matheson invented another version of events. On 14 May 1992 Western Leader reporter Fiona Stewart quoted her as saying ‘I’m just so relieved it is finally over’, and ‘I’m also pleased liability has been admitted and some recognition of that has been made.’\(^3\) (my emphasis). Given her new-found passion for accuracy as evidenced by her pursuit of perceived errors in Professor Linda Bryder’s book \textit{A History of the “Unfortunate Experiment” at National Women's Hospital}, will Matheson now apologise for her own inaccuracy?

Matheson also made much of the fact that the Dean of the Auckland Medical School, Professor Derek North, publicly apologised on 27 May 1992 for the ‘stress and distress caused to many of the women involved in the treatment programme in the 1970s at National Women's Hospital’. This fell short of an admission of liability. It was also made solely on the Dean’s initiative, with official information showing he had not formally canvassed Medical School or other university staff for their views.

Whom was he apologising to? Not the 3018 patients who had not complained, and surely not the 19 who for a comparatively small amount of money had agreed that there was no fault in their management. And why, if he was so concerned, had the dean stood by while university lawyers drove a rather hard bargain?

Matheson has an appetite for litigation. In 1986 she successfully took a complaint against her GP to the Medical Disciplinary Committee for failing to take smears in the 6 years between her discharge from NWH in September 1979 and developing cervical cancer in September 1985. In 2010 her focus is on proving that she had invasive cancer of the cervix when she was discharged from NWH in 1979.
Her recent letter to the NZMJ incorporates excerpts from her NWH case notes. The entry for 27 January 1976 reads ‘Biopsy cervix = carcinoma of cervix’. However, the version in the Cartwright Report, where all of her case notes were reproduced in Appendix 11 (p. 282) reads: ‘Biopsy of cervix = carcinoma in situ’. This is a significant error and one she should retract.

Dr Charlotte Paul, one of the Inquiry's medical advisors, has also confirmed that Matheson did not have cancer on discharge in 1979. In her 1988 BMJ article, Paul cited Coney and Bunkle’s 1987 Metro article, which stated that Matheson's final histology report in 1979 ‘clearly showed she still had cancer’. Paul corrected this diagnosis, adding '[carcinoma in situ]' to the original quote. She did not, however, clarify or explain the difference.

Matheson cites the cross-examination of Professor Per Kolstad at the Inquiry as proof that she had, in his words, suffered ‘terrifying mismanagement’. In fact, Kolstad was a very poor witness. He muddled patients’ notes and became flustered when he realised that the temporising which he criticised was by his friend the late Dr McIndoe, and not by Green. Kolstad also remarked that on discharge Matheson could and should have had smears taken by her GP after 1979; this would have been unthinkable had she had cancer. The official body which ruled on Matheson’s management/condition did not support Kolstad’s interpretation of her management at NWH. The Medical Disciplinary Committee which adjudicated on her 1986 complaint did remark on Green’s overly optimistic letter to her GP but did not criticise the treatment she had received.

There are numerous claims in Matheson’s letter with which one could take issue, but I will restrict myself to one further example of her selective and misleading statements. One of her criticisms of Bryder was that ‘there is little point quoting specialists who say that a patient need have only three negative smears before discharge as opposed to my five’. The specialist who said that at the Inquiry was Professor Ralph Richart—Coney, Bunkle and Matheson's own star witness. She cannot have it both ways.

Watching the events surrounding the Cartwright Inquiry unfold from 1987, and as someone who knew the Green family, I became increasingly concerned by the injustices I observed. I began to attend the Inquiry hearings, collect news clippings, write letters, and request information under the Official Information Act. Based on this accumulated knowledge, I believe the injustices and misrepresentations of what happened at National Women's Hospital in the 1960s and 1970s have continued to the present day.

Shortly before his death, Herbert Green remarked to me that ‘Sometimes a kind of madness takes over a society. The only thing to do is wait it out.’ Perhaps the waiting is nearly over.

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Clare Matheson’s inaccurate reporting

In her letter to the editor published in the 27 August 2010 issue of the NZMJ, A patient’s response to recent criticisms of the findings in the report of the Cervical Cancer Inquiry 1988, your correspondent Clare Matheson apparently quotes the pathohistologic report for the biopsy of her cervix performed “re 27-1-76”. Seemingly incontrovertibly, she states “Biopsy cervix=Carcinoma of cervix – Excision appears incomplete”.

If she had checked how that report was recorded in the Cartwright Report she could have seen that its accurate duplication there in Appendix 11, on p.282, was not as “Carcinoma of the cervix” but as “Carcinoma insitu of the cervix”. Curiously perhaps, C Matheson had already quoted the phrase correctly and fully, p.69, in her own 1989 book “Fate Cries Enough” (which she freely offers you as her reference #14). If ‘Any thinking person must wonder how many other errors there are’, then here is one requiring correction. She must know the significance of her serious omission for the doctors involved.

Echoes! One is reminded that it was with the word ‘cancer’—not the words ‘carcinoma in situ’—that Ms Sandra Coney and another of your correspondents of 27 August, Ms Phillida Bunkle, had referred [Metro June 1987, p.56] to “Ruth’s” 1979 discharge from National Women’s Hospital: “as the final histology clearly showed, she still had cancer”.

It can be observed however, that even Prof C Paul, when quoting that pair’s same assertion in the Brit Med J [27Aug1988; p.533], was obliged to refute such a claim by expanding the clause they used to “‘she still had cancer [carcinoma in situ].’ “. Yet recently the New Zealand Herald persisted with this unfounded myth (“No accounting for mistakes”, issue of Saturday, 16 July 2010).

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A farsighted energy strategy would benefit health in New Zealand

The government’s draft national energy strategy\(^1\) is a curious mix of activism on fossil fuel exploration and lack of ambition on energy sustainability. At one and the same time it champions petroleum extraction but is passive about reshaping transport energy, and takes a low-key approach to energy efficiency targets. On the plus side, it recognises that a lower carbon economy presents opportunities, and that warm, dry homes are important. On the minus side, it fails to recognise the importance of a stronger national greenhouse gas target, and the inadequacy of the current emissions trading scheme (ETS).\(^2\)

Perhaps the single most important aspect of the draft strategy is its position on renewable energy. As the climate change evidence becomes more and more concerning, and in the face of rising prices for fossil fuels, the benefits of renewable energy generation (not just electricity generation) assume high importance. The draft strategy “retains the aspirational, but achievable, target that 90 per cent of electricity generation be from renewable sources by 2025 (in an average hydrological year)”.\(^1\)

This target is far from adequate in our view; a more appropriate target with clearer signalling value is 100% by 2030 (for an average hydrological year in terms of water supply to hydro lakes). As a small dynamic developed country, New Zealand should be an international exemplar in terms of responding to the very serious climate change problem,\(^3\)\(^4\) by developing an efficient low-carbon economy powered by renewable energy.

Moreover, given current levels of renewable electricity generation, the 100% target is within reach, even though the last 5% could be relatively costly. Furthermore, New Zealand should also have a target for use of renewable energy in the transport sector of at least 50% by 2040 (i.e., of all trips, whether public transport or private vehicles, half should be powered by renewable electricity or indigenous biofuel).

Below we detail the likely health benefits of adopting such targets. We also detail previous examples of international leadership by this country.

Health benefits for New Zealanders from shifting to an energy efficient and low-carbon economy—

- Benefits to respiratory and cardiovascular health from reduced air pollution if there was greater use of low-emissions vehicles\(^5\) e.g., electric cars and buses with both being supplied with renewably-generated electricity. Air pollution is a public health concern in New Zealand given the results of modelling work on mortality impacts\(^6\) and studies in Christchurch linking particulate levels in ambient air with respiratory symptoms\(^7\) and hospitalisations.\(^8\)\(^9\)

- Benefits from injury reduction associated with a reduction in road traffic volumes\(^10\)—which would occur with a shift from private cars to more energy efficient public transport.
• Energy conservation involving improved home insulation will cost-effectively benefit health as shown in New Zealand research.\textsuperscript{11,12} New Zealand studies also indicate that home insulation is associated with lower endotoxin levels\textsuperscript{13} and lower dust mite allergen levels.\textsuperscript{14} The low quality of New Zealand housing\textsuperscript{15} suggests major gains are possible from home energy efficiency measures in terms of health and cost savings to householders.

• Possible general health benefits to low-income New Zealanders if an expanded renewable energy sector helped generate net employment and reduced unemployment (e.g. as modelled for Scotland\textsuperscript{16} and Germany\textsuperscript{17})

• Likely benefits from reduced risks to occupational health associated with reduced dependence of fossil fuel extraction industries (e.g., respiratory disease and injuries from coal mining).\textsuperscript{18}

• Health benefits of avoiding pollution associated with the industrial processes foreshadowed in the draft strategy—of coal conversion to liquids, methanol and urea. There are also benefits from avoiding the significant risks of failure of systems for carbon capture and storage in New Zealand.

To achieve these targets a strong pricing scheme for greenhouse gas emissions would be required such as an upgrade to the currently weak ETS. In addition, much stronger energy efficiency policies in relation to transport—where the government currently lacks a coherent strategy – would be needed.

Better integration of urban design with transport planning is vital, as are a marked reallocation from new highway building to urban public transport, travel demand management, and measures to support walking and cycling. Such measures would trigger other health benefits:

• Gains from a shift to increased physical activity associated with active transport (e.g. cycling and walking)—even when considering potentially increased injury risk.\textsuperscript{5 19 20}

• Possible mental health benefits from improvements in urban planning (e.g. less suburban sprawl) and reductions in time spent commuting in private vehicles.\textsuperscript{21}

It is likely that these health benefits will collectively exceed the potential adverse health effects from any increased fuel poverty with higher electricity prices. The latter could also be ameliorated by targeted electricity subsidies (e.g. to low-income elderly residents), subsidies for wood pellets for use in home heating for low-income populations, or tax-benefit adjustments. Similarly, adverse health impacts from wind turbine noise can be minimised with appropriate placement of turbines and sound insulation of nearby buildings.

In addition to such health benefits are the economic benefits of greater energy security for New Zealand. The greater use of local renewable energy for transport will reduce national vulnerability to sudden oil price hikes from oil-producing cartels and conflict in petro-states. Oil and natural gas prices are also very likely to rise long-term as international demand continues to outpace supply.
Past leadership by New Zealand—The small size of this country has not prevented it from international leadership roles in the past. These include being the first country to give women the vote, being one of the first countries to establish key aspects of the welfare state, and being a key player in the establishment of the United Nations. New Zealand has also provided strong opposition to nuclear testing (with Australia), been the first country to develop national legislation against nuclear weapons in 1987, and has used its “honest broker” status to facilitate conflict resolution (e.g. in Bougainville).

This history suggests that New Zealand can make bold policy decisions and should continue to do so by having renewable energy, energy efficiency and energy conservation strategies that are exemplars to the world community as we collectively struggle to limit planetary damage from climate change.

The costs of being un-strategic—New Zealand also faces a key choice between a ‘she’ll be right’ approach to energy and carbon challenges on the one hand, and a proactive strategy on the other. There is now mounting evidence that China (New Zealand’s second largest trading partner) and Korea are tracking firmly towards a green economy. For example, China has recently announced it will introduce an ETS to its power sector in 2011, and it has very ambitious renewable energy transition targets, partly motivated by air quality and health concerns. Korea too is investing heavily in renewable energy.

Both China and Korea will be looking for their trading partners to be adopting complementary strategies, and expecting New Zealand to work with them in developing or exploiting new energy technologies such as solar water, photovoltaics and battery electric vehicles. If New Zealand does so, it increases the likelihood of preserving trading opportunities and New Zealand’s clean, green reputation in critical and growing markets. This will have long-term benefits for New Zealand’s prosperity and the health of our population.

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The doctor’s dilemma

Rather than responding to Phillida Bunkle’s letter¹ point by point I wish to draw readers’ attention to one example as an indication of the way in which she distorts evidence, that relating to expert witness Dr Joe Jordan and his discussion of Patient 60/64.²

Bunkle wrote:

“The “dilemma” Jordan referred to in the passage quoted by Professor Bryder, was a discussion about the decision to be made about patient 60/64 in 1981…The sentence in Jordan’s statement which occurs immediately before that quoted by Professor Bryder, but which she omits, could not be more explicit. He said, “I think that some definitive treatment to the vaginal vault lesion should have been instituted in the early 1960’s, and at the latest in October 1965, when the vaginal vault biopsy confirmed the presence of severe dysplasia.” The full text of Jordan’s evidence to the Inquiry, thus, makes clear his view, that the “dilemma” was created by the more than twenty years of prevarication about diagnosis and delays in treatment.’

Bunkle’s claim that this ‘dilemma’ related to 1981 and followed ‘more than twenty years of prevarication about diagnosis and delays in treatment’ shows a misreading of the case notes and Jordan’s commentary.

The case history of Patient 60/64, as set out by Jordan, reveals the patient had a cone biopsy and hysterecomy in 1960. She continued to have positive cytology. In 1973 she was found to have vaginal carcinoma in situ and vulval carcinoma in situ following a DNCB. Jordan noted ‘At this stage, Professor Green discussed treatment with the radiotherapist who felt that radiotherapy was not indicated because there was no invasive carcinoma. The alternative was a vaginectomy with vulvectomy, in the absence of symptoms, it was decided to await events with the hope and expectation that the lesion would not become malignant. She continued to have positive cytology and in 1977, she underwent a vulvovaginectomy.’ In 1981 she was referred to a urologist who confirmed the presence of squamous cell carcinoma of the urethra.

Dr Jordan then wrote his ‘Comments’ on the case, which Bunkle refers to. Here is the ‘full text’ which she asks for:

‘This would appear to be a rare case of generalised cancerisation of the lower genital tract in which the cervix, vagina and vulva are involved. I think that some definitive treatment to the vaginal vault lesion should have been instituted in the early 1960s, and at the latest in October 1965, when the vaginal vault biopsy confirmed the presence of severe dysplasia.

Again, one sympathises with the dilemma faced by Professor Green knowing that the treatment of this was either removal of the upper vagina or radiotherapy. Both procedures carrying a high morbidity and almost certainly, removing or interfering seriously with sexual function.

At the time Professor Green was still of the opinion that carcinoma in situ rarely, if ever progressed to invasive carcinoma and so decided to leave well alone. On the other hand, this course of action in a patient known to have squamous cell carcinoma in the cone biopsy in 1960, carried a certain element of risk.'
At the end of the day, the main problem seems to have related to the urethra and the vulva and it is probably that the squamous cell carcinoma found at the urethra meatus in 1981 by the urologist, occurred in spite of the fact that the vaginal vault disease had been left untreated for many years.

It is probable that even if she had had radiotherapy or vaginectomy to remove the vaginal vault lesion, that she would still at the end of the day, have had a squamous cell carcinoma of the vulva and urethra.’

Jordan’s discussion shows:

1. The ‘dilemma’ related to Green’s clinical decisions in the 1960s.
2. Green made his clinical decisions in conjunction with others, for example the Auckland Hospital radiotherapist.
3. Jordan acknowledged that the squamous cell carcinoma of the urethra occurred in spite of the fact that the vaginal vault disease had been left untreated, and that ‘It is probable that even if she had had radiotherapy or vaginectomy to remove the vaginal vault lesion, that she would still at the end of the day, have had a squamous cell carcinoma of the vulva and urethra.’

While I am not suggesting that Dr Jordan was not critical of Green’s clinical decisions—he most certainly was—his commentary also shows an awareness of the complexity of the situation; there were no simple answers. As an Oxford community health study stated in relation to carcinoma in situ in 1988, ‘The medical dilemma is to know when to treat and when to leave alone.’

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2. Jordan J. Evidence in Chief, presented to the Committee of Inquiry into Allegations concerning the treatment of cervical cancer at National Women’s Hospital and into other related matters, 10 September 1987, ‘Summary of case histories’, pp. 3-5.
Response to letters by Paul and Bunkle

The 27 August 2010 issue of the NZMJ contained further examples of the orchestrated campaign which has sought to undermine Professor Linda Bryder’s book, *The History of the 'Unfortunate Experiment' at National Women’s Hospital*, since its publication in August 2009. One of the repeated refrains has been that Bryder—‘without any legal or medical qualifications’—fails to understand the medical science at the heart of the story.¹ This angle was taken up by Associate Professor Jo Manning in the introduction to *The Cartwright Papers*, where she wrote ‘Given that Bryder is a social historian and not a medical scientist, a striking aspect of this book is her lack of deference to the expertise of other specialists in their field.’²

It is surprising therefore that Ms Phillida Bunkle, who is not medically or legally qualified, undertook the task of responding to Sir Iain Chalmers’ critique of Professor Charlotte Paul, with an interpretation of case notes and a commentary on the legal system, under the heading ‘Reassessing Cartwright—the factual record’ on 27 August.

While Bunkle defended Paul, Paul’s contribution to the 27 August issue of the NZMJ took the form of a response to Dr Paul Patten’s letter of 30 July, which challenged her claim that Professor Bryder had a ‘particular relationship … with gynaecologists whose work she was researching’.³

Paul denied categorically that she was referring to Professor Colin Mantell and Dr Tony Baird, stating that a close reading would show otherwise. Careful scrutiny of her comments in *The Cartwright Papers*, however, suggests otherwise.

Paul’s allegation was highlighted in Manning’s introduction to *The Cartwright Papers*, where she stated that ‘Paul’s reading of Bryder’s *History* corroborates her earlier surmise (Chapter 4) that the author’s work is influenced by a particular relationship she formed with some doctors at National Women’s while conducting the research.’³

Paul herself first referred to this on p.97 of *The Cartwright Papers*, where she alleged that ‘a group of gynaecologists appears to have guided her [Bryder] in determining whom she should take seriously and whom she should not’. She returned to this theme on p.118, where she noted that Bryder’s book had been ‘effusively welcomed by two other senior gynaecologists’, and that ‘All this adds weight to my earlier surmise (see chapter 4) that Bryder’s work was influenced by the particular relationship she formed with gynaecologists whose story she was researching.’ The footnotes for this section refer to Mantell and Baird; they make no mention of any other gynaecologist.

This view was also promoted by Paul’s colleague, Professor Sir David Skegg, in the New Zealand Herald of 19 September 2009, where he was quoted as saying: “This leaves her open to the accusation that she may have been ‘captured’ by some of the people who were unhappy with Judge Cartwright’s findings”, says Skegg. “One of the doctors interviewed and quoted [by Bryder] was found by the judge to have misled..."
the public during the inquiry.’ The two doctors named by Judge Cartwright on p.172 of her report were Mantell and Baird.

Closer examination of Bryder’s book demonstrates the absurdity and mischief in the claims of a particular relationship. Mantell was interviewed by Bryder’s research assistant in relation to the history of National Women’s Hospital but this was not used in *The Unfortunate Experiment*, and the only reference to the interview with Baird was his comment about Green’s views on Caesarean birth (p.85).

The other references to Baird in the book are to a statement he made at the 1988 Inquiry (p.85); a comment made by Coney about Baird in 1988 (p.106); Coney’s debate with Baird and Bruce Faris in 1990 (p.156); a reference to a letter by Baird in the *NZMJ* in 2004 (p.157); a 1988 letter written by Baird, held in the Joan Donley archives (p.162); his appearance as one of six individuals who questioned Coney’s interpretation of events in 1988 (p.173); his 1990 letter to the *New Zealand Herald* (p.178) and a 1993 quote about Baird by patients’ advocate Lynda Williams (p.179).

It is hard to see how a discerning reader could interpret any of this as evidence of Bryder being ‘captured’. When Baird sought redress from the publishers of *The Cartwright Papers* for these allegations the response he received was that the particular relationship derived from the fact he had been interviewed by Bryder’s research assistant. If such were the case, then no historian would be free from similar accusations of bias!

In her letter, Bunkle made many other comments which merit a response, but I will draw attention to just one—her statement that ‘It also follows from the judicial status of the Inquiry that when its findings are contested in the media the judge cannot defend herself.’ She might have added that judges are expected to be impartial. This did not inhibit Dame Silvia from addressing the 2008 ‘Twenty Years After’ conference in Auckland, which celebrated the anniversary of the publication of the Cartwright Report and was the basis of *The Cartwright Papers*.

Interestingly, the Bridget Williams Books online advertising of *The Cartwright Papers* still names Dame Silvia as one of the contributors, though her paper did not appear in the published volume. Many would view the continued inclusion of her name on the website (more than 8 months after publication of *The Cartwright Papers*) as an endorsement of its contents and conclusions.

In this context consideration might be given to one of Dame Silvia’s comments at the anniversary celebrations: ‘But I realise now that this was a drama unfolding in the nation’s living rooms, a drama in which there were goodies and baddies, and for all time, I was placed with the goodies, if not by the medical profession.’

Fortunately, most historians, including Bryder, do not take such a simplistic approach to their subject.

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Derek A Dow
Honorary Senior Lecturer
University of Auckland
References:

1. Letter to the editor from Professor Linda Holloway and Professor Charlotte Paul, Listener, 22 August 2009, p.7; Professor Sir David Skegg, New Zealand Herald, 19 September 2009.
3. ibid, p.1.
4. Personal communication from Dr Baird, September 2010.
A particular relationship: part 2

I don’t know what a chap is supposed to think; there I was, clearly identified in Chapter 6 of The Cartwright Papers, with reference to the Report of the Inquiry, as having extraordinary powers to lead Professor Bryder astray through a particular relationship—despite having met her only briefly before she started her work on the history of women’s health in New Zealand.

Now, Professor Charlotte Paul writes¹ (NZMJ 27 August 2010) that it is not me. I have yet to receive the acknowledgement she says she has made to me but her publisher seems to support the concoction of a particular relationship because I was interviewed by Professor Bryder’s research assistant.

Anyway, I shall just keep on being a simple clinician, doing my best to help women who need medical care, facing the daily dilemmas of specialist practice secure in the knowledge that what I do now will be criticised retrospectively in the future.

It is clear that academic freedom now means that University staff have no code of conduct and the Dunedin group can continue to pillory Herb Green and Linda Bryder with the blessing of the Vice-Chancellor.

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MAH (Tony) Baird
Auckland Uro Gynaecology
Parnell, Auckland

Reference:
Sidney Scott McCann


Born 12 August 1923 in Belfast, Northern Ireland. Died 31 July 2010 after a long illness. He studied medicine at Queens University, Belfast 1941–1947.

In 1948–1951 he had house surgeon and house physician posts at Portadown Hospital near Belfast and short general practice work in the same area. He was the Medical Officer for two trips to Australia in 1951 with P & O Steam Navigation Company; meeting his future wife Dr Judith McCann QSO on the second trip. He returned to Queens University, Belfast to study Public Health & Obstetrics in 1951. He came to NZ in 1952 joining the Faris Medical Practice at Hauraki Corner, Takapuna, where he worked for the next 20 years in a large general practice with a busy maternity component.

Anaesthetic Registrar at Auckland Hospital and National Women’s Hospital (1969) posts followed by private anaesthetic practice on the North Shore for the next 17 years mainly at Lister Hospital, Devonport Naval base and North Shore Hospital Obstetric Unit.

He retired in 1989. He had four children with wife Judith. Eleven grand-children & one great grand-child. Wife Judith died in September 1999 after a long illness.

Scott suffered from peripheral vascular disease and two below knee amputations (2005 and 2009) followed by the complications from a stroke May 2009. This affected his speech and ability to swallow.

Personal qualities of good humour, integrity, loyalty and compassion endeared him to his patients and colleagues alike and his fortitude and courage this past year have been an inspiration to many people.

Scott from Port Stewart Lough Neagh Ireland, always lived by the water—Takapuna on the beach and Lake Taupo. There Scott and Judy raised their children steeped in water sports and the mountains now traditionally carried on by all his offspring.

His 40 years as a member of the North Shore Golf club will always be remembered for his fondness of collecting lost golf balls.

A colleague and friend, Dr Duncan Finlayson (North Shore, Auckland) wrote this obituary.
Derek Alexander Larnder

OBE for Services to Dermatology

Dermatologist Dr Derek Larnder was liked and trusted throughout Canterbury. His dedication to caring, his consideration of the underlying causes of skin problems and his production of reliable medications endeared him to thousands of patients.

Larnder died on 3 December 2009 in Christchurch, aged 87. One of the old school of doctors, he epitomised his generation’s altruism and dedication to helping people in need.

His daughter, Sarah, says her father did not like to see people suffering and worked hard to find ways to help them. His level of commitment to patients limited his available time. However, he was a loving father who made time for the family and ensured all felt important.

His wife, Prue, says he packed an enormous amount of work into his life. Although never boastful, he took satisfaction from his professional and voluntary service and quiet pride in his achievements.

The awards and honours he received meant much to him but, to anyone meeting him, he was a down-to-earth and gentle man.

Larnder was born in Timaru and began school at Waimataitai Primary School, before the family moved south to Waimate. He excelled academically and in sport at Waimate High School, completing his secondary education at Timaru Boys’ High School.

Studying medicine at the University of Otago in the 1940s prevented him from serving overseas in World War II, but he took part in army medical training camps. He demonstrated prodigious powers of memory as a student and, soon after, as a lecturer in anatomy. He frequently astounded his classes by calling the roll of 120 forwards and backwards, without notes.

Larnder graduated as a doctor in 1947 and worked at Rotorua and Wanganui hospitals, as well as lecturing at the University of Otago. He enjoyed travel and made tours as a ship’s surgeon, including one on a troopship taking British forces to the Korean War.

His heart was in surgery and he went to England for specialist study, achieving his qualifications in 1955. However, a medical allergy made surgical work impractical
and he switched to his second choice, dermatology. Back in England for further study, he met and married English nurse Prue Daniel.

He returned to New Zealand in 1959, settling in Christchurch. He worked as a consultant to Christchurch Hospital and in private practice in Colombo St. Prue says he enjoyed his work and the people he met. “He was a great admirer of the old-fashioned general practitioner and liked to be available for his patients,” she says. His manner with patients inspired trust.

He understood that skin conditions were often related to stress. His rapport with people helped treat “the inner man” by giving hope and the confidence to cope with life, thus reducing stress levels. Larnder laconically explained the link between stress and skin complaints with the line, “If you’re bitchy, you’re itchy”.

He was invited in 1981 to join the Order of St Lazarus, an ancient, universal Christian organisation dedicated to the care and assistance of the poor and the sick. The order was formed to combat leprosy and membership became an important part of Larnder’s life. He hosted a pan-Pacific symposium on leprosy and did much voluntary work for the order. With its help, he launched Christchurch’s first free clinics for skin cancer checks, in 1986. The popularity and success of the clinics led to their extension throughout the South Island and to the Chatham Islands.

Larnder suffered a brain haemorrhage in 1986, but recovered and returned to work. Three years later, the Order of St Lazarus awarded him its prestigious Order of Merit and the Queen awarded him the OBE for services to dermatology. He retired in 1993.

His participation in professional associations included roles of president of the New Zealand Dermatological Society, and co-founder and president of the Psoriasis Association. He was the only doctor in Australasia to become an honorary overseas member of the British Association for Dermatologists and was a fellow of the American Academy of Dermatology. He was Senior Commonwealth Fellow in Dermatology in 1972.

In his retirement, Larnder at last found time for sport and travel. He and Prue travelled extensively.

Derek Alexander Larnder, born Timaru, 22 June 1922; died Christchurch, 3 December 2009. Survived by wife Prue, daughters Sarah, Bridget, Katie and Diana and nine grandchildren.

Mike Crean wrote this obituary; it first appeared in The Press newspaper (Christchurch).