INFORMATION FOR AUTHORS
First page following cover

EDITORIALS
229 What price accountability? The Editors
230 The future of New Zealand’s public health system. From top-down to bottom-up? The Editors

ORIGINAL ARTICLES
231 Improved clinical management of retinoblastoma through gene testing Anthony Raizis, Richard Clemett, Rob Corbett, Julie McGaughran, Jane Evens, Peter George
234 Acute gastroenteritis associated with seafood privately imported from the Pacific Islands Vanessa Thornton, Wayne Hazell, Greg Simmons
237 Could laboratory-based notification improve the control of foodborne illness in New Zealand Greg Simmons, Robyn Whittaker, Kerry Boyle, Arthur J Morris, Arlo Upton, Lester Calder
241 Infant bed-sharing among Pacific families in New Zealand Janis Paterson, Colin Tukuitonga, Sarnia Butler, Maynard Williams

BRIEF REPORT
244 Auckland paediatric liver transplant experience 1990-2000 Janine Smith, Alison Wesley, Simon Chin, Jane Harding

PRELIMINARY REPORT
246 Can cancer centres in New Zealand help the Cancer Registry generate survival data? A pilot study in prostate cancer TKA Evans, DS Lamb, DA Cornes, J Fraser, CA Johnson, DA Hamilton

VIEWPOINT
247 Bioterrorism in the Northern Hemisphere and potential impact on New Zealand Nick Wilson, Douglas Lush

MEDICOLEGAL DIARY
251 Enduring powers of attorney Jonathan Coates

From July 2002 the New Zealand Medical Journal will be published electronically: there will be no paper version. Members and subscribers, to ensure you receive regular updates, send your email address to shani@nzma.org.nz
Editor: Gary Nicholls
Deputy Editors: Philip Bagshaw, Evan Begg, Peter Moller, Les Toop, Christine Winterbourn
Biostatistician: Chris Frampton  Ethicist: Grant Gillett
Emeritus: Pat Alley, John Allison, Jim Clayton, Roy Holmes, John Neutze
Editorial Board: George Abbott, Bruce Arroll, Sue Bagshaw, Gil Barbezat, Richard Beasley, Lutz Beckert, Ross Blair, Antony Braithwaite, Stephen Chambers, Barry M Colls, Garth Cooper, Brett Delahunt, Matt Doogue, Pat Farr, Jane Harding, Andrew Hornblow, Geoffrey Horne, Rod Jackson, Peter Joyce, Martin Kennedy, Graham Le Gros, Tony Macknight, Tim Maling, Jim Mann, Colin Mantell, Lynette Murdoch, Bryan Parry, Neil Pearce, David Perez, Anthony Reeve, Ian Reid, Mark Richards, Andrê van Rij, Justin Roake, Peter Roberts, Bridget Robinson, Prudence Scott, Norman Sharpe, David Skegg, Bruce Smaill, Rob Smith, Ian St George, Andy Tie, Ian Town, Colin Tukuitonga, Harvey White

I Information for authors
From July 2002 the New Zealand Medical Journal will be published electronically: there will be no paper version. More information about the electronic NZMJ will be available soon for contributors.

Guidelines for authors are in accordance with the Uniform Requirements for Manuscripts submitted to Biomedical Journals. Full details are printed in NZ Med J 1997; 110: 9-17, Med Educ 1999; 33: 66-78 and are on the NZ Medical Association website – www.nzma.org.nz. Authors should be aware of the broad general readership of the Journal. Brevity and clear expression are essential. Most papers should be 2200 words or less, the maximum being 3000 words and 30 references. For papers accepted for publication which exceed three printed pages (around 3,000 words) there will be a page charge of $450 plus GST for each printed page. Letters should not exceed 400 words and ten references. Case reports must be no longer than 600 words, with up to six references and no more than one Figure or Table. Requirements for letters, obituaries and editorials are on the website. All material submitted to the Journal is assumed to be sent to it exclusively unless otherwise stated.

In, or with your covering letter, the following is required:

1. Each author must give a signed personal statement of agreement to publish the paper or letter.
2. One (or more) author must state: “I (we) accept full responsibility for the conduct of the study, had access to the data, and controlled the decision to publish”.
3. Authors must state whether potential conflicts of interest do or do not exist.
4. All sources of funding must be stated explicitly and this information will be published with the paper.

The paper: Papers are to be written in English and typewritten in double spacing on white A4 paper with a 25 mm margin at each side. Send three copies of the paper. Wherever possible, the article should also be submitted on a 3.5-inch disk. Although Word 5.1 (or later version) is the program of choice, other word-processing programs are acceptable. Organise the paper as follows:

Title page – the title should be brief without abbreviations. Authors’ names, with only one first name and no degrees should be accompanied by position and workplace at the time of the study. Corresponding author details with phone, fax and email should be given, and the text word count noted.

Abstract page – this must not exceed 200 words and should describe the core of the paper’s message, including essential numerical data. Use four headings: Aims, Methods, Results, Conclusions.

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

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

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

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

The Journal does not hold itself responsible for statements made by any contributors. Statements or opinions expressed in the Journal reflect the views of the author(s) and do not reflect official policy of the New Zealand Medical Association unless so stated.

Addresses
Editorial: All editorial correspondence is sent to Professor Nicholls, c/o Department of Medicine, Christchurch Hospital, PO Box 4345 Christchurch, New Zealand. Telephone (03) 364 1116; Facsimile (03) 364 1115; email barbara.griffin@chmeds.ac.nz
Advertising: All correspondence is to be sent to the Advertising Manager, Print Advertising, 83-91 Captain Springs Road, PO Box 13 128 Onehunga, Auckland. Telephone (09) 634-4982; Facsimile (09) 634-4951; email printad.auck@xtra.co.nz or PO Box 27194, Upper Willis Street, Wellington. Telephone (04) 801-6187; Facsimile (04) 801-6261; email printad.wgtn@xtra.co.nz
Circulation: All correspondence about circulation, subscriptions, change of address and missing numbers is sent to Chief Executive Officer, New Zealand Medical Association, PO Box 156, Wellington. Telephone (04) 472-4741; Facsimile (04) 471-0838. email nzmed@nzma.org.nz
Publisher: The Journal is published by Southern Colour Print, PO Box 920, Dunedin. Telephone (03) 455-0554; Facsimile (03) 455-0359
Subscriptions: New Zealand – standard mail NZ$255.15, fastpost NZ$272.25 (GST incl); overseas surface mail NZ$280.00, overseas airmail – South Pacific/Australia NZ$340.00; America/Asia/India/Europe NZ$420.00; Africa/Middle East NZ$490.00. All subscription enquiries to NZ Medical Association, as for Circulation above.

New Zealand Medical Journal 24 May 2002
Accountability is a laudable concept but comes at a price. We live in a culture of increasing intolerance to uncertainty in which blame, litigation and punitive compensation have become commonplace. This culture has been exploited by the media. Health continues to provide endless opportunities for awkward questions in parliament to put political opponents on the spot. This negative publicity and the mechanisms for accountability it has ushered in, result in an increasing feeling among health care professionals that they are over regulated and over scrutinised. Policy makers, funders and regulators need to understand and accept that there are inevitable uncertainties in health care. Paradoxically, these groups do not appear to be subjected to the same level of accountability.

Few would dispute that there have been some unfortunate, high profile New Zealand episodes that could have been prevented by better ethical and professional systems of scrutiny. However, many at the receiving end of the current overly bureaucratic accountability systems would argue that the pendulum has now swung too far in the opposite direction. According to recent surveys, morale in most parts of the health system is at an all time low. A consistent theme among the health reforms of the 90’s demonstrated, patients would be better served if they were treated in the evidence-free and incompetent manner that health professionals feel more positive about the time required to record accountability data?

It must be realistic and it comes with a price that must be borne by all.
The Editors


The future of New Zealand’s public health system. From top-down to bottom-up?

The past decade has seen our public health system directed centrally by the financial imperative (efficiency), centralised control of disparate local circumstances, and top-down management. In this we have followed the United Kingdom’s (UK) National Health Service (NHS). The UK, at last realising there is a real crisis, has made two decisions. The first is to inject an extra 40 million pounds sterling into the NHS. The second, and much more important decision, appears to be that there will be “another round of inspection, monitoring and audit” to “trace every single penny spent to ensure that it goes where ministers require”. A recent paper in the British Medical Journal entitled “The rise of regulation in the NHS” elaborates on new monitoring agencies.” In an article in The Times of London entitled Leaving the running of the NHS to the professionals, Simon Jenkins predicts the outcome will be a disaster. “The NHS seems unable to push its performance into Europe’s premier league. It cannot handle more money and convert it into a better service”. The reason given is that “it has for two decades endured a leadership and staff encumbered by insensitive monitoring, by national standardisation and persistent political exposure. Imposing on the service yet more of the same can only be catastrophic. It will waste more money and exasperate more staff. The case for privatisation will become overwhelming”. It is all very well to criticise a plan. What does Simon Jenkins propose instead? “The key to sensitive health-care is deregulating the painful task of rationing to frontline professionals.” Yes, he is suggesting that central authorities need to trust health professionals to define and address the unique health concerns of their local populations. He states that autonomous professional judgement is the essence of the welfare state. Along the same lines, Onora O’Neill concluded her Reith Lecture this year by stating: “If we want a culture of public service, professionals and public servants must in the end be free to serve the public rather than their paymasters.”

In New Zealand, organisation of and advice regarding the public health system over the past 10-15 years has generally involved carefully selected health professionals with little input from those elected by their peers. At a local level also and with few exceptions, input by health professionals elected by their peers into organising delivery of services, has been minimal. It is hardly surprising that the repeated implicit votes of no-confidence in the judgement of health professionals from corporate managers, the Ministry of Health, Treasury and a long line of ministers of health, has had a negative impact on our public health system and the morale of its workers. Continuation of the current centralised, ideologically driven approach will ensure further deterioration of the public health system in New Zealand, ongoing wastage of precious health dollars and further calls for privatisation. We need less (not more) accountability to central bureaucracy. Simon Jenkins states, the NHS “needs a minister who is brave enough to call off his rottweillers, his auditors, his league tables, his commissioners. It needs doctors and nurses ready to answer to their patients for the money they spend….Like the rest of the public services, it needs professionals ready to profess their craft, not to count government beans.”

The views of Simon Jenkins relating to the NHS apply equally to New Zealand. Politicians and Treasury need to understand that they must harness the skills and energy of their health professionals – or lose them. A change in direction is needed urgently. The Editors

1. Jenkins S. Leave running the NHS to the professionals. The Times 2002 April 19.
Improved clinical management of retinoblastoma through gene testing

Anthony Raizis, Scientific Officer, Department of Molecular Pathology, Christchurch School of Medicine; Richard Clemett, Ophthalmologist, Department of Ophthalmology; Rob Corbett, Paediatric Oncologist, Department of Paediatrics, Christchurch Hospital, Christchurch; Julie McGaughran, Geneticist, Northern Regional Genetic Services, Auckland; Jane Evans, Pathologist, Department of Anatomical Pathology, Christchurch School of Medicine, Christchurch; Peter George, Pathologist, Department of Molecular Pathology, Christchurch School of Medicine, Christchurch.

Abstract

Aims. To investigate the relative benefits of retinoblastoma gene testing over conventional ophthalmological screening methods in a New Zealand setting, and to determine the importance of tumour material in resolving germline status.

Methods. Three cases of gene testing are described to illustrate the clinical advantages over conventional ophthalmological screening. To determine the role of tumour material in resolving germline status, 24 New Zealand families were tested, of which tumour material was available for eight.

Results. In the three cases reported, we found genetic testing of the RB1 gene resulted in clinically significant benefits and cost savings. When fresh tumour was available for high molecular weight DNA extraction, germline status was resolved in 8/8 (100%) cases. In these cases tumour mutations were not present in the corresponding peripheral blood DNA, indicating that the tumours were sporadic. In the absence of tumour DNA, mutations were identified in only 8/13 (62%) heritable cases. Germline status remains unresolved in all of the three cases of unilateral tumour without a family history or tumour DNA.

Conclusions. Our experience indicates that retinoblastoma gene testing has significant benefits to the affected individuals and their families in New Zealand. Moreover, DNA extracted from fresh tumour allows retinoblastoma germline status in most cases to be defined. Without tumour material, the germline status of potentially sporadic cases will remain undetermined since the absence of detectable RB1 coding region mutations does not exclude all possible mutations in the RB1 gene, which is too large for DNA analysis. A lack of conclusive results will mean that infants will be subjected to the unnecessary inconvenience of surveillance under general anaesthesia.

NZ Med J 2001; 115: 231-4

Retinoblastoma is a malignant tumour that originates from embryonic retinal cells. It is the most common intraocular cancer of infants with a frequency of 1 in 18 000 live births. Early genetic diagnosis and focal treatment, while lesions can be still be ablated, is central to an effective management strategy.

Inheritance of a mutation in one copy of the RB1 gene leads to a hereditary predisposition to retinoblastoma. Tumour development is initiated when a mutation of the second allele occurs. Somatic RB1 mutations in embryonal retinal tissue lead to non-hereditary unilateral retinoblastomas from a solitary focus. However, some unilateral tumours are the result of inherited mutations that confer risk to the affected individual with a risk of subsequent malignancy.

Germline mutations generally result in bilateral disease and/or multiple tumours. There is a 3% risk of an associated intracranial retinoblastoma and a high risk (6-35%) of a second non-ocular malignancy later in life, especially when external beam radiotherapy is used for ocular treatment.

Here we report the genetic analysis of three retinoblastoma pedigrees to demonstrate outcomes, cost savings and other implications for families with either sporadic or heritable disease. We also investigate the role tumour tissue plays in the resolution of germline status of sporadic unilateral cases.

Methods

Tumour and blood DNA extraction. Each tumour sample was snap frozen in liquid nitrogen and subsequently stored at -80°C. High molecular weight tumour DNA was extracted by SDS/proteinase K treatment, followed by phenol/chloroform extraction. Peripheral blood (5mL) was collected in EDTA tubes and DNA extracted as previously described.

Polymerase chain reaction (PCR). Exons of the retinoblastoma gene were amplified in two steps. In the first step long PCR was used to amplify segments containing several RB exons in a single multiplex with previously described primer sequences. Using the long PCR products as templates, individual exons were amplified using 26 primer pairs containing a GC clamp in a single multiplex PCR reaction.

Two-dimensional gene scanning. Mutations were located with a two-dimensional gene scanning (TDGS) technique that combines a size separation step with denaturing gradient gel electrophoresis (DGGE). The products obtained in the multiplex reaction were separated electrophoretically, according to size, through a native 10% acrylamide gel. The DNA fragments were then separated in a second dimension through a denaturing gradient of 0-50% urea-formamide. Electrophoresis was done in a TDGS electrophoresis tank (CBS Scientific Inc, California) according to manufacturer's instructions.

DNA sequencing. PCR products showing abnormal mobility on two-dimensional gene scanning were sequenced using the dideoxy chain termination method. Labelled [3P]-dATP as well as Thermosequenase were both supplied by Amersham and used according to manufacturer's instructions.

Restriction enzyme analysis of mutations. In some cases the presence of mutations was confirmed by using an appropriate restriction enzyme based assay.

DNA methylation. Analysis of DNA methylation was done by the bisulphite method as previously described.

Results

DNA testing was done for a total of 24 New Zealand families and is summarised in Table 1. Tumour samples were collected from eight patients, whilst in the remaining 16 families only the proband's peripheral blood DNA was available for genetic analysis. The results of testing three affected with retinoblastoma are presented.

Case 1. A child of four months with unilateral retinoblastoma was treated by enucleation. In the absence of
gene testing, 3-4 examinations under general anaesthesia (EUA's) per year would be required to exclude disease in the contralateral eye. Since neither parent was affected, and the infant’s tumour was unilateral, there were no clinical clues to suggest a germline RB1 mutation. To establish germline status in this family genetic testing of tumour DNA was performed. TDGS revealed a mutation in exon 14, which was homozygous. Subsequent sequence analysis revealed that a nonsense mutation R455X was homozygous in the tumour DNA (Figure 1). Although both alleles in tumour DNA had this mutation, the sequence of DNA from the infant's peripheral blood was normal. This result indicates that the child did not have a germline mutation and no longer required regular ophthalmological screening to detect a second tumour.

**Table 1. Frequency of RB1 mutation detection in New Zealand cases of retinoblastoma.**

<table>
<thead>
<tr>
<th>Cases involving small mutations, deletions and insertions (alleles)</th>
<th>Family history/bilateral</th>
<th>No family history/unilateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood DNA</td>
<td>Tumour available</td>
<td>No tumour available</td>
</tr>
<tr>
<td>8(8)</td>
<td>4(8)</td>
<td>0</td>
</tr>
<tr>
<td>Cases involving DNA methylation (alleles)</td>
<td>N/A</td>
<td>4(8)</td>
</tr>
<tr>
<td>Cases involving DNA methylation (alleles)</td>
<td>N/A</td>
<td>4(8)</td>
</tr>
<tr>
<td>Total Number of affected alleles detected</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>Total cases (expected affected alleles)</td>
<td>13(13)</td>
<td>8(16)</td>
</tr>
<tr>
<td>Percentage of alleles resolved</td>
<td>62%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Figure 1. Sequence analysis of the R455X mutation in case 1. Blood DNA (left four lanes) and tumour DNA (right four lanes) was amplified by PCR and the products sequenced. The mutated sequence (circled right) is homozygous for the R455X mutation, with no mutation observed in blood DNA (circled left). The base change is a cytosine to a thymidine (underlined) resulting in a stop codon (TGA). Figure shows sequence of the antisense strand.

**Case 2.** A child at two months was diagnosed with trilateral retinoblastoma ie, affecting both eyes and a primitive neuroectodermal tumour (PNET) in the region of the pineal gland. Enucleation of the right eye was followed by intensive chemotherapy and stereo-tactic radiotherapy for the midline tumour. Although tumour DNA was unavailable, the occurrence of trilateral disease was strong evidence of a germline mutation. Other family members including parents and two young sisters were clinically unaffected. However, regular EUAs were considered necessary for these sisters, due to the 50% risk that they might carry RB1 mutations.

TDGS identified a mutation in exon 3 of the RB1 gene from the affected infant's peripheral blood DNA. Sequence analysis showed that this corresponded to a 304-305delTG frameshift deletion. Figure 2 shows that while the proband has a two base pair deletion, neither parent nor the two siblings have the mutation. This result indicates that none of the other family members are germline carriers of the RB1 mutation, although it remains possible that one of the parents might be a germline mosaic. This result confirms that the affected infant has a germline mutation with a high risk of developing a second malignant tumour and so regular surveillance by EUA is required. In contrast the two sisters no longer require EUA and the parents do not have increased risk of osteosarcoma. Because of the risk that one of the parents may be a gonadal mosaic any future siblings will need to be offered gene testing or screened with regular EUA.

**Case 3.** A first child was diagnosed with a large retinoblastoma adjacent to the optic disc in one eye and multiple small tumours in the second eye at three months of age. The child’s mother had a past history of unilateral retinoblastoma but did not have genetic counselling prior to her pregnancy. Upon birth of a second child, cord blood was collected for gene testing.

TDGS of peripheral blood DNA derived from the mother identified a mutation in exon 18 and sequencing of genomic DNA showed that this mutation was an R579X mutation, predicting truncation and inactivation of the RB1 gene. A restriction-based assay (Figure 3) confirmed that the mutated allele is present in DNA from both the mother and first child, but absent in the newborn infant. This result indicates that the second newborn did not inherit the genetic susceptibility to retinoblastoma, and does not need regular examination under general anaesthesia.

**Tumour DNA analysis.** The role of tumour DNA analysis was compared with peripheral blood analysis alone in 24 families referred for gene testing. Tumour DNA was available from eight affected infants all of whom had unilateral disease and no family history of retinoblastoma. Peripheral blood DNA was obtained from the affected members of sixteen additional families of which thirteen were heritable.

Mutations were identified in 16/16 alleles (Table 1) from the eight tumour DNA samples analysed. In the eight tumours where mutations were found on both alleles, the corresponding peripheral blood DNA was normal, confirming that they were sporadic.
No mutations were detected in the three cases with unilateral disease and where there was no family history and no tumour DNA available. The absence of detectable mutations means that germline status is unresolved, and those at risk in these families must continue with EUA examinations.

In the remaining thirteen heritable cases it was possible to detect mutations in the peripheral blood DNA of eight probands, indicating that a significant proportion of heritable cases can be resolved without tumour DNA.

Discussion

The three cases we report illustrate the use and benefits of RB1 gene testing. In the first case the child was found not to carry the pathogenic germline mutation, such that there was no increased risk for a second malignant tumour. Nor was there a need for frequent EUA follow-up to detect subsequent tumours in this child or in future sibs. Here the availability of tumour DNA was critical to determining risk of future tumours. When analysing blood DNA alone, the absence of detectable mutations in the RB1 gene coding regions does not mean that the gene is unaffected. Some germline carriers are likely to be caused by mutations in introns, which are far too large for DNA sequencing. Gene analysis of blood DNA alone does not mean that an infant is unaffected unless it can be shown that the mutation(s) in tumour DNA are absent in peripheral blood DNA ie, germline DNA.

The second case demonstrated a new germline mutation, occurring in either the developing foetus or the gonads of one parent. The affected child has a significant risk of developing a second tumour and requires careful clinical surveillance. The affected child has a 50% probability of passing the mutant RB1 gene to his/her children. Here we found that the affected child's siblings did not carry the 304-305delTG mutation indicating that they are not at risk and do not need to continue their frequent EUAs.

Future children could by analysed early for the RB1 mutation through amniocentesis or by testing of cord blood.

In the third case, both the mother and first child were found to be carriers of an R579X mutation. Genetic analysis of cord blood revealed that this second child did not have the mutation. In the absence of gene testing, the second child would have received frequent EUAs for the first five years of life.

In these three cases, gene testing allowed four siblings to be discharged from ophthalmologic follow-up, an estimated lifetime cost saving of at least NZ$100 000. This alone more than covers the cost of all genetic testing done in New Zealand.

To date we have examined 24 cases of retinoblastoma, but fresh tumour samples were available for only eight. Table 1 shows that, where tumours were available, 8/8 unilateral cases were resolved which is similar to the experience of others.12 These mutations were not detected in peripheral blood DNA. Clearly, had tumour DNA not been available, then the absence of mutations in blood DNA would have been inconclusive. This is illustrated by the three unilateral cases without a family history (Table 1). Here the lack of tumour DNA means that their germline status cannot be resolved.

The identification of mutations in only 62% of clinically heritable cases is typical of international experience.13 Overall, 42% of all heritable cases are caused by point mutations, 26% by small deletions/insertions, 15% large deletions and 17% are not detectable by current methods.13 These remaining unresolved heritable cases are likely to have unidentified deletions or mutations within introns. These will require alternative techniques for mutation detection.

Our results emphasise that an important aspect of DNA analysis for patients with unilateral disease is the routine collection and storage of tumour material (Table 1). Once mutations are identified in tumour DNA, their presence or absence in blood DNA (germline) can easily be determined.

Ophthalmologists should arrange a suitable strategy for harvesting tumour, whenever an eye is excised for retinoblastoma. Contamination of adjacent tissues during tumour harvesting may interfere with the histological diagnosis and estimation of prognosis. Preferably the patient should be referred to a centre where tumour extraction and storage is routine. It now appears negligent not to provide for genetic testing at the time of excision of an eye, given the beneficial impact of analysis on management of the infant and on the family. Recovery of DNA from tumours embedded in paraffin blocks is now possible for gene analysis, but analysis is considerably simpler and cheaper when using fresh tumour material.

Gene testing for retinoblastoma from overseas laboratories has been under-utilised in New Zealand due to the high cost for tests and the long delay in obtaining a result. As a consequence many retinoblastoma families in New Zealand have not been offered gene testing. We have now demonstrated that application of this technology within New Zealand can have a useful impact on affected individuals and their families.

Although the ethics of genetic testing have been widely discussed,14 we have not yet encountered a parent who has declined genetic testing. Parents understand the need to establish risk and where a germline mutation is identified, life-long clinical surveillance to ensure preservation of sight and life is mandatory.

Clinicians considering referral for retinoblastoma genetic analysis may obtain more information and contact details at the following WEB site: http://www.chmeds.ac.nz/research/retinoblastoma.html

Acknowledgements. This work was supported by the Canterbury District Health Board and the McClelland Trust. We would also like to thank Drs Diane Kenwright, Joanne Dixon and Alice Christian for their valuable assistance in obtaining samples.

Correspondence. Dr Peter George, Department of Molecular Pathology, Christchurch School of Medicine, PO Box 4345, Christchurch. Fax: (03) 364-0545; email: peter.george@cdhb.govt.nz

Figure 3. Restriction analysis of the R579X mutation in case 3. Exon 18 of RB1 was amplified and the PCR products cleaved with DdeI which cleaves the mutant allele 5’CTGAG-3’ but not the wild-type allele 5’CCGAG-3’. N: normal allele and M mutant allele. Lane 1 is a normal control, lane 2 is the mother, lane 3 is the first child and lane 4 is cord blood derived from the newborn child.
Acute gastroenteritis associated with seafood privately imported from the Pacific Islands


Abstract

Aims. To investigate a potential link between consumption of food privately imported from the Pacific Islands and presentation with acute gastroenteritis to Middlemore Hospital Emergency Department.

Methods. This was a three month prospective observational case study that included patients aged greater than fifteen years presenting with acute gastroenteritis and a history of food privately imported from the Pacific Islands. Data included case demographics, symptoms, island of food origin and type. Stool and blood samples were collected and analysed.

Results. Of 358 patients who presented to Middlemore Emergency Department during the study period with gastroenteritis, 34 (9.4%) had a history of consumption of food privately imported from the Pacific Islands. The seafood came from Tonga (23 cases), Samoa (10 cases) or Niue (1 case). The implicated seafood was shellfish (28 cases), jellyfish (2 cases), fish intestine (2 cases), seaweed or seaslug (1 case each). Fourteen patients (41%) provided stool samples; all were culture positive for Vibrio parahaemolyticus (VPH).

Conclusions. This case series confirms a link between acute VPH gastroenteritis and consumption of seafood privately imported from the Pacific Islands. A number of public health initiatives to reduce the burden of VPH gastroenteritis among Auckland's Pacific Islanders have commenced. The Ministries of Health, Agriculture and Forestry are considering tighter controls or banning food privately imported from the Pacific Islands.

MIDDLEMORE HOSPITAL

MIDDLEMORE HOSPITAL

Middlemore Hospital is situated in Manukau in the heart of South Auckland. The population of Manukau includes 20.7% who are Polynesian. This is the highest proportion of Polynesians in any New Zealand district.1 Middlemore Hospital Emergency Department (ED) assesses approximately 70 000 patients annually. Emergency Physicians at Middlemore Hospital noticed a subgroup of patients presenting with acute gastroenteritis who, prior to presentation, had consumed food privately imported from the Pacific Islands. Auckland Public Health Protection had also received case reports of acute gastroenteritis associated with consumption of seafood privately imported from the Pacific Islands. Prospective studies have not been performed to substantiate the significance of this food as a potential source of illness.

Methods

A prospective observational case series was performed at Middlemore Hospital. Cases were defined as those aged greater than fifteen years presenting to the ED from the 1st December 1999 until the 29th February 2000 with acute gastroenteritis and a history of consumption of food privately imported from the Pacific Islands. The clinical diagnosis of gastroenteritis was made if the patient reported diarrhoea (defined as loose or frequent motions).2

The triage nurse identified potential cases. ED doctors or nurses involved in the patients' care completed a standardised form recording patient characteristics including demographic details, symptoms, food source, food type and island of food origin. The following laboratory tests were requested: faecal samples, a full blood count, electrolytes, and liver function tests. The fecal specimens were cultured on selective media for VPH gastroenteritis. This allowed a calculation of the proportion of cases having a history of consumption of food privately imported from the Pacific Islands.

Results

Analysis of the PIMS database of emergency department presentations identified 358 patients coded with the gastroenteritis (GGAST) diagnostic category over the 3-month study period. 34 (9.5%) of the 358 patients were identified as cases having a history of consumption of food privately imported from the Pacific Islands. Of the 34 cases, 25 (74%) were female. Cases’ age ranged from 16-77 years with a median of 37 years. Thirteen (38%) cases were aged between fifteen and 30 years, sixteen (47%) were between 30 and 50 years and five (14%) were older than 50 years. Twenty-four (70%) of the cases identified themselves as food privately imported from the Pacific Islands.

being of Tongan ethnicity, nine (26%) as Samoan and the remaining patient as a Cook Island Maori. 33 cases (97%) reported both diarrhoea and abdominal pain. Vomiting occurred in 23 (67%) of cases. The source in 32 (94%) cases was food privately imported by a relative or friend with only two (6%) cases consuming food they had personally imported. The food had been imported from Tonga in 23 (68%) cases, from Samoa in ten (29%) cases and Niue was reported as the source in one case (3%). The food type was universally seafood, being shellfish in 28 (82%) of cases. Of the remaining six (18%) cases, two had consumed jellyfish, two fish intestine and one patient each had consumed seaweed and sea slug.

Fourteen (41%) of the 34 patients submitted at least one stool sample for culture, of which all were positive for VPH: no other pathogens were identified. Full blood counts, electrolytes and liver function tests were taken from 31 patients. All had an elevated white cell count ranging from 11.4 x 10⁹/L to 26.0 x 10⁹/L (normal 4.0 - 11.0 x 10⁹). Five of the 31 cases had abnormal urea and electrolytes; two with mildly elevated urea of 7.8 mmol/L (normal 3.2-7.7 mmol/L), a further two with a plasma potassium of 3.1 mmol/L and one with a potassium of 6.1 mmol/L (normal 3.5-6.0 mmol/L). Three patients had isolated mildly elevated aspartate transaminase (AST) levels.

Five (15%) of the 34 cases in the study group were admitted to hospital. One was admitted to the ED Observation Ward for six hours. Four patients were admitted; three were pregnant and stayed overnight for observation and one was a 77 year-old female who had ongoing abdominal pain and diarrhoea.

In order to put the results of the stool analysis of the case series into perspective, the results of Middlemore Hospital Laboratory stool cultures for bacterial pathogens in 1999 were reviewed. Only 115 gastroenteritis patients (from 2249 samples) cultured for bacterial causes of diarrhoea were confirmed with a pathogen, of which eleven (9.2%) were positive for VPH. Over the three-month period of the study three cases of culture-proven VPH were identified by the laboratory outside the study group. A chart review indicated that all three patients had also reported consuming seafood imported from the Pacific Islands.

Inquiries were made of the Tongan Ministry of Health concerning the possibility of an outbreak of VPH gastroenteritis in Tonga during the study period. Tonga’s only hospital, Vaiola Hospital in Nuku’alofa, does not routinely culture stools for VPH. During the month of December 1999, a significant increase occurred in requests for stool cultures on gastroenteritis cases compared to the preceding or following months (18 versus 3 and 10 requests respectively; personal communication Dr Malaki ‘Ake, Chief Medical Officer, Public Health, Ministry of Health, Tonga, 14th September, 2001). It is difficult to know whether an increase in the level of VPH contamination of Tongan seafood had occurred and was reflected in the Auckland case series. No increase in gastroenteritis or in seafood-related illness was noted in Samoa (personal communication Dr Nualofa Potoi, Department of Health, Western Samoa, 30th August, 2001).

The disease notification database (EpiSurv) for the Auckland region was reviewed to identify sporadic cases or outbreaks (two or more linked cases) of VPH gastroenteritis between the years 1995 and 2000. In the Auckland population the rate of notified VPH gastroenteritis was 1.6/100 000/year and for those of Pacific Island ethnicity, 15.3/100 000/year. All notified cases were in Pacific Islanders and no outbreaks were identified where seafood originating in New Zealand had been consumed. The first reported outbreak of VPH gastroenteritis was documented in July 1999 with seven people ill and two hospitalised after consuming raw shellfish privately imported from Tonga. Over the period of this study, Auckland Public Health Protection investigated three outbreaks where VPH was confirmed on stool culture in at least one case from each outbreak. These outbreaks involved a total of sixteen cases with a median number of cases of five per outbreak (range 3-8). Patients were reluctant to give detailed information on the source of food during the follow up investigations. There has only been one report of indigenous VPH infection in the Auckland region – an outbreak of gastroenteritis associated with recreationally harvested mussels in Southeast Auckland in 1983 (personal communication, Dorothy-Jean McCoubrey, Ministry of Agriculture and Forestry, 2nd October 2001).

Discussion
This study supports a link between acute VPH gastroenteritis in Auckland and the consumption of privately imported seafood from the Pacific Islands. Given that only 16% of New Zealanders seek medical attention for foodborne illness, there were probably numerous cases of VPH gastroenteritis during the study period that were not identified through this case series at Middlemore Hospital.1

The true burden of acute VPH gastroenteritis in New Zealand is uncertain but there is likely to be a significant level of non-diagnosis and non-notification of cases. Only 25% of patients presenting with acute gastroenteritis to general practitioners in New Zealand collect stool samples for culture.1 In many community laboratories only selected samples are tested for *Vibrio* spp.. Moreover, medical practitioners are only encouraged to notify cases of acute gastroenteritis (under the Infectious and Notifiable Diseases Schedule of the Health Act 1956) if they are ‘high risk’ such as health care workers, childcare and food workers or are linked to a suspected common source.

VPH is a halophilic gram-negative bacillus, widely distributed and occurring naturally in the tropical marine environment.4 VPH has a pronounced seasonal variance, with higher numbers present in seawater during the warmer summer months. Shellfish are filter feeders and acquire the bacterium as part of their normal flora.5 The bacillus is seldom isolated from shellfish in water below 15°C but is consistently recovered in water exceeding 15°C.6 It withstands freezing but does not multiply at temperatures below 10°C.7 Symptoms usually commence within 4-30 hours of the ingestion of contaminated food and consist of watery diarrhoea, abdominal cramps, nausea, vomiting and fever.8 Illness is self-limiting and antibiotics are not indicated. Infection is not directly transmissible from person to person. VPH has long been recognised as an important cause of gastroenteritis after consumption of raw or undercooked shellfish or seafood.9 It can also cause wound infection and primary septicaemia particularly in the immunocompromised.10-11

All of the patients in this series had reported eating seafood prior to the onset of illness. This seafood was predominantly shellfish, imported on a 2-4 hour flight from the Pacific Islands usually as hand luggage in a jar containing salt water. This seafood is generally regarded as a delicacy and is usually purchased from the local markets in the Pacific Islands after commercial harvest. There are no existing data to estimate the frequency of consumption of privately imported seafood by Pacific Island people in New Zealand. Data collected during the 2001 Pacific Island Food Safety Campaign suggested a high level of exposure to seafood privately imported from the Pacific Islands (Fakalago P, Pacific Island home food safety radio campaign, evaluation report. Public Health Protection, Auckland District Health Board; 2001: unpublished), 56% (95% Confidence Interval 47-65%) of 124 Samoan, Tongan and Cook Island respondents aged fifteen years and over, reported consuming seafood brought back by their families and friends from the Pacific Islands in the preceding twelve months. Tonga was a prominent source (67% of cases) of implicated

New Zealand Medical Journal 24 May 2002
Looking to the future, Auckland Public Health Protection lobbied the Ministry of Health in 2000 to ban the private importation of seafood from the Pacific Islands. Discussions on tighter border controls or even a ban on the private importation of seafood from the Pacific Islands are being considered by the Ministries of Health, and Agriculture and Forestry (personal communication Mr Jim Wilson, Ministry of Health, 27 September 2001). Larger prospective studies in this area are required to aid policy information.

Acknowledgements. We are grateful to Dr Kim Yates, Emergency Physician at Middlemore Hospital and Dr Susan Taylor at Middlemore Microbiology Laboratory, Dr Maika Kinahoi-Veikune a member of the Pacific Integrated Care Team at Middlemore Hospital has provided valuable cultural assistance. We also thank the Emergency Department nurses, resident doctors, and medical records personnel.

Correspondence. Dr Vanessa Thornton. Middlemore Hospital Emergency Department, Private Bag, Auckland. Fax: (09) 270 9725; email: vthornton@middlemore.co.nz


INTERESTED IN THE BIGGER PICTURE?

A career in Public Health Medicine will give you an exceptional opportunity to improve health at a population level. Public Health Medicine will also offer you a wide variety of challenging and interesting roles in New Zealand and internationally.

As a Public Health Physician, you could choose to lead the development of health care strategy and policy, or you could choose to be involved in the design and implementation of public health programmes. Exciting careers are also possible in academic public health or in consultancy.

The career path is flexible, which will enable you to tailor work commitments to meet your specific interests.

Applications are invited to the Australasian Faculty of Public Health Medicine Training Programme, from New Zealand registered medical practitioners who wish to specialise in public health medicine. The Faculty places a high priority on training doctors of Maori or Pacific ethnic affiliation who want to work to improve the health of Maori or Pacific people respectively.

It is desirable that you have a high quality academic record and some clinical experience. You will also need the ability to think strategically, and to take a longer-term perspective on health issues. You should also have excellent written and verbal communication skills and analytical skills.

A tax-free grant of $38,000 per annum in the first year of training will be awarded to successful applicants. Positions will be available from 1 December 2002. Applications close on 30 June 2002.

For further information on the training programme contact us at: Australasian Faculty of Public Health Medicine New Zealand Office PO Box 10233, Wellington Website: www.afphm.org.nz Telephone: (04) 472 9183 Fax: (04) 472 9184 Email: afphm@afphm.org.nz

APPLICATIONS INVITED FOR PUBLIC HEALTH MEDICINE TRAINING PROGRAMME
Could laboratory-based notification improve the control of foodborne illness in New Zealand?

Greg Simmons, Public Health Physician; Robyn Whittaker, Public Health Medicine Registrar; Kerry Boyle, Data Analyst, Auckland Regional Public Health; Arthur J Morris, Clinical Microbiologist, Diagnostic Medlab and Auckland Hospital; Ario Upton, Microbiology Registrar, Middlemore Hospital; Lester Calder, Public Health Physician, Auckland Regional Public Health, Auckland.

Abstract

Aims. To estimate the completeness and timeliness of notifications of seven potentially foodborne diseases in Auckland.

Methods. The diseases audited were shigellosis, salmonellosis, campylobacteriosis, yersiniosis, listeriosis, hepatitis A and verocytotoxigenic (VTEC) _E. coli_ infections. Hospital and community laboratory-confirmed cases for the calendar year 2000 were audited against those notified to the Auckland Regional Public Health Service. Cases were matched on disease, name, date of birth, gender and National Health Index number.

Results. There were 3182 laboratory-confirmed cases of the seven diseases identified of which 77% had been notified to the Auckland Regional Public Health Service. The proportion of laboratory-confirmed cases notified ranged from a 65% for hepatitis A to 100% for VTEC infection. The median delay between laboratory confirmation and practitioner notification was two days. Notification of all laboratory-confirmed cases would have resulted in an estimated 145 additional investigations in the year 2000.

Conclusion. A change to laboratory-based notification could improve public health investigation and control of foodborne disease in New Zealand.

By international standards, New Zealand suffers high rates of illness due to foodborne enteric pathogens. An estimated 19,000 New Zealanders consult their general practitioners with foodborne gastroenteritis each year. Public health action to control foodborne illness is dependent upon complete and timely notification. This enables public health authorities to conduct effective surveillance (involving the systematic collection and use of epidemiological information) and control of transmission by follow-up of individual cases or outbreaks. Under the Health Act (1956), medical practitioners are required to report notifiable diseases to the Medical Officer of Health. However, only a small proportion of cases of foodborne illness present to a medical practitioner and a still smaller proportion undergo stool testing. In only a minority of these will a notifiable pathogen be identified, and of these, a variable proportion are actually notified. Therefore, notified cases represent the tip of the food borne disease ‘pyramid’.

In the UK one case is reported to national surveillance for every 1.4 laboratory identifications and 136 community cases. In contrast to New Zealand’s ‘passive’ surveillance system, which has remained essentially unchanged from the type first developed in England in the late 1880’s, many countries are now implementing ‘active’ foodborne illness surveillance systems. These involve the collection and integration of laboratory-based data, outbreak surveillance data and epidemiological studies aimed at documenting and monitoring each level of the foodborne diseases pyramid. This approach enables not only improved estimates of the prevalence of food borne illness but also enhanced outbreak investigation and the identification of sources and modes of transmission. This audit aimed to assess the completeness and timeliness of the notification system, by comparing notifications to laboratory-confirmed cases of seven selected foodborne diseases in Auckland.

Laboratory data. Laboratory-confirmed case data were confined to the 2000 calendar year. Data were collected from three major hospital laboratories, Auckland District Health Board (ADHB) Laboratory Services (LabPlus), North Shore Hospital Laboratory, Middlemore Hospital Laboratory and from Auckland’s only (at that time) community laboratory, Diagnostic Medlab, which services general practitioners and non-hospital accident and medical clinics. Anti-hepatitis A IgM positive test results were provided by Auckland Virology Laboratory (LabPlus), Diagnostic Medlab and Middlemore Hospital.

Data from the four sources differed in format, but included disease type, date of positive test, family name, first name, national health index number (NHI), date of birth, age and gender. LabPlus data did not include the date of positive test, therefore three days were added to the date of specimen arrival to derive an estimate of date of reporting a positive test. Two or more positive results for the same individual and disease within three months were considered to be repeated tests for the same illness. In these instances only the earliest date of a positive test was used.

Notification data. Data were extracted from EpiSurv, the computerised national notifiable diseases reporting system. All EpiSurv notifications for the seven diseases between 1/1/00 and 31/3/01 were included. This period was three months greater than that for laboratory-confirmed cases (1/1/00-31/12/00) and allowed for a delay in case notification of three months from laboratory confirmation. The fields extracted were disease type, date of notification, family name, first name, NHI number, date of birth, age and sex. Repeat notifications for the same individual and disease within three months were considered to be a duplicate notification and the earliest date of notification used.

Data matching. After discarding duplicates, the data sets were matched by family name, first name and NHI number. Notification and laboratory databases were checked manually for misspelt names and other errors to ensure no matches were missed. Notifications that did not appear on the laboratory databases were individually checked with each laboratory to determine whether they were laboratory-confirmed.

Notification delay. The delay in notification was calculated as the difference between the date of reporting the positive laboratory test and the date of notification. Differences greater than two weeks and negative differences (where notification was made before a positive result) were all manually checked against notification files and the date of confirmation by the laboratory. These data were then analysed using the statistical function of Microsoft Excel.

Permission to perform the audit was sought from the Auckland Ethics Committee. The chairperson considered this to be an audit not requiring formal ethics committee approval.

Results

Positive isolates were confirmed for 3182 cases of potentially food borne illness (excluding repeat specimens) in the twelve month period.
Source of notification. The majority of notifications were made by general practitioners (88.0%) followed by hospital practitioners (8.2%), hospital laboratories (2.4%), and the Institute of Environmental Science and Research (ESR) Laboratory (0.2%). A very small proportion were self-notified (0.3%), or from other sources (0.5%), and for 0.4% the source was unknown (Table 1). Almost all VTEC and listeriosis cases were notified by hospitals or the ESR Laboratory. Approximately half of the hepatitis A notifications were made by Auckland Virology Laboratory, under an arrangement with Auckland Regional Public Health Service (PHS) to send a copy of all positive results to a Medical Officer of Health. The majority of shigellosis (63%), campylobacteriosis (91%) and yersiniosis (90%) notifications were made by general practitioners.

Completeness of notification. For the food borne diseases audited, 77% of positive isolates were notified. Notification rates were highest for serious diseases with small numbers of cases, that is VTEC infection (100%) and listeriosis (90%). Hepatitis A had the lowest notification rate of 65%. The proportions of notified laboratory-confirmed food borne diseases are shown in Table 2. Notified cases of hepatitis A were younger than non-notified cases (mean age 23 years vs 42 years, p<0.001). Notified cases of yersiniosis were younger than non-notified cases (19 years vs 32 years, p<0.01). Otherwise there were no significant differences in age or gender between notified and non-notified cases.

Notification rates were compared for hospital practice (all hospital laboratory data) and community practice (all Diagnostic Medlab data including those hepatitis A tests requested by community practitioners) (Table 3). There was no significant difference between notification rates for community and hospital-based practitioners (χ² = 0.07, df=1, p = 0.798).

Notification delay. The median, 25th and 75th percentiles and maximum notification delays are shown in Table 2. The median delay in VTEC notification was two days, with two of the five cases being notified on the same day as the result was reported. The maximum delay in notification occurred ten days after a presumptive positive result, however this notification was made on the same day the definitive result from ESR was reported. The median delay in listeriosis notification was zero days, with five of the nine cases being notified on the day of the positive result, with a maximum delay of eight days.

The maximum delay of notification for shigellosis was 75 days. This was an outlier as the second longest delay was thirteen days. There were some very long delays in the notification of campylobacteriosis (66 to 224 days) where cases had multiple tests and were notified on a subsequent positive result.

Cases which would have been investigated if notified. All cases of VTEC infection, listeriosis, hepatitis A, shigellosis, and salmonellosis are investigated by the PHS to identify a source and to give advice in order to reduce transmission and prevent outbreaks. Table 2 shows the estimated number of cases who would have been investigated, had they been notified. This does not include sixteen positive Shigella and 26 positive Salmonella carriers identified during routine screening in refugees. These carriers were assumed to have been infected overseas and were screened while resident at the Mangere Refugee Centre. Cases of yersiniosis and campylobacteriosis are not routinely investigated unless they occur in ‘high-risk’ settings, including food handlers, childcare attendants and children aged less than five years and health-care workers. During the 2000 calendar year 5.7% of yersiniosis and 7.5% of campylobacteriosis notifications were investigated. If the proportions investigated were extrapolated to the non-notified cases, we estimate a further two cases of yersiniosis and 44 cases of campylobacteriosis would have been investigated, and for all seven diseases a total of 145 extra cases would have been investigated had notification been complete.

Discussion

An important level of non-notification of food borne illness exists in Auckland, particularly for highly infectious diseases such as hepatitis A infection, shigellosis and salmonellosis. The non-notification of 35% of the laboratory-confirmed hepatitis A cases in the year 2000 is of considerable public health importance, given that foodborne outbreaks are known to occur in New Zealand and that community outbreaks are not uncommon in Auckland. Secondary cases can be prevented with immunoglobulin. The relatively low rate of hepatitis A notification despite laboratory reporting was due to two reasons. Firstly, one Auckland laboratory did not routinely notify hepatitis A cases; secondly, the paper reports sent by the notifying laboratory were incompletely reconciled with the notification database due to human error. Similarly, the non-notification of 12% of laboratory-confirmed shigellosis and 18% of salmonellosis is likely to adversely affect public health, as all notified cases are investigated in order to identify outbreaks and prevent person to person transmission.

Compared to other studies of comparable notification systems outside New Zealand, the level of completeness of notification in Auckland is high. The proportion of laboratory-based cases notified for campylobacteriosis (76%), salmonellosis (82%) and shigellosis (88%) infections exceed those found in the UK (47% for salmonellosis and campylobacteriosis and 70% for shigellosis). This UK audit was of a ‘passive’ medical practitioner notification system.

<table>
<thead>
<tr>
<th>Source</th>
<th>VTEC infection</th>
<th>Listeriosis</th>
<th>Hepatitis A</th>
<th>Shigelliosis</th>
<th>Yersiniosis</th>
<th>Salmonellosis</th>
<th>Campylobacteriosis</th>
<th>Total (proportion)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR laboratory</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Hospital laboratory</td>
<td>2</td>
<td>2</td>
<td>17</td>
<td>10</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Hospital doctor</td>
<td>0</td>
<td>6</td>
<td>6</td>
<td>10</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>General practitioner</td>
<td>1</td>
<td>0</td>
<td>11</td>
<td>35</td>
<td>127</td>
<td>235</td>
<td>2035</td>
<td>2462 (88.0%)</td>
</tr>
<tr>
<td>School</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Self-notification</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Part of outbreak</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other Public Health</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Unit</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Unknown</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>5</td>
<td>9</td>
<td>35</td>
<td>56</td>
<td>141</td>
<td>316</td>
<td>2235</td>
<td>2797</td>
</tr>
</tbody>
</table>
A system for payment of a fee to medical practitioners for each case notified previously existed in New Zealand and consideration could be given to its reintroduction. A survey of South Island general practitioners found that 62% believed they should be paid for notifying a case with the fee of the order of $17.50.10 However, given the environment of fiscal constraint currently affecting Government health expenditure, it is unlikely that this option would be considered attractive to funders. Conversely, it has been shown that increasing an existing reimbursement may not have a positive effect in terms of improving the notification practices of clinicians.18 Other suggested strategies to improve notification include the delegation of notification duties to practice nurses (although the majority of notifications are currently made by practice nurses) and targeting less recently graduated medical practitioners to promote the importance of notification. Laboratory-based notification has been found to be more timely than medical practitioner reporting,17 although not by all researchers.19 Laboratory-based reporting is effective in improving completeness of notification, the quality of data and in reducing notification delay where information technology is applied in the form of electronic data transfer systems.20-21 Automated systems for electronic data transfer from laboratories are also attractive in that there is significant potential to reduce the total burden of data entry. Based on the difficulties experienced during this audit with respect to the variation in the types of laboratory and public health surveillance software, there is likely to be a considerable amount of work required in building an infrastructure for integrated electronic laboratory-based notification in New Zealand. The development costs of computer software and secure systems for data transfer to facilitate notification will be faced by both laboratories and public health services. The ongoing costs of maintaining systems for laboratory-based notification are likely to be significant. Ethical and privacy issues in adopting such a system include the need for secure networks for the transmission of personal information and careful protocols around communication between the laboratory, the practitioner, the patient and the public health service. In order to preserve the integrity of the practitioner-patient relationship the patient needs to be informed of their diagnosis by their doctor before the public health service contacts the case. A law change is needed to facilitate laboratory notification as there is still much uncertainty among laboratory management about the implications of the Privacy Act (1993) for laboratory reporting. Rule 11 of the Health Information Privacy Code (1994) permits the disclosure of health information “…to prevent or lessen a serious and imminent threat to public health…”12. The authors are not aware of any notifications consciously made by laboratories under this rule, even though illnesses such as VTEC infection and listeriosis warrant it. We consider that the Public Health

### Table 3. Comparison of the completeness of notification between hospital and community practitioners.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Community Practice</th>
<th>Hospital Practice</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of cases</td>
<td>Proportion notified (95% CI)</td>
</tr>
<tr>
<td>Campylobacteriosis</td>
<td>2298</td>
<td>76% (74-78)</td>
</tr>
<tr>
<td>Yersiniosis</td>
<td>149</td>
<td>81% (73-87)</td>
</tr>
<tr>
<td>Shigellosis</td>
<td>43</td>
<td>88% (75-96)</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>316</td>
<td>83% (79-87)</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>38</td>
<td>68% (51-82)</td>
</tr>
<tr>
<td>Total</td>
<td>2844</td>
<td>77% (76-79)</td>
</tr>
</tbody>
</table>

*Based on DML data plus positive hepatitis A results from LabPlus.

Early recognition and investigation of outbreaks of foodborne illness have been shown to have significant public health and economic benefit.11 Delay in the investigation of cases and recognition of outbreaks already exists by virtue of the interval between onset of illness and the case seeking medical attention, sample collection and laboratory confirmation. It was not the aim of this study to investigate pre-diagnostic delay. However, we found that the post-diagnostic delay in notification for most foodborne diseases was a median of two days. VTEC infection should be notified to the PHS as soon as possible owing to the seriousness of the disease and the need to rapidly identify and control outbreaks. The median delay of two days between laboratory confirmation and notification of VTEC gastroenteritis disadvantages urgent public health investigation. While four of the five VTEC cases were notified by laboratories, two of these were notified by the Enteric Reference Laboratory, Portia. These were notified six and ten days following presumptive positive hospital laboratory results. Therefore hospital laboratory-based notification would still have improved this delay.

In some regions public health services have already developed informal relationships to facilitate early and complete notification of laboratory-confirmed cases. Auckland is no exception, with the 80% of VTEC and 33% of listeriosis cases being notified by laboratory personnel. A range of strategies could be promoted to improve the proportion of laboratory-confirmed cases notified by medical practitioners. It has been suggested that feedback of preventative action taken as the result of notification may be the most effective way to improve notification.14 However, this information is already provided to medical practitioners in New Zealand using a number of regional and national bulletins.

*Estimated from the proportion of notified ‘high-risk’ cases investigated in 2000, 7.5% of 589 campylobacteriosis cases and 5.7% of 34 yersiniosis cases.

### Table 2. Notification completeness and delay.

<table>
<thead>
<tr>
<th>Notified Disease</th>
<th>Number of laboratory-confirmed cases (n)</th>
<th>Proportion of cases notified (95% confidence interval)</th>
<th>Median notification delay (25th, 75th Centiles, maximum)</th>
<th>Estimated number of uninvestigated cases (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VTEC Infection</td>
<td>2500</td>
<td>76% (75-78)</td>
<td>2 days (1, 3, 224)</td>
<td>44 (41, 47)</td>
</tr>
<tr>
<td>Shigellosis</td>
<td>76</td>
<td>88% (77-94)</td>
<td>2 days (0, 3, 75)</td>
<td>0</td>
</tr>
<tr>
<td>Yersiniosis</td>
<td>90</td>
<td>79% (72-85)</td>
<td>2 days (1, 4, 24)</td>
<td><em>2</em> (1, 3)</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>82% (77-85)</td>
<td>82% (51-77)</td>
<td>2 days (1, 4, 57)</td>
<td>71 (57, 87)</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>65% (51-77)</td>
<td>1 day (0, 3, 14)</td>
<td>1 day (0, 3, 14)</td>
<td>19 (12, 27)</td>
</tr>
<tr>
<td>Campylobacteriosis</td>
<td>76% (75-78)</td>
<td>2 days (1, 3, 224)</td>
<td>2 days (1, 3, 224)</td>
<td>44 (41, 47)</td>
</tr>
<tr>
<td>Listeriosis</td>
<td>90% (56-100%)</td>
<td>2 days (0, 2, 8)</td>
<td>0 days (0, 2, 8)</td>
<td>1 (0, 4)</td>
</tr>
<tr>
<td>Total</td>
<td>3182</td>
<td>77% (76-79%)</td>
<td>2 days (1, 4, 224)</td>
<td>143 (136, 155)</td>
</tr>
</tbody>
</table>

*Estimated from DML data plus positive hepatitis A results from LabPlus.

Table 2. Notification completeness and delay.
Bill currently being drafted should mandate laboratory-based notification in New Zealand while providing unequivocal protection to the notifiers. This will reduce the apparent confusion among the medical profession, laboratories and public health services concerning the application of the privacy legislation in New Zealand.

The introduction of laboratory-based notification would not obviate the need for medical practitioners to notify. The requirement for some diseases to be notified on suspicion is an important one, especially for VTEC infection, where syndromic presentation is common (haemolytic uraemic syndrome) and in the absence of a positive stool VTEC isolate the case may otherwise never come to the attention of public health authorities.

We consider that a change to laboratory-based notification could improve the public health investigation and control of foodborne illness in New Zealand.

Acknowledgements. We are grateful to the following people who supplied data: Dr Margaret Crosson, LabPlus, Auckland Hospital; Dr Sally Roberts and Mr Colin Swager, North Shore Hospital; Drs Susan Taylor and Selwyn Lang, Middlemore Hospital Microbiology Laboratory; Mr David Riley, Diagnostic Medlab. We thank Dr Myint Lin for assistance in setting up the audit.

Correspondence. Dr Greg Simmons, Auckland Public Health Protection, Private Bag 92 605, Symonds St, Auckland. Fax: (09) 630 7431, email: greg@adhb.govt.nz.

Fish offer clue to human fertility decline

The steady drop in male fertility in Britain could be caused by men ingesting female hormones in drinking water drawn from rivers containing recycled sewage, according to government researchers.

Extensive work for the Environment Agency shows that in some rivers from which drinking water is taken all the male fish of some species have become feminised. This is blamed on the presence of trace quantities of chemicals in the water.

According to Susan Jobling of Brunel University in west London, the same might be happening to the human population. Over the past 30 years human sperm counts have fallen by half as the birth pill has become increasingly used. Millions of contraceptive pills are taken every day and the synthetic oestrogen, known as ethanol oestradiol, is discharged and flushed into rivers, where it remains active for a month.

In some parts of London water is said to have passed through seven lots of kidneys before it reaches the sea. The Lea, which is a tributary of the Thames, would have no flow in the summer months but for discharges from sewage works.

“This issue is not just about fish,” Dr Jobling told the BBC. “Everything we eat, put on our skin, throw down the drain, ends up in the sewage treatment works and ultimately in the river. One could argue that we are actually living in a sea of chemicals. I think there are very real reasons to be worried about whether male reproductive health could also be affected in the same way as fish.”

The worst example cited in the research was the River Aire, which runs through Bradford and Leeds. All the roach caught in this river were female.

Alarm about chemicals affecting human health has been raised many times by environmental groups, and the first case of feminising of fish was discovered four years ago. The Environment Agency launched a comprehensive study, but doubted at the time that the findings would be significant. Now the agency is considering ordering water companies to remove the hormones.

Infant bed-sharing among Pacific families in New Zealand

Janis Paterson, Associate Professor, Co-Director, Pacific Islands Families: First Two Years of Life Study, Auckland University of Technology; Colin Tukuitonga, Pacific Health Research Centre, Department of Maori & Pacific Island Health, University of Auckland, Co-Director, Pacific Islands Families: First Two Years of Life Study; Samia Butler, Research Fellow, Pacific Islands Families: First Two Years of Life Study, Auckland University of Technology; Maynard Williams, Senior Research Fellow and Statistician, Faculty of Health Studies, Auckland University of Technology, Auckland.

Abstract

Aim. To describe infant bed-sharing among Pacific families in New Zealand.

Methods. The data were gathered as part of the Pacific Island Families: First Two Years of Life (PIF) Study in which 1376 mothers were interviewed when their infants were six-weeks-old. Maternal reports of infant bed-sharing practices were assessed by questions about infant sleep location and the number of people who usually shared a mattress with the infant.

Results. Over half of the mothers (54.9%) reported that their infants shared a mattress with other people, 44.2% sharing with one other person, the remainder sharing with two or more people. Of the bed-sharing infants, 4.7% slept on a mattress on top of the bed, and 4.7% only slept part of the night in the shared bed.

Conclusions. Together with effective information delivery, the educational and housing issues that many Pacific families in New Zealand face need to be addressed so that parents can make informed decisions about infant care practices.

Results

There has been a substantial reduction in the rate of Sudden Infant Death Syndrome (SIDS) mortality in New Zealand.1,2 However, there is concern that Pacific SIDS mortality rates may either be remaining constant or increasing.3,4 Three modifiable SIDS risk factors were initially identified: prone sleep position, lack of breastfeeding and maternal smoking.5 Further investigation identified bed-sharing as a potential SIDS risk factor,5,6 particularly if the mother is a smoker,5,7 and for infants under 4 months.8 There is ongoing discussion about the role of bed-sharing in SIDS7,8,9,10 as this infant care practice is common in many parts of the world where SIDS is rarely identified.11 In addition, there are reported physiological benefits associated with close contact between infants and caregivers.12,13 Conversely, other reports suggest that infant bed-sharing may present potential infant health risks such as accidental asphyxia,14,15 and hyperthermia.16,17,18

Ethnic differences have been found in several New Zealand studies with the prevalence of bed-sharing substantially higher for Pacific infants than infants of other ethnicities (Maori, Non-Maori/Non-Pacific).17,22,23 Infant bed-sharing has also been shown to be culturally diverse with many Pacific infants who share a bed with their parents sleeping in a raised position in a bassinet or carrycot on the parental mattress.24 Due to the concern about Pacific infant SIDS mortality in New Zealand and to clarify the nature of Pacific infant bed-sharing, questions were designed to examine the type and extent of bed sharing and the maternal and socio-demographic factors associated with this infant care practice.

Methods

Data were collected as part of the Pacific Islands Families: First Two Years of Life (PIF) Study. The PIF Study is a longitudinal investigation of a cohort of 1398 infants born at Middlemore Hospital, South Auckland during the year 2000. Middlemore Hospital has the largest number of Pacific births in New Zealand and is representative of the major Pacific ethnicities. All potential child participants were selected from live births at Middlemore Hospital where the child had at least one parent who identified as being of a Pacific Island ethnicity and also a New Zealand permanent resident. All procedures and interview protocols had ethical approval from the National Ethics Committee.

Approximately six-weeks after the birth of the child, Pacific interviewers, fluent in both English and a Pacific language, visited the mothers in their homes. Once eligibility criteria were confirmed and informed consent was gained, mothers participated in one-hour interviews concerning the health and development of the child and family functioning. This interview was carried out in the preferred language of the mother. Detailed information about the cohort and procedures is described elsewhere.19 Mothers were asked where the infant usually slept during the night, if the infant usually shared a mattress with anyone, and if so, how many people usually shared with the infant.

Table 1 lists the maternal and socio-demographic factors expected to influence infant bed-sharing. These were assessed by univariate and multivariate logistic regression procedures with adjustments for non-independence of twin data.

Results

The cohort was made up of 87.8% of all eligible Pacific births that occurred in the period 15 March to 17 December, 2000. Of the 1376 mothers (1.7% gave birth to twins), 47.2% self identified their major ethnic group as Samoan, 21% as Tongan, 16.9% as Cook Islands Maori, 4.3% as Niuean, 3.4% as Other Pacific (includes mothers identifying equally with two or more Pacific groups, equally with Pacific and Non-Pacific groups, or with Pacific groups other than Samoan, Tongan, Cook Island or Niuean), and 7.2% as Non-Pacific. The mean (SD) age of mothers was 27 (6.2) years, 80.5% were married or in defacto partnerships, 33% of mothers were New Zealand-born and 27.4% had post-school qualifications.

Over half of the mothers (54.9%) reported that their infants shared a mattress with other people, 44.2% sharing with one other person, the remainder sharing with two or more people. Of the bed-sharing infants, 4.7% slept on a mattress on top of the bed, and 4.7% slept part of the night in the shared bed.

Table 1 lists the variables examined for potential association with infant bed-sharing. For the categories within each variable the numbers and percentages of infants who shared a bed, along with the associated odds ratios of the variables are shown. Tongan ethnicity, Pacific birth place, lack of post-school qualifications, and having more than five children were significantly associated with infant bed-sharing (p<0.001). Living in overcrowded conditions, limited English fluency, living in a married relationship or being non-partnered, maternal reports of lack of SIDS awareness, and a
high level of alignment with the Pacific way of life and customs, or low levels of alignment with both Pacific and New Zealand way of life and customs were also significantly associated with infant bed-sharing (p<0.001). In addition, being in an older age group, not smoking (p<0.01), and exclusive breastfeeding (p<0.05) were associated with infant bed sharing. Household income did not reach significance in the univariate analyses.

To adjust for potential confounding effects all variables listed in Table 1 were entered into a multiple logistic regression model. When controlling for the effects of all other variables, factors which remained significantly associated (p<0.05) with infant bed-sharing were Tongan ethnicity, being Pacific-born, not being fluent in English, and reporting overcrowded living conditions. Table 2 presents adjusted odds ratios of variables attaining significance in the multiple logistic regression model.

Discussion

Ethnic differences in infant bed-sharing were found in several studies with bed-sharing being substantially higher for Pacific infants, which suggests that this is a strong cultural practice among Pacific families living in New Zealand. In a 1998 study, bed-sharing last night with three-month infants was 5.7% for Non-Maori/Non-Pacific infants, 20.2% for Maori infants, and 40.4% for Pacific infants. However, in that study it was also found that 51% of bed-sharing Pacific infants did not share the mattress with their parents but slept in a raised position in a bassinet or carrycot on the parental mattress. PIF findings show a higher overall

Table 1. Numbers (row percentages) and univariate odds ratios for infant bed-sharing by selected variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Category</th>
<th>Infant shares bed</th>
<th>Univariate odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>54 (48.6)</td>
<td>57 (51.4)</td>
<td>1.00 (0.71, 1.58)</td>
</tr>
<tr>
<td>20-29</td>
<td>362 (49.2)</td>
<td>366 (50.3)</td>
<td>1.06 (0.61, 1.11)</td>
</tr>
<tr>
<td>30-39</td>
<td>318 (62.7)</td>
<td>189 (37.3)</td>
<td>1.74 (1.15, 2.63)</td>
</tr>
<tr>
<td>40+</td>
<td>33 (71.7)</td>
<td>13 (28.3)</td>
<td>2.53 (1.20, 5.35)†</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Samoan</td>
<td>144 (52.3)</td>
<td>314 (47.7)</td>
<td>1.00</td>
</tr>
<tr>
<td>Cook Island Maori</td>
<td>110 (47.2)</td>
<td>123 (52.8)</td>
<td>0.82 (0.34, 1.00)</td>
</tr>
<tr>
<td>Niuean</td>
<td>23 (37.7)</td>
<td>38 (62.3)</td>
<td>0.58 (0.34, 0.61)†</td>
</tr>
<tr>
<td>Tongan</td>
<td>210 (78.2)</td>
<td>64 (21.8)</td>
<td>3.23 (2.15, 4.45)‡</td>
</tr>
<tr>
<td>Other Pacific</td>
<td>24 (51.1)</td>
<td>23 (48.9)</td>
<td>0.94 (0.30, 0.76)</td>
</tr>
<tr>
<td>Non Pacific</td>
<td>36 (36.0)</td>
<td>64 (64.0)</td>
<td>0.97 (0.30, 0.76)†</td>
</tr>
<tr>
<td>Social marital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partnered, legally married</td>
<td>472 (59.8)</td>
<td>317 (40.2)</td>
<td>1.00 (0.36, 0.61)‡</td>
</tr>
<tr>
<td>Status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partnered, defacto</td>
<td>135 (40.9)</td>
<td>195 (59.1)</td>
<td>0.47 (0.11, 0.87)</td>
</tr>
<tr>
<td>Non partnered</td>
<td>160 (58.6)</td>
<td>113 (41.4)</td>
<td>0.98 (0.34, 0.69)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post school qualification</td>
<td>183 (47.7)</td>
<td>201 (52.3)</td>
<td>1.00</td>
</tr>
<tr>
<td>Secondary school qualification</td>
<td>247 (52.7)</td>
<td>222 (47.3)</td>
<td>1.27 (0.96, 1.66)</td>
</tr>
<tr>
<td>English Fluency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>377 (41.7)</td>
<td>486 (56.3)</td>
<td>1.00 (2.82, 4.52)‡</td>
</tr>
<tr>
<td>No</td>
<td>390 (71.6)</td>
<td>140 (28.4)</td>
<td>3.57 (2.82, 4.52)‡</td>
</tr>
<tr>
<td>Parity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>186 (50.1)</td>
<td>185 (49.9)</td>
<td>1.00</td>
</tr>
<tr>
<td>2-4</td>
<td>412 (52.8)</td>
<td>368 (47.2)</td>
<td>1.13 (0.88, 1.44)</td>
</tr>
<tr>
<td>S</td>
<td>240 (71.7)</td>
<td>91 (28.3)</td>
<td>2.41 (1.69, 3.45)‡</td>
</tr>
<tr>
<td>Unknown</td>
<td>9 (47.4)</td>
<td>10 (52.6)</td>
<td>0.90 (0.36, 2.23)</td>
</tr>
<tr>
<td>Born in NZ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>160 (34.8)</td>
<td>300 (65.2)</td>
<td>1.00 (2.73, 4.45)‡</td>
</tr>
<tr>
<td>No</td>
<td>607 (65.1)</td>
<td>326 (34.9)</td>
<td>3.48 (2.73, 4.45)‡</td>
</tr>
<tr>
<td>Smoked yesterday</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>610 (58.7)</td>
<td>429 (41.3)</td>
<td>1.73 (1.35, 2.22)‡</td>
</tr>
<tr>
<td>Yes</td>
<td>156 (44.6)</td>
<td>194 (55.4)</td>
<td>1.00</td>
</tr>
<tr>
<td>Heard advice re SIDS prevention</td>
<td>305 (60.9)</td>
<td>196 (39.1)</td>
<td>1.44 (1.15, 1.80)†</td>
</tr>
<tr>
<td>Cultural Alignment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High NZ, Low Pacific Is</td>
<td>175 (59.6)</td>
<td>260 (40.4)</td>
<td>1.00 (2.78, 4.87)‡</td>
</tr>
<tr>
<td>Low NZ, High Pacific Is</td>
<td>318 (70.8)</td>
<td>131 (29.2)</td>
<td>3.68 (1.29, 2.45)‡</td>
</tr>
<tr>
<td>High NZ, High Pacific Is</td>
<td>126 (53.4)</td>
<td>110 (46.6)</td>
<td>1.78 (1.37, 2.58)‡</td>
</tr>
<tr>
<td>Low NZ, Low Pacific Is</td>
<td>140 (54.9)</td>
<td>115 (45.1)</td>
<td>1.88 (1.37, 2.58)‡</td>
</tr>
<tr>
<td>Fed baby first 6 weeks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast milk only</td>
<td>402 (58.9)</td>
<td>281 (41.1)</td>
<td>1.42 (1.02, 1.98)§</td>
</tr>
<tr>
<td>Combination breast milk &amp; formula</td>
<td>276 (52.1)</td>
<td>254 (47.9)</td>
<td>1.08 (0.77, 1.52)§</td>
</tr>
<tr>
<td>Formula only</td>
<td>89 (49.4)</td>
<td>91 (50.6)</td>
<td>1.00</td>
</tr>
<tr>
<td>Other variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Home overcrowded</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not at all</td>
<td>485 (50.4)</td>
<td>478 (49.6)</td>
<td>1.00 (1.32, 2.23)§</td>
</tr>
<tr>
<td>To some extent</td>
<td>202 (61.3)</td>
<td>117 (38.7)</td>
<td>1.71 (1.32, 2.23)§</td>
</tr>
<tr>
<td>A great deal</td>
<td>80 (72.1)</td>
<td>31 (27.9)</td>
<td>2.52 (1.63, 3.89)§</td>
</tr>
<tr>
<td>Household Income</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$10 000</td>
<td>57 (61.3)</td>
<td>33 (36.7)</td>
<td>0.79 (0.92, 1.45)§</td>
</tr>
<tr>
<td>$10 000-$20 000</td>
<td>221 (58.8)</td>
<td>155 (41.2)</td>
<td>1.42 (0.81, 2.44)§</td>
</tr>
<tr>
<td>$20 000-$30 000</td>
<td>249 (51.0)</td>
<td>204 (45.0)</td>
<td>1.23 (0.73, 2.10)§</td>
</tr>
<tr>
<td>$30 000-$40 000</td>
<td>129 (49.6)</td>
<td>131 (50.4)</td>
<td>0.98 (0.56, 1.70)§</td>
</tr>
<tr>
<td>$40 000-$50 000</td>
<td>37 (56.4)</td>
<td>34 (43.6)</td>
<td>1.23 (0.65, 2.32)§</td>
</tr>
<tr>
<td>&gt;$50 000</td>
<td>32 (50.8)</td>
<td>31 (49.2)</td>
<td>1.00</td>
</tr>
<tr>
<td>Unknown</td>
<td>22 (44.0)</td>
<td>28 (56.0)</td>
<td>0.78 (0.36, 1.66)§</td>
</tr>
</tbody>
</table>

*p<0.05; †p<0.01; ‡p<0.001.
rate of infant bed-sharing but a considerably lower rate (4.7%) of those infants reportedly sleeping on top of the bed. In addition, only 4.7% of these infants slept part of the night with another person. This is in contrast to earlier findings that 50% of infant bed-sharers slept less than two hours in their parents’ bed.9

Table 2. Adjusted odds of infant bed-sharing for variables attaining significance in a multiple logistic regression (n=1377).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Category</th>
<th>Adjusted odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Samoan</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Cook Island Maori</td>
<td>1.26</td>
<td>(0.86, 1.85)</td>
</tr>
<tr>
<td>Niuean</td>
<td>0.85</td>
<td>(0.46, 1.57)</td>
</tr>
<tr>
<td>Tongan</td>
<td>3.53</td>
<td>(2.44, 5.10)</td>
</tr>
<tr>
<td>Other Pacific§</td>
<td>1.76</td>
<td>(0.90, 3.45)</td>
</tr>
<tr>
<td>Non Pacific</td>
<td>1.42</td>
<td>(0.83, 2.44)</td>
</tr>
<tr>
<td>Born in NZ</td>
<td>Yes</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>1.86</td>
</tr>
<tr>
<td>English Fluency</td>
<td>Yes</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>1.76</td>
</tr>
<tr>
<td>Home overcrowded</td>
<td>Not at all</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>‘To some extent’</td>
<td>1.60</td>
</tr>
<tr>
<td></td>
<td>‘A great deal’</td>
<td>1.75</td>
</tr>
</tbody>
</table>

*p<0.05; †p<0.01; §§p<0.001. §Includes mothers identifying equally with two or more Pacific Island groups, equally with Pacific Island and non Pacific Island groups, or with Pacific Island groups other than Tongan, Samoan, Cook Island Maori or Niuean.

There has been considerable promotion through SIDS prevention programmes and the media in New Zealand about SIDS risk factors. However, information about the possible risks associated with bed-sharing has not been consistently delivered.27 These findings indicate that, despite efforts to inform people about this potential SIDS risk, a number of Pacific parents place their six-week old infants in a bed with other people. Further, the failure of differences in bed-sharing rates between non-smoking and smoking and exclusive breastfeeding and combination/non-breastfeeding mothers to reach significance in the multivariate model suggests that more emphasis needs to be placed on promoting the role of protective SIDS factors and the interaction of such factors with infant bed-sharing risk.

Ethnicity was associated with infant bed-sharing, with Tongan mothers reporting the highest rate (78.25%). The adjusted odds of Tongan infant bed-sharing was 3.5 times greater than that for Samoan infants which may be explained as a strong cultural infant care practice in this ethnic group. Compared to New Zealand-born mothers, their Pacific-born counterparts were significantly more likely to report infant bed-sharing. As there are no specific SIDS preventative programmes in the Pacific Islands nations Pacific-born mothers may be unaware that infant bed-sharing, particularly in some circumstances, may pose a health risk to their infants. In relation to this factor, mothers who reported not being fluent in English were significantly more likely to report infant bed-sharing.

The association of socioeconomic factors with SIDS26,27 demonstrates a need to address issues such as poverty and unemployment together with prevention efforts. The significant association between infant bed-sharing and maternal ratings of overcrowding suggests that housing problems among Pacific families may play an important role in infant care practices.

Other studies have shown that less educated mothers, mothers with more than five children and unemployed mothers are significantly more likely to practice infant bed-sharing.21 Although these factors only reached univariate significance in the PIF study data they indicate that a number of factors, other than culture, have an impact on infant bed-sharing.

Although only significant at the univariate level, mothers who reported no awareness of SIDS risk factors were more likely to put their infants in a bed-sharing situation. The delivery of information about infant bed-sharing and the protective and risk factors that interact with this practice needs to be tailored to specific Pacific groups. Together with effective information delivery, the educational and housing issues that many Pacific families in New Zealand face need to be addressed so that parents can make informed decisions about infant care practices.

Correspondence. Dr Janis Paterson, Faculty of Health Studies, Auckland University of Technology, Private Bag 92006, Auckland. Fax: (09) 917 9877; email: janis.paterson@aut.ac.nz

Auckland paediatric liver transplant experience 1990-2000

Janine Smith, Paediatric Registrar; Alison Wesley, Specialist Paediatrician; Simon Chin, Specialist Paediatrician, Department of Gastroenterology, Starship Hospital; Jane Harding, Professor of Neonatology, National Womens’ Hospital, Auckland.

Abstract

Aims. New Zealand is establishing its own Paediatric Liver Transplant Service. However there have been no readily available data on the experience of New Zealand paediatric transplant recipients to date. The aim of our study was to determine numbers and indications for transplantation at present, current outcomes and to estimate the likely demand for the service in the future.

Methods. A retrospective search of computerised records was performed on children cared for at Starship Hospital from 1990 to 2000.

Results. Seventeen children received eighteen transplants. The indication for transplantation was biliary atresia in the majority of patients (11/17, 65%). A higher proportion of Maori and Pacific Island children received transplants than would be expected from their proportion in the population (59 vs 29%, p<0.01). Significant and often multiple complications occurred post transplantation in the majority of children, but overall outcomes were good.

Conclusions. A New Zealand Paediatric Liver Transplant Program is likely to perform about six transplants per year.

The first paediatric liver transplant was performed in 1963 on a three year old with biliary atresia.1 With improved perioperative management, surgical techniques and immunosuppression resulting in improved survival,2,3 the indications for transplantation have expanded, and liver transplantation has become a treatment option for an increasing number of children.4

The first New Zealand child received a liver transplant in 1988. Since then most New Zealand children have been transplanted at the Queensland Liver Transplant Service, Brisbane. In 2000 the first paediatric liver transplant was performed in New Zealand and there are planned. However, there has been no registry of New Zealand paediatric recipients and therefore no readily available information on the likely demand for this service in New Zealand, or the workload implications. The aim of this study was to estimate likely demand, the indications for transplantation, complications and outcome. These data would be useful to evaluate any changes with the establishment of a New Zealand paediatric liver transplant service.

Methods

The Queensland Liver Transplant Service were asked to provide details of New Zealand children transplanted. Recipients cared for at Starship Hospital were identified from computerised records of inpatient, day stay and outpatient attendances from July 1990 to April 2000. Data not available from the medical record were sought from the Queensland Liver Transplant Unit database or the New Zealand Liver Transplant Trust.

Results

A total of 54 New Zealand children have been referred to the Queensland liver transplant service. 47 liver transplants were performed and eight patients died post transplantation. Eighteen of these 47 transplants (38%) in seventeen children came from the Auckland region. This report concerns the seventeen children identified from searching the medical records as receiving their post-transplant care at Starship Hospital. These thirteen boys and four girls received eighteen transplants. One child required a second transplant for autoimmune hepatitis. One child was transplanted in New Zealand and the remainder in Brisbane. The median age at transplant was 2.4 years, (range 0.8-15). The median length of followup was 3.7 years (range 0.2-12).

The main indication for liver transplantation was biliary atresia (11/17, 65%). Other indications included one each of: α1 antitrypsin deficiency, cryptogenic cirrhosis, hepatopulmonary syndrome, hepatoblastoma, non-syndromic bile duct paucity and fulminant hepatic failure.

Six Maori and four Pacific Island children received liver transplants. This is a significantly higher number than would be expected from the population, (59% versus 29% in the population under 15 years, p< 0.015). Eight of these children received a transplant for biliary atresia, one for neonatal hepatitis and one for hepatoblastoma. At the time of review, thirteen children were more than one year post transplantation and sixteen were still alive (94%). The one year survival rate for child and graft was 93%. One child died at the transplant center six months post transplantation from multiple complications including chronic rejection, chronic lung disease, prematurity and cerebral atrophy.

Synthetic liver dysfunction and jaundice occurred in fifteen children prior to transplantation. The two children without synthetic liver dysfunction had hepatoblastoma and hepatopulmonary syndrome. Other complications of chronic liver disease included recurrent bleeding varices (3), cholangitis (8), ascites (5) and encephalopathy (1). Only five children had an uncomplicated post transplantation course (Table 1). Common complications included biliary strictures (intra and extra hepatic) (6), vascular complications (hepatic artery thromboses (2), hepatic artery stricture (1), portal vein (2) and inferior vena cava thrombosis (1)) and infection (cholangitis (7), bacterial sepsis (1), viral (7)). Cholangitis, infection, thrombosis, and biliary strictures frequently occurred in the same child. Complications occurring less frequently included gastrointestinal protein loss, recurrent duodenal ulcer, autoimmune hepatitis and short stature. Complications presumed secondary to immunosuppressive medications included decreased glomerular filtration rate (7), renal impairment (3) and hypertension (3). Two children require anti-hypertensive medication. All children of school age were participating in a full time school program.

Discussion

This study provides the first collated information on the likely service demands for a New Zealand Paediatric liver transplant
service. Children in our study represent approximately one third of New Zealand paediatric liver transplants. Therefore a New Zealand service could expect to perform liver transplants on up to six children annually. Families transplanted in Queensland moved to Australia for 14 - 71 weeks (median 24 weeks). With transplants performed in New Zealand that time should be shorter and less disruptive.

This study may have underestimated the total number of New Zealand paediatric liver transplant recipients. The majority of transplants occurred in Brisbane, but we cannot exclude the possibility that individual health boards in New Zealand referred to other centres. Children cared for in Auckland may also not be representative of those elsewhere in the county. Although this was a retrospective review of medical records, it is unlikely that we missed significant complications. It is also unlikely that we missed earlier cases because we could not search computerised records prior to 1990, since outpatient records allowed us to detect the first New Zealand child to receive a liver transplant in 1988.

Biliary atresia was the indication for transplantation in the majority of our children (65%). This is consistent with the indications reported elsewhere. However, there was an excess of Maori or Pacific Island children amongst Auckland paediatric transplant recipients. This over-representation has previously been reported in patients with biliary atresia (58%), and eight of the ten Maori and Pacific Island patients in our review had biliary atresia. We speculate that this may lead to a higher number of transplants in New Zealand than might be predicted on a population basis.

In this study survival one year post transplantation was 93% for child and graft. This is similar to one year survival rates in larger series (now approach 90%) with four to eight year survival rates of 70-85%. We were concerned that managing transplant recipients in a setting geographically isolated from existing transplant recipients will be made to Starship Hospital, resulting in an increase in workload out of proportion to the number of transplants performed. This study provides baseline data for ongoing monitoring of the New Zealand Paediatric Liver Transplant Program.

Presented as part of The Paediatric Society of New Zealand, 53rd Annual Scientific Meeting, Napier, New Zealand, 30th November 2000.

Correspondence. Jane Harding, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 373 7497; email: j.harding@auckland.ac.nz

HA = hepatic artery, PV = portal vein, IVC = inferior vena cava, EBV = Epstein-Barr virus, HHV6 = Human herpes virus 6, CMV = cytomegalovirus.
Can cancer centres in New Zealand help the Cancer Registry generate survival data? A pilot study in prostate cancer

TKA Evans, Radiation Oncology Registrar; DS Lamb, Head of Radiation Service; DA Cornes, Clinical Trials Coordinator, Wellington Cancer Centre; J Fraser, Chief Analyst, New Zealand Health Information Service; CA Johnson, Radiation Oncologist; DA Hamilton, Radiation Oncologist, Wellington Cancer Centre, Wellington Hospital, Wellington.

Abstract

Aims. One of the current limitations of reports issued by the New Zealand Cancer Registry (NZCR) is that the only measure of the success of treatment is provided by the mortality ratio. A pilot study was therefore carried out to see if collaboration between cancer centres and the NZCR might allow the generation of more meaningful survival data that could be used for the audit of treatment outcome. Methods. Clinical details of patients seen at the Wellington Cancer Centre (WCC), in whom a diagnosis of prostate cancer was made in 1997, were provided to the NZCR. These details were matched with registration and mortality data held by the NZCR. Results. WCC records identified 82 patients who were diagnosed with prostate cancer in 1997. Of these, the NZCR registered 60 (73%) in 1997, 3 (4%) prior to 1997, and 14 (17%) after 1997. Five patients (6%) were not registered at all. In the cohort of 82 patients, 17 (21%) had subsequently died. Of these, 11 (65%) had been treated with palliative intent, and six (35%) with radical intent. Of those patients treated radically, three had died of prostate cancer and three of other causes. Conclusions. Cooperation between Cancer Centres and the NZCR would allow the NZCR to generate useful survival data. This could help evaluate the impact on survival of specific treatments and interventions, such as screening programs. Regional variations in outcome could be detected. The exercise is feasible, without compromising patient confidentiality.

The New Zealand Cancer Registry (NZCR) has collected data since 1948. However, in the early years the registration of patients was voluntary, so data were far from complete. A dramatic improvement occurred when the New Zealand Cancer Registry Act (1993) came into effect on 1 July 1994. This made it compulsory for any person in charge of a pathology laboratory to report a new case of malignant disease to the NZCR, and registration of new cases subsequently became much more comprehensive. Incidence data are currently added to mortality data taken from death certificates details, and the mortality ratio for all types of cancer is determined.

However, the mortality ratio is a poor measure of the effectiveness of treatment. This is because the ratio can fluctuate substantially from year to year, merely due to more or less patients being registered, and/or changes in the number of deaths because of a different mix of early and late stage cancer. In contrast, survival data on cohorts of patients can be used for national and international benchmarking of treatment outcome, for accurate assessment of the impact of screening programs, and for the development of an effective cancer control program.41

At the present, the NZCR does not collect the clinical information that is necessary to produce survival data. However, the Wellington Cancer Centre (WCC) routinely documents in the patient file details of the clinical staging, treatment given, and intent of treatment. This pilot study was performed to see whether it is possible to marry this clinical information with data held in the NZCR. If it is, then this flow of information could occur more widely throughout New Zealand, at least for specified cancers, and survival data could become a standard part of reporting by the NZCR.

Methods

Details on patients with a new diagnosis of prostate cancer seen at the WCC during 1997 were extracted from medical records. The year 1997 was selected to avoid problems with the interpretation of staging, as a new version of the TNM classification was introduced that year. In each case, the following were recorded: name, date of birth, National Hospital Index (NHI) number, date of diagnosis, TNM stage, Gleason grade and score, pre-treatment prostate specific antigen (PSA) level, treatment intent, and primary treatment modality.

This information was then forwarded to the NZCR, where it was matched with their data. Data from the NZCR comes from four main sources; histology reports, hospital generated information about inpatient discharges from the National Minimum Dataset, death certificates, and autopsy reports. These enable the following to be determined; date of pathological diagnosis, the ICD-O (morphology) code, the last known survival date (date of latest entry on the National Minimum Dataset), and the date and cause of death (if applicable). The combined data from the WCC and NZCR were then analysed to determine how many patients had died, and to define the tumour characteristics of the deceased patients. The NZCR also provided information on the number and age of patients registered with prostate cancer in 1997 who were not seen in the WCC during that year. The NZCR list of patients was compared with a list of patients seen at WCC in the years 1998-2000, to determine what proportion of patients registered in 1997 were seen at any time in the WCC.

Ethical approval for this pilot study was granted by the Wellington Ethics Committee, who were satisfied that patient confidentiality was protected.

Results

Survival. At the time of analysis (March 2001), 17 (21%) of the 82 WCC patients diagnosed in 1997 had subsequently died. Of those, 6 (35%) were treated with radical intent, and 11 (65%) with palliative intent. Deaths of patients treated radically were evenly divided between death from cancer and death from other causes.

Registration. Table 1 shows that 82 patients seen in the WCC had prostate cancer first diagnosed in 1997. Of these, 60 (73%) were registered with prostate cancer on the NZCR in 1997. Fourteen (17%) were registered after 1997, and 5 (6%) were not registered at all. Three (4%) were incorrectly recorded on the register as being diagnosed with invasive prostate cancer prior to 1997.
The impact of the terrorist attacks on the United States on 11 September 2001 has been felt around the world. For New Zealand it probably had impacts on psychological stress and significant economic impacts eg, arising from a decline in tourist numbers and in global financial confidence. Furthermore, since these attacks have also been bioterrorist attacks within the United States associated with anthrax contaminated mail. As well as resulting in five deaths, these attacks have triggered numerous ‘white powder’ incidents around the world (including over 200 in New Zealand). This article considers in more detail the potential risks and impact on New Zealand of bioterrorist attacks in the Northern Hemisphere. It is based on Medline searches for literature on bioterrorism and for literature on preventable causes of death in New Zealand so as to put the threat of bioterrorism into a public health context.

The definition of terrorism used in this article is “the instrumental use or threatened use of violence by an organisation or individual against innocent civilian targets in furtherance of a political, religious, or ideological objective”.

Particular assumptions made included: (i) That direct terrorist attacks on New Zealand with bioweapons are extremely unlikely and that it is more relevant to consider the results of such attacks on a Northern Hemisphere country; (ii) That the terrorist use of bioweapons probably exceeds the risk of inter-state conflict involving such weapons (with the latter not being considered directly in this article).

**Risk assessment**

It has been suggested that the low cost of producing some bioweapons, and the ability of resulting diseases to spread in the population, are properties that might make such weapons

<table>
<thead>
<tr>
<th>Year of Registration</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1995</td>
<td>1</td>
</tr>
<tr>
<td>1996</td>
<td>2</td>
</tr>
<tr>
<td>1997</td>
<td>60</td>
</tr>
<tr>
<td>1998</td>
<td>10</td>
</tr>
<tr>
<td>1999</td>
<td>1</td>
</tr>
<tr>
<td>2000</td>
<td>2</td>
</tr>
<tr>
<td>2001</td>
<td>1</td>
</tr>
<tr>
<td>Not registered</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
</tr>
</tbody>
</table>
attractive to terrorists. Furthermore, terrorist groups could potentially obtain weaponised materials and scientific expertise from the bioweapons research and development programmes that probably exist in eighteen countries. Some of these countries may even support and sponsor terrorist organisations. Many thousands of scientists are thought to have been involved in the biological warfare programme of the former Soviet Union alone.

Terrorist use of bioweapons has already occurred, as with the anthrax-contaminated mail in the United States in 2001. Another attack in the United States was when a religious cult contaminated restaurant salad bars with salmonella in 1984, resulting in at least 751 cases of salmonellosis. A Working Group on Civilian Biodefense in the United States has conducted detailed reviews of the following potential bioweapons: anthrax, botulinum toxin, plague, smallpox and tularemia (Table 1). The only one of these diseases to occur naturally in New Zealand is botulism – with only one reported outbreak in the past two decades. Clostridium botulinum is the organism that produces this toxin and it is ubiquitous in soil. Livestock infection with anthrax has not been detected in New Zealand since 1954.

Well-resourced terrorist groups or ones sponsored by a state could conceivably genetically engineer new bioweapons. There have been claims that Russian scientists have developed antibiotic resistance in strains of tularemia,7 potentially obtain weaponised materials and scientific expertise from the bioweapons research and development programmes that probably exist in eighteen countries. Some of these countries may even support and sponsor terrorist organisations. Many thousands of scientists are thought to have been involved in the biological warfare programme of the former Soviet Union alone.7

Well-resourced terrorist groups or ones sponsored by a state could conceivably genetically engineer new bioweapons. There have been claims that Russian scientists have developed antibiotic resistance in strains of tularemia,7 potentially obtain weaponised materials and scientific expertise from the bioweapons research and development programmes that probably exist in eighteen countries. Some of these countries may even support and sponsor terrorist organisations. Many thousands of scientists are thought to have been involved in the biological warfare programme of the former Soviet Union alone.7

Terrorist use of bioweapons has already occurred, as with the anthrax-contaminated mail in the United States in 2001. Another attack in the United States was when a religious cult contaminated restaurant salad bars with salmonella in 1984, resulting in at least 751 cases of salmonellosis. A Working Group on Civilian Biodefense in the United States has conducted detailed reviews of the following potential bioweapons: anthrax, botulinum toxin, plague, smallpox and tularemia (Table 1). The only one of these diseases to occur naturally in New Zealand is botulism – with only one reported outbreak in the past two decades. Clostridium botulinum is the organism that produces this toxin and it is ubiquitous in soil. Livestock infection with anthrax has not been detected in New Zealand since 1954.

Despite these concerns about the use of bioweapons, terrorists are probably still likely to prefer conventional weapons since such weapons can be more precisely targeted (eg, as with the attacks on the World Trade Centre or the Federal Building in Oklahoma City, USA). There are also many difficulties in delivering bioweapons in a manner that could inflict mass casualties. For example, modification of crop

dusters to allow for the dispersal of bioweapons is apparently technically complex (at least to achieve a particle size capable of evading respiratory tract defences). It has also been noted that the scientists in the Japanese religious cult Aum Shinrikyo, tried for some years to master bioweapons and were still unsuccessful. Indeed, an estimated twelve attempts to release either anthrax or botulinum toxin all failed to produce any known casualties. Yet this cult included many scientists and had total resources estimated at over a billion US dollars.

Some analysts have considered that United States Government officials have had a tendency to exaggerate the threat of chemical and biological terrorism. Others have suggested this exaggerated risk perception has been reinforced by sensational reporting in the press and an obsessive fascination with catastrophic terrorism in movies and best-selling books.7

**Potential impact on New Zealand**

Bioweapons used in Northern Hemisphere that could spread to New Zealand include smallpox, plague and genetically engineered bioweapons.

**Smallpox.** A smallpox attack could occur if a terrorist organisation was supported by a state that held remaining (illegal) supplies of this agent. After such an attack, an infected person could travel to New Zealand prior to any symptoms developing (given the incubation period – Table 1). Furthermore, the disease could then spread from person-to-person within New Zealand. An exercise involving a simulated bioterrorist attack on the United States with smallpox has recently been conducted.11 In this simulation, smallpox spread to 25 other American states and to fifteen other countries within thirteen days. After three months and the depletion of vaccine stocks, there were three million cases, one million deaths and civil disorder in response to government failure to control the situation.

Another model also suggests that substantial disease spread is possible before control measures are enacted, with each

---

**Table 1. Key characteristics of potential biological weapon agents.**

<table>
<thead>
<tr>
<th>Biological agent</th>
<th>Rationale for consideration as a potential bioweapon</th>
<th>Has been weaponised at some time</th>
<th>Has been used as a weapon (since 1900)</th>
<th>Incubation period in humans</th>
<th>Person-to-person spread?</th>
<th>Vaccine commercially available</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smallpox*</td>
<td>Would spread widely and rapidly in a highly susceptible population, high fatality rate</td>
<td>Former Soviet Union1</td>
<td>No</td>
<td>12-14 days (range: 7-17 days)</td>
<td>Yes but supply is very limited &amp; current quality is problematic</td>
<td></td>
</tr>
<tr>
<td>Plague*</td>
<td>Availability around the world, capacity for mass production and aerosol dissemination, potential for secondary spread</td>
<td>Former Soviet Union1</td>
<td>By Japan (World War II)</td>
<td>1-6 days (from aerosol distribution)</td>
<td>Yes</td>
<td>Vaccine withdrawn from market in 1999</td>
</tr>
<tr>
<td>Anthrax*</td>
<td>Availability around the world, capacity for mass production and aerosol dissemination, high fatality rate</td>
<td>Russia,7 Iraq,9 &amp; formerly: the UK, US &amp; Japan</td>
<td>WWI, terrorist/s in 2001 (USA); Attempted by Aum Shinrikyo7</td>
<td>1-7 days (range up to 60 days)</td>
<td>No</td>
<td>Yes but supply is very limited</td>
</tr>
<tr>
<td>Botulism toxin**</td>
<td>Extreme potency and lethality; ease of production, transport and misuse; can contaminate food</td>
<td>Iraq,9 Soviet Union</td>
<td>Attempted by Aum Shinrikyo7</td>
<td>An intoxication; rapid onset of symptoms</td>
<td>No – an intoxication</td>
<td>Antitoxin &amp; toxoid available but are problematic</td>
</tr>
<tr>
<td>Tularemia**</td>
<td>Extreme infectivity, ease of dissemination, substantial capacity to cause illness and death</td>
<td>Previously by the US, Soviet Union</td>
<td>Possibly in World War II</td>
<td>3-5 days (range 1-14 days)</td>
<td>Not documented</td>
<td>Vaccine under review by the US FDA</td>
</tr>
</tbody>
</table>
case leading to between four and six cases of smallpox (but possibly as many as 10-12). Widespread panic has been associated with smallpox outbreaks in the past and such panic could be exacerbated if there were inadequate vaccine available or if health authorities were not perceived to be in control of the situation. One psychiatrist has described bioweapons as more like weapons of ‘mass hysteria’ rather than ‘mass destruction’. Mitigating factors in smallpox outbreaks in New Zealand would be as follows:

- The provision of warnings to health workers so that surveillance and prompt diagnosis of disease among exposed people arriving in New Zealand could be undertaken. As smallpox is not infectious until the rash appears, surveillance for the initial pre-rash symptoms would be desirable (ie, high fever, malaise, and prostration with headache and backache). Information to assist in distinguishing smallpox from chickenpox would be particularly critical.
- The prior immunisation of key health and emergency personnel with smallpox vaccine is another option. Also, if smallpox vaccine supplies were available then immunisation could be used for outbreak control (eg, for vaccinating incoming travellers or for ring vaccination). While New Zealand currently has no secure access to its own supplies of smallpox vaccine, there are limited supplies held by the WHO that could be used to vaccinate key personnel in countries with outbreaks. The model by Meltzer et al considered that a combined vaccination and quarantine campaign could stop a smallpox outbreak if a daily quarantine rate of 25% were achieved and vaccination reduced transmission by ≥ 33%,.*
- The use of population-based interventions such as short-term voluntary home curfew for anyone developing a fever and advice to call for urgent medical assessment if a rash developed, are options. Restrictions on assembly of groups or closure of public transportation systems in selected areas could also be considered. Different modelling work has suggested that quarantine alone could stop an outbreak in the United States in which 100 people were initially infected in a bioterrorist attack. However, such control would require a minimum daily removal rate by quarantine of 50% of those with overt symptoms.
- If imported cases arrived in the New Zealand summer, outbreaks would be less likely given that the survival of aerosolised virus is inversely proportional to both temperature and humidity (eg, disease outbreaks in the European summer used to be rare compared to winter and spring). The historical New Zealand experience with a number of smallpox outbreaks suggests that a mix of vaccination and population measures (limiting travel and school attendance by non-vaccinated individuals) were effective in controlling outbreaks. The largest outbreak was in 1912-1914 with a total of around 2100 cases and 55 deaths. Plague. Terrorist distribution of pneumonic plague is unlikely for various reasons including the technical difficulties of distributing the organism. Nevertheless, after a covert terrorist attack involving the aerosolised dissemination of plague, an infected person could travel from the Northern Hemisphere to New Zealand prior to any symptoms developing (given the incubation period – Table 1). If such a person then developed primary pneumonic plague, there would be some risk of secondary cases. But this risk is probably small given the rarity of pneumonic plague outbreaks despite an average of 1700 cases of plague being reported annually on average in the last 50 years. Exceptions do occur however, as in Madagascar in 1997 when one person transmitted pneumonic plague to eighteen other people. However, once a case of imported plague was diagnosed in New Zealand, antibiotic treatment of cases and basic infection control measures would probably rapidly contain any outbreak. Of the past four cases of pneumonic plague in New Zealand (between 1902 and 1911), none resulted in secondary cases.

Engineered bioweapons. The impact of such bioweapons can not be easily predicted. At worst they would spread rapidly from person-to-person, have high fatality rates, and would be resistant to vaccines and antibiotic therapy. This could mean that much of the population would be at risk of infection if there were delays in instituting population control measures (eg, voluntary home curfews, restrictions on assembly of groups, and closure of public transportation systems). However, the evidence to date is that genetically engineered organisms tend to struggle to survive in the environment or quickly lose their imbibed characteristics. Anthrax. Since anthrax does not spread from person-to-person, this disease is of little threat to New Zealand as an imported disease. Nevertheless, it is possible that ‘copy cat’ anthrax attacks involving contaminated mail will occur again in the Northern Hemisphere. A small risk for New Zealanders would therefore exist from contact with international mail that had been cross-contaminated with anthrax spores leaking from letters used in the attacks (assuming that small numbers of spores can cause infection). Comparisons of bioterrorism with other public health concerns

The threat posed by bioterrorism can be compared to the threat from pandemic influenza to New Zealand. Some work on influenza has indicated that “the probability of a future global influenza pandemic is very high within the next few decades and it is very likely to reach New Zealand”. It has been conservatively estimated that in the absence of large-scale public health interventions that 800 to 4100 New Zealanders are expected to die in the next global influenza pandemic (Table 2).
around the world in nearly 100 years is only equivalent to the number of deaths from smoking in New Zealand over ten days. Furthermore, New Zealand is still experiencing a meningococcal disease epidemic with 508 notified cases in the year 2000. The burden of campylobacteriosis is also remarkably high at 8163 notified cases in 2000.

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Deaths caused</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>All acts of terrorism in NZ in the last 100 years</td>
<td>2</td>
<td>Both due to conventional weapons: the Rainbow Warrior bombing (1985); the bombing of the Wellington Trades Hall Building (1984).</td>
</tr>
<tr>
<td>All acts of chemical/biological weapon terrorism in the world 1900- May 1999</td>
<td>123</td>
<td>The major events have involved chemical weapons eg, 19 deaths from pesticide in water (Philippines, 1987); and 21 deaths from poison gas (Turkish village, 1994).</td>
</tr>
<tr>
<td>Possible deaths in a future influenza pandemic in NZ</td>
<td>800–4100</td>
<td>Model results (see Table 2).</td>
</tr>
</tbody>
</table>

Discussion
Summary of risk and impact. Given the historical evidence and the characteristics of bioweapons, it appears unlikely that terrorists will use these weapons to produce mass casualties. However, low-level usage with small outbreaks may continue (such as with anthrax distribution via mail). The terrorist use of bioweapons will also continue to be a concern in a world where many countries have bioweapons programmes and with advances in genetic engineering. Similarly, some risk will persist while the forces which breed terrorism persist throughout much of the world — including unresolved conflicts, poverty, inequality and environmental degradation.

Although the risk of terrorists using smallpox is probably very unlikely, any such outbreak in the Northern Hemisphere would pose some risk of spreading to New Zealand with subsequent outbreaks in this country. Pneumonic plague could also spread to this country, but both smallpox and plague would probably be fairly controllable if cases were detected and reported early.

Limitations of this analysis. This analysis has only considered a limited range of the many possible bioweapons that could be used. Furthermore, there are major uncertainties concerning the potential development of future genetically engineered bioweapons. The future course of terrorist activity is itself very uncertain, along with the complex trade-offs that terrorists might make between using conventional weapons, bioweapons (against people or agriculture), chemical weapons, nuclear weapons and even cyber-attacks on the Internet.

Comparisons with other threats to public health. The risk and potential impact of pandemic influenza on New Zealand is likely to exceed that of terrorist use of bioweapons. Similarly, there are many preventable risk behaviours such as smoking and physical inactivity that cause thousands of premature deaths in New Zealand each year. This impact suggests that it would be unwise to divert significant public health resources away from these more major concerns.

Possible primary preventive measures. Governments could do more to actively address the root causes of terrorism by supporting conflict resolution in areas of tension such as the Middle East and supporting health development and sustainable development in countries suffering from high disease rates, poverty and environmental degradation. Most developed countries are relatively poor international aid donors, giving less than the United Nation's target of 0.7% of GDP and much less than that provided by Denmark (1.01% of GDP).

The New Zealand Government has been a strong supporter of the destruction of remaining smallpox stocks and has supported the Biological Weapons Convention. However, more work is required by governments to enhance international support for this Convention along with establishing stronger mechanisms to actively enforce it. Concerned citizens and non-government organisations also have key roles in encouraging all governments to progress these disarmament activities.

Measures to reduce impact. New Zealand has a range of options to reduce the potential impact of diseases resulting from the use of bioweapons elsewhere:

- **Public health infrastructure.** Improvements could be made to the public health infrastructure and particularly in strengthening disease surveillance and outbreak control systems. These are desirable anyway for controlling other disease outbreaks (such as food-borne disease outbreaks) and would assist with controlling a likely future influenza pandemic. Indeed, the Ministry of Health has already invested in various influenza pandemic planning activities (including the world's first simulation of a national pandemic response in early 2002).
- **Public health legislation.** There could be additional provisions made to new public health legislation to assist with controlling diseases such as smallpox.
- **Health professional education.** There could be improved health professional education concerning diseases arising from bioweapons (particularly to assist in the early detection of smallpox).
- **Vaccine stockpiles.** Consideration could be given to following forthcoming WHO recommendations on securing access to smallpox vaccine. A first step might be to stockpile enough vaccine to cover key health and emergency workers and to be able to conduct 'ring vaccination' around outbreaks.
- **Contingency planning.** There could be further refinement of contingency plans for enhanced border control in the event of the use of bioweapons (particularly for an outbreak of smallpox in the Northern Hemisphere). However, the significant limitations of quarantine measures need to be recognised along with other lessons from past preparedness efforts against bioterrorism.

Summary
Given the historical evidence, and the characteristics of biological weapons, it appears unlikely that terrorists will use these weapons to produce mass casualties. Yet this terrorist threat will continue to be a concern while many countries still have bioweapon programmes, with advances in genetic engineering, and while determinants of terrorism persist around the world (eg, unresolved conflicts, poverty, inequality and environmental degradation). Terrorist use of
smallpox, pneumonic plague and genetically engineered pathogens in the Northern Hemisphere could lead to imported cases reaching New Zealand and some risk of ongoing disease outbreaks. However, a range of disease control measures are available that could substantially limit the size of any resulting outbreaks. The risk of terrorist use of bioweapons needs to be considered in the context of the more important risk of pandemic influenza on New Zealand, the many thousands of preventable deaths in each year in this country (eg, from smoking and physical inactivity), and the current epidemic of meningococcal disease. Nevertheless, attention needs to be given to the primary prevention of terrorism and to preparatory measures that improve the country’s public health infrastructure.

Correspondence. Dr Nick Wilson, 367A Karori Road, Karori, Wellington. Fax: (04) 476 3646; email: nwilson@actrix.gen.nz

Disclaimer. The views in this article do not necessarily represent those of the authors’ employing organisations.

33. OECD. Development Assistance Committee (DAC) members, date of membership and their aid at a glance (February 2000). Internet: [http://www1.oecd.org/dac/html/aidatagl.htm].

MEDICOLEGAL DIARY

Enduring powers of attorney
Jonathan Coates, Senior Associate, Buddle Findlay, Wellington.


When a practitioner is confronted with a patient who is unable to make decisions in relation to his or her treatment options, either as a result of temporary or permanent incapacity, the practitioner must consider whether there is any other person who is available and entitled to consent on behalf of the patient for the procedure.1 Where the attorney does have authority to act, the practitioner will be able to satisfy him or herself that the attorney is authorised to make the particular decision.2 The donor may authorise the attorney to act on his or her behalf unless the donor is ‘mentally incapacitated’.

The Protection of Personal and Property Rights Act 1988 (“PPP Act”) allows a competent person (“the donor”) to appoint someone with an enduring power of attorney to have responsibility for decisions about the donor’s property or personal care and welfare. The donor may authorise the attorney to act on his or her behalf in relation to personal care and welfare issues either generally or for specific matters. By and large it is for the donor to set out what powers he or she wishes to delegate to the attorney. These powers must be set out in a document which formally appoints the attorney. The donor can include restrictions and conditions on the attorney’s authority. Where time allows, practitioners should ask to see the document appointing the attorney before relying on the attorney’s authority to make a decision on the patient’s behalf. In this way, the practitioner will be able to satisfy him or herself that the attorney is authorised to make the particular decision. There are a number of other matters practitioners should consider when confronted with a person with an enduring power of attorney:

1. The attorney will have no power to make decisions on the donor’s behalf unless the donor is ‘mentally incapable’. A patient is ‘mentally incapable’ if he or she lacks, wholly or partly, the capacity to understand the nature, and to foresee the consequences, of decisions relating to his or her personal care and welfare. Alternatively, a patient is ‘mentally incapable’ if he or she has the capacity to understand the nature, and to foresee the consequences, of decisions in respect of matters relating to his or her personal care and welfare, but wholly lacks the capacity to communicate decisions in respect of such matters.

2. Where the attorney does have authority to act, the decisions made by the attorney will have the same effect as if those decisions had been made by the patient.
3. The appointment of the attorney must be in writing, signed by the donor and witnessed by a person other than the attorney. The document must also be signed by the attorney and witnessed by a person other than the donor.

4. An enduring power of attorney cannot appoint more than one person to act as attorney.

5. The attorney must be 20 years of age or older at the time the document was signed.

6. There are limits to what the donor can authorise the attorney to do. These prohibitions include (but are not limited to):2
   (a) Refusing consent to any standard medical treatment or procedure intended to save the patient's life or to prevent serious damage to the patient's health; or
   (b) Consenting to the patient taking part in any medical experiment (except for the purpose of saving the patient's life or of preventing serious damage to the patient's health).

   The prohibition on refusing treatment intended to save the patient's life might appear to be exactly the type of decision a donor may want an attorney to make in the event that he or she becomes incompetent (eg turning off a life support system). Donors who are anxious to ensure their views on refusing life-sustaining treatment are respected should prepare an advance directive in addition to the enduring power of attorney.

7. If the practitioner is concerned about the attorney's decision, he/she should consider asking the Court to review the attorney's decision.

To what lengths should a practitioner go to find out whether an incompetent patient has appointed an attorney? The practitioner will be required to do what is reasonable in the circumstances. In an emergency, it may not be possible to take any steps to find out if there is an attorney. Where there is more time available, practitioners should ask those close to the incompetent patient if they are aware whether or not there is an enduring power of attorney. The steps required will very much depend on the exigencies of the situation. Like all medical decisions, the important thing will be for the practitioner to be able to justify the steps he or she has taken.

Correspondence. Jonathan Coates, Buddle Findlay, PO Box 2694, Wellington. email: jonathan.coates@buddlefindlay.com

2. See the Third Schedule Protection of Personal and Property Rights Act for a full list of the prohibitions.