

1. Editorial

The interface of human and animal health in New Zealand – a personal perspective

The term "one medicine" was coined 20 years ago to focus attention on the similarity between human and veterinary health interests.¹ Veterinary medicine plays an essential role in protecting and promoting public health, especially in the prevention and control of zoonotic diseases.² Zoonotic disease agents account for most emerging human pathogens and over half of known human pathogens.³ Currently both human and animal health authorities are concerned about pandemic influenza H1N1 worldwide, highly pathogenic avian influenza H5N1 in Asia, and Q fever in the Netherlands. Other examples include the transmissible spongiform encephalopathies and the burden of neglected zoonotic disease suffered by developing or socially disrupted populations. New Zealand has been spared from some of these threats but has, both in current times and historically, experienced significant zoonotic disease burdens.⁴ Past and present foodborne zoonoses of importance include salmonellosis and campylobacteriosis;⁵ direct zoonoses include hydatidosis, leptospirosis,⁶ and *Salmonella* Brandenburg and *Salmonella* Typhimurium DT 160.⁷

The challenges health professionals face now are likely to continue to expand in the future as a result of a changing world. These changes may include increased disruption of ecosystems by development, changes in climate, and further disruption of human populations by conflict. Increased co-operation between veterinary and human health communities will be necessary to meet future challenges in public health. How well is New Zealand prepared?

I believe we are well placed. Several institutions, including government, research institutes and academia, have created positions to strengthen our capabilities in controlling diseases in a changing world. It is not possible in this article to detail all these groups and individuals, however, I wish to give a few examples that are close to me. Nigel French, the professor of Food Safety and Veterinary Public Health at Massey University, has built strong links with health professionals across the country including those from organisations such as the New Zealand Food Safety Authority and the Institute of Environmental Science and Research (ESR).

Tui Shadbolt is the MidCentral Public Health Unit Health Protection Coordinator. She has recently completed her Master's in Veterinary Studies in veterinary public health, the first health professional working in human health to undertake this degree. Later in this publication she co-authors a report outlining a collaborative investigation into a *Salmonella* outbreak affecting both humans and cattle.

The Biosecurity arm of the Ministry of Agriculture and Forestry (MAFBNZ) has a broad mandate that acknowledges the links between the health of people, wildlife and production animals. Paul White joined MAFBNZ as Team Manager, Animal Surveillance in May 2009. Paul's previous position was in the Public Health Intelligence team of the Ministry of Health. MAFBNZ's Principal Advisor Human Health is Doug Lush, a public health physician with 20 years experience in communicable disease control. Erik Van Eyndhoven is Principal Advisor Conservation, and his work includes ensuring that systems are in place to adequately protect our wildlife from communicable disease.

Ange Bissielo, a former vet with Massey training in epidemiology and veterinary public health, has recently joined the staff of ESR at the National

Centre for Biosecurity and Infectious Disease, itself a major initiative to bring together animal and human health on to one site at Wallaceville. Here ESR, MAFBNZ, AgResearch, and ASureQuality come together to provide diagnostic and epidemiological services and research into zoonotic disease. Secondments have already occurred with MAF's Investigation and Diagnostic Centre providing assistance to ESR during the Influenza A (H1N1) pandemic response and ESR providing training for MAF staff in microbiology.

Since April 2009, I have been in a novel co-joint position instigated by ESR and Massey's Institute of Veterinary, Animal and Biomedical Sciences. Like Nigel French, my first training is as a veterinarian. Advising on antimicrobial use in dairy herds, counselling worried parents about ringworm in their child's puppy, and sensitively handling the euthanasia of a much loved pet is all veterinary public health. For me it took learning the common language of epidemiology to see the broader possibilities of my field more clearly.

This joint position between Massey and ESR that I now hold is about working at the human-animal interface, by providing veterinary and epidemiological expertise in a human health setting. This is best exemplified through our work in leptospirosis. Through close cooperation, Massey researchers, ESR staff and others are involved with a number of projects on leptospirosis, New Zealand's most common occupationally acquired infectious disease. These include serological studies of prevalence and incidence in meat workers and farmers. Another current research project is investigating the effect of climate change on infectious diseases of humans in New Zealand.

Working in a new field is challenging. Inspired by the other specialists I have encountered I want to learn about areas that pertain more to human than to animal health. These include the social determinants of health, ethnicity, measuring environmental exposures, and health inequality. Some veterinary health areas are irrelevant to the human sphere, such as disease freedom and barriers to trade. However there is much common ground, especially when we consider what we do jointly, as opposed to where we come from. Inspiration can be taken from Calvin Schwabe, who in 1984 stated that 'Improved human health is the sole among veterinary medicine's several benefits to society that arises from virtually all of veterinarians' diverse activities... There is now and always has been only one medicine', (cited by 2).

References

1. Zinsstag J, et al. 2005. Potential of cooperation between human and animal health to strengthen health systems. *Lancet* 366(9503): 2142-5.
2. Sargeant JM 2008. The influence of veterinary epidemiology on public health: past, present and future. *Prev Vet Med* 86(3-4): 250-9.
3. Taylor LH, Latham SM, and Woolhouse ME 2001. Risk factors for human disease emergence. *Philosophical Transactions of the Royal Society B: Biological Sciences* 356: 983-989.
4. Crump JA, Murdoch DR, and Baker MG 2001. Emerging Infectious Diseases in an Island Ecosystem: The New Zealand Perspective. *Emerging Infectious Diseases* 7(5): 767-72.
5. Baker MG, Sneyd E, and Wilson NA 2007. Is the major increase in notified campylobacteriosis in New Zealand real? *Epidemiol Infect* 135(1): 163-70.
6. Thornley CN, et al. 2002. Changing epidemiology of human leptospirosis in New Zealand. *Epidemiol Infect* 128(1): 29-36.
7. Thornley CN, et al. 2003. First incursion of *Salmonella* enterica serotype typhimurium DT160 into New Zealand. *Emerg Infect Dis* 9(4): 493-5.

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VACCINE PREVENTABLE DISEASE

Hepatitis B

- **Notifications:** 26 notifications in the quarter (2008, 9); 58 notifications over the last 12 months (2008, 50) giving a rate of 1.4 cases per 100,000 population (2008, 1.2); not a statistically significant increase
- **Comments:** there has been a statistically significant quarterly increase from the previous quarter (12 cases) and from the same quarter last year (9 cases). Cases were aged between 18 and 71 years, with no cases under the age of 16 years

Invasive Pneumococcal Disease

- **Notifications:** 258 notifications in the quarter
- **Comments:** Cases were aged between 9 days and 101 years, with 18 cases under the age of two years

2. Notifiable Disease Surveillance

The following is a summary of disease notifications for the July - September quarter of 2009 and cumulative notifications and rates calculated for a 12-month period (October 2008 - September 2009). For comparative purposes notification numbers and rates are presented in brackets for the same periods in the previous year. A robust method of constructing 95% confidence intervals is used to determine 'statistically significant differences' throughout this report unless otherwise stated [see Newcombe, R. G. and D. G. Altman. Proportions and their differences. In: *Statistics with Confidence*. 2000. BMJ Books. Bristol]. Data contained within this report are based on information recorded in EpiSurv by public health service staff up to 7 October 2009. As this information may be updated over time, these data should be regarded as provisional.

National surveillance data tables are available online (www.surv.esr.cri.nz).

- *Note:* Invasive pneumococcal disease became notifiable on 17 October 2008 therefore comparisons between quarters and 12-month rates are not valid

Measles

- *Notifications:* 215 notifications in the quarter (2008, 1); 258 notifications in the last 12 months (2008, 18) giving a rate of 6.0 cases per 100,000 population (2008, 0.4); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (13 cases) and from the same quarter last year (1 case); 107 notifications were laboratory confirmed

Menigococcal Disease

- *Notifications:* 63 notifications in the quarter (2008, 46); 137 notifications over the last 12 months (2008, 118) giving a rate of 3.2 per 100,000 population (2008, 2.8); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (23 cases) reflecting the expected seasonal peak of cases in winter/spring. Cases were distributed by age as follows: 9 (less than 1 year), 20 (1-4 years), 3 (5-9 years), 1 (10-14 years), 30 (15 years and over); 10 cases were the epidemic strain

Mumps

- *Notifications:* 26 notifications in the quarter (2008, 17); 62 notifications over the last 12 months (2008, 89) giving a rate of 1.5 cases per 100,000 population (2008, 2.1); a statistically significant decrease
- *Comments:* six notifications were laboratory confirmed

Pertussis

- *Notifications:* 347 notifications in the quarter (2008, 99); 1,182 notifications over the last 12 months (2008, 309) giving a rate of 27.7 cases per 100,000 population (2008, 7.2); a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the same quarter last year (99 cases)

INFECTIOUS RESPIRATORY DISEASES

Non Seasonal Influenza A (H1N1)

- *Notifications:* 2,422 notifications in the quarter
- *Comments:* cases were distributed by age as follows: 117 (less than 1 year), 160 (1-4 years), 340 (5-14 years), 570 (15-24 years), 708 (25-44 years), 437 (45-64 years), 74 (65 years and over) and for the remaining 16 cases age was unknown; 2,384 cases were laboratory confirmed
- *Note:* non seasonal influenza became notifiable on 29 April 2009 therefore comparisons between quarters and 12-month rates are not valid

Tuberculosis Disease

- *Notifications:* 97 notifications in the quarter (2008, 75); 318 notifications over the last 12 months (2008, 296) giving a rate of 7.4 per 100,000 population (2008, 6.9); not a statistically significant increase
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (69 cases); 69 notifications were laboratory confirmed; 95 were new cases and two were relapse or reactivation cases

BLOOD- AND TISSUE-BORNE INFECTIONS

Hepatitis C

- *Notifications:* 14 notifications in the quarter (2008, 7); 40 notifications over the last 12 months (2008, 23) giving a rate of 0.9 cases per 100,000 population (2008, 0.5); a statistically significant increase
- *Comments:* Notified cases were aged between 12 months and 76 years, with two cases under the age of 16 years

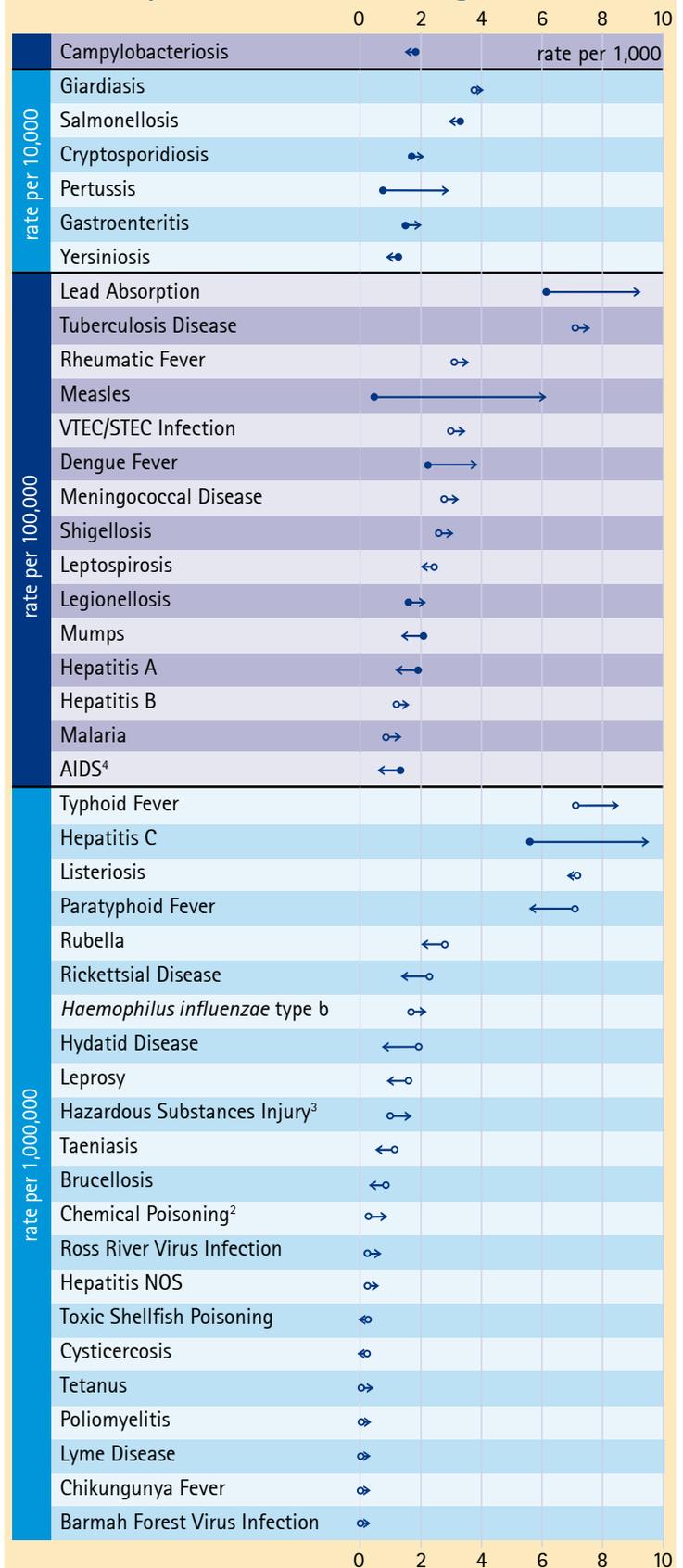
ENTERIC INFECTIONS

Campylobacteriosis

- *Notifications:* 1,548 notifications in the quarter (2008, 1,533); 7,036 notifications over the last 12 months (2008, 7,401) giving a rate of 164.8 cases per 100,000 population (2008, 173.4); a statistically significant decrease
- *Comments:* there has been a statistically significant quarterly increase from the previous quarter (1,245 cases)

National Surveillance Data

12-Monthly Notification Rate Changes⁽¹⁾



Notifications per 1,000 or 10,000 or 100,000 or 1,000,000 persons

Rate Change Symbol Key:

- Rate increase from the previous 12-month period
- Rate decrease from the previous 12-month period
- Statistically significant rate change
- Statistically non-significant rate change

¹ Rates are calculated for the 12-month period October 2008 – September 2009 and compared to previous 12-month rates

² From the environment

³ Hazardous Substance Injury became notifiable in EpiSurv as of 19 September 2007

⁴ Data provided by the AIDS Epidemiology Group, University of Otago. Note: changes in the 12 month notification rate should be interpreted with caution as this often reflects late notifications

Gastroenteritis

- **Notifications:** 247 notifications in the quarter (2008, 141); 794 notifications over the last 12 months (2008, 615) giving a rate of 18.6 cases per 100,000 population (2008, 14.4); a statistically significant increase
- **Comments:** there has been a statistically significant quarterly increase from the previous quarter (154 cases) and from the same quarter last year (141 cases). Note that this is not a notifiable disease per se except in persons with a suspected common source or with a high risk occupation, and the term 'gastroenteritis' provides a catch-all category for enteric diseases that are not notifiable and for syndromic reports that come through public health units, including direct reports from the public where the causative pathogen may never be known

Salmonellosis

- **Notifications:** 214 notifications in the quarter (2008, 231); 1,221 notifications over the last 12 months (2008, 1,358) giving a rate of 28.6 cases per 100,000 population (2008, 31.8); a statistically significant decrease

ENVIRONMENTAL EXPOSURES & INFECTIONS

Cryptosporidiosis

- **Notifications:** 234 notifications in the quarter (2008, 274); 846 notifications over the last 12 months (2008, 722) giving a rate of 19.8 cases per 100,000 population (2008, 16.9); a statistically significant increase
- **Comments:** there has been a statistically significant quarterly increase from the previous quarter (164 cases)

Hepatitis A

- **Notifications:** 14 notifications in the quarter (2008, 38); 52 notifications over the last 12 months (2008, 82) giving a rate of 1.2 cases per 100,000 population (2008, 1.9); a statistically significant decrease
- **Comments:** there has been a statistically significant quarterly decrease from the same quarter last year (38 cases). Cases were aged between 3 and 79 years, with five cases under the age of 16 years

Lead Absorption

- **Notifications:** 87 notifications in the quarter (2008, 98); 388 notifications over the last 12 months (2008, 257) giving a rate of 9.1 cases per 100,000 population (2008, 6.0); a statistically significant increase
- **Comments:** there has been a statistically significant quarterly decrease from the previous quarter (119 cases). Cases were distributed by age as follows: 1 (1-4 years), 9 (15-24 years), 33 (25-44 years), 39 (45-64 years), and 5 (65 years and over); 75 male cases, 8 female cases and 4 of unknown sex; 36 cases recorded an occupation that involved exposure to lead: painter (5), bricklayer (2), radiator fitter/repairer (2), builder (1), engineer (1), glass processing worker (1), policeman (1), scrap metal worker (1), and for 22 cases their occupation was not specified

Legionellosis

- **Notifications:** 15 notifications in the quarter (2008, 10); 89 notifications over the last 12 months (2008, 63) giving a rate of 2.1 cases per 100,000 population (2008, 1.5); a statistically significant increase

Leptospirosis

- **Notifications:** 18 notifications in the quarter (2008, 34); 87 notifications over the last 12 months (2008, 97) giving a rate of 2.0 cases per 100,000 population (2008, 2.3); not a statistically significant decrease
- **Comments:** there has been a statistically significant quarterly decrease from the same quarter last year (34 cases). There were 13 male cases, and 5 female cases; 7 farmers, 5 meat process workers, and grain mill worker, insurance agent, student (each 1 case), and for three cases occupation was not stated.

Yersiniosis

- **Notifications:** 88 notifications in the quarter (2008, 113); 434 notifications over the last 12 months (2008, 538) giving a rate of 10.2 per 100,000 population (2008, 12.6); a statistically significant decrease

NEW, EXOTIC & IMPORTED INFECTIONS

Dengue Fever

- **Notifications:** 15 notifications in the quarter (2008, 30); 162 notifications over the last 12 months (2008, 94) giving a rate of 3.8 cases per 100,000 population (2008, 2.2); a statistically significant increase
- **Comments:** there has been a statistically significant quarterly decrease from the previous quarter (45 cases) and from the same quarter last year (30 cases); all cases were laboratory confirmed; all cases were overseas during the incubation period and the places visited were Cook Islands (5), Thailand (4), Samoa (3), Fiji (1), New Caledonia (1), and Tonga (1).

3. Other Surveillance Reports

Antimicrobial susceptibility among invasive isolates

Streptococcus pneumoniae, *Neisseria meningitidis* and *Haemophilus influenzae* isolated from normally sterile sites are routinely referred to ESR for the national laboratory-based surveillance of invasive disease due to these organisms. The antimicrobial susceptibility of all viable invasive isolates of these three organisms referred in 2008 was tested. More detailed information is available on http://www.surv.esr.cri.nz/antimicrobial/antimicrobial_resistance.php.

Streptococcus pneumoniae

The previous penicillin susceptibility breakpoints for *S. pneumoniae*, which were universally applied to all pneumococcal infections, were predicated on the need to ensure successful treatment for pneumococcal meningitis. However, it had become clear that the outcomes for pneumococcal pneumonia caused by penicillin non-susceptible strains in patients treated with parenteral penicillin were no different to those in patients treated with other antibiotics. The application of the universal breakpoints meant that many pneumococcal infections outside the central nervous system were being unnecessarily treated with newer broad-spectrum and more expensive antibiotics when penicillin would have been effective.

Therefore in 2008, the Clinical and Laboratory Standards Institute (CLSI) published new interpretive standards for pneumococcal penicillin minimum inhibitory concentrations (MICs) and introduced different criteria for the parenteral treatment of meningitis, the parenteral treatment of non-meningitis infections, and the oral treatment of non-meningitis infections. Different cefotaxime MIC interpretive standards for meningitis and non-meningitis infections have been in place since 2002.

The antimicrobial susceptibility of 630 invasive *S. pneumoniae* isolates was tested in 2008. The penicillin and cefotaxime susceptibility of these isolates, interpreted according to each of the CLSI interpretive standards, is shown in Table 1.

Table 1. Penicillin and cefotaxime susceptibility among isolates (n=630) from invasive pneumococcal disease cases, 2008

CLSI interpretive standard ¹	Percent (number)		
	Susceptible	Intermediate	Resistant
penicillin			
parenteral treatment of meningitis	77.9 (491)	- ²	22.1 (139)
parenteral treatment of non-meningitis infections	99.5 (627)	0.5 (3)	0.0 (0)
oral treatment of infections	77.9 (491)	14.6 (92)	7.5 (47)
cefotaxime			
parenteral treatment of meningitis	84.9 (535)	10.0 (63)	5.1 (32)
parenteral treatment of non-meningitis infections	94.9 (598)	3.0 (19)	2.1 (13)

¹ The penicillin and cefotaxime MICs of all 630 isolates were interpreted according to each of the CLSI interpretive standards for these antibiotics

² There is no intermediate category for penicillin parenteral treatment of meningitis

The rates of resistance to other antibiotics in 2008 included 12% erythromycin resistance, 5% constitutive clindamycin resistance with another 0.5% inducible clindamycin resistance, 30% co-trimoxazole resistance and 8% tetracycline resistance. Seven percent of isolates had combined penicillin (meningitis interpretation) and erythromycin resistance. Among the penicillin-resistant isolates (meningitis interpretation), 27% (37/139) were multiresistant to ≥ 3 additional antibiotics, commonly co-trimoxazole, erythromycin and tetracycline with or without cefotaxime resistance. All isolates were susceptible to vancomycin and moxifloxacin.

Based on the meningitis interpretive standards, 90% of penicillin-resistant isolates and 94% of cefotaxime-resistant isolates were serotypes included in the 7-valent pneumococcal conjugate vaccine (PCV-7), which was added to the New Zealand childhood immunisation schedule on 1 June 2008. Among cases <2 years of age, all penicillin-resistant, cefotaxime-intermediate and cefotaxime-resistant isolates were serotypes included in PCV-7.

Neisseria meningitidis

The antimicrobial susceptibility of all 79 meningococcal isolates from cases of invasive disease in 2008 was tested. There was no resistance to penicillin, ceftriaxone, rifampicin or ciprofloxacin. Twenty-seven percent of isolates had reduced penicillin susceptibility, with MICs of 0.12–0.5 mg/L. Isolates with reduced penicillin susceptibility have been increasing over the last 10 years. However, meningococcal infections due to such isolates are still treatable with penicillin.

Haemophilus influenzae

The antimicrobial susceptibility of 64 invasive *H. influenzae* isolates was tested in 2008. Nine of the 64 isolates were serotype b. Seventeen percent of isolates produced β -lactamase and another 25% were ampicillin resistant but β -lactamase negative. There was no resistance to cefotaxime or rifampicin.

Reported by Helen Heffernan, Communicable Disease Programme, ESR

Exposure to lead-based paint at an Early Childhood Centre

In December 2008 lead-based paint in poor condition was identified on the exterior and interior walls of an Early Childhood Centre (ECC) in Wellington. Enquiries revealed that the paint had been flaking and peeling for at least four years. Flakes of paint were clearly visible on the ground in the children's outdoor play area. Results of interior dust samples found that on many surfaces lead was present in amounts exceeding 'safe' dust lead loadings as set by the US Environmental Protection Agency's (US EPA) Final Rule (2001) clearance standards.¹

The ECC is licensed to care for 22 children (including a maximum of four under two-year olds). All children attend full time. At the time of the investigation 21 children were enrolled comprising of a fairly evenly spread age distribution of two, three and four year olds together with two 'under twos'. The length of time the children had attended the centre ranged from a few weeks to four years with most having attended for at least one year. There were approximately equal numbers of males and females and the majority identified as being of Pacific ethnicity.

Children are at much higher risk of adverse health effects from lead than adults. Young children are especially vulnerable as their rapidly developing nervous systems are particularly sensitive to the effects of lead. In addition increased absorption of lead from the gut occurs in children compared with adults and there is increased likelihood that preschool children will place contaminated objects in their mouth.² Iron deficiency anaemia, which increases lead absorption, is relatively more prevalent in young Pacific children.³ Due to the vulnerability of the population at risk and their attendance characteristics, a decision was made to offer blood lead testing to all the children.

In total 18 children were tested. The results ranged from <0.1 $\mu\text{mol/L}$ to 0.23 $\mu\text{mol/L}$, all well below the notification threshold of 0.48 $\mu\text{mol/L}$. The blood lead levels did not appear to vary in any significant way with respect to ethnicity, age, gender or length of time that the child had attended the centre. Of note, due to the Christmas break and a subsequent move of the centre to temporary premises, the blood tests were taken five weeks after exposure to the lead paint had stopped. This is approximately equivalent to one half life of lead in the blood.⁴ However even allowing

for doubling of the results, no children would have had a blood lead level requiring notification.

This investigation provides useful information on the necessity to perform mass blood lead testing on children, following their exposure to a lead hazard. In this type of setting there can be significant parental pressure to undertake blood tests on children where there is much less risk than was the case at this ECC. Our results provide evidence that even in the presence of a clearly documented lead hazard the blood lead levels of children exposed may not become significantly raised. This may be useful information for those who find themselves in a position of managing risk communication to the public on similar hazards in the future.

References

1. USEPA. 2001. *Lead: Identification of Dangerous Levels of Lead: Final rule*. 40 CFR, Part 745, 5 January 2001.
2. Ministry of Health. 2007. *The Environmental Case Management of Lead-exposed Persons: Guidelines for Public Health Units*. Wellington: Ministry of Health.
3. Wilson C, Grant CC and Wall CR 1999. Iron deficiency anaemia and adverse dietary habits in hospitalised children. *New Zealand Medical Journal* 112(1089): 203–6.
4. Bull S 2007. Lead – Toxicological overview. HPA Compendium of chemical hazards; Health Protection Agency.

Reported by Sarah Gray, Public Health Registrar, Deborah Read, Medical Officer of Health, Annette Nesdale, Medical Officer of Health, Regional Public Health

4. Outbreak Surveillance

The following information is a summary of the outbreak trends for New Zealand, from data collected in the last quarter (July – September 2009). Comparisons are made to the previous quarter (April – June 2009), and to the same quarter in the previous year (July – September 2008). Note that the outbreak data in this section are notified to ESR by the Public Health Services.

General

- 194 outbreaks notified in this quarter (2,432 cases)
- 118 are 'final' reports (1,957 cases); 76 are 'interim' reports (475 cases) that have yet to be finalised and closed

All data following are pertaining to final reports only.

- 16.6 cases on average per outbreak, compared with 21.6 cases per outbreak in the previous quarter (13.6 cases per outbreak in the same quarter of last year)
- 66 hospitalisations: norovirus (52), *Salmonella* (8), *Neisseria meningitidis* (2), rotavirus (2), *Campylobacter* (1), and influenza A H1N1 (1)
- Six deaths: norovirus (5) and influenza A H1N1 (1)

Pathogens

- 46 norovirus outbreaks (1,188 cases) during this quarter
- 33 'gastroenteritis' outbreaks (486 cases)
- 7 rotavirus outbreaks (91 cases)
- 6 *Salmonella* outbreaks (29 cases)
- 5 *Cryptosporidium* outbreaks (14 cases)
- 5 influenza-like illness outbreaks (98 cases)
- 4 *Giardia* outbreaks (9 cases)
- 2 *Bordetella pertussis* outbreaks (7 cases)
- 2 *Campylobacter* outbreaks (5 cases)
- 2 influenza A H1N1 outbreaks (15 cases)
- 1 *Clostridium perfringens* outbreak (2 cases)
- 1 *Escherichia coli* O157 outbreak (3 cases)
- 1 measles outbreak (4 cases)
- 1 *N. meningitidis* outbreak (2 cases)
- 1 *Shigella* outbreak (2 cases)
- 1 *Yersinia* outbreak (2 cases)

Modes of Transmission

Note that reporting allows for multiple modes of transmission to be selected. In many instances no mode of transmission is selected for outbreaks notified to ESR, consequently, numbers may not add up to the total number of outbreaks reported.

- 94 person-to-person, from (non-sexual) contact with an infected person (including droplets): 41 norovirus (1,145 cases), 18 gastroenteritis (264 cases), 7 rotavirus (91 cases), 5 *Cryptosporidium* (14 cases), 5 influenza-like illness (98 cases), 4 *Giardia* (9 cases), 4 *Salmonella* (11 cases), 2 *B. pertussis* (7 cases), 2 *Campylobacter* (5 cases), 2 influenza A H1N1 (15 cases), 1 *E. coli* O157 (3 cases), 1 measles (4 cases), 1 *N. meningitidis* (2 cases), and 1 *Shigella* (2 cases)
- 25 foodborne, from consumption of contaminated food or drink (excluding water): 12 gastroenteritis (74 cases), 9 norovirus (98 cases), 1 *Campylobacter* (3 cases), 1 *C. perfringens* (2 cases), 1 *Salmonella* (2 cases), and 1 *Yersinia* (2 cases)
- 24 environmental, from contact with an environmental source (e.g. swimming): 13 norovirus (370 cases), 5 influenza-like illness (98 cases), 4 gastroenteritis (56 cases), 1 influenza A H1N1 (2 cases), and 1 *Salmonella* (2 cases)
- 5 waterborne, from consumption of contaminated drinking water: 2 *Cryptosporidium* (6 cases), 2 *Salmonella* (6 cases), and 1 *Giardia* (2 cases)
- 3 zoonotic: 1 *Campylobacter* (3 cases), 1 *Cryptosporidium* (3 cases), and 1 *Yersinia* (2 cases)
- 1 'other' mode of transmission: 1 *N. meningitidis* (2 cases) (via unidentified carrier)
- 9 'unknown' mode of transmission: 7 gastroenteritis (157 cases), 1 norovirus (2 cases), and 1 *Salmonella* (14 cases)

Circumstances of Exposure/Transmission

Common 'settings' where exposure/transmission occurred or contaminated food/beverage was prepared for consumption are identified below. Note that multiple settings can be selected and in many instances no settings are selected in outbreaks notified to ESR.

- 32 home: 8 gastroenteritis (30 cases), 4 *Cryptosporidium* (10 cases), 4 *Giardia* (9 cases), 4 norovirus (23 cases), 4 *Salmonella* (13 cases), 2 *B. pertussis* (7 cases), 1 *Campylobacter* (3 cases), 1 *E. coli* O157 (3 cases), 1 influenza A H1N1 (13 cases), 1 measles (4 cases), 1 rotavirus (3 cases), and 1 *Shigella* (2 cases)
- 45 rest home: 32 norovirus (1029 cases), 9 gastroenteritis (272 cases), 3 rotavirus (45 cases), and 1 *Campylobacter* (2 cases)
- 22 hospital (continuing care): 16 norovirus (499 cases) and 6 gastroenteritis (152 cases)
- 14 café: 8 gastroenteritis (31 cases), 4 norovirus (11 cases), 1 *C. perfringens* (2 cases), and 1 *Yersinia* (2 cases)
- 9 childcare: 4 influenza-like illness (61 cases), 3 rotavirus (43 cases), and 2 norovirus (29 cases)
- 7 takeaways: 5 gastroenteritis (14 cases) and 2 norovirus (13 cases)
- 4 workplace: 2 gastroenteritis (107 cases), 1 *B. pertussis* (5 cases), and 1 norovirus (17 cases)
- 3 hospital (acute care): 2 norovirus (48 cases) and 1 gastroenteritis (4 cases)
- 2 supermarket: 1 gastroenteritis (2 cases) and 1 norovirus (17 cases)
- 1 caterer: norovirus (17 cases)
- 1 hostel: gastroenteritis (11 cases)
- 1 school: influenza-like illness (37 cases)
- 1 tangi: influenza A H1N1 (13 cases)
- 6 'other setting': 1 *Cryptosporidium* (4 cases), 1 *E. coli* O157 (3 cases), 1 influenza A H1N1 (2 cases), 1 measles (4 cases), 1 *N. meningitidis* (2 cases), and 1 *Salmonella* (2 cases)
- 2 outbreaks with no setting selected: 1 gastroenteritis (4 cases) and 1 *Salmonella* (14 cases)

5. Outbreak Case Reports

Measles outbreak in Canterbury 2009

The first case of the current measles outbreak in Christchurch was an unimmunised 16-year old girl who was notified on 8th June 2009. She gave no history of travel. In the following three weeks, two 15 year old boys who attended Christchurch Boys High School were notified. Neither had been in contact with the index case. By 8 July another 10 students from the same school had been notified and the eventual scale of the outbreak was only just becoming apparent.

Case definitions were as per the *Communicable Disease Control Manual*.¹ However, the definition of a clinically compatible illness required modification to allow for undocumented "fever" and a maculopapular rash of unknown duration. This is because the temperature was often not recorded and patients presented at the onset of the rash.

Contact tracing was implemented for all cases but due to the time delay from onset of symptoms to notification, prophylaxis was not always possible. By 4 August 2009 there had been 96 cases notified. MMR was given as prophylaxis to unimmunised contacts within 72 hours of exposure (ring vaccination). Human Normal Immunoglobulin (IG) was initially given to unimmunised contacts between three and six days post exposure who did not receive MMR, pregnant contacts or those who were immune suppressed. It was subsequently limited to contacts who were pregnant or immune suppressed.

At this stage, because of the unsustainable workload (staff were also managing public health aspects of the pandemic response), General Practitioners assumed responsibility for management of cases and contacts. Practices were provided with comprehensive guidelines. In addition, a letter was sent to all schools and preschools via the Ministry of Health and Ministry of Education advising of contact management expectations within these environments, including exclusion of unimmunised children for 14 days after exposure to a case.

As the number of notified cases continued to increase, local changes were made to the National Immunisation Schedule. The MMR vaccinations previously given at 15 months (MMR1) and 4 years (MMR2) were brought forward to 12 and 13 months respectively. To make the change manageable for primary care, this younger age group was identified as the priority with all older children being recalled for either their first or second MMR later. On 7 September the Ministry of Health advised primary care to recall 13 to 20 year olds who had not had two MMR doses.

Amongst the problems encountered was the difficulty in determining the status of a case. Often a patient who met the case definition on clinical +/- epidemiological grounds had contradictory (negative) or no laboratory results. Reasons for this included early timing of testing with respect to disease onset, or the lack of serological testing (especially in young children). Another complicating factor was determining the significance of previous measles immunisation. This latter factor resulted in some cases having modified measles; being laboratory proven (after repeat serology) but failing to meet the clinical criteria.

By 8 October in the Canterbury District Health Board region there had been 126 confirmed and 43 probable measles cases including 17 who had been hospitalised. There were no deaths and no admissions to the intensive care unit. As a response to the request for improved immunisation coverage at least 9,000 children had been vaccinated with MMR since 5 August in Canterbury.

1. Ministry of Health. 1998. Communicable Disease Control Manual. Wellington: Ministry of Health.

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A collaborative investigation of a *Salmonella* Typhimurium phage type 156 outbreak

MidCentral Public Health Service (PHS) recognised a cluster of *Salmonella* cases during August 2009. Case 1 was a worker on a dairy farm (onset 30 July), and it was noted that a veterinarian had recently diagnosed salmonellosis in sick cows on the farm. Case 2, a 10 year old boy (onset 29 July), had stayed on the same farm during his incubation period.

He had no direct contact with cows but had consumed unpasteurised milk from the farm the day prior to illness onset. Case 3 (onset 4 August) was the owner of the farm. She had direct contact with sick calves and consumes unpasteurised milk on a daily basis. All three cases were from separate households on the same farm property.

Typing results of all three cases were positive for *Salmonella* Typhimurium phage type 156 (PT156). Subsequently, the younger sibling of Case 2 was identified as the same phage type (onset 11 August). He had stayed in the same house as Case 2 during his incubation period. He had no direct contact with cows and had not consumed unpasteurised milk. Family members living in the households of Cases 2 and 3 had also consumed unpasteurised milk during the same time period, but did not become unwell.

Existing relationships between MidCentral PHS and the Institute of Veterinary, Animal and Biomedical Sciences (IVABS) at Massey University were used to contact the veterinarian involved and to confirm that the animal specimens were the same phage type. The vet provided advice about sampling for potential ongoing sources of animal infection.

An investigation of the farm premises was undertaken on 19 August. The farm had experienced a higher rate of herd illness and death than usual (two thirds of deaths from June to August were attributed to salmonellosis), with the first deaths noted on 24 July. All calves and sick cows had been treated with antibiotics and herd vaccination had commenced on 28 July.

A number of environmental samples were tested. Feed and maize silage samples were negative for *Salmonella*. Unpasteurised milk and environmental samples from a farm bike and the feed pad were positive for *S. Typhimurium* PT156. Bore/Tank water samples were negative for faecal coliforms (including *Salmonella*) but a tap water sample did not meet the drinking water standards.¹ *Escherichia coli* was detected at 3.1 MPN/100ml. Advice had previously been given to treat the tank water and repair a defect in a tank lid.

Outbreaks of salmonellosis on dairy farms in New Zealand are sporadic. The size of this outbreak would have led to significant environmental contamination. Contamination of animal feed was excluded as a source. Cows can maintain a carrier status of *S. Typhimurium* and when under stress this can increase pathogen shedding as a source of contamination.²

In 2008, 4% of non-human *Salmonella* isolates typed by the Enteric Reference Laboratory were *S. Typhimurium* PT156, predominantly of bovine origin. This phage type accounted for around 9% of all typed *Salmonella* isolates from bovine sources.³ In the same year, *S. Typhimurium* PT156 accounted for only 5% of typed human *Salmonella* isolates.⁴ The most likely source of human infections with this phage type will be of bovine origin.

Although the source of the outbreak within this dairy herd was not determined, the human cases were a result of exposure to these infected animals. Case 4 is presumed to be the result of household contact with Case 2. Although unpasteurised milk consumption could be the source for two of the cases, there are other potential sources to consider. It is likely that there was significant environmental contamination around the farm that cases were also exposed to. Advice was provided around milk pasteurisation and reducing environmental contamination within farm households.

The investigation of this outbreak was a collaborative effort between animal and human health. The investigation of potential sources and control of the outbreak required veterinarian knowledge and action. Vets who identify outbreaks in animals can provide advice to reduce the exposure of people to the animals or their products. This can significantly reduce the human burden of disease.

References

1. Ministry of Health. 2008. *Drinking-water Standards for New Zealand 2005 (Revised 2008)*. Wellington: Ministry of Health.
2. Smith BP 1996. *Salmonellosis in ruminants*. In: Smith BP (ed). *Large Animal Internal Medicine, 2nd ed*. St Louis: Mosby-Year Book.
3. Non-human *Salmonella* isolates 2008 [cited 30 October 2009]. Available from: http://www.surv.esr.cri.nz/enteric_reference/nonhuman_salmonella.php?we_objectID=1950
4. Human *Salmonella* isolates 2008 [cited 30 October 2009]. Available from: http://www.surv.esr.cri.nz/PDF_surveillance/ERL/HumSalm/2008/HumSalm2008.pdf

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6. Laboratory Surveillance

Laboratory-based surveillance of legionellosis in New Zealand

Legionellosis is a notifiable disease resulting from the opportunistic infection by bacteria belonging to the genus *Legionella*. Infection with *Legionella* is an important cause of community-acquired pneumonia, occurring both sporadically and in outbreaks. *Legionellae* are widespread in aquatic and damp soil environments which are considered the natural habitats of these bacteria. Their ubiquitous presence in soils and aquatic environments makes it difficult to prevent their entry into engineered water reticulation systems. Under suitable conditions they can grow and proliferate to levels where the potential for transmission to humans is significant.

There are currently at least 50 different *Legionella* species identified comprising 70 distinct serogroups, of which 40 are known to be pathogenic to humans. The predominant species responsible for disease in New Zealand are *L. pneumophila* and *L. longbeachae*. Annually approximately 50% of cases are attributed to *L. pneumophila* serogroup 1 and 40% to *L. longbeachae* serogroup 1, although these figures fluctuate from year to year (Table 2). The remaining 10% of cases are attributed to other *Legionella* species. These commonly consist of *L. bozemanae*, *L. dumoffii*, *L. feeleii*, *L. gormanii*, *L. jordanis*, *L. micdadei*, and *L. sainthelehsi*.

The two most common clinical manifestations of legionellosis are Legionnaires' disease and Pontiac fever, with Legionnaires' disease cases being more prevalent because the illness results in medical intervention and the case numbers are captured in the disease surveillance statistics. Not all persons exposed to the bacterium will develop symptoms. This has been demonstrated by seroconversion without concomitant disease. The resulting circulating antibodies following infection are not protective and do not confer immunity.

Infections by *L. pneumophila* strains are commonly associated with exposure to a contaminated water source – either a domestic drinking water supply (usually un-chlorinated), or recreational water (usually a spa or swimming pool). In situations where aerosols of *Legionella*-contaminated water are generated, such as in cooling towers or humidifiers or vehicle washes, the potential for outbreaks is increased because of the increased numbers of people potentially exposed to the source.

Infections by *L. longbeachae* have long been associated with composts and potting mixes, so are common amongst gardeners. The mechanism of infection from this material is not fully understood, but is likely to be caused by the inhalation of aerosolised particles created when handling the material. Many other *Legionella* species have been cultured from composted material including *L. pneumophila*, *L. bozemanae*, *L. dumoffii*, *L. feeleii*, *L. gormanii*, *L. jordanis*, *L. micdadei*, and *L. sainthelehsi*.

The annual notification data shows there are between 50 and 100 laboratory confirmed cases each year, giving an annual rate of between 1.3 and 2.3 per 100,000 population with between one to five deaths reported each year. Most cases occur as sporadic isolated cases, either community-acquired, or travel-associated, and more rarely as nosocomial infections. Fortunately, from a public health view point, there is no evidence that *Legionella* can be spread person-to-person. The predominant predisposing factors that have been recognised are diabetes, cancer or blood disease, and immunosuppressant treatment.

Diagnostic Testing

A number of methods are currently available for the diagnosis of legionellosis. These are *Legionella* culture, detection of *Legionella*-specific antibodies in serum, urinary antigen detection, and the detection of *Legionella* nucleic acid by PCR. The sensitivity of diagnostic tests range from 60–70% and no one test exceeds 90% for specificity. No individual diagnostic test fulfils the requirements of clinicians, microbiologists and epidemiologists. For this reason the examination of different specimen types with different tests in parallel is strongly recommended. The occurrence of false-positive results particularly for serological and PCR tests demonstrate the value of routine confirmatory testing procedures. All clinical samples giving positive results for serology or PCR tests are required to be sent to the *Legionella* Reference Laboratory for confirmation.

Most cases of legionellosis in New Zealand are diagnosed serologically using indirect fluorescent antibody testing methods to detect a rise in *Legionella*-specific antibodies in serum. Serological testing does not have an impact on patient management because seroconversion occurs relatively late (usually within three to six weeks after the onset of symptoms, occasionally longer and sometime not at all) in the course of infection. There may also be the occasion where a serial bleed over a longer period than three weeks is required to demonstrate a seroconversion or a four-fold titre rise. Test sensitivity is increased using methods that detect both IgG and IgM antibody classes, since some studies have shown the immune response is primarily IgM. Serological testing is a retrospective test and serves as confirmation of suspected cases. Therefore there is a need for additional tests to diagnose legionellosis in the early stage of disease, such as the urinary antigen test (UAT) or molecular-based tests.

Culture is still considered the 'gold standard' method for the diagnosis of legionellosis even though it requires special media and the appropriate processing of clinical samples. *Legionella* do not colonise humans and cannot be isolated from healthy people. Culture is strongly encouraged by the Legionella Reference Laboratory as it enables matching of clinical isolates with any available from the environmental tracing exercise. However, its relative low sensitivity and the reliance on the availability of a lower respiratory tract sample make it inadequate as the sole diagnostic test. Culture yield usually depends on the severity of disease, with those with severe disease more likely to be culture-positive. This finding probably reflects the pathogen load in the specimen, with those with milder disease having lower numbers and therefore less likely to be culture-positive.

For effective surveillance of strains and disease there is a requirement for clinical samples from suspected cases to be sent to the Legionella Reference Laboratory for confirmatory testing. We would also encourage sending lower respiratory samples for both culture and molecular testing. These materials are invaluable for both source tracing and for enabling the genetic matching of strains with those present in New Zealand and for comparison with those from other countries.

Table 2: Comparison of predominant *Legionella* species causing disease and the method of diagnosis since 2004 in New Zealand

Legionellosis cases – lab confirmed by year		2004	2005	2006	2007	2008	2009 [†]
Legionella species	<i>L. pneumophila</i> serogroup 1	19	40	22	18	21	18
	<i>L. pneumophila</i> other than serogroup 1	9	8	8	11	4	7
	<i>L. longbeachae</i>	23	25	11	26	38	23
	All other <i>Legionella</i> species	23	11	12	17	11	15
	Total	75	84	54	72	74	63
Method of diagnosis	Culture	4	9	3	10	17	13
	Serology	65	48	40	57	39	38
	PCR supported by culture or serology	1	2	1	3	6	3
	PCR alone	3	2	2	0	2	0
	PCR supported by UAT	0	4	1	0	0	1
	UAT supported by culture or serology	1	2	5	1	5	0
	Urinary antigen test alone	1	17	1	1	5	8
	Total	75	84	54	72	74	63

†: Part year (until end of October 2009)

Reported by David Harte, Legionella Reference Laboratory, Communicable Disease Group, ESR

Laboratory-based surveillance of invasive *Listeria monocytogenes* in New Zealand

Listeriosis, which is primarily a food-borne infection, was made a notifiable disease in New Zealand in 1969, following an outbreak involving 13 cases in Auckland. Statistics since then have been based on notifications and the receipt of isolates from cases by the Communicable Disease Group at ESR in Porirua. It is believed that almost all diagnosed infections are reported to ESR through the laboratory-based system, because of the long-standing co-operative arrangement in existence with the referring laboratories. In addition, close co-operation with the Public Health Surveillance Group at ESR enables non-receipt of an isolate from a notified case to be promptly followed up.

On receipt in the Special Bacteriology laboratory at ESR, isolates of *L. monocytogenes* have their identity confirmed and O antigen serotype determined; the majority of New Zealand strains are either serotype O1/2 or serotype O4. Isolates are stored and DNA macrorestriction analysis by pulsed-field gel electrophoresis (PFGE) is done annually using test procedures based on the international PulseNet protocol.

PFGE is a molecular fingerprinting tool that generates a barcode-like fingerprint from each isolate, and enables comparison of strains to detect a possible common source. Isolates that are indistinguishable by PFGE analysis can then be followed up, without the distraction of unrelated cases.

If there is an apparent cluster or upsurge of cases during the year, PFGE typing can be done promptly at ESR on isolates from cases and from suspect food sources. This enables timely public health action to be taken, if required.

In 2008, isolates from 23 patients were received, from 6 perinatal and 17 non-perinatal cases. PFGE analysis of the isolates identified 4 groups of 5, 3, 3 and 2 indistinguishable strains. All the cases occurred in different locations and time, and no epidemiological evidence exists to indicate if they were linked.

So far this year (2009) the laboratory has been asked to do PFGE on two clusters of cases: 4 Auckland serotype O4 isolates in June/July which proved to be distinct (unrelated), and 6 serotype O4 isolates from Waikato, Auckland and Hutt in September where two groups of 2 isolates proved to be indistinguishable (possibly related). Suspect sources of the infections have not yet been determined.

Reported by Pat Short, Special Bacteriology Laboratory, Communicable Disease Group ESR

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