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Surveillance of antimicrobial resistance in New Zealand

Maggie Brett, Scientist, ESR; Rod Ellis-Pegler,* Infectious Disease Physician, Auckland Hospital

Antimicrobial resistance is now acknowledged as a serious public health issue not only in hospital settings but also in the community. Surveillance information on the prevalence and trends in antimicrobial resistance contributes to the formulation of local and national antimicrobial treatment guidelines and infection control policies, to the development of strategies to contain the emergence and spread of resistance, and to the measurement of the effectiveness of intervention strategies. Antimicrobial resistance data collected by the Institute of Environmental Science and Research (ESR) between 1988 and 1999 show an increase in antimicrobial resistance among many, but not all, clinical pathogens. In particular there were increases in the prevalence of methicillin-resistant *Staphylococcus aureus*, and *Streptococcus pneumoniae* resistant to penicillin and third-generation cephalosporins. The limitations of the current surveillance systems and suggestions for improving surveillance are discussed.

It took the medical profession until the last decade of the 20th century to finally acknowledge antimicrobial resistance as a serious public health issue. Just 60 years after the first parenteral use of benzylpenicillin, a nanosecond in evolutionary time, really worrying increases in antimicrobial resistance have been reported from all corners of the globe. No longer is it just the pathogens of the hospitalised and immune compromised, which we have struggled with for years; now it is increasingly the common bacterial pathogens of our communities, important contributing causes to the syndromes which general practitioners see daily.

These increases in resistance threaten our health on many fronts. For example, they threaten to varying degrees the generally easy management of conditions such as urinary tract infections, pneumonia, peritonitis and postpartum breast infections; they threaten less common but dramatic areas of antimicrobial triumph like meningitis, endocarditis and tuberculosis; they threaten all our surgical advances and in particular transplant surgery; and they threaten the increasing survival of our tiniest neonates and of those with malignancy, especially haematological malignancy. And all these examples are from the developed world only.

Data on the current prevalence and trends in antimicrobial resistance are needed to:

- formulate local and national antimicrobial treatment guidelines

- establish appropriate infection control strategies, particularly in the hospital environment
- implement strategies to avoid, delay and perhaps even reverse the emergence of resistance
- measure if intervention strategies are working.

Over the last 2-3 years, various international and national expert groups have considered and reported on the problem of increasing antimicrobial resistance.¹⁻³ In New Zealand, the Ministry of Health convened an Antimicrobial Resistance Working Group in 1997 to examine the issues facing New Zealand. All these groups agree that surveillance of antimicrobial resistance is a key element of any strategy to contain the emergence and spread of resistance. This article reports on the methods and results of national surveillance of antimicrobial resistance in New Zealand.

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*Correspondence: Dr Rod Ellis-Pegler, Infectious Disease Physician, Infectious Disease Unit, Auckland Hospital, Private Bag, Auckland. Email: rodep@ahsl.co.nz

Surveillance methods

In New Zealand, the national surveillance of antimicrobial resistance among human pathogens has been undertaken since the 1970s by what is now known as the Institute of Environmental Science and Research (ESR), on behalf of the Ministry of Health.

ESR uses four principal systems of antimicrobial resistance surveillance:

1 Collated clinical laboratory data: ESR collects and collates antimicrobial resistance data generated from routine diagnostic susceptibility testing in hospital and community laboratories. Currently about 20 clinical laboratories contribute data. Data for a large range of organisms are collected, for example, *Staphylococcus aureus*, *Escherichia coli*, *Streptococcus pneumoniae* and *Haemophilus influenzae*. The collated New Zealand data are forwarded to the World Health Organization (WHO) Antimicrobial Surveillance Programme for the Western Pacific Region, and the WHO collates and promulgates these data regionally.

In a similar way, ESR collects and collates antimicrobial resistance data for *Mycobacterium tuberculosis* from the three New Zealand reference mycobacteriology laboratories at Green Lane, Waikato and Wellington Hospitals. The laboratory data are matched with the tuberculosis (TB) notifications. The New Zealand data are submitted to the WHO for their global Antituberculous Drug Resistance Surveillance Programme.

2 Ongoing monitoring of *S pneumoniae*, *Neisseria meningitidis* and *H influenzae* from invasive disease: Isolates of these organisms from bacteraemias and sterile sites (invasive disease) are routinely referred to ESR for further epidemiological evaluation. This evaluation includes antimicrobial susceptibility testing. The *N meningitidis* isolates are matched with meningococcal disease notifications. This matching indicates there is full referral of isolates from culture-positive cases of meningococcal disease. As neither pneumococcal disease nor non-serotype b *H influenzae* invasive disease are notifiable, it is not known how complete or representative the referral of isolates from these diseases is.

3 Antibiotic-resistant bacteria monitoring scheme: Important antimicrobial-resistant bacteria, for example, multiresistant methicillin-resistant *S aureus* (MMRSA), *S pneumoniae* with reduced penicillin susceptibility, vancomycin-resistant Enterococci, and beta-lactamase producing *Neisseria gonorrhoeae*, are targeted for increased surveillance. ESR requests clinical laboratories to refer all isolates of these resistant organisms for further susceptibility testing and investigation. The antibiotic-resistant bacteria included in the scheme vary over time, and reflect emerging resistance and other resistance which warrants enhanced surveillance.

4 National point prevalence surveys: Periodic, usually annual, surveys of the prevalence of resistance among a specific organism using a sample of purpose-collected isolates. For these surveys, clinical laboratories are requested to refer to ESR all isolates of the selected pathogen for a specific period. The antimicrobial susceptibility of the referred isolates is tested at ESR. Since 1976, surveys of, for example, *S aureus*, *E coli*, *Enterococcus*, and *N gonorrhoeae* have been carried out. Surveys of a particular organism are often repeated after several years to monitor trends over time.

Since the late 1970s, ESR has followed the National Committee of Clinical Laboratory Standards (NCCLS) guidelines for interpreting susceptibility testing results.⁴ ESR has used predominantly agar dilution and, more recently, E-tests to determine minimum inhibitory concentrations (MICs).

A questionnaire completed by 32 clinical laboratories in 1999

indicated that 21 laboratories used the Kirby-Bauer diffusion method following NCCLS guidelines, five laboratories used commercial antimicrobial susceptibility systems, four laboratories used agar break-point methods, and two laboratories used the Stokes comparative disc diffusion method.⁵ All isolates of *M tuberculosis* were tested by a radiometric method (BACTEC) and results were interpreted according to the manufacturer's instructions.

Results and prevalence of resistance

National collation of clinical laboratory data began in 1988, and therefore the 12 year period 1988-1999 has been analysed for trends. The prevalence of resistance among common, important clinical pathogens, which have been continuously monitored from 1988, is presented in Table 1. The data are grouped into four three-year cumulative periods up to and inclusive of 1999. The data in Table 1 are derived principally from the first two surveillance systems described above. Where appropriate, data from the other two surveillance systems are also included in the following results section.

Staphylococcus aureus: In a 1999 national point prevalence survey, only 1.9% (11/583) of *S aureus* were methicillin resistant. In contrast, data collated from clinical laboratories for the same year indicated a resistance rate of 5.8%.⁶ This is an example of how two surveillance systems can indicate quite different rates of resistance. Notwithstanding this, based on the collated clinical laboratory data, there has been an inexorable increase in the prevalence of methicillin resistance over the 12 years to 1999. There is a large variation in the prevalence of methicillin-resistant *S aureus* (MRSA) in different parts of the country, with the highest rates in recent years being consistently recorded in the Auckland and Wellington areas.⁷

Among MRSA there has been a reduction in resistance to co-trimoxazole and also, but to a lesser extent, erythromycin. Therefore, in general practice co-trimoxazole is a useful alternative for the treatment of patients with moderately severe MRSA infections. These generally lower rates of resistance to non-beta lactam antimicrobial agents among MRSA since 1994 reflect the increasing prevalence through the middle and late 1990s of the nonmultiresistant strains, designated the WSPP (Western Samoan phage pattern) MRSA. However, since 1998 there has been a notable increase in the prevalence of multiresistant MRSA strains, in particular the British epidemic strain, EMRSA-15.⁸

There has been a dramatic increase in mupirocin resistance among *S aureus* since the early 1990s. Therefore, mupirocin susceptibility needs to be checked before it is prescribed. No vancomycin-intermediate or glycopeptide-intermediate *S aureus* (VISA or GISA)⁹ have been isolated in New Zealand yet.

Streptococcus pneumoniae: As in other parts of the world, there has been an increase in resistance, particularly to benzylpenicillin, among *S pneumoniae*. This increase in resistance is less marked among invasive isolates. The reduction in penicillin susceptibility, and accompanying changes in third-generation cephalosporin susceptibility, have driven changes in the recommendations for the antimicrobial management of pneumococcal meningitis.¹⁰ But these reductions in susceptibility have not led to widespread changes in the recommendations for antimicrobial treatment of other pneumococcal infections.¹¹ This is because the reduction in susceptibility is usually relatively minor and therefore only impacts on the success of therapy in sites of poor penicillin penetration, such as the cerebrospinal fluid and, albeit to a lesser extent, the middle ear. It does not usually impact on the success of therapy at sites where there is good penicillin penetration, such as the lungs, sinuses and blood stream.

Enterococcus: Unlike the international trends of increasing

vancomycin and amoxicillin resistance among enterococci,^{12,13} vancomycin-resistant enterococci (VRE) are very rare in New Zealand and amoxicillin-resistant enterococci are also uncommon. The first VRE was confirmed in 1996 and up to the end of 1999, VRE had only been isolated from a total of seven patients. The relative rarity of isolates of *E faecium* in New Zealand explains this in part, as this species is more commonly resistant than *E faecalis*.¹³

Escherichia coli: Resistance to amoxicillin/clavulanic acid among urinary *E coli* nearly doubled over the 12 years 1988-99. There was a smaller increase in trimethoprim resistance, and fluoroquinolone resistance emerged. Resistance to nitrofurantoin has changed little and remains very uncommon. This antibacterial could and should be used more often for community urinary tract infections.

Invasive isolates of *E coli* are predominantly sensitive to second-generation cephalosporins, so the use of third-generation drugs in suspected Gram negative septicaemia, where these organisms are such dominant contributors, is more fashion than science. *E coli*

remain doggedly susceptible to gentamicin.

Pseudomonas aeruginosa: *P aeruginosa* has long been regarded as presenting a challenge due to both intrinsic and acquired resistance. However, its resistance in New Zealand is generally strikingly stable. For example, only 2.8% tobramycin resistance was recorded in the latest three-year period, 1997-99. There does not appear to be a trend of increasing gentamicin resistance, and fluoroquinolone resistance has remained stable at around 8-10% throughout the 1990s.

Haemophilus influenzae: The susceptibility of non-invasive isolates is stable, with the exception of an increase in amoxicillin resistance during the last three years.

Since the introduction of *H influenzae* type b vaccination in 1994, there have been far fewer invasive isolates. Resistance to amoxicillin/clavulanic acid and cefuroxime among these isolates is very uncommon and these agents are appropriate and effective treatment for invasive *H influenzae* disease outside the central nervous system.

Table 1: Prevalence of antimicrobial resistance, 1988-99

| Pathogen | Antimicrobial | Percent resistance ¹ (number tested) | | | |
|---|--|---|--------------|--------------|---------------|
| | | 1988-1990 | 1991-1993 | 1994-1996 | 1997-1999 |
| <i>S aureus</i> ² | methicillin | 0.5 (37466) | 0.6 (42839) | 2.8 (58283) | 4.9 (136356) |
| | erythromycin | 8.1 (34900) | 6.8 (40425) | 8.0 (54870) | 10.8 (134350) |
| | co-trimoxazole | 1.0 (11783) | 1.1 (27469) | 0.8 (32926) | 0.6 (91391) |
| | mupirocin | NA ³ | 0 (16) | 10.1 (9291) | 18.2 (37173) |
| | Methicillin-resistant <i>S aureus</i> ⁴ | erythromycin | 52.9 (263) | 58.2 (701) | 31.5 (2249) |
| | co-trimoxazole | 24.3 (263) | 24.8 (701) | 8.6 (2249) | 1.8 (1303) |
| | mupirocin | 4.6 (263) | 2.0 (701) | 6.4 (2244) | 6.0 (1303) |
| | rifampicin | 1.1 (263) | 13.0 (701) | 0.3 (2249) | 0.8 (1303) |
| <i>S pneumoniae</i> , non-invasive disease ² | penicillin ⁵ | 1.8 (2372) | 0.8 (3720) | 9.5 (7076) | 19.0 (10976) |
| | erythromycin | 1.8 (2334) | 1.3 (3554) | 8.3 (6832) | 14.5 (11212) |
| | tetracycline | 5.6 (1760) | 1.7 (3376) | 10.5 (5019) | 11.2 (5993) |
| <i>S pneumoniae</i> , invasive disease ⁶ | penicillin ⁵ | 1.0 (382) | 1.4 (694) | 3.4 (989) | 15.0 (1182) |
| | erythromycin | 0.8 (382) | 1.9 (694) | 2.6 (989) | 4.1 (853) |
| | cefotaxime ⁵ | 0.3 (382) | 0.1 (694) | 1.8 (989) | 7.3 (1182) |
| <i>Enterococcus spp</i> ² | amoxicillin ⁷ | 1.6 (6127) | 2.3 (2573) | 1.5 (7373) | 2.4 (17548) |
| | vancomycin | NA | 0 (148) | 0.2 (1141) | 0.5 (4752) |
| <i>E coli</i> , urinary isolates ² | amoxicillin ⁷ | NA | 56.2 (29394) | 55.9 (48706) | 56.0 (138712) |
| | amoxicillin/clavulanic acid | NA | 6.9 (27249) | 10.6 (42666) | 12.2 (136326) |
| | trimethoprim | NA | 18.8 (29340) | 19.6 (48098) | 22.6 (111710) |
| | nitrofurantoin | NA | 2.2 (28331) | 1.6 (48123) | 1.7 (124362) |
| | fluoroquinolone | NA | 0.2 (7014) | 0.5 (40032) | 0.6 (118917) |
| <i>E coli</i> , non-urinary isolates ² | amoxicillin/clavulanic acid | NA | 18.3 (2318) | 22.8 (7358) | 21.8 (15948) |
| | cefuroxime | NA | 2.3 (1158) | 3.2 (6309) | 4.5 (6893) |
| | gentamicin | NA | 0.5 (3200) | 0.8 (10352) | 0.9 (13789) |
| | fluoroquinolone | NA | 0.1 (728) | 0.5 (4717) | 0.8 (10800) |
| <i>P aeruginosa</i> ² | gentamicin | 6.5 (11832) | 5.8 (5918) | 12.5 (9556) | 9.5 (20542) |
| | tobramycin | 1.4 (1759) | 3.1 (2535) | 3.9 (6757) | 2.8 (11033) |
| | ceftazidime | NA | 6.6 (1006) | 5.0 (4832) | 5.2 (11147) |
| | fluoroquinolone | NA | 8.4 (1652) | 8.8 (8123) | 9.9 (16551) |
| <i>H influenzae</i> , non-invasive disease ² | amoxicillin ⁷ | 10.2 (4347) | 8.4 (4131) | 12.0 (12244) | 19.3 (18852) |
| | amox/clav | 0.9 (555) | 1.1 (1136) | 1.1 (9839) | 0.6 (15040) |
| | co-trimoxazole | NA | 11.4 (1581) | 11.9 (6605) | 14.7 (13964) |
| | tetracycline | NA | 1.7 (2082) | 1.0 (7810) | 1.5 (13007) |
| <i>H influenzae</i> , invasive disease ⁶ | amoxicillin ⁷ | 14.9 (388) | 13.2 (478) | 21.8 (179) | 11.5 (122) |
| | amoxicillin/clavulanic acid | 0.3 (388) | 0.2 (478) | 3.4 (179) | 1.6 (122) |
| | cefuroxime | 0.3 (388) | 0.8 (478) | 3.4 (179) | 4.9 (122) |
| <i>N meningitidis</i> , invasive disease ⁶ | penicillin ⁸ | 2.2 (139) | 2.1 (291) | 3.9 (659) | 7.9 (431) |
| | rifampicin | 0 (139) | 0.3 (291) | 0 (659) | 0 (431) |
| <i>N gonorrhoeae</i> ^{2,3} | penicillin | NA | 16.4 (85) | 11.6 (879) | 10.4 (1437) |
| | fluoroquinolone | NA | 0 (85) | 0.7 (864) | 1.8 (1437) |
| <i>M tuberculosis</i> ² | isoniazid | NA | NA | 4.6 (438) | 8.2 (757) |
| | rifampicin | NA | NA | 0.7 (438) | 1.3 (757) |
| | multidrug resistant ¹⁰ | NA | NA | 0.7 (438) | 0.9 (757) |

Notes: 1 intermediate resistance not included in resistant category unless otherwise stated (refer footnotes 5 and 8 below)

2 collated clinical laboratory data

3 NA = not available

4 MRSA data from ESR for 1988-1998

5 includes intermediate resistant and resistant isolates

6 invasive disease isolates tested at ESR

7 ampicillin used in laboratory testing

8 reduced susceptibility (MIC 0.12-0.25 mg/l)

9 data from northern North Island only

10 resistant to at least isoniazid and rifampicin

Neisseria meningitidis: Reduced susceptibility to benzyl-penicillin, although currently only at a maximum MIC of 0.25 mg/l, increased sharply to 18.5% of isolates in 1999, increasing the 1997-99 three-year average to 7.9%. However, there are no data that show patients with invasive disease due to strains with reduced susceptibility do less well when treated with benzylpenicillin than those infected with fully sensitive strains. Therefore benzylpenicillin remains the drug of choice.¹⁴

Neisseria gonorrhoeae: Data are only available from the northern North Island. Penicillin (amoxycillin) resistance appears to be falling with the change to routine quinolone treatment of gonorrhoea. However, concomitant with the increasing use of quinolones for both gonococcal and other infections, there is an upward trend in fluoroquinolone resistance among *N gonorrhoeae*.

Mycobacterium tuberculosis: Isoniazid resistance is increasing. Multidrug resistance (MDR), resistance to at least isoniazid and rifampicin, is rare but drives initial four drug regimens for this disease, particularly in Pacific Island and Asian immigrants. All cases of MDR TB in New Zealand have been in people born overseas. Whether the frequency of MDR warrants all immigrants initially receiving a four-drug regimen, with its increased complexity and toxicity, is debatable.

Intestinal pathogens: The prevalence of resistance among the commonest of these pathogens, *Campylobacter*, *Salmonella*, *Yersinia* and *Shigella*, has not been continuously or comprehensively monitored. However, ESR monitored *Salmonella* susceptibilities continuously between 1972 and 1982, and intermittently since then. There has been little significant change in resistance over that period.¹⁵ ESR now participates in the WHO global Salmonella Monitoring Programme and in 2000 resumed antimicrobial susceptibility testing of 20% of the isolates referred for speciation.

The primary purpose of monitoring antimicrobial resistance in *Salmonella* is to provide an indication of the possible effects of antibacterial use in animals as well as humans. Antimicrobial treatment of uncomplicated salmonellosis is not recommended, and therefore data on resistance among *Salmonella* are not collected primarily to guide therapy.¹⁶

In contrast with *Salmonella*, a point prevalence survey of *Shigella* in 1996 showed a high rate of ampicillin (42%) and co-trimoxazole (57%) resistance in 106 isolates.¹⁷ Consequently, quinolones are recommended for the minority of shigellosis cases where antibiotic treatment is indicated.

No national data on *Campylobacter* susceptibilities are available in New Zealand, but antibiotics are rarely indicated for diarrhoeal disease due to this organism. A 1998 study in the Auckland area found low rates of erythromycin (1.5%) and fluoroquinolone (2.5%) resistance amongst 202 *Campylobacter* isolates.¹⁸

Discussion

There are clear and mostly obvious limitations to the epidemiological and scientific reliability of these data; these limitations are not unique to New Zealand. The limitations, which particularly apply to the collation of locally derived clinical laboratory susceptibility testing results, include:

- Because participation is voluntary, the number of clinical laboratories contributing their susceptibility testing results has varied from 7 to 21 between 1988 and 1999. In the early 1990s, the data were fragmentary because many laboratories were unable to extract data from computer systems. Some laboratories only contributed spasmodically. However, since 1998, at least 20 laboratories have consistently reported results.
- Antimicrobial susceptibility testing methodologies are still not uniform, although the majority of clinical laboratories are now using NCCLS guideline methods. All clinical laboratories participate in quality assurance programmes.⁵

- A clinical laboratory's catchment area may no longer be confined to the surrounding geographic area. For example, a laboratory in Christchurch processes specimens from all over the South Island and from Hawkes Bay. Therefore, any analysis and comparisons of regional differences in resistance rates, based on the locations of the laboratories, may be invalid.
- While attempts are made to eliminate duplicate specimens from the same patient, not all laboratories contributing data are able to eliminate repeat isolates. As re-sampling is more likely when there is treatment failure, data which include repeat isolates will be skewed to indicate a greater rate of resistance than is real. This effect may account, at least to some extent, for the data from the 1999 point prevalence survey indicating a much lower prevalence of MRSA than the collated clinical laboratory data (1.9 vs 5.8%).⁶
- Another factor that needs to be considered, especially when using surveillance data to guide empirical therapy, is that the actual isolates tested may not fully represent the actual population of that organism. Medical practitioners make decisions about when to take specimens for laboratory investigation. The best example here is the recent trend, driven almost entirely by short term economic pressures, to empirically treat first time urinary tract infections in young women. There will thus be a bias to test treatment failures, which will result in testing a more resistant subset of organisms. The data generated by the testing of this more resistant subset will then drive empiric decisions inappropriately.

What can be done to enhance the surveillance of antimicrobial resistance in New Zealand? Some of the current limitations of the system of collating data from clinical laboratories need to be addressed. All clinical laboratories need to be encouraged to submit their available data. The inclusion of duplicate isolates needs to be minimized. To judge the accuracy of the antimicrobial susceptibility data on invasive isolates of *S pneumoniae* and *H influenzae*, we need to know how complete or representative the referral of these isolates to ESR is. The Antibiotic-Resistant Bacteria Monitoring Scheme and the national point prevalence antibacterial resistance surveys need to continue. Perhaps most importantly of all, we need prospective studies which isolate and test the causative organisms from all patients presenting to general practitioners with particular syndromes. When this was done recently in Christchurch, only 11.9% of the *E coli* isolated from all women with urinary tract infections presenting to general practitioners were resistant to trimethoprim (Chambers S. Personal communication, 2001), compared with a rate of 18% among routine isolates tested by the local laboratory and a national rate of 22.6%. The message is clear.

Recent attention has focused on the impact of antibacterial use in agriculture on human health.¹⁹ There is general agreement that some form of monitoring of resistance in agricultural settings is needed, but there is much debate over the best way to do it.^{20,21} In New Zealand some resistance data on relatively small numbers of animal pathogens are collected, but the process is much more rudimentary than that for human health. We need to consider if the surveillance of resistance among animal pathogens should be enhanced in terms of selecting animals and isolates to test, laboratory testing and quality control, and central collation. We need to consider if the antimicrobial susceptibility of non-pathogenic animal flora, and some plant and environmental isolates, should be monitored to detect potential origins of antimicrobial resistance genes. While it would be 'interesting' to have all these data, their collection would be an expensive exercise and one that needs to be justified. Nevertheless, complex integrated systems of surveillance are being initiated in Denmark, France and the United States.²²⁻⁴ We need to continue to talk about the best ways to monitor

antimicrobial resistance in New Zealand, and the best way to use the surveillance data to control the spread of resistance and the emergence of new resistance.

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Surveillance and control notes

High incidence of malaria among defence personnel returning from East Timor

Malaria notifications more than doubled in 2000, with 111 cases notified compared with 46 in 1999. Most of the excess cases in 2000 were in military personnel: 74 of the 97 cases for whom occupation was recorded. Of these cases, 72 had recently returned from East Timor. Among the military personnel the malarial species was determined for 65 cases, of which 56 (86.2%) were *Plasmodium vivax* infection and four (7.1%) were *P falciparum*. A further five cases (8.9%) were diagnosed with a combination of *P vivax* and *P falciparum* infection.

In 2000, there were 37 cases of malaria in people who did not list military occupations. Overseas travel information was recorded for 34 of these 37 cases. The most commonly visited country was Papua New Guinea (12 cases), followed by India (6 cases) and Africa (6 cases). The malarial species was determined for 33 cases, of which 22 (66.7%) were diagnosed with *P vivax* infection, six (18.2%) with *P falciparum*, two with *P malariae*, and one with *P ovale*. One further case was diagnosed with a combination of *P vivax* and *P falciparum* infection, and another with *P vivax* and *P malariae*.

Additional information on malaria among military personnel in East Timor has been provided by the New Zealand Defence Force.

The first 6-month rotation of personnel to East Timor returned to New Zealand in May 2000. There were 60 cases of malaria in this group of 665 personnel, giving an attack rate of 18 045 per 100 000 person years of exposure. Most (70.0%) of the cases in this group were diagnosed in the June 2000 to February 2001 period following their return to New Zealand. Malaria rates were lower in the second 6-month rotation of personnel, which returned to New Zealand in November 2000. There were two cases diagnosed among this second group while they were in East Timor, and a further 10 cases between their return to New Zealand and February 2001: an attack rate of 3609 per 100 000. Among the average 80 airforce personnel based in East Timor, only two malaria cases had been diagnosed as at February 2001.

The factors that contributed to the high incidence of malaria in military personnel are under investigation. The first rotation was in East Timor during the wet season, which may account for their much higher rate of infection. Malaria prevention measures used by the military in East Timor included DEET repellent, permethrin-impregnated clothing and bed nets, prophylactic use of doxycycline, and an additional eradication regime of primaquine upon return to New Zealand. (Data on defence personnel reported by Dr Donald Stewart, Captain Tony Lewis, Lt Colonel Julie Leighton, and Wing Commander Len Bagnall, New Zealand Defence Force.)

Surveillance data

National surveillance data - January 2001

| Disease ¹ | Current year - 2001 ² | | | Previous year - 2000 | | | Trends - January 2001 |
|------------------------------------|----------------------------------|-------------------------------|---------------------------|----------------------|-------------------------------|----------------------------|-----------------------|
| | Jan 2001 cases | Cumulative total year-to-date | Current rate ³ | Jan 2000 cases | Cumulative total year-to-date | Previous rate ³ | |
| AIDS | 5 | 5 | 0.9 | 1 | 1 | 0.9 | |
| Campylobacteriosis | 1050 | 1050 | 233.5 | 1024 | 1024 | 230.0 | |
| Cholera | 0 | 0 | 0 | 0 | 0 | 0 | |
| Creutzfeldt-Jakob disease | 0 | 0 | 0.1 | 0 | 0 | 0.1 | |
| Cryptosporidiosis | 35 | 35 | 21.8 | 20 | 20 | 26.0 | *** |
| Dengue fever | 0 | 0 | 0.2 | 0 | 0 | 0.2 | |
| Gastroenteritis ⁴ | 101 | 101 | 21.7 | 47 | 47 | 16.7 | *** |
| Giardiasis | 105 | 105 | 46.0 | 124 | 124 | 49.2 | |
| <i>H influenzae</i> type b disease | 1 | 1 | 0.4 | 1 | 1 | 0.3 | |
| Hepatitis A | 3 | 3 | 3.0 | 3 | 3 | 3.0 | |
| Hepatitis B (acute) ⁵ | 4 | 4 | 1.9 | 15 | 15 | 2.9 | ** |
| Hepatitis C (acute) ⁵ | 1 | 1 | 2.1 | 8 | 8 | 2.7 | |
| Hydatid disease | 1 | 1 | 0.1 | 0 | 0 | 0.2 | |
| Influenza ⁶ | 3 | 3 | 7.0 | 1 | 1 | 22.0 | *** |
| Lead absorption | 20 | 20 | 3.8 | 7 | 7 | 4.1 | |
| Legionellosis ⁶ | 6 | 6 | 2.0 | 5 | 5 | 1.9 | |
| Leprosy | 0 | 0 | 0.1 | 0 | 0 | 0.3 | |
| Leptospirosis | 10 | 10 | 2.8 | 9 | 9 | 1.9 | ** |
| Listeriosis | 3 | 3 | 0.6 | 4 | 4 | 0.6 | |
| Malaria | 6 | 6 | 3.1 | 7 | 7 | 1.3 | *** 131 |
| Measles | 5 | 5 | 1.7 | 9 | 9 | 3.0 | *** |
| Meningococcal disease | 47 | 47 | 13.9 | 23 | 23 | 14.0 | |
| Mumps | 2 | 2 | 1.4 | 3 | 3 | 1.5 | |
| Paratyphoid | 1 | 1 | 0.6 | 0 | 0 | 0.4 | |
| Pertussis | 303 | 303 | 116.4 | 236 | 236 | 35.3 | *** 230 |
| Rheumatic fever | 3 | 3 | 3.2 | 5 | 5 | 1.7 | *** |
| Rubella | 0 | 0 | 0.7 | 2 | 2 | 1.0 | |
| Salmonellosis | 254 | 254 | 54.2 | 99 | 99 | 53.2 | |
| Shigellosis | 9 | 9 | 3.1 | 11 | 11 | 3.9 | |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0.1 | |
| Tuberculosis | 39 | 39 | 10.4 | 23 | 23 | 12.0 | * |
| Typhoid | 4 | 4 | 0.6 | 2 | 2 | 0.3 | * 120 |
| VTEC/STEC infection | 5 | 5 | 2.0 | 3 | 3 | 1.8 | |
| Yersiniosis | 57 | 57 | 11.1 | 51 | 51 | 13.2 | * |

Notes: 1 Other notifiable infectious diseases reported in January: Nil

2 These data are provisional

3 Rate is based on the cumulative total for the current year (12 months up to and including January 2001) or the previous year (12 months up to and including January 2000), expressed as cases per 100 000

4 Cases of gastroenteritis from a common source or foodborne intoxication (eg, staphylococcal intoxication or toxic shellfish poisoning)

5 Only acute cases of this disease are currently notifiable

6 Surveillance data based on laboratory-reported cases only

7 Percentage change is the difference between the number of cases in the current year (12 months up to and including January 2001) and the previous year (12 months up to and including January 2000). This difference is expressed as a percentage of the number of cases in the previous year.

Surveillance data

Surveillance data by health district - January 2001

Cases this month Current rate¹

| Disease | Cases for January 2001, ² and current rate ^{1,2} by health district ^{3,4} | | | | | | | | | | | | | | | | | | | | | | |
|-----------------------------|--|---------|--------------|------------|---------|----------|------------|----------|---------|-------|----------|---------|------------|----------|----------|-----------|------------|------|-------------|------------|------------|------------|-------|
| | Northern | | | | Midland | | | | | | Central | | | | | | Southern | | | | | | |
| | Northland | NW Auck | Central Auck | South Auck | Waikato | Tauranga | Eastem BoP | Gisborne | Rotorua | Taupo | Taranaki | Ruapehu | Hawkes Bay | Wanganui | Manawatu | Wairarapa | Wellington | Hutt | Nelson-Marl | West Coast | Canterbury | South Cant | Otago |
| AIDS ³ | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Campylobacteriosis | 18 | 110 | 82 | 72 | 94 | 34 | 5 | 7 | 5 | 8 | 21 | 3 | 48 | 7 | 23 | 12 | 94 | 40 | 22 | 7 | 221 | 39 | 49 |
| Cholera | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Creutzfeldt-Jakob disease | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Cryptosporidiosis | 1 | 2 | 0 | 2 | 1 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 12 | 2 | 1 | 0 | 6 | 0 | 0 | 0 | 2 | 3 | 1 |
| Dengue fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Gastroenteritis | 2 | 11 | 10 | 7 | 0 | 0 | 0 | 0 | 3 | 0 | 1 | 0 | 0 | 1 | 45 | 1 | 0 | 0 | 5 | 3 | 10 | 0 | 2 |
| Giardiasis | 4 | 17 | 16 | 8 | 10 | 3 | 1 | 1 | 3 | 1 | 2 | 0 | 5 | 0 | 1 | 0 | 9 | 5 | 0 | 1 | 12 | 3 | 1 |
| H influenzae type b disease | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Hepatitis A | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Hepatitis B | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Hepatitis C | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Hydatids | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Influenza ⁵ | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Lead absorption | 0 | 2 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 4 | 1 | 3 | 0 | 0 | 2 | 0 | 1 | 0 | 2 | 0 | 1 |
| Legionellosis ⁵ | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 1 |
| Leprosy | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Leptospirosis | 0 | 1 | 1 | 0 | 6 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Listeriosis | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 |
| Malaria | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| Measles | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Meningococcal disease | 4 | 3 | 5 | 15 | 2 | 1 | 1 | 1 | 2 | 2 | 0 | 0 | 1 | 2 | 2 | 1 | 2 | 1 | 0 | 0 | 1 | 0 | 1 |
| Mumps | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Paratyphoid | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Pertussis | 8 | 19 | 16 | 20 | 35 | 8 | 2 | 1 | 2 | 2 | 2 | 10 | 1 | 0 | 6 | 14 | 9 | 47 | 7 | 59 | 11 | 19 | 5 |
| Rheumatic fever | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Salmonellosis | 10 | 25 | 27 | 13 | 24 | 0 | 0 | 1 | 9 | 0 | 13 | 0 | 9 | 8 | 10 | 3 | 12 | 6 | 24 | 1 | 22 | 15 | 12 |
| Shigellosis | 0 | 2 | 2 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Tuberculosis | 0 | 3 | 10 | 6 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 7 | 2 | 0 | 0 | 4 | 0 | 1 |
| Typhoid | 0 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| VTEC/STEC infection | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| Yersiniosis | 0 | 6 | 7 | 5 | 3 | 2 | 2 | 2 | 2 | 4 | 0 | 0 | 2 | 0 | 0 | 1 | 2 | 4 | 0 | 9 | 3 | 0 | 3 |

Notes: 1 Current rate is based on the cumulative total for the 12 months up to and including January 2001, expressed as cases per 100 000
 2 These data are provisional
 3 AIDS data is reported for the greater Auckland and Wellington areas, rather than by health district
 4 Further data are available from the local medical officer of health
 5 Surveillance data based on laboratory-reported cases only

Public health abstracts

Hip protectors reduce risk of hip fracture in the elderly

A study in Finland has demonstrated that wearing anatomically designed external hip protectors can reduce by $\geq 50\%$ the risk of hip fractures in the elderly. The study involved 1801 ambulatory but frail adults aged ≥ 70 years, selected from community-based elderly care units in Finland. The units were randomly designated to treatment status (all subjects received hip protectors) or control status (no subjects used a protector). Subjects were followed for approximately two years, and during this time the rate of hip fracture in the hip protector group was 21.3 per 1000 person years, compared with 46.0 in the control group: a relative hazard in the treatment group of 0.4. The number needed to treat for one year to prevent one hip fracture was 41 persons (Kannus P, Parkkari J, Niemi S, et al. Prevention of hip fracture in elderly people with use of a hip protector.

N Engl J Med 2000; 343: 1506-13).

Editorial note: Hip fractures in the elderly, the vast majority of which are the result of a fall, cause significant morbidity and mortality. About a quarter of those injured die within a year, and the first-year direct cost in New Zealand was estimated in the mid-1990s to be almost \$42 million annually – a figure likely to be well short of the full current and future burden. Therefore, despite some difficulty regarding long-term compliance with wearing external hip protectors, the growing evidence for their efficacy and their relative low cost indicates that they have a large potential for cost saving and improving well being in the elderly. Hip protectors designed and manufactured in New Zealand are available from High Tech Bodywear Ltd, phone/fax 09 627 9596.

Outbreak of listeriosis in Finland linked to butter

An outbreak of listeriosis in Finland, June 1998-April 1999, was linked to the consumption of pasteurised butter produced by a Finnish dairy. Twenty-five cases infected with the same strain of *Listeria monocytogenes* serotype 3a were identified. Six cases died. Patients with the outbreak strain were more likely to have been admitted to a particular tertiary care hospital and to be immune compromised, than patients with other strains of *L. monocytogenes*. The outbreak strain was identified in packaged butter served at the hospital and in the source dairy, which began supplying butter to the hospital in June 1998. The outbreak ceased after the product was recalled, and no further cases due to the outbreak strain were detected after cleaning and restarting the production facility (Lyytikäinen O, Autio T, Maijala R, et al. An outbreak of *Listeria monocytogenes* serotype 3a infections from butter in Finland. J Infect Dis 2000; 181: 1838-41).

Editorial note: Between 1969 and 2000, there were five documented outbreaks of listeriosis in New Zealand. *L. monocytogenes* is widespread in the environment, being found in soil, water, green plants and animals, and consequently in food products of dairy, meat and vegetable origin. Although pasteurisation and sufficient heating or cooking of such products will kill the bacterium, subsequent contamination during processing, at the retail outlet, or in the home, are important preventable causes of infection. Many major New Zealand food industries have HACCP (hazard analysis and critical control point)-based food safety programmes (FSPs) in place to assure food safety, or are in the process of developing them. The Ministry of Health is working to assist other food production and service businesses to implement FSPs as soon as possible.

Travel health

Leptospirosis outbreak in adventure sport competitors

Participants in the Eco-Challenge-Sabah 2000 multisport expedition race in Borneo, Malaysia, experienced a high rate of leptospirosis. The event ran from 20 August to 3 September 2000. Teams spent from 6 to 12 days on the course, which included jungle trekking, canoe paddling, and open water swimming. Of the 304 athletes who competed, 158 were interviewed by the United States Centers for Disease Control and Prevention, and 68 (44%) met the clinical case definition for leptospirosis (acute onset of fever, with at least two of the following symptoms: chills, myalgias, headache, diarrhoea, or conjunctivitis). Specimens were obtained from 39 of these athletes, and 32 had serological evidence of leptospirosis or were culture positive. The risk of illness was significantly associated with exposure during the river swim (Update: Outbreak of acute febrile illness among athletes participating in Eco-Challenge-Sabah 2000 - Borneo, Malaysia, 2000. MMWR 2001; 50: 21-4).

Editorial note: A four-person team of New Zealanders competed in this Eco-Challenge event. All reported taking doxycycline prophylaxis and none became ill. Overseas travel remains an uncommon risk factor for notified leptospirosis in this country. Among the 99 cases in 2000, 4.7% (3 of the 64 for whom travel information was recorded) had a history of overseas travel during the incubation period. Travellers should be informed of this disease risk if their plans are likely to involve exposure to potentially contaminated water, soil or infected animals. If the risk of exposure is high, then prophylaxis with doxycycline 200 mg weekly could be considered. Medical practitioners should also consider leptospirosis in the differential diagnosis of febrile illness in the returning traveller, particularly if the patient has a history of exposure to fresh water.

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Phone: (04) 914 0700
Fax: (04) 914 0770



Phone: (04) 496 2000
Fax: (04) 496 2340

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Scientific Editor, New Zealand Public Health Report, ESR, PO Box 50-348, Porirua, Wellington, New Zealand. Phone: (04) 914 0700; Fax: (04) 914 0770; Email: michael.baker@esr.cri.nz

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