

Victorian Infectious Diseases Bulletin

The Water We Drink: Assessing its Impact on Human Health

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The provision of high quality drinking water has been vital throughout history. The advent of chlorination and the ability to identify many disease-causing micro-organisms have led to a reduction in disease caused by contaminated drinking water in developed countries.¹ Until recently it was assumed that drinking water caused minimal disease and that it was a simple engineering matter to maintain a clean, non-contaminated water supply.

However, an outbreak of cryptosporidiosis traced to the water supply in Milwaukee in the United States raised questions about our understanding of water-borne disease, of what defines good quality drinking water and of how it should be measured. The Milwaukee outbreak caused an estimated 400 000 cases of gastroenteritis in 14 days, with massive absenteeism and community disruption.² Milwaukee's water quality was within required limits during the outbreak, suggesting that reliance on routine water tests to predict safe water was not adequate.

QUALITY DRINKING WATER

A Canadian study measuring the endemic (background) level of gastroenteritis resulting from tap water highlighted the issues of good water quality, laboratory water test results and their relationship to health outcomes.³ This study compared the incidence of gastroenteritis in people drinking tap water (which met appropriate standards for the region) with another group drinking water filtered to a high standard with reverse osmosis filters. The reported incidence of gastroenteritis was 30 per cent higher in the group using tap water, suggesting the difference may be attributed to the presence of micro-organisms in the water. Despite structural weaknesses in its design, this study raised concerns about the limitations of relying on laboratory water tests alone to assess water quality.

The Canadian study, the outbreak in Milwaukee and other recent outbreaks have occurred where the drinking water met appropriate guidelines or standards.⁴ This suggests that conforming to guidelines and standards based on routine tests such

as total coliforms, faecal coliforms and heterotrophic plate counts does not imply that the water is necessarily safe from protozoa and viruses. Given that tests for protozoa and viruses are often unreliable, difficult to perform and expensive, they are not routinely performed.⁵⁻⁷

An additional problem is that the acceptable level of a micro-organism — *Cryptosporidium* for example — in a water supply is also uncertain because we do not know the infective dose of such organisms and the sensitivity of the water test is uncertain.⁸⁻¹⁰ The problem of protozoal testing has been highlighted in Sydney, where *Cryptosporidium* and *Giardia* have been found in the water supply but there has been no measurable increase in disease. Thus, there is no certainty as to what level of contamination leads to disease.

We need to change the emphasis in the way we measure water quality. Water testing combined with the measurement of human disease attributable to water is a more powerful method for assessing water quality.

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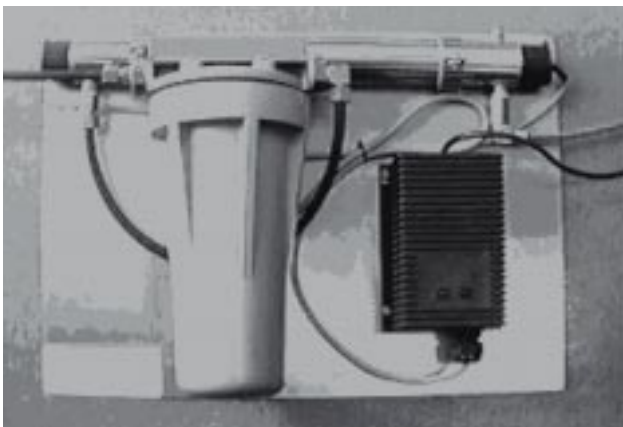


Victoria

THE MELBOURNE WATER QUALITY STUDY

The Water Quality Study is a randomised, controlled trial in the south-east suburbs of Melbourne. The Department of Epidemiology and Preventive Medicine is conducting the study under the auspices of the Cooperative Research Centre for Water Quality and Treatment. The study will measure the difference, if any, in the levels of gastroenteritis in people drinking treated and untreated tap water. It will combine the direct measurement of human health with water quality data. Six hundred families using chlorinated but unfiltered water have been randomly allocated to receive either real or sham water treatment units (see photo), which they will use as their main supply of drinking water for 18 months.

Over the study period, data on gastroenteritis symptoms are being collected by means of a weekly health diary. Participants also complete a questionnaire following episodes of gastroenteritis and a control questionnaire when they are well. The families complete a food frequency questionnaire at the end of summer and winter, and on three occasions they complete a water consumption questionnaire. Faecal specimens obtained when participants did not have gastroenteritis (before water treatment units were installed) and during gastroenteritis episodes (after water treatment units were installed) are being analysed for causative organisms. Mains water samples are also being tested for a range of pathogens.



Water treatment unit

The results of the Water Quality Study will provide extremely valuable information on causes of community acquired gastroenteritis, and they will indicate whether a public health benefit could be expected from the introduction of more expensive methods of water treatment in Melbourne and other cities with similar water supplies. Another important role of the study is to provide information about the non-water causes of community-based gastroenteritis, including their sources, the causative organisms and the routes of transmission.

OTHER STUDIES

While the Water Quality Study will provide useful information on specific pathogens, this is not its primary focus. To supplement the Water Quality Study, a series of

case control studies of *Cryptosporidium* and *Giardia* are being planned.

The *Cryptosporidium* case control study has recently commenced in Melbourne and is soon to begin in Adelaide. These studies will be useful in assessing the relative contribution of water as a risk factor for cryptosporidiosis and in setting acceptable levels of *Cryptosporidium* oocysts in drinking water.

If these studies show no association between cases of cryptosporidiosis and drinking tap water during a period of time when *Cryptosporidium* oocysts are detected in drinking water, then it would be reasonable to infer that current levels of oocysts in drinking water are not associated with significant disease. Alternatively if the study does show an association between cases of cryptosporidiosis and drinking water, then it would indicate that water treatment is required to reduce the number of oocysts in drinking water.

COMMUNITY MONITORING

In addition to the studies mentioned above, it would be useful to have a system that can continually monitor the level of gastroenteritis in the community. Most surveillance systems throughout the world are associated with considerable delays and may not be sensitive enough to detect outbreaks of water-borne gastroenteritis. Such surveillance is unable to identify the cause of an outbreak without further specific studies. There remains a need for a rapid surveillance system that can link measures of gastroenteritis to water quality data.

A preliminary review has investigated the potential of rapid proxy markers for community diarrhoea incidence such as numbers of requests for faecal analysis, the attendance of children with gastroenteritis at hospital casualty departments, and sales of anti-diarrhoeal medicines. Further research is being conducted on the use of these novel sources of timely information, and on their link to water quality information. New methods of computer analysis, such as the training of artificial neural networks, are being investigated, along with new methods of presenting the findings.

It is hoped that automating the scanning of large numbers of data items, and presenting anomalies in easily understood ways, will lead to earlier detection of outbreaks and faster reaction by the health authorities.

The provision of high quality drinking water is fundamental to community health, but there are still challenges to achieving and maintaining such a water supply. We believe that these challenges are best met by using the integrated approach of monitoring water quality and measuring human disease attributable to water, rather than simply monitoring micro-organisms in the water.

The Department of Epidemiology and Preventative Medicine, Monash University, web site is:
<http://www.med.monash.edu.au/epidemiology/>

The Cooperative Research Centre web site is:
<http://www.med.monash.edu.au/epidemiology/crc>

ACKNOWLEDGEMENTS

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Infectious Diseases News

IN THIS ISSUE

WATER SAFETY IN CONTEXT

Public concern about water quality is understandably high. During July and August, residents of Sydney were advised to boil their water following protozoal contamination of the water supply. In September, Victoria's Chief Health Officer Dr Graham Rouch cleared the water supply for the suburb of Heathmont following an isolated case of giardiasis. In this issue, Margaret Hellard *et al.* (page 21) highlight the problems caused by highly sensitive water tests where there is no certainty as to what level of contamination causes disease. They call for a change in the way we measure water quality and outline studies in Victoria which will assist in the development of an integrated approach of testing water supplies and measuring water-borne disease in humans.

LEGIONNAIRES' DISEASE

Graham Tallis and Neil Formica (page 24) describe an outbreak of Legionnaires' disease in Melbourne's western suburbs, while Maria Yates (page 25) summarises the advantages and disadvantages of the urinary antigen test used in identifying this outbreak. Agnes Tan (page 26) discusses the molecular subtyping of the most common *Legionella* isolated from patients with Legionnaires' disease.

IMMUNISATION COVERAGE

Preliminary immunisation coverage data from the Australian Childhood Immunisation Register are included in the bulletin for each local government area (page 29).

Overall, 83 per cent of Victorian children aged 12-15 months had completed the primary course of vaccination (the third dose of diphtheria-tetanus-pertussis (DTP), oral polio and *Haemophilus influenzae* type b [Hib] vaccines) while 66 per cent of children aged 24-27 months were fully immunised for their age.

SETTING NATIONAL PRIORITIES FOR NOTIFICATION OF COMMUNICABLE DISEASES

The National Public Health Partnership Group recommended at its meeting in August 1997 that there should be an agreed list of notifiable food-borne infections. It referred this recommendation to the Legislation Review Working Group which, in cooperation with the Communicable Diseases Network of Australia New Zealand (CDNANZ), has agreed to examine this issue in the broader context of communicable disease notification. A workshop involving State and Territory representatives and other key stakeholders was convened on 21 September 1988, establishing draft principles which will underpin the revised list of nationally notifiable communicable diseases now being developed.

HEPATITIS C ACTION PLAN UNDER REVIEW

The Commonwealth Department of Health and Family Services is reviewing the National Hepatitis C Action Plan and the Nationally Coordinated Hepatitis C Education and Prevention Approach. Both plans were developed to help manage hepatitis C in Australia. The purpose of the review is to map progress over the past few years and recommend strategies for future management of the virus. A report is expected soon.

VALE DR CHESTER VOON

It is with deep regret that we note the passing of Dr Chester Voon. Chester was the Manager of the STD/Blood-Borne Virus Program at the Department of Human Services for six years. He will be sadly missed by all those who knew and worked with him.



NEW IMMUNISATION COORDINATOR

Dr Rosemary Lester, Immunisation Coordinator for the past several years, has been appointed Acting Head of Prevention and Child Health Surveillance. The new section, created as a result of restructuring within the Department of Human Services, will be responsible for immunisation, genetics, perinatal/paediatric surveillance and cancer screening. Dr Melissa Morgan, who has worked closely with Dr Lester over the past two years, has been appointed Acting Immunisation Coordinator.

ERRATUM

We apologise for the incorrect telephone and fax numbers for correspondence which were printed in the last issue. You will find the correct telephone and fax numbers for correspondence and subscriptions on the back page of this issue.

A Cluster of Legionnaires' Disease in July 1998

Graham Tallis and Neil Formica, Disease Control Victoria, Department of Human Services

Legionnaires' Disease is caused by bacteria of the Legionella genus. When inhaled into the lungs on fine water droplets, it can cause a severe form of pneumonia. The disease often affects older persons with risk factors such as smoking, diabetes, excess alcohol consumption, chronic lung disease or immune suppression. Outbreaks may be associated with water systems, especially cooling towers of large air-conditioning systems. Cooling towers and other water systems are routinely treated, usually with chemicals, to minimise the risk of contamination.

In Victoria, about 20–40 cases are notified each year (usually during summer and autumn), and up to 20 per cent of these may be fatal. Over two days during winter (30 June – 1 July 1998), Disease Control Victoria was notified of three cases in women who lived or worked in a western suburbs' municipality. This was considered likely to represent an outbreak. We report a summary of the outbreak investigation.

THE FIRST THREE CASES

The first case was a 73-year-old woman who had been admitted to intensive care with symptoms of fever, dyspnoea and cough. Diagnosis was based on a positive *Legionella pneumophila* serogroup (SG) 1 urinary antigen test. *L. pneumophila* was subsequently cultured from a bronchial aspirate. The patient died in intensive care on 30 June. The second case was a 67-year-old woman admitted to hospital with left upper lobe pneumonia. Diagnosis was again based on a positive urinary antigen. *L. pneumophila* SG1 was subsequently cultured from sputum. She was treated successfully and discharged after a full recovery. The third case was a 53-year-old woman who also required intensive care. As with the first two cases, initial diagnosis was based on a positive urinary antigen. *L. pneumophila* SG1 was subsequently cultured from a bronchial aspirate.

Interviews with either the case or a near relative established the movements of the three cases during their incubation periods. These interviews indicated a likely common source of infection for at least two cases who frequented the same suburban shopping and business district.

COOLING TOWERS

Cooling towers in the area were inspected and sampled for microbiological testing. Two towers were subsequently shown to be contaminated with *L. pneumophila* SG1, and were immediately decontaminated.

The microbiological and epidemiological evidence suggested a common source outbreak, so a media release was issued to alert the public to symptoms of Legionnaires' disease. Subsequently, all general practitioners in the region received a 'health alert' advising them of the outbreak and of the appropriate diagnostic tests and management of Legionnaires' disease. Hospitals in the area were also alerted.

TWO MORE CASES

Active surveillance was undertaken in the two workforces where the contaminated cooling towers were located but no further cases were identified. A further two cases were identified through enhanced community surveillance. The fourth case was a 49-year-old woman admitted to hospital on 14 July with pneumonia. She made an uneventful recovery. The fifth case, a 75-year-old man admitted to hospital on 13 July with pneumonia, also made an uneventful recovery. Diagnosis was based on a positive *L. pneumophila* SG1 urinary antigen test in both cases.

Both of these cases identified exposure to the same suburban shopping centre, strengthening the hypothesis that there was a local environmental source associated with the cases. The incubation period of both cases indicated infection before the effective decontamination of each tower on 9 July. Workforce and community surveillance did not identify further cases.

LABORATORY DIAGNOSES

All isolates of *L. pneumophila* SG1 were subtyped by pulsed-field gel electrophoresis (PFGE), which indicated that isolates from the first and third cases were indistinguishable from those of the second tower. Isolates from the second case and the first tower differed from this pattern and from each other.

Results Of Pulsed-Field Gel Electrophoresis, Legionellosis Investigation, Victoria, 1998		
Isolate	Date of sample	Subtype
Case 1	28 June	Strain A
Case 2	1 July	Strain B
Case 3	28 June	Strain A
Tower 1	1 July	Strain C
Tower 2	3 July	Strain A

The Environmental Health Unit has continued to liaise with the water treatment companies engaged to maintain the towers that tested positive for Legionella bacteria. The companies received a report on the inspection of the cooling towers, their maintenance and recommendations for improvement.

CONCLUSION

On the basis of the epidemiological and microbiological results, it is highly likely that the first and third cases were infected through exposure to the second cooling tower. The second case did not give a history of potential exposure to either of the contaminated cooling towers, and subtyping supports the conclusion that this case probably acquired her infection from a different, incidental source.

The fourth and fifth cases were potentially exposed to the contaminated towers and were likely part of the cluster. Molecular subtyping cannot be used to infer whether the second tower was the source of their infection because no organisms were cultured from these patients.

These results must be interpreted with caution. The frequency of the many different subtypes of *L. pneumophila* SG1 in the environment remains unknown. While urinary antigen testing is increasingly used for the diagnosis of Legionnaires' disease, and while it has benefits for both patient care and rapid detection of outbreaks, it is important to always culture the organism to help determine whether there is a common source for an outbreak. The subtyping methods used here illustrate this point.

The Legionella Urinary Antigen Test: VIDRL's Experience

Maria Yates, Victorian Infectious Diseases Reference Laboratory

Legionella pneumophila serogroup 1 is the most frequently identified Legionella species worldwide (in both clusters and sporadic activity of legionellosis). Soluble antigens of this serogroup can be detected in the urine of patients with Legionnaires' disease using the Binax Legionella urinary antigen enzyme immunoassay test. The Victorian Infectious Diseases Reference Laboratory (VIDRL) has been using this test for the past three years, accruing a database of over 600 tests.

ADVANTAGES OF THE TEST

The urinary antigen test has many features that make it useful when investigating cases of suspected legionellosis, especially when a rapid diagnosis is required to assist in focusing antimicrobial therapy or when clusters of legionellosis may require urgent public health intervention. Sputum is not always readily available by non-invasive means, whereas urine is easy to obtain and requires no complicated storage or transport media. The enzyme immunoassay format used in this test is rapid and familiar to laboratory workers.

The manufacturers indicate a sensitivity of 97.7 per cent and a specificity of 100 per cent when compared with another commercial immunoassay. VIDRL has relied on culture validation when both the urinary antigen test and culture have been available for comparison, and for episodes during cluster and sporadic activity. In our hands, the sensitivity has been closer to 90 per cent (38 of 43 culture positive cases).

POTENTIAL PROBLEMS

We have noted important reasons for failure to achieve 100 per cent sensitivity when compared with optimally collected respiratory samples for culture. These reasons

may not be evident when studies of sensitivity/specificity have relied on samples from *Legionella* outbreaks without including sporadic cases. Such reasons have included: established acute renal failure at presentation; the dilutional effects of some urine samples; inappropriate timing of samples; and the individual variability of inoculum of organisms at the time of presentation, especially from patients with less severe disease.

Noting these important provisos, VIDRL has been happy with the performance and reproducibility of the assay, which has proven particularly useful for rapid diagnosis in outbreaks of legionellosis. However, a negative urinary antigen test does not exclude a *Legionella* infection, and culture remains the diagnostic gold standard.

Occasionally other species of *Legionella*, such as *L. longbeachae*, can cause pneumonia indistinguishable from Legionnaires' disease. The urinary antigen test will not identify this organism, which will require an alternate means of diagnosis such as culture. We continue to recommend sending a sputum sample for culture for *Legionella* from all patients presenting with severe community acquired respiratory disease where *Legionella* is suspected.

Molecular Subtyping of *Legionella pneumophila* Serogroup 1 Isolates by Pulsed-Field Gel Electrophoresis

Agnes Tan, Microbiological Diagnostic Unit, University of Melbourne

L. pneumophila serogroup 1 (SG1) is the most common *Legionella* isolated from patients with legionellosis (Legionnaires' disease) and from cooling towers. Characterisation of these bacteria to the strain level may link human isolates with cooling tower or other environmental isolates, thus identifying sources of infection. Increasingly, laboratories are using molecular typing methods such as pulsed-field gel electrophoresis (PFGE) for this purpose.

PFGE involves embedding *L. pneumophila* SG1 organisms in an agarose gel, lysing the cells in situ, then digesting the chromosomal DNA with an enzyme (restriction endonuclease) which cuts the DNA strands only at sites with specific short nucleotide sequences. Mutations in the genome affecting these sites alter the number of places at which the DNA is cut, resulting in different numbers and lengths of DNA fragments.

Often, laboratories use two or more restriction enzymes in separate runs to increase the ability to differentiate strains. The Microbiological Diagnostic Unit uses two enzymes, *Xba*I and *Sfi*I, for *Legionella* isolates.

The DNA fragments produced from the digestion are separated by alternating electric fields (the PFGE process). As the electric field across the gel is changed, the DNA molecules change conformation and reorient themselves before migrating in the direction of the electric field. The time they take to reorient is directly proportional to the molecular weight of the fragment.

Larger fragments spend more time in each switching cycle reorienting before they can migrate through the gel, and at the completion of the process, they have migrated a shorter distance than that migrated by smaller fragments.

The result of the process is a 'fingerprint' of the genome of the organism, with 'bands' comprising fragments of DNA positioned in the gel according to their molecular weight. Staining the gel with ethidium bromide renders these bands visible.

The DNA fingerprints or band patterns of different isolates of *L. pneumophila* SG1 may then be compared to determine their relatedness. A proposed rule of thumb is that isolates exhibiting up to three band differences are considered to be

epidemiologically related, or insignificant variants of the one pattern.¹ At the Microbiological Diagnostic Unit, we also compare the pattern for each new isolate with those of previous isolates, and we allocate the same number if a match is found (or a new number if there is no match). A letter, in addition to the pattern number, identifies the variants of a strain. Thus, pattern 2A is a variant of pattern 2, and both patterns are unrelated to pattern 3. Laboratories combine the pattern numbers for each of the restriction enzymes to form an overall PFGE pattern number.

A drawback of PFGE is that there is no universal scheme available for naming and comparing the band patterns produced. Therefore, PFGE results must be interpreted in the context of each individual laboratory's experience with the organism in question²⁻³.

The Microbiological Diagnostic Unit commenced routine subtyping of *L. pneumophila* SG1 by PFGE in 1994. To mid-1998, we have used PFGE on more than 440 isolates of *L. pneumophila* SG1 (derived from both human cases and environmental sources). Each of the two enzymes mentioned above has produced over 40 different PFGE patterns from among Victorian *L. pneumophila* SG1 isolates. These PFGE patterns indicate that there is a greater diversity of molecular subtypes in environmental cultures than among isolates from humans. However, there appears to be one predominant strain of *L. pneumophila* SG1 among human and environmental isolates in Victoria.

The Microbiological Diagnostic Unit had not previously seen the PFGE pattern of a *L. pneumophila* SG1 strain involved in the recent cluster of Legionnaires' disease cases in Melbourne. This supported the epidemiological and microbiological evidence linking several of the cases to a particular environmental source of the organism.

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Using 16S rRNA Gene Sequencing to Detect and Classify Unusual Pathogens

Wee Tee, Victorian Infectious Diseases Reference Laboratory

To the great joy of microbiologists, it is now possible to identify bacteria that have never been cultured using 16S rRNA gene sequencing. This remarkable technique has arisen from technological advances (the automation of DNA gene sequencing, polymerase chain reaction [PCR] and computerised databases) and fundamental microbiological research (ribosomal RNA genes, molecular evolution and taxonomy).

Generally, 16S rRNA gene sequencing can be used to classify all bacteria, because all bacterial cells have a characteristic 16S rRNA gene. The 16S rRNA gene is about 1500 bases long but has regions that are identical to other bacteria (conserved regions) and other regions that vary between genera and species. The conserved regions can be used as targets for PCR primers which should bind to all bacteria, and the variable regions contain the gene sequence used to differentiate bacteria. These PCR primers are specific for bacteria and mostly do not bind to the DNAs extracted from humans, fungi or protozoa.

16S rRNA gene sequencing consists of three parts: DNA extraction, polymerase chain reaction (PCR) and DNA gene sequencing. In theory, any bacterial DNA present in the clinical samples can be extracted and its 16S rRNA gene fragment can be amplified using the universal primers or broad range primers. Samples containing a small number of bacteria or even dead bacteria can also be used. The resulting PCR product (the fragment containing the 16S rRNA genes) is then sequenced using an automated DNA gene sequencer.

The gene sequence can be used in rapid computerised database searches (such as Genbank and the other ribosomal databases) to identify or classify the bacteria to species or genus level. The rRNA databases contain thousands of rRNA sequences of bacteria, including most reference strains and newly discovered species. These databases are updated daily.

During the past decade or so, a number of 'novel' infectious agents have emerged and have been linked with previously unrecognised bacterial diseases. New bacteria that were previously unculturable or culture resistant, including some unusual *Mycobacterium* species, have been identified using this technique.

At the Victorian Infectious Diseases Reference Laboratory, we have used this technique extensively over the past three years to classify *Campylobacter*, *Helicobacter* and other unusual micro-organisms.¹⁻³ From isolates or cultures, we have successfully identified three new species causing human infections, two of which are yet to be named.⁴

Recently, we have also applied this technique to definitively identify an unusual *Mycobacterium* species and a culture-resistant *Mycobacterium* species. The first case involved a patient with *M. abscessus* pneumonia. Conventional tests using biochemical reactions were unable to differentiate this organism from *M. chelonae*. Dr Daniel O'Brien (see box below) highlighted the clinical significance of this definitive identification.

An Unusual Mycobacterium

Presented to the Melbourne Infectious Diseases Group by Dr Daniel O'Brien, Victorian Infectious Diseases Service

Earlier this year, a 29-year-old man presented with an illness that was recognised as a cavitating pneumonia. He had insulin-dependent diabetes mellitus and was a heavy smoker.

The pathogenic organism responsible for the pneumonia was initially thought to be *M. tuberculosis*, but was later found to be a rapidly growing non-tuberculous *Mycobacterium*. *M. tuberculosis* is a relatively slow growing species. However, laboratory biochemical and sensitivity testing could not determine the precise species, being unable to distinguish between two related rapidly growing species, *M. abscessus* and *M. chelonae*. The former species is more common but conventional laboratory tests had tentatively identified the organism in this case as *M. chelonae*.

The distinction was important because clinical management decisions on the need for surgery, the choice of medication and the duration of therapy depended on knowing the exact species. To try to resolve the species' identity, the organism was sent to the Victorian Infectious Diseases Reference Laboratory for analysis using 16S rRNA methods. This resolved the species' identity and confirmed that the organism in this case was the more common *M. abscessus*.

The second case involved a patient with chronic cough. Acid fast bacilli were seen on several occasions but attempts at culture using conventional media were unsuccessful. Inoculated Bactec bottles were referred to us, and we used 16S rRNA gene sequencing to identify an organism indicative of *M. genavense*.

In summary, this powerful technique permits accurate identification of known and novel bacteria of clinical and public health significance from both cultures and clinical specimens. Its wide application has the potential to greatly increase our understanding of the role of bacterial pathogens in human disease, including diseases not previously recognised as infectious.

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Immunisation Update

Stephen Pellisier, Enhanced Measles Control Campaign, Department of Human Services

ENHANCED MEASLES CONTROL CAMPAIGN

Federal Minister for Health and Family Services, Dr Michael Wooldridge, officially launched the Victorian component of the national measles control campaign at Chirnside Park Primary School in Melbourne's outer eastern suburbs on 3 August. The program is now in full swing, with all primary school children being offered measles-mumps-rubella (MMR) vaccination. Feedback from metropolitan and rural areas indicates high coverage in schools already visited. The school data collection forms are constantly flowing in, with excellent responses to the schools' immunisation days.

A follow-up media campaign will occur 15–28 November 1998. The campaign will be aimed at children who have missed the school immunisation days, as well as those who have slipped through the immunisation providers' absentee processes.

IMMUNISATION PROMOTIONAL MATERIAL

Given the recent amendment to the Immunisation Schedule (see box), various immunisation educational and promotional materials have become outdated. A revised 'Understanding childhood immunisation' booklet is now available, and the School Entry Immunisation Certificate pamphlet 'A great start to school life' and its associated poster are also still available. You can order these items from the Department of Human Services.

Reminder: Vaccine Schedule Changes

Children should now receive the fifth dose of diphtheria-tetanus-pertussis (DTP) vaccine and the fourth dose of oral polio vaccine (OPV) before school entry (at 4–5 years of age). These vaccines, traditionally given in the first year of primary school, are now administered at preschool age in line with other States and Territories.

The second dose MMR vaccine (previously recommended at 10–16 years of age) is now also recommended before school entry (at 4–5 years of age).

IMMUNISATION COVERAGE

Preliminary data from the Australian Childhood Immunisation Register (ACIR) is shown in the table. The data are currently being checked and may be subject to review. Coverage rates are based on two cohorts of children aged 12–<15 months and 24–<27 months as at 31 March 1998, and they include vaccinations recorded by the ACIR up until 31 August 1998.

The table has been sorted in order of coverage for children aged 12–<15 months. The birth cohort within each local government area is proportionally allocated on the basis of postcode. This had led to some anomalies when comparing the birth cohort and the proportion immunised, particularly for those with smaller numbers in the respective cohorts.

Children aged 12–<15 months were considered fully immunised if they had received a third dose of DTP (DTP3) and OPV, plus either a second or third dose (depending on the vaccine) against *Haemophilus influenzae* type b. Children aged 24–<27 months were considered fully immunised if they had received DTP4, OPV3 and MMR vaccines.

The data indicate that immunisation coverage in Victoria is above the national average, but only eight of 78 local government areas have more than 90 per cent of children aged 12–<15 months who have completed the primary course of vaccination. Coverage rates among children at 24–<27 months are much lower, which suggests that we still have much to do to achieve full immunisation of 90 per cent of children by 2 years of age, and/or that we need to improve reporting of immunisation to the ACIR.

Immunisation Coverage for Children Aged 12–<15 Months and 24–<27 Months (at 31 March 1998), by
Local Government Area, Australian Childhood Register, 31 August 1998

	Local Government Area		Children Aged 12–<15 Months	% DTP3	% Fully Immunised	Children Aged 24–<27 Months	% DTP4	% MMR	% Fully Immunised
1	Pyrenees	S	12	98.7	98.7	21	79.0	79.0	68.9
2	Horsham	RC	68	96.8	95.3	66	98.4	97.0	89.3
3	Glenelg	S	69	94.4	94.4	66	77.7	80.7	59.7
4	Golden Plains	S	25	92.0	92.0	30	83.1	92.2	79.8
5	Ararat	RC	46	91.9	91.9	44	90.1	89.1	83.7
6	Moorabool	S	76	94.2	91.6	99	85.3	88.6	74.2
7	Wodonga	RC	138	91.3	90.6	141	81.5	89.7	74.8
8	Moyne	S	35	93.3	90.5	55	71.7	87.7	64.2
9	Indigo	S	35	88.4	88.4	49	76.0	83.2	58.6
10	Warrnambool	C	111	89.2	88.3	101	87.1	89.1	79.2
11	East Gippsland	S	116	90.8	88.2	126	70.5	84.5	62.6
12	Wellington	S	139	88.1	88.0	124	78.7	90.5	74.2
13	Loddon	S	27	87.9	87.5	33	79.1	84.4	68.8
14	Moira	S	96	87.3	87.3	100	77.7	87.4	68.4
15	Swan Hill	RC	74	90.8	86.9	72	86.6	92.0	77.1
16	Hepburn	S	50	86.8	86.8	52	69.1	84.4	61.2
17	Maroondah	C	306	87.8	86.6	307	79.8	87.8	70.1
18	Hobsons Bay	C	275	87.4	86.3	260	80.9	86.3	71.0
19	Ballarat	C	250	87.3	86.1	302	77.0	86.6	70.5
20	Greater Geelong	C	602	87.3	86.0	593	82.2	90.9	74.5
21	Corangamite	S	83	88.3	85.9	60	76.1	79.7	63.3
22	Yarriambiak	S	23	94.6	85.8	22	95.3	100.0	90.8
23	Stonnington	C	179	87.1	85.6	207	79.0	77.9	63.0
24	Central Goldfields	S	39	85.3	85.3	34	69.4	80.8	60.9
25	Colac-Otway	S	68	86.7	85.2	70	63.7	78.1	56.1
26	LaTrobe	S	256	87.8	85.2	262	77.4	92.8	70.4
27	Boroondara	C	325	85.8	85.2	425	81.5	85.4	71.6
28	Wyndham	C	332	86.4	85.2	443	75.2	88.9	66.6
29	Knox	C	467	85.9	85.1	476	81.6	89.5	72.5
30	Casey	C	757	86.3	85.1	799	79.7	87.3	70.3
31	Mitchell	S	90	85.9	84.9	107	77.7	88.8	68.5
32	Nillumbik	S	198	86.0	84.9	203	76.9	86.5	71.3
33	Whitehorse	C	369	85.7	84.9	428	76.0	84.4	68.0
34	South Gippsland	S	82	86.9	84.5	99	77.0	86.1	69.0
35	Gannawarra	S	45	88.9	84.4	39	89.7	87.2	74.4
36	Kingston	C	377	86.3	84.2	433	80.4	87.4	71.4
37	Southern Grampians	S	49	88.2	84.1	56	85.7	91.1	75.0
38	Wangaratta	RC	86	85.0	83.9	79	74.3	76.9	55.5
39	Banyule	C	396	85.3	83.8	359	79.2	88.4	71.1
40	Hume	C	544	85.6	83.8	561	78.6	88.9	69.9

(Table cont)

	Local Government Area		Children Aged 12-<15 Months	% DTP3	% Fully Immunised	Children Aged 24-<27 Months	% DTP4	% MMR	% Fully Immunised
41	Northern Grampians	S	40	83.7	83.7	42	83.3	82.4	61.1
42	Delatite	S	56	89.0	83.7	62	82.8	92.4	74.5
43	Darebin	C	434	85.2	83.6	419	74.2	82.8	65.1
44	Baw Baw	S	119	83.7	83.4	136	77.9	89.8	71.7
45	Monash	C	418	84.4	83.4	397	69.6	81.9	60.7
46	West Wimmera	S	18	83.3	83.3	18	83.3	94.4	83.3
47	Mildura	RC	159	83.9	83.3	163	72.4	83.5	68.3
48	Cardinia	S	151	83.9	83.0	200	77.2	89.6	71.1
49	Bayside	C	266	84.7	82.9	237	74.0	82.4	63.6
50	Greater Bendigo	C	305	84.5	82.9	273	74.6	85.0	66.3
51	Frankston	C	388	84.7	82.9	391	76.8	87.4	70.1
52	Moonee Valley	C	370	83.9	82.8	335	81.1	86.5	71.5
53	Macedon Ranges	S	126	84.3	82.7	139	74.7	86.1	61.3
54	Melton	S	228	82.9	82.5	210	75.7	86.7	66.2
55	Whittlesea	C	430	84.0	82.4	424	83.1	75.1	61.6
56	Maribyrnong	C	255	85.0	82.1	228	71.8	80.7	56.2
57	Moreland	C	457	84.4	82.0	490	78.9	85.7	70.5
58	Towong	S	16	87.8	81.7	19	63.0	94.2	37.0
59	Bass Coast	S	66	83.8	81.6	64	85.7	92.8	72.5
60	Greater Dandenong	C	485	83.8	81.2	482	74.8	78.7	57.7
61	Buloke	S	23	84.8	80.8	23	79.1	91.5	66.2
62	Greater Shepparton	C	209	81.7	80.2	191	76.9	83.8	67.3
63	Campaspe	S	127	81.1	79.6	110	78.7	76.9	59.8
64	Yarra	C	181	81.0	79.4	170	71.5	87.7	64.2
65	Yarra Ranges	S	502	80.7	78.8	510	67.7	80.6	56.9
66	Manningham	C	296	79.6	78.5	260	70.7	79.8	63.4
67	Mornington Peninsula	S	351	79.6	78.5	402	75.2	84.8	67.6
68	Glen Eira	C	368	78.4	77.3	318	74.6	81.4	60.8
69	Hindmarsh	S	30	76.7	76.7	22	86.4	95.5	81.8
70	Port Phillip	C	158	76.1	74.4	173	62.6	79.1	54.6
71	Melbourne	C	91	74.1	73.7	85	67.7	72.9	57.0
72	Surf Coast	S	65	82.7	73.5	55	66.3	79.3	56.7
73	Brimbank	C	492	75.7	72.1	589	72.2	81.8	57.7
74	Mount Alexander	S	46	72.2	70.0	59	60.1	71.9	60.1
75	Queenscliff	B	7	66.7	66.7	4	100.0	100.0	100.0
76	Murrindindi	S	44	71.3	66.7	39	60.3	75.4	51.5
77	Strathbogie	S	24	66.2	66.2	21	83.6	87.0	69.3
78	Alpine	S	27	59.3	59.3	36	71.9	69.1	44.1
	Victoria		15273	84.5	83.0	15735	76.9	85.2	66.9
	Australia		63219	82.4	80.2	64775	76.0	82.5	63.8

The data relating to children aged 24–27 months should be considered as preliminary. The proportions fully immunised appear low compared with the proportions for individual vaccines. The Health Insurance Commission are checking these calculations.

Gonorrhoea Infection in Victoria ... on the Rise?

Dennis Rhodes, Jane Hocking, Nick Crofts and Alison Rodger, Epidemiology and Social Research Unit, Macfarlane Burnet Centre for Medical Research

Gonorrhoea in Victoria declined steadily until 1991 when 337 cases were recorded, and it has since fluctuated around this figure. However, recent surveillance data indicate that this pattern may be changing.

INTRODUCTION

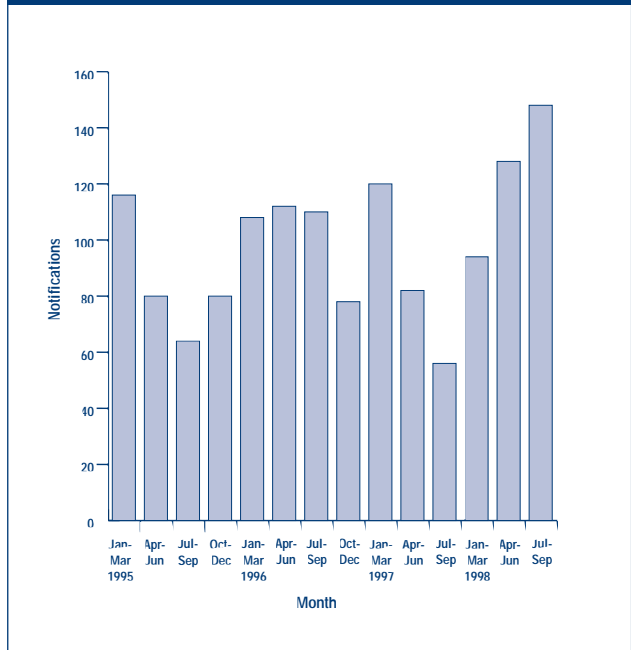
Gonorrhoea is caused by the bacterium *Neisseria gonorrhoeae* which infects the mucous membranes and is usually transmitted through sexual contact. The most common presenting symptom in males is a purulent urethral discharge, although asymptomatic carriage can occur. Rectal infection is usually asymptomatic, but may cause pruritus, tenesmus and discharge. Pharyngeal infection is usually asymptomatic. Females often experience a mild initial urethritis or cervicitis a few days after exposure, which usually passes unnoticed. Later, endometritis, salpingitis or pelvic peritonitis may develop, with the accompanying risk of subsequent impaired fertility.

VICTORIAN DATA

There were 280 reported cases of gonorrhoea during the first six months of 1998, compared with 203 cases for the same period in 1997 — an increase of 37 per cent. During 1997, 348 individuals were reported with gonorrhoea. This reverses the Victorian trend evident at the end of 1997, of a decline in the number of reported cases (see figure 1). It is, however, consistent with the recent rise in gonorrhoea reported in New South Wales¹ and overseas, principally the United Kingdom.²

In the six months to 30 June 1998, 93 per cent (n = 260) of cases were male, compared with 90 per cent for 1997. The majority of these cases identified as homosexual men — 57 per cent in 1998 and 55 per cent in 1997. However, data on sexuality are unknown for a significant number of individuals (18 per cent and 14 per cent respectively) (table 1).

Figure 1: Number of People from whom *N. gonorrhoeae* was Isolated, By Quarter of Specimen Collection, Victoria, 1995–98



Urethral isolates accounted for most of the additional cases for the first six months of 1998, with nine more cases (a 20 per cent increase) among heterosexual men and 43 more cases (a 37 per cent increase) among homosexual men, relative to cases in the same period in 1997.

Table 1: *N. gonorrhoeae*, by Gender and Sexuality, Per Quarter 1997–98

		Jan–Mar 1997	Apr–Jun 1997	Jul–Sept 1997	Oct–Dec 1997	Jan–Mar 1998	Apr–Jun 1998
Heterosexual	Male	22	21	10	26	19	33
	Female	9	6	5	8	9	9
Homosexual	Male	69	48	27	49	68	92
	Female	1	0	0	0	0	0
Unknown	Male	17	8	9	8	34	14
	Female	2	0	1	2	1	1
All Persons	Male	108	77	46	83	121	139
	Female	12	6	6	10	10	10
Total		120	83	52	93	131	149

Table 2: *N. gonorrhoeae*, by Site, Per Quarter 1997–98 (Total Isolates)

Site	Jan–Mar 1997	Apr–Jun 1997	Jul–Sept 1997	Oct–Dec 1997	Jan–Mar 1998	Apr–Jun 1998
Urethral	90	66	31	69	104	108
Vaginal/ Cervical	11	6	6	7	9	9
Rectal	17	11	9	13	12	26
Pharynx	13	2	3	5	7	5
Other	2	0	3	2	1	1
Total	133	85	52	96	133	149

Rectal isolates (n = 38) increased by 10 cases during the first six months of 1998, rising by 36 per cent from the number in the same period in 1997 (Table 2). Although the total number of cases has risen this year, the proportion of isolates from the rectum has remained stable (approximately 13.5 per cent in 1996, 1997 and 1998).

HOW CAN WE INTERPRET THESE DATA?

From the surveillance data we can identify a definite increase in gonorrhoea in Victoria. Whether this can be interpreted as an increase in unprotected anal sex (that is, a change in behaviour), or simply as an increase in overall sexual activity in the community, it causes concern (particularly given evidence of the role of gonorrhoea in amplifying the risk of transmission of HIV).³⁻⁶

The data highlight the need for improved control of gonorrhoea transmission through surveillance and the treatment of infection and, most importantly, through preventative educational campaigns focused on safer sexual practices. These issues are particularly relevant in the gay community, in which HIV remains the most prevalent and in which the major increase in gonorrhoea infection has occurred.

ANTIBIOTIC SUSCEPTIBILITY

Susceptibility data are available to the end of June 1998, but only for total isolate numbers. Specific epidemiological breakdowns of this data will be available at the end of 1998.

For historical purposes, penicillin and tetracycline sensitivities are reported. Only seven (2.5 per cent) of 280 isolates were fully 'sensitive' to penicillin, 166 (59 per cent) were 'less sensitive' and 104 (37.1 per cent) were classified as 'resistant'. Only 19 isolates (6.8 per cent) produced penicillinase (PPNG). Eleven isolates demonstrated high-level tetracycline resistance (TRNG). Oral ciprofloxacin (500 milligrams) currently remains the treatment of choice for gonorrhoea: only six isolates were classified as 'resistant' to this treatment, with a further six isolates classified as 'less sensitive'. All 280 isolates were fully sensitive to ceftriaxone.

TREATMENT GUIDELINES

The current recommended therapy for uncomplicated genital gonococcal infection in Victoria is:

- ciprofloxacin 500 milligrams orally as a single dose, OR

- ceftriaxone 250 milligrams IM (with 1 per cent lignocaine) as a single dose, OR
- spectinomycin 2 grams IM as a single dose.

Each should be taken with doxycycline 100 milligrams orally, 12-hourly for 10 days for co-existing chlamydial infection.

Ciprofloxacin should not be used for infection acquired overseas, during pregnancy, or by children or adolescents. Patients with pharyngeal or anorectal infection should be given ceftriaxone 250 milligrams IM as a single dose.

Disseminated gonococcal infection, pelvic inflammatory disease and epididymitis require more intensive and prolonged treatment. Further information is available in *Antibiotic Guidelines 1998/1999 Edition 10* or the Venereology Society of Victoria's *National Management Guidelines for Sexually Transmissible Diseases and Genital Infections 1997*.

The Microbiological Diagnostic Unit, University of Melbourne collected the data for this report, and it also performs the antibiotic sensitivity testing. The unit receives epidemiological data from both the diagnosing doctor and the contact tracers from the Department of Human Services.

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Surveillance Briefs

The Department of Human Services receives notifications of infectious diseases from medical practitioners and laboratories. These notifications prompt investigation and action to control infectious diseases in Victoria. For some diseases, investigation is instigated on the basis of clinical suspicion in the absence of laboratory confirmation.

This section includes a summary of infectious disease notifications received until 30 September 1998 and historical comparisons at both the State and Regional level (tables 4 and 5). Summary data at local government level can be obtained by contacting Ross Andrews, Disease Control Victoria, Department of Human Services (03 9637 4121). The 1997 and 1998 data are currently under review and may be subject to change. Data for notifications of hepatitis B and hepatitis C are incomplete and have not been included in this report.

MENINGOCOCCAL INFECTION

Notifications of meningococcal infection have increased in recent months, in keeping with the usual seasonal variation (figure 1). There were 21 apparant sporadic cases reported with onset of symptoms in August or September. Overall, the number of cases reported this year remains fewer than for the same period in 1997 (table 4). As in previous years, cases have predominantly been among children younger than five years of age with a second peak among adolescents and young adults. Of the 48 cases notified this year, 20 have been younger then 5 years of age (including 12 aged under one year) and 12 have been aged 15 to 24 years. Of those meningococci which were typable, 12 were Group B and seven Group C. There have been 4 deaths from meningococcal infection this year: three infants aged 3 months, 10 months and 16 months respectively (one Group B, two not typed); and a 32 year old female (Group C).

Information on the management of contacts and prophylaxis was included in VIDB 1998;1:13.

ENHANCED PERTUSSIS SURVEILLANCE

Although pertussis (whooping cough) notifications have continued to decline, we expect the number of notifications to increase over the spring/summer months (figure 2).

The Department of Human Services is currently undertaking an enhanced pertussis surveillance program and is keen to obtain nasopharyngeal aspirates from patients with suspected pertussis as early in the illness as possible. At present as serological tests for the diagnosis of pertussis are not standardised, nasopharyngeal culture is the preferred method of diagnosis. Obtaining nasopharyngeal aspirates is often difficult because the time between onset of cough and diagnosis may be several weeks, by which time the organism will not be present in the nasopharynx.

The Department currently offers a free service within the metropolitan Melbourne where pertussis surveillance nurses can arrange nasopharyngeal aspirates to be taken (contact Annette Clancy or Sue Thorpe on 9345 6664). The criteria for referral include:

- Cough for less than one month.
- Less than 3 days of erythromycin, Rulide or Bactrim therapy (preferably no antibiotic therapy) and
- Less than 14 days of another antibiotic

Further information about the project can be obtained by contacting Ross Andrews, Disease Control Victoria, 9637 4121.

Figure 1: Meningococcal Infection Notifications
By Month of Onset, Victoria, January 1993 to September 1998

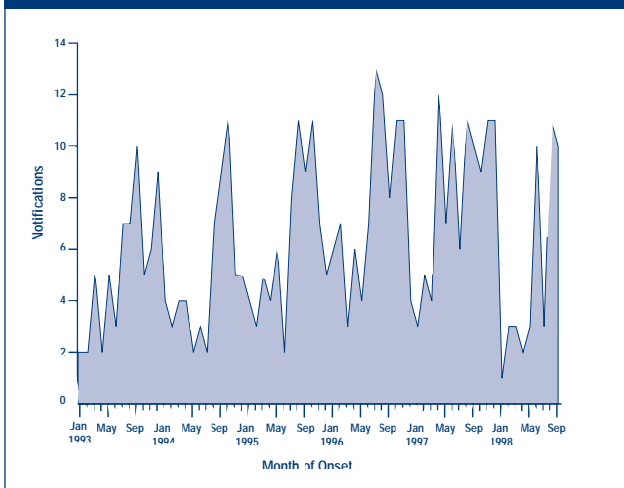
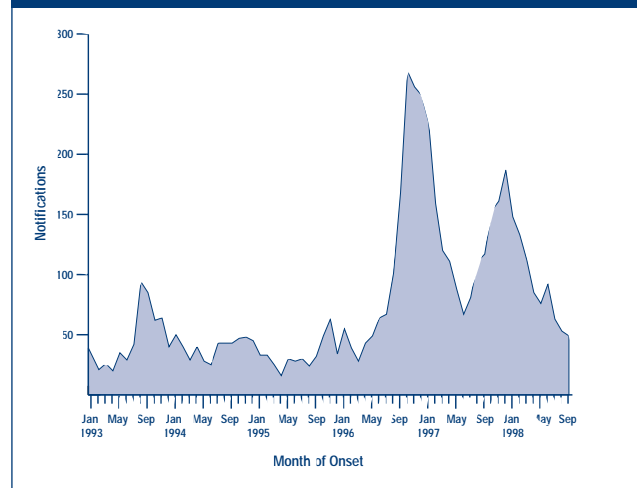


Figure 2: Pertussis Notifications
By Month of Onset, Victoria, January 1993 to September 1998



ENTERIC DISEASES

Shigellosis - a travel experience?

Shigellosis is an acute bacterial disease characterised by diarrhoea, fever, nausea, vomiting and abdominal pain. Notifications of shigellosis are 72% higher than for the same period last year and have already exceeded the 1997 total (table 4). Notifications peaked in January and now appear to be declining (figure 3). Although most cases were resident in the Eastern and Southern Metropolitan Region (table 4), cases have been of varying serotypes and no epidemiological links have been established. The cases ranged from 10 months to 79 years of age, most (72, 79 per cent) were aged 18 years or more. A history of recent overseas travel was reported for 22 cases (24 per cent).

Campylobacteriosis increases

Campylobacter infections are the most commonly notified cause of bacterial diarrhoea in Victoria. Although total notifications for the year to date are lower than the same period last year (table 4), notifications of campylobacteriosis have increased in the last quarter (figure 4). There were 947 notifications received between from July - September 1998. These cases resided in both rural and metropolitan areas, with the majority (208, 22 per cent) younger than 5 years of age (including 64 aged under one year). This is a typical pattern for campylobacteriosis.

Salmonellosis expected to increase

Notifications of salmonellosis usually increase over the spring/summer months. There have been 81 notifications of salmonellosis with onset of symptoms in September, which is a slight increase on previous months (figure 5).

INFLUENZA SURVEILLANCE

Sentinel General Practices

A network of metropolitan and rural sentinel general practitioners provided consultation rates for patients with influenza-like symptoms as a proportion of the number of patients seen each week between April and September of this year. In addition, GPs also sent nose and throat swabs and an acute serum specimen to the Victorian Infectious Diseases Reference Laboratory. Convalescent serum samples were collected by either the GP or a public health nurse. The collection of convalescent samples is still in progress.

Antibody response to influenza A and B was estimated for both acute and convalescent samples and immunofluorescence was performed on the nasal and pharyngeal specimens before they were cultured for influenza and other viruses. A diagnosis of influenza was considered to be laboratory confirmed if any of the laboratory tests was positive for influenza.

Laboratory Isolates

In addition to data from general practitioners, laboratories at the Royal Children's Hospital and the Monash Medical Centre provided a record of their influenza identifications over the same period.

Figure 3: Shigella Notifications
By Month of Onset, January 1993 to September 1998

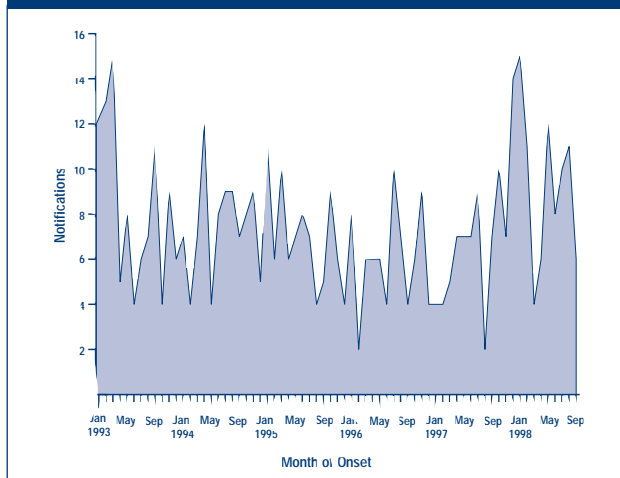


Figure 4: Campylobacteriosis Notifications
By Month of Onset, January 1993 to September 1998

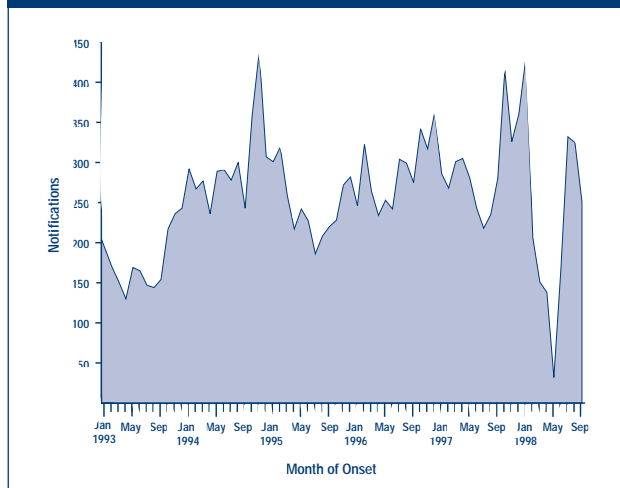


Figure 5: Salmonellosis Notifications
By Month of Onset, Victoria, January 1993 to September 1998

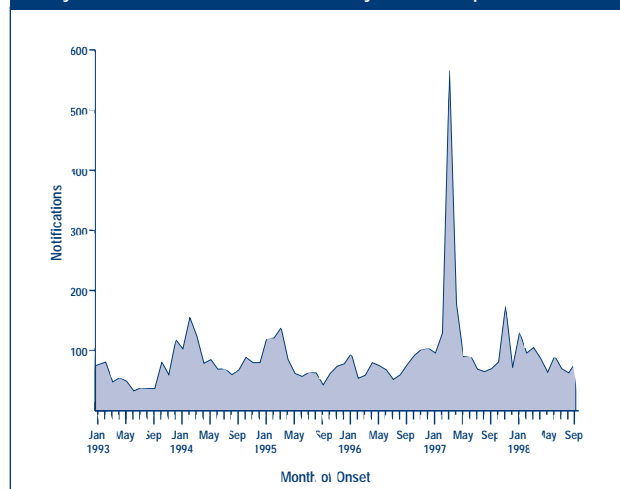


Figure 6: Influenza Consultation Rates Reported Through Sentinel General Practitioners-By Fortnight and Year, Victoria 1997-98

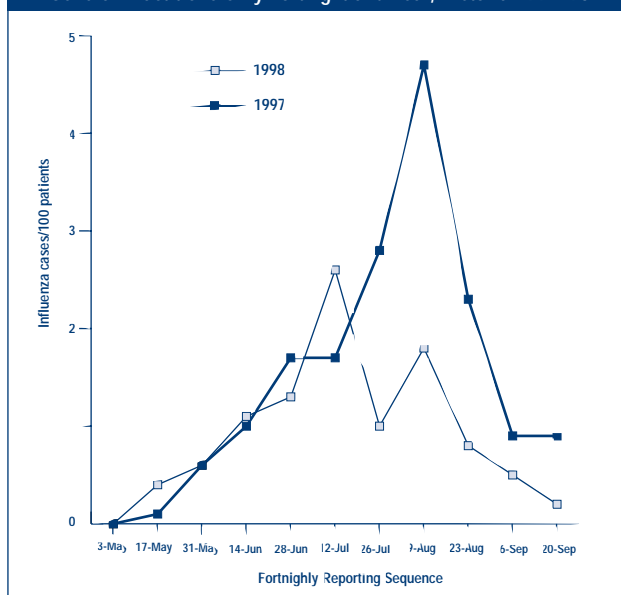
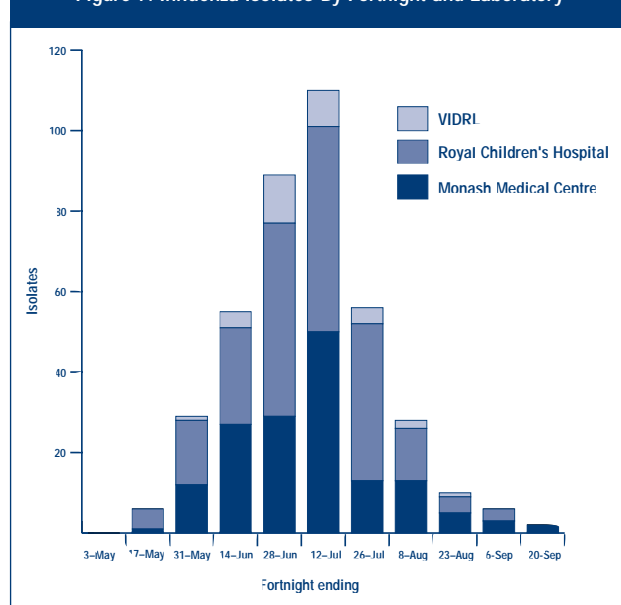


Figure 7: Influenza Isolates-By Fortnight and Laboratory



Results

Figure 6 illustrates the general practitioner consultation rate data, comparing the current year with 1997. In 1998 the influenza season peaked earlier and at a lower level than in 1997. The pattern of the consultation rate is very similar to that from the laboratories (figure 7), although the laboratory based surveillance includes much younger patients than the general practice surveillance. The second small peak seen in GP surveillance is not mirrored in the laboratory surveillance.

As expected the 1998 influenza season was predominantly influenza A (Sydney-like). Over the past two decades the

pattern of influenza epidemics in Victoria has been for influenza A to predominate in one year, as has occurred in 1998, with the alternate years characterised by the co-circulation of influenza A and B, as was the case in 1997. In 1999 we expect both influenza A and B to be circulating.

More than 400 patients satisfying the criteria for the clinical diagnosis of influenza were notified as part of the sentinel surveillance program. They ranged in age from less than one year old to 97 years, but were predominantly adults from their late twenties to their fifties. The relatively small number of people aged over 65 years (n=36) may reflect the success of the influenza vaccination campaign. There was no significant difference in the number of male and female patients notified.

109 patients were included in laboratory surveillance. Of these, 43 (39%) have been confirmed as having had influenza (table 1). If only those patients for whom complete data are available are considered, then the clinical suspicion of influenza was confirmed in 43/84 (51%) cases.

Table 1: Laboratory Confirmed Influenza Cases-By Method Of Diagnosis, Victoria 1998

Method of Diagnosis	No of Cases
Culture Only	6
Immunofluorescence Only	8
Serology Only	18
Any Combination	11
Total Influenza	43

As expected, vaccination rates were highest in patients aged over 65 years (71%). The vaccination rate in all those less than 65 years old was only 7%. Four patients had viruses other than influenza, a rhinovirus, adenovirus, a respiratory syncytial virus (RSV) and an enterovirus.

A detailed report of the surveillance program will be available once all remaining convalescent sera are collected and tested.

HIV/AIDS SURVEILLANCE

From July 1 1998, the HIV/AIDS Surveillance Program will report the number of HIV and AIDS notifications on the basis of date of notification rather than date of diagnosis. This has contributed to the drop in HIV notifications for this quarter.

There were 12 males and one female notified with HIV during this last quarter (table 2). While the drop in numbers can be attributed in part to the change in our reporting methods, the numbers have been generally down relative to last year. For the 12 months from 1 October 1997 to 30 September 1998, there were 121 HIV notifications compared with 188 notifications for 1997. Men reporting male sexual contact still make up the majority of notifications accounting for over 75% of notifications during the last 12 months.

Of the 8 men notified with AIDS during this quarter, 6 reported male to male sexual contact as their exposure risk (table 3). During the 12 months between 1 October 1997 and 30 September 1998, there were 49 males and 2 females diagnosed with AIDS. These numbers are low compared with the 61 notifications for 1997. Male to male sexual contact is still the predominant risk factor accounting for 65% of notifications in the last 12 months.

GONORRHOEA INFECTIONS

Rhodes *et al* (page 31) have reported an increase in notifications of gonorrhoea infection in Victoria. A similar increase in isolates of *Neisseria gonorrhoeae* has also been noted by the Bacteriology department at the Victorian Infectious Diseases Reference Laboratory (VIDRL). This increase is demonstrated by the comparison of positive cultures seen at VIDRL in 1997 and 1998 by month (figure 8). There has been no alteration in method of detection used by VIDRL during this period and samples have been received from the same clinics.

From January-September 1998, there were 95 culture positive *N.gonorrhoeae* at VIDRL. All isolates were from male patients. Twenty-four of the isolates were from rectal sites, not always in combination with other sites. Three isolates were grown from a throat swab only.

There were two beta-lactamase producing isolates. Nine percent (11/125) were resistant to penicillin by chromosomally mediated mechanisms, with Minimal Inhibitory Concentrations of at least 1.0 ug/mL (performed by MDU).

Eight of the culture positive individuals had coexisting infections with various other pathogens including *Chlamydia trachomatis*, Hepatitis B and HIV. Seven people had had previous episodes of *Neisseria gonorrhoeae* identified on culture at VIDRL.

There was a single case of urethritis caused by *Neisseria meningitidis* during this period.

Whilst the VIDRL data are a subset of that reported by Rhodes *et al.*, a similar trend is evident. The data highlights that clinics which VIDRL serves can be effective sentinel clinics to monitor the incidence of gonorrhoea infection in Victoria.

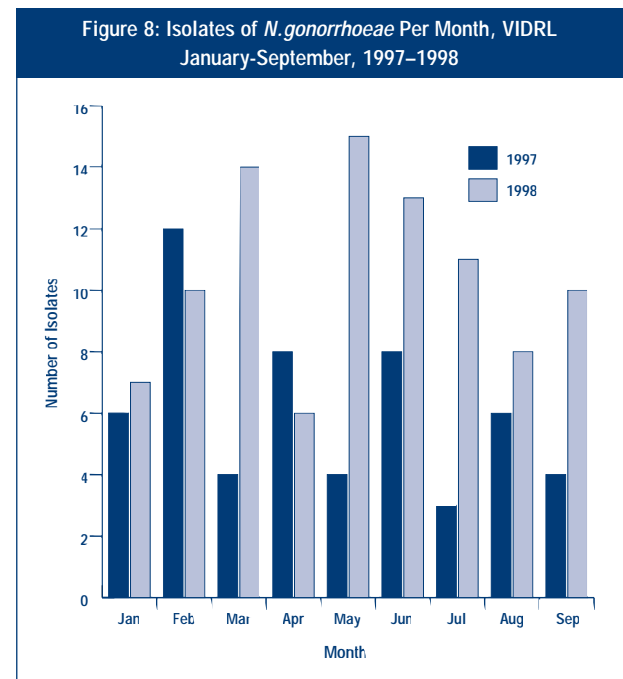


Table 2: Number of cases of notified HIV by exposure category July 1 1998 to September 30 1998, October 1 1997 to September 30 1998 and cumulative to September 30 1998, Victoria

Exposure Category	Cases Diagnosed July 98-Sept 98		Cases Diagnosed Oct 97- Sept 98		Cumulative Diagnoses to end Sept 1998		
	Male	Female	Male	Female	Male	Female	Total*
Male homo/bisexual	12	-	92	-	3111	-	3124
Male homo/bisexual & IDU	0	-	6	-	174	-	177
IDU	0	0	3	1	98	35	135
Heterosexual	0	1	11	3	124	115	239
Person from specified country	0	0	1	1	44	21	65
Haemophilia/related disorder	0	0	1	0	99	1	100
Transfusion recipient	0	0	0	0	19	13	32
Other	0	0	2	0	6	9	15
Under investigation	0	0	0	0	4	0	4
Unavailable	0	0	0	0	90	3	115
Total	12	1	116	5	3769	197	*4006

* Includes 14 cases whose sex is given as transsexual and 26 cases for whom no gender is reported

Table 3: Number of cases of notified AIDS by exposure category July 1 1998 to September 30 1998, October 1 1997 to September 30 1998 and cumulative to September 30 1998, Victoria

Exposure Category	Cases Diagnosed July 98-Sept 98		Cases Diagnosed Oct 97-Sept 98		Cumulative Diagnoses to end Sept 1998		
	Male	Female	Male	Female	Male	Female	Total*
Male homo/bisexual	6	-	32	-	1428	-	1431
Male homo/bisexual & IDU	1	-	2	-	89	-	91
IDU	0	0	0	0	15	9	24
Heterosexual	0	0	10	2	51	44	95
Person from specified country	0	0	1	0	11	6	17
Haemophilia/related disorder	0	0	0	0	34	1	35
Transfusion recipient	0	0	0	0	7	4	11
Other	0	0	2	0	0	1	1
Under investigation	1	0	2	0	2	2	4
Unavailable	0	0	0	0	13	0	13
Total	8	0	49	2	1650	67	*1722

* Includes 5 people for whom gender is given as transsexual

Table 4: Notifications of Infectious Diseases by Department of Human Services Region Victoria, 1 January to 30 September 1998 and Historical Comparisons

DISEASE	Barwon South-Western		Gippsland		Grampians		Hume		Loddon Mallee		Eastern Metropolitan		Northern Metropolitan		Southern Metropolitan		Western Metropolitan		Unknown		Victoria		
	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1997 total
Enteric Diseases																							
Amoebiasis	1	0	1	1	1	0	1	1	0	0	13	7	12	15	19	20	12	11	3	2	63	57	76
Campylobacteriosis	117	111	120	128	51	67	106	118	95	94	467	555	349	341	490	643	231	314	40	56	2066	2427	3612
Cholera	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1
Food/Water Borne Illness																							
– cryptosporidiosis	1	0	14	8	1	0	12	0	5	1	37	1	44	0	71	2	15	0	6	0	206	12	15
– other	3	2	1	4	2	0	2	1	1	1	13	40	9	7	14	85	5	4	2	4	52	148	189
Giardiasis	50	58	27	43	33	39	36	43	36	24	189	180	127	163	188	186	98	90	18	20	802	846	1071
Hepatitis A	1	6	14	2	3	4	3	36	30	40	33	47	17	66	23	69	18	22	4	9	146	301	363
Listeriosis	0	0	0	0	0	0	0	1	1	0	1	4	3	1	2	3	1	4	0	0	8	13	15
Paratyphoid	0	0	0	0	0	0	0	0	0	0	2	2	0	1	2	2	0	0	0	0	4	5	6
Salmonellosis	55	49	40	48	25	25	31	40	48	41	145	329	153	143	186	518	98	151	39	24	820	1368	1690
Shigellosis	3	1	2	0	1	0	1	3	2	7	27	7	16	8	27	14	11	9	1	4	91	53	79
Typhoid	0	0	0	0	0	0	0	1	0	0	1	5	4	6	0	2	4	0	0	0	9	14	16
Yersiniosis	1	1	0	1	1	1	1	1	0	0	7	2	2	1	5	3	6	2	1	1	24	13	15
Vaccine Preventable Diseases																							
<i>Haemophilus Influezae</i>																							
type b Infections	0	0	0	1	0	0	2	0	0	0	0	1	2	0	1	1	0	0	0	0	5	3	6
Measles	6	1	1	4	0	3	0	5	1	2	9	7	3	17	8	5	1	16	1	4	30	64	91
Mumps	0	0	2	2	1	3	3	10	2	2	4	13	9	9	8	10	7	5	0	1	36	55	66
Pertussis	34	38	212	93	25	31	52	57	65	138	145	240	96	248	224	232	84	126	23	24	960	1227	1679
Rubella	5	11	8	2	0	13	9	17	3	44	40	46	28	45	39	54	19	23	6	7	157	262	371
Tetanus	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	1	1	1
Vector Borne Diseases																							
Arboviral Infections																							
– Flavivirus	0	0	1	1	2	0	2	0	2	0	3	2	1	1	5	2	1	0	1	0	18	6	6
– Barmah Forest Virus	2	0	7	12	0	0	2	5	0	21	1	1	0	0	0	0	0	0	2	2	14	41	43
– Ross River Virus	3	43	6	10	5	112	1	139	6	466	3	70	2	54	4	65	0	21	1	22	31	1002	1017
– Not further specified	0	0	7	2	0	0	3	8	0	22	0	4	2	3	0	1	0	1	0	1	12	42	46
Malaria	0	1	1	2	1	2	6	4	1	0	16	15	15	15	20	21	3	8	4	8	67	76	90
Zoonoses																							
Brucellosis	0	0	0	0	2	1	0	0	0	0	1	0	0	0	1	0	0	0	0	0	4	1	3
Hydatid Disease	0	1	1	0	1	0	0	1	0	0	5	4	3	5	3	2	3	4	0	0	16	17	31
Leptospirosis	3	3	5	4	0	0	1	4	0	0	0	0	0	0	0	1	0	0	1	0	9	13	27
Psittacosis	0	4	0	5	1	1	2	1	3	1	4	9	5	4	5	5	3	5	0	0	23	35	39
Q Fever	3	0	1	1	2	2	4	4	2	4	1	0	1	0	1	3	2	4	0	0	17	18	24
Taeniasis	0	0	0	0	0	0	0	0	0	0	0	0	2	2	0	0	1	3	0	0	3	5	5
Typhus	0	0	0	0	1	0	0	1	1	0	0	0	1	0	0	1	0	0	0	0	3	2	2
Other Infectious Notifiable Diseases																							
Hepatitis (viral, unspecified)	0	0	0	0	0	0	0	0	0	0	0	0	1	2	1	0	0	0	0	1	2	3	4
Legionellosis	3	2	0	1	0	1	0	0	0	0	3	2	5	5	5	9	17	6	1	0	34	26	30
Meningococcal Infection	3	4	4	5	1	1	6	1	2	6	11	12	4	14	10	18	3	7	3	1	47	69	99
Tuberculosis	3	5	3	3	4	4	4	2	2	0	41	23	37	55	48	50	62	33	3	7	207	182	282
Total	395	503	572	496	210	390	381	594	425	1043	1794	2342	1566	2563	2278	3245	1259	1756	641	620	9469	13404	17969

Notes

1 Data for notifications of hepatitis B and hepatitis C have not been included due to problems in transferring the data to a new database

2 The data are preliminary figures only and may be subject to revision

Table 5: Rates Per 100,000 Population of Infectious Diseases by Department of Human Services Region Victoria, 1 January to 30 September 1998 and Historical Comparisons

DISEASE	Barwon South-Western		Gippsland		Grampians		Hume		Loddon Mallee		Eastern Metropolitan		Northern Metropolitan		Southern Metropolitan		Western Metropolitan		Unknown		Victoria		
	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	1998 ytd	1997 ytd	total
Enteric Diseases																							
Amoebiasis	0.3	0.0	0.4	0.4	0.5	0.0	0.4	0.4	0.0	0.0	1.4	0.7	1.6	2.0	1.8	1.9	2.1	1.9	0.10	0.0	1.4	1.2	1.7
Campylobacteriosis	35.8	33.9	51.3	54.7	25.3	33.3	44.4	49.4	33.8	33.4	49.5	58.8	47.1	46.0	46.0	60.4	40.4	55.0	0.9	1.2	44.9	52.7	78.4
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Food/Water Borne Illness – cryptosporidiosis	0.3	0.0	6.0	3.4	0.5	0.0	5.0	0.0	1.8	0.4	3.9	0.1	5.9	0.0	6.7	0.2	2.6	0.0	0.1	0.0	4.5	0.3	0.3
– other	0.9	0.6	0.4	1.7	1.0	0.0	0.8	0.4	0.4	0.4	1.4	4.2	1.2	0.9	1.3	8.0	0.9	0.7	0.0	0.1	1.1	3.2	4.1
Giardiasis	15.3	17.7	11.5	18.4	16.4	19.4	15.1	18.0	12.8	8.5	20.0	19.1	17.1	22.0	17.6	17.5	17.2	15.8	0.4	0.4	17.4	18.4	23.3
Hepatitis A	0.3	1.8	6.0	0.9	1.5	2.0	1.3	15.1	10.7	14.2	3.5	5.0	2.3	8.9	2.2	6.5	3.2	3.9	0.1	0.2	3.2	6.5	7.9
Listeriosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0	0.1	0.4	0.4	0.1	0.2	0.3	0.2	0.7	0.0	0.0	0.2	0.3	0.3
Paratyphoid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0	0.1	0.2	0.2	0.0	0.0	0.0	0.0	0.1	0.1	0.1
Salmonellosis	16.8	15.0	17.1	20.5	12.4	12.4	13.0	16.8	17.1	14.6	15.4	34.8	20.6	19.3	17.5	48.6	17.2	26.4	0.8	0.5	17.8	29.7	36.7
Shigellosis	0.9	0.3	0.9	0.0	0.5	0.0	0.4	1.3	0.7	2.5	2.9	0.7	2.2	1.1	2.5	1.3	1.9	1.6	0.0	0.1	2.0	1.2	1.7
Typhoid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.5	0.5	0.8	0.0	0.2	0.7	0.0	0.0	0.0	0.2	0.3	0.3
Yersiniosis	0.3	0.3	0.0	0.4	0.5	0.5	0.4	0.4	0.0	0.0	0.7	0.2	0.3	0.1	0.5	0.3	1.1	0.4	0.0	0.0	0.5	0.3	0.3
Vaccine Preventable Diseases																							
Haemophilus <i>Influenzae</i> type b Infections	0.0	0.0	0.0	0.4	0.0	0.0	0.8	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.1
Measles	1.8	0.3	0.4	1.7	0.0	1.5	0.0	2.1	0.4	0.7	1.0	0.7	0.4	2.3	0.8	0.5	0.2	2.8	0.0	0.1	0.7	1.4	2.0
Mumps	0.0	0.0	0.9	0.9	0.5	1.5	1.3	4.2	0.7	0.7	0.4	1.4	1.2	1.2	0.8	0.9	1.2	0.9	0.0	0.0	0.8	1.2	1.4
Pertussis	10.4	11.6	90.6	39.7	12.4	15.4	21.8	23.9	23.1	49.0	15.4	25.4	12.9	33.4	21.0	21.8	14.7	22.1	0.5	0.5	20.8	26.6	36.5
Rubella	1.5	3.4	3.4	0.9	0.0	6.5	3.8	7.1	1.1	15.6	4.2	4.9	3.8	6.1	3.7	5.1	3.3	4.0	0.1	0.2	3.4	5.7	8.1
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0
Vector Borne Diseases																							
Arboviral Infections																							
– Flavivirus	0.0	0.0	0.4	0.4	1.0	0.0	0.8	0.0	0.7	0.0	0.3	0.2	0.1	0.1	0.5	0.2	0.2	0.0	0.0	0.0	0.4	0.1	0.1
– Barmah Forest Virus	0.6	0.0	3.0	5.1	0.0	0.0	0.8	2.1	0.0	7.5	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.9	0.9
– Ross River Virus	0.9	13.1	2.6	4.3	2.5	55.6	0.4	58.2	2.1	165.6	0.3	7.4	0.3	7.3	0.4	6.1	0.0	3.7	0.0	0.5	0.7	21.8	22.1
– Not further specified	0.0	0.0	3.0	0.9	0.0	0.0	1.3	3.4	0.0	7.8	0.0	0.4	0.3	0.4	0.0	0.1	0.0	0.2	0.0	0.0	0.3	0.9	1.0
Malaria	0.0	0.3	0.4	0.9	0.5	1.0	2.5	1.7	0.4	0.0	1.7	1.6	2.0	2.0	1.9	2.0	0.5	1.4	0.1	0.2	1.5	1.7	2.0
Zoonoses																							
Brucellosis	0.0	0.0	0.0	0.0	1.0	0.5	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Hydatid Disease	0.0	0.3	0.4	0.0	0.5	0.0	0.0	0.4	0.0	0.0	0.5	0.4	0.4	0.7	0.3	0.2	0.5	0.7	0.0	0.0	0.3	0.4	0.7
Leptospirosis	0.9	0.9	2.1	1.7	0.0	0.0	0.4	1.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.3	0.6
Psittacosis	0.0	1.2	0.0	2.1	0.5	0.5	0.8	0.4	1.1	0.4	0.4	1.0	0.7	0.5	0.5	0.5	0.5	0.9	0.0	0.0	0.5	0.8	0.8
Q Fever	0.9	0.0	0.4	0.4	1.0	1.0	1.7	1.7	0.7	1.4	0.1	0.0	0.1	0.0	0.1	0.3	0.4	0.7	0.0	0.0	0.4	0.4	0.5
Taeniasis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.3	0.0	0.0	0.2	0.5	0.0	0.0	0.1	0.1	0.1
Typhus	0.0	0.0	0.0	0.0	0.5	0.0	0.0	0.4	0.4	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Other Infectious Notifiable Diseases																							
Hepatitis (viral, unspecified)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1
Legionellosis	0.9	0.6	0.0	0.4	0.0	0.5	0.0	0.0	0.0	0.0	0.3	0.2	0.7	0.7	0.5	0.8	3.0	1.1	0.0	0.0	0.7	0.6	0.7
Meningococcal Infection	0.9	1.2	1.7	2.1	0.5	0.5	2.5	0.4	0.7	2.1	1.2	1.3	0.5	1.9	0.9	1.7	0.5	1.2	0.1	0.0	1.0	1.5	2.1
Tuberculosis	0.9	1.5	1.3	1.3	2.0	2.0	1.7	0.8	0.7	0.0	4.3	2.4	5.0	7.4	4.5	4.7	10.9	5.8	0.1	0.2	4.5	4.0	6.1
Total	120.7	153.7	244.4	211.9	104.3	193.7	159.6	248.8	151.1	370.7	190.0	248.0	211.2	345.7	213.8	304.6	220.4	307.3	13.9	13.5	205.6	291.1	390.2

Notes

- 1 Data for notifications of hepatitis B and hepatitis C have not been included due to problems in transferring the data to a new database
- 2 The data are preliminary figures only and may be subject to revision

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