

Victorian Infectious Diseases Bulletin

Bairnsdale Ulcer Goes Global

Paul Johnson, Melbourne *Mycobacterium ulcerans* Study Team

Fifty years ago the Australians McCallum, Buckle, Sissons and Tolhurst were the first to describe a new mycobacterial infection of humans—Bairnsdale ulcer. This chronic, progressive necrotising skin infection eventually heals if untreated, but sufferers are often left with functional and cosmetic deformities. Drug therapy is ineffective, so the principles of management are to aim for an early diagnosis and to surgically remove ulcers before they become too large.

Buruli ulcer, the African version of the disease, is becoming more significant than leprosy in several West African countries. The situation is now so serious that the World Health Organization (WHO) has just announced the creation of the Global Buruli Ulcer Initiative to focus attention on the disease.

CLUSTERING

No one yet understands how people become exposed to *Mycobacterium ulcerans* (MU), the bacteria that causes Bairnsdale ulcer and Buruli ulcer. However, outbreaks always seem to occur in clusters that are geographically localised.

Current thinking is that MU is transmitted from an unknown environmental niche—probably microenvironments within swamps, rivers or lakes—to humans (and occasionally animals). But no one has successfully isolated MU from the environment, despite the relative ease with which it may be cultured from the undermined edges of ulcers.

Australia has scattered pockets of endemicity. An unsolved mystery of MU is that temperate Victoria and tropical north Queensland are two major foci, yet New South Wales, South Australia and most of Western Australia remain disease free.

CHANGING PATTERNS

The epidemiology of MU in Victoria changed abruptly in the 1990s. Following the first descriptions in the State, all infections for the next 40 years occurred in people with a history of contact with east Gippsland. Then in 1982 and 1983, ulcers affected three people in the Tooradin-

Warnete region at the north end of Westernport Bay, implying that MU had either jumped at least 200 kilometres westward or long lain dormant until some unknown factor caused it to reawaken.

Nothing happened for seven years until MU reappeared even nearer to Melbourne. A new focus in the Frankston–Langwarrin region became active in 1990. Despite being 40 kilometres to the south east of the GPO, the focus is now very much in suburban Melbourne with at least 12 infections reported over eight years.

A spectacular cluster of 29 cases occurred in a tiny region in east Cowes on Phillip Island in 1993–94. Isolated cases have also been reported from Crib Point, Cranbourne, Hoppers Crossing, The Gurdies, Mornington, and even Bendigo.

MYCOBACTERIUM ULCERANS IN THE ENVIRONMENT

No one really knows how the infection interacts with the environment. However, the outbreak at Phillip Island provided a significant opportunity to learn more. We proposed on epidemiological grounds that common exposure to a golf course's recycled water irrigation system and/or a nearby swamp may explain these cases.

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Human
Services



Peoplefirst

Alterations to the irrigation system and drainage of the swamp preceded an abrupt reduction in new cases. Attempting to prove the hypothesis, the Melbourne MU Study Team developed a polymerase chain reaction (PCR) based assay for testing environmental samples. Using this new tool, we were able to show that MU was present in the irrigation system and the swamp but not in other samples collected on Phillip Island and elsewhere.

This is the first time that anyone in the world has been able to demonstrate MU in the environment, despite the 'smoking gun' epidemiology which indicates that it must be there.

PCR FOR DIAGNOSIS

Using the same PCR target, we have also developed a rapid MU diagnostic test which appears to be completely specific and highly sensitive. A definitive diagnosis can now be made within 24 hours—a significant advance compared with the 8–12 weeks it may take by conventional methods to identify MU. We have used the PCR to diagnose 23 new cases of MU infection (21 people, one possum and one alpaca!) in the past two years.

Phillip Island is now famous among MU experts worldwide. The Second International *Mycobacterium ulcerans* Conference was held in Melbourne in April, with the conference dinner at a spectacular Phillip Island venue overlooking rolling Bass Strait surf. Dr Kingsley Asiedu, Director of the Global Buruli Ulcer Initiative, announced at the conference that Melbourne will be one of three world reference centres for MU, in recognition of the study team's work.

WHO BURULI ULCER TASKFORCE

John Hayman and Paul Johnson (members of the Melbourne MU Study Team) have also been appointed as advisors to the WHO Buruli Ulcer Taskforce, and will travel to Cote d'Ivoire with other members of our group in July to attend the launch of the Global Buruli Ulcer Initiative. West Africa is a long way from Phillip Island, and it is not at all clear that the progress we have made here will translate into a useful public health intervention under such different circumstances.

In some west African valleys, up to 20 per cent of villagers have ulcers, and 80 per cent of these people will be left with a significant deformity. These stark facts challenge us to hope that we will be able to make at least a small contribution in Africa, and spur us to continue the 50 year Australian tradition of research into *Mycobacterium ulcerans*.

The Melbourne Mycobacterium ulcerans Study Team is: Paul Johnson, John Hayman, Tim Stinear, Frances Oppedisano, Bruce Ross, Lui Marino, Roy Robbins-Browne, Mark Veitch, Aina Sievers, Paul Flood, Jason Harney, Travis Gooding, Andrew Kemp, Dianne Campbell, David Leslie, Christine Drummond and others. We gratefully acknowledge the financial support of the Victorian Department of Human Services and the Royal Children's Hospital Research Institute.

Infectious Diseases News

UPDATED UPDATE

The *Victorian Infectious Diseases Bulletin* replaces *Update* and will continue on a quarterly basis. It is hoped that this publication will be a forum for exchange of related information about infectious diseases, particularly as they affect Victoria.

The bulletin is free and the mailing list includes medical practitioners, laboratory personnel, maternal and child health nurses, environmental health officers, infection control practitioners and many more interested readers. To obtain your copy of the bulletin, contribute or comment, see the back page.

BAIRNSDALE/BURULI ULCER—A WORLD FIRST

Australian researchers of *Mycobacterium ulcerans* (MU) continue to make breakthroughs. First identified in Australia, MU causes a chronic skin infection referred to as Bairnsdale ulcer. It is now more widely known as Buruli ulcer, the West African version of the same disease. In this issue, Paul Johnson reports on the development of a PCR assay for identifying MU in the environment and the extension of this assay as a rapid diagnostic test for patients.

TACKLING MEASLES IN AUSTRALIA

An enhanced measles control campaign has now commenced, but Mike Catton and Heath Kelly (page 10) stress the critical role of laboratory testing in the diagnosis of measles. General practitioners should encourage laboratory confirmation for patients in whom measles is suspected.

PLANS TO MAKE HUS/VTEC NOTIFIABLE

Regulations have been proposed to make haemolytic uraemic syndrome (HUS) and verotoxin-producing *Escherichia coli* (VTEC) infection notifiable in Victoria. VTEC are widespread in animals that are harvested for human consumption and may cause serious and potentially preventable illness in humans. To obtain a copy of the proposed regulations and the Regulatory Impact Statement, contact the Infectious Diseases Unit, Department of Human Services (03 9637 4121).

Karl Bettelheim (page 6) clarifies the difference between VTEC and enterohaemorrhagic *E. coli* (EHEC) and stresses the importance of testing for non-0157 VTEC as well as 0157 VTEC.

CRYPTOSPORIDIOSIS IN SWIMMING POOLS

Martyn Kirk et al. (on this page) report the results of active surveillance initiated by the Department of Human Services to identify cases of cryptosporidiosis. The surveillance identified 125 cases of cryptosporidiosis in a two-month period, including several clusters associated with swimming pools.

NEW LOCATION FOR THE VIDRL

The Victorian Infectious Diseases Reference Laboratory (VIDRL) began as the pathology service within the former Fairfield Hospital.

Now part of the North Western Health Care Network, VIDRL operates as a Statewide reference laboratory. VIDRL moved to its new premises, 10 Wreckyn Street, North Melbourne (03 9342 2600) on 4 May 1998.

CONFERENCE CALLS FOR IMPROVED SURVEILLANCE OF FOODBORNE DISEASE

The second national Public Health Association food safety conference was held in Brisbane on 28–29 May 1998. The conference considered the evidence for current and future action in preventing foodborne illness. A major issue was the need for improved surveillance and national reporting of foodborne illness.

Participants discussed the concepts of legal evidence and scientific evidence and their role in shaping actions. They also debated the evidence for the change to a HACCP-based system, the benefits and costs of this change, and the likely outcomes from different perspectives.

A wide variety of persons attended the conference, including public health practitioners, regulators, scientists, industry groups and consumer representatives. Organisers will soon circulate the conference resolutions and also forward them to the Public Health Association for further consideration.

Clusters of *Cryptosporidium* Infection in Victoria

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Cryptosporidiosis is a parasitic infection spread via the faecal–oral route. Health authorities linked recent outbreaks of cryptosporidiosis in other Australian States to public swimming pools. The infection is not a notifiable disease in Victoria, but active surveillance initiated by the Department of Human Services identified 125 cases of cryptosporidiosis between 9 March and 15 May 1998. Several clusters were identified, including some associated with swimming pools. We report the results of these investigations.

INTRODUCTION

Cryptosporidiosis is a parasitic infection which often causes profuse watery diarrhoea. Vomiting, fever and abdominal pain can also occur. *Cryptosporidium* oocysts—the infectious stage—are spread via the faecal–oral route. Infected patients may shed the oocysts in faeces at the beginning of their illness (1–12 days after exposure) until several weeks after symptoms cease.¹ There is no specific treatment for cryptosporidiosis other than rehydration when indicated. Diagnosis is by specialised staining and microscopy which should be specifically requested from pathology laboratories.

Outbreaks of cryptosporidiosis have been associated with municipal water supplies, infected animals, contaminated foods and child care centres.^{1,2} Recent outbreaks in Queensland, New South Wales and the Australian Capital Territory were linked to public swimming pools.³

Cryptosporidiosis is not a notifiable disease in Victoria. However, following the outbreaks reported in other Australian States, the Department of Human Services initiated active surveillance for cryptosporidiosis in Victoria. We identified several clusters, some of which were associated with swimming pools.

METHODS

During the week commencing 9 March 1998, Department of Human Services staff contacted the major pathology providers in Melbourne to ascertain if they had observed an increase in cases of *Cryptosporidium* infections in recent months. Following this, all Victorian pathology services were asked to report identification of *Cryptosporidium* from patient faeces as part of a voluntary surveillance scheme. The Department also sent a letter to general practitioners informing them about cryptosporidiosis and its association with swimming pools.

We defined a case as someone who had *Cryptosporidium* oocysts identified in faeces. All reported cases were contacted by telephone to ascertain personal details, clinical history, recent exposure to various sources of drinking water, attendance at child care centres and use of swimming pools. We reviewed the case series to identify common exposures.

Where two or more cases had used the same swimming pool facility within the incubation period of their illness, the Department contacted the local council environmental health officer and the manager of the swimming pool. Pool managers were advised to institute a series of precautionary actions (box).

Victorian council environmental health officers received guidelines for managing cryptosporidiosis to distribute to swimming pool managers. The guidelines were adapted from a Centers for Disease Control and Prevention fact sheet about *Cryptosporidium* (see http://www.cdc.gov/ncidod/dpd/pool_op.htm).

Standard instructions for the management of public swimming pools where two or more cases of cryptosporidiosis were epidemiologically linked

- Hyperchlorinate the pool with at least 14 milligrams of free chlorine per litre of water for at least 12 hours.
- Erect signs advising people to shower before using the pool, and to not swim if they had diarrhoea or a gastrointestinal illness in the previous week.
- Formulate a written policy for instances where people accidentally defecate in the pool.

Active surveillance was initiated in selected swimming pools with large clusters of cases, using a systematic sample of 50 people from each pool's swim school enrolment list. Telephone interviewers asked respondents if they had experienced gastrointestinal symptoms in the previous month and if they had swum in the pool. Those with diarrhoea were asked to supply a faecal specimen, which was transported to the Microbiological Diagnostic Unit at Melbourne University.

A Kinyoun acid fast stain and a haematoxylin stain was performed to identify *Cryptosporidium* oocysts and other parasites. Faecal samples were cultured for bacterial pathogens. The Victorian Infectious Diseases Reference Laboratory used electronmicroscopy to examine faecal specimens for viruses.

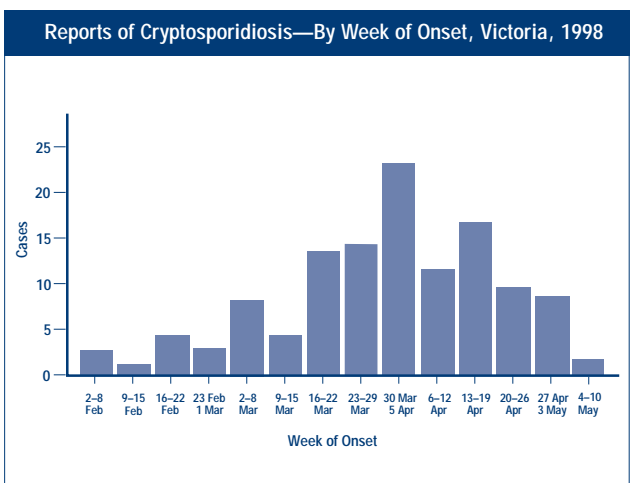
RESULTS

In early March, the Water Quality Study—a large community intervention trial conducted by the Cooperative Research Centre for Water Quality and Treatment—first indicated an increase in cases of cryptosporidiosis.⁴ There were nine cases from six families in the last month, compared with one case for the previous four months. Seven of the nine cases had used the same swimming pool in the incubation period of their illness.

A number of pathology laboratories subsequently reported an increase in diagnoses of *Cryptosporidium* infection. Laboratories and general practitioners had notified the Department's Infectious Diseases Unit of 125 cases of cryptosporidiosis by 15 May.

The majority of cases (105 of 125—that is, 84 per cent) developed symptoms in March or April (figure). Males and females were equally represented and the median age was 5 years (range 1–61 years). We detected several clusters in different postcodes, including seven clusters associated with swimming pools (table). One cluster of four cases was epidemiologically linked to a child care centre. We could not identify any epidemiological link for the remaining cases, some of which are still under investigation.

Cryptosporidium oocysts were identified in 13 (32 per cent) of 41 faecal specimens obtained from swimming pool patrons who reported gastroenteritis. *Enteromonas hominis*, *Blastocystis hominis* and *Chilomastix mesnili*—which are of doubtful significance as human pathogens—were also identified in the faeces of patients with *Cryptosporidium*. No bacterial or viral pathogens were identified from faeces.



Action Implemented at Public Swimming Pools Following Two or More Epidemiologically Linked Cases of Cryptosporidiosis or Identification of *Cryptosporidium* from a Pool Water Sample, Victoria, 1998

Pool	Location	No. of Cases	Pool Water Test for <i>Cryptosporidium</i>	Pool Treatment
1	South eastern suburbs	19	Negative on three occasions	Hyperchlorination; change of filter media two weeks before investigation
2	Southern suburbs	13	No	Hyperchlorination on two occasions
3	Northern suburbs	13	Backwash water positive	Hyperchlorination; pool water drained; change of filter media
4	Eastern suburbs	2	No	Hyperchlorination
5	Eastern suburbs	2	No	Hyperchlorination
6	Northern suburbs	2	No	Hyperchlorination
7	Southern suburbs	2	No	Hyperchlorination
8	Western suburbs	1	Positive	Hyperchlorination

DISCUSSION

Given the absence of routine surveillance for cryptosporidiosis, the Water Quality Study provided an early indication of increased incidence in Victoria. That study's participants are encouraged to supply faecal specimens as soon as they have any gastrointestinal symptoms. Although the Water Quality Study investigates the contribution of drinking water to endemic gastroenteritis, routine testing of faecal specimens for *Cryptosporidium* detected the outbreak, which may have otherwise gone unnoticed.

Notified cases of cryptosporidiosis were predominantly young children and occurred in late summer and early autumn. This pattern was similar to that observed in other Australian States.⁵ There are no routine surveillance data against which to compare the reported incidence of cryptosporidiosis in Victoria, but these cases probably represent a typical seasonal pattern rather than an unusual outbreak.

A 1995 sentinel surveillance scheme for cryptosporidiosis identified 588 cases in Victoria between 1 January and 15 May—far more than for the same period in 1998 (personal communication, Mark Veitch, Microbiological Diagnostic Unit 1998). The sentinel data also showed the highest incidence in summer and identified several clusters in metropolitan Melbourne and rural Victoria.

Health agencies have reported similar epidemics in other States in previous years, but were unable to identify a definitive source.^{6,7} Prior to 1998, only one Australian swimming pool associated outbreak of cryptosporidiosis was reported in the literature.⁸ Our active surveillance identified 13 cases of cryptosporidiosis among 42 swimming pool patrons with gastroenteritis (32 per cent). This high proportion suggests that swimming pools may play an important role in the aetiology of *Cryptosporidium* infections.

Cryptosporidium oocysts are highly resistant to levels of chlorine normally present in swimming pools.^{8,9} This may make pools, particularly children's pools, a focus for *Cryptosporidium* infections. To prevent these infections, pool patrons should not swim while suffering from, and immediately following, a gastrointestinal illness. Toddlers

suffering from cryptosporidiosis should not swim in public swimming pools for four weeks after diarrhoea ceases, while adults may recommence swimming one week after diarrhoea ceases if they shower carefully before entering the pool. Swimming pool managers should endeavour to minimise the risk of pool contamination (box).

Australian State health authorities differ in their recommended advice about testing swimming pools for *Cryptosporidium* oocysts. Two tests for *Cryptosporidium* in water are commonly used in Australia: one relies on microscopy and the other uses polymerase chain reaction (PCR) technology.¹⁰

Microscopy does not indicate whether oocysts are viable, and the assay may be affected by chlorine. The PCR assay does detect viability, but does not indicate whether oocysts are infectious. Health agencies have difficulties interpreting the results of positive and negative results, and both tests can be expensive. Thus, the Department of Human Services has not routinely tested swimming pool water for *Cryptosporidium*.

Health agencies also differ in their recommended treatment for swimming pools associated with clusters of cryptosporidiosis. Some States recommend that pools drain all water, clean the facility and change the filter media. Others require pool managers to dose the water with chlorine dioxide.

The current treatment regime for Victorian swimming pools is hyperchlorination of the water. Some Victorian pool managers now hyperchlorinate their swimming pools each week, as a precautionary measure. This treatment is based on laboratory evidence of the concentration of chlorine for a set time that is required to destroy *Cryptosporidium* oocysts.⁹ We are unsure whether this information is applicable for *Cryptosporidium* in swimming pool water.

This report focused on clusters of cryptosporidiosis associated with swimming pools, but it is important to consider other modes of transmission when investigating reported cases—for example, we identified one cluster associated with a child care centre in a rural Victorian town.

To identify risk factors for *Cryptosporidium* infections the Department is collaborating with the Cooperative Research Centre for Water Quality and Treatment on a case control study.

Many uncertainties surround the epidemiology and management of *Cryptosporidium* infections. A national consensus conference is planned for later this year to clarify such issues and develop a research agenda.

ACKNOWLEDGMENTS

We thank Victorian environmental health officers, laboratories and general practitioners who assisted with the investigations.

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VTEC and EHEC: Verocytotoxigenic and Enterohaemorrhagic *E. coli*

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Research during the second half of the 1970s found that certain strains of *Escherichia coli*, isolated from patients with diarrhoea, produce a toxin or toxins capable of causing distinctive cytopathic effects on vero cells.¹ These organisms became known as verocytotoxigenic *E. coli* (VTEC) and human infections with VTEC have now been described throughout the world.^{2,3} However, the scientific community was first alerted to the importance of VTEC in 1982 following a report of two hamburger-linked outbreaks in the United States associated with *E. coli* 0157:H7 (considered a rare serotype at the time).⁴

VTEC VERSUS EHEC

The production of the verocytotoxins is an essential prerequisite for the organisms to cause human infection, but other virulence factors seem to be associated with these organisms—including specific adhesins and an unusual haemolysin. Not all pathogenic strains necessarily carry all these factors but they must carry at least some of them. Thus, while VTEC have been frequently isolated from the faeces of domestic animals (such as cattle and sheep) and from the dressed carcasses,⁵ EHEC are those strains shown to cause human infection. This adds to the problem because VTEC are widely dispersed in nature and probably have a role in rumen ecology but only some VTEC are EHEC.⁶

SEROTYPES AFFECTING HUMANS

Serotypes 0157:H7 and 0157:H– (the non-motile variant of 0157:H7) are most frequently reported from cases of human disease. Symptoms vary from mild diarrhoea to the haemolytic uraemic syndrome, which can be associated with high morbidity and mortality. The 0157 serogroup have some unusual features, particularly an inability to ferment sorbitol, making them comparatively

easy to isolate and characterise from patient specimens. Australian studies have shown other serotypes (especially 0111:H–) are present in this country,³ so it was no surprise when 0111:H– was the predominant strain of VTEC isolated in the first Australian outbreak which occurred in Adelaide in 1995. Other serotypes including strains of 0157:H7 and 0157:H– were also found.⁷

It is easier to isolate the 0157 VTEC than the non-0157 VTEC, but the latter should always be attempted so as to improve understanding of the epidemiology of these organisms. The South Australian outbreak, for example, could have been wrongly described as an 0157 rather than 0111:H– outbreak.⁸ Similarly, other internationally reported outbreaks might have been wrongly attributed to VTEC of serotype 0157:H7 if authorities had not attempted to isolate non-0157 VTEC.

The *E. coli* Reference Laboratory has operated at the Victorian Infectious Diseases Reference Laboratory for 10 years with a full set of *E. coli* O and H antisera, establishing a unique set of data on VTEC serotypes in Australia. Recently described tests for the production of enterohaemolysin may assist laboratories to isolate more non-0157 VTEC.⁹

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Responding to the Emergence of Vancomycin-Resistant Enterococci

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Vancomycin-resistant enterococci (VRE) are several species of the genus Enterococcus with acquired resistance to the glycopeptide antibiotic vancomycin. Since the late 1980s VRE have become an increasingly common cause of hospital-acquired infection in major medical centres in the northern hemisphere.

BACKGROUND

The two species of VRE of greatest clinical and epidemiological importance are *E. faecium* and *E. faecalis*. Several other species, particularly *E. gallinarum* and *E. casseliflavus*, have intrinsic resistance to vancomycin, but are less commonly associated with human infection and do not significantly contribute to epidemic or endemic nosocomial infection. Rather, they are important in the sense that these species must be rapidly distinguished from vancomycin-resistant strains of *E. faecium* and *E. faecalis*.

E. faecium and *E. faecalis* may cause urinary tract and surgical wound infections, bloodstream infections and endocarditis. The relatively low virulence of these pathogens and the availability of effective antibiotic therapy meant that enterococcal infection, while relatively common, did not pose a particular threat to the susceptible population until the late 1980s.

Why then have certain enterococci gained an unfortunate reputation as ‘superbugs’?

- Strains of *E. faecium* and *E. faecalis* have acquired resistance to vancomycin.
- These strains have occurred as endemic and epidemic nosocomial pathogens in health care facilities, particularly in the United States.
- Critically ill hospitalised patients appear most likely to acquire VRE as colonising or infecting pathogens, and are vulnerable to complications and death in association with VRE infections.
- Enterococci (including VRE) may acquire resistance to other antibiotics, leaving few, if any, therapeutic options for treating infections. Infection with beta-lactamase-producing VRE with acquired high-level gentamicin resistance may be practically untreatable.

Isolates of *E. faecium* and *E. faecalis* can be characterised as VanA, VanB or VanD phenotype by their specific pattern of resistance to the glycopeptide antibiotics vancomycin and teicoplanin. Corresponding genetic markers (VanA and VanB) can be identified by polymerase chain reaction (PCR). By contrast, VanC resistance pattern is an intrinsic characteristic of *E. gallinarum* and *E. casseliflavus*, and there are genetic subtypes associated with the VanC phenotype. Additional transferable mechanisms may confer resistance on VRE to beta-lactam antibiotics and aminoglycosides.

VRE should not be confused with another pathogen recently incorporated under the bacterial ‘superbug’ rubric. There is concern that resistance to vancomycin may transfer from enterococci to *Staphylococcus aureus*, thereby generating a fearful pathogen—one that is highly transmissible, virulent and, if able to resist other antibiotics, potentially untreatable. Strains of *S. aureus* with an intermediate level of resistance to vancomycin were recovered from clinical specimens in Japan and the United States in 1997. However, the vancomycin resistance detected in these strains of *S. aureus* was unrelated to the mechanisms that confer transferable vancomycin resistance on enterococci.

EPIDEMIOLOGY

Enterococci naturally inhabit the gut of animals, including humans. Various factors appear to contribute to the emergence of VRE as pathogens in health care institutions. These include hospitals’ widespread therapeutic and prophylactic use of antibiotics including vancomycin and third generation cephalosporins.

Colonisation of the gut usually precedes invasive infection, and asymptomatic colonisation is much more common than clinically important infection. Colonisation may occur in settings where invasive infections have not been reported. Gut carriage of VRE may persist for months, with or without associated infection.

Infection of individuals with VRE has been associated with vancomycin therapy, therapy with multiple antibiotics, severe underlying disease, surgery, invasive procedures, prolonged or repeated hospitalisation, and proximity to known cases of VRE infection. Nevertheless, VRE are uncommon pathogens in persons without risk factors, and are rarely genuinely community-acquired infections.

Once human colonisation is established, VRE can spread in hospitals by person-to-person transmission—directly by hand and indirectly via contaminated environment or equipment. A combination of nosocomial transmission and continuing selection pressure contribute to endemicity within health care settings. Molecular typing of VRE is useful to define the epidemiology of VRE within, between and beyond institutions. Pulsed-field gel electrophoresis (PFGE) is emerging as a useful typing method.

INTERNATIONAL PERSPECTIVE

VRE were first reported in the United States and United Kingdom in the mid-1980s. Hospitals that had reported VRE were widely distributed in these countries by the mid-1990s. The proportion of nosocomial infections resulting from VRE in the United States rose from 0.3 per cent in 1989 to 14.2 per cent in 1996.

LOCAL PERSPECTIVE

The first reported case of VRE infection in Australia occurred in 1994.^{1,2} The National Health and Medical Research Council in November 1996 reported 15 isolates of vancomycin-resistant *E. faecium* and *E. faecalis* in Australia, mostly VanB phenotype.³ Infection resulting from VRE with beta-lactamase and high-level gentamicin resistance appears to be an uncommon problem in Australia. Invasive infection with VRE is still rare in Victoria.

CONTROL STRATEGIES

General principles for the control of VRE are outlined below. All health care facilities should be prepared to address the care of persons with VRE. Hospital infection control personnel should be the key educators and strategists. The Department of Human Services has produced local guidelines to inform health care workers about VRE, including a recent document on VRE and methicillin-resistant *S. aureus* (MRSA) in long term care facilities.⁵⁻⁸

General Principles for the Control of VRE⁴⁻⁷

- Use all antibiotics prudently. Restriction of the use of vancomycin and third generation cephalosporins to well defined, mostly therapeutic indications, can reduce the incidence of VRE infection without adverse sequelae.
- Enforce appropriate infection control practices when caring for persons infected or colonised with VRE. Use contact precautions including gloves and gown when directly caring for an infected/colonised patient. Hand washing using 4 per cent chlorhexidine gluconate and an intelligent awareness (enhanced by education) of the modes of VRE transmission are crucial. Physical isolation of infected/colonised person(s) in a designated room helps focus attention on reducing transmission by health care workers.
- Undertake surveillance for VRE. The intensity of surveillance depends on the particular health care setting and whether VRE have already been identified. Identification of clinically significant enterococci to species level and screening for vancomycin resistance will suffice for many settings. More active surveillance of selected faecal samples is needed if VRE have been detected or if the health care setting has a high risk (for example, intensive care, oncology, dialysis and transplant environments).

For infection control advice in relation to VRE contact Genevieve Ryan (03 9344 4575 or pager 016 373 344).

The Microbiological Diagnostic Unit at the University of Melbourne provides a reference diagnostic and molecular typing service for suspected VRE, using conventional bacteriological identification and PCR for resistance genes and speciation, and PFGE for typing. We encourage laboratories to submit VRE for testing and storage to help create a resource to better define the molecular epidemiology of local VRE. The Victorian Hospital Pathogens Surveillance Scheme collates reports of (currently) rare VRE bloodstream infection.

VRE are a serious and expensive problem in many technologically advanced hospitals worldwide. Four years after the first case in Victoria, clinically important isolates still appear to be rare but we must keep informed, keep looking and wash our hands.

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Case Reports from the Melbourne Infectious Diseases Group

Grant Jenkin, Melbourne Infectious Diseases Group

The Melbourne Infectious Diseases Group (MIDG) was established in 1985 to share information and promote contacts among those working in the field of infectious diseases in Melbourne. Starting simply as fireside meetings in the doctors' sitting room at Fairfield Hospital, the MIDG is now a fortnightly breakfast meeting with approximately 40-50 people attending regularly. Its focus has remained the presentation of clinical histories of patients, and it forms an important component of the training of microbiologists and physicians specialising in infectious diseases.

This year an email service (cyberMIDG) was established. A summary of the clinical case discussions is emailed to all those on cyberMIDG. Other information such as outbreak alerts, meeting announcements and infectious diseases employment advertisements are also posted. CyberMIDG has a mailing list of 60, including expatriates working in the United States, the United Kingdom and the Pacific Islands.

For further information contact Grant Jenkin (03 9342 2612 or email: grant.jenkin@nwchn.org.au).

'A SWOLLEN THIGH'

Presented by Dr Kas Thursky, Registrar, Victorian Infectious Diseases Service

A 63-year-old Greek born man, who had immigrated to Australia in 1954, presented with a three-day history of painful swelling of the left thigh with associated fevers, rigors and generalised itch. He had recently returned from a one-month holiday in Noosa, Queensland, but there was no history of trauma or bites to the thigh.

Following his admission to hospital, intravenous flucloxacillin was commenced for a presumed spontaneous pyomyositis. Investigations at that time included a computerised tomography scan of the leg which showed fluid within the muscles of the inner thigh. A needle aspiration of this fluid showed inflammatory cells but did not reveal a causative organism. After one week, the patient underwent open surgical drainage of the area which revealed multiple cysts, confirmed microscopically as hydatid cysts.

Hydatid cysts result from the ingestion of eggs of *Echinococcus*, a dog tapeworm. Dogs in sheep-raising areas of the world, such as Australia and Southern Europe, transmit the disease. The latter region is where this patient likely acquired his infection. The infection in humans

results in cysts, usually in the liver or lungs, but can involve any organ including musculoskeletal tissues in 1-4 per cent of patients. Cysts enlarge slowly and can present many years after infection. A sudden onset of symptoms, as occurred in this patient, can result from rupture or secondary bacterial infection of the cyst.

Diagnosis of muscle cysts preoperatively is often problematic because imaging may not be diagnostic and can look like sarcoma. Serology by immunoelectrophoresis (Arc-5) or ELISA is specific but insensitive for muscle hydatidosis. Indeed initial serology was negative in this patient.

The patient had no evidence of hydatid disease elsewhere and he was commenced on albendazole 400 milligrams BD to treat possible residual disease, using the usual cycling regimen of 30 days on treatment followed by 14 days off treatment.

His course was complicated by the need for further surgery to remove residual hydatid cysts that were not visible during the initial surgery. He was then changed to continuous albendazole therapy and praziquantel was added (40 milligrams per kilogram, cycling seven days on and seven days off for three cycles). He currently remains afebrile and is tolerating antimicrobials.

The management of symptomatic hydatids has previously been predominantly surgical, and surgery remains an important component of treatment, particularly for extrahepatic disease. However, a percutaneous ultrasound guided technique (known as PAIR) to drain liver hydatid cysts and instil a scolicidal agent appears to be effective and safe. This PAIR technique was recently successfully performed at the Royal Melbourne Hospital on a patient with a liver hydatid cyst.

Albendazole is now recognised to have a role—either alone or as an adjuvant to surgery or PAIR—and continuous monitored therapy over three to six months is probably more effective than cyclical therapy. Human studies with small numbers suggest that praziquantel may have a role, in combination with albendazole, in preventing the spread of hydatids if cyst contents spill during the operation.

The follow-up plan for this patient is repeat imaging of the thigh with ultrasound at three to six monthly intervals, probably over a five-year period, and a repeat of his Arc-5 serology over the next two years. If this test is positive at diagnosis or immediately after treatment, it will generally become negative within two years if no viable cysts remain.

'A KEEN GARDENER'

Presented by Dr Rob Baird, clinical microbiologist, Melbourne Pathology

A 59-year-old Perth woman who was a keen gardener had discovered a lesion 'like a boil' under her chin. It had developed over the four weeks since she had noticed it into a 5-centimetre, linear, raised lesion with associated upper cervical lymphadenopathy and nodules along the

line of the neck lymphatics. Initial diagnoses included tinea or pustular furunculosis, but no improvement was noted after treatment with garlic, tea tree oil or oral flucloxacillin. A biopsy in Perth showed granulomas and possible yeast-like bodies but culture was negative.

Visiting Melbourne, the woman saw a general practitioner who sent a scab of the lesion for further culture. White colonies grew on blood agar within three days and (Sabouraud's) black volcano colonies were noted on a fungal culture medium. These were confirmed microscopically as *Sporothrix schenckii*.

This fungus is isolated from soil and plants in tropical and subtropical areas. Eighty cases have been reported in Australia since 1951; these were predominantly from Queensland and northern New South Wales but a solitary case was reported from Perth. This patient had the lymphocutaneous form of *S. schenckii*, which classically occurs following a prick from a rose thorn, although she could not remember any specific injury to the chin.

The differential diagnosis of this presentation includes nontuberculous mycobacteria, nocardiosis and cutaneous leishmaniasis, and biopsy is often required for diagnosis. Histology may be diagnostic if the sparse yeast forms are seen, and culture of the scab may be more successful than from tissue biopsy. Lesions tend to persist for months to years so treatment is advised.

Traditionally potassium iodide drops have been used effectively, but side effects may be problematic. Thus, oral azoles are now more often used. Itraconazole is more active than fluconazole; however, this patient responded to fluconazole given for four weeks, with resolution of the lymphadenopathy.

Key Role for Laboratories in Controlling Measles in Australia

Mike Catton and Heath Kelly, Victorian Infectious Diseases Reference Laboratory

The Fairfield Infectious Diseases Hospital in Melbourne admitted 282 patients suffering from measles in 1922–23. Fourteen of those admitted (5 per cent) died. This was not surprising because epidemics were recognised at the hospital every second year before the measles vaccine was introduced.

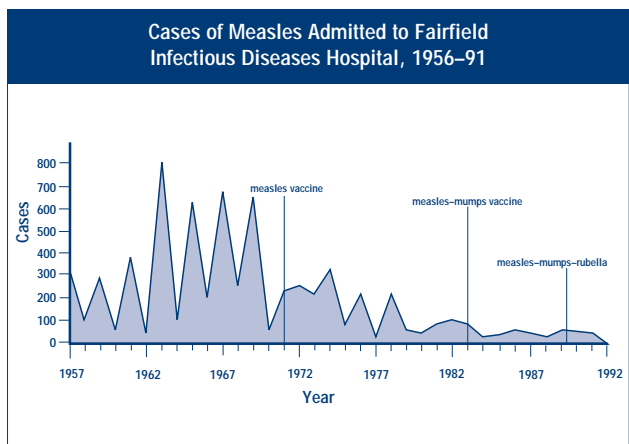
Improving immunisation rates mean that changes are expected in the epidemiology of measles infections:

- A decrease in the magnitude of measles epidemics.
- An increase in the time between those epidemics.
- An increase in the average age of measles patients.
- An increase in the proportion of measles cases occurring in vaccinated children.

and

- A decrease in the measles case fatality rate.

The experience of the Fairfield Infectious Diseases Hospital between 1957 and 1991 illustrates the first two of these changes as measles immunisation was introduced into Victoria (figure).



The Commonwealth Government is now sponsoring a measles-mumps-rubella (MMR) vaccination catch-up campaign targeting preschool and primary school children (see below). The campaign is loosely based on similar successful campaigns conducted by the Pan American Health Organisation (PAHO). These PAHO campaigns have interrupted the transmission of measles in Belize, Brazil, Chile, Colombia, Costa Rica, Cuba, El Salvador, Guatemala, Jamaica, Nicaragua, Panama and Peru. Other countries including Finland, the United Kingdom and the United States have also been successful in interrupting the transmission of measles in recent years.

Enhanced laboratory support is an essential component of the measles control campaign. The Victorian Infectious Diseases Reference Laboratory is offering this support in a number of ways, including:

- Measles virus direct immunofluorescence and PCR for rapid measles diagnosis.
- Measles virus culture for confirmation of rapid tests.

- Measles strain genotyping to differentiate measles virus acquired in Australia from those acquired elsewhere.
 - Plaque reduction neutralisation antibody assays for the resolution of ambiguous serological results.
 - Laboratory differentiation of illnesses diagnosed as measles. These include rubella, human parvovirus and roseola infantum.
- and
- A serological survey at the completion of the measles catch-up campaign to estimate the proportion of Victorian children who may still be susceptible to measles infection.

Australia should be able to interrupt the spread of locally acquired measles early in the next century. But it means achieving high vaccination coverage and endeavouring to test all suspected cases of measles to confirm the diagnosis.

Immunisation Update

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

MAJOR CHANGES AFFECT ALL IMMUNISATION PROVIDERS

The fifth dose of DTP and fourth dose of OPV (previously given in the first year of primary school) and the second dose of measles-mumps-rubella (MMR) vaccine (previously given at 10-16 years of age) is now recommended for children before school entry (at 4-5 years of age). This major change to the vaccination schedule was officially announced on 9 July 1998. To complement these changes, the School Entry Certificate is being amended.

The DTP and OPV vaccines, traditionally given in first year of primary school, are being moved forward to preschool age, aligning with all other States and Territories and easing the burden on school immunisation programs. Already some immunisation providers have begun to promote this change.

ENHANCED MEASLES CONTROL CAMPAIGN

In 1995 the Australian Bureau of Statistics estimated that 91.7 per cent of 6-year-old children in Australia had been vaccinated against measles. A rate of at least 95 per cent is required to control this highly infectious disease and higher rates are necessary to eliminate the virus. The Enhanced Measles Control Campaign is the first phase of Australia's long range plan to eliminate measles from the continent. The campaign will involve four major strategies:

- **Amendment to the immunisation schedule:** As noted above, the immunisation schedule has now been amended so the second MMR dose is administered when children are aged 4-5 years rather than 10-16 years.

- **Catch-up campaign:** An Australia-wide catch-up campaign will be carried out between July and November 1998 to immunise all primary school children with MMR vaccine.
 - **Preschool campaign:** The Health Insurance Commission will send letters to parents of children noted on the Australian Child Immunisation Register as being overdue for the 12 month MMR vaccination. The aim is to ensure high coverage for the first dose.
- and
- **Secondary school campaign:** Parents of all children in secondary school will receive a letter asking them to check their child's immunisation status.

ADVERTISING CAMPAIGN

A high profile advertising campaign will run throughout the program. Advertising began one month before the school sessions and includes television, radio and the printed media.

For further information contact Stephen Pellissier, Coordinator, Enhanced Measles Control Campaign (03 9637 4136).

Review of Sexually Transmissible Diseases in Victoria in 1997

Elaine Stevenson, Nick Crofts and Alison Rodger, Epidemiology and Social Research Unit, Macfarlane Burnet Centre for Medical Research

This is a summary of the 1997 annual report on surveillance of sexually transmissible diseases (STDs) in Victoria. The annual report includes detailed Victorian data on human immunodeficiency virus (HIV), acquired immunodeficiency syndrome (AIDS) and related diseases, comparative data from earlier years and related research.

In Victoria, exposure data are available for almost all diagnoses of HIV and AIDS. Data for syphilis and gonorrhoea have been gradually upgraded in recent years with the integration of clinical and laboratory based surveillance. Improved surveillance of genital chlamydia cases since 1995 resulted in the 1997 notifications being the highest ever recorded in Victoria, and several detailed reports on Victorian surveillance methods are now available.¹⁻³

Information on infection and disease resulting from HIV is collected via three separate mechanisms: statutory notification of all AIDS diagnoses; nonidentifying laboratory and clinical reports of new HIV diagnoses; and basic laboratory data on all HIV tests conducted in Victoria. Notification of new HIV diagnoses has also been a statutory requirement since September 1996.

DECREASING DIAGNOSES OF AIDS

During 1997, 68 people were diagnosed with AIDS in Victoria—a reduction from 138 in 1996 and 172 in 1995. This downward trend in AIDS diagnoses has also been observed nationally and is consistent with changes in treatment of HIV-related disease because the group with the largest decline in incidence are those who have known about their positive HIV status for five or more years.

Most AIDS diagnoses (76 per cent) in 1997, as in previous years, occurred among men with a history of homosexual contact, 6 per cent of whom also reported a history of injecting drug use. There were six AIDS diagnoses in women during the year, all but two of whom reported infection via heterosexual contact. They increased the total number of Victorian AIDS diagnoses among women to 67 (4 per cent of all diagnoses in the State). A total of 1682 people had been diagnosed with AIDS in Victoria by the end of 1997—1318 (78 per cent) of whom were known to have died.

HIV DIAGNOSES IN VICTORIA

There were 187 people newly diagnosed with HIV in 1997, bringing the total number of Victorian HIV diagnoses to 3923 (24 per cent of Australian HIV diagnoses). Most HIV diagnoses during the year (74 per cent) were among men with a reported history of homosexual contact.

Fifteen people (8 per cent of the year's diagnoses) had a history of injecting drug use, with most (eight of the 15) also reporting male homosexual contact. Seven per cent of people diagnosed with HIV during the year were women, of whom 79 per cent (n = 11) reported heterosexual contact as their only risk exposure. Forty-nine people newly diagnosed with HIV during the year (26 per cent of the total) had evidence of either a seroconversion illness and/or a prior negative HIV test in the twelve months preceding their HIV diagnosis.

FALLING GONORRHOEA NOTIFICATIONS

There were 353 cases of gonorrhoea reported in Victoria in 1997. This total was lower than that of the previous year (397 in 1996) and was the third lowest since data were first collected in 1983. The male:female ratio fell slightly for 1997, reflecting an increased number of diagnoses among women (n = 36) compared with the number in 1996 (n = 31) and 1995 (n = 24). Despite this, the rate of diagnosis among men continues to be at least 10 times that for women. There were 185 cases (52 per cent) diagnosed in men with a history of homosexual contact compared with 251 (63 per cent) in the previous year.

Almost all men infected through homosexual contact (94 per cent, n = 173) acquired their infection locally. But 27 of the 101 men who reported infection through heterosexual contact acquired their infection overseas.

INFECTIOUS SYPHILIS: STILL UNCOMMON

Like most other notifiable infectious diseases, syphilis is notifiable by both doctors and laboratories. But laboratory results for syphilis do not readily discriminate between past (treated) infection, other spirochetal infections such as yaws or leptospirosis, and late or latent syphilis.

To combat this problem a laboratory based supplementary surveillance system for syphilis was introduced in 1990 to enable accurate staging of this disease and to collect detailed information on sexual orientation and reason for testing. It is now possible to separate reports of past adequately treated infection and other treponemal (nonsyphilis) infections from those representing current disease.

The same number of cases of infectious syphilis (16) were notified in 1997 and 1996, although there were more cases (172) reported with evidence of current (infectious or non-infectious) syphilis than in 1996 (101) and 1995 (156).

Infectious (recently acquired) syphilis continues to remain uncommon in Victoria. Forty-three per cent of those cases diagnosed during 1997 were symptomatic or the partners of symptomatic individuals. The remaining cases were diagnosed through routine STD screening or antenatal testing.

INCREASING AWARENESS OF GENITAL CHLAMYDIA

The number of chlamydia notifications has risen steadily over the past few years, possibly reflecting efforts to improve the notification system,¹ changes in diagnostic testing requested for *Chlamydia trachomatis* (including the availability of urine testing via polymerase chain reaction) or an increase in incidence. Despite improvements in notification of this disease, it is likely that chlamydia remains substantially underreported as a result of the diagnostic practices of general practitioners and their use of presumptive treatment without testing.^{2,4,5}

Genital chlamydia remains the most common notifiable STD. There were 2116 notifications received in 1997 compared with 1596 for 1996. Most infections were diagnosed in people younger than 30 years of age. Contrasting most other STDs, diagnoses in women outnumbered those in men by a factor of two. Information from the enhanced surveillance system implemented

during 1997 suggests that most men with chlamydia were tested because symptoms were present, whereas a substantial proportion of women with chlamydia were tested in relation to screening.

Further information appears in the report Surveillance of Sexually Transmissible Diseases in Victoria, 1997 which is available from the STD/BBV Program within the Department of Human Services (03 9637 4184).

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Surveillance Briefs

This section includes a summary of notifications received until 30 June 1998 and historical comparisons at both the State (table 1) and Regional level (table 2). Summary data at the local government level can be obtained by contacting Ross Andrews, Department of Human Services (03 9637 4121).

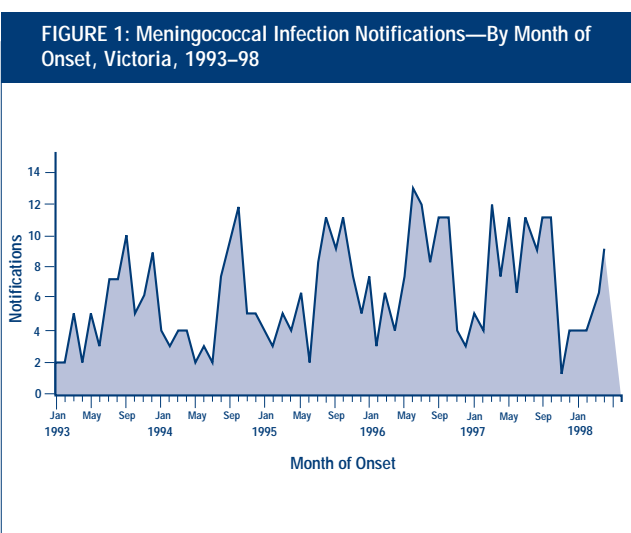
The 1997 and 1998 data are currently under review and may be subject to change. Data for notifications of campylobacteriosis, giardiasis, hepatitis B and hepatitis C have not been included in this report, due to problems in transferring data to a new database. Summary data of sexually transmissible diseases in 1997 have been included as a separate report (see Stevenson et al., page 12). The 1998 data will be included in the next edition of the bulletin.

MENINGOCOCCAL INFECTIONS EXPECTED TO INCREASE

While meningococcal infections are uncommon, the reported incidence of the disease often peaks about this time of year (figure 1). During recent years there has been a steady increase in the number of notified meningococcal infections, although fewer cases have been reported in Victoria in 1998 than for the same period in 1997 (table 1).

All ages may be affected, but most cases occur among children younger than 5 years of age, with a second peak among adolescents and young adults. Of the 24 cases notified to date this year, ten have been under 5 years of age and six of these cases were under one year. The youngest case was a one-week-old twin, who responded well to treatment.

All cases have occurred sporadically. Nine of those meningococci which were typable were Group B and three were Group C.



MANAGEMENT OF CONTACTS

Close contacts of the patient, such as family and household members, must receive prophylaxis, preferably within 24–48 hours of diagnosis. It is the responsibility of the treating hospital to arrange antibiotic prophylaxis of family contacts, which should be provided free of charge. The Department of Human Services will undertake the follow-up of other contacts such as staff and children in day care centres, and those who have shared drinks with the patient during the ten days preceding the onset of illness. Surveillance letters are given to contacts.

Medical, nursing staff or ambulance officers are not at increased risk (unless they have attempted mouth-to-mouth resuscitation) and do not need prophylaxis.

PROPHYLAXIS

Rifampicin is the drug of choice for prophylaxis in the following doses:

Adults	600 mg twice daily for two days
Children one month of age and older	10 mg/kg twice daily for two days
Children up to one month of age	5 mg/kg twice daily for two days

An alternative to rifampicin is ceftriaxone (5 milligrams per kilogram, to a maximum of 250 milligrams) as a single intra-muscular dose. *Ceftriaxone is the drug of choice in pregnancy.* Ciprofloxacin as a single dose of 500 milligrams orally can also be used in adults and children older than 12 years, provided they also weigh more than 40 kilograms. Like rifampicin, ciprofloxacin should not be used in pregnancy.

INFLUENZA SEASON ALREADY ESTABLISHED

SENTINEL SURVEILLANCE

Six rural general practices and 11 metropolitan practices contribute data on influenza-like illness according to the ASPREN (Australian Sentinel Practice Research and Education Network) criteria (box). Additional surveillance data are provided from the emergency departments of the Royal Melbourne Hospital, the Alfred Hospital, the Austin Hospital and the Monash Medical Centre. Since hospital patients are more likely to be seen later in the course of their illness than patients in general practice, the ASPREN criteria have been modified for use in hospitals.

Nose and throat swabs and serum samples, obtained from patients who satisfy the ASPREN criteria, are forwarded to the Victorian Infectious Diseases Reference Laboratory (VIDRL). Direct immunofluorescence is performed on the swabs which are then cultured for influenza and other respiratory viruses. Complement fixation tests are used to determine antibody status for influenza A and B.

ASPREN CRITERIA FOR THE CLINICAL DIAGNOSIS OF INFLUENZA

When there is an influenza epidemic, a patient should satisfy four of the following criteria; when there is no epidemic, a patient should satisfy six of the criteria to be clinically diagnosed as having influenza:

- 1 Onset within 12 hours
- 2 Cough
- 3 Rigors or chills
- 4 Fever
- 5 Prostration/weakness
- 6 Myalgia, widespread aches and pains
- 7 No significant respiratory symptoms other than redness of the nasal mucous membrane and throat
- 8 Influenza in close contacts

CONSULTATION RATES INCREASING

To the end of June, 163 patients satisfying the ASPREN criteria for the clinical diagnosis of influenza have been recorded in general practice and a further 14 have been recorded at the participating hospitals.

The GP consultation rate for influenza-like illness in 1998 does not appear to be rising as rapidly as at the corresponding time last year (figure 2). However, data for the first week in July suggest that the rate is approaching 3 per cent, indicating that the flu season is already well established. This conclusion is supported by the large number of cases of influenza from non-surveillance patients at the Royal Children's Hospital and the Monash Medical Centre (figure 3). The larger numbers seen at these two locations, compared with the number seen at VIDRL, reflect the early phase of an influenza epidemic among younger patients.

There have been 13 influenza A diagnoses by immunofluorescence or culture and a further 17 were weakly positive by serology. There has been one influenza B diagnosed by serology only. All the influenza A isolates typed to date, either at VIDRL or CSL, have been A Sydney-like.

Historical data from VIDRL indicate that significant numbers of influenza A and B co-circulate in alternate years, with influenza A dominating in the intervening years. 1997 was a significant influenza year in many parts of Victoria, with influenza A and B co-circulating. It is probable that this season will be as significant, but will predominantly be an influenza A season.

A detailed report of influenza surveillance will appear in the next edition of this bulletin.

FIGURE 2: Influenza Consultation Rates Reported Through Sentinel Surveillance—By Fortnight and Year, Victoria, 1997–98

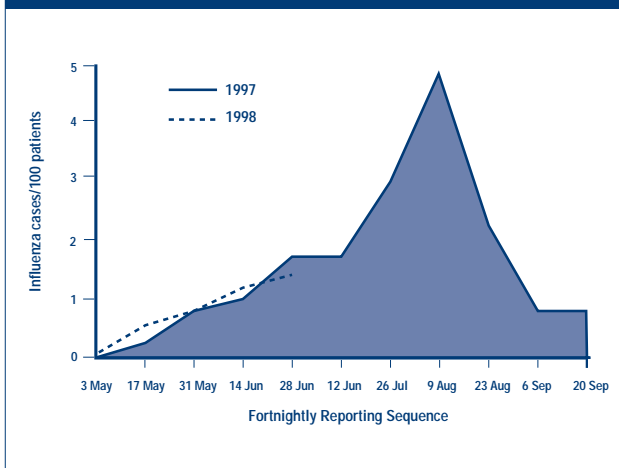
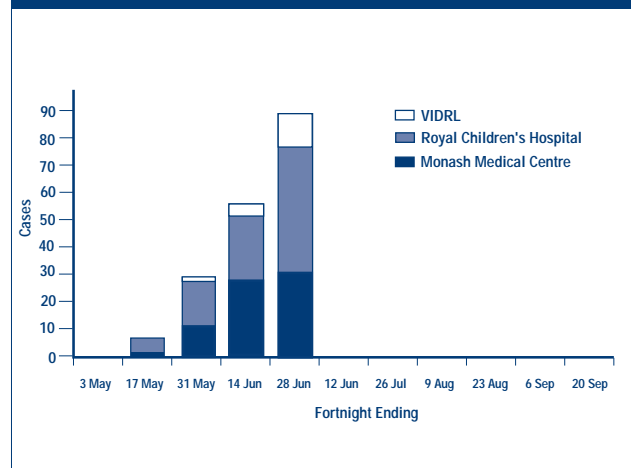


FIGURE 3: Influenza Cases—By Fortnight and Laboratory



New Zealand Outbreak of Swimming Pool Associated Cryptosporidiosis

We have included this summary because of the marked similarities between the New Zealand outbreak of cryptosporidiosis and the Victorian investigation reported in this edition of the bulletin.

New Zealand experienced its first documented outbreak of cryptosporidiosis in March this year. The outbreak occurred in the Hutt Health District—just north of Wellington. The public health unit investigated the outbreak, using enhanced surveillance for cases of cryptosporidiosis, a case control study, and by auditing local swimming pools. One hundred and twenty two cases were notified to the public health unit between 1 January and 30 April this year. The majority (46 per cent) of cases were under the age of 4 years, and 72 per cent of cases had swum in a swimming pool in the two weeks prior to their illness. Amongst 53 cases enrolled in a case control study, swimming in any swimming pool and in one particular pool in the Hutt Valley were strongly associated with illness. Having someone in the house with recent gastroenteritis and a case wearing nappies were also associated with illness.

The public health unit also collected water samples from four swimming pools in the Hutt Health District. Water from the pool implicated in the outbreak was positive for *Cryptosporidium* oocysts. The Hutt City council closed this pool for cleaning. No *Cryptosporidium* were detected after the pool was cleaned. Two other pools were closed as a precautionary measure. The public health unit advised swimming pool operators to warn people with recent diarrhoea not to swim, to exclude children in nappies from swimming, to encourage people to shower before

swimming, to maintain high standards of hygiene in the facility, and to respond appropriately when patrons accidentally defecated in the pool.

More details of the outbreak can be found in the *New Zealand Public Health Report*, which can be obtained from their website at: <http://www.moh.govt.nz/phg/phr.htm>.¹ In their report Baker *et al.* reinforced the importance of person-to-person spread in the aetiology of cryptosporidiosis. The authors did not believe that swimming pool managers should routinely test swimming pool water for *Cryptosporidium*, due to the high costs and difficulties in interpreting results. They recommended that the control of swimming pool associated cryptosporidiosis should focus on preventing contamination of pools by educating the public about hygiene, and about not swimming after gastroenteritis. They emphasised the fact that pools should be appropriately designed and managed, and that the risk of gastroenteritis associated with swimming in public pools must be balanced with the benefits of swimming and the role it plays in preventing childhood drownings.

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IS IT MEASLES?

The Department of Human Services received 94 notifications of measles to the end of June 1998. In comparison there were 104 notifications for all of 1997 and 95 notifications in 1996.

As part of an enhanced measles surveillance program, the Department offers to arrange laboratory confirmation of all notified cases. To date, 81 of 94 cases reported this year (87 per cent) have had blood drawn for the estimation of measles IgM. Only 13 of the 81 patients tested (16 per cent) were confirmed as having measles. However, the apparent positive predictive value of a clinical notification for true measles (16 per cent) is exaggerated by the inclusion of nine cases notified as part of two separate clusters. If these nine cases are excluded, clinical notification of measles predicts true sporadic measles in only 5 per cent of cases.

The 81 notifications of measles for which there were serological results were compared with the standard measles case definition to determine if this would improve the prediction of measles based on clinical criteria (box). There was only a marginal improvement in the accuracy of prediction using these criteria. The measles case definition is neither sensitive (69 per cent), nor specific (63 per cent) and only predicted measles 26 per cent of the time in this six months.

The case definition for the clinical diagnosis of measles is:

- A generalised maculopapular rash lasting three or more days.
- A fever of at least 38°C if measured.
- Cough or coryza or conjunctivitis or Koplik's spots.

These data emphasise that laboratory confirmation of clinical diagnoses will become even more important as measles becomes increasingly uncommon.

CHOLERA CASE IMPORTED

A 41-year-old man fell ill three days after returning from a holiday in Bali where he had eaten prawns and crayfish from a barbecue on the beach. He developed watery diarrhoea which recurred several times during the day. The diarrhoea ceased three days later and he remained well with no other symptoms. A faecal specimen obtained by his local doctor was confirmed on culture as *Vibrio cholerae* serogroup O1 (Ogawa).

CRYPTOSPORIDIOSIS INCREASE DUE TO SURVEILLANCE

Cryptosporidiosis is not a notifiable disease in Victoria. The dramatic increase in the number of reports received for the year to date (table 1) reflects active surveillance implemented to investigate a cluster of cryptosporidiosis cases earlier this year and voluntary reporting by a number of laboratories as part of an ongoing case control study to identify risk factors for *Cryptosporidium* infections (see Kirk *et al.*, page 3).

SALMONELLOSIS REMAINS MODERATE

Notifications of salmonellosis for the year to date have remained moderate and, with the exception of early 1997, are similar to levels in previous years (figure 4).

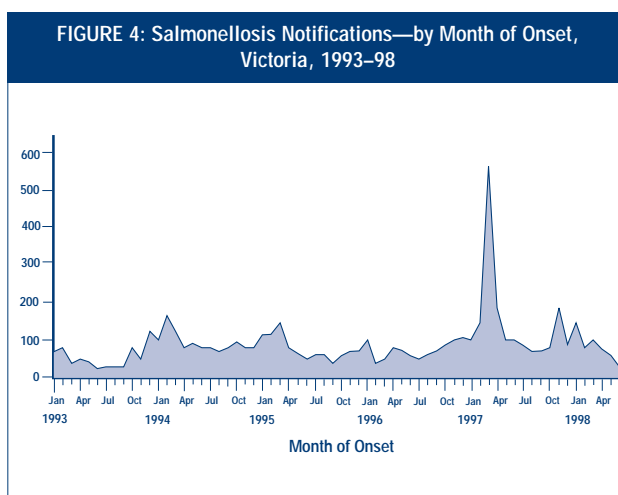


TABLE 1: Notifications of Infectious Diseases Received from 1 January 1998 to 30 June 1998 and Historical Comparisons, Victoria

DISEASE	1998 ytd	1997 ytd	1996 ytd	1995 ytd	1997 total	1996 total	1995 total
Amoebiasis	27	44	35	42	75	75	72
Arboviral infections	59	1072	155	43	1106	187	55
Brucellosis	2	1	2	1	3	3	2
Cholera	1	0	0	0	1	0	0
Food/waterborne illness							
– cryptosporidiosis	193	10	3	33	15	18	43
– other	46	128	18	21	189	53	32
<i>Haemophilus influenzae</i> type B	5	3	7	9	6	11	14
Hepatitis A	110	273	341	84	366	464	231
Hepatitis (viral, unspecified)	2	2	6	2	3	9	5
Hydatid disease	8	4	5	4	32	16	14
Legionellosis	30	23	22	14	30	37	23
Leptospirosis	5	12	35	17	27	74	58
Listeriosis	6	11	9	14	15	19	23
Malaria	47	53	57	54	90	112	110
Measles	94	55	57	62	102	96	155
Meningococcal infection	24	42	33	25	99	93	75
Mumps	30	43	24	40	65	50	78
Pertussis	481	948	235	171	1684	1201	391
Psittacosis	16	31	35	49	39	62	147
Q fever	14	14	27	21	24	62	67
Rubella	100	150	523	146	370	804	1165
Salmonellosis	610	1155	424	614	1698	916	984
Shigellosis	58	35	32	46	79	71	85
Taeniasis	3	4	4	4	5	7	13
Tetanus	1	1	0	2	1	1	4
Tuberculosis	129	187	151	151	352	302	301
Typhoid/paratyphoid	10	14	14	22	23	24	28
Typhus	3	0	6	1	1	9	6
Yersiniosis	19	9	7	15	15	15	27
Total	2133	4324	2267	1707	6516	4791	4208

Notes

- 1 Data for notifications of campylobacteriosis, giardiasis, hepatitis B and hepatitis C have not been included due to problems in transferring data to a new database.
- 2 'ytd' refers to 'year to date' (that is, 1 January–30 June in the respective year).
- 3 The 1997 and 1998 data are preliminary figures only and may be subject to revision.

TABLE 2A: Notifications of Infectious Diseases—By Department of Human Services Region and Historical Comparisons, Victoria, 1 January 1998 to 30 June 1998

DISEASE	Non-Metropolitan Regions											
	Barwon South-Western		Grampians		Loddon Mallee		Hume		Gippsland		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
Amoebiasis	0	0	1	0	0	0	0	0	0	1	27	44
Arboviral infections	3	38	7	120	7	473	8	172	16	20	59	1072
Brucellosis	0	0	1	1	0	0	0	0	0	0	2	1
Food/waterborne illness												
– cryptosporidiosis	0	0	1	0	5	1	9	0	9	7	193	10
– other	1	2	3	0	1	1	2	1	1	4	46	128
<i>Haemophilus influenzae</i> type B	0	0	0	0	0	0	2	0	0	1	5	3
Hepatitis A	0	6	2	2	26	38	2	37	5	1	110	273
Hepatitis (viral, unspecified)	0	0	0	0	0	0	0	0	0	0	2	2
Hydatid disease	0	0	1	0	0	0	0	0	0	0	8	4
Legionellosis	2	2	0	1	0	0	0	0	0	1	30	23
Leprosy	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	0	3	0	0	0	0	0	4	5	3	5	12
Listeriosis	0	0	1	0	1	0	0	1	0	0	6	11
Malaria	0	2	1	0	1	0	4	4	0	1	47	53
Measles	2	1	1	4	3	2	5	6	3	2	94	55
Meningococcal infection	0	2	0	1	1	4	6	1	1	2	24	42
Mumps	0	0	2	1	2	3	3	4	2	2	30	43
Pertussis	19	28	12	24	25	113	23	61	113	62	481	948
Psittacosis	0	4	0	0	2	0	2	1	0	5	16	31
Q fever	2	0	3	2	1	1	3	5	1	1	14	14
Rubella	3	4	0	7	3	7	8	11	5	1	100	150
Salmonellosis	43	37	21	21	30	30	28	37	31	32	610	1155
Shigellosis	2	0	0	0	1	5	1	1	1	0	58	35
Taeniasis	0	0	0	0	0	0	0	0	0	0	3	4
Tetanus	0	0	1	0	0	1	0	0	0	0	1	1
Tuberculosis	3	4	1	5	3	2	3	4	1	1	129	187
Typhoid/paratyphoid	0	0	0	0	0	0	0	1	0	0	10	14
Typhus	0	0	0	0	2	0	0	0	0	0	3	0
Yersiniosis	1	1	1	0	0	0	1	1	0	1	19	9
Total	81	134	60	189	114	681	111	353	194	148	2133	4324

(continued)

TABLE 2A: Notifications of Infectious Diseases—By Department of Human Services Region and Historical Comparisons, Victoria, 1 January 1998 to 30 June 1998

DISEASE	Metropolitan Regions											
	Western Metropolitan		Northern Metropolitan		Eastern Metropolitan		Southern Metropolitan		Unknown or Interstate		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
Amoebiasis	6	9	5	10	5	6	9	18	1	0	27	44
Arboviral infections	0	23	4	59	3	77	7	67	4	23	59	1072
Brucellosis	0	0	0	0	1	0	0	0	0	0	2	1
Food/waterborne illness												
– cryptosporidiosis	16	0	43	0	31	1	73	1	6	0	193	10
– other	4	4	7	4	11	23	14	85	2	4	46	128
<i>Haemophilus influenzae</i> type B	0	0	2	0	0	1	1	1	0	0	5	3
Hepatitis A	11	19	14	59	24	40	22	63	4	8	110	273
Hepatitis (viral, unspecified)	0	0	1	1	0	0	1	0	0	1	2	2
Hydatid disease	3	2	1	1	3	1	0	0	0	0	8	4
Legionellosis	14	4	4	4	3	2	6	9	1	0	30	23
Leptospirosis	0	0	0	0	0	0	0	1	0	1	5	12
Listeriosis	1	4	1	1	0	3	2	2	0	0	6	11
Malaria	1	6	13	10	11	7	12	16	4	7	47	53
Measles	12	13	18	14	25	4	25	4	0	5	94	55
Meningococcal Infection	1	4	3	10	5	6	5	12	2	0	24	42
Mumps	7	4	7	9	1	13	6	5	0	2	30	43
Pertussis	35	90	55	190	67	182	118	183	14	15	481	948
Psittacosis	4	5	1	3	3	10	4	3	0	0	16	31
Q fever	1	4	1	0	1	0	1	1	0	0	14	14
Rubella	12	14	20	28	23	36	21	36	5	6	100	150
Salmonellosis	71	123	125	105	98	300	127	457	36	13	610	1155
Shigellosis	6	7	12	6	11	5	23	9	1	2	58	35
Taeniasis	1	2	2	2	0	0	0	0	0	0	3	4
Tetanus	0	0	0	0	0	0	0	0	0	0	1	1
Tuberculosis	43	34	16	52	27	30	31	53	1	2	129	187
Typhoid/paratyphoid	2	0	3	4	2	5	3	4	0	0	10	14
Typhus	0	0	1	0	0	0	0	0	0	0	3	0
Yersiniosis	5	2	2	1	5	0	4	2	0	1	19	9
Total	256	373	361	573	360	752	515	1032	81	90	2133	4324

Notes

- 1 Data for notifications of campylobacteriosis, giardiasis, hepatitis B and hepatitis C have not been included due to problems in transferring data to a new database.
- 2 'ytd' refers to 'year to date' (that is, 1 January–30 June in the respective year).
- 3 The 1997 and 1998 data are preliminary figures only and may be subject to revision.
- 4 Notification data are available at local government level. For further information contact Ross Andrews (03 9637 4121).

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The *Victorian Infectious Diseases Bulletin* is published quarterly and provides summaries of infectious diseases surveillance data, local news and reports. Topics include outbreak investigations, infection control procedures, clinical cases of general interest, brief reports on original research and laboratory science.

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Opinions expressed in the bulletin are those of the authors and not necessarily those of the Department of Human Services. Data are subject to revision.

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