

Victorian Infectious Diseases Bulletin

Vaccination to Halt Meningococcal Outbreak at School

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In the last week of August 1999, the Disease Control Section was notified of three students from the same school with meningococcal septicaemia. Outbreaks of meningococcal infection, defined as two or more cases linked in time or place, are relatively uncommon in Victoria. This report summarises the public health response to the outbreak.

PUBLIC HEALTH RESPONSE

The public health response to outbreaks of meningococcal infection includes gathering a description of the outbreak, conducting active surveillance of the population at risk, undertaking specific measures to prevent further transmission, educating the community, and running microbiological testing to determine the sub-type(s) involved.

In this outbreak, we undertook detailed interviews with the cases' families to identify close contacts and to further elicit common links between cases. Our aim was to identify the population at risk. Close contacts require antibiotic prophylaxis to eliminate nasopharyngeal carriage of meningococci from asymptomatic contacts and thereby prevent further transmission between carriers and susceptible individuals. Prophylaxis may not prevent contacts from acquiring disease, and there is no evidence that prophylaxis can abort disease in those already incubating the infection.

Discussion with the cases' parents and the school staff identified several important links.

1. The first two cases were close friends. The third case was unknown to the first two, and attended a different school campus.

2. The three students had recently been involved in *different* extension school camp activities: one had gone to Canberra, one had gone to Darwin and one had gone on a beach retreat day.

3. The school camps to Canberra and Darwin had effectively involved the entire year 9 group from one campus. The beach retreat had involved year 10 students from both campuses.

PROPHYLAXIS

School contacts usually do not require prophylaxis.¹ However, given the recent timing of the camps and the closeness of the camp living quarters of the students, we decided to treat the entire year 9 group on one campus, the year 10 children who had attended the retreat, and the usual family and close contacts. The rationale was that the two school groups were likely to contain a pool of asymptomatic carriers, who were at high risk of transmitting the infection to other students. Mass chemoprophylaxis aims to interrupt this process.

RATIONALE FOR IMMUNISATION

Immunisation can be undertaken to prevent cases if the organism is identified as a vaccine-preventable strain. In Victoria, almost all meningococcal disease is caused by serogroups B and C, in a ratio of roughly

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2:1.² There is no vaccine for group B, but an effective group C vaccine can be used in specific groups at increased risk (although it is not very effective in young children). Immunisation is recommended if the age-specific attack rate is greater than 40 per 100 000. The current group C vaccines induce protective antibodies in 10–14 days in 90 per cent recipients over the age of 2 years.³

In this outbreak, although only a single blood culture sample from one case was positive, the isolate was identified as a group C meningococcus. The use of new molecular techniques subsequently confirmed the other two cases as also being group C. Thus we decided to protect the population of the school and the family contacts of the cases by immunisation. Given the delay in providing protection, immunisation complements rather than supplants chemoprophylaxis in the management of outbreaks. We vaccinated 1530 of the 1600 children and staff on a single day; there were no major problems except one child fainting. Absentees were vaccinated in a mop-up campaign two days later.

We communicated via the school with the parents of children whom we identified initially as being in the chemoprophylaxis target group, and later as candidates for immunisation. This approach achieved a very high rate of signed parental consent to the immunisation campaign. Communication with the general public was effected through regular press conferences and the distribution of press kits that summarised the local situation and the rationale for public health decisions. There have been no further cases linked to this outbreak.

LESSONS FOR THE FUTURE

Given the volume and content of telephone calls to the Disease Control Section during this outbreak, it is clear that the general public do not fully understand the rationale for distributing prophylactic antibiotics. It is also clear that the demand for protective vaccines is high when people are frightened by a disease such as this one. Both chemoprophylaxis and immunisation are important tools in the public health equipment box, to be used with caution and based on scientific evidence. The media can be a useful ally in the dissemination of important information at crucial stages in this process.

A new group C conjugate vaccine has recently been released in Britain.⁴ This vaccine uses the meningococcal surface polysaccharide used in the presently available vaccine, and by conjugating this to protein, overcomes the shortcomings of the presently available vaccine. Unlike conventional vaccines for group C disease, the new conjugate vaccine provides protection in children from as young as two months of age, and appears to induce immunological memory, so has longer duration of effect. An evaluation of the suitability of this vaccine in the Australian context will need to be made.

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Infectious Diseases News

VIDB READERSHIP SURVEY

Our thanks to all those readers who answered our readership survey. This issue of the bulletin is only being sent to those who indicated that they wished to stay on the mailing list. We are sending a reminder letter to those who didn't respond that they must return their survey form to receive the bulletin. We will report the results of the survey in a future issue.

MENINGOCOCCAL VACCINATION AT SCHOOL

Graham Tallis, Kath Taylor and Priscilla Robinson report (page 65) on a vaccination program at a school following a cluster of three cases of meningococcal septicaemia. This year in Victoria, notifications of meningococcal infection have been higher than those in previous years.

ADVICE FOR SUMMER

Three articles in this issue remind us about public health during the summer season. Tony Gherardin provides advice on travelling safely. Mark Veitch deals with food safety and gastrointestinal illness during summer. Scott Bowden, Julian Druce and Heath Kelly write about the survival of HIV, hepatitis B and hepatitis C in the environment and how to deal with discarded needles.

CLUSTERS OF MEASLES IN VICTORIA

Since the large outbreak of measles from February to May this year, sporadic cases and clusters of measles have been identified in Victoria. The most recent cluster has involved the East Timorese evacuees who are staying at the safe haven in Puckapunyal. Officers from the Disease Control

Section assisted with immunisation sessions (conducted by the health services on site at Puckapunyal) to prevent further cases.

Further, Ian Jennens reports (page 73) on a case of transverse myelitis following measles infection linked with a Ferntree Gully cluster. For more information on measles in Victoria since the outbreak see Surveillance Briefs.

1997 ANNUAL REPORT ON THE WEB

The Victorian annual report for notifiable infectious diseases in 1997 has been published. The Department of Human Services received 17 925 notifications in 1997—up 11 per cent from the number in 1996. You can find the 1997 annual report and previous years reports at <http://www.dhs.vic.gov.au/phd/9903122/index.htm>, or contact the Department for a hard copy (03 9637 4126).

NEW FAX NUMBER FOR NOTIFICATIONS

We now have a new 1300 number for receiving faxed notifications of infectious diseases. Calls made to the Department's two 1300 numbers from within the 03 area code (including Albury–Wodonga) are charged at the same rate as a local call. These numbers are reserved for notifications only, and should not be used for general calls or information about health alerts.

Notifications of Infectious Diseases

Telephone: 1300 65 1160

Facsimile: 1300 65 1170

Preventing Infections in Overseas Travellers

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Australians are active travellers and among the most travelled populations of all countries around the world. Approximately 4 million people travel overseas each year, with the majority travelling for recreational purposes. A large number of these travellers will acquire an infection along the way. Travel increases a person's potential exposure to infectious diseases, and disease in travellers is frequent. The spectrum of disease ranges from the common to the unusual and rare, and from the trivial to the life threatening. Identifying the particular infectious disease(s) to which each traveller may be exposed will determine necessary strategies. This approach is the basis of modern travel medicine practice. The four major strategies for prevention of infections in travel medicine are advice, vaccinations, prophylactic medication and self-treatment.

PRE-TRAVEL ADVICE

Pre-travel advice involves educating the traveller about the potential risks of infectious disease. Specific education will hopefully lead to behavioural change in the individual, which will reduce the risk of exposure. We have scant data on travellers' compliance with such or on the effectiveness of pre-travel counselling.

FOOD AND WATER

Food-borne and water-borne diseases are the most common threats to travellers, and about 40 per cent of all international travellers experience traveller's diarrhoea. Various *E. coli* pathotypes (particularly enterohaemorrhagic *E. coli*) are a common cause, as are many other enteric pathogens. Before travelling, overseas travellers should seek advice on safe food and beverages, and remember the popular rule, 'Cook it, peel it or leave it'.

INSECTS

Several insect-borne diseases are common risks for international travellers, and avoiding insects is the best prevention. Malaria is clearly an important and potentially life-threatening parasitic disease, while dengue fever is an increasingly common arboviral infection.

Travellers may be exposed to many insect-borne diseases, involving a range of arthropod species. They should use a suitable, effective diethyl-*m*-toluamide (DEET)-containing repellent. DEET has been shown to be effective and has a favourable toxicity profile. Further, wearing long-sleeved, light-coloured clothing reduces mosquito bites, as does impregnating clothing with the insecticide permethrin. Sleeping under a permethrin-impregnated net reduces the risk of malaria. Knock-down sprays may also have a role.

ANIMALS AND PARASITES

Advice about avoiding animals is an important message for travellers; in particular, rabies is present in many popular tourist destinations, and dog bites or licks are not uncommon. Fresh water exposure in some countries, notably in Africa and parts of South America and South East Asia where schistosomiasis is endemic, can lead to infection and should be avoided. Also, travellers who do not use footwear are at risk of acquiring transdermal infections.

SEXUALLY TRANSMITTED DISEASES

Travellers need to remember the risk of infectious disease acquired through unsafe sexual practice. The rates of many sexually transmitted diseases in developing countries are poorly defined, but may be very high in selected populations. Travellers should be encouraged to carry condoms and always practise safe sex.



Eggs of Schistosoma haematobium present in urine (Courtesy Norbert Ryan, Victorian Infectious Diseases Reference Laboratory)

TRAVEL VACCINATIONS

A range of routine and travel-related vaccines may be relevant to travellers. Routine immunisation schedules, including all paediatric vaccines for children, should be up-to-date for all travellers. Polio remains endemic in Africa and parts of Asia, and a booster of sabin oral polio vaccine is recommended for previously immunised travellers whose last booster was more than 10 years ago. Previously non-immunised adults should be primarily immunised with inactivated polio vaccine.

Combined adult diphtheria and tetanus vaccine should be given to all adults whose last booster was more than 10 years ago. Diphtheria has unfortunately re-emerged in eastern Europe, as routine public health initiatives have declined. Travellers aged 15–30 years should receive a measles/mumps/rubella vaccine if they have not had the clinical disease or two previous measles vaccines.

Influenza and pneumococcal vaccines should be offered for all the normal indications, and influenza vaccine may be considered for travellers to countries where evidence of influenza activity is being reported. We have no data that quantify the benefits of influenza vaccine for travellers.

Hepatitis A vaccine should be offered to most travellers to developing countries. This disease remains the most common vaccine-preventable disease acquired by travellers. Protection with vaccine is close to 100 per cent and is long lasting; further, the vaccine is suitable for individuals aged 2 years and older.

Protection against typhoid fever can be facilitated with parenteral Vi antigen vaccine or oral live attenuated vaccine. Both vaccines confer about 70–80 per cent

protection — for about three years for the Vi vaccine and one to five years for the oral vaccine, depending on the number of doses used. The older, whole cell vaccine has been discontinued. Cholera vaccine in Australia, the old, whole cell vaccine, is not recommended because it is poorly immunogenic and produces a high rate of adverse reactions.

The threshold for offering hepatitis B vaccination to travellers is becoming lower, as we adopt a universal vaccination policy against hepatitis B. Travellers who will undertake higher risk activities in their travels, or who intend to travel for an extended period (especially in countries where hepatitis B is endemic), should be vaccinated.

Other vaccines may be relevant to the specific itinerary. Yellow fever vaccine, a live attenuated strain, provides long-term immunity against yellow fever and is relevant to travellers to sub-Saharan Africa and the Amazon. Japanese encephalitis vaccine is an inactivated, whole cell vaccine that provides at least three years protection against this mosquito-borne virus endemic in rural Asia. The vaccine is indicated for travellers who will have extended rural exposures in Asia, Papua New Guinea or the Torres Strait.

Meningococcal vaccine will provide good protection against serogroups A, C, Y and W-135 for three years. Serogroups A and C are implicated in epidemic disease, which occurs in specific areas of the world. Travellers to sub-Saharan Africa, north India and Nepal or pilgrims on the Haj should be vaccinated.

Human diploid cell vaccine against rabies is a safe, highly effective vaccine, and it is recommended for travellers who will spend more than three months in countries with endemic rabies — that is, countries in Asia, Africa and Latin America. Rabies pre-exposure immunisation can be given using an intradermal method, which is substantially cheaper. This method is not suitable for all travellers and should be performed by experienced vaccinators only.

Travellers to areas with a high endemicity of tick-borne encephalitis — such as forested rural areas of eastern Europe and parts of Asia — should obtain this vaccine at their destination. Finally, the exact role of BCG vaccine for tuberculosis prevention in travellers remains controversial. In general, BCG may be offered to Mantoux-negative travellers spending more than 12 months in countries where tuberculosis is endemic. The emergence of multidrug-resistant tuberculosis has highlighted the need for better vaccines. The actual risk of tuberculosis for travellers is not well defined.

PROPHYLAXIS FOR TRAVELLERS

Prophylactic medication or post-exposure prophylaxis is another strategy to reduce the risk of disease after exposure to infectious agents. An individual's compliance is a key determinant in the effectiveness of drug prophylaxis.

Malaria is the main infectious disease to which drug prophylaxis applies. Patients must understand that 'no bites means no risk' and that they can significantly reduce their risk when in malaria zones by following the simple measures already mentioned.

Where the perceived risk of disease is high, such as in Africa, the Amazon, parts of Asia and western Oceania, the need to use chemoprophylaxis must be considered. Several factors influence the decision to choose chemoprophylaxis, including the level of malaria transmission, the local resistance pattern and the degree of exposure.

Currently mefloquine or doxycycline is the most appropriate recommendation for malaria prophylaxis for areas with chloroquine-resistant malaria. Each agent has specific contraindications and potential for side-effects. Where both are contraindicated, chloroquine (either alone or with proguanil), should be considered; otherwise, standby treatment alone may be an option. Currently, the combination drug Malarone offers effective and safe standby therapy for chloroquine-resistant falciparum malaria. The advent of self-test diagnostic kits for travellers has enhanced the role of standby treatment.

Prophylaxis against traveller's diarrhoea is not routinely recommended, although there may be special circumstances in which antibiotic prophylaxis may be justified, such as for competition-level sports people and Heads of State.

A more recent role for chemoprophylaxis is triple-drug anti retroviral prophylaxis for potential exposure to HIV. This may be appropriate for travellers such as health workers in aid programs. The logistics, costs and ethical issues surrounding safe provision and management of this new prophylaxis are still evolving. Hopefully, this situation is not routine, but rather underscores the dynamic nature of the field of prevention and early intervention.

EARLY INTERVENTION

Early intervention to treat infectious diseases in travellers can be important, and certain medications have proved to be consistently useful.

A quinolone antibiotic such as norfloxacin for treatment of traveller's diarrhoea shortens the length and severity of the illness. Tinidazole carried and taken at the onset of symptoms consistent with giardiasis may be appropriate for treatment, and may prevent chronic symptoms. Standby treatment for malaria has been discussed.

Travellers can treat simple infectious episodes by using a broad-spectrum antibiotic suitable for sinusitis, acute exacerbation of bronchitis or common bacterial skin infections. Clotrimazole (or equivalent) is useful for treating candidiasis, especially if induced by doxycycline for malaria prevention. Topical eye and ear antibiotics are also useful, as is topical iodine antiseptic. All of these items are components of a good traveller's medical kit.

From a public health perspective, protecting the community against imported exotic diseases, particularly where conditions receptive to endemicity occur, is an important reason to minimise traveller's exposure to infectious diseases. This is especially true when emergent disease is occurring as a result of old infectious agents causing disease in new ways or new contexts. For developing countries to have to allocate scarce resources to treating preventable disease in visitors is also a concern.

Gastroenteritis in Victoria: Recent Statistics and How Not to Be One

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The incidence of many infectious diseases varies through the year, and notifications of the common bacterial and protozoal agents of gastroenteritis regularly peak in summer. The basis of this phenomenon is not clear, and may involve the prevalence of pathogens in animal hosts and agricultural environments, riskier patterns of food handling and consumption, and other behaviours (such as swimming) that favour the spread of gastroenteritis during the warmer months. This article considers the incidence of sporadic and outbreak-related gastroenteritis in Victoria and offers some preventive strategies for the home kitchen.

HOW COMMON IS GASTROENTERITIS?

Estimates based on local data¹ and a recent study in the United Kingdom² suggest that roughly 20–25 per cent of the population annually experience infectious gastroenteritis. In Victoria, this would mean around one million episodes of such illness each year. How does this estimate compare with the numbers of apparently sporadic cases and outbreaks identified by surveillance?

In the 12 months to 30 September 1999, approximately 7600 cases of infection (mostly gastroenteritis) with *Campylobacter* (5095 cases), *Salmonella* (1366 cases), *Shigella* (109 cases), *Yersinia* (17 cases), *Giardia* (884 cases) and *Cryptosporidium* (119 cases) were reported to the Victorian Department of Human Services.

In the same 12 months, 110 outbreaks or clusters of gastrointestinal illness were reported to the department. These outbreaks affected 2437 persons (a median outbreak size of 14 cases). The majority of outbreaks were attributed to otherwise non-notifiable pathogens (particularly viruses) which were identified or suspected as the causative agent of 56 outbreaks. A substantial proportion of the 28 outbreaks of unknown aetiology may also have been due to viruses.³ Five outbreaks were due to toxin-producing bacteria (*Bacillus cereus*, *Clostridium perfringens*, *Staphylococcus aureus*) and five were due to marine intoxications (ciguatera, paralytic shellfish poisoning, scombroid). Twelve outbreaks were due to *Salmonella* serovars. No reported outbreaks were caused by the most commonly notified cause of gastroenteritis, *Campylobacter* infection.

Most 'gastro' is a mild illness, and fewer than 25 per cent of affected persons seek medical care.^{2,4} Few persons who do visit a doctor with gastroenteritis are investigated by faecal examination,¹ and the yield from routine investigation is low.⁵ Thus only a tiny proportion of cases of gastroenteritis are identified through passive surveillance systems. In published estimates of the burden of bacterial gastroenteritis, community cases have been estimated by applying multipliers from three to 38 to notified cases of *Salmonella* infection, and from 13 to 77 to notified cases of *Campylobacter* infection.^{2,6,7}

We can anticipate greater understanding of the significance of viral gastroenteritis as rapid viral diagnostic tests establish the specific viral aetiology of sporadic illnesses and outbreaks. Viral infections are an important cause of gastroenteritis linked with 'eating out',⁸ but it is often difficult to determine whether infection is spread by meeting, greeting or eating.

'Everyone who gets ill or dies, does so within a few hours of the last thing they ate. This in no way implies causation.'⁹ The food most vivid in the memory of an ill person is not necessarily the cause of their gastroenteritis; food may not even be the vehicle for the infection. Potentially food-borne pathogens may reach humans by routes other than food, such as drinking and recreational water, other infected humans or animals, and the inanimate environment. The proportion of particular infections spread by each route is difficult to determine, and varies from pathogen to pathogen. An estimated 80 per cent of *Campylobacter* infections are food-borne, as are 95 per cent of *Salmonella* infections and 40 per cent of Norwalk-like virus infections.⁶

PREVENTING FOOD-BORNE ILLNESS

Various initiatives to improve food safety 'from the paddock to the plate' are underway both locally and nationally. Many food industry sectors have adopted food safety programs based on a process for risk identification and control known as Hazard Analysis Critical Control Points (HACCP). Additional approaches are also available. Irradiation kills *Campylobacter* on raw poultry, for example, and the World Health Organisation promotes irradiation as a means to reduce human illness due to *Campylobacter* enteritis.¹⁰ Public acceptance of this approach remains problematic.¹¹

But what about the home, where consumers have near complete control over the safety of the food they eat? Hazards lurk in our kitchens.¹² A recent survey of the household food handling of Australians demonstrated widespread deficiencies in our knowledge of simple safety practices and neglect of such practices.¹³

Understanding of food safety issues across communities and generations must precede long-term behavioural change in household food-handling practices. This should start with an appreciation that many raw foods must be handled *as if* contaminated (at the farm or thereafter). Unfortunately, this notion competes with the hopeful misunderstanding that our daily staples and habits bear us no risk of harm. The labels 'fresh' and 'organic' do not necessarily mean 'safe'.

Various agencies provide simple strategies to address the threat of food poisoning from home cooking.^{14,15} Locally produced information is available at the Food Safety Victoria Web site¹⁶ and the Public Health and Development Division (Department of Human Services) Web site¹⁷ (under 'Food and Nutrition').

Simple Tips for Food Safety^{14,15}

Clean	Wash your hands and kitchen surfaces frequently. Wash raw fruit and vegetables in clean water.
Separate	Do not cross-contaminate cooked foods with raw foods.
Cook	Cook (or reheat) food thoroughly.
Chill	Refrigerate all food promptly.

Beyond safe food, remember personal hygiene (see box). Ill persons pose a hazard to others. Symptomatic food-handlers, health-care workers, child-care attendees and school pupils are excluded from work or attendance until they are well.

Victorian regulations require notification of cases of specific enteric infections. Any clinician or laboratory that identifies a notifiable pathogen must notify the department. A timely public health response is only possible if notifications are prompt, particularly for suspected food-borne or water-borne illness.

Advice to Reduce Transmission of Infectious Gastroenteritis

- Take care around family, friends and other workers, because most cases of gastroenteritis pose an infectious risk to close contacts.
- Maintain good personal hygiene, particularly hand-washing.
- Cases of gastroenteritis in the household may contaminate toilets and bathrooms. At such times, clean fixtures thoroughly with a chlorine-based disinfectant to try to reduce the risk of hand-to-mouth transmission of pathogens.
- Cover spilt faeces or vomitus with a towel, wipe up immediately, and clean the area with warm water and detergent. Finally, disinfect the area with a chlorine-based agent.
- Do not prepare meals for others while you are ill with diarrhoea or vomiting.

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Stability of Blood-borne Viruses in the Environment and the Risk of Infection

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The hepatitis B virus (HBV), hepatitis C virus (HCV) and human immunodeficiency virus (HIV) are blood-borne viruses and represent potential occupational hazards to health care workers and environmental hazards to other people. Exposure is usually due to sharps or needlestick injuries. HBV appears to be more efficiently transmitted than HCV, which in turn is more efficiently transmitted than HIV. In the health care setting, immunisation against HBV and adherence to universal blood and body fluid guidelines help to minimize the risk of transmission. The risk of transmission of these viruses following accidental environmental exposure partly depends on their stability in the environment.

The contribution to infection with blood-borne viruses from environmental contamination is difficult to assess. A common question is how long these viruses survive in the environment. The inability to grow HBV and HCV virus in culture has hampered efforts to carry out appropriate assays to determine the risk of infection following accidental environmental exposure to these viruses. Thus we are unable to answer the question of time. In contrast, the replication of HIV in lymphocytes of human origin has enabled quite detailed studies of the stability of HIV under various environmental conditions.

HEPATITIS B VIRUS (HBV)

For HBV, one early report resorted to inoculation of a chimpanzee after leaving dried blood at room temperature for one week.¹ Post-inoculation follow-up showed successful infection of the chimp, indicating the potential for contaminated surfaces to contribute to viral transmission. Further, hepatitis B surface antigen (HBsAg) has been used as a marker of contamination in laboratory environments.² Substantial amounts of HBsAg have been found on laboratory surfaces. However, the excess amount of HBsAg over HBV found in blood probably limits the conclusions that can be drawn from such studies. Nevertheless, the presence of HBsAg in the environment demonstrates the potential for viral transmission.

With the development of polymerase chain reaction (PCR) technology, HBV DNA has also been used as a surrogate marker to indicate the potential for transmission. Indeed, HBV DNA has been found on medical devices such as tonometers after disinfection.³ Again, as with HBsAg, PCR positivity does not necessarily equate with infectivity and may exaggerate the potential for virus viability.

Perhaps the most useful model for studying the stability of HBV has proven to be duck HBV. This virus shares similar biological and structural features with HBV and can be either cultured in duck hepatocytes or directly inoculated into ducklings, which are very sensitive to infection. Professor Yvonne Cossart from Sydney University has used the duck model to evaluate a number of disinfection and sterilisation procedures. These studies show that the virus is very stable in the external environment and that standard disinfection methods are compromised in the presence of blood.⁴

HEPATITIS C VIRUS (HCV)

At the Victorian Infectious Diseases Reference Laboratory (VIDRL), we have used a similar model system for

estimating the stability of HCV. For infectivity evaluation, we have used bovine viral diarrhoea virus, which is from the same virus family and closely related to HCV.

We left blood samples containing both HCV and bovine viral diarrhoea virus at room temperature in loosely sealed tubes for several weeks. At set intervals, we tested tubes by PCR to monitor the genetic stability of HCV RNA; we also tested by infectivity assays for the viability of bovine viral diarrhoea virus. The HCV RNA was very stable, being detected throughout the study period. Importantly, the infectivity assays showed that bovine viral diarrhoea virus was still viable after two weeks. The implication from this work is that HCV, in the right environment (such as blood and other protein-containing physiological solutions), may remain viable for some time in the environment.

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

Unlike HBV and HCV, HIV can be grown in cell culture systems, so infectivity data relate to this virus directly. Many studies have been conducted, examining varying conditions. Variables include investigation of cell-free versus cell-associated virus, dried or aqueous suspensions of virus, different temperatures, and the effect of organic material in the virus suspension.^{5,6}

We have examined the survival of cell-free and cell-associated HIV, in both dried and aqueous suspensions of the virus, and at temperatures of 4°C, room temperature and 37°C. Our results are similar to those of other investigators, and are published on the VIDRL homepage (<http://www.vidrl.org.au>). In general, we found that a blood sample with a relatively high HIV viral load could be infectious for up to eight weeks at 4°C, two to four weeks at room temperature, and one to two weeks at 37°C. Even HIV in dried blood can be potentially infectious under these circumstances.

RISK OF INFECTION

Because it is possible to culture HIV, we can provide more reliable estimates for HIV than for HBV or HCV (neither of which can be cultured) of how long the virus remains potentially infectious in the environment. At ambient environmental temperatures, studies done at VIDRL showed that HIV could survive for up to two weeks in the environment. Using bovine diarrhoea virus, a surrogate virus for HCV, we also showed virus viability in the environment for a similar period. Other studies showed that HBV is likely to be able to survive even longer.

These studies provide a guideline to the potential risks from accidental exposure encountered in the environment, such as the risk from a needlestick injury. However, these are laboratory studies and the real risk depends on many other factors, including:

- Whether a needle found in the environment contained blood.
- If it did, whether the blood was fresh or dried.
- Whether the blood contained any viruses.
- If there were any viruses, and if so, their concentration in the original sample.
- The temperatures to which the virus has been exposed.
- The length of time that a contaminated needle or other sharp object has been in the environment.
- Whether the exposed person has an injury to the skin.

Given all these variables, the risk of infection following accidental environmental exposure is likely to be very low. There is a wealth of data on the risk and management of health care workers following exposure to blood-borne viruses,⁷ but we have been unable to find a published risk estimate following environmental exposure. Further, there is no published case report of any person becoming infected with HIV, HBV or HCV following accidental environmental exposure.

However, a theoretical risk of HIV infection following a random needlestick injury in the health care setting is an estimated one in ten million when the prevalence of HIV infection is 1:1000.⁸ The risk from an environmental exposure is likely to be even lower, and a recent *CDC Update* estimated that the risk of HIV infection after environmental exposure is essentially zero.⁹ Although the

prevalence of HBV and HCV in Australia is higher than that of HIV, similar assumptions could be used in estimating the risk of hepatitis B or C infection following accidental environmental exposure.

These considerations indicate that the risk of infection with any of the blood-borne viruses following unexpected environmental exposure is likely to be very low. However this risk is higher for, health care workers and they should always take appropriate precautions following an accidental injury or when handling potentially infectious material. For information on handling discarded needles see box.

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When you find a syringe

1. Take a hard plastic container with a screw-top lid (for example, a fruit juice or detergent bottle) to where the syringe is located.
2. Pick up the syringe by the barrel end. DO NOT REPLACE SYRINGE CAP because this may increase the risk of injury.
3. Place the syringe in the container and replace the container lid.
4. Call your local council or one of the needle and syringe program hotlines (listed below) to find out where you can dispose of it.

If you suffer a needlestick injury

Gently encourage the injury to bleed and wash it with soap and water. Seek medical advice from your treating doctor.

NEEDLE AND SYRINGE PROGRAM HOTLINES

Southern HIV/Hep/AIDS—Resource Prevention Service (0417 345 750)
– Services Frankston and neighbouring areas

Melbourne Inner City AIDS—Prevention Program (03 9417 5125)
– Services the City of Yarra

North Eastern AIDS—Prevention Program (0418 996 838)

– Services the cities of Banyule, Nillumbik, Darebin and Whittlesea

AIDS Prevention and Support Unit (1800 673 046)

– Services the cities of Greater Dandenong, Casey and Cardinia and neighbouring areas

Western Regions AIDS Prevention (BH: 03 9688 0257, AH: 03 9689 6115)

– Services the City of Maribyrnong

AIDS Prevention and Health Awareness Program (03 9304 2140)

– Services the cities of Moreland, Hume and Moonee Valley

CBD Syringe Disposal Project (03 9654 2198)

– Services the City of Melbourne

AIDS Prevention Program (0418 175 249)

– Services the City of Port Phillip

For more information on the Needle and Syringe Program contact Jeffrey Milne at the Department of Human Services (03 9637 4106 or jeffrey.milne@dhs.vic.gov.au).

Transverse Myelitis Complicating Acute Measles

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A 24-year-old single woman was admitted to hospital on 11 September 1999 with a provisional diagnosis of acute measles. Her symptoms had begun six days earlier with the development of a non-specific febrile illness. After a three-day prodrome she developed a rash, had a prominent dry cough and suffered some abdominal pains. She was seen by her local doctor then by an emergency department of a public hospital. Following blood tests, she was diagnosed as having a 'viral illness' and treated with intravenous fluids before being discharged to go home.

She was admitted the following day (day six of illness) to a private hospital, still unwell with a temperature of 39.5°C and a tachycardia of 120. A generalised maculopapular rash was noted, coalescing on her trunk. She had bilateral conjunctivitis. Her FBE revealed a mild neutropenia of 2.4 with a lymphopenia of 0.2. A chest X-ray was clear. She had not travelled overseas recently or had contact with animals. There were no recognised links with other cases of measles, but she worked in a sales position in her family business and attended social gatherings frequented by large numbers of young people. She believed she had received her childhood vaccinations. A provisional diagnosis of measles was entertained.

The woman was admitted to a single room and treated with IV fluids and paracetamol. She remained febrile and miserable with fever up to 38.5°C until 14 September (day nine). On 15 September her fever settled, but she noted difficulty voiding urine and was found to have urinary retention. During the day she noted increasing difficulty walking, showing unsteadiness and leg weakness. Clinically she had mild leg weakness with brisk reflexes in

her arms and legs and down-going plantar responses.

A sensory level at T3 was detected and she was diagnosed as having transverse myelitis. The woman was treated with methyl prednisolone 1gm IV daily for three days. An MRI of her brain and thoracic cord was normal, with no cord compression or intrinsic cord abnormality detected.

Two weeks following commencement of her neurological condition she made a steady recovery. She was walking short to moderate distances without difficulty and had no problem voiding urine. An NPA performed on 13 September had insufficient cells for direct immuno fluorescence. Culture for measles virus was negative. EBV and rubella serology was consistent with past exposure or vaccination, with positive IgG and negative IgM. Her measles serology was suggestive of recent infection with a positive IgG and IgM.

Measles pneumonia and post-measles encephalomyelitis are recognised severe complications of measles. Measles itself has become an uncommon disease and, as in this case, may not be recognised early in the illness. Multiple presentations to local doctors and emergency centres increase the risk of the virus spreading to susceptible populations, including medical staff and other patients. This case reminds us that measles not only is a severe illness in its own right — with a high rate of hospitalisation — but can also have severe complications. Clinicians need to be alert for these late immune mediated complications, because early treatment with immunomodulating agents such as steroids may reduce the degree of damage to the brain or spinal cord.

Immunisation Update

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CHILDHOOD IMMUNISATION REGISTER REPORT

Data cited in this report are based on the Australian Childhood Immunisation Register (ACIR) Coverage Report. The ACIR report measured immunisation coverage at 30 June 1999 for children aged 12–<15 months and 24–<27 months at 31 March 1999. Only vaccines administered before 12 months of age were included in the coverage calculation for the former age group, and only those vaccines administered before 24 months of age were included in the coverage calculation for the latter age group.

The following table (Table 1) groups immunisation coverage by local government area for the two birth cohorts. For a copy of the ACIR report listing immunisation coverage against individual vaccines for each local government area, contact Michele Wend (03 9637 4143 or michele.wend@dhs.vic.gov.au).

Congratulations to immunisation providers in the municipalities of Ararat, Hindmarsh, Horsham, Moynes, the Pyrenees and West Wimmera for achieving coverage of 95 per cent or higher for children at 12 months of age. West Wimmera gets a special mention for achieving 100 per cent

coverage for this age group, as does Ararat for increasing its coverage from 84 per cent at the last estimate (31 March 1999) to 97 per cent.

The percentage of local government areas with coverage below 80 per cent for children at 12 months fell from 15 per cent at 31 March 1999 to 8 per cent in this reporting period. Thirty seven per cent of all local government areas achieved coverage of 90 per cent or higher, compared with only 26 per cent in the previous reporting period. These great results reflect the cooperative efforts of all immunisation providers working to improve coverage in their respective areas.

For children at 24 months of age, the coverage data also show improvement. Seventeen per cent of municipalities achieved a coverage of 80 per cent or greater, compared with 12 per cent in the last reporting period.

Overall, coverage in Victoria of children at 12 months of age increased by 1.4 per cent over the three-month period — up from 86.5 per cent to 87.9 per cent. Coverage of children aged 24 months of age increased from 72 per cent to 74.7 per cent.

Table 1: Childhood Immunisation Coverage, by Local Government Area (LGA)—Victoria, 1999

Age Group	% Fully Immunised	Local Government Area (LGA)	Total LGAs (% LGAs)
12–<15 months	95%+	Ararat (RC), Hindmarsh (S), Horsham (RC), Moyne (S), Pyrenees (S), West Wimmera (S)	6 (8%)
	90–94%	Ballarat (C), Banyule (C), Baw Baw (S), Campaspe (S), Casey (C), Central Goldfields (S), Corangamite (S), Frankston (C), Gannawarra (S), Golden Plains (S), Indigo (S), Knox (C), Loddon (S), Macedon Ranges (S), Melton (S), Mildura (RC), Mitchell (S), Strathbogrie (S), Swan Hill (RC), Whitehorse (C), Whittlesea (C), Wodonga (RC), Yarriambiack (S)	23 (29%)
	85–89%	Alpine (S), Bass Coast (S), Bayside (C), Boroondara (C), Brimbank (C), Colac-Otway (S), Delatite (S), Glenelg (S), Greater Bendigo (C), Greater Geelong (C), Greater Shepparton (C), Hobsons Bay (C), Hume (C), Kingston (C), Manningham (C), Maroondah (C), Monash (C), Moonee Valley (C), Moorabool (S), Moreland (C), Mornington Peninsula (S), Murrindindi (S), Nillumbik (S), Southern Grampians (S), Stonnington (C), Warrnambool (C), Wyndham (C), Yarra (C), Yarra Ranges (S)	29 (37%)
	80–84%	Buloke (S), Cardinia (S), Darebin (C), East Gippsland (S), Glen Eira (C), Greater Dandenong (C), Hepburn (S), La Trobe (S), Maribyrnong (C), Melbourne (C), Moira (S), Northern Grampians (S), South Gippsland (S), Wangaratta (RC)	14 (18%)
	<80%	Mt Alexander (S), Port Phillip (C), Queenscliffe (B), Surf Coast (S), Towong (S), Wellington (S)	6 (8%)
24–<27 months	95%+		
	90–94%	Horsham (RC), West Wimmera (S), Yarriambiack (S)	3 (4%)
	85–89%	Ararat (RC), Gannawarra (S), Pyrenees (S)	3 (4%)
	80–84%	Ballarat (C), Golden Plains (S), Mildura (RC), Nillumbik (S), Stonnington (C), Towong (S), Wodonga (RC)	7 (9%)
	<80%	Alpine (S), Banyule (C), Bass Coast (S), Baw Baw (S), Bayside (C), Boroondara (C), Brimbank (C), Buloke (S), Campaspe (S), Cardinia (S), Casey (C), Central Goldfields (S), Colac-Otway (S), Corangamite (S), Darebin (C), Delatite (S), East Gippsland (S), Frankston (C), Glen Eira (C), Glenelg (S), Greater Bendigo (C), Greater Dandenong (C), Greater Geelong (C), Greater Shepparton (C), Hepburn (S), Hindmarsh (S), Hobsons Bay (C), Hume (C), Indigo (S), Kingston (C), Knox (C), La Trobe (S), Loddon (S), Macedon Ranges (S), Manningham (C), Maribyrnong (C), Maroondah (C), Melbourne (C), Melton (S), Mitchell (S), Moira (S), Monash (C), Moonee Valley (C), Moorabool (S), Moreland (C), Mornington Peninsula (S), Mt Alexander (S), Moyne (S), Murrindindi (S), Northern Grampians (S), Port Phillip (C), Queenscliffe (B), South Gippsland (S), Southern Grampians (S), Strathbogrie (S), Surf Coast (S), Swan Hill (RC), Wangaratta (RC), Warrnambool (C), Wellington (S), Whitehorse (C), Whittlesea (C), Wyndham (C), Yarra (C), Yarra Ranges (S)	65 (83%)

PRE-ADOLESCENT HEPATITIS B IMMUNISATION PROGRAM IN 1998

In 1998, a hepatitis B immunisation program commenced for year 7 students in Victorian secondary schools. The coverage data tabulated below (Table 2) are for the final dose of the three-dose course of hepatitis B immunisations (although data were not available for six municipalities). We gratefully acknowledge the assistance of the Clinical

Epidemiology and Biostatistics Unit of the Royal Children’s Hospital in compiling the data.

Overall coverage for the third dose of hepatitis B vaccine in Victorian secondary schools was 76 per cent, with metropolitan schools achieving 74.6 per cent and rural schools achieving 79.6 per cent. The results achieved in this first year of the program are excellent, with 26 municipalities (36 per cent) achieving coverage of 85 per cent or higher.

Table 2: Coverage for Third-dose Hepatitis B Immunisation in Year 7 students, by Local Government Area (LGA)—Victoria 1998

% Fully Immunised	Local Government Area (LGA)	Total LGAs (% LGAs)
95%+	Buloke (S), Gannawarra (S), Hepburn (S), Horsham (RC), Moyne (S), Murrindindi (S), Northern Grampians (S)	7 (10%)
90–94%	Ararat (RC), Delatite (S), Hindmarsh (S), Loddon (S), Towong (S), West Wimmera (S)	6 (8%)
85–89%	Alpine (S), Ballarat (C), Campaspe (S), Central Goldfields (S), Corangamite (S), La Trobe (S), Melton (S), Monash (C), Moreland (C), Southern Grampians (S), Wangaratta (RC), Whittlesea (C), Wodonga (RC)	13 (18%)
80–84%	Banyule (C), Baw Baw (S), Boroondara (C), Colac-Otway (S), Frankston (C), Greater Bendigo (C), Melbourne (C), Moira (S), Moorabool (S), Nillumbik (S), Pyrenees (S), South Gippsland (S), Stonnington (C), Strathbogrie (S), Warrnambool (C), Wellington (S), Yarra Ranges (S)	17 (24%)
<80%	Bass Coast (S), Bayside (C), Brimbank (C), Cardinia (S), Casey (C), Darebin (C), Glen Eira (C), Glenelg (S), Greater Dandenong (C), Greater Geelong (C), Hobsons Bay (C), Hume (C), Indigo (S), Kingston (C), Knox (C), Macedon Ranges (S), Manningham (C), Maribyrnong (C), Maroondah (C), Mildura (RC), Mitchell (S), Moonee Valley (C), Mornington Peninsula (S), Port Phillip (C), Swan Hill (RC), Whitehorse (C), Wyndham (C), Yarra (C), Yarriambiack (S)	29 (40%)

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 initiated on the basis of clinical suspicion in the absence of laboratory confirmation. Prompt notification of infectious diseases is an integral component of prompt public health action. Please do not delay. To notify, call 1300 65 1160 or fax 1300 65 1170.

This section includes a summary of infectious disease notifications received until 30 September 1999 and historical comparisons with 1998 data at both the State and regional level (Table 4). There have been no notifications of anthrax, Australian arbo encephalitis, botulism, diphtheria, leprosy, plague, poliomyelitis, rabies, primary amoebic meningo-encephalitis, typhus, viral haemorrhagic fevers or yellow fever. For summary data at local government level, contact Martyn Kirk, Disease Control Section, Department of Human Services (03 9637 4121). Data may be subject to revision.

This section now also includes surveillance data from the Victorian Infectious Diseases Reference Laboratory (VIDRL). The VIDRL Fortnightly Surveillance Bulletin can be accessed online at <http://www.vic.gov.au/vidrl/>. For surveillance data related to sexually transmissible diseases, see page 79. General information on the control of infectious diseases (the Blue Book) can be found online at http://www.dhs.vic.gov.au/phd/hprot/inf_dis/bluebook/index.htm

ENHANCED MEASLES SURVEILLANCE

The onset of illness for the last case in the 1999 measles outbreak in Victoria was 2 May 1999. Since then, two sporadic cases of measles have been laboratory confirmed and two clusters of disease have been identified.

SPORADIC CASE 1

A 12-year-old male from East Java arrived in Melbourne on 8 May 1999. He was visiting family. He developed prodromal symptoms on 15 May and was serologically confirmed as having measles. The case's mother could not identify a source of infection, and the child had no history of prior immunisation with a measles-containing vaccine. No secondary cases were related to this case.

SPORADIC CASE 2

A 24-year-old male from Mulgrave developed prodromal symptoms on 6 May 1999, and was later serologically confirmed as having measles. He had recently returned from a trip to the Gold Coast where he had attended a large theme park on 25 May 1999. A ride attendant who worked on that day also developed measles, but no source for either of these cases was identified. The case did not report having received a measles-containing vaccine, and no secondary cases were related to this case.

FERNTREE GULLY CLUSTER

A 16-year-old male arrived in Melbourne following a flight from London, via Singapore, on 2 August 1999. On holidays, he was staying with his brothers, aged 22 years and 24 years, at Ferntree Gully. He developed prodromal symptoms on the 17 August 1999, and was at home when his brothers held a party, attended by 40 young adults, on 20 August 1999. The Disease Control Section undertook active surveillance for measles in the party attendees, and only one further case was identified — a 20-year-old female whose onset of illness was 29 August 1999.

A further four cases were identified in people with links to the Ferntree Gully area, although no direct epidemiological link between cases could be identified. A 25-year-old female (with onset of illness on 2 September 1999), a 29-year-old female (with onset of illness on 3 September 1999) and a 23-year-old female (with onset of illness on 7 September 1999) were all laboratory confirmed cases. The last case developed transverse myelitis as a complication of her infection.

The fourth case, a 28-year-old male surgical registrar who works in a teaching hospital that services the Ferntree Gully area, experienced onset of illness on 24 September 1999. No source for his infection was found, despite follow up of cases of febrile rash illness that were seen in the hospital's Emergency Department during the period in which he would have acquired infection.

PUCKAPUNYAL SAFE HAVEN CLUSTER

There have been 11 cases of measles in the East Timorese evacuees at Puckapunyal. The cases have been aged 1–13 years, and the onset of illness occurred from 21 September to 5 October 1999. Measles cases have also been reported at the Darwin reception center and the Leeuwin Haven in Western Australia. Dates of movement and onset dates indicate that no transmission of measles occurred among the East Timorese at Puckapunyal, with all cases originally infected in Darwin.

A 26-year-old volunteer who was not immunised attended the Puckapunyal Haven on 20 September 1999 and subsequently developed measles, with onset of illness on 1 October 1999. She spent four days in Bendigo Hospital with her illness. Her 10-month-old daughter received immunoglobulin on 6 October 1999, but went on to develop symptoms of measles infection on 14 October 1999.

This cluster of cases in the East Timorese provides an interesting contrast to the epidemiology of measles in the general Victorian community. All of the 11 cases in the East Timorese occurred in age groups in which we no longer see cases in Victoria. Lack of access to immunisation services in East Timor has left the younger age groups non-immune. The only transmission at the Haven involved the Australian volunteer and subsequently her daughter (who was too young to be immunised and who would not have received any maternal antibody from her mother).

Both of the sporadic cases and the index case in each of the clusters imported their measles infection from outside Victoria. Subsequent transmission of infection only occurred to young adults and those too young to be immunised. These findings provide evidence of episodic interruption of measles transmission within Victoria, and reinforce both the two-dose mumps/measles/rubella immunisation policy and the success of the Measles Control Campaign held last year.

OUTBREAKS OF GASTROINTESTINAL ILLNESS

Overall, 27 outbreaks of gastrointestinal illness were reported to the Disease Control Section (Department of Human Services) during July–September 1999 (Table 1).

A medical practitioner reported an outbreak of gastroenteritis to the Disease Control Section in July. The practitioner had a patient who had become ill after attending a ball two days prior. The doctor also reported that other attendees at the function were ill. The function organiser was contacted and a list of guests was obtained, from which 75 people were randomly selected and interviewed. Thirty-four of the 75 interviewees reported gastrointestinal illness. One hundred per cent of cases had diarrhoea, 82 per cent had abdominal cramps, and 17 per cent reported vomiting. The incubation period ranged between five and 17 hours after consumption of the meal.

Those people who ate the chicken vol au vent were nine times more likely to be ill than those who did not eat this meal. Five faecal specimens were collected, and *Clostridium perfringens* enterotoxin was detected in two specimens that had high levels of *Clostridium perfringens* bacteria. When the preparation of the suspect food was investigated, it seemed that cooling and reheating the vol au vents might have been the problem.

An outbreak of gastrointestinal illness consisting mainly of diarrhoea was notified to the Disease Control Section in early July. Cases had dined at a restaurant and consumed a smorgasbord meal six days before the outbreak was notified. The incubation period was four to 19 hours after consuming the suspect meal. A total of 38 people were interviewed, of whom 14 were ill.

Epidemiological analysis of information contained in the questionnaires revealed that those who consumed the chicken Vietnamese dish were 18 times more likely to have been ill than those who did not eat it. The relative risk from eating rice was also high (RR 12) but because most people who ate the rice also ate the chicken, it is not possible to be certain about which of these foods was the vehicle of infection. Six faecal samples were collected, but no bacterial or viral pathogens were isolated. No leftover foods were available for sampling. After inspecting the premises and investigating the chicken dish preparation, found food-handling procedures that were conducive to the growth of pathogens were identified.

Nine gastrointestinal illness outbreaks in child care centres have been reported over the three months to October. Most have been viral outbreaks, with some secondary cases typically in household contacts. Council environmental health officers have been diligent in ensuring that the centres adhere to good infection control procedures; in particular, the officers provide advice on handwashing, nappy changing and disinfection procedures in an outbreak setting.

HEPATITIS A

Since late 1998, notifications of hepatitis A in injecting drug users have increased in some parts of Melbourne, particularly the southern suburbs (Figure 1). Injecting drug users represent approximately 40 per cent of the total number of cases of hepatitis A cases in Victoria for the year to date, and other States have also recently documented outbreaks of hepatitis A among injecting drug users.

Table 1: Outbreaks of Gastrointestinal Illness Reported to the Disease Control Section, 1 July to 30 September 1999

Setting	Outbreaks	Persons Affected	Pathogen/Toxin (No. of Outbreaks)
Restaurant/ reception	7	77	Unknown (5) <i>Clostridium perfringens</i> (2)
Privately catered function	1	35	Norwalk virus (1)
Aged/disability/health care institution	6	105	Suspect viral (5) Norwalk virus (1)
Family/social gathering	2	10	Unknown (2)
Children's service	9	148	Rotavirus (6) Norwalk virus (1) Suspect viral (1) Unknown (1)
Workplace	1	8	Unknown (1)
Conference	1	11	Suspect viral (1)
Total	27	394	

Good personal hygiene, proper food-handling practices and timely use of normal immunoglobulin can prevent the transmission of hepatitis A. The exact mode of transmission among injecting drug users is unclear, which hampers efforts to prevent the spread of the disease.

The *Guidelines for the Control of Communicable Diseases* recommend normal immunoglobulin (0.02 millilitres per kilogram of body weight) for some contacts of hepatitis A — that is, household and sexual contacts, staff and children in close contact with a case in a child care centre, and other food handlers in an establishment (if the case is a food handler). Immunoglobulin must be given within seven to ten days of exposure to be effective.

Figure 1: Notifications of Hepatitis A among Injecting Drug Users (IDUs) and their Contacts and Non-IDUs, in Victoria, January–September 1999

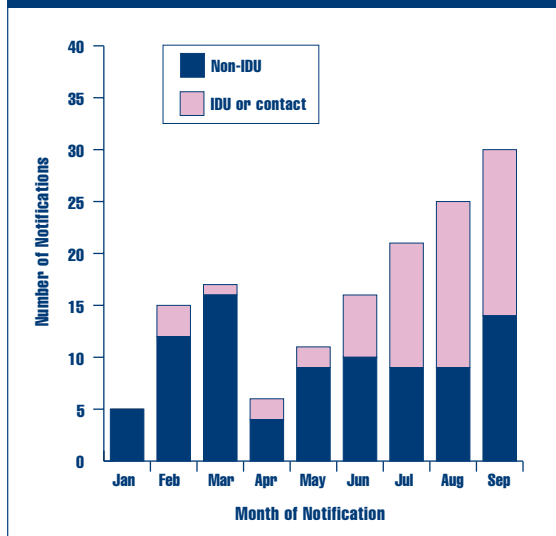
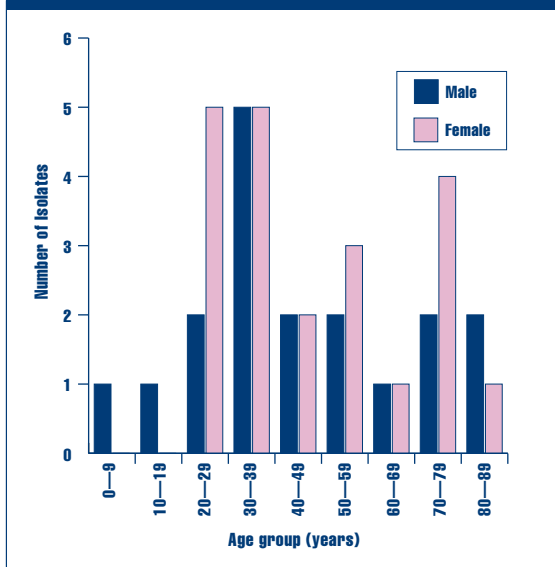


Figure 2: New *M. tuberculosis* Isolates from Victorian Residents, by Age Group and Sex, VIDRL Mycobacterium Reference Laboratory, March–June 1999



TUBERCULOSIS

The Mycobacterium Reference Laboratory at VIDRL prepared this report. Given the slow-growing nature of *Mycobacterium* spp, the report is limited to the second quarter of 1999. Most specimens (both primary and referred) and isolates are from Victorian patients. The majority of non-Victorian specimens originate in the Northern Territory and the Solomon Islands.

COMMENTS

- One fully sensitive *M. tuberculosis* strain was isolated from blood and spinal collection of a 52-year-old, HIV-positive male.
- *M. bovis* was isolated from the bronchial washings and bone marrow of a 53-year-old male with miliary tuberculosis
- *M. kansasii* was isolated from the sputum and bronchial washings from two male patients aged 62 years and 28 years respectively.
- *M. marinum* was isolated from an arm biopsy of a 32-year-old male.
- Two resistant *M. Tuberculosis* strains were isolated from Victorian patients, and one from a South Australian patient.

Table 2: Specimens Submitted to the VIDRL Mycobacterium Reference Laboratory, by Month, March–June 1999

Primary Specimens	<i>M. tb</i> Isolates	New Victorian <i>M. tb</i> Isolates	Non <i>M. tb</i> Isolates	Negatives	Total
April	19	4	12	412	443
May	8	4	16	382	406
June	8	5	11	347	366
Referred Specimens	<i>M. tb</i> Isolates	New Victorian <i>M. tb</i> Isolates	Non <i>M. tb</i> Isolates	Negatives	Total
April	41	14	75		116
May	8	5	53		61
June	15	7	62		77
Grand Total	99	39	229	1141	1469

Table 3 Extra-pulmonary *M. tuberculosis* Isolates and Resistant Isolates Detected by VIDRL Mycobacterium Reference Laboratory, by Month, March–June 1999

Site	April	May	June
Pulmonary	13	6	5
Extra-pulmonary	5	3	7
Extra-pulmonary Site Details	Lymph Node (x3) CSF Urine	Lymph node (x2) Omentum*, Ovary*, Peritoneal fluid*, Pleural fluid*	Lymph node (x4) Endometrium Lumbar mass, Blood/Paraspinal collection
Antibiotic Resistance	No resistant isolates	1x resistance to Isoniazid	1x resistance to Isoniazid 1x resistance to Pyrazinamide

* Isolated from one patient.

Table 4: Notifications of Infectious Diseases, by Department of Human Services Region, Victoria, 1 January to 31 September 1999 and Historical Comparisons

Disease	Barwon-South		Grampians		Loddon-Mallee		Hume		Gippsland		Western		Northern		Eastern		Southern		Victoria		Total	
	1999/98	1998/97	1999/98	1998/97	1999/98	1998/97	1999/98	1998/97	1999/98	1998/97	Metropolitan	1999/98	Metropolitan	1999/98	Metropolitan	1999/98	Metropolitan	1999/98	Metropolitan	1999/98		1998/97
Blood-borne diseases																						
Hepatitis B—acute	0	0	1	2	2	2	3	3	1	5	2	13	10	8	12	9	4	14	30	55	69	92
Hepatitis B—chronic/unknown	14	18	8	7	19	16	14	18	15	19	15	500	357	370	284	287	107	532	548	1855	1456	2203
Hepatitis C	234	220	101	93	183	188	200	152	193	193	636	617	832	737	832	560	249	1251	1568	5025	5370	6883
Enteric diseases																						
Amoebiasis	3	1	6	1	1	1	3	1	0	0	1	15	10	15	12	16	5	23	26	83	61	84
Campylobacter infection	172	138	98	76	100	120	171	133	220	158	485	344	424	500	424	622	229	944	971	3354	2650	4115
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2
Food/water/environmental																						
Cryptosporidium	2	1	3	1	7	3	13	11	10	14	4	4	15	13	44	7	14	13	89	29	73	221
—Other	5	4	3	4	5	2	14	2	3	5	8	31	5	55	8	36	15	54	34	101	14	307
Giardiasis	60	48	30	33	36	35	52	38	35	27	63	98	124	109	124	126	75	236	296	14	23	797
Haemolytic Uraemic Syndrome	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	2	1
Hepatitis A	5	1	4	3	3	30	4	3	8	14	14	17	18	18	16	22	4	66	49	2	5	146
Listeriosis	1	0	1	1	0	1	1	0	0	0	2	1	0	0	3	1	0	4	4	0	0	10
Paratyphoid	0	0	0	0	0	0	0	0	0	0	2	2	0	2	0	0	2	3	0	0	5	4
Salmonellosis	81	55	44	27	30	51	42	31	43	39	144	101	168	144	144	140	60	282	260	35	1014	1128
Shigellosis	2	3	1	1	2	2	3	1	3	2	12	11	11	11	16	13	8	31	47	3	1	81
Typhoid	0	0	0	0	0	0	0	0	0	0	3	4	3	5	1	1	0	5	1	0	14	10
Verotoxin-producing E.coli	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0	1	1	0	1	4
Yersiniosis	0	1	0	1	0	0	0	1	0	0	6	6	5	5	2	1	2	4	10	1	16	24
Other infectious notifiable diseases																						
Legionellosis	2	2	0	0	1	0	0	0	1	0	13	19	5	18	5	8	2	15	6	1	59	35
Meningococcal infection	7	2	3	2	3	2	7	6	5	4	11	3	16	16	4	15	4	30	17	2	3	99
Tuberculosis	9	5	4	4	5	2	11	4	2	2	66	56	34	56	34	46	11	77	68	3	0	279
Vaccine-preventable diseases																						
Type b	0	0	0	0	0	0	0	0	0	0	1	0	0	0	2	1	0	1	1	0	0	3
Measles	0	0	2	0	2	0	1	2	2	2	37	2	31	4	4	10	8	10	14	4	2	99
Mumps	0	0	4	1	1	2	7	2	1	3	14	8	9	9	9	4	2	7	10	6	0	53
Pertussis	26	34	22	27	30	66	52	47	89	212	93	86	73	73	91	99	53	124	312	25	21	633
Rubella	4	6	3	0	2	3	9	8	0	8	11	19	16	16	28	21	10	26	69	4	7	96
Tetanus	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	1
Vector-borne diseases																						
Forest	0	1	0	0	1	0	3	2	5	7	0	0	0	1	0	0	0	0	1	2	2	12
Arbovirus—Flavivirus	0	0	0	2	0	2	0	2	0	1	0	1	1	1	1	0	1	0	6	0	1	17
Arbovirus—not further specified	0	0	3	0	6	0	4	3	30	7	0	0	2	1	2	0	0	1	0	3	0	48
Arbovirus—Ross River	6	3	15	5	43	6	41	1	65	5	7	0	16	16	2	13	1	30	6	13	1	2491
Arbovirus—Sindbis	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Malaria	2	0	2	1	3	1	2	6	2	1	4	4	7	7	17	9	6	22	28	5	4	58
Zoonoses																						
Brucellosis	0	0	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0	1	0	0	2
Hydatid disease	2	0	0	1	0	0	1	0	1	1	3	3	2	3	3	1	0	2	6	2	0	14
Leptospirosis	5	3	0	0	3	0	5	1	3	0	0	0	0	0	0	0	0	2	0	2	0	16
Psittacosis	2	0	4	1	3	3	3	2	1	0	4	3	5	5	5	10	2	18	7	1	0	53
Q fever	4	4	0	2	3	2	5	4	2	1	1	1	0	0	1	0	1	3	1	5	1	23
Taeniasis	1	0	0	0	0	0	0	0	0	0	2	1	4	4	2	1	0	0	0	0	0	8
Total	650	550	362	296	495	541	676	485	748	727	2149	1003	2269	2137	2080	874	3829	4491	1355	1561	14613	18352
Population	327210		201291		281356		238729		234077		571339		741434		944278		1065350		4605064			

Notes

1. Verotoxin-producing *E. coli* was made notifiable from 27 October 1998
2. The data are preliminary figures only and may be subject to revision
3. ABS estimated residential population data as at July 1997

Victorian STD Surveillance Report

This report is produced by the Epidemiology and Social Research Unit of the Macfarlane Burnet Centre for Medical Research, on behalf of the Disease Control Section (Department of Human Services), in cooperation with the Melbourne Sexual Health Centre; the Microbiological Diagnostic Unit, University of Melbourne; the Victorian Infectious Diseases Reference Laboratory; and the Victorian Collaborative Group on HIV and AIDS Surveillance. The Victorian Department of Human Services and the Victorian Health Promotion Foundation jointly fund the Epidemiology and Social Research Unit to conduct surveillance and related research into sexually transmissible diseases and blood-borne viruses.

Research Unit, Macfarlane Burnet Centre for Medical Research, on (03) 9282 2290 or the Disease Control Section, Victorian Department of Human Services, on (03) 9637 4184.

All data in this report are provisional and subject to revision as further information becomes available.

SUMMARY

- HIV and AIDS—notifications have remained stable for HIV and slightly increased for AIDS.
- Chlamydia—notifications continue to rise, the highest number of infections were reported in the 20–29 year age group
- Reported cases of gonorrhoea infection decreased by 31 percent this quarter although the overall trend is still upwards.

- Syphilis—notifications have increased by a total of 14 for the quarter

ACQUIRED IMMUNE DEFICIENCY SYNDROME

Notifications for AIDS increased slightly this quarter. All 12 cases notified between 1 July and 30 September 1999 occurred in men. Six occurred in men who have sex with men and four in heterosexual men.

Table 1: Number of Cases of AIDS Notified in Victoria, 1 July 1999 to 30 September 1999, 1 October 1998 to 30 September 1999 and Cumulative

Exposure Category	Cases Notified Jul 99–Sep 99		Cases Notified Jul 98–Sep 99		Cumulative Notifications to 30 Sep 99		
	Male	Female	Male	Female	Male	Female	Total*
homo/bisexual	6	0	41	0	1469	0	1473
Male homo/bisexual & IDU	0	0	3	0	94	0	97
IDU	0	0	1	1	16	10	26
Heterosexual	4	0	6	1	56	45	101
Person from specified country	1	0	1	0	12	6	18
Haemophilia/related disorder	0	0	2	0	36	1	37
Transfusion recipient	0	0	1	1	8	5	13
Other	1	0	1	0	1	1	2
Under investigation	0	0	0	0	11	0	11
Unavailable		0	4	0	6	2	8
Total	12	0	60	3	1709	70	1786

*Includes seven persons for whom gender is given as transsexual

Two AIDS-related deaths were notified during the third quarter of 1999. The total number of AIDS-related deaths for the year ending 30 September 1999 was 24. All but one of these deaths were in males.

Table 2: Number of Deaths from AIDS in Victoria, 1 July 1999 to 30 September 1999, 1 October 1998 to 30 September 1999 and Cumulative

Exposure Category	Deaths 1 Jul 99–Sep 99		Deaths Jul 98–Sep 99		Cumulative Deaths 30 Sep 99		Total*
	Male	Female	Male	Female	Male	Female	
Male homo/bisexual	2	0	19	0	1171	0	1174
Male homo/bisexual & IDU	0	0	0	0	69	0	72
IDU	0	0	0	0	12	5	17
Heterosexual	0	0	1	1	28	35	63
Person from specified country	0	0	1	0	5	3	8
Haemophilia/related disorder	0	0	0	0	26	1	27
Transfusion recipient	0	0	0	0	6	4	10
Other	0	0	0	0	0	0	0
Under investigation	0	0	0	0	9	0	9
Unavailable	0	0	2	0	3	2	5
Total	2	0	23	1	1329	50	1385

*Includes 6 people for whom gender is given as transsexual

HUMAN IMMUNODEFICIENCY VIRUS INFECTION

There were 31 HIV notifications during the third quarter of 1999, 28 males and three females. Male-to-male sexual contact was the principal exposure category accounting

for 68 percent of notifications. The majority of cases, 68 percent, were notified in people aged between 20 and 40 years. The average age was 34 years (range: 11–72 years). Two cases were notified in children under the age of 13.

Table 3: Number of Cases of HIV Notified in Victoria by Age Group, 1 July 1999 to 30 September 1999, 1 October 1998 to 30 September 1999 and Cumulative

Age Group	Cases Notified Jul 99–Sep 99		Cases Notified Oct 98–Sept 99		Cumulative Notifications to 30 Sep 99		Total*
	Male	Female	Male	Female	Male	Female	
0–12	1	1	3	1	34	10	44
13–19	1	0	4	0	99	11	111
20–29	8	2	40	4	1430	90	1535
30–39	11	0	46	4	1383	52	1442
40–49	4	0	23	1	588	25	615
50+	3	0	19	1	286	22	308
Unavailable	0	0	0	0	101	1	117
Total	28	3	135	11	3921	211	4172

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

Table 4: Number of Cases of HIV Notified in Victoria by Exposure Category, 1 July 1999 to 30 September 1999, 1 October 1998 to 30 September 1999 and Cumulative

Exposure Category	Cases Notified Jul 99–Sep 99		Cases Notified Oct 98–Sep 99		Cumulative Notifications to 30 Sep 99		Total*
	Male	Female	Male	Female	Male	Female	
Male homo/bisexual	13	0	84	0	3208	0	3221
Male homo/bisexual & IDU	6	0	13	0	191	0	193
IDU	1	0	6	0	104	35	142
Heterosexual	5	2	15	6	145	127	272
Person from specified country	0	0	8	3	53	24	77
Haemophilia/related disorder	1	0	1	0	100	1	101
Transfusion recipient	0	1	1	2	4	15	35
Other	1	0	1	0	3	7	11
Under investigation	1	0	3	0	3	0	3
Unavailable		0	3	0	93	2	117
Total	28	3	135	11	3921	211	4172

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

The proportion of notifications for HIV from people who identify as IDU has remained stable, accounting for 4.1 percent of the total for the year from 1 October 1998 to

30 September 1999. Historically the proportion of notifications for HIV in which IDU was the principal exposure category has ranged from 0 percent to 5.9 percent.

Figure 1: Proportion of Notifications for Which IDU was the Principal Exposure Category, 1983 to September 1999, Victoria

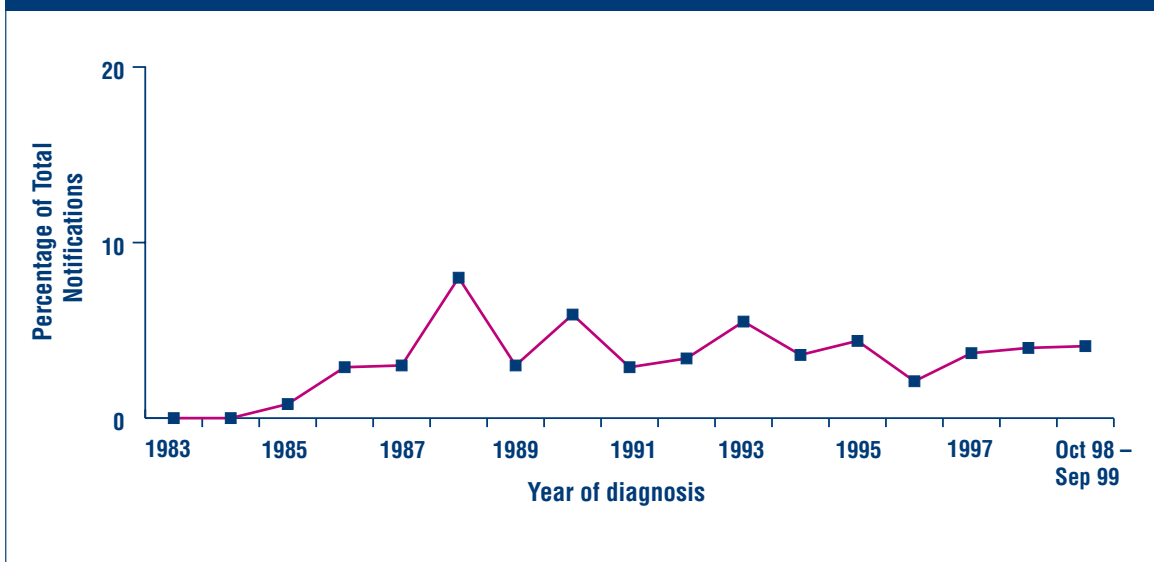


Table 5: Number of Notified Cases of HIV in Victoria by Time Since Last Negative Test or Seroconversion Illness, 1 July 1999 to 30 September 1999 and 1 October 1998 to 30 September 1999

Time Between HIV Diagnosis and Negative Test and/or Seroconversion Illness	Cases diagnosed Jul 99–Sep 99			Cases diagnosed Oct 98–Sep 99		
	Male	Female	Total*	Male	Female	Total*
Less than 1 year	5	1	6	23	2	25
1 year to less than 3 years	6	0	6	3	1	4
3 or more years	5	0	5	1	0	1
No test or illness information provided	12	2	14	108	8	116
Total	28	3	31	135	11	146

*includes one case for whom gender is not reported

Of the 31 new HIV infections diagnosed this quarter, six (19 percent) had had a negative HIV test and/or seroconversion illness in the previous 12 months and are regarded as incident infections. In the 12 months preceding September 1999 there were 25 incident infections, representing 17 percent of the total.

CHLAMYDIA

There has been a small decrease in notifications for chlamydia in the third quarter of this year. However, the 12 month data show a 7 percent increase. The 1999 calendar year-to-date figures indicate a 22 percent (n=524) increase on the 1998 year-to-date figures. The highest number of infections (n=515) were diagnosed in people under the age of 30.

Table 6: Number of Cases of *Chlamydia trachomatis* Notified in Victoria by Age and Sex, 1 July 1999 to 30 September 1999 and 1 October 1998 to 30 September 1999, Victoria

Age Group	Chlamydia Notifications Jul–Sep 99*				Chlamydia Notifications Oct 98–Sep 99			
	Male	Female	Unknown	Total	Male	Female	Unknown	Total
0–12 years	4	3	0	7	12	10	0	22
13–19 years	11	77	0	88	40	287	0	327
20–29 years	156	268	1	425	628	1093	3	1724
30–39 years	74	76	1	151	316	267	1	584
40–49 years	24	20	1	45	106	79	1	186
50+ years	10	4	0	14	40	17	0	57
Unavailable	0	3	0	3	2	6	0	8
Total	279	451	3	733	1144	1759	5	2908

*Include 6 cases with *C. trachomatis* eye infections

GONORRHOEA

Notifications for gonorrhoea during the third quarter decreased by 31 percent compared with the previous quarter. This reduction was made almost exclusively by a reduction in cases of urethral and rectal gonorrhoea notified in gay and bisexual males. The majority of cases,

56 percent (n=83) were in men who have sex with men, although this compares with 67 percent (n= 142) for the previous quarter. Heterosexual males accounted for 30 percent (n=44) of cases, an increase from 21 percent (n=44) last quarter. The number of cases acquired from overseas declined by almost half (47 percent, n=8).

Table 7: Number of Isolations of *N. gonorrhoeae* in Victoria by Sexual Orientation and Gender, 1 July to 30 September 1999

Gender		Site of Isolation						Total
		Urethral	Vaginal	Cervical	Rectal	Pharyngeal	Other	
Heterosexual	Male	41	0	0	3	0	0	44
	Female	0	5	2	0	0	0	7
Homo/bisexual	Male	56	0	0	19	8	0	83
	Female	0	0	1	0	0	0	1
Unavailable	Male	12	0	0	0	0	1	13
	Female	0	0	0	0	0	0	0
Total		109	5	3	22	8	1	148

Table 8: *N. gonorrhoeae* Notifications by Age Group, 1 July 1999 to 30 September 1999

Age Group	Male	Female	Total	Proportion (per cent)
0–12 years	1	0	1	1
13–19 years	8	0	8	5
20–29 years	42	7	49	33
30–39 years	69	0	69	47
40–49 years	15	1	16	11
50+ years	5	0	5	3
Unavailable	0	0	0	0
Total	140	8	148	100

SYPHILIS

Table 9: Number of Syphilis Notifications in Victoria by Sex and Category of Disease, 1 July 1999 to 30 September 1999

	Male	Female	Total*
Primary syphilis	1	0	2
Secondary syphilis	1	0	1
Latent syphilis–early	0	1	1
Latent syphilis–late	1	2	3
Latent syphilis–unknown	6	2	8
Other late syphilis	1	2	3
Neurosyphilis	1	1	2
Past treated	11	3	14
Unknown	18	6	25*
Total	40	17	58

* includes 1 person for whom gender was unknown

Reports on surveillance data for STDs in Victoria are generally available approximately six weeks after the end of each quarter from Disease Control Section, Department of Human Services.

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DISCLAIMER

All data in this report are provisional and subject to revision as further information becomes available.

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Contributions are invited on any topic dealing with the control of infectious diseases. These may be in the form of articles, short reports or letters. Submissions should be in Microsoft Word IBM-compatible format with Vancouver-style references. We encourage submissions in electronic format. Original data from which graphs and figures have been prepared should be included. Submissions will be edited to conform with the style of the bulletin.

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