Rubella infection in pregnancy

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Abstract

It is over 50 years since a syndrome of congenital abnormalities following maternal rubella infection was first recognised. Despite the potentially devastating effects of the congenital rubella syndrome, immunisation rates are not optimal and infections in pregnancy still occur. Four cases of rubella infection occurring in pregnancy are presented. Laboratory diagnosis of primary infection and reinfection is discussed, and the need for full immunisation in childhood, and of women of child-bearing age is reiterated. *Commun Dis Intell* 1999;23:93-96.

Introduction

Rubella remains a common community infection and continues to be a risk to pregnant women who have either not been immunised or who have waning immunity. Four recent cases of rubella infection occurring in pregnancy highlight the potential risk to the developing foetus of both primary infection and reinfection. For each case, the gestation period stated is the time since the last menstrual period.

Case Studies

Case 1

A 30 year old primigravid woman had routine antenatal investigations at 9 weeks gestation (07/01/97), at which time her rubella IgG was <10 IU/mL by ELISA and rubella IgM was negative. During the 11th and 12th week of pregnancy she had contact with a male co-worker who was diagnosed as having rubella. At that time (24/01/97), repeat testing revealed a rubella IgG of 12 IU/mL. The rubella IgM level remained negative. Subsequent testing two weeks later revealed an IgG >130 IU/mL and positive IgM antibodies (confirmed by IgM sucrose density ultracentrifugation, the reference method). One day later she developed fever, a rash lasting two days and arthralgia. She had previously received rubella vaccination when at school. Repeat testing in parallel of all three samples demonstrated levels for the first two samples that fluctuated between 8 and 17 IU/mL. Her antibody levels prior to exposure were low and non-protective rather than absent. In primary rubella infection, antibodies appear as the rash fades.[1] The detection of IgG in high titre one day prior to the onset of rash is evidence of a rapid

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antibody response consistent with rubella reinfection rather than primary infection.[3] A high IgG avidity index also suggested reinfection. The rash however, was a clinical sign that viraemia had occurred. The patient elected to terminate the pregnancy. Testing of the products of conception did not demonstrate rubella infection, either by standard viral culture or by polymerase chain reaction.[2]

Case 2

A 31 year old woman developed a rash at 17 weeks gestation (5/11/96). Although reinfection was suspected because of a history of vaccination, initial antenatal serology (14/10/96) demonstrated a titre of <10 IU/mL, indicating no evidence of previous vaccination or infection. Serum collected at the time of the rash showed detectable IgM and IgG antibodies, the latter in high titre. Repeat testing of the first serum sample revealed a detectable titre of 18 IU/mL. A third serum sample, collected two weeks prior to the onset of her illness, was retrieved from another laboratory. Testing demonstrated similarly low level IgG titres. Serological testing, including avidity studies was consistent with rubella reinfection. The patient was advised that rubella reinfection at 17 weeks gestation posed little, if any, risk to the foetus. A normal term infant was delivered by vacuum extraction. No foetal abnormalities were evident at birth or on review at six weeks of age.

Case 3

At 22 weeks gestation, a 24 year old aboriginal woman presented unwell with fever and rash (20/11/96) to her general practitioner. Vaccination history was unknown. Rubella specific IgG and IgM was detected. Stored serum from unrelated investigations was retrieved and failed to demonstrate rubella antibodies on the 20/08/96. Documentation of IgG seroconversion confirmed a diagnosis of primary rubella infection. The pregnancy continued to term and a normal foetus was delivered spontaneously. Fortunately, infection at this stage of gestation poses very little risk to the foetus. Although no laboratory investigations or audiometry assessment were performed on the baby, early development has proceeded normally.

Case 4

A 23 year old primigravid woman without a history of rubella vaccination developed a typical rubella illness at 10 weeks gestation. Her mother, a health care worker, did not believe in the benefits of immunisation. A childhood illness characterised by rash was considered by the mother to have been rubella. Serum collected at the time of onset of the rash (13/01/97) contained no demonstrable IgG or IgM rubella antibodies. One week later she seroconverted, with development of elevated IgG and IgM antibody levels. Sucrose density ultracentrifugation confirmed a true IgM elevation. The patient elected to continue with her pregnancy, despite the likelihood of primary infection having occurred at 10 weeks gestation. Subsequently, a male infant was delivered at term. Although there was no evidence of embryopathy clinically at birth, Auditory Brainstem Reaction testing showed responses at 70db but not below and a skeletal survey showed celery stick appearance of the distal femora and proximal tibiae consistent with congenital rubella syndrome. Throat, eye and urine cultures grew rubella virus and the peripheral blood rubella IgG and IgM were positive. On follow up soon after birth, repeat audiology showed minimal hearing loss only.

Methods

In all cases, IgG and IgM antibody assays were performed by a plate ELISA method. IgM detection was by the indirect method. Quantitative results are expressed in international units (IU) with calibration being performed against reference standards of 10, 27, 42, 80 and 130 IU/mL. IgM confirmation was performed by Queensland Health Scientific Services using sucrose density ultracentrifugation, followed by an indirect ELISA assay and expressed as a qualitative result. In all four cases, sources of potential cross-reacting antibodies, such as infection with CMV, EBV, Toxoplasma and Parvovirus were excluded.

Avidity testing was performed for all patients at a later date and was not available at the time of clinical decision making. The IgG ELISA assay had 6.0 M urea added to it. Dissociation of weakly formed antigen–antibody complexes after challenge with a mild protein denaturant (for example, urea) is characteristic of a primary infection whereas rubella reinfection is characterised by highly avid antigen-antibody complexes.[3, 4]

Serological results for rubella antibody testing, including avidity studies are shown in Table 1. The avidity studies confirm the earlier serological diagnoses of rubella reinfection (cases 1 and 2) and primary infection (cases 3 and 4).

Discussion

Two cases of rubella reinfection and 2 cases of rubella primary infection occurring in pregnancy are presented. Distinguishing between the two types of rubella infection can be difficult but is of considerable clinical importance. The risk of foetal abnormality is far greater following primary infection than reinfection, though a number of reports in recent years have demonstrated that reinfection carries a small but definite risk of long term sequelae.

The estimated risks of foetal damage following primary infection is highest when infection occurs in the first 8 weeks after the last menstrual period, when 90 – 100% of foetuses will become infected and up to 100% of the infected foetuses will develop major clinical defects.[5] Such defects typically include those affecting the heart, vision and auditory function. The risk of both foetal infection and the incidence and severity of congenital defects progressively declines after the first trimester and the risk of any defects after 17 weeks gestation is rare, though may account for some cases of deafness observed after rubella infection in pregnancy.[5] It is important to note that some features of congenital rubella syndrome, such as deafness, may not be detected at birth, and so careful follow up is required.

The risk of foetal infection following maternal reinfection has been variably estimated as 0[6, 7] to 30%,[3] though it is generally accepted that less than 5% of foetuses will become infected when maternal reinfection occurs within the first trimester of pregnancy[3, 5] and that a proportion less than this will develop congenital defects. No cases of rubella reinfection infecting the foetus have been reported after 12 weeks gestation.[8] Most reinfections are

Table 1. Patient results: Rubella serology, avidity testing and characterisation of rubella primary infection from reinfection

	Serology measurements (IU/mL, positive or negative)			
Test	Case 1	Case 2	Case 3	Case 4
Initial antenatal serology	IgG <10	IgG <10	IgG <10	IgG <10
Repeat initial serology (tested in parallel)	IgG 17 IgM neg	IgG 18I gM neg	IgG<1 0IgM neg	IgG<10
Testing at time of rash (tested in parallel)	IgG>130IgM pos IgM UC pos	IgG 130 IgM pos IgM UC equiv	IgG >25 IgM pos IgM UC pos	IgG >25 IgM pos IgM UC pos
Follow-up testing	Not done	IgG >130 IgM pos IgM UC pos	Not done	Not done
Avidity studies	High	High	Low	Low
Diagnosis	Reinfection	Reinfection	Primary Infection	Primary Infection

UC = sucrose density ultracentifugation; to separate IgM from IgG in a serum sample

asymptomatic.[8] Maternal rash is a clinical sign of viraemia but is seldom noted in cases of rubella reinfection, though some women report a non-specific illness.[8, 9] When rash does occur with rubella reinfection, as occurred in the first two cases presented, the risk of foetal damage may more closely match that for primary infection at equivalent gestation, though this has never been clearly documented.

The incidence of congenital rubella infection is monitored by the Australian Paediatric Surveillance Unit (APSU). From May 1993 to December 1997, there have been 24 cases of congenital rubella infection reported to the APSU, of which 5 were born without defects.[9] The estimated incidence in Australia of congenital rubella infection with defects is 1.5/100 000 live births. Seven cases born in 1996 were reported.[10] Two cases had a history of maternal vaccination and represent possible rubella reinfection (or vaccine failure). Both infants had congenital defects; one infant died.

When a pregnant patient has contact with a known or suspected rubella case, or has a non-specific viral-like illness with or without rash, clinicians are advised to perform serial rubella antibody tests, regardless of vaccination status. Congenital rubella syndrome has been documented to occur in Australia despite documented pre-pregnancy levels considered to afford good immunity.[11-13]

While some authorities, notably in the United Kingdom, require proven evidence of successful seroconversion following either vaccination or wild type infection to establish a diagnosis of reinfection, this documentation is commonly lacking in everyday practice. Most reinfections occur in subjects previously vaccinated. Evidence of vaccine efficacy is not usually sought until a woman presents with her first pregnancy. The distinction between primary and secondary infection is ultimately in the hands of the serology laboratory. A single IgG antibody measurement of less than 10 IU/mL would be reported as showing no evidence of prior rubella vaccination or infection by most laboratories, including our own (and hence susceptible to primary infection). A value of 10-15 IU/mL would be reported by our laboratory as indicating that antibodies are detectable but at a level not necessarily providing protection from (re)infection. Repeat testing of the same sample may give results variably suggesting that the patient is, or is not, at risk for primary infection yet still be within the range of two standard deviations (SD) of the cut-off of 10 IU/mL. Calculation of distribution parameters for the reference standard of 10 IU/mL revealed a range within 2SD of 7.2-12.8 IU/mL for the ELISA assay. It is important that testing laboratories investigate possible cases of rubella infection in pregnancy by careful, reproducible parallel testing. Laboratories should be aware of the coefficients of variation for their assay.

Serum samples that predate or occur within 7 - 10 days of a presumed rubella exposure can be extremely valuable in determining pre-exposure immune status to enable one to establish whether a significant rise in IgG antibody level subsequently occurs. It may be necessary to pursue a history of unrelated serological testing or previous rubella antibody measurement in order to discover a source of stored serum (as was done for cases 2 and 3). While IgM was detected in our two cases of reinfection, this does not invariably occur.[13] A significant rise in IgG level is required to diagnose rubella reinfection serologically. Unlike primary infection, reinfection is characterised by high avidity antibody binding. Avidity testing was performed by our laboratory but at a later date. It requires careful technique but is a useful adjunct to antibody detection. However, unless the testing laboratory is regularly performing avidity testing, turn around time may not be rapid enough for a clinician and patient contemplating termination of pregnancy.

The schoolgirl rubella vaccination programme commenced in 1970-71. In 1988-89 combined measles, mumps and rubella (MMR) vaccination was recommended for all infants aged 12 months. Australian states and territories introduced vaccination of all teenage boys and girls in the period 1994-96, replacing the schoolgirl vaccination programme. More recently (1998), the age for the second MMR vaccine has been lowered to age 4 - 5 years,

principally to improve immunity against measles in children. While eradication of measles and rubella is now a real possibility, there remains a large pool of rubella susceptible males, typically aged between 10 - 25 years, in the community today. Unfortunately childhood vaccination in this country has reached worryingly low levels. When surveyed in April 1995, only 35% of children aged two years were fully vaccinated, although the rubella vaccination rate was higher (81%).[14] As a greater proportion of the community acquires antibodies through vaccination rather than naturally occurring disease, primary disease will become less common. Infections encountered are more likely to be reinfections, generally seen in those with low post-vaccination antibody titres.

In the past, women were at the greatest risk of exposure through contact with their own children. Now susceptible women are at most risk of becoming infected by contact with infected fellow students or male co-workers. Migrant women may be more likely not to have been vaccinated prior to becoming pregnant.[15]

As it is clear that immunity following vaccination, especially a single dose in adolescence, may decline over time, the importance of checking antibody titres with each and every pregnancy must be stressed. A pregnant woman with no or low immunity needs to be vaccinated immediately after delivery and antibody status checked after 3 months. It is important that vaccination not be given in the three months following administration of immunoglobulin (with the exception of anti-D Rh immunoglobulin) or whole blood transfusion, as there may be some interference with antibody response to the vaccine. Ideally, antibody status could be checked prior to a planned pregnancy so that vaccination could be given, if indicated, prior to conception. This may be especially applicable where first pregnancies are occurring many years after vaccination. It is recommended that women wait 2 months following vaccination with live attenuated rubella virus before conceiving.[16] Where vaccination has inadvertently occurred during pregnancy, no documented cases of foetal abnormality have been recorded.[16] Whenever a pregnant woman has had contact with an illness that might be rubella, clinicians should be encouraged to check immune status and look for evidence of acquired infection. This requires appropriately timed serological investigation; at least 28 days (maximum incubation period plus 7 days) after a rubella contact should be allowed to reliably detect an antibody response. Clinical illness cannot be relied upon to detect most cases of reinfection.

Congenital rubella syndrome remains a preventable disease provided that the current childhood immunisation schedule is successfully implemented and that protective immunity is maintained in women of child-bearing age.

When infection does occur in pregnancy, careful serological investigation can help distinguish between primary infection and reinfection, in order that patients can be best informed of the potential risks to the foetus.

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Meningococcal disease and the law: does non-notification really happen?

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Abstract

In Victoria, legislation clearly makes the notification of clinical or confirmed cases of meningococcal disease mandatory. Statistical modelling suggests that meningococcal disease is significantly under-notified, and that incorrect codes might be being ascribed to some in-patient episodes. The aims of this study were (i) to test the assumption that cases identified as non-notified cases were true cases, and (ii) to identify the reasons for non-detection on the hospital separation database and non-notification to the infectious diseases unit. Of 26 cases not identified on the in-patient dataset, the main causes were either being given completely incorrect ICD-9-CM codes (11 cases) or being given codes for a different type of meningitis (8 cases). Of 29 non-notified admissions, most were clinically (17) or microbiologically (6) confirmed cases, although 5 were coded in error and were not cases of meningococcal disease. Therefore, although the allocation of incorrect ICD-9-CM codes at separation was a major reason for discrepancy, non-notification was a real and recent problem. It is also possible that some clinical staff did not understand the relationship between *Neisseria meningitidis* and meningococcal disease, the public health implications of this infection, or the law relating to it. *Commun Dis Intell* 1999;23:97-101.

Introduction

Following European settlement, fear of imported diseases crossing state boundaries resulted in the introduction of state public health acts, [1,2] which were mainly adapted British public health law, to assist with quarantine and control of population movement. Eventually the 1908 Commonwealth Quarantine Act [3] brought about the protection of national boundaries from imported diseases. The Quarantine Act states that the Governor General (GG) may provide, or arrange for the provision of:

'..... teaching, research and advisory service for or in relation to the improvement of health or the prevention of disease ....'

Clause 35A(2) provided the GG with the ability to nominate any disease as subject to quarantine if it was judged necessary. Today, in each of the States and Territories, this responsibility is delegated to the Chief General Manager (CGM).

The States developed their own internal mechanisms for the notification and control of communicable and infectious diseases. In Victoria, the relevant legislation relating to infectious and communicable disease is contained in the Victorian Health Act 1958 [4] and amendments (Part VI Division 3 Clauses 121 and 126, and Division 9 Clause 138; Part VIII Clause 146 and Division 4 Clauses 421 and 142). Clause 9 of the Victorian Health Act, which relates to disease notification, states that:

'The CGM may make regulations for or with respect to - (a) prescribing diseases ..... the occurrence or existence of which must be notified to the CGM';

The Regulations referred to are the Health (Infectious Diseases) Regulations 1990,[5] Schedule 2, which includes meningococcal infections on the list of Group A diseases. Group A diseases …

'.... should be notified to the Health Department Victoria by telephone or fax upon initial diagnosis (presumptive or confirmed) with written confirmation to follow within seven days.'

In addition, in 1996 the National Health and Medical Research Council produced Australian guidelines for meningococcal disease control which further set out the process of notification of the disease in this country. [6]

If all doctors understood and abided by the law, all cases of meningococcal disease (whether clinical or microbiologically confirmed), would be notified to the Infectious Diseases Unit promptly, and there would be no non-notified cases. Non-notification of communicable diseases, including meningococcal disease, has been noted as a problem in some communities internationally.[7,8,9]

In 1996 a study was conducted in Victoria to determine the extent of under-notification of meningococcal disease.[11] The study compared three datasets, which should have comprehensively and independently recorded cases of meningococcal disease:

- the then Department of Human Services Infectious Diseases Unit's Infectious Diseases Epidemiology Surveillance System (IDESS);
- the Melbourne University Microbiological Diagnostic Unit's (MDU) Victorian Hospitals Pathogen Surveillance System (VHPSS); and
- the Department of Human Services Epidemiology Unit's Victorian Inpatient Minimum Dataset (VIMD), where one of the first three listed International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes indicated that the admission was for meningococcal infection (036.0-036.9).

Initial matching of cases was undertaken using the hospital unit record (UR) - number, gender, date of birth and age, postcode of residence, and admitting hospital. In addition

name codes (derived from a combination of first and second name initial letters) were available from the IDESS and VIMD datasets. Using log-linear modelling, for the years 1988 -1994, a significant under-notification was demonstrated. Whilst 576 cases were identified overall, only 251 cases (43.6%) were identified in all three datasets, and initial modelling suggested an under-notification of 90 cases (95%, CI 58.2,139.3).[11] However, when the model was applied to 1995 and 1996 data, it became very unstable, resulting in an inability to produce a clear and unambiguous estimate of the total number of cases. This effect probably occurred because a productive collaboration between the Infectious Diseases Unit and Microbiological Diagnostic Unit had been established, and IDESS and VHPSS were no longer independent of each other. However, the application of techniques such as capture-recapture to these data suggested that there was still significant under-notification. Detection was estimated as only 94.9% of all probable cases (95%, limits 92.7% - 97.2%).

Although several studies have been designed to estimate the total number of cases of meningococcal disease by using modelling techniques such as capture-recapture methods, in only one has an attempt been made to validate their datasets.[10] However, the authors of this American study included only cases which were confirmed by positive microbiology or microscopy, ignoring clinical cases. In Victoria in 1996, 26% of notified cases of meningococcal disease had no positive laboratory confirmed results, and were therefore considered to be clinical cases. It is important that clinical cases be included in case counts. Overall, in Australia 12% of cases of meningococcal disease included in the National Notifiable Diseases Surveillance System were unconfirmed. [6]

The current study was designed to (i) test the assumption that cases identified as non-notified cases were true cases, (ii) identify reasons why these cases were not notified despite clear legislation, and (iii) identify the reasons why some notified cases escaped detection on the hospital separation database. As the issue of notification to VHPSS is now a historical problem, VHPSS-only cases were not included in this study.

Methods

To be identified on the IDESS, a person must be notified to the Infectious Diseases Unit as having a provisional or confirmed diagnosis of meningococcal disease. To be identified in the VIMD, a person must have an ICD-9-CM relating to meningococcal infection (codes 036.0-036.9). People who have meningococcal disease should appear on both of these data sets.

In this study, a case was a person with meningococcal disease who was either

- notified to IDESS but not identified on the VIMD; or
- identified on the VIMD but not notified to the IDESS.

A data collection form was developed which captured key information relating to the remaining discrepant admissions, including clinical signs and symptoms, diagnostic tests, results, and discharge ICD-9-CM codes.

Ethical approval for this study was given by the Human Ethics Committee of the Department of Human Services, and in addition was noted by the Human Ethics Committees of the participating hospitals.

Results

For the years 1990 -1995, of 483 notifications and admissions for meningococcal disease, there were 121 which were known only to either IDESS or VIMD.

Of these, 4 were not hospitalised in Victoria, and were therefore not eligible for inclusion on the VIMD, accounting for this discrepancy. Access to their records was not sought. A further 24 cases were excluded from the study as they were either admitted to a private hospital (where record access is difficult), or could not be identified at the admitting hospital, or the admitting hospital could not be identified. Therefore 93 discrepant cases remained for inclusion in this study (Table 1).

On retrieving the hospital records, 19 pairs were matched with complete information (including complete name, and listed ICD-9-CM codes 36.0-9), making 55 unmatched cases and 19 matched cases. Therefore the identified sample of 93 cases was reduced to 74 records, of which 19 were no longer discrepant.

The remaining 55 discrepant admissions are discussed below, and summarised in Table 2.

Twenty-six cases were known to IDESS but not identified on VIMD for one of the following reasons:

- Eight people had codes that attributed their disease to other types of bacterial or viral infections. Some of

Table 1. Summary of records of menigococcal disease in this study sample

Total identified discrepant notifications and admissions	121
• Ineligible for inclusion to VIMD (never admitted or hospitalised interstate), therefore reason for discrepancy already clear	4
• Admitted to private hospital (logistic and legal problems relate to the retrieval of information relating to private patients admitted to private hospitals in Victoria)	6
• Records unidentifiable at admitting hospital	15
• No record of admitting hospital	3
Total number of records not requested for reasons of access or identification	28
• Known to IDESS, not identified on VIMD	45
• Identified on VIMD, not notified to IDESS	48
Total number of records for which access was requested	93

Table 2. Reasons for dataset discrepancies, meningococcal disease, Victoria, 1990-1995

	(b) Known only to IDESS	(a) Known only to VIMD	TOTAL
Matched with complete details	19	19	19/2
ICD-9-CM code differs from ascribed code; data entry error	11	5	16
ICD-9-CM code for different type of meningitis	8		8
Meningococcal disease not listed on problem ICD-9-CM coding summary	2		2
Microbiologically shown not to be a case	2		2
ICD-9-CM codes not listed	2		2
Clinical case with meningococcal disease ICD-9-CM code		17	17
Culture +ve case, not notified (including 1 recrudescence)		6	6
Clinical record states case notified, but not notified to IDESS		1	1

these were to the wrong organism (pneumococcus, for example) and others to an unspecified organism. Two of these had (incorrect) codes indicative of systemic gonococcal infection.

- Two had chronic conditions that were listed in detail, and meningococcal infection codes were ignored.
- Eleven had codes unrelated to meningococcal disease or any other type of meningitis. Most ascribed codes related to the main presenting symptoms; convulsions or diarrhoea, for example. Several had codes incorrectly transcribed, such as '36.0' instead of '036.0'.
- Two cases had no ICD-9-CM codes listed on the separation sheet.
- One was ascribed a code completely unrelated to any signs, symptoms, or final diagnosis.
- Two people were eventually shown to not have meningococcal disease (for example, one child had echovirus type 30 on CSF culture). Although these two were no longer cases they were not 'un-notified'.

Twenty-nine cases were known to VIMD but not identified on IDESS for one of the following reasons:

- An incorrect ICD-9-CM code was assigned or entered for 5 people who did not have meningococcal disease, and who should have had codes of '036' instead of '36' (heart vessel procedure) or '8361' (knee reconstruction).
- One culture positive case was recorded in the patient record as having been notified, but the Department had no record of the communication.
- Seventeen people had clinical meningococcal disease, and technically should have had a code related to bacterial meningitis of unknown origin.
- Six cases were microbiologically confirmed (either culture positive or gram-negative diplococci identified microscopically) but were not notified. In examining these case records, it was not clear whether the consultant staff were unaware of the regulations concerning this infection, or whether there was no understanding of the connection between *'Neisseria meningitidis'* and 'meningococcal disease', for example:

' ... grew Neisseria meningitidis from CSF and blood; however antigen negative therefore ? cause of (this person)'s bacterial septicaemia....'

The problem of non-notification is not simply historical (Table 3). For instance, in the most recent study year, 1995, 5 cases were not notified. None had a lumbar puncture performed, although all had blood cultures collected, of which one was culture-positive. Two of these presented with unusual and interesting clinical histories. Three had a characteristic rash, 3 had a severe headache and 1 had neurological signs. All recovered with the administration of penicillin and ceftriaxone, however no mention was made in any of the case notes suggesting that any close contacts had received prophylaxis. Two cases should have had particularly careful public health management; one was a recurrent case, and the other a secondary, or possibly co-primary case.

Table 3. Number of discrepant cases of meningococcal disease, 1990-1995, by year and dataset record

	Notified to HACS, not identified on VIMD	Identified on VIMD, not notified to IDESS	TOTAL
1990	2	2	4
1991		1	2
1992	7	13	29
1993	7	4	17
1994	5	3	9
1995	5	6	13
TOTAL	26	29	74

Several comments on the patient records demonstrated a reluctance on the part of staff to divulge any information about their patients to the Infectious Diseases Unit, which needed it for outbreak control, for example:

' Dr ..... phoned from the (Health Department. He) wanted to know ..... however I told him that only the patient could give permission for this information to be released .....'

Discussion

An assumption in the use of log-linear modelling and capture-recapture techniques for the estimation of total populations and events, is that all cases occurring in more than one dataset are matched. Despite carefully matching criteria, 19 people who could not be matched in the original project,[11] were matched in this study with more complete information.

Four cases were not admitted to hospital in Victoria, making it impossible for inclusion on the VIMD. It is possible that other Victorian residents may have been admitted to hospital interstate, or were not admitted to hospital at all, who were also not notified to IDESS.

Inaccurate public hospital discharge data have previously been noted to be a problem in terms of both epidemiological accuracy[12] and financial renumeration.[13] Incorrectly being assigned an ICD-9-CM code for meningococcal disease accounted for 5 of 29 VIMD 'cases'. It is interesting that on the VIMD, 17 non-notified clinical cases were identified only because they were given a technically incorrect code; the code for bacterial meningitis or septicaemia caused by unknown organism (ICD-9-CM codes 320.9 or 038.9) would have been more accurate. It is likely that other non-notified clinical cases of meningococcal disease have occurred, who were given the correct ICD-9-CM code (such as 320.9 or 038.9), and who were not identifiable by the methods used in this study.

The one study, conducted in New York, designed to validate the completeness of notification of meningococcal disease by examining the records of notified and admitted cases, did not include clinical cases**.**[10] Although the conclusion of these authors was that their combined datasets identified 93% of all cases; the inclusion criteria for both databases was identical, 'positive culture or microscopy', therefore they were not really independent of each other. In Victoria, many notified cases of meningococcal disease are not able to be confirmed by existing laboratory techniques (between 25% and 50% of cases since 1990 have not had an isolate submitted to the State Meningococcal Reference Laboratory.)[14]

The problem of laboratory-positive cases not being notified to the Infectious Diseases Unit accounted for 6 of 29 'un-notified' admissions. It is of concern that disease caused by *Neisseria meningitidis* is *ever* not recognised as being meningococcal disease, and therefore not notified. Ward staff should be aware of the importance of prompt notification of both suspected and confirmed cases of all manifestations of meningococcal disease.

It is commendable that ward staff are generally unwilling to divulge information about their patients. In this study the apparent unwillingness of ward staff to divulge information to public health staff has been noted. Some staff appear not to understand the contact tracing process involved in communicable diseases and are diffident about exposing the close contacts of cases to scrutiny. All cases, whether private patients or not, should be notified by law. The law covers issues of confidentiality and identification equally for all patients, whether being treated in private or public hospitals. The Commonwealth Privacy Act 1988[15] contains legislation relevant to health personnel involved in outbreak investigations. This is a situation that is common in the follow-up of contacts of cases of meningococcal disease, and precludes further disclosure of personal details of cases or their contacts, except in very unusual circumstances:

' .... shall not disclose ..... unless (the) record-keeper believes (disclosure) will lessen a serious and imminent threat to the life and health of the individual concerned or another person.'

Mechanisms should be explored for ensuring that private patients are afforded the same public health follow-up as their public patient counterparts, so that the former are not disadvantaged by their private patient status.

It should be noted that of the 24 clinical and confirmed but non-notified cases, 2 were secondary cases. Without consistent notification it is impossible to ascertain some important epidemiological features including; accurate counts of co-primary or secondary cases, rates of clinical versus confirmed cases, and efficacy of chemoprophylaxis and vaccination programmes. [7, 10] Some clinical staff undertake the identification of contacts and prescription of appropriate antibiotics without notifying the Health Department. In the event of an admission for meningococcal disease it is common for many people to claim to be close contacts. Whilst it is important to institute prophylactic treatment promptly, for clinical reasons it is also important to ensure that *only* close contacts are treated. In Victoria a legal clause exists in the Victorian Health Act 1958 (Division 4, Clause 421) which could be used to enforce this point:

'Every person who - (a) knowingly makes any false or misleading statement in any application, notice or report'

The last point to emphasise with regard to notification is very clear in the Victorian Health Act 1958, Clause 422:

' ... Every person who does not do anything directed to be done ..... shall be guilty of an offence against this Act.'

Therefore, as meningococcal disease is listed in Schedule 2 as a notifiable disease in Victoria, its notification is obligatory, regardless of whether cases are *suspected or confirmed*. Disclosure of personal details of cases or close contacts by any hospital staff, to anyone other than people closely involved with the family or health protection staff, is technically in breach of the law. The relatives of cases are likely to be upset and unable to give rational informed consent for public dissemination of distressing details. It should be impossible for health protection staff to first hear about a new case through the media or from a worried teacher or neighbour, rather than directly from a colleague.

Complete notification enables effective public health management of single cases, early identification of outbreaks and secondary cases, the distribution of appropriate information and advice for communities, and rational information for media distribution. It makes the impact of preventive programmes measurable. Without complete notification the incidence of this frightening disease will be underestimated, and consequently the costs of public health strategies and preventive programmes overestimated.

This study has shown that, despite laws which stipulate that suspected and confirmed cases of meningococcal

disease shall be notified, for several reasons, clinicians fail to always do so.

Acknowledgements

Dr Kath Taylor and Dr Michael Ackland, Public Health Branch, Department of Human Services Victoria, and the CEOs and CMROs of all participating hospitals for their help and support.

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An outbreak of *Salmonella* Typhimurium RDNC A045 at a wedding feast in South Australia

Peter Brennan,[1,2] Rosalind Holland,[1] Robert Hall[1] and Scott Cameron[1]

Abstract

In April 1998 an outbreak of salmonellosis amongst guests at a wedding feast was investigated. Of the 58 attendees interviewed 38 (66%) subsequently developed gastrointestinal symptoms. Stool cultures from 7 cases grew *Salmonella* Typhimurium RDNC A045. Food samples were culture-negative for *Salmonella* spp. A cohort study implicated spatchcock (RR 2.5, 95% CI 1.09-5.77) and scampi (RR 2.0, 95% CI 1.05-3.89). Temperature abuse and cross-contamination within the kitchen during preparation and cooking are likely to have been the main contributing factors to this outbreak. Control measures included staff education in safe food handling and improvements in poultry processing methods to minimise carcass contamination. *Commun Dis Intell* 1999;23:101-103

Introduction

In South Australia between 300 and 600 notifications of salmonellosis are received annually. Of these the most common serovar is *Salmonella* Typhimurium (62% in 1997) with a predominance of phage types 9, 64 and 135. *Salmonella* Typhimurium designated as 'Reacts Does Not Conform' (RDNC) occur much less frequently with about 12 cases per year (South Australian Department of Human Services, unpublished data).

On 23 April 1998 the Communicable Disease Control Branch was notified of two laboratory proven cases of salmonellosis. They were from a group of 61 people who had attended a wedding. Enquiries revealed that at least 6 (10%) had a gastrointestinal illness. The only common feature amongst the 61 people was attendance at the wedding. The caterer reported that all foods were prepared and served on site.

An investigation was conducted to determine the extent and source of the outbreak.

Methods

Epidemiological investigation

A questionnaire was developed based on information from a menu and list of staff and guests. A cohort study was conducted to determine whether any food or drink consumed at the wedding was associated with illness. A case was defined as any of the attendees, including staff,

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who suffered a gastrointestinal illness between the wedding on 18 April 1998 and the time of interview. The questionnaire was conducted by telephone between 24 and 27 April. The interviewers inquired about symptoms of illness, onset time and foods and drinks consumed from the menu items. Food risks and risk ratios for illness were calculated using Epi Info 6.

Environmental investigation

Methods of food preparation were determined from the caterer, the cook and the serving staff by telephone interview. The kitchen facilities, food storage and preparation areas were inspected by the local government environmental health officer (EHO). There was no leftover food available for culture, but a sample of scampi, which was from another batch, was sampled for culture. Faecal specimens from 7 cases were sent for culture and typing.

The processing plant that supplied spatchcocks for the wedding was inspected and fresh and frozen samples were taken for culture (spatchcocks are 4 week old broiler chickens, which are processed at specialty plants).

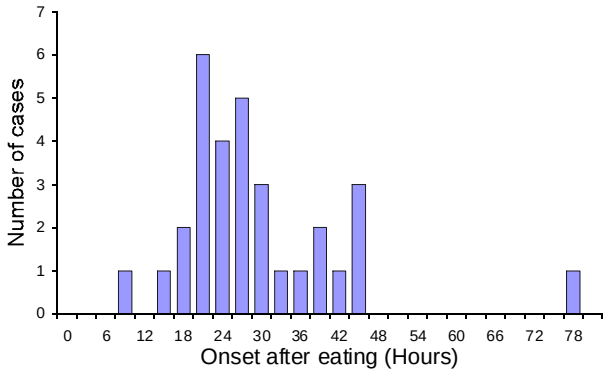
Results

Epidemiological investigation

Of the 61 guests, 58 (95%) were interviewed. The guests came from various states (SA - 48, NSW - 5, Vic - 2, WA - 2 and Qld - 1). Twenty people reported no illness after the wedding. Symptoms consistent with the case definition were reported by 38 (66%). The only common feature identified in cases was attendance at the wedding. Among the cases the male to female ratio was 1.2: 1 and the age range was 10 to 68 (median = 38). The majority, 36 (95%), had onset of illness on the 19 or 20 April 1998 with a median incubation period of 26 hours (see Figure 1). The symptoms described by the 38 cases were: diarrhoea (100%), abdominal pain (92%), fever (92%), nausea (73%), vomiting (35%) and bloody diarrhoea (3%). Stool specimens from seven cases grew *Salmonella* Typhimurium on standard enteric media and were subtyped using the Colindale method as RDNC A045.

The foods with the highest risk ratio for illness were spatchcock (RR = 2.51, 95% CI 1.09-5.77) and scampi (RR = 2.02, 95% CI 1.05- 3.89) (Table 1). Lower (although statistically significant) risk also occurred for the terrine (RR = 1.76, 95% CI: 1.13-2.75) and fetta filo parcels (RR = 1.53, CI 1.08-2.16). No statistically significant risk was found with the other foods or beverages.

Figure 1. Number of cases of gastrointestinal illness after the wedding feast, by onset time



Environmental investigation

Food preparation and handling

A large proportion of the food was prepared on the day of the wedding or the previous day. Foods were pre-cooked and reheated on the day, or cooked just prior to serving. The foods were prepared and stored overnight in the refrigerator or iced in polystyrene containers. The methods of preparation for all foods were reviewed.

The spatchcocks were purchased and delivered frozen. On Friday 17 April they were thawed in cold water for 3 hours and then stuffed with a mixture of herbs and spices, cooked rice, pine nuts and sultanas. They were then baked for one hour then transferred to iced polystyrene containers. On the day of the wedding the spatchcock were split in half and then reheated. They were served on

Table 1. Foods consumed at the wedding feast by reported illness

Food	Illnesss:Food Specific Risk[1]	RR	95% CI
Bacon	19/25	1.32	0.91-1.90
Filo parcels	17/20	1.53	1.08-2.16
Kangaroo	19/23	1.48	1.04-2.10
Chicken wings	19/28	1.07	0.74-1.56
Devilled eggs	14/17	1.43	1.01-2.02
Olives	22/28	1.52	1.02-2.27
Dolmades	22/28	1.52	1.02-2.27
Salami	11/15	1.20	0.81-1.78
Crackers with pesto	25/34	1.30	0.86-1.96
Vegetable terrine	26/32	1.76	1.13-2.75
Trout	32/47	1.25	0.70-2.22
Scampi	31/41	2.02	1.05-3.89
Spatchcock	34/44	2.51	1.09-5.77
Lamb shanks	19/26	1.23	0.85-1.78
Snapper	26/36	1.31	0.84-2.05
Salad	26/36	1.32	0.86-2.04
Bread roll	12/20	0.89	0.58-1.36
Bread roll/butter	18/22	1.46	1.02-2.09
Red wine	34/50	1.36	0.66-2.79
White wine	12/20	0.90	0.58-1.38
Champagne	21/35	0.82	0.57-1.20

1. Food Specific Risk in those ill

CI = Confidence Interval

RR = Risk ratio

a clean earthenware platter with a bed of triple washed mixed lettuces.

The scampi were purchased frozen and delivered on Friday and left at room temperature for approximately 2-3 hours before being placed in the refrigerator overnight. A lime mayonnaise, to be served with the scampi, was prepared on the Friday using raw eggs, garlic, olive oil and spices and stored overnight in the refrigerator. On removal from the fridge on the evening of the wedding the scampi were still frozen and were thawed at room temperature for approximately 4 hours. They were then cooked for about 2 minutes per side on a BBQ hot plate and served with the lime mayonnaise. A Caesar salad dressing was also prepared using raw eggs.

Function site investigation

The cook and the serving staff had consumed some of the food at the function and a number of them subsequently developed gastroenteritis. None were ill before the wedding.

The food preparation area and refrigerator space in the kitchen was limited. On inspection the temperature of the fridge was found to be adequate for food storage. Advice on food preparation and storage was given on each of three visits by the local EHO.

No *Salmonella* spp. were identified in the scampi.

Spatchcock processing investigation

The inspection revealed faecal contamination of carcasses in the initial processing stages. Advice on how to avoid this was provided. Six specimens were taken from 3 different batches of spatchcocks. The actual batch of spatchcock that was served at the wedding was not known. Of the six specimens two grew *Salmonella* Senftenburg and one of these further grew *Salmonella* Typhimurium untypable (C. Murray, Med Vet IMVS Laboratory Adelaide, personal communication).

Discussion

This report describes the investigation of a well-defined *Salmonella* Typhimurium outbreak of a previously unrecognised phage type (RDNC A045) with a high attack rate (66%) suggestive of a high level of contamination.[1] It also illustrates some of the difficulties in identifying a source of contamination and indicates that small-scale catering operations continue to be a source of foodborne disease.

Numerous foods were implicated including; spatchcock, scampi, terrine and fetta filo parcels, suggesting cross contamination. On the basis of biological plausibility, food specific risk (34/44) and risk ratio, spatchcock was further investigated as a source of contamination. Spatchcock was found to be subject to inadequate temperature control and was identified as a high risk food (RR = 2.51).

Spatchcock could be expected to carry *Salmonella* spp. at the same rate (25-35%) as other poultry,[2] and this was confirmed (2/6 positive for *Salmonella* spp.) in this local investigation.

Inadequate thawing, storage and possibly cooking of the foods in the kitchen may have allowed the organisms to multiply and spread to other foods. Limited food preparation and refrigeration spaces are likely to have increased the possibility of cross contamination.

Salmonella food poisoning, related to small catered functions, continues to be a public health concern. Timely investigation of potentially related notified cases can assist with outbreak detection. Rapid typing of *Salmonella* spp. isolates assists in the identification of clusters of infection and in the tracing their source. This study reconfirms the need to educate those involved in food handling at all levels with regard to safe thawing, handling, storage and cooking of foodstuffs.

Limitations of the investigation

Selection bias may have occurred as a result of the inability to contact 3 attendees despite numerous attempts, though this would be unlikely to effect any outcomes. Measurement bias may have occurred as part of the 'loose' definition of the illness, although all 'cases' had had a diarrhoeal illness. Recall bias may be a contributor. A number of attendees had difficulty recalling exactly what they ate, or the amount, due to the nature of the function; a progressive feast served sequentially on trays, resulting in people tending to try many of the 17 or more foods. Unfortunately no samples of food from the wedding feast were available for culture, so microbiological confirmation of contamination was not possible.

Recommendations

Effective catering operations require an adequate clean preparation area with appropriate staffing and equipment. Staff should be trained in safe food handling practices to allow for appropriate preparation, cooking and serving of food. This study emphasises the need for food safety plans, as well as education and review of small-scale catering practice.[3,4]

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Communicable Diseases Surveillance

Highlights

Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. The Australian Sentinel Practice Research Network (ASPREN) is a general practitioner-based sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Vaccine preventable diseases

The number of measles cases is higher in this period, largely because of the outbreak which is mainly in Victoria.

Details of the outbreak are summarised below and on the web page at: 'http://www.health.gov.au/pubhlth/alert.htm'.

Statistics at 15 April 1999 relating to the Victorian outbreak

- the index case was a young adult who had returned from Bali;
- the date of onset for the index case was 11 Februrary 1999;
- 66 cases of measles have been reported to Victorian Health authorities;
- 25 persons have been admitted to hospital;
- all 6 cases aged between 0 and 8 years were unimmunised and
- 5 cases of vaccine failure have been identified (all had received one dose of a measles-only vaccine).

The number of notifications for pertussis infection remains relatively low, with the number of cases having onset in February 1999 being the lowest since July 1996.

Tables

There were 7,238 notifications to the National Notifiable Diseases Surveillance System (NNDSS) in the four week period, 3 to 30 March 1999 (Tables 1 and 2). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 1).

There were 1,542 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the four week period, 25 February to 24 March 1999 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 9 to 12, ending 28 March 1999, are included in this issue of *CDI* (Table 5).

Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 3 to 30 March 1999

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
H. influenzae type b infection	0	1	1	1	0	0	1	0	4	3	13	6
Measles	2	4	0	2	0	1	48	2	59	29	98	100
Mumps	1	2	0	1	0	0	4	3	11	18	28	48
Pertussis	5	70	1	58	7	6	103	4	254	503	961	2,664
Rubella[3]	3	2	1	8	1	0	8	2	25	53	85	179
Tetanus	0	0	0	0	0	0	0	0	0	0	0	2

NN. Not Notifiable

1. No notification of poliomyelitis has been received since 1978.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. Includes congenital rubella.

Table 2. Notifications of diseases received by State and Territory health authorities in the period 3 to 30 March 1999

Disease[1,2,3,4]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998[5]
Arbovirus infection (NEC)	0	0	0	3	0	1	8	0	12	5	28	17
Barmah Forest virus infection	0	24	0	24	0	0	1	1	50	63	160	180
Brucellosis	0	0	0	1	0	0	0	0	1	2	5	14
Campylobacteriosis[6]	44	-	27	280	338	32	306	80	1,107	867	3,439	3,049
Chancroid	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydial infection (NEC)[7]	20	NN	53	392	74	20	208	143	910	826	2,753	2,430
Cholera	0	0	0	0	0	0	0	0	0	1	1	2
Dengue	0	1	0	12	0	0	0	2	15	106	124	185
Donovanosis	0	NN	0	0	NN	0	0	0	0	3	5	17
Gonococcal infection[8]	4	74	88	118	22	2	0	73	381	391	1,213	1,194
Haemolytic uraemic syndrome[9]	NN	1	NN	1	0	0	NN	0	2	1	7	2
Hepatitis A	3	49	6	42	9	1	18	21	149	266	487	842
Hepatitis B incident	3	3	4	2	3	1	0	2	18	19	77	68
Hepatitis B unspecified[10]	3	136	0	65	0	2	157	11	374	614	1,489	1,797
Hepatitis C incident	12	0	0	-	3	0	0	8	23	33	81	64
Hepatitis C unspecified[5,10]	23	505	39	254	77	35	362	69	1,364	1,965	4,875	5,576
Hepatitis (NEC)[11]	0	0	0	0	0	0	0	NN	0	2	1	6
Hydatid infection	0	0	0	0	0	0	2	0	2	1	7	9
Legionellosis	0	2	0	3	4	1	20	4	34	19	93	55
Leprosy	0	0	0	0	0	0	0	0	0	0	0	1
Leptospirosis	0	1	1	24	0	0	4	0	30	12	97	38
Listeriosis	0	2	0	0	0	0	2	0	4	7	14	20
Malaria	3	12	2	31	2	0	3	0	53	40	221	162
Meningococcal infection	0	17	0	6	0	0	5	6	34	11	96	47
Ornithosis	0	NN	0	0	0	0	2	0	2	1	16	6
Q Fever	0	11	0	33	0	0	1	1	46	37	127	125
Ross River virus infection	1	161	8	479	4	11	24	23	711	580	1,870	1,104
Salmonellosis (NEC)	6	127	46	296	482	32	177	58	1,224	800	3,019	2,591
Shigellosis[6]	1	-	15	18	12	0	17	10	73	61	178	180
SLTEC, VTEC[12]	NN	0	NN	NN	3	0	NN	NN	3	1	10	4
Syphilis[13]	1	27	41	72	1	1	0	0	143	116	454	321
TTP[14]	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis	1	50	1	8	0	2	31	1	94	94	337	320
Typhoid[15]	0	3	0	0	2	0	2	1	8	9	21	35
Yersiniosis (NEC)[6]	1	-	0	13	4	0	0	0	18	12	59	84

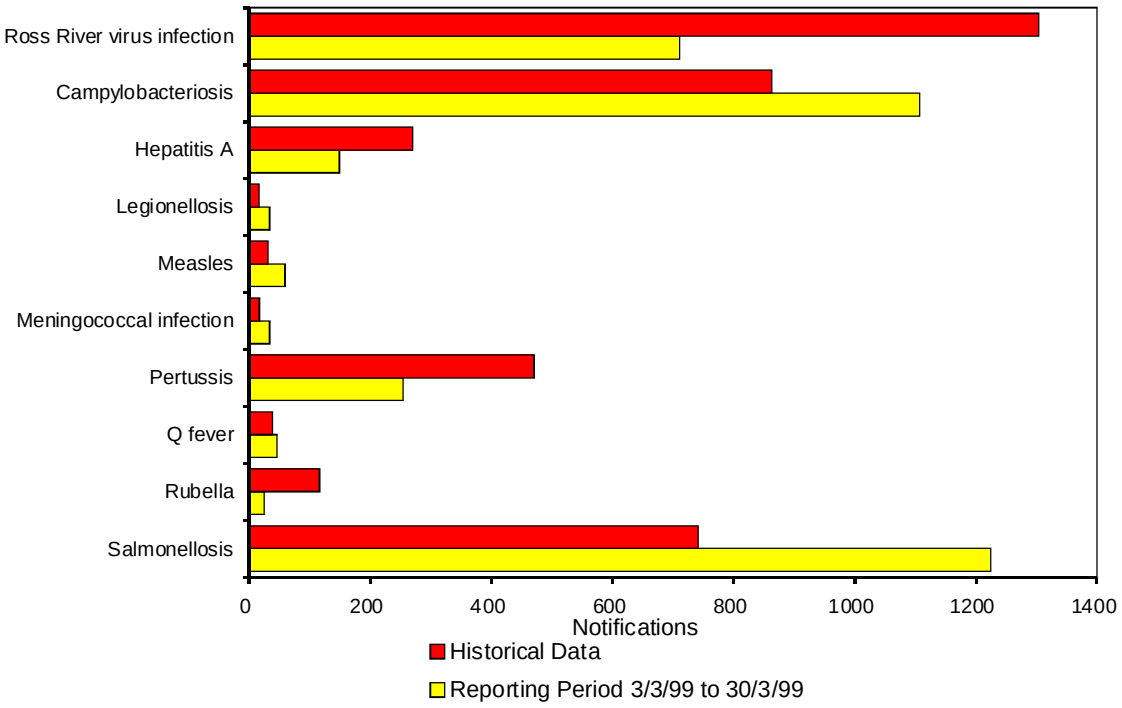
1. Diseases preventable by routine childhood immunisation are presented in Table 1.
2. For HIV and AIDS, see Tables 6 and 7.
3. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
4. No notifications have been received during 1999 for the following rare diseases: lymphogranuloma venereum, botulism, plague, rabies, yellow fever, or other viral haemorrhagic fevers.
5. Data from Victoria for 1998 are incomplete.
6. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
7. WA: genital only.
8. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
9. Nationally reportable from August 1998.
10. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of testings being carried out.
11. Includes hepatitis D and E.
12. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC) became nationally reportable in August 1998.
13. Includes congenital syphilis.
14. Thromotic thrombocytopaenic purpura became nationally reportable in August 1998.
15. NSW, Qld: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Figure 1. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in the corresponding 4 week periods of the last 3 years and the 2 week periods immediately preceding and following those.

Table 3. Virology and serology laboratory reports by contributing laboratories for the reporting period 25 February to 24 March 1999

State or Territory	Laboratory	Reports
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	22
	New Children's Hospital, Westmead	114
	Royal Prince Alfred Hospital, Camperdown	41
	South West Area Pathology Service, Liverpool	57
Queensland	Queensland Medical Laboratory, West End	473
	Townsville General Hospital	20
Tasmania	Northern Tasmanian Pathology Service, Launceston	6
	Royal Hobart Hospital, Hobart	10
Victoria	Monash Medical Centre, Melbourne	29
	Royal Children's Hospital, Melbourne	76
	Victorian Infectious Diseases Reference Laboratory, Fairfield	131
Western Australia	PathCentre Virology, Perth	525
	Princess Margaret Hospital, Perth	38
TOTAL		1,542

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 25 February to 24 March 1999, and total reports for the year

	State or Territory[1]								Total this period	Total reported in *CDI* in 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles, mumps, rubella										
Measles virus		1					35	2	38	48
Mumps virus								7	7	16
Rubella virus			1	1		1	1	1	5	21
Hepatitis viruses										
Hepatitis A virus			1	4		2		16	23	111
Arboviruses										
Ross River virus		7	5	196			5	42	255	569
Barmah Forest virus		1	1	6				4	12	34
Dengue type 3				1					1	23
Dengue not typed						1		5	6	17
Kunjin virus								1	1	1
Flavivirus (unspecified)				3					3	11
Adenoviruses										
Adenovirus type 2							1		1	6
Adenovirus type 3							4		4	13
Adenovirus type 4							2		2	3
Adenovirus type 5							1		1	1
Adenovirus type 7							1		1	1
Adenovirus type 10							1		1	1
Adenovirus type 19	1								1	1
Adenovirus type 37							2		2	4
Adenovirus type 40								5	5	18
Adenovirus not typed/pending		38		1	1	1	8	17	66	361
Herpes viruses										
Herpes virus type 6								2	2	2
Cytomegalovirus		7		19			25	11	62	311
Varicella-zoster virus		14		25			8	48	95	520
Epstein-Barr virus		5	2	51		1	13	36	108	729
Other DNA viruses										
Molluscum contagiosum								1	1	3
Parvovirus				3		1	14	15	33	114
Picorna virus family										
Coxsackievirus B5							1		1	2
Echovirus type 2							1		1	1
Echovirus type 5		1							1	2
Echovirus type 6		6							6	9
Echovirus type 9		1							1	17
Echovirus type 11	1	5							6	25
Echovirus type 22		5							5	11
Echovirus type 30		4							4	18
Poliovirus type 1 (uncharacterised)							2		2	6
Poliovirus type 1 (vaccine strain)						1			1	1
Rhinovirus (all types)		16						15	31	113
Enterovirus not typed/pending		1	3				6	69	79	227

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 25 February to 24 March 1999, and total reports for the year (continued)

	State or Territory[1]									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Total reported in *CDI* in 1999
Ortho/paramyxoviruses										
Influenza A virus		8		1			5	11	25	194
Influenza B virus								8	8	28
Parainfluenza virus type 2								1	1	8
Parainfluenza virus type 3		32		1	1		1	15	50	248
Respiratory syncytial virus	1	38	1	24		2	4	40	110	273
Other RNA viruses										
HTLV-1				1					1	4
Rotavirus		10					4	49	63	286
Norwalk agent							9		9	29
Other										
Chlamydia trachomatis - A-K							1		1	1
Chlamydia trachomatis not typed		31	18	75		7	2	109	242	798
Chlamydia psittaci							2	1	3	20
Chlamydia spp typing pending								1	1	1
Chlamydia species				1					1	3
Mycoplasma pneumoniae		12	1	15			35	7	70	382
Coxiella burnetii (Q fever)		1		12			2	2	17	48
Rickettsia spp - other								1	1	3
Bordetella pertussis		4		13			35	4	56	171
Legionella pneumophila								1	1	5
Legionella longbeachae								3	3	20
Cryptococcus species		1							1	1
Leptospira hardjo								1	1	2
TOTAL	3	250	33	453	2	17	233	551	1,542	5,899

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.

Table 5. Australian Sentinel Practice Research Network reports, weeks 9 to 12, 1999

Week number	9		10		11		12	
Week ending on	7 March 1999		14 March 1999		21 March 1999		28 March 1999	
Doctors reporting	50		47		56		54	
Total encounters	6401		6574		7427		7669	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	18	2.8	27	4.1	10	1.3	24	3.1
Rubella	2	0.3	0	0.0	0	0.0	2	0.3
Measles	0	0.0	0	0.0	0	0.0	2	0.3
Chickenpox	8	1.2	5	0.8	9	1.2	11	1.4
New diagnosis of asthma	5	0.8	11	1.7	10	1.3	5	0.7
Post operative wound sepsis	6	0.9	13	2.0	12	1.6	12	1.6
Gastroenteritis	62	9.7	65	9.9	67	9.0	60	7.8

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of more than 40 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1999;23:55.

LabVISE is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence every four weeks. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1999;23:58.

ASPREN currently comprises about 100 general practitioners from throughout the country. Up to 9,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance in 1999. CDI reports the consultation rates for seven of these. For further information, including case definitions, see CDI 1999;23:55-56.

Additional Reports

HIV and AIDS Surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report , and annually in HIV/AIDS and related diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648; Facsimile: (02) 9332 1837; http://www.med.unsw.edu.qu/ncherc.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 30 November 1998, as reported to 28 February 1999, are included in this issue of CDI (Tables 6 and 7).

Table 6. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 30 November 1998, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998
HIV diagnoses	Female	2	7	0	1	2	0	1	0	13	8	87	73
	Male	3	32	1	5	5	0	18	1	65	52	588	651
	Sex not reported	0	1	0	0	0	0	0	0	1	0	8	12
	Total[1]	5	40	1	6	7	0	19	1	79	60	683	737
AIDS diagnoses	Female	0	1	0	0	0	0	0	0	1	0	13	24
	Male	0	3	0	2	1	0	1	0	7	26	216	295
	Total[1]	0	4	0	2	1	0	1	0	8	26	229	319
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	2	8	14
	Male	0	3	0	2	0	0	2	0	7	10	106	201
	Total[1]	0	3	0	2	0	0	2	0	7	12	114	216

1. Persons whose sex was reported as transgender are included in the totals.

Table 7. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 28 February 1999, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	23	579	8	132	57	4	199	100	1,102
	Male	187	10,488	102	1,859	647	77	3,738	869	17,967
	Sex not reported	0	259	0	0	0	0	24	0	283
	Total[1]	210	11,345	110	1,998	704	81	3,974	972	19,394
AIDS diagnoses	Female	8	168	0	45	20	2	65	25	333
	Male	83	4,498	32	780	326	43	1,563	343	7,668
	Total[1]	91	4,677	32	827	346	45	1,635	370	8,023
AIDS deaths	Female	2	113	0	30	15	2	47	16	225
	Male	62	3,082	24	540	224	28	1,228	244	5,432
	Total[1]	64	3,202	24	572	239	30	1,281	261	5,673

1. Persons whose sex was reported as transgender are included in the totals.

Childhood Immunisation Coverage

Tables 8 and 9 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 October and 31 December 1997 and at 24 months of age for the cohort born between 1 October and 31 December 1996, according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in CDI 1998;22:36-37.

Table 8. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 1997; assessment date 31 December 1998

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,062	22,009	823	11,700	4,594	1,547	15,520	6,067	63,322
Diphtheria,Tetanus, Pertussis (%)	88.1	84.2	80.2	88.6	88.2	87.8	87.2	85.5	86.3
Poliomyelitis (%)	88.0	83.7	79.3	87.4	88.1	87.9	87.3	85.4	85.9
Haemophilus influenzae type b (%)	87.9	84.3	81.7	88.9	88.3	87.8	87.5	85.3	86.4
Fully Immunised (%)	87.7	82.7	74.6	86.5	87.5	87.2	86.5	84.4	84.9
Change in fully immunised since last quarter (%)	-0.9	+0.2	-0.7	+0.6	+0.9	+2.1	+0.5	+0.1	+0.4

Table 9. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 1996; assessment date 31 December 1998[1]

	State or Territory								
Vaccine	ACT	NSW	NT[1]	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,113	22,251	890	11,824	4,671	1,654	16,145	6,318	64,866
Diphtheria,Tetanus, Pertussis (%)	83.4	79.2	66.1	84.3	80.9	81.9	81.8	79.4	80.9
Poliomyelitis (%)	88.4	83.2	70.9	89.7	85.5	88.5	87.6	82.5	85.6
Haemophilus influenzae type b (%)	82.3	79.2	69.7	84.2	80.8	81.9	82.1	79.4	81.0
Measles, Mumps, Rubella (%)	88.1	84.3	74.7	90.5	85.4	87.2	87.8	84.3	86.4
Fully Immunised (%)[2]	77.7	66.9	54.6	77.5	68.1	71.6	72.0	66.0	70.3
Change in fully immunised since last quarter (%)	+2.3	+0.1	-0.2	+1.6	+0.9	+3.9	+2.0	+4.4	+1.5

1. The 12 months age data for this cohort was published in *CDI* 1998;22:170.
2. These data relating to 2 year old children should be considered as preliminary. The proportions shown as "fully immunised" appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Aged Care. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone 02 6124 6607.

Overseas briefs

Source: World Health Organization (WHO)
This material has been condensed from information on the WHO internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the CDI homepage.

Nipah virus

Malaysia and Singapore

The United States Centers for Disease Control and Prevention (CDC) have confirmed that of the 15 blood samples from abattoir workers received from Singapore for testing, 11 (including 1 death) tested positive for Nipah virus. No additional cases have been reported in Singapore during the past two weeks and the outbreak there has now ended.

The outbreak of encephalitis is still ongoing in Malaysia. For up-to-date information on the number of cases and deaths, please see the web site of the Department of Public Health, Ministry of Health, Malaysia at: "http://dph.gov.my/press/press2/cases.htm"

The Nipah virus is a new virus. It is similar to the Hendra virus which was responsible for the deaths of two humans and some race horses in Australia in 1994. However, genetic analysis of the new virus shows significant differences. Experts at CDC have noted that transmission of the virus has been confined to persons who have had direct contact with infected pigs. Currently, there is no evidence that the virus can be transmitted from human to human. Travellers to Malaysia should be aware of this outbreak of febrile encephalitis, which thus far has involved only those closely associated with pig farms. No travel restrictions are indicated at this time.

A report of the outbreak can be found in the article, " Outbreak of Hendra-Like Virus - Malaysia and Singapore, 1998-1999" in *MMWR*, April 9, 1999 48(13); 265-269 at: "http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00056866.htm"

Influenza A(H9N2)

Hong Kong Special Administrative Region of China

Influenza A(H9N2) viruses have been identified in two hospitalised children, ages 1 and 4 years, in Hong Kong SAR. One of the children is from Kowloon, and the other from Hong Kong Island.

Further genetic analysis of the human virus isolates from 2 hospitalized children in Hong Kong SAR in March 1999, confirmed to be influenza A(H9N2) by the WHO Collaborating Centre for Influenza in London (United Kingdom) WHO Collaborating Centres in Atlanta (United States), has revealed that the viruses are genetically closely related to, but distinct from, influenza A/Quail/Hong Kong/GI/97(H9N2) isolates detected in 1997 during the influenza A(H5N1) outbreak in Hong Kong SAR.

Studies on the spread of A(H9N2) viruses between cages of chickens indicate that quail H9N2 virus is transmitted by aerosol more effectively than by faecal transmission.

In China, 5 human cases of influenza A(H9N2) were apparently identified in March 1999, but laboratory confirmation of the virus was not reported. Of the 5 cases, the youngest was 1 year old and the oldest was a man in his 70s. All patients apparently had mild influenza-like symptoms and recovered with no medical complications.

Acute haemorrhagic fever syndrome

Southern Sudan

During the last week of March, an outbreak of an unidentified haemorrhagic fever was reported from Rumbek county. The number of cases and deaths is unknown. Samples were immediately sent for testing to the National Institute for Virology in South Africa. A team from WHO has now reached the area in order to: (1) continue the investigation of the etiology of the outbreak; (2) provide barrier nursing material and training to health care workers; and (3) determine the need for additional on-site personnel.

Cholera

Madagascar

Cholera cases have been reported in Madagascar for the first time. Between 24 March and 12 April, 278 cases of acute diarrhoea were reported. The first cholera case confirmed in the laboratory was diagnosed as *V.cholerae* O1 Ogawa. The areas affected are in the west of Madagascar in the districts of Antsohihy and Mahajanga (Mahajanga Province).

Strict control measures have been put in place and a task force from the Ministry of Health went immediately to the area.

Brazil

The Ministry of Health has reported an outbreak of cholera in the municipality of Paranagua, Parana State. The cases occurred in the villages of Guarani and Araça. Up to 31 March, a total of 235 cases (205 confirmed) and 3 deaths had been reported. Strict investigative and control measures are being implemented, including inspection and training of food vendors and distribution of health education material.

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Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereering process. Instructions to authors can be found in *CDI* 1999;23:59.