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Communicable Diseases Intelligence

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Editor Dr I.F. Cook

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1809 reports were processed during this period.

Nineteen cases of Q fever (17 males, 1 female, 1 not stated) were reported during this period (NSW [18], SA [1]). The age of patients ranged from 16 to 44 years. No exposure details were provided.

Congenital rubella syndrome was diagnosed in an 8 day old boy with thrombocytopenia cerebral calcification and low birth weight for delivery date. Igm to rubella was identified in the child's serum using ELISA testing.

The child's mother had been tested for rubella antibody status during the first trimester of her first pregnancy. However, the patient moved during that pregnancy and post-partal vaccination against rubella was not carried out. During the second pregnancy, the woman came into contact with an outbreak of rubella, but did not develop any clinical symptoms of the disease.

Currently the National Health and Medical Research Council recommends that women should be routinely tested for rubella antibody status in each pregnancy and non-immune women be vaccinated before the next pregnancy.

Measles virus was isolated from a nasophanyngeal swab from a 1 year old boy, one month after vaccination with measles/mumps vaccine. A Morbilli form rash was seen although no Koplik spots were seen.

- The Bulletin is compiled and distributed by the Public Health Section, Communicable Diseases Branch, Department of Community Services and Health.
- Contributions are solicited, and do not preclude later publication elsewhere.
- Material appearing in the Bulletin may be quoted provided suitable acknowledgement is made.
- Figures given may be subject to revision.

OVERSEAS BRIEFS:

1. ADVICE ON MENINGOCOCCAL MENINGITIS VACCINATION

. Travellers to Mecca

For the coming Hajj pilgrimage season (from late July) authorities in Saudi Arabia require vaccination of pilgrims visiting Mecca.

. Travellers to Sudan and adjacent areas

WHO has released an update on the epidemic of group A meningococcal meningitis in Sudan; 13,404 cases have been recorded with 900 deaths. Vaccination is recommended for travellers to Sudan and adjacent areas.

Availability of the vaccine

The appropriate vaccine is bivalent A/C meningococcal meningitis vaccine, Mencevax (R) AC (Smith, Kline & French). This vaccine is available at the vaccinees own expense from the Commonwealth Serum Laboratories in Melbourne (03) 389 1276.

A single dose of the vaccine provides effective immunity one to two weeks after administration and protection for 1-3 years.

2. TRAVEL RESTRICTIONS ON HIV INFECTED PERSONS

Travel restrictions on HIV infected persons (based on AIDS Newsletter March 1988), were published in CDI 88/11.

Since that time the following additions, amendments and corrections have been received.

Additions

Country	Type of Visitor	Type of Restriction
Belize	Foreign workers or migrants	Must have evidence of negative HIV test in past 3 months
Cyprus	Foreign nationals working in nightclubs or cabaret, students from Africa	Tested on arrival
France	There is a clear statement that a positive HIV test is not grounds for refusal of resident status, in the absence of clinical disease	
Mongolia	Foreign students	Must be certified antibody negative
Papua New Guinea	Foreign workers	Must be HIV antibody negative

Additions cont

Country	Type of Visitor	Type of Restriction
Poland	Foreign students	Must be certified HIV antibody negative
Thailand	Foreign nationals seeking residence who suffer from AIDS	Refused entry
United States of America	All aliens seeking residence	Tested for HIV antibody. HIV infection listed as a 'dangerous, contageous disease'

Amendments

Country	Type of Visitor	Type of Restriction
Austria (Klagenfurt only)	Foreign workers applying for residence permits	Must be certified antibody negative
Costa Rica	Crews of all ships, foreigners applying for residence visa	Must be certified antibody negative
Philippines	All immigrants, refugees, illegal residents, visitors for longer than 6 months, all foreign sailors, and aliens seeking a change in status from temporary to permanent resident	From 18.4.88 must be certified antibody negative
Qatar	Foreign nationals applying for work permits, except diplomats	Compulsory testing, or have an AIDS certificate issued in the past 6 months
Syria	Foreign nationals applying for work permits, foreign students	Compulsory testing on arrival at specified centres
United Arab Emirats (UAE)	Foreign nationals applying for work or resident permits, except diplomats	Compulsory testing in UAE

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Corrections

Country	Type of Visitor	Type of Restriction
Bulgaria	Foreign nationals staying more than one month	Compulsory testing within **1-3 days** of arrival
Japan	There is at present no restriction on travellers from abroad, and no restriction is proposed in the legislation that is pending.	

PAEDIATRIC MYCOPLASMA PNEUMONIAE INFECTIONS

(Contributed by Dr S.J. MacInnes, Microbiology Department, Royal Children's Hospital, Melbourne, Victoria.)

Mycoplasma pneumoniae usually causes a relatively mild respiratory tract disease, but a wide range of extrapulmonary syndromes can occur. Infection is endemic in the general population with epidemics occurring in 4-6 year cycles.

M.pneumoniae causes 20% of all pneumonias in the general population and up to 50% in closed populations. The disease is most commonly seen in the 5 to 18 year age group[1].

Diagnosis is usually based on clinical judgement and serology, either complement fixation or detection of specific IgM and a rise in specific IgG. Culture can also be performed and a gene probe is available.

One hundred and thirty five cases of *M.pneumoniae* infection were diagnosed at the Royal Children's Hospital, Melbourne from October 1986 to March 1988 inclusive. This figure represents a current epidemic as there was only one other case in 1986 and only two cases in 1985.

The clinical presentation of the 135 cases is shown in Table 1.

Table 1: Clinical presentation of *M.pneumoniae* cases

Primary Diagnosis	Number	Accompanied by:	
Pneumonia*	105	Erythema multiforme	1
Resp. Tract. Infection#	20	Other rashes	3
Croup	3	Erythema nodosum	1
Bronchiolitis	3	Guillan Barre'	1
PUO	1	Febrile encephalopathy	1
Acute polyarthritis	1		
Erythema multiforme	1		
Erythema nodosum	1		
Total	135		

* with radiological changes.
\# no radiological changes or no further information.

Diagnosis was based on a combination of serology and culture, where possible. Serology was carried out using an enzyme immunosorbent assay (ELISA) to detect specific *M.pneumoniae* IgM and/or a significant rise in specific *M.pneumoniae* IgG.[2] Culture was carried out using modified Hayflick's agar[3] and SP4 broth[3].

Of the 135 cases reported diagnosis was by:

- serology and culture for 29 cases;
- serology only for 79 cases (culture not performed on 65); and
- culture only for 26 cases (serology not performed on 22).

Figure 1 shows the distribution of cases during the period October 1986 to March 1988.

FIGURE 1

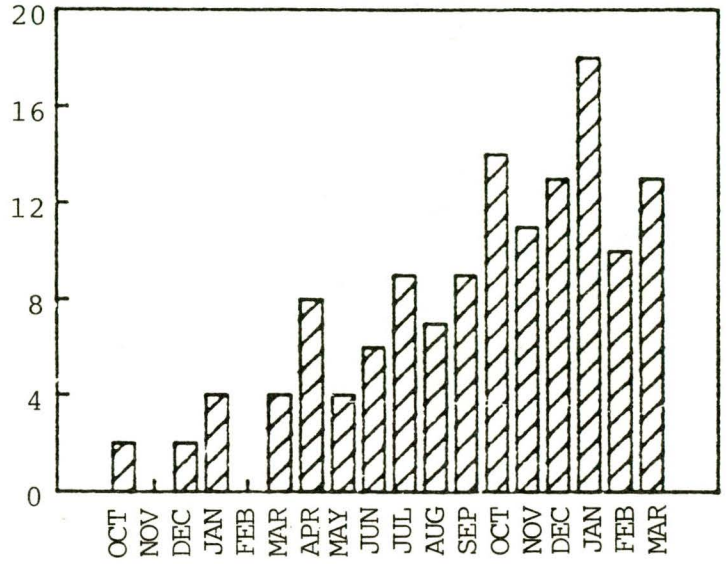
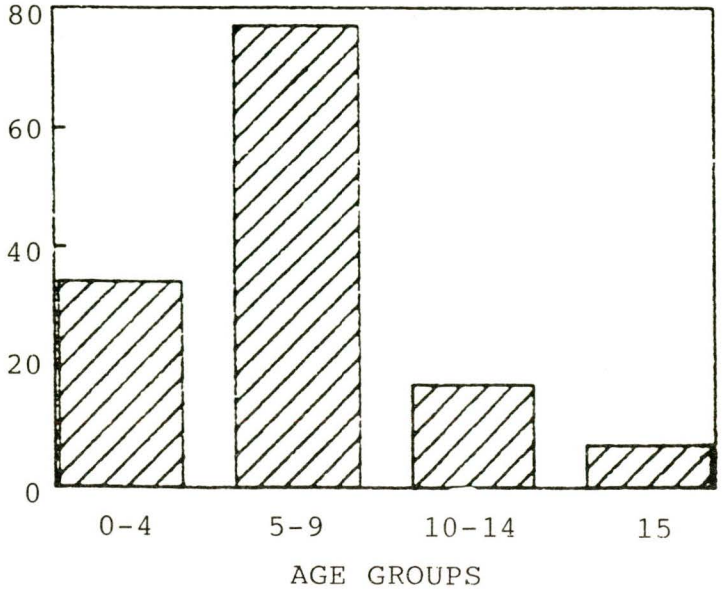


Figure 2 shows the age distribution of cases. The majority of cases were in the 5-9 year old age group.

FIGURE 2

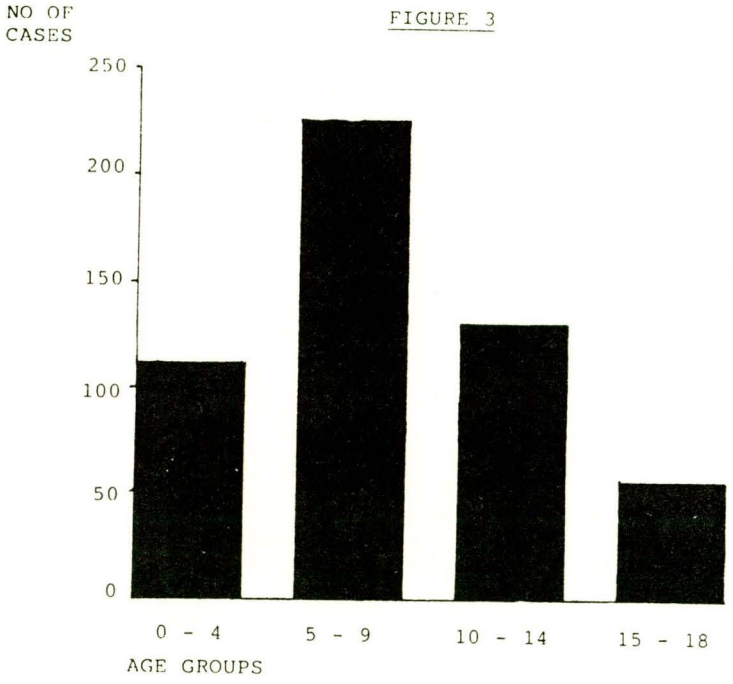


CDI Editorial Comment:

M.pneumoniae is usually characterised by low grade fever, myalgia, malaise and headache followed by pharyngitis and tracheobronchitis [1].

In 1987, 993 cases of *M.pneumoniae* were reported to CDI with 528 cases in the 0-18 years age group. The age distribution of the latter cases are shown in Figure 3. This figure shows a similar age distribution as found in the above report.

FIGURE 3



Pulmonary infiltrates develop in only 3-10% of persons infected with *M.pneumoniae*. Radiologically the pneumonia is described as atypical and is usually visualized in the lower lobes of the lungs, however upper lobe, multilobe and bilateral pulmonary infiltrates have also been reported [4].

Most patients with *M.pneumoniae* infection have an uneventful recovery whether treatment was given or not. Lung abscess, pneumatocele formation, extensive lobar consolidation and large pleural effusions have been described in cases of laboratory confirmed (either serology or culture) *M.pneumoniae* infection [5].

The extrapulmonary manifestations of *M.pneumoniae* infection are diverse and can involve nearly every organ system. Complications may include meningoencephalitis, haemolytic anaemia and mucocutaneous lesions. Myocardial involvement with *M.pneumoniae* occurs in up to 4.5% of patients infected with the organism [6].

Diagnosis is based on clinical judgement and serology and culture is performed where possible (though identification takes up to 3 weeks). The determination of IgM cold agglutinins which appear during the first or second week of illness may be useful in making a tentative diagnosis of *M.pneumoniae*.

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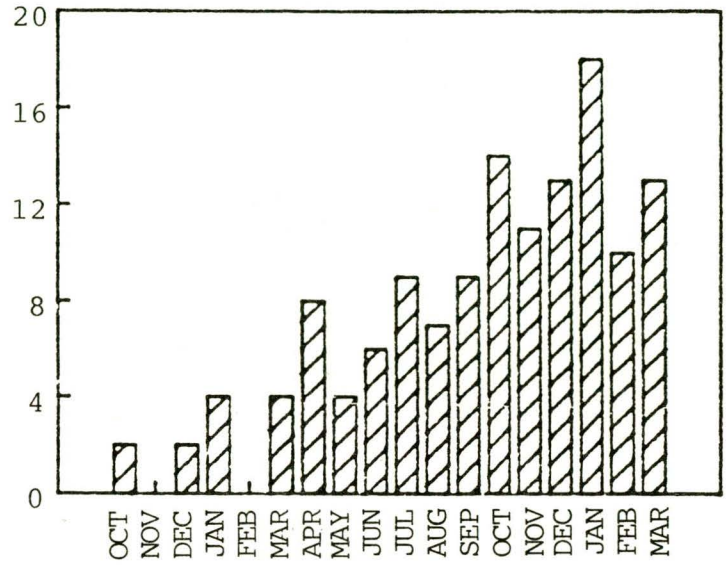

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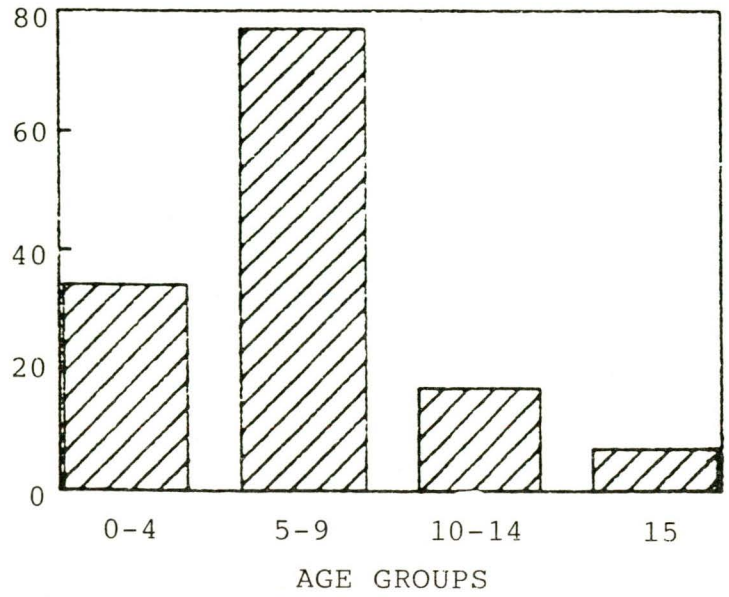
FIGURE 2



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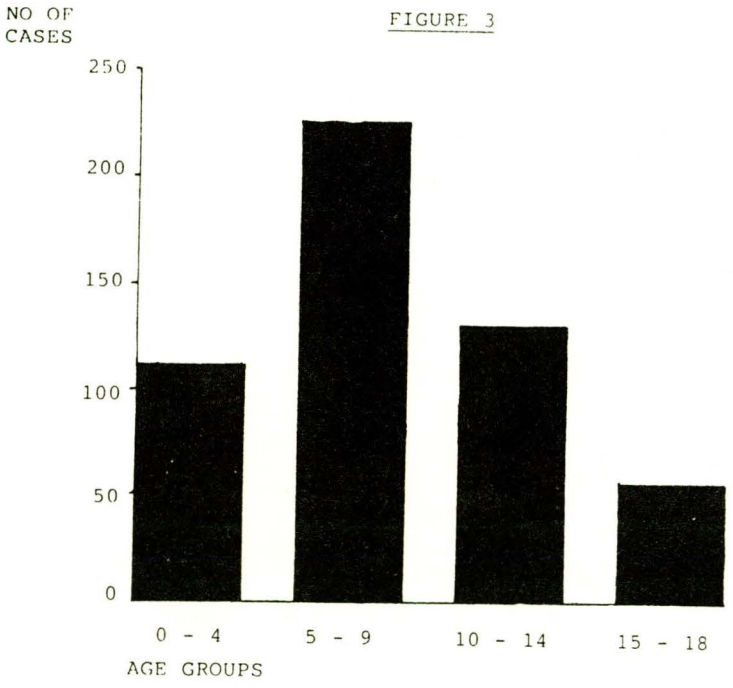

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Diagnosis is based on clinical judgement and serology and culture is performed where possible (though identification takes up to 3 weeks). The determination of IgM cold agglutinins which appear during the first or second week of illness may be useful in making a tentative diagnosis of *M.pneumoniae*.

Erythromycin and tetracycline are effective antibiotic agents for the treatment of respiratory tract disease due to *M.pneumoniae*. Neither drug eliminates the organism from the respiratory tract, however the duration of cough and fever and the time taken for resolution of pneumonia are significantly reduced in treated patients[6].

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PREVENTION OF PERINATAL TRANSMISSION OF HEPATITIS B VIRUS

(Based on MMWR Vol. 37, No. 22, June 10 1988)

Transmission of hepatitis B virus (HBV) from mother to infant during the perinatal period represents one of the most efficient modes of HBV infection and often leads to severe long-term sequelae. Infants born to mothers positive for hepatitis B surface antigen (HBsAg) and hepatitis B "e" antigen (HBeAg) have a 70%-90% chance of acquiring perinatal HBV infection, and 85%-90% of infected infants will become chronic HBV carriers[1,2]. It has been estimated that more than 25% of these carriers will die from primary hepatocellular carcinoma or cirrhosis of the liver[3]. These deaths usually occur during adulthood, when familial and financial responsibilities make them particularly devastating.

In the United States, an estimated 16,500 births occur to HBsAg-positive women each year (about 4,300 of whom are also HBeAg-positive), and approximately 3,500 of these infants become chronic HBV carriers. Antenatal screening of all pregnant women would identify those who are HBsAg-positive and thus would allow treatment of their newborns with hepatitis B immunoglobulin (HBIG) and hepatitis B (HB) vaccine, a regimen that is 85%-95% effective in preventing the development of the HBV chronic carrier state[2,4-6].

In 1984, the Immunization Practices Advisory Committee (ACIP) of the United States recommended that pregnant women in certain groups at high risk for HBV infection be screened for HBsAg during an antenatal visit and, if found to be HBsAg-positive, that their new borns receive HBIG and HB vaccine at birth[7]. No data are available regarding the proportion of high-risk women currently being screened in clinical practice, but several studies and the experience of public health workers indicate that major problems have been encountered in implementing these recommendations[8-12]. These include:

. concerns about the sensitivity, specificity, and practicality of the current ACIP guidelines for identifying HBV carrier mothers;

- lack of knowledge among antenatal health-care providers about the risks of perinatal transmission of HBV and about recommended screening and treatment procedures;

- poor coordination among medical-care workers who provide treatment and follow-up of mothers and infants; and

- refusal of some public and private third-party health insurers to reimburse for HBV screening of pregnant women and treatment of their infants.

In addition, concern has been expressed that these recommendations may not be practical or applicable in some U.S. jurisdictions where HBV infection is highly endemic, such as parts of Alaska and certain Pacific Islands.

The problems encountered in implementing the currently recommended strategy of screening high-risk women have been examined by a number of investigators:

- Recent studies in several large inner-city hospitals, where all pregnant women were tested for HBsAg, have found that only about 35%-65% of HBsAg-positive mothers would have been identified by following the current ACIP guidelines [8-12]. In these studies, the prevalence of HBsAg in inner-city black (0.4%-1.5%) and Hispanic women was higher than expected.

- Several investigators expressed concern that many health-care providers are too busy or may be reluctant to obtain the sexual and drug-use history necessary to identify high-risk patients for screening.

- In addition, persons providing health care to pregnant women often are not aware of the risk of perinatal transmission of HBV and of the recommended screening and treatment guidelines. In one study, 40% of obstetricians could name no more than two groups at high risk for HBV infection, and only 28% knew the recommended treatment for infants born to HBV carrier mothers (CDC, unpublished data).

Given these limitations, it is now evident that routine screening of all pregnant women is the only strategy that will provide acceptable control of perinatal transmission of HBV infection in the United States. Screening the approximately 3.5 million pregnant women per year for HBsAg would identify 16,500 positive women and allow treatment that would prevent about 3,500 infants from becoming HBV carriers. Recent studies also indicate that the costs and benefits of universal testing of mothers are comparable to those encountered in other widely implemented programs of antenatal and blood-donor screening [13-14].

HBsAg testing should be done early in pregnancy when other routine antenatal testing is done. The HBsAg test is widely available and can be added to the routine antenatal "panel" of tests without requiring additional patient visits. The

advantages of making HBsAg testing routine during early pregnancy include:

- the identification of HBV carrier mothers is not dependent on the health-care providers identifying high-risk women or on ordering HBsAg as a special test;
- the availability of test results before delivery so that infants can receive HBIG and vaccine without delay after birth; and
- appropriate counselling of families before delivery[15].

As more than 90% of women found to be HBsAg-positive on routine screening will be HBV carriers, routine follow-up testing later in pregnancy is not necessary. In special situations, such as when the mother is thought to have acute hepatitis B, when there has been a history of exposure to hepatitis B, or when particularly high-risk behaviour such as parenteral drug use has occurred during the pregnancy, an additional HBsAg test can be ordered during the third trimester. Few women in populations at low risk for HBV infection will have a change in HBsAg status during subsequent pregnancies. However, because of the expected benefits of making HBsAg testing a routine part of each antenatal panel, testing should be done during each pregnancy.

Women who present for delivery without antenatal care or without medical records documenting the results of HBsAg screening should have the HBsAg test done as soon as possible after admission, since delay in administration of HBIG to infants of carrier mothers will decrease the efficacy of therapy. In the studies that demonstrated the highest efficacy (85%-95%) of combined HBIG and HB vaccine prophylaxis, HBIG was administered within 2-12 hours after birth[2,4-6]. In one study in which only HBIG was used for prophylaxis, no efficacy was found if HBIG was given more than 7 days after birth, and a significant decrease in efficacy was observed if it was given more than 48 hours after birth[16]. Only one-third of U.S. hospitals currently perform the HBsAg test as an in-house procedure, and many of these have technicians who are trained to do the test available on only one shift. Hospitals that cannot rapidly test for HBsAg should either develop this capability or arrange for testing to be done at a local laboratory or blood bank where test results can be obtained within 24 hours.

The commercially available HBsAg tests have an extremely high sensitivity and specificity if positive tests are repeated and confirmed by neutralisation as recommended by the manufacturers of the reagent kits. Testing for other markers of HBV infection, such as HBeAg, is not necessary for maternal screening. Mothers who are positive for both HBsAg and HBeAg have the highest likelihood of transmitting HBV to their newborns. However, infants of mothers who are HBsAg-positive but HBeAg-negative may become infected and develop severe, even fatal, fulminant hepatitis B during infancy[17,18]. For this reason, HBIG and HB vaccine treatment of all babies born to HBsAg-positive women is recommended.

HBsAg-positive mothers identified during screening may have HBV-related acute or chronic liver disease and should be evaluated by a physician. Identification of women who are HBV carriers through antenatal screening presents an opportunity to vaccinate susceptible household members and sexual partners of HBV carriers, as previously recommended[19]. Screening and vaccination of susceptible contacts should be done by the family's paediatrician, primary health-care provider, or the physician evaluating the clinical status of the HBsAg-positive pregnant woman.

Implementation of the recommendations to prevent perinatal transmission requires maternal screening, treatment of the newborn in the hospital, and administration of subsequent doses of HB vaccine to the infant during paediatric visits at 1 and 6 months of age. This multistep process requires effective transfer of information among several groups of health-care providers, knowledge of recommended treatment, and availability of HBIG and vaccine at separate facilities. Treatment failures due to lack of communication among health-care providers can occur, especially in situations where antenatal, obstetric, and paediatric care are provided in different facilities[20].

These deficiencies can be overcome by:

- Central coordination of the treatment of these infants by city, county, or state health departments would improve the education of the health-care providers involved and increase the likelihood that proper treatment is provided.
- Universal vaccination of newborns with HB vaccine is recommended to prevent disease transmission both during the perinatal period and during childhood, in certain populations under U.S. jurisdiction, including Alaskan Natives and Pacific Islanders, as well as in many other parts of the world, where HBV infection is highly endemic in the general population[21] and transmission occurs primarily during childhood. Several studies have shown that HB vaccine given without HBIG will prevent 70%-85% of perinatal HBV infections and 95% of early childhood infections[22,23].

- Vaccinating all children in areas with highly endemic HBV infection where antenatal screening is impractical because the population is isolated, laboratory facilities are not available, and/or health-care budgets and personnel are limited. In these areas, control of HBV infection can be better achieved by directing available resources into programs to vaccinate all children with HB vaccine. Programs for screening all mothers for HBsAg and providing HBIG to infants born to carrier mothers are costly and will add only modestly to disease prevention. They should be considered only after the program for universal vaccination of children has been implemented.

RECOMMENDATIONS

ALL PREGNANT WOMEN SHOULD BE ROUTINELY TESTED FOR HBsAg DURING AN EARLY ANTENATAL VISIT IN EACH PREGNANCY.

This testing should be done at the same time that other routine antenatal screening tests are ordered. In special situations,

such as when acute hepatitis is suspected, when there has been a history of exposure to hepatitis, or when the mother has a particularly high-risk behaviour such as intravenous drug use, an additional HBsAg test can be ordered later in the pregnancy.

If a woman has not been screened antenatally or if test results are not available at the time of admission for delivery, HBsAg testing should be done at the time of admission, or as soon as possible thereafter. If the mother is identified as HBsAg-positive more than 1 month after giving birth, the infant should first be tested for HBsAg; if negative, the infant should be treated with HBIG and HB vaccine. Hospitals where infants are delivered should have HBsAg testing capabilities or should be able to obtain HBsAg results within 24 hours from a local laboratory.

If a serum specimen is positive for HBsAg, the same specimen should be tested again, and then the test results should be confirmed by neutralization. It is unnecessary to test for other HBV markers during maternal screening, although HBsAg-positive mothers identified during screening may have HBV-related acute or chronic liver disease and should be evaluated by their physician.

Infants born to HBsAg-positive mothers should receive HBIG (0.5 mL) intramuscularly (IM) once they are physiologically stable, preferably within 12 hours after birth. HB vaccine, either plasma-derived (10 µg per dose) or recombinant (5 µg per dose), should be administered IM in three doses of 0.5 mL each. The first dose should be given concurrently with HBIG but at a different site. If vaccine is not immediately available, the first dose can be given within 7 days after birth. The second and third doses should be given 1 month and 6 months after the first.

Testing the infant for HBsAg and its antibody (anti-HBs) is recommended at 12-15 months of age to monitor the effectiveness of therapy. If HBsAg is not detectable and anti-HBs is present, the child can be considered protected. Testing for antibody to hepatitis B core antigen (anti-HBc) is not useful, since maternal anti-HBc can persist for more than a year. HBIG and HB vaccination do not interfere with the routine childhood immunizations.

Household members and sexual partners of HBV carriers identified through antenatal screening should be tested to determine susceptibility to HBV infection and, if susceptible, should receive HB vaccine. Screening and vaccination of susceptible contacts should be done by the family's paediatrician, primary health-care provider, or the physician evaluating the clinical status of the HBsAg-positive pregnant woman.

Obstetric and paediatric staff should be notified directly about HBsAg-positive mothers so that the neonate can receive therapy without delay after birth and follow-up doses of vaccine can be given. Hospitals, as well as state, county, and city health departments, should establish programs to educate appropriate health-care providers about perinatal transmission of HBV and its control through maternal screening, treatment of infants, and vaccination of susceptible household and sexual contacts of HBV carrier women.

Programs to coordinate the activities of those providing antenatal care, hospital-based obstetrical services, and baby health care services must be established to assure proper follow-up and treatment of infants born to HBsAg-positive mothers and other susceptible household and sexual contacts.

In populations under U.S. jurisdiction in which hepatitis B infection is highly endemic, including certain Alaskan Native and Pacific Island groups, vaccination of all newborns with HB vaccine is the most effective strategy for HB control. In these populations, such vaccination programs should be given highest priority. In areas where HBsAg screening of mothers and use of HBIG in infants born to HBV carrier mothers are not practical, the vaccination of all newborns with HB vaccine should be considered the appropriate treatment.

Editorial Note:

Hepatitis B vaccine is the first human vaccine that can prevent both serious chronic disease and a uniformly fatal type of cancer. These recommendations, developed in consultation with representatives of the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics, represent a major step toward the control of perinatal hepatitis B transmission in the United States.

CDI EDITORIAL COMMENT

In 1987 the Australian Government adopted a neonate hepatitis B program which offers HB vaccine and HBIG free of charge for the vaccination of babies born to women in high risk community groups where the carrier rates exceed 5%. Screening for HBsAg is not a prerequisite and vaccination is not compulsory for babies born to mothers in these high risk groups. To establish whether a pregnant woman is in a high risk group involves some detailed questioning and this is unlikely to occur in the context of a conventional visit to the physician. Risk assessment is up to the discretion of the health care provider and depends on the providers knowledge of hepatitis B epidemiology and prevention. Methods of high risk assesssment have therefore been found to miss a number of carrier mothers who are usually asymptomatic.[8,14]

The advantage offerred by the United States policy, where all pregnant women are screened for HBsAg as part of their routine antenatal screening program, is the identification of pregnant women who are hepatitis B carriers regardless of their high risk status defined by a risk assessment. Their neonates can then be offered HB vaccine and HBIG. Such screening of all pregnant women for HBsAg permits the use of HBIG to be restricted to babies born of women who are HBsAg positive, thereby constituting a substantial financial saving when the cost of HBIG is approximately $50 per dose. A study conducted in the United States found that routine screening for HBsAg,and immunisation of neonates of HBsAg positive mothers with HB vaccine and HBIG, was cost effective at a prevalence rate of hepatitis B of 0.06%, which is significantly lower than their national prevalence of 0.2%[13]. In Australia the national prevalence of hepatitis B is approximately 0.1-0.2%.

Another strategy which is in the process of being adopted by several countries (New Zealand, Alaska, People's Republic of China, American Samoa, Taiwan and Singapore) involves the immunisation of all neonates with HB vaccine as part of the routine childhood immunisation schedule. The vaccination of all neonates would break the perinatal transmission cycle and would ultimately reduce the incidence of hepatitis B and its sequelae. This could only occur if vaccine compliance was high and the protection afforded by the vaccine was longlasting. However, even a program where all neonates were given HB vaccine would also require HBsAg screening of pregnant women as those babies born to carrier mothers require HBIG as soon as possible (within 24 hours) after birth to prevent acute hepatitis. An antenatal screening program would therefore be required to complement a universal neonate HB vaccine program. A program whereby all neonates would receive HB vaccine (and HBIG where necessary) would also reduce the horizontal transmission which occurs between prepubertal children where there is interaction between high and low prevalence groups[24,25]. In New Zealand an extended HB vaccine program has been established (which offers HB vaccine to all infants and preschoolers) as a result of high levels of horizontal transmission of hepatitis B[25].

The high cost and lower acceptance (due to fears of HIV contamination) of the HB plasma derived vaccine has ruled out its use for mass immunisation programs of children in the past. However, the development of a recombinant DNA (rDNA) HB vaccine has considerably reduced the cost of the vaccine (less than half price) as compared to the plasma derived HB vaccines. The rDNA HB vaccine is also more consistent in quality and can be produced in sufficient quantities for mass immunisation programs. Further improvements in the rDNA HB vaccine technology is likely to reduce the cost even further.

The ideal approach towards controlling hepatitis B would be a program of immunisation with HB vaccine for all neonates in conjunction with routine antenatal screening of pregnant women for HBsAg and the administration of HBIG to babies born to HBsAg positive mothers.

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GONOCOCCAL SURVEILLANCE - AUSTRALIA

(Contributed by the Australian Gonococcal Surveillance Programme (AGSP). Co-ordinator, Dr J.W. Tapsall, The Prince of Wales Hospital, Sydney, NSW 2031)

This report provides details of penicillin sensitivity of 473 strains of gonococci isolated in participating laboratories over the period 1 October to 31 December 1987 (Table 1). The sensitivity of the isolates to penicillin was determined by a standardised techniques[1].

Table 1: Penicillin sensitivity of isolates of N. gonorrhoea 1 October - 31 December 1987

Centre	Percentage of isolates					
	Sensitive*		Less Sensitive**		PPNG	
Brisbane	24	(23.7)	53	(55.9)	5	(16.1)
Sydney	11.3	(6.2)	49	(49.3)	21.6	(23.2)
Melbourne	13.8	(14.7)	47.4	(56.0)	24	(12.9)
Adelaide	10.3	(20.8)	67.2	(55.8)	8.6	(11.7)
Perth	16.2	(28.4)	54	(32.9)	10.8	(14.8)

* Sensitive MIC = 0.004-.016 mg/L

** Less Sensitive MIC = 0.06-0.25 mg/L

Figures in parenthesis represent data for the same period in 1986.

The chromosomally-mediated intrinsic resistance to penicillin has a bimodal distribution into categories designated as 'sensitive' or 'less sensitive' (see table footnotes). A smaller number of isolates not in these categories are not included in the table. The gradual increase in intrinsic resistance noted over many years continues. High level intrinsic resistance (MIC 1.0 mg/L or greater) was detected in a small number of isolates from four centres. Strains of this type represented less than 2% of total isolates.

Penicillinase-producing gonococci continued to account for a large proportion of strains, the 77 PPNG detected representing 16.2% of all isolates in this quarter. As has been the case over the past few years, a significant proportion of PPNG isolated in Sydney and Melbourne were cultured from patients who contracted their disease locally. However, these were the only centres in which local spread of PPNG was confirmed whereas in the equivalent period in 1986[2] sustained domestic transmission was occurring in other areas.

Again a significant decrease was noted in the number of gonococci isolated in this quarter. In the equivalent quarter in 1986, 1077 strains were isolated, and the 473 strains reported here represents a further decrease from the 560 isolates in the previous quarter (July-September 1987, CDI 88/9).

REFERENCE

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SALMONELLA STANLEY CONTAMINATION OF COCONUT

(Contributed by Dr B Oliver, Mr K Bradshaw [Health Department Victoria] and Dr J R L Forsyth [Microbiological Diagnostic Unit])

In Victoria between June and November 1987, *Salmonella stanley* was cultured from 51 patients: from faecal samples of 50 patients with gastroenteritis and from joint fluid of a boy with acute arthritis of the hip.

Fifty four contacts from 19 families were screened; mostly those in which members other than the index case had suggestive symptoms. Nine out of 20 contacts with symptoms, and 6 out of 34 without symptoms, yielded *S. stanley*.

Epidemiology

Apart from a peak of 11 cases from 9-22 August, cases presented at a steady rate of 6 per fortnight through July and August. However, from September there was a slow and irregular decline in the number of new cases. The last patient in this outbreak became ill on 12 November.

Most cases occured among toddler age groups; 21 (41%) of those patients who visited a doctor were 1 or 2 years old and 10 (20%) 3 to 5 years. Only 3 (6%) were less that 1 year old.

Geographically, most cases were in the Eastern half of Victoria, with clustering in the outer Eastern suburbs of Melbourne and along the Princes Highway to Bairnsdale. Minimal time/place clustering was present was observed in this outbreak of *S. stanley*.

Clinical

Retrospective questioning revealed that:

- 48/51 (94%) of the cases who attended doctors and 7/15 (47%) of those found on screening had experienced diarrhoea. The diarrhoea was of variable intensity and was often prolonged, lasting 5 days or more.

- Rectal bleeding was reported by 23/51 (45%), ranging from spotting to grossly bloody diarrhoea. Several started to bleed after 3 or 4 days' diarrhoea, others presented with grossly bloody stools. One child was hospitalised for observation of a possible intussusseption.

- Vomiting, (transient in many cases) occured in 47% of cases, abdominal pain in 49%, and fever in 37%.

. A prodromal malaise of 2 days or more was experienced by at least 3/51 cases (6%).

Blood culture was positive in a girl aged 5 with symptoms of septicaemia and a girl aged 6 with febrile gastroenteritis. One boy, aged 7, had infective arthritis of the hip, presenting after an uncertain period of malaise.

Eleven other patients with acute gastroenteritis needed hospital admission. Also a child who had an exacerbation of a long standing diarrhoeal disorder was admitted for investigation. At least one other patient, a boy aged 14, had a 10% weight loss, recorded as 1 stone.

The source of infection

This was eventually identified as desiccated coconut. The batch responsible for the outbreak consisted of a single day's production and had been packed in Malaysia for distribution by a supermarket chain in Victoria. the discovery was made after recovering *S. stanley* from coconut in a canister in a household with cases. Further investigation of other affected families yielded two with contaminated coconut - in one case in the original packet. Finally, *S. stanley* was also isolated from unopened packets of the same batch retrieved from supermarkets. This salmonella was not isolated from batches of other production dates.

Many people must have had unrecognised infections. Therefore, it is possible that in hot weather many more patients would have been severely ill owing to dehydration.

Because cases seemed to occur sporadically and because many of those affected had been away on holiday, takeaway food products were initially suspected. However, this pattern actually reflected usage of a medium-life commodity from household shelves. Recognition and investigation of each case depended on culture and serotyping of the organism, by which time memory of food taken was fading. Even when the vehicle was known, 20% of families who were questioned again denied using desiccated coconut and others said it was only used in cake mixes which were cooked. Among these was the household from which the original packet yielded the organism.

While central Government has always to be wary about invading private households, systematic investigation and culture of pantry contents might have identified the vehicle considerably earlier.

MALARIA SCREENING USING THE FLUORESCENT ANTIBODY TECHNIQUE

(Submitted by A. Findlay, Malaria Reference Laboratory, State Health Laboratory, Queensland)

The Queesland Malaria Reference Laboratory has provided a screening service for migrants arriving from South East Asia and South America for many years. No cases of malaria have ever been detected in this population after three separate blood film examinations on each migrant. During the 1986/87 period 1187 blood films were examined.

Malarial antibodies appear in the bloodstream 7-12 days after infection, (perhaps even before a parasitaemia develops), and

Malarial antibodies appear in the bloodstream 7-12 days after infection, (perhaps even before a parasitaemia develops), and will have disappeared approximately six months after effective treatment[1].

It became obvious to the Laboratory that when testing this population a serological method may provide a less labour-intensive and probably a more sensitive method (given the large number of negatives) of detecting possible malaria carriers.

The fluorescent technique was selected in preference to other antibody detection methods such as ELISA, because it is well known that the former is non-specific and at low titres will detect antibodies, not only to the *P.falciparum* substrate but also to *P.vivax* and *P.malariae*. Thus it provides a means of identifying people who may have malaria amongst a large population who are negative. It is remotely possible that a newly infected person may be missed because there has been insufficient time for antibodies to develop, but in this case the lack of immunity will result in the patient presenting with clinical symptoms within a very brief time period.

The method has now been used for seven months and 170 patients have been tested. Five positive reactions have been detected. All but one patient was recalled for subsequent blood film examination and no malarial parasites were found.

The test is thought to be specific for malaria at appropriate dilutions and our experience has confirmed this. Whilst an occasional sample in the migrant population has been inexplicably positive, large numbers of random laboratory patients (including approximately 20 who have had demonstrable anti-nuclear antibodies to HEp 2 cells) have shown no fluorescence when tested with *P.falciparum*.

The fluorescent technique has also been used to survey the incidence of malaria antibodies in the resident population of Boigu, one of the Torres Strait Islands near the Papua New Guinea Coast. Nine per cent of 173 residents tested were found to have detectable malaria antibodies.

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CORRIGENDUM: HIV TESTING QUALITY ASSURANCE PANEL

Data contained in Tables 1, 2, 3, 4, 5 and 6 of the article entitled 'Anti-HIV Testing Quality Assurance Programme Panel No. 2/1987 - NSW Red Cross Blood Transfusion Service' published in the previous issue of CDI (CDI 88/12) have been corrupted due to reformatting. The corrected data are shown in pages .. of this issue and readers are requested to substitute old tables (p15, 16, 18, 20, 22 and 24 of CDI 88/12) with the new corrected data.

Table 1: Results of samples in 'CH' dilution series

TEST-SYSTEMS		CASE 1				
		DILUTION SERIES 'CH' SAMPLES # A/HIV DILUTION $10^{-2.0}$		DILUTION $10^{-1.5}$		BLANK # a/HIV
		287/1	287/3	287/6	287/11	287/12
ABBOTT-(rDNA)	SAMPLE	+	+	+	+	-
	No. Pos/n	45/45	45/45	45/45	45/45	34/40
Cut-off mean abs ± SD	Mean absorbance	1.937	1.927	1.933	1.957	0.059
= 0.198 ± 0.053	± SD	± 0.175	± 0.175	± 0.233	± 0.168	± 0.093
BEHRING (Competitive Binding Assay)	SAMPLE	+	+	+	+	-
	No. Pos/n	4/4	4/4	4/4	4/4	4/4
Cut-off mean abs ± SD	Mean absorbance	0.121	0.089	0.104	0.104	1.185
= 0.594 ± 0.239	± SD	± 0.081	± 0.034	± 0.016	± 0.123	± 0.345
DU PONT	SAMPLE	+	+	+	+	-
	No. Pos/n	3/3	3/3	3/3	3/3	3/3
Cut-off mean abs ± SD	Mean absorbance	1.266	1.139	1.756	1.660	0.051
= 0.594 ± 0.084	± SD	± 0.522	± 0.291	± 0.256	± 0.281	± 0.033
GENETIC SYSTEMS	SAMPLE	+	+	+	+	-
	No. Pos/n	15/15	15/15	15/15	15/15	11/11
Cut-off mean abs ± SD	Mean absorbance	0.927	0.808	1.318	1.263	0.022
= 0.264 ± 0.012	± SD	± 0.177	± 0.236	± 0.277	± 0.389	± 0.013
ORGANON-TEKNIKA	SAMPLE	+	+	+	+	-
	No. Pos/n	11/11	11/11	11/11	11/11	8/8
Cut-off mean abs ± SD	Mean absorbance	1.0975	1.117	1.315	1.414	0.084
= 0.309 ± 0.092	± SD	± 0.466	± 0.441	± 0.373	± 0.339	± 0.036
ORGANON-BIONETICS	SAMPLE	+	+	+	+	-
	No. Pos/n	2/2	2/2	2/2	2/2	2/2
Cut-off mean abs ± SD	Mean absorbance	0.485	0.450	0.482	0.470	0.068
= 0.213 ± 0.035	± SD	± 0.054	± 0.045	± 0.004	± 0.006	± 0.000
WELLCOME (Competitive Binding Assay)	SAMPLE	+	+	+	+	-
	No. Pos/n	26/27	27/27	25/26	26/27	23/23
Cut-off mean abs ± SD	Mean absorbance	0.160	0.152	0.120	0.129	0.945
= 0.599 ± 0.265	± SD	± 0.080	± 0.064	± 0.091	± 0.109	± 0.281
VIRGO-ENI	SAMPLE	+	+	+	+	-
	No. Pos/n	1	1	1	1	1
Cut-off absorbance = 0.117	Mean absorbance	0.796	0.688	1.157	1.183	0.011

Table 2: Results of samples in 'BM' dilution series

TEST-SYSTEMS		CASE 2				
		DILUTION SERIES 'BM' SAMPLES # A/HIV DILUTION $10^{-3.5}$		DILUTION $10^{-3.0}$		BLANK # a/HIV
		287/2	287/13	287/8	287/10	287/12
ABBOTT-(rDNA)	SAMPLE	-	-	+	+	-
	No. Pos/n	30/39	36/39	45/45	45/45	34/40
Cut-off mean abs ± SD	Mean absorbance	0.161	0.148	0.397	0.392	0.059
= 0.198 ± 0.053	± SD	± 0.061	± 0.036	± 0.097	± 0.104	± 0.093
BEHRING	SAMPLE	-	-	(*)	(*)	-
(Competitive Binding Assay)	No. Pos/n	4/4	3/4	3/4	2/4	4/4
Cut-off mean abs ± SD	Mean absorbance	0.759	0.751	0.529	0.651	1.185
= 0.594 ± 0.239	± SD	± 0.175	± 0.217	± 0.118	± 0.203	± 0.345
DU PONT	SAMPLE	-	-	-	-	-
	No. Pos/n	3/3	3/3	3/3	3/3	3/3
Cut-off mean abs ± SD	Mean absorbance	0.051	0.087	0.185	0.146	0.051
= 0.594 ± 0.084	± SD	± 0.034	± 0.036	± 0.051	± 0.073	± 0.033
GENETIC SYSTEMS	SAMPLE	-	-	-	-	-
	No. Pos/n	11/11	11/11	12/12	12/12	11/11
Cut-off mean abs ± SD	Mean absorbance	0.053	0.040	0.089	0.091	0.022
= 0.264 ± 0.012	± SD	± 0.034	± 0.013	± 0.022	± 0.028	± 0.013
ORGANON-TEKNIKA	SAMPLE	-	-	-	-	-
	No. Pos/n	9/9	9/9	9/10	10/10	8/8
Cut-off mean abs ± SD	Mean absorbance	0.120	0.120	0.209	0.192	0.084
= 0.309 ± 0.092	± SD	± 0.053	± 0.050	± 0.087	± 0.078	± 0.036
ORGANON-BIONETICS	SAMPLE	-	-	+	+	-
	No. Pos/n	2/2	2/2	2/2	2/2	2/2
Cut-off mean abs ± SD	Mean absorbance	0.135	0.138	0.257	0.220	0.068
= 0.213 ± 0.035	± SD	± 0.009	± 0.000	± 0.060	± 0.010	± 0.000
WELLCOME (Competitive	SAMPLE	-	-	+	+	-
Binding Assay)	No. Pos/n	20/26	19/26	25/27	26/27	23/23
Cut-off mean abs ± SD	Mean absorbance	0.629	0.680	0.414	0.402	0.945
= 0.599 ± 0.265	± SD	± 0.240	± 0.235	± 0.153	± 0.145	± 0.281
VIRGO-ENI	SAMPLE	-	-	-	-	-
	No. Pos/n	1	1	1	1	1
Cut-off absorbance	Mean absorbance	0.017	0.007	0.051	0.042	0.011
= 0.117						

(*) Equivocal result

Table 3: Results of samples in 'AO' dilution series

TEST-SYSTEMS		CASE 4				
		DILUTION SERIES 'AO'				
		SAMPLES # A/HIV DILUTION $10^{-3.0}$		DILUTION $10^{-2.5}$		BLANK # a/HIV
		287/7	287/16	287/5	287/14	287/12
ABBOTT-(rDNA)	SAMPLE	+	+	+	+	-
	No. Pos/n	45/45	44/44	45/45	45/45	34/40
Cut-off mean abs ± SD	Mean absorbance	0.572	0.577	1.170	1.142	0.059
= 0.198 ± 0.053	± SD	± 0.123	± 0.126	± 0.272	± 0.283	± 0.093
BEHRING	SAMPLE	-	-	+	+	-
(Competitive Binding Assay)	No. Pos/n	4/4	4/4	4/4	4/4	4/4
Cut-off mean abs ± SD	Mean absorbance	0.770	0.826	0.549	0.420	1.185
= 0.594 ± 0.239	± SD	± 0.202	± 0.200	± 0.182	± 0.108	± 0.345
DU PONT	SAMPLE	-	-	-	-	-
	No. Pos/n	3/3	3/3	3/3	3/3	3/3
Cut-off mean abs ± SD	Mean absorbance	0.132	0.126	0.238	0.375	0.051
= 0.594 ± 0.084	± SD	± 0.040	± 0.042	± 0.134	± 0.108	± 0.033
GENETIC SYSTEMS	SAMPLE	-	-	-	-	-
	No. Pos/n	11/11	11/11	12/12	12/12	11/11
Cut-off mean abs ± SD	Mean absorbance	0.070	0.075	0.155	0.167	0.022
= 0.264 ± 0.012	± SD	± 0.015	± 0.024	± 0.032	± 0.041	± 0.013
ORGANON-TEKNIKA	SAMPLE	-	-	-	-	-
	No. Pos/n	8/10	9/9	7/10	7/11	8/8
Cut-off mean abs ± SD	Mean absorbance	0.188	0.150	0.333	0.299	0.084
= 0.309 ± 0.092	± SD	± 0.118	± 0.067	± 0.232	± 0.158	± 0.036
ORGANON-BIONETICS	SAMPLE	-	-	-	+	-
	No. Pos/n	2/2	2/2	2/2	2/2	2/2
Cut-off mean abs ± SD	Mean absorbance	0.136	0.127	0.314	0.251	0.068
= 0.213 ± 0.035	± SD	± 0.000	± 0.000	± 0.032	± 0.016	± 0.000
WELLCOME (Competitive	SAMPLE	-	-	+	+	-
Bind Assay)	No. Pos/n	20/26	18/24	23/27	24/27	23/23
Cut-off mean abs ± SD	Mean absorbance	0.651	0.695	0.454	0.448	0.945
= 0.599 ± 0.265	± SD	± 0.274	± 0.259	± 0.200	± 0.163	± 0.281
VIRGO-ENI	SAMPLE	-	-	-	-	-
	No. Pos/n	1	1	1	1	1
Cut-off absorbance	Mean absorbance	0.027	0.027	0.095	0.083	0.011
= 0.117						

Table 4: Results of samples in 'EC' dilution series

TEST-SYSTEMS		CASE 5		
		DILUTION SERIES 'EC'		
		SAMPLES # A/HIV		BLANK
		DILUTION $10^{-3.0}$	DILUTION $10^{-2.5}$	# a/HIV
		287/17	287/9	287/12
ABBOTT-(rDNA)	SAMPLE	+	+	-
	No. Pos/n	44/45	45/45	34/40
Cut-off mean abs ± SD	Mean absorbance	0.777	1.410	0.059
= 0.198 ± 0.053	± SD	± 0.209	± 0.372	± 0.093
BEHRING	SAMPLE	-	+	-
(Competitive Binding Assay)	No. Pos/n	3/4	4/4	4/4
Cut-off mean abs ± SD	Mean absorbance	0.657	0.360	1.185
= 0.594 ± 0.239	± SD	± 0.140	± 0.039	± 0.345
DU PONT	SAMPLE	-	-	-
	No. Pos/n	3/3	2/3	3/3
Cut-off mean abs ± SD	Mean absorbance	0.145	0.508	0.051
= 0.594 ± 0.084	± SD	± 0.100	± 0.208	± 0.033
GENETIC SYSTEMS	SAMPLE	-	+	-
	No. Pos/n	11/11	7/13	11/11
Cut-off mean abs ± SD	Mean absorbance	0.103	0.257	0.022
= 0.264 ± 0.012	± SD	± 0.035	± 0.072	± 0.013
ORGANON-TEKNIKA	SAMPLE	-	+	-
	No. Pos/n	9/10	8/11	8/8
Cut-off mean abs ± SD	Mean absorbance	0.193	0.396	0.084
= 0.309 ± 0.092	± SD	± 0.092	± 0.236	± 0.036
ORGANON-BIONETICS	SAMPLE	+	+	-
	No. Pos/n	2/2	2/2	2/2
Cut-off mean abs ± SD	Mean absorbance	0.225	0.352	0.068
= 0.213 ± 0.035	± SD	± 0.005	± 0.013	± 0.000
WELLCOME (Competitive	SAMPLE	-	+	-
Binding Assay)	No. Pos/n	19/25	26/27	23/23
Cut-off mean abs ± SD	Mean absorbance	0.651	0.411	0.945
= 0.599 ± 0.265	± SD	± 0.224	± 0.145	± 0.281
VIRGO-ENI	SAMPLE	-	+	-
	No. Pos/n	1	1	1
Cut-off absorbance	Mean absorbance	0.053	0.154	0.011
= 0.117				

Table 5: Results of samples in 'OR' dilution series

TEST-SYSTEMS		CASE 7		
		DILUTION SERIES 'OR'		
		SAMPLES # A/HIV DILUTION $10^{-2.0}$	SAMPLES # A/HIV DILUTION $10^{-1.5}$	BLANK # a/HIV
		287/19	287/18	287/12
ABBOTT-(rDNA)	SAMPLE	+	+	-
	No. Pos/n	44/44	45/45	34/40
Cut-off mean abs ± SD	Mean absorbance	0.772	1.354	0.059
= 0.198 ± 0.053	± SD	± 0.186	± 0.286	± 0.093
BEHRING (Competitive Binding Assay)	SAMPLE	-	+	-
	No. Pos/n	4/4	3/4	4/4
Cut-off mean abs ± SD	Mean absorbance	0.704	0.612	1.185
= 0.594 ± 0.239	± SD	± 0.200	± 0.242	± 0.345
DU PONT	SAMPLE	-	-	-
	No. Pos/n	3/3	2/3	3/3
Cut-off mean abs ± SD	Mean absorbance	0.241	0.361	0.051
= 0.594 ± 0.084	± SD	± 0.133	± 0.197	± 0.033
GENETIC SYSTEMS	SAMPLE	-	+	-
	No. Pos/n	11/11	7/13	11/11
Cut-off mean abs ± SD	Mean absorbance	0.108	0.271	0.022
= 0.264 ± 0.012	± SD	± 0.034	± 0.089	± 0.013
ORGANON-TEKNIKA	SAMPLE	-	-	-
	No. Pos/n	8/9	6/10	8/8
Cut-off mean abs ± SD	Mean absorbance	0.211	0.271	0.084
= 0.309 ± 0.092	± SD	± 0.206	± 0.084	± 0.036
ORGANON-BIONETICS	SAMPLE	-	+	-
	No. Pos/n	2/2	2/2	2/2
Cut-off mean abs ± SD	Mean absorbance	0.177	0.312	0.068
= 0.213 ± 0.035	± SD	± 0.007	± 0.013	± 0.000
WELLCOME (Competitive Binding Assay)	SAMPLE	-	+	-
	No. Pos/n	22/24	22/27	23/23
Cut-off mean abs ± SD	Mean absorbance	0.745	0.507	0.945
= 0.599 ± 0.265	± SD	± 0.274	± 0.145	± 0.180
VIRGO-ENI	SAMPLE	-	+	-
	No. Pos/n	1	1	1
Cut-off absorbance = 0.117	Mean absorbance	0.044	0.119	0.011

Table 6: Results of samples 'late', 'early' and 'false positive' HIV infection

TEST-SYSTEMS		CASE 3 'LATE' HIV INFECTION # a/HIV	CASE 6 'EARLY' HIV INFECTION # a/HIV	CASE 8 'FALSE-POSITIVE' # a/HIV	BLANK # a/HIV
		287/4	287/15	287/20	287/12
ABBOTT-(rDNA)	SAMPLE	+	+	-	-
	No. Pos/n	45/45	45/45	39/39	34/40
Cut-off mean abs + SD	Mean absorbance	1.849	1.409	0.090	0.059
= 0.198 ± 0.053	± SD	± 0.280	± 0.370	± 0.028	± 0.093
BEHRING (Competitive Binding Assay)	SAMPLE	+	+	-	-
	No. Pos/n	4/4	4/4	4/4	4/4
Cut-off mean abs + SD	Mean absorbance	0.101	0.427	1.075	1.135
= 0.594 ± 0.239	± SD	± 0.055	± 0.230	± 0.325	± 0.345
DU PONT	SAMPLE	+	+	+	-
	No. Pos/n	3/3	3/3	3/3	3/3
Cut-off mean abs + SD	Mean absorbance	1.533	1.673	1.556	0.051
= 0.594 ± 0.084	± SD	± 0.180	± 0.250	± 0.627	± 0.033
GENETIC SYSTEMS	SAMPLE	+	+	-	-
	No. Pos/n	15/15	15/15	9/14	11/11
Cut-off mean abs + SD	Mean absorbance	1.150	0.931	0.317	0.022
= 0.264 ± 0.012	± SD	± 0.320	± 0.206	± 0.112	± 0.013
ORGANON-TEKNIKA	SAMPLE	+	+	-	-
	No. Pos/n	11/11	8/11	9/9	8/8
Cut-off mean abs + SD	Mean absorbance	1.361	0.449	0.101	0.084
= 0.309 ± 0.092	± SD	± 0.357	± 0.277	± 0.023	± 0.036
ORGANON-BIONETICS	SAMPLE	+	+	-	-
	No. Pos/n	2/2	2/2	2/2	2/2
Cut-off mean abs + SD	Mean absorbance	0.511	0.345	0.092	0.068
= 0.213 ± 0.035	± SD	± 0.033	± 0.015	± 0.000	± 0.000
WELLCOME (Competitive Binding Assay)	SAMPLE	+	+	+	-
	No. Pos/n	26/27	27/27	23/23	23/23
Cut-off mean abs + SD	Mean absorbance	0.135	0.354	0.898	0.945
= 0.599 ± 0.265	± SD	± 0.080	± 0.127	± 0.301	± 0.281
VIRGO-ENI	SAMPLE	+	+	+	-
	No. Pos/n	1	1	1	1
Cut-off absorbance	Mean absorbance	1.138	0.222	0.143	0.011
= 0.117					

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

TOTAL VIRAL ISOLATIONS BASED ON DATE OF REPORTING
PERIOD - FORTNIGHTLY
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

Period 15-6-88 to 28-6-88.

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA)
3. CODE 110 - IMVS(SA)
4. CODE 111 - RCH(VIC)
5. CODE 112 - ICPMR(NSW) WVH(ACT)
6. CODE 113 - PHH POW(NSW)
7. CODE 114 - RAHC(NSW)
8. CODE 115 - STATE LAB(QLD)

		65	019	065	110	111	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	0	1	2	2	0	7	12	2	6	32
0101	ADENOVIRUS TYPE 1	0	0	3	0	0	3	0	0	0	6
0102	ADENOVIRUS TYPE 2	0	2	1	1	4	2	0	0	0	10
0103	ADENOVIRUS TYPE 3	0	0	0	0	1	8	0	0	0	9
0104	ADENOVIRUS TYPE 4	0	4	0	0	0	1	0	0	0	5
0105	ADENOVIRUS TYPE 5	0	1	0	1	1	0	1	0	0	4
0106	ADENOVIRUS TYPE 6	0	0	1	0	0	1	0	0	0	2
0107	ADENOVIRUS TYPE 7	0	0	0	0	0	2	0	0	0	2
0108	ADENOVIRUS TYPE 8	0	1	1	0	0	4	1	0	0	7
0109	ADENOVIRUS TYPE 9	0	0	1	0	0	3	0	0	0	4
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	1	0	0	0	1
0127	ADENOVIRUS TYPE 27	0	0	0	0	0	1	0	0	0	1
0130	ADENOVIRUS TYPE 30	0	1	0	0	0	0	0	0	0	1
0135	ADENOVIRUS TYPE 35	0	2	0	0	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	2	0	6	1	0	9
0201	INFLUENZA A VIRUS	0	0	7	1	0	3	3	0	0	14
0203	INFLUENZA B VIRUS	0	0	0	1	0	0	1	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	0	4	6	2	16	5	0	2	1	36
0302	PARAINFLUENZA VIRUS TYPE 2	0	5	0	1	15	1	0	0	0	22
0303	PARAINFLUENZA VIRUS TYPE 3	0	2	0	3	2	1	0	0	0	8
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	15	8	33	42	55	21	38	50	262
0500	RHINOVIRUS (ALL TYPES)	0	3	1	2	1	5	2	0	10	24
0600	MYCOPLASMA PNEUMONIAE	0	8	0	5	6	35	3	0	0	57
0700	ORNITHOSIS-PSITTACOSIS	0	1	0	0	0	4	0	0	0	5
0809	COXSACKIEVIRUS A9	0	3	0	0	2	14	1	0	0	20
0816	COXSACKIEVIRUS A16	0	1	0	0	0	0	0	0	0	1
0901	COXSACKIEVIRUS B1	0	0	0	0	1	1	0	0	0	2
0902	COXSACKIEVIRUS B2	0	0	0	0	0	6	0	0	0	6
0905	COXSACKIEVIRUS B5	0	2	0	0	1	10	2	0	0	15
1001	ECHOVIRUS TYPE 1	0	0	0	0	0	3	0	0	0	3
1004	ECHOVIRUS TYPE 4	0	6	0	0	0	1	0	0	0	7
1006	ECHOVIRUS TYPE 6	0	0	0	0	0	1	0	0	0	1
1007	ECHOVIRUS TYPE 7	0	0	0	0	0	1	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	0	0	0	0	1	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	0	0	0	2	0	0	0	0	2
1016	ECHOVIRUS TYPE 16	0	0	0	0	0	1	0	0	0	1
1017	ECHOVIRUS TYPE 17	0	0	0	0	1	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	1	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	1	0	0	0	0	1	0	2
1025	ECHOVIRUS TYPE 25	0	2	0	0	1	0	0	0	0	3
1030	ECHOVIRUS TYPE 30	0	1	0	0	6	0	0	0	0	7
1100	POLIOVIRUS NOT TYPED	0	0	0	0	4	0	4	0	0	8
1101	POLIOVIRUS TYPE 1	0	1	0	0	0	1	0	0	0	2
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	2	0	0	0	2
1103	POLIOVIRUS TYPE 3	0	0	0	0	0	2	0	0	0	2
1200	MUMPS VIRUS	0	0	0	0	0	2	1	0	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	55	4	0	1	61
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	3	7	1	0	0	0	1	0	12
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	2	5	0	1	9	6	0	0	23
1303	VARICELLA-ZOSTER VIRUS	0	3	4	0	1	9	4	0	1	22
1306	HERPES SIMPLEX TYPE 1	0	41	25	20	0	42	14	0	37	179
1307	HERPES SIMPLEX TYPE 2	1	70	46	22	0	147	27	0	62	375
1399	HERPES VIRUS TYPING PENDING	0	6	0	1	5	0	0	0	0	12
1401	COXIELLA BURNETI	0	0	0	1	0	18	0	0	0	19
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	8	4	0	10	22
1521	MEASLES VIRUS	0	1	0	0	2	1	3	0	0	7
1522	RUBELLA VIRUS	0	2	0	1	0	7	1	0	0	11
1532	HEPATITIS B ANTIGEN	0	12	3	10	2	51	11	1	9	99
1535	HEPATITIS A ANTIBODY	0	2	5	4	1	9	1	0	3	25
1541	CHLAMYDIA A - C. TRACHOMATIS	0	0	49	19	0	36	5	0	13	122
1556	CMV - CYTOMEGALOVIRUS	0	23	5	5	6	28	9	0	12	88
1563	CORONAVIRUS	0	0	0	0	0	1	0	0	0	1
1564	ROTAVIRUS	0	0	6	33	0	7	3	2	0	51
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	6	0	27	4	0	37
9992	ROSS RIVER VIRUS	0	7	1	0	0	11	6	0	0	25
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	0	2	0	2
TOTAL		1	239	188	169	132	628	183	54	215	1809

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

Period 15-6-88 to 28-6-88.

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	8	0	0	0	17	0	0	0	0	26
0101	ADENOVIRUS TYPE 1	1	2	0	0	0	3	0	0	0	0	6
0102	ADENOVIRUS TYPE 2	0	4	0	0	0	5	0	0	0	0	9
0103	ADENOVIRUS TYPE 3	1	4	0	0	0	1	0	0	0	0	6
0105	ADENOVIRUS TYPE 5	0	2	0	0	0	1	0	0	0	0	3
0106	ADENOVIRUS TYPE 6	0	1	0	0	0	1	0	0	0	0	2
0107	ADENOVIRUS TYPE 7	0	1	0	0	0	0	0	0	0	0	1
0109	ADENOVIRUS TYPE 9	0	1	0	0	0	2	0	0	0	0	3
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	1	0	0	0	0	1
0127	ADENOVIRUS TYPE 27	0	0	0	0	0	1	0	0	0	0	1
0130	ADENOVIRUS TYPE 30	0	0	0	0	0	0	0	0	0	1	1
0199	ADENOVIRUS TYPING PENDING	0	3	0	0	0	0	0	0	0	0	3
0201	INFLUENZA A VIRUS	1	7	0	0	1	0	0	0	0	0	9
0203	INFLUENZA B VIRUS	0	2	0	0	0	0	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	1	35	0	0	0	0	0	0	0	0	36
0302	PARAINFLUENZA VIRUS TYPE 2	0	22	0	0	0	0	0	0	0	0	22
0303	PARAINFLUENZA VIRUS TYPE 3	1	5	0	0	0	0	0	0	0	0	6
0400	RESPIRATORY SYNCYTIAL VIRUS (R	5	252	1	0	1	0	0	0	0	1	260
0500	RHINOVIRUS (ALL TYPES)	0	22	0	1	0	0	0	0	0	0	23
0600	MYCOPLASMA PNEUMONIAE	8	38	1	0	1	0	0	0	0	1	49
0700	ORNITHOSIS-PSITTACOSIS	1	3	1	0	0	0	0	0	0	0	5
0809	COXSACKIEVIRUS A9	3	1	0	10	0	0	0	0	0	1	15
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	0	0	1	1
0901	COXSACKIEVIRUS B1	0	1	0	0	0	1	0	0	0	0	2
0902	COXSACKIEVIRUS B2	0	0	0	0	0	3	0	0	0	0	3
0905	COXSACKIEVIRUS B5	1	1	0	4	0	3	0	0	0	0	9
1001	ECHOVIRUS TYPE 1	0	0	0	1	0	1	0	0	0	0	2
1004	ECHOVIRUS TYPE 4	0	0	0	7	0	0	0	0	0	0	7
1006	ECHOVIRUS TYPE 6	1	0	0	0	0	0	0	0	0	0	1
1007	ECHOVIRUS TYPE 7	1	0	0	0	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	0	0	1	0	0	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	1	0	1	0	0	0	0	0	0	2
1016	ECHOVIRUS TYPE 16	0	0	0	0	0	1	0	0	0	0	1
1017	ECHOVIRUS TYPE 17	0	1	0	0	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	1	0	0	0	0	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	1	0	0	0	1	0	0	0	0	2
1025	ECHOVIRUS TYPE 25	0	1	0	2	0	0	0	0	0	0	3
1030	ECHOVIRUS TYPE 30	0	0	0	7	0	0	0	0	0	0	7
1100	POLIOVIRUS NOT TYPED	0	2	0	0	0	4	0	0	0	0	6
1101	POLIOVIRUS TYPE 1	1	0	0	0	0	1	0	0	0	0	2
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	2	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	0	0	0	1	0	1	0	0	0	0	2
1200	MUMPS VIRUS	0	1	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	15	2	0	0	4	0	0	0	0	27	48
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	1	0	0	0	0	0	0	1	5	7
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	11	0	0	0	0	0	0	0	0	0	11
1303	VARICELLA-ZOSTER VIRUS	1	1	0	0	0	0	0	0	0	13	15
1306	HERPES SIMPLEX TYPE 1	9	13	0	0	0	0	0	0	1	78	101
1307	HERPES SIMPLEX TYPE 2	19	0	0	0	0	0	0	0	0	49	68
1399	HERPES VIRUS TYPING PENDING	1	3	1	0	0	1	0	0	0	3	9
1401	COXIELLA BURNETI	9	0	0	0	0	0	0	0	0	0	9
1502	PICORNIA VIRUS - NOT TYPED = E	0	8	0	3	2	4	0	0	0	1	18
1521	MEASLES VIRUS	1	0	0	0	1	0	0	0	0	3	5
1522	RUBELLA VIRUS	1	2	0	0	0	0	0	0	0	3	6
1532	HEPATITIS B ANTIGEN	52	0	0	0	0	0	40	0	0	0	92
1535	HEPATITIS A ANTIBODY	3	0	0	0	0	0	19	0	0	0	22
1541	CHLAMYDIA A - C. TRACHOMATIS	17	0	0	0	0	0	0	0	0	0	17
1556	CMV - CYTOMEGALOVIRUS	16	21	0	0	1	2	2	1	4	0	47
1563	CORONAVIRUS	0	0	0	0	0	1	0	0	0	0	1
1564	ROTAVIRUS	0	0	0	0	0	51	0	0	0	0	51
1599	ENTEROVIRUS TYPING PENDING	0	7	0	4	0	22	0	0	0	1	34
9992	ROSS RIVER VIRUS	6	0	0	0	0	0	0	0	0	2	8
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	2	0	0	0	0	2
TOTAL		189	480	4	42	11	133	61	1	6	190	1117

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

Period 15-6-88 to 28-6-88.

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	2	0	0	0	1	0	1	1	1	0	6
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	0	0	0	1	1
0103	ADENOVIRUS TYPE 3	3	0	0	0	0	0	0	0	0	0	3
0104	ADENOVIRUS TYPE 4	5	0	0	0	0	0	0	0	0	0	5
0105	ADENOVIRUS TYPE 5	0	0	0	0	0	0	0	1	0	0	[illegible]
0107	ADENOVIRUS TYPE 7	1	0	0	0	0	0	0	0	0	0	[illegible]
0108	ADENOVIRUS TYPE 8	7	0	0	0	0	0	0	0	0	0	7
0109	ADENOVIRUS TYPE 9	1	0	0	0	0	0	0	0	0	0	1
0135	ADENOVIRUS TYPE 35	2	0	0	0	0	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	5	0	0	0	0	0	0	1	0	0	6
0201	INFLUENZA A VIRUS	0	0	0	0	1	0	3	1	0	0	5
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	1	0	0	0	0	1	0	0	2
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	1	1	0	0	2
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	0	0	1
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	2	5	1	0	8
0809	COXSACKIEVIRUS A9	0	0	0	0	0	0	2	1	2	0	5
0902	COXSACKIEVIRUS B2	0	0	0	0	0	0	1	0	2	0	3
0905	COXSACKIEVIRUS B5	0	0	0	0	0	0	3	2	0	1	6
1001	ECHOVIRUS TYPE 1	0	0	0	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	0	0	0	2	2
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	1	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	2	9	0	0	0	0	0	0	2	0	13
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	5	0	0	0	0	0	0	0	0	5
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	1	3	0	0	0	1	5	2	0	12
1303	VARICELLA-ZOSTER VIRUS	1	1	0	0	1	0	0	0	4	0	7
1306	HERPES SIMPLEX TYPE 1	4	71	0	0	0	0	0	1	2	0	78
1307	HERPES SIMPLEX TYPE 2	0	307	0	0	0	0	0	0	0	0	307
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	0	2	0	3
1401	COXIELLA BURNETI	0	0	0	0	0	0	6	2	2	0	10
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	1	0	0	0	0	1	2	0	[illegible]
1521	MEASLES VIRUS	0	0	0	0	0	0	0	1	1	0	[illegible]
1522	RUBELLA VIRUS	0	0	0	0	0	1	0	1	3	0	[illegible]
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	7	0	7
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	1	0	2	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	1	103	0	0	0	0	0	0	1	0	105
1556	CMV - CYTOMEGALOVIRUS	1	2	0	3	0	8	1	9	17	0	41
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	1	1	3
9992	ROSS RIVER VIRUS	0	0	0	0	14	0	0	2	1	0	17
TOTAL		35	500	6	3	17	9	22	39	56	5	692