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VIRUS REPORTING SCHEME: A total of 1 402 reports were processed for this period.

Nineteen cases of Q fever were reported, 1 from South Australia, 4 from Queensland and 14 from New South Wales. Occupational exposure data were only available for 3 of the Queensland cases:-

- a 43 year old male farmer from Chinchilla;
- a 34 year old male shearer from Roma; and
- a 33 year old male meatworker from Maryborough.

None of these nineteen patients was involved in the Q fever vaccine field trial conducted in South Australia.

Echovirus type 5 was isolated from the faeces of a 28 year old female who presented at 34 weeks gestation with pyrexia, skin rash and arthralgia. She subsequently delivered twins, one of whom developed meningitis at the age of 7 days; and echovirus type 5 was isolated from its cerebrospinal fluid. Central nervous system infections associated with echoviruses, in children under 1 year of age, may lead to neurologic sequelae and mental impairment. This does not appear to happen in older children.

Echovirus type 5 and type 11 have also been reported by Fairfield Hospital (Victoria) to be the predominant viruses causing aseptic meningitis in the summer months of this year.

Poliovirus type 3 was isolated from the faeces of a 4 month old male who underwent medical investigation for microcephaly.

Herpes Simplex virus type 1 was isolated from the bronchial washings of a 73 year old male suffering from a severe lower respiratory tract infection. Chest radiography indicated progressive pulmonary fibrosis.

Adenovirus type 1 was isolated from the urine of a 33 year old male who developed a urinary tract infection following renal transplantation.

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ACUTE SCHISTOSOMIASIS AMONG AMERICANS RAFTING THE OMO RIVER - ETHIOPIA
(based on JAMA Vol. 251/No. 4, 27 January 1987)

Acute schistosomiasis, sometimes called "Katayama syndrome", is a clinical syndrome that occurs within a few months after primary infection with Schistosoma mansoni and Schistosoma japonicum. The maturation of worms in the body may precipitate an illness, similiar to serum sickness, characterised by fever, eosinophilia, malaise, anorexia, weigth loss, fatigue, cough, lymphadenopathy, and hepatosplenomegaly. Acute schistosomiasis is distinct from the chronic schistosomiasis, which is more frequently seen in long-term residents of endemic areas, and the diagnosis can be difficult to establish, especially early in the course of the disease.

A small outbreak of schistosomiasis among a group of Americans and Swedish adventurers was recently investigated. In the autumn of 1981, a man (index case) had organised a private rafting expedition down the Omo River in south western Ethiopia with ten other persons. The eleven members gathered in Addis Ababa on 2 and 3 November, travelled to the Omo River on 6 November, and began the raft trip on 7 November. The group left the Omo River on 26 November at its confluence with the Mui River about 560 km downstream, flew to Addis Ababa on 28 November, and from there to their respective homes.

CASE REPORT

On 24 October 1981, a 41 year old man visited his physician in preparation for a rafting trip on the Omo River. He was immunised with tetanus toxoid and received a prescription of chloroquine phosphate for malaria prophylaxis. A complete blood cell count revealed:

. a haemoglobin level of 16.7 g/dL
. a haemotocrit value of 53.4%
. a WBC count of 7 600 /cu mm with
  - 61% segmented neutrophils,
  - 31% lymphocytes,
  - 5% monocytes,
  - 1% eosinophils, and
  - 2% basophils

The patient participated in the river expedition for four weeks in November, returning to his home in Colorado, in early December. Beginning on 5 December, he noted gradual onset of headache, chills, sweats, generalised malaise, and weakness and consulted the hospital outpatient clinic on 13 December. Physical examination was unrevealing:

. a haemoglobin level of 14.8 g/dL
. a haematocrit value of 44%
. a WBC count of 7 200 /cu mm with

  - 46% segmented neutrophils (6% band forms)
  - 15% lymphocytes(5% atypical lymphocytes)
  - 2% monocytes
  - 22% eosinophils (absolute oesinophil count, 1580/cu mm)
  - 4% basophils

Platelets were adequate in number, total bilirubin level was 0.8 mg/dL and SGPT level was 51 units. A malaria smear was negative. The patient's symptoms improved spontaneously, with a return of his strength and vigor, disappearance of fever, and return of appetite.

On 29 December, he returned to outpatient complaining of a recurrence of fever, chills, malaise, anorexia and extreme fatigue. Physical examination again disclosed no abnormalities, however his WBC count was 11 600/ cu mm, with 36% oesinophils (absolute oesinophil count 4 180/ cu mm). Malaria smears were negative. The patient continued to have intermittent episodes of malaise, prostration, chills and fever, night sweats and weight loss.

On 13 January, the patient was re-examined and physical examination was again unrevealing, however his WBC count was 29 500/ cu mm with 57% eosinophils (absolute oesinophil count 16 815/ cu mm). A battery of blood chemistry tests and liver function tests and a urinalysis showed normal results. A thick film for malaria was negative. No ova or parasites were detected by formalin - ether concentration of stool. A medical specialist consultant suggested filarial diseases, schistosomiasis, visceral larva migrans, and trichinosis as diagnostic possibilities.

Stool specimens examined by formalin - ether concentration and by the modified Ritchie concentration technique (MRCT) at the Centers for Disease Control (CDC) in Atlanta were found to contain ova of Schistosoma mansoni (ten eggs per gram of faeces). Urine sediment had no schistosome eggs and thick films had no malaria parasites or microfilariae.

The patient was treated with a single dose of oxamniquine (30mg/kg). Examination by the MRCT of 3g of stool taken six weeks after treatment revealed no schistosome ova. The patient has resumed his work and he has had no recurrence of his symptoms.

EPIDEMIOLOGIC INVESTIGATION

All 11 rafting trip participants were informed of their exposure and requested to reply to questionnaires concerning basic personal data, previous travel, specific exposures such as swimming or bathing in various pools along the Omo River, use of oils or lotions (ie sunscreens), towel drying habits, and symptoms.

A. CLINICAL DIAGNOSIS

Five of the 11 expeditioners experienced mild clinical symptoms, and signs compatible with acute schistosomiasis:

- median onset occurred approximately three weeks after the group left the Omo River;
- eosinophilia was the most reliable sign; the median peak oesinophil count was 2 280/ cu mm (range, 1152/ cu mm to 16 815/ cu mm).

All five persons with oesinophilia and at least one other symptom had S. mansoni eggs in their stool and positive finding for schistosomiasis by indirect immunofluorescence (IIF), while

only one of six asymptomatic persons had eggs and positive findings for schistosomiasis by IIF (P = .01 by Fishers exact test).

Every person without S. mansoni in their stool had negative findings by IIF. A few serum specimens produced weak reactions to trichinosis and filariasis, and stools from two person also contained cyst of Entamoeba histolytica, but they were not associated with illness. Egg counts from four infected asymptomatic persons ranged from three to 15 S. mansoni eggs per gram of stool. Similarly, only a few eggs were seen in non quantitative formalin - ether concentrations of stool from the other two infected expeditioners.

All five expeditioners with negative test results in January remained asymptomatic. Three of these had negative findings by MRCT (3g of stool) and negative findings for schistosomiasis by IIF again in March.

B. QUESTIONNAIRE INFORMATION

Questionnaire results for ten respondents revealed that only three pools were used for swimming by all six infected expeditioners (on Nov. 13, 18 and 19). These three pools were also used by three of the four responding uninfected expeditioners. In addition, infected persons were less likely to have towel dried on a regular basis after becoming wet during the last portion of the trip (one of six infected vs three of four noninfected, P =.12).

All six person with S. mansoni infection were treated with oxamniquine, 30 mg/kg, in a single oral dose. Side effects were frequent but mild and transient. Among the six, five reported some distortion of reality and two reported frank hallucinations. MRCT examination of 3g of stool taken from five infected adventurers approximately one month after treatment showed no S. mansoni ova. Three nonquantitative formalin - ether, concentrations of stool and a rectal biopsy specimen from the remaining infected adventurer also showed no schistosome ova.

COMMENT

Although the outbreak was small, the high attack rate of illness (45%) and infection (55%) among these rafters emphasises the potential magnitude of the problem for travellers to the area. The Mui River served as a source for two reported outbreaks of acute schistosomiasis among tourists in the Omo National Park (a game reserve) in Ethiopia. Most of these tourists swam or bathed in clear, cool pools they presumed were safe. Similarly, the rafters often bathed in inviting pools in side streams. Trips to remote territory in Ethiopia have increased in popularity during the past several years, and at least one commercial organisation now arranges rafting trips. In fact, the CDC has received anecdotal reports of persons infected on other trips. Physicians probably will continue to see persons with acute schistosomiasis from these trips as well as other exposures.

The low egg counts for ill persons in this outbreak are not unusual for acute S. mansoni infections. The low egg counts observed in this outbreak would not reflect early detection of

illness; egg counts in humans exposed only once show no tendency to increase from six weeks to 18 months after exposure. More likely, they represented a light worm burden.

While the day of exposure or the onset of egg excretion could not be precisely determined, several ill adventurers probably had onset of symptoms in advance of onset of egg excretion. Early symptoms are associated in time with the migration and maturation of the schistosome. After penetrating the skin, schistosomula (juvenile worms) of S. mansoni migrate through the lymphatics and veins to the lungs. On the 8th day after infection, they begin to appear in the portal vasculature of the liver, where they grow and mature sexually. Beginning at four weeks after infection, they migrate again to the mesenteric veins, where oviposition begins. Stool specimens usually became positive for eggs from 40 to 50 days (range, 34 to 97 days) after exposure, while incubation periods for symptoms were shorter (nine to 87 days). Thus, in acute schistosomiasis, symptoms can appear before eggs can be detected by stool examination.

Light infections and the delayed onset of egg excretion can hinder parasitological diagnosis. Four symptomatic persons had consulted their physicians before confirmation of their S. mansoni infections. In two instances, stools were examined during the prepatent period and were negative for ova. In the index case, a light infection was not detected by routine laboratory methods. The MRCT, used successfully in this outbreak to detect light infections, is a quantitative formalin - ether method. Because of the delay between symptoms and onset of egg excretion, even a sensitive method like the MRCT technique may need to be repeated for up to 13 weeks after exposure.

In this outbreak, treatment with a single 30 mg/kg dose of oxamniquine produced a symptomatic and parasitic cure of all infected persons. The observed side effects were anticipated and the patients forewarned. With such prejudice the high rates of subjectively defined psychogenic side effects cannot be interpreted as contradicting previous experience with oxamniquine nor can there rates, based on a small group, be determined to be substantially different from previous experience. An equally effective drug, praziquantel, has recently been licensed in the United States, and is effective against all important species of Schistosoma in humans. Given the relative safety of these drugs, it seems reasonable that all parasitologically confirmed schistosome infections be treated.

Several preventive measures could be recommended for future travellers. However, it should be emphasised that the river rafters were aware that the Mui River at the end point of the trip was a source of schistosomiasis. Consequently, they iodinated all drinking and bath water, but only during the last three to four days of the trip. Preventive measures are aimed at avoiding contact with schistosome cercariae either through avoiding presumably infected water or by killing the cercariae with iodination (1ppm for 30 minutes) and chlorination (1ppm for 30 minutes) or heat the water (to 50°C or more for five minutes) before using it. Experimental evidence suggests that frequent (approximately every ten minutes), brisk towel drying

could remove and kill the cercariae before they can penetrate the skin. This measure is easily practised even after accidental dunkings and could theoretically prevent infection in some people and reduce the numbers of penetrating cercariae and consequent severity of disease in others. Regardless of precautions, a history of travel to Africa or to other endemic areas in a patient with a febrile illness accompanied by eosinophilia should alert the physician to the possibility of acute schistosomiasis.

A POINT-SOURCE EPIDEMIC OF LEPTOSPIROSIS
(Based on Postgraduate Medicine Vol. 80/No 8, 1 December 1986)

Leptospirosis is a zoonotic disease that has traditionally been considered an occupational hazard of sewer and abattoir workers, farmers, and others exposed to animals, particularly rats. Animal reservoirs include rats, other rodents, dogs, cats, cattle, hogs, and horses. Asymptomatic infection appears to be common, particularly in rats, and organisms can be excreted in the urine of infected animals (including asymptomatic immunised pets) for many months.

The organism survives in a warm, moist environment and enters the human body through abraded or diseased skin, the respiratory tract, or mucous membranes. Direct person-to-person transmission rarely, if ever, occurs. The notifiable incidence of leptospirosis is high in Australia (1.33 cases/100 000 population/year) compared with the United States (0.05 cases/100 000/person/years), but the actual incidence is probably much higher because of failure to diagnose the disease and to report it.

In recent years many cases have resulted from outdoor recreational exposure to contaminated water or from contact with infected pets, which may or may not be symptomatic. Thus, the following point-source epidemic reflects the changing epidemiologic pattern of the disease.

Four of ten adults who had participated in a kayaking trip on a creek near Columbia, Missouri, experienced febrile illness within 17 days of the trip. In cases 1 and 2 the diagnosis of leptospirosis was confirmed by seroconversion.

. Case 1 - in early December 1985 a previously healthy 32 year old man presented with a febrile illness. Fever had developed abruptly four days previously; an elevated temperature had persisted at a level of 39°C to 40°C since then. Fever was accompanied by intermittently shaking chills, night sweats, severe headache and malaise, photophobia, myalgias and anorexia. Skin rash and respiratory, urinary, and gastrointestinal symptoms were absent. Ten days before the onset of illness he had been kayaking on a local creek that was swollen by recent rains and water run off from adjacent land, including a livestock farm. While kayaking he had capsized and ingested several mouthfuls of water.

On examination he appeared ill and had:

- oral temperature of 39.5°C
- pulse rate of 75 beats/min, and
- blood pressure of 118/68 mm Hg

No rash, lymphadenopathy, hepatic or splenic enlargement, conjunctivitis, or signs of localised infection were present. Laboratory findings were as follows:

- WBCs 5 400/cu mm with
  51% segmented neutrophils,
  33% bands,
  1% metamyelocytes,
  10% lymphocytes, and
  5% monocytes
- Haematocrit 35%

- Serum chemistry:
  glucose 128 mg/dL
  sodium 131 mEq/L
  potassium 3.5 mEq/L
  chloride 93 mEq/L
  carbon dioxide 30 mEq/L

- serum biochemistry:
  creatinine 1.4 mEq/L
  BUN 9 mg/dL
  SGOT 124 units/L
  SGPT 99 units/L
  total bilirubin 1.7 mg/dL

- serum enzyme activities (creatine kinase, lactate dehydrogenase, and alkaline phosphatase) were at normal levels.

Findings of urinalysis and chest X-ray were normal. Lumbar puncture yielded clear, colourless fluid with

- 4 mononuclear cells and 6 red cells/cu mm
- protein content of 44 mg/dL, and
- glucose content of 77 mg/dL.

The patient was admitted to hospital where intravenous chloramphenicol was initiated after blood, urine and CSF was obtained for culture. During the first three days he had intermittent fever and night sweats. His symptoms gradually resolved, transaminase and bilirubin levels normalised, and he was discharged on the fifth day. All cultures and antibody titres for Leptospira, Brucella and Salmonella were negative.

Soon after discharge he regained his premorbid health. Three weeks after the onset of illness, the leptospiral macroagglutination test was positive. Specific microagglutination tests demonstrated positive titres to five serotypes. The highest titre was 1:3 200 for the djasiman and andaman A serotypes.

. Case 2 - Two days after patient 1 was admitted, a 43 year old man presented with a four-day history of high fever, chills, malaise, and severe headache and myalgias. Except for a temperature of 39°C and pulse rate of 82 beats/min, results of his examination were normal. The total WBC count was 8 700/cu mm^3 with a marked left shift. Urinalysis showed pyuria and proteinuria; transminase levels were elevated to three times normal.

The patient had kayaked on the local creek with patient 1 and had also capsized and ingested some water. He was treated with oral doxycycline (100 mg twice a day) for suspected leptospirosis and was followed as an outpatient. He recovered completely within a few days. A serologic test for leptospiral antibodies during the acute phase was negative, but a macroagglutination test on convalescent-phase serum was positive. A titre of 1:800 was found for the djasiman serotype.

In cases 3 and 4 the diagnosis was presumptive in the absence of bacteriologic or serologic documentation.

. Case 3 - One day after patient 2 was seen, a 25 year old woman presented with a two-day history of high fever, chills, headache, myalgias, malaise and abdominal pain. Except for a temperature of 39°C and a pulse rate of 88 beats/min, findings on physical examination were normal. Urinalysis showed pyuria, and her liver and renal function test results were normal. She was treated with oral doxycycline (100 mg twice a day) and recovered within three days. Acute and convalescent serologic tests for leptospiral antibodies were negative. She had been on the same kayaking trip as patients 1 and 2 and had accidentally ingested some creek water.

. Case 4 - a 23 year old woman, who had also been on the kayaking trip, presented one day after patient 3 with a one-day history of similar symptoms. Except for a temperature of 40° and pulse rate of 76 beats/min, findings on physical examination were normal. She was treated with oral doxycycline and recovered within four days. Acute and convalescent leptospiral antibody titres were negative.

Acute and convalescent leptospiral serologic tests in the six participants who did not become ill were also negative. At the time of the trip the creek was swollen by recent rains and the water was noted to be malodorous, suggesting contamination by waste from a nearby livestock farm. Illness occurred in all four kayakers who inadvertently swallowed creek water and in none of those who did not.

Leptospirosis was considered as a possible diagnosis when patient 1 was admitted. Typhoid fever was also considered

because of the pulse-temperature disparity and the history of exposure to contaminated water; thus, chloramphenicol therapy was initiated. Two days later, when patient 2 presented, it appeared certain that both men had leptospirosis, and the second patient was started on therapy with doxycycline. Patients 3 and 4 presented earlier in the course of their illnesses, received doxycycline, and experienced symptoms for a shorter period. The early antibiotic therapy probably aborted antibody responses, accounting for the negative convalescent-phase serologic tests in these patients. The uniformity of exposure, the temporal relationship, and the similarity of clinical manifestations strongly suggested that these four people had the same disease.

On repeated examination all four patients had consistently normal heart rate in the presence of high fever. This 'relative bradycardia' is a common finding in typhoid fever and psittacosis but has not been reported in leptospirosis. The explanation for this finding in these patients is not clear. It is unlikely that they had either typhoid fever or psittacosis. A pulse-temperature discrepancy may be an unrecognised effect of infection by a specific serotype of L interrogans (probably djasiman). Since these four persons regularly engaged in physical activity, it is also possible that the observed heart rates represented significant elevations above their usual baseline rates.

DIAGNOSIS AND TREATMENT

The clinical picture and severity of leptospirosis are variable, ranging from mild nonspecific febrile illness to a fatal condition. The course is biphasic in about half the cases. Following an incubation period of 4 to 20 days, the leptospiremic phase typically begins abruptly and is characterised by fever, chills, intense headache, variable but often severe muscle pains, and profound malaise. Photophobia and gastrointestinal symptoms are also common. Conjunctival erythema and muscle tenderness may be subtle and therefore missed.

A wide spectrum of laboratory abnormalities may occur. The white blood cell count is variable. Serum transaminase levels are elevated in approximately half the cases but are rarely increased more than five times normal. Serum creatine phosphokinase may also be increased, and in 25% of cases some degree of azotemia is present. The serum bilirubin level is usually normal or only mildly elevated, but jaundice occurs in about 10% of cases, indicating a more severe form of the disease. Pyuria and proteinuria are common. During the initial febrile phase, the organism can often be recovered from the blood with the use of a special medium (Fletcher's). The initial febrile characteristically lasts four to nine days.

In about half the cases a second febrile period occurs from two to ten days after the initial defervescence and lasts from a couple of days to more than a week. The fever is usually lower and the patient is often less symptomatic than during the leptospiremic phase. However, during this immune phase, meningeal involvement and hepatitis are relatively common; serious complications, such as endocarditis and myocarditis,

may develop in rare cases. During this phase the organism can be recovered from urine but not blood. In some cases the leptospiremic and immune phases overlap and are not clearly distinguishable.

A severe, life-threatening form of leptospirosis (Well's disease) develops in about 5% of the cases. This is characterised by various combinations of hepatic failure, renal failure, haemorrhage, shock, meningitis, and myocarditis. In some series the mortality rate from Well's disease is as high as 40%, while the rate of anicteric leptospirosis is less than 1%.

The diagnosis of leptospirosis should be considered in a child, adolescent, or adult with fever, chills, severe headache, and myalgias combined with recent exposure to animals or a wet outdoor environment. Findings such as photophobia, conjunctivitis, muscle tenderness, jaundice, abnormal urinalysis results, elevated transaminase levels, and azotemia should enhance the suspicion. Differential diagnosis includes eliminating a variety of bacterial and viral infections; differentiation from Rocky Mountain spotted fever and brucellosis may be particularly difficult. Definitive diagnosis depends on culturing the organism or demonstrating an antibody response, findings that are frequently delayed one to three weeks. If the optimum culture medium is not readily available, organisms remain viable for 11 days in blood specimens treated with sodium oxalate.

There are well over 100 serotypes of L interrogans, many of which are pathogenic for humans. To screen serums for leptospiral antibodies, a macroagglutination test using pooled antigen is done. A positive screening test is followed by a microagglutination test to identify serotype-specific antibodies. There is considerable cross-reactivity in antibody responses between the serotypes. The highest titre usually denotes the infecting serotype. The pattern of antibody findings in cases 1 and 2 suggests infection with the djasiman serotype. Concurrent infection with more than one serotype does occur.

Although leptospirosis is usually self-limited, the course may be protracted and involve considerable morbidity. Numerous antibiotics, including penicillin, tetracycline, chloramphenicol (Chloromycetin), and erythromycin, have in vitro antileptospiral activity. However, until recently the clinical value of antibiotic therapy in the management of the disease was uncertain. A randomised controlled clinical trial (1) has shown doxycycline in an oral dosage of 100 mg twice a day to be effective in reducing the duration of symptoms and preventing leptospiruria, when started in the first two or three days of illness.

Little evidence exists to indicate that antibiotic treatment initiated after the first three or four days affects the course of the disease. Because of the usual delay in establishing the diagnosis, the decision regarding antibiotic therapy is based on the nature and duration of symptoms, an estimate of the likelihood of the diagnosis, and an assessment of the potential

risks of treatment. The optimal duration of antibiotic therapy is uncertain; a course of seven to ten days is sufficient in most cases.

PUBLIC HEALTH MEASURES

Prevention of leptospirosis involves several public health strategies. Control of rodents is a crucial component. The rodent-proofing of buildings, use of traps and poisons, and proper disposal of garbage can markedly reduce contact of rats with livestock, pets and humans. Control of leptospirosis in pets has focused on periodic immunisation, which reduces the risk of disease but does not completely prevent subclinical infection and leptospiruria. The risk of infection in pets can be minimised by preventing their contact with rats and other wild animals.

The incidence of the disease among previously high-risk occupational groups has been decreased by using waterproof garments, by protecting mucous membranes and open wounds, and by reducing contact with animals and their urine. The efficacy of chemoprophylaxis with doxycycline has been demonstrated[2]. This approach may be indicated in certain high-risk endemic tropical areas. Avoiding contact with water that is malodorous or otherwise manifests evidence of contamination is a commonsense approach to preventing this and other diseases.

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BRUCELLOSIS IN A GROUP OF TRAVELLERS OF SPAIN
(based on JAMA Vol. 251, No 4, 27 Jan 1984)

Human brucellosis has become an uncommon disease in the United States and Australia for the past decade. Nevertheless, brucellosis remains an important infection of man in other parts of the world, expecially southern Europe, Latin America, and central Asia. The widespread distribution of brucellosis poses a potential health risk to travellers, particularly those whose taste for local foods may find expression in unpasteurised dairy products.

A cluster of six cases of bacteremic brucellosis reported below, in a group of American travellers to Spain, illustrates the transmissibility of brucellosis to travellers and emphasises the importance of an epidemiologic investigation of cases.

CASE REPORT

On 7 April 1980, a previously healthy 17-year-old girl began experiencing headache, fever, chills, and myalgia. Five days later she was admitted to the infirmary at her boarding

school, where the examining physician found no apparent cause for fever. Her haemoglobin level was 13.4 g/dL, the leukocyte count was 6 400/uL, and results of two slide tests for infection mononucleosis heterophil antibodies, were negative. Her symptoms grew worse, and her temperature intermittently rose above 39°C despite administration of aspirin and treatment with cephalexin monohydrate for four days.

The patient was examined by a second physician on 21 April and a blood culture specimen was obtained. Because no diagnosis was established, the patient was brought to a university medical centre on 25 April. She denied intravenous drug use, blood transfusion, or recent exposure to animals. She noted that a class mate (patient 2) at her school was experiencing similar symptoms and that both had participated in a school-sponsored trip to Spain during 4-14 January 1980. Physical examination disclosed no abnormal findings other than apparent fatigue and an oral temperature of 38.3oC. Haemoglobin level, leukocyte count, and ESR were normal, and aspartate and alanine aminotransferase levels were slightly elevated. Hospital admission was declined, but the patient returned for treatment the following day when a Brucella agglutinin titre of 1280 was detected by a rapid tube-dilution method. Subsequently blood culture specimens collected on 21 April and 25 April yielded Brucella melitensis.

Treatment with tetracycline hydrochloride, 2.0 g/day for five weeks, and streptomycin sulphate, 1.0 g/day for two weeks, was administered. During the second day of treatment, the patient became afebrile; however, posterior neck soreness in the region of the cervical spine began. There were no signs of meningeal irritation and a Tc 99m hydroxy-diphosphonate bone scan was normal. The neck pain resolved, and the patient felt well enough to return to school on 5 May. Fatigue and intermittent headaches persisted for several more weeks, but no relapse occurred during the following two years while contact with the patient was maintained.

EPIDEMIOLOGIC INVESTIGATION

The possibility that the patients infection was part of an outbreak involving her travel group or school prompted a search for additional cases. The physician treating the classmate (patient 2) with similar symptoms was advised to test the Brucella agglutinin titre, and the physician also tested the titre of another ill student-traveller (patient 3) under his care.

On 1 May 1980, serum specimens were obtained from the 24 travellers who had not yet been tested and also from 23 consenting, randomly selected classmates who had not gone on the trip.

Rapid slide tests performed at the hospital where two ill classmates (patients 2 and 3) of the first patient were being treated showed Brucella agglutinin titres above 320. The diagnosis of brucellosis was subsequently confirmed by standard

tube test titres of 1280 and by isolation of B melitensis from cultures of blood. Patients 2 and 3 had participated with patient 1 in the trip to Spain. Testing of serum specimens from the other 17 student and seven adult travellers identified three more students (patients 4 through 6) with Brucella agglutinin titres of 640 or greater; blood culture specimens from these students also yielded B melitenisi. Brucella agglutinin titres of the remaining 21 travellers and 23 control students who had not gone on the trip were below 40. The clustering of cases of brucellosis in six of the 27 travellers compared with none of 23 control students was statistically significant ($P < .05$, Fisher's exact test).

Food-history questionnaires administered in May 1980, four months after the trip, were completed by five of the patients and then unaffected students. Specific meals were rarely remembered, and no single meat or dairy item was consumed significantly more often by patients than controls ($P > .10$). However, the mean number of times patients reported eating foods containing cheese (usually cheese omelets and cheese sandwiches) was 9.4 compared with 5.0 for controls. The greater consumption by patients of foods containing cheese was statistically significant ($P < .01$, Mann-Whitney U tests).

An itinerary for breakfast and dinner generally was planned, and the entire travel group usually ate their meals together. Locations for lunch were selected on an impromtu basis, and the six patients described themselves as adventuresome in their frequent choice of small restaurants and taverns for this meal.

The mode of transmission of brucellosis to the travellers described in this report could not be retrospectively established. However, results from the food-history questionnaire are compatible with evidence from previous cases implicating unpasteurised dairy products. The students who became infected consumed cheese - containing food items significantly more often than did noninfected student travellers. Furthermore the cheese that they ate either had not been cooked (sandwiches) or had only been briefly heated (omelets). The frequency of Brucella contamination of goat's milk cheese in Spain, has not been reported, but serosurveys demonstrated a mean infection rate of 6% to 7% in goats. In Iran, where infection of goats also is a problem, B melitensis was isolated from 8.3% of cheese specimens sampled from retail shops in one area.

CLINICAL AND MICROBIOLOGICAL FEATURES

Clinical characteristics of the six patients are summarised below:

- the incubation period, calculated from the midpoint of the trip, ranged from 5 to 17 weeks.
- symptoms were primarily systemic, and included most commonly chills, fever, fatigue and sweats.
- located musculoskeletal symptoms were reported by patients 1 through 4:
  - patients 1,2 and 3 had back or neck pain, and patient 3 had been treated with traction before brucellosis was diagnosed.

- in patient 4, septic arthritis developed in his right knee after a glucocorticoid injection into an area of suspected tendonitis adjacent to the joint. The injection was administered after systemic symptoms had begun, and later culture of synovial fluid yielded B melitensis.

Before recognition of the outbreak, patients 1 through 4 made nine visits to four different physicians. The patients were confirmed in the school infirmary for 16 days and in hospitals for 11 days before diagnosis. In contrast, the diagnosis of brucellosis in patients 5 and 6 was made by Brucella agglutination testing 3 to 7 days before the onset of symptoms. Treatment was withheld by their physician until symptoms began, at which time positive blood culture specimens were obtained.

Two to five blood culture specimens were collected from each of the six patients. At least one specimen from each patient and 15 (79%) of the 19 total specimens yielded B melitensis. Specimens from which B melitensis was recovered gave a positive reading in the radiometric system 4 to 8 days after inoculation. Isolates of B melitensis from all six patients were reported to be biotype 1. The isolates were identical in their response to all biochemical and serological tests.

Two clinical features of brucellosis in the patients described in this report may by useful in the approach to other suspected cases. First, joint or skeletal complaints, which previously have been described in brucellosis, seems to be a clue to the diagnosis. Such complaints were reported by all four patients whose symptoms lasted for at least a week. Back or neck pain occurred in patients 1 through 3, and patient 4 had septic arthritis of a knee. Although the glucocorticoid injection that patient 4 received could have induced infection in the knee, infection of the joint or bone probably was already present as the cause of the presumed tendonitis. Second, blood culture specimens from each of the six patients yielded B melitensis, and the combined rate of recovery from all blood culture specimens was 79%. Although earlier reports described low rates of recovery of Brucella from blood culture specimens, recent clusters of acute cases have been characterised by positive blood cultures. Consequently, it seems that prolonged incubation of several blood culture specimens is a sensitive method for confirming acute B melitensis infection.

Epidemiologic investigation of patients with brucellosis had been important in identifying additional undiagnosed cases. The present investigation facilitated the diagnosis of brucellosis in five patients and lessened the cost of illness. Three of the patients (patients 2 through 4) had had symptoms for one to ten weeks when the investigation began. These three had made seven visits to physicians, and two of the patients had been admitted to hospitals. In comparison, a presumptive diagnosis of brucellosis was made in two other patients by Brucella agglutination testing before the onset of symptoms. Although their physician withheld therapy until symptoms began, illness was mild and no diagnostic tests other than blood cultures were required.

CONCLUSION

Because the clinical features of brucellosis are not distinctive, epidemiologic information often is important in suggesting the diagnosis. Most cases of brucellosis have occurred in persons with occupational or avocational exposure to animals or laboratory cultures of Brucella. These persons usually have been workers in the livestock and meat-processing industries or were veterinarians, hunters, or microbiology laboratory personnel. In addition, several reports indicate that travellers to areas where brucellosis is endemic, may acquire this infection. Travel to Spain, which is a major location of brucellosis, prompted Brucella agglutination testing of patient 1 in the present cluster of cases.

Other countries that have reported large numbers of cases of brucellosis include Mexico, Argentina, Peru, Italy, Greece, and Iran. Among these countries, Mexico has been implicated most frequently in cases of brucellosis in travellers from the US. Several other reports have described clusters of cases in Mexican - Americans who have either travelled to Mexico or consumed goat cheese purchased in Mexico.

Based on the occurrence of a number of cases of brucellosis reported since the early seventies in travellers to endemic areas, it seems appropriate for physicians to advise prospective travellers of precautions. Travellers should be instructed that because of the risk of brucellosis or other zoonosis, they should not consume unpasteurised milk or products such as cheese made from unpasteurised milk.

NOTICE TO READERS

ICI Australia Operations Pty Ltd has advised CDI that the antimalaria agent, Proguanil hydrochloride, distributed as Paludrine, will not be available until at least the end of 1987.

1986 RUBELLA EPIDEMIC IN SOUTH EAST QUEENSLAND

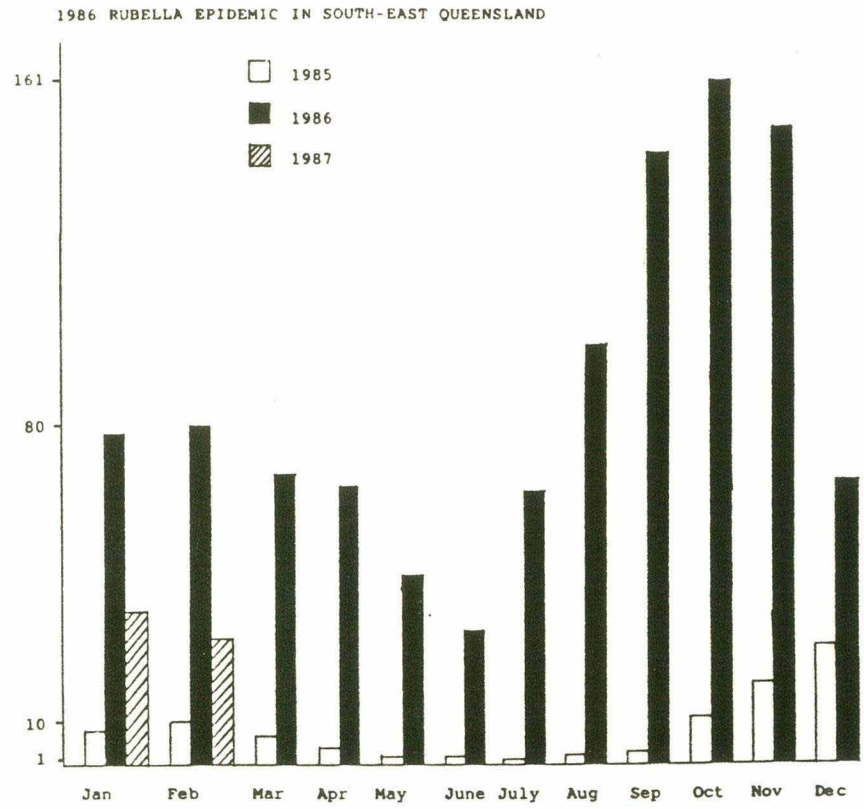
(Contributed by Dr D M Wyatt and Dr W R Forgan-Smith, Queensland Medical Laboratory)

Queensland Medical Laboratory has reported a marked increase in rubella cases during 1986 with 1021 cases recorded compared with 97 reported in 1985 (Figure). The majority of cases were from patients residing in South-East Queensland.

Ten of the cases were pregnant women, two of which appeared to have been reinfected following a waning vaccine induced immunity to rubella.

The cases reported for 1986 represented 4 per cent of the total sera submitted for testing at Queensland Medical Laboratory in that year.

FIGURE:



CDI Editorial Comment:-

It is important to monitor the rubella vaccination program and therefore all proven or suspected cases of congenital rubella should be repofted to the State Health Authority. The current National Health and Medical Research Council recommendations for rubella vaccine deployment is as follows:

(i) All girls between their tenth and sixteenth birthdays should be routinely offered rubella vaccine. A history of a previous attack of rubella should be disregarded in view of the difficulty of being certain of the diagnosis. Schoolgirls are not tested serologically prior to vaccination as this is impractical and costly. It is important that uptake be very high in this program. Girls who are immune are not disadvantaged by the vaccination.

(ii) Women of child bearing age should be tested prior to pregnancy. All seronegative women of child-bearing age, provided they are not pregnant, should be offered rubella vaccine. As rubella vaccine virus can cause fetal infection, it is desirable that the vaccine should NOT be administered to any women who may be pregnant. Those administering the vaccine should be careful to instruct women to whom it is given that they should not become pregnant for at least two full menstrual cycles. However, to date, there have not been any congenital rubella-like defects in the live born infants (about 400) of seronegative mothers vaccinated during or just before pregnancy.

> Based on this experience, rubella vaccination during pregnancy need not be the reason to recommend interruption of pregnancy. The final decision must be made by the patient and her physician. The American Advisory Committee for Immunization Practices (ACIP) at present, believes that the risk of vaccination associated malformations is so small as to be negligible.

A convenient time to immunise seronegative women is when prescribing contraceptives and in the immediate post-partum period. All women should be informed of their immune status in writing.

Certain groups of women such as school teachers, nursery and play group staff, community health staff, staff of children's hospitals and obstetrics units may be at special risk of contracting rubella. Staff and students, both male and female, working in antenatal clinics may, if they become naturally infected, transmit rubella to patients in the early stage of pregnancy. Individuals in all these groups including all medical students and trainee nurses should have their antibody status determined and, if found to be seronegative, should be offered vaccination both for their own protection and for the protection of seronegative pregnant women.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 9/3/87 - 22/3/87 BULLETIN NUMBER 87/6
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED		2	5		5		7	1	20
0101 ADENOVIRUS TYPE 1					4				4
0102 ADENOVIRUS TYPE 2	1				7				8
0103 ADENOVIRUS TYPE 3				1		1			2
0105 ADENOVIRUS TYPE 5					6	1		1	8
0106 ADENOVIRUS TYPE 6					1				1
0107 ADENOVIRUS TYPE 7						1			1
0108 ADENOVIRUS TYPE 8				1		1			2
0109 ADENOVIRUS TYPE 9	1								1
0111 ADENOVIRUS TYPE 11				1					1
0113 ADENOVIRUS TYPE 13	1								1
0122 ADENOVIRUS TYPE 22	1								1
0199 ADENOVIRUS TYPING PENDING	2		1						3
0201 INFLUENZA A VIRUS	1								1
0203 INFLUENZA B VIRUS	1								1
0301 PARAINFLUENZA VIRUS TYPE 1	1				1				2
0302 PARAINFLUENZA VIRUS TYPE 2					2		2	2	6
0303 PARAINFLUENZA VIRUS TYPE 3	1				1		4	3	9
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	3		1	3	1	3	2	4	17
0500 RHINOVIRUS (ALL TYPES)					7	7	2		16
0600 MYCOPLASMA PNEUMONIAE	11		2		1	1	2	4	21
0700 ORNITHOSIS-PSITTACOSIS	1								1
0902 COXSACKIEVIRUS B2					1				1
0903 COXSACKIEVIRUS B3						1			1
1003 ECHOVIRUS TYPE 3					1				1
1005 ECHOVIRUS TYPE 5	1			4					5
1007 ECHOVIRUS TYPE 7					2				2
1011 ECHOVIRUS TYPE 11	9	2		2	8		3		24
1014 ECHOVIRUS TYPE 14					1		1		2
1018 ECHOVIRUS TYPE 18	1								1
1020 ECHOVIRUS TYPE 20					1	1			2
1021 ECHOVIRUS TYPE 21					1				1
1022 ECHOVIRUS TYPE 22					1				1
1100 POLIOVIRUS NOT TYPED			1		2				3
1102 POLIOVIRUS TYPE 2	1	1		1					3
1103 POLIOVIRUS TYPE 3	1			1					2
1200 MUMPS VIRUS	1						1	2	4
1300 HERPES VIRUS GROUP-NOT TYPED	28			1			4	1	34
1301 HERPES SIMPLEX VIRUS NOT-TYPED		8						1	9
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	11		2		1	3	7	5	29
1303 VARICELLA-ZOSTER VIRUS	4	1	2	1			3	2	13
1306 HERPES SIMPLEX TYPE 1	38		7	31		23	40	23	162
1307 HERPES SIMPLEX TYPE 2	102		27	59		18	70	47	323
1399 HERPES VIRUS TYPING PENDING				2	4				6
1401 COXIELLA BURNETI	13		1			1	4		19
1502 PICORNA VIRUS-NOT TYPED	9		9				16		34
1521 MEASLES VIRUS	1		1					8	10
1522 RUBELLA VIRUS	5			1			16	1	23
1532 HEPATITIS B ANTIGEN	79	3	4	18		16	31	16	167
1535 HEPATITIS A ANTIBODY	12		1	2		1	3	4	23
1541 CHLAMYDIA A - C TRACHOMATIS	28	1	1			110	18	85	243
1556 CMV - CYTOMEGALOVIRUS	4		5	26	8	4	10	8	65
1564 ROTAVIRUS	4					16	1	1	22
1571 ENTEROVIRUS TYPE 71 (BRCR)					1				1
1599 ENTEROVIRUS TYPING PENDING		1	9						10
9992 ROSS RIVER VIRUS			3				17	7	27
9997 KUNJIN VIRUS							1		1
9998 ARBO. GROUP B. ...							1		1
Total	377	19	82	155	68	209	266	226	1,402

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 9/3/87 - 22/3/87 BULLETIN NO 87/6

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir-atory	Enceph-alitis	Mening-itis	Para-lysis	CNS other unspec	GI	Hepa-tic	CVS	Urin-ary	Skin/ mucs memb
0100 ADENOVIRUS NOT TYPED.....							1				
0101 ADENOVIRUS TYPE 1.....		5									
0102 ADENOVIRUS TYPE 2.....		5					1				
0103 ADENOVIRUS TYPE 3.....		1									
0105 ADENOVIRUS TYPE 5.....		4					3				
0106 ADENOVIRUS TYPE 6.....		1									
0107 ADENOVIRUS TYPE 7.....							1				
0109 ADENOVIRUS TYPE 9.....							1				
0111 ADENOVIRUS TYPE 11.....										1	
0113 ADENOVIRUS TYPE 13.....							1				
0122 ADENOVIRUS TYPE 22.....							1				
0201 INFLUENZA A VIRUS.....		1									
0203 INFLUENZA B VIRUS.....		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		2									
0302 PARAINFLUENZA VIRUS TYPE 2....	1	5									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	6				1		1			1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		17									
0500 RHINOVIRUS (ALL TYPES).....		16									
0600 MYCOPLASMA PNEUMONIAE.....	3	12									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0902 COXSACKIEVIRUS B2.....				1							
0903 COXSACKIEVIRUS B3.....		1									
1005 ECHOVIRUS TYPE 5.....	1			3							1
1007 ECHOVIRUS TYPE 7.....		1		1			1				
1011 ECHOVIRUS TYPE 11.....		6		9			5				
1014 ECHOVIRUS TYPE 14.....		1									
1018 ECHOVIRUS TYPE 18.....							1				
1020 ECHOVIRUS TYPE 20.....		2									1
1021 ECHOVIRUS TYPE 21.....											
1022 ECHOVIRUS TYPE 22.....		1									
1102 POLIOVIRUS TYPE 2.....							1				
1103 POLIOVIRUS TYPE 3.....							1				
1200 MUMPS VIRUS.....	3										
1300 HERPES VIRUS GROUP-NOT TYPED..	2										1
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	7	2				2		3			
1303 VARICELLA-ZOSTER VIRUS.....	3	1							1		7
1306 HERPES SIMPLEX TYPE 1.....	6	6				1				2	87
1307 HERPES SIMPLEX TYPE 2.....	10									1	67
1401 COXIELLA BURNETI.....	6			1					1		
1502 PICORNA VIRUS-NOT TYPED.....	1					3	10				
1521 MEASLES VIRUS.....	2	7	1			1					1
1522 RUBELLA VIRUS.....	2	1						1			13
1532 HEPATITIS B ANTIGEN.....	48							109			
1535 HEPATITIS A ANTIBODY.....	3							20			
1541 CHLAMYDIA A - C.TRACHOMATIS...	20										
1556 CMV - CYTOMEGALOVIRUS.....	8	6						1	1	5	3
1564 ROTAVIRUS.....							22				
1571 ENTEROVIRUS TYPE 71 (BRCR)....											1
9992 ROSS RIVER VIRUS.....	4		1					1			5
9997 KUNJIN VIRUS.....	1										
Total.....	132	112	2	15		8	50	136	3	9	189

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 9/3/87 - 22/3/87 BULLETIN NO 87/6
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0102 ADENOVIRUS TYPE 2.....							1		1	
0103 ADENOVIRUS TYPE 3.....	1							1		
0105 ADENOVIRUS TYPE 5.....	1						1		1	
0106 ADENOVIRUS TYPE 6.....								1		
0108 ADENOVIRUS TYPE 8.....	2									
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0500 RHINOVIRUS (ALL TYPES).....								1		
0600 MYCOPLASMA PNEUMONIAE.....			2				3	3	1	
1003 ECHOVIRUS TYPE 3.....							1			
1005 ECHOVIRUS TYPE 5.....								1		
1011 ECHOVIRUS TYPE 11.....								3	1	1
1014 ECHOVIRUS TYPE 14.....					1					
1102 POLIOVIRUS TYPE 2.....									2	
1103 POLIOVIRUS TYPE 3.....									1	
1200 MUMPS VIRUS.....								1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			12	5				5		
1303 VARICELLA-ZOSTER VIRUS.....		1							1	
1306 HERPES SIMPLEX TYPE 1.....	3	56						1	2	
1307 HERPES SIMPLEX TYPE 2.....		246								
1401 COXIELLA BURNETI.....							4	7	1	
1522 RUBELLA VIRUS.....			5		5	2		3	1	
1532 HEPATITIS B ANTIGEN.....		3							7	
1541 CHLAMYDIA A - C.TRACHOMATIS...		221							2	
1556 CMV - CYTOMEGALOVIRUS.....		2	1	1		9	2	5	22	
9992 ROSS RIVER VIRUS.....					18			5		
Total.....	7	529	20	6	24	11	12	38	43	1

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 13 - 29 November 1986 to 31 December 1986 Bulletin...87/6....

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis				1	1				2	56
Ankylostomiasis				2	3		NN		5	* 39
Anthrax									-	-
Arbovirus infection	6	1	131		NN				138	1 431
Brucellosis									-	13
Campylobacter infections	137		NN	130	16	NN	1	NN	284	* 2 943
Chancroid				NN	1				1	10
Cholera									-	-
Congenital rubella syndrome			NN			NN		NN	-	1
Diphtheria							2		2	45
Donovanosis			25	NN	10		3		38	170
Giardiasis	40		NN	74	20	NN	NN	NN	134	* 1 312
Genital herpes	48		23	25	NN	NN	1	3	100	1 404
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	3
Gonorrhoea	89		209	40	208	6	30	7	589	4 992
Hepatitis A (infectious)	7	16	6	23	10		5		67	* 1 684
Hepatitis B (serum)	35	11	38	3	38	1		2	128	1 780
Hepatitis – unspecified	3		1	2	NN	NN			6	* 145
Hydatid disease				1		1			2	13
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	5	1	NN			NN		NN	6	63
Leprosy	1								1	25
Leptospirosis		4	7	2		7			20	191
Lymphogranuloma venereum			1	NN	NN	NN		NN	1	6
Marburg disease			NN			NN		NN	-	-
Malaria	8	5	36	2	7	3		3	64	* 730
									-	-
Meningococcal infections	2		1	3		NN			6	* 50

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	256		77	50	NN	NN	NN	NN	383	4 466
Ornithosis				1					1	44
Pertussis (whooping cough)	12		NN	6	69	NN	1	NN	88	* 601
Plague									-	-
Poliomyelitis									-	1
Q. fever	17	1	26	1					45	355
Rabies				NN		NN		NN	-	-
Salmonella infections	63	15	29	33	19	1	11	1	172	2 479
Shigella infections	5		11	3	19		19		57	831
Smallpox									-	-
Syphilis	25		125	1	31		93	2	277	2 323
Tetanus									-	6
Trachoma		NN			79	NN	NN		79	234
Tuberculosis (all forms)	16	27	14	12	11		2	NN	82	1 065
Typhoid fever	3		1					1	5	42
Typhus (all forms)									-	14
Vibrio parahaemolyticus infections			NN			NN		NN	-	6
Yellow fever									-	-
Yersinia infections	5		NN	1		NN		NN	6	* 77

NN - Not Notifiable

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon voluntary reporting by medical practitioners etc.)

* Adjustment to the Cumulative Total since last report:

Ankylostomiasis	-1	South Australia	Meningococcal inf.	+1	South Aust.
Campylobacter inf.	+1	South Australia	Pertussis	+2	South Aust.
Giardiasis	-6	South Australia	Yersinia inf.	-1	South Aust.
Hepatitis A	-3	South Australia			
Hepatitis unspec.	+1	South Australia			
Malaria	-2	South Australia			