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Contents:

Editor *Dr Robert Hall*

IMPORTANT NOTICE: Some issues of the last CDI were missing the second leaf. Should your copy be missing pages 3 and 4, please insert the leaf provided at the end of this issue.

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1307 reports were processed during this period.

Four reports of Q fever (3 males - 46 years, 48 years and a 35-year-old farmer - and 1 a 12-year-old female) were received during this period.

Eight influenza reports were received during this period - 3 influenza A (including one subtype H_3N_2) and 5 influenza B. So far this year twelve cases of influenza A (including 1 H_3N_2 and 1 H_1N_1) and 16 cases of influenza B have been reported.

The seasonal increase in respiratory syncytial virus continues with 430 reports received during this period. Reports by collection date since the beginning of the year are:

Jan	Feb	Mar	Apr	May	June
10	11	38	76	288	665

Following last year's echovirus type 9 outbreak in late 1988 in WA, increased activity in Victoria has been observed. Sixty percent (39/63) of echovirus type 9 cases have been reported

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- Contributions are solicited, and do not preclude later publication elsewhere.

- Figures given may be subject to revision.

from Fairfield Hospital with a monthly incidence ranging from 1 to 12. Of the 63 cases reported, severe morbidity was observed with 37 cases of meningitis and one of encephalitis.

Increased activity has been observed this year compared to last year for:

- adenovirus type 3: Sixty-two (62) reports have been received so far this year compared to last year (87 reports). Over 40% (19/62) of these reports orignated from IMVS, SA with 30%, 21% and 6% being reported by Victoria, NSW and Western Australia respectively. Respiratory and ocular symptoms predominated. Sixty-one per cent of cases occurred in children under 5 years of age;

- adenovirus type 19: Ten reports have been received so far this year compared to 4 reports for each of the previous two years. Ocular symptoms predominated. Four cases were in children under 1 month of age; the remaining cases were in adults over 20 years of age. Seven of the 10 cases were reported by Westmead Hospital, NSW;

- parainfluenza type 2: So far this year 128 reports have been received compared to 130 reports in 1988 and 81 in 1987. Seventy-seven per cent of cases were in children under 5 years of age, and another 19% were aged from 5 to 14 years.

OVERSEAS BRIEFS:

1. TRAVELLERS TO SAUDI ARABIA - POSSIBLE QUARANTINE IMPLICATIONS

For some years now, Saudi Arabia has required all pilgrims and visitors to holy places to be in possession of a valid certificate of vaccination against meningococcal meningitis issued not more than 2 years and not less than 10 days before the date of arrival. Those not in possession of a valid certification will be vaccinated on arrival.

All travellers arriving from the following list of countries who do not possess a valid vaccination certificate will be subject to health checks on arrival and if suspected of having meningitis, will be quarantined:

Benin	Ethiopia	Pakistan
Burkino Faso	India	Senegal
Cameroon	Mauritania	Sudan
Chad	Morocco	Syrian Arab Republic
Cote d'Ivoire	Niger	Togo
Egypt	Nigeria	Yemen

2. DENGUE IN MALAYSIA

(Based on MMWR, 23 June 1989, P194)

The Ministry of Health, Malaysia, has reported an outbreak of dengue fever and dengue haemorrhagic fever in Johore State, Pahang State and the Federal Territory (see below). No information has been provided on serotype(s) involved.

	Dengue fever	Dengue haemorrhagic fever	Deaths
Johore State	43	29	4
Pahang State	55	3	0
Federal Territory	45	18	0
Total	143	50	4

Q FEVER AND Q FEVER VACCINE (QVAX)

On 5 March 1989 the Australian Drug Evaluation Committee approved CSL's Q fever vaccine (QVAX) for general marketing. This vaccine is now available in limited quantities.

As vaccination of persons who have already been exposed to Q fever, either through clinical or subclinical infection, can result in severe local adverse reactions, it is essential that potential vaccinees first be tested for evidence of humoral and cell mediated immunity. If evidence of prior immunity is present, vaccination is contraindicated.

The following article is prepared from information put together by CSL which will be used to acquaint appropriate personnel with the zoonosis, Q fever, and with its prevention.

The organism

The disease is caused by a microorganism from the genus coxiella, *Coxiella burnetii*, the only member of the genus.

Coxiellas have some similarities to other rickettsias but an unusual difference is that the *C. burnetii* exists in two antigenic phases. Except for the phase differences, there is little antigenic difference between coxiella strains from different hosts and different places. The organism exists in the wild as Phase I.

Q fever is a zoonosis of cattle, sheep, rodents and their attendant ticks. A wildlife cycle also exists involving, in Australia, animals such as the bandicoot and the kangaroo.

C. burnetii can be found in the milk, excreta, and placenta of infected farm animals, particularly goats, sheep and cows. Dogs and cats may sometimes be infected and excrete the organism at parturition.

C. burnetii has very high resistance to drying and can remain viable at 4°C for more than a year on fomites.

Transmission

Human Q fever is acquired by inhalation of infected aerosols. Air sampling experiments have shown that *C. burnetii* may be disseminated as a primary aerosol at parturition of an infected animal. Alternatively, because the organism is resistant to heat, drying and sunlight, a *C. burnetii*-laden dust often forms

from contaminated birth fluids, blood, faeces, or urine. Such dusts may be disseminated by dry and windy weather or carried on fomites such as wool, hides, farmworkers' clothing, straw, and packing materials, to be released later as secondary aerosols in other environments. Farm dogs, particularly if fed on infected placentas, and farmyard chickens may contribute their own excreta to this part of the cycle.

Lower but significant concentrations of the organism are found in the udders and milk of infected cows. Drinking infected raw milk accounts for some sporadic cases. The protective or neutralising properties of whey antibody in infected milk are a possible explanation for the low incidence of clinical Q fever arising from this mode of transmission. Carcass meat is not infective.

Within the herd or flock infection is probably maintained by inhalation of infected dusts and aerosols. Newborn or immature, nonimmune animals are infected from parturient animals or the contaminated environment. After a brief rickettsemia, the coxiella remains latent until the animal becomes pregnant when the infection is reactivated and the organism is excreted at term. In some herds the organism may perhaps be transferred from udder to udder by the milking process or by animal bedding.

Man-to-man infection is extremely uncommon, but in rare instances patients with *C. burnetii* in their sputum, urine or placenta have been identified as sources of infection. A significant incidence of infection has been reported among laboratory workers handling infected tissues, specimens, and laboratory animals, and among medical and paramedical personnel attending autopsies on cases of Q fever.

Wildlife resevoirs are of little direct relevance to the human infection. On rare occasions a connection between the two cycles may be established, as when the infection is transmitted to sheep by ticks that have previously fed on kangaroos, or when infectious ticks' faeces are inhaled by man. Man rarely gets Q fever from a tick bite. A brief report of Q fever infection with a dirty knife which had been used to skin a kangaroo, was published in *CDI* recently [1].

Clinical manifestations

In man the incubation period for Q fever is two to four weeks, commonly 19-21 days (occasionally up to 60 days), and the symptoms are a fever of acute onset that seldom exceeds 40-41°C, chills, a particularly severe headache, cough, and muscle pain (a presentation often misdiagnosed as influenza). The fever mostly lasts from one to two weeks. The primary site of infection is in the lungs, and various degrees of pneumonitis may result, depending on the extent of infection. There is frequently biochemical evidence of hepatic involvement (elevated alkaline phosphatase, raised aminotransferase, and increased bilirubin) and overt jaundice may occur.

Q fever is a well documented cause of chronic endocarditis, especially in persons with prior mitral or aortic valve disease. Apart from the prolonged fluctuating fever, chills, night sweats, extreme malaise and thrombolic or embolic

incidents, endocarditis patients may also present with a purpuric rash, thrombocytopenia, hepatomegaly, and/or splenomegaly. These signs and symptoms may help to differentiate Q fever endocarditis from subacute bacterial endocarditis. Recent reports have described a chronic hepatitis (without endocarditis) but experience with this complication is still limited.

Q fever is treated with broad spectrum antibiotics, the antibiotic of first choice is tetracycline. Chloramphenicol is also effective and rifampicin may be of value in chronic infection.

Death from acute Q fever is extremely rare and usually occurs only in elderly and diseased patients. Following recovery from acute Q fever, most patients can expect life-long immunity. Reports of second and subsequent attacks of Q fever are probably due to the misidentification of a recurrent post Q fever debility syndrome (PQDS) of unknown pathogenic mechanism, as an attack of acute Q fever. It is estimated that as many as 20-40% of acute Q fever cases in abattoir workers are followed by PQDS.

Epidemiology

Prevalence and distribution

Because of occupational exposure, Q fever is primarily a disease of men rather than of women, and usually affects those between 20 and 60 years of age. Symptomatic infection in childhood is rare, but subclinical infection, which may be acquired by ingestion of infected unpasteurised milk, is not uncommon. In areas of high Q fever endemicity human fetal infection has been demonstrated, but whether such fetal infection has any later effect on the developing child is unknown.

Q fever is a well recognised occupational hazard for:

- abattoir workers;
- farm workers;
- shepherds;
- shearers;
- wool sorters;
- dairy workers;
- veterinary personnel (including veterinary students); and
- pelt and hide tanners.

New recruits to these occupations bear the brunt of the infection as persons in these occupations eventually become immune.

Similarly, non-immune visitors to contaminated environments are particularly at risk. Cases of Q fever have been reported in:

- maintenance engineers servicing equipment in abattoir slaughtering lines or cold stores;
- catering school students;
- research workers obtaining animal tissues;
- veterinary school technicians;
- fire assessors for insurance companies; and
- agricultural machinery salesmen visiting farms.

In some countries - eg, Switzerland - Q fever has been observed in the general population along routes used to drove sheep from winter to summer pastures. This 'locality' infection is probably rare in Australia, although Q fever cases have been detected in the vicinity of abattoir stockyards and have been connected with the cleaning of trucks or railway cars used to transport livestock.

In many regions a seasonal variation in incidence occurs, related in most instances to farming activity. Higher prevalences are to be expected at times of calving, lambing or shearing. Dry summers encourage airborne dissemination. Apart from domestic livestock as the main source of infection, maintenance cycles in wild animals - bush rodents, kangaroos - may sometimes be the source of infection for man, but direct infection from them is rare.

Q fever is found throughout the world but in Sweden, Norway, Iceland and New Zealand it is rare. The few reported Scandinavian patients were probably infected while travelling or working in the Mediterranean region, where Q fever is common.

Q fever vaccine trial

Various forms of inactivated whole cell *C. burnetii* vaccines have been used for many years in laboratory workers handling the organism but industrially exposed groups had not been vaccinated.

Starting in 1981, a clinical trial of a formalin-inactivated vaccine made from *C. burnetii*, Henzerling strain in Phase I antigenic state, was mounted in four South Australian abattoirs.

Volunteers among abattoir workers, employees of other firms on the site, and visitors to the abattoirs were serotested and skintested to determine whether they were already immune and sensitised to vaccine. Subjects without significant immune markers were then inoculated subcutaneously with a dose of the inactivated Q fever vaccine.

During the period 1981-88, over 4000 subjects in the four abattoirs were vaccinated. There have been no Q fever cases among those vaccinees who had time (>15 days) to develop immunity after vaccination and before exposure to infection. There were, however, eight Q fever cases among vaccinees inoculated during the incubation period of the natural infection. On the other hand, there were 97 Q fever cases in unvaccinated persons working in or visiting the abattoir environments. Four hundred and sixty-four (464) vaccinees from two abattoirs were followed up intensively for adverse reactions (see Table 1).

In addition the following uncommon or chronic reactions were noted:

- 1 vaccinee noticed a small mobile lump under the skin at the vaccination site which lasted for 2 months and then resolved.
- 1 vaccinee had an abscess at the site of injection.
- 1 vaccinee complained of transient dizzy spells. He had head trauma and similar spells prior to vaccination.
- 1 vaccinee developed an indurated lump at the site of inoculation.

Table 1: Common Adverse reactions to Q fever vaccine - Samcour and Dandy Jacobs abattoirs

Reactions observed among 464 vaccinees	Number	(%)
Temperature >38°C	1	(0.2)
Headache	44	(9.5)
Local tenderness	223	(48.0)
Local erythema	154	(33.0)
Other reactions (a)	70	(15.0)
Later reactions (b)	6	(1.3)

(a) 'Other reactions' included aching joints, swollen glands, flu-like symptoms, feeling faint, itching or induration at site of injection.
(b) 'Later reactions' were coincidental respiratory tract infections.

The first two vaccinees had a complement fixing antibody titre or 2.5 or greater and should not have been vaccinated. The fourth vaccinee was antibody negative by a sensitive test (immunofluorescence) and considered to be skin test negative in the prevaccination 'screen' but may have been exposed to Q fever as a child in Eastern Europe before migration to Australia.

The 'risk factor' for chronic reaction to Q fever vaccine appears to be long-standing cell mediated immunity or sensitivity, coupled with a decline of antibody to low or undetectable levels. In these circumstances, the correct performance of the skin test is even more critical. Errors in dilution of the skin test antigen (ie, from the vaccine), inadequate mixing of the diluted skin test antigen, or in retaining and reusing the diluted antigen for more than a few hours (thereby weakening the dose by absorption of the coxiellas to the glass of the container) may lead to a false-negative result and so to a reaction when the vaccine is given.

Immune responses to Q fever and to vaccination

Both humoral and cell mediated immunity are developed in Q fever. Cell mediated immunity is of primary importance with intracellular organisms such as *C. burnetii* and can be detected by a skin test using a dilute suspension of vaccine. The method for this test is described in the Q fever product information appended to this article (see page 9). *In vitro* tests for cell mediated immunity such as stimulation of peripheral blood mononuclear cells can also be used.

Serological tests for antibody to Q fever are:

. Complement fixation:
  - widely used for the diagnosis of clinical Q fever cases;
  - useful for detection of antibody in strong dilutions of serum before vaccination; and
  - not suitable for measurement of antibody development after vaccination;

. Immunofluorescence (particularly immunoglobulin class analysis):
  - sensitive and valuable for detection of IgM antibody during acute phase of clinical Q fever; and
  - useful for detection of antibody after vaccination (equivalent sensitivity to ELISA and RIA;

. Enzyme linked immunosorbent assay (ELISA); and

. Radioimmunoassay (RIA).

Antibody responses in acute Q fever

Acute Q fever will result in a fourfold increase in complement fixing (CF) antibody against Phase II antigen or immunofluorescence-IgM antibody against Phase I and Phase II antigens.

Detection of IgM is particularly useful in the small proportion of patients (approximately 10%) with equivocal results in complement fixation tests.

Antibody responses in chronic Q fever

High CF antibody titres to Phase I and Phase II antigens of *C. burnetii* are diagnostic. Serotesting by immunofluorescence (IF) shows high IgG and IgA antibody titres to both Phase I and Phase II antigen and low or no antibody in the IgM immunoglobulin class. Care is needed with the performance of IgM antibody tests as a majority of Q fever endocarditis patients have large amounts or IgM rheumatoid factor, which accounts for the generally raised level of IgM reported for these cases.

Antibody responses after vaccination

Prevaccination CF and skin test is not an absolute test of immunity but is done primarily to exclude individuals who might develop severe reactions at the inoculation site when vaccinated. Some subjects with negative antibody and skin tests have positive lymphocyte stimulation indices indicating minor or threshold levels of immunity or sensitisation to *C. burnetii*.

Antibody responses (tabulated below) are different in high risk groups (eg, abattoir workers) as compared with low risk groups (eg, research workers or others making infrequent visits to the abattoirs)(see Table 2).

The greater precentage of vaccinees developing antibodies in the 'high risk' group seems to indicate the presence of a low level of immunity in these vaccinees (not detected by antibody or skin testing before vaccination) which is 'boosted' by vaccination.

Table 2: Antibody responses to Q fever vaccine - differences between high and low risk groups

	High risk groups	Low risk groups
Antibody production	Positive response in: . about 80% of vaccinees within 3 months of vaccination; . about 60% of vaccinees after 20-60 months.	Positive antibody response in about 30% of vaccinees.
Cellular immunity (positive lymphocyte proliferation indices)	Approximately 80% positive. Response is maintained up to 5 years (the longest period tested so far).	

Vaccine use

Q fever vaccine is different from most other vaccines in that, as already emphasised, vaccinees who are already immune may suffer unpleasant local reactions. The immunity may be the result of a previous vaccination or it may result from a previous clinical or subclinical Q fever infection.

Many workers commencing work at an abattoir rapidly acquire immunity early in their career, and approximately 50% of workers have markers of immunity after 10 years in the abattoir.

Another major difference which affects the use of this vaccine is that markers of cell mediated immunity are more significant than those of humoral immunity. Potential vaccinees must therefore be screened for humoral and cell mediated immunity (skintest) before the vaccine is used. The use of the vaccine in people with either of these markers is contraindicated.

More detailed information on the use of the vaccine and on testing of prospective vaccinees is included in the Q fever vaccine product information appended to this article.

REFERENCE

1. Q fever from a stab wound. CDI 89/9 p1.

APPENDIX: Q FEVER VACCINE PRODUCT INFORMATION

(May 1989)

Description

The vaccine consists of a purified killed suspension of *Coxiella burnetii*, the cause of Q fever. It is prepared from Phase I Henzerling strain of *C. burnetii* grown in the yolk sacs

of embryonated eggs. The organisms are extracted, inactivated with formalin and freed from excess egg proteins by fractionation and ultracentrifugation. Thiomersal 0.01% w/v is added as a preservative.

Phase I vaccines have been shown to be highly antigenic and protective against challenge [1,2].

Immunology

Serological response to vaccination is chiefly IgM antibody to *C. burnetii* Phase I antigen. In weakly seropositive subjects it is largely IgG antibody to Phase I and Phase II antigens [3]. Although the seroconversion rate may be low (50% to 80%) cell mediated immunity develops [4] and the vaccine has been shown in field trails to be protective.

Indications

To protect susceptible individuals against infection with *C. burnetii.*

C. burnetii is a common infection in cattle, sheep and goats. The infection may be transmitted to man through the handling of infected tissue e.g. slaughtering of animals, washing of carcasses or by handling infected uterine or placental tissue. The organism is mainly transmitted by the respiratory route by droplet infection through aerosols, or after drying as infective dust.

Abattoir workers, veterinarians and laboratory workers handling potentially infected tissue should be considered for vaccination.

Contraindications

Persons shown to be immune by serological investigation or sensitive to the organism by skin test.

Persons sensitive to egg proteins.

Warnings

Currently no information is available concerning efficacy and safety of Q fever vaccine in immunodeficient or immuno-suppressed individuals.

Precautions

Prior to vaccination, persons must have serum antibody estimations and skin tests to exclude those likely to have hypersensitivity reactions to the vaccine.

Vaccination during the incubation period of the disease does not prevent the development of Q fever.

Serology

Antibody studies should be performed by complement fixation tests at serum dilutions of 1 in 2.5, 1 in 5 and 1 in 10 against Phase II antigen of *C. burnetii.*

Individuals positive at 1 in 2.5 or greater must not be vaccinated.

Skin test

Skin tests are performed by diluting 0.1 mL of vaccine in 30 mL of Sodium Chloride Injection. 0.1 mL of the diluted vaccine is injected intradermally into the volar surface of the forearm using methylated spirits as a skin cleansing agent (commercial isopropyl alcohol skin wipes are not satisfactory). A positive reaction is indicated by redness or induration at the site of injection after 72 hours. Individuals giving such a reaction must not be vaccinated.

Use in pregnancy

Safety of use in pregnancy has not been established in a positive sense. Benefits of vaccination should be weighed against potential risks. Q fever vaccine is inactivated and as with inactivated bacterial vaccines is not considered to be deleterious in pregnancy. It is however prudent not to vaccinate in pregnancy so as to avoid chance association of vaccination with complications of pregnancy.

Use during lactation

No information is available.

Use in paediatrics

No information is available.

Drug interactions

No information is available.

Adverse reactions

Vaccination of immune, or subjects hyperimmunised by repeated vaccination, may result in severe local or general reactions with local abscess formation in a few instances. Non-immune subjects commonly show local tenderness and erythema at the inoculation site. Local induration or oedema is rare. General symptoms are infrequent and include mild influenza-like symptoms, rarely fever, chills and minor sweating.

Dosage and administration

Shake the container gently before use. One dose of 0.5 mL is given by subcutaneous injection (NOT INTRAMUSCULAR) after ascertaining that serology and skin testing have been performed. There is no definite information concerning the duration of immunity produced by the vaccine, and until this information becomes available revaccination or booster doses of the vaccine are not recommended because of the risk of severe local adverse reactions.

Overdosage

No information is available.

Presentation

Q fever vaccine is available in ampoules of 0.5 mL containing 25 ug of antigen.

Storage

Store protected from light at 2 to 8°C. Do not freeze.

References

1. Fiset P. Vaccination against Q fever. Proceedings of First International Conference on vaccines against viral and rickettsial disease of man. Pan American Health Organization. 1967 Scientific Publication 147:528-31.
2. Ormsbee RA, et al. The influence of phase on the protective potency of Q fever vaccine. J Immunol 1964;92:404-12.
3. Worswick D, Marmion BP. Antibody responses in acute and chronic Q fever and in subjects vaccinated against Q fever. J Med Microbiol 1985;19:281-6.
4. Izzo AA, Marmion BP, Worswick DA. Markers of cell mediated immunity after vaccination with an inactivated whole cell Q fever vaccine. J Infect Dis 1988;157:781-9.

Manufacturer and distributor

Commonwealth Serum Laboratories
45 Poplar Road
Parkville Victoria 3052.
Australia

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 22/6/89 TO 5/7/89

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA) PMH(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)

		019	065	110	111	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	1	0	0	5	2	12	20
0102	ADENOVIRUS TYPE 2	0	0	1	0	1	0	0	0	2
0103	ADENOVIRUS TYPE 3	2	0	3	0	0	0	0	0	5
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	1	0	3	0	0	0	0	0	4
0107	ADENOVIRUS TYPE 7	1	0	0	0	0	0	0	0	1
0116	ADENOVIRUS TYPE 16	0	1	0	0	0	0	0	0	1
0119	ADENOVIRUS TYPE 19	1	0	0	0	0	0	0	0	1
0122	ADENOVIRUS TYPE 22	1	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	0	0	6	0	0	1	0	8
0201	INFLUENZA A VIRUS	0	0	2	0	0	0	0	0	2
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	1	0	0	0	0	1
0203	INFLUENZA B VIRUS	0	4	1	0	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	1	3	0	0	0	2	6
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	5	2	1	0	0	8	17
0303	PARAINFLUENZA VIRUS TYPE 3	3	1	2	4	0	1	0	1	12
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	1	0	0	0	7	8
0400	RESPIRATORY SYNCYTIAL VIRUS (R	16	5	180	62	22	15	37	93	430
0500	RHINOVIRUS (ALL TYPES)	5	2	11	6	2	0	0	2	28
0600	MYCOPLASMA PNEUMONIAE	2	1	15	5	4	2	0	0	29
0700	ORNITHOSIS-PSITTACOSIS	0	0	3	0	1	1	0	0	5
0900	COXSACKIEVIRUS GROUP B - NOT T	0	0	2	0	0	0	0	0	2
0904	COXSACKIEVIRUS B4	0	0	3	0	0	0	0	0	3
1009	ECHOVIRUS TYPE 9	3	0	0	0	0	0	0	0	3
1013	ECHOVIRUS TYPE 13	0	0	0	0	1	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	0	0	0	0	0	1	0	1
1030	ECHOVIRUS TYPE 30	1	0	0	0	0	0	1	0	2
1101	POLIOVIRUS TYPE 1	1	0	2	0	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	0	1	0	0	0	0	0	1
1200	MUMPS VIRUS	0	1	0	0	0	1	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	3	0	0	10	2	0	3	18
1301	HERPES SIMPLEX VIRUS - NOT TYP	5	6	0	0	0	0	0	0	11
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	13	9	0	1	4	3	0	30
1303	VARICELLA-ZOSTER VIRUS	1	7	2	0	1	1	0	4	16
1306	HERPES SIMPLEX TYPE 1	42	22	15	1	3	4	0	16	103
1307	HERPES SIMPLEX TYPE 2	55	37	15	0	7	18	0	20	152
1399	HERPES VIRUS TYPING PENDING	2	0	0	0	0	0	0	0	2
1401	COXIELLA BURNETII	3	0	0	0	0	1	0	0	4
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	0	16	16
1514	MOLLUSCUM CONTAGIOSUM	0	1	0	0	0	0	0	0	1
1521	MEASLES VIRUS	0	0	0	0	1	0	0	0	1
1522	RUBELLA VIRUS	1	1	4	0	1	0	0	0	7
1532	HEPATITIS B ANTIGEN	0	33	11	0	6	6	1	71	128
1535	HEPATITIS A ANTIBODY	0	2	5	0	0	1	0	0	8
1541	CHLAMYDIA A - C. TRACHOMATIS	11	33	0	0	10	0	0	27	81
1556	CMV - CYTOMEGALOVIRUS	42	13	5	5	2	1	3	10	81
1564	ROTAVIRUS	1	0	0	0	0	5	0	3	9
1599	ENTEROVIRUS TYPING PENDING	0	0	0	4	0	5	2	0	11
9992	ROSS RIVER VIRUS	6	8	3	0	0	0	0	6	23
TOTAL		209	194	305	100	74	73	51	301	1307

MALTA - COMMUNICABLE DISEASES INTELLIGENCE

VI - IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 22/6/89 TO 5/7/89

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	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	7	0	0	0	0	10	0	0	0	0	17
0102	ADENOVIRUS TYPE 2	0	1	0	0	0	0	1	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	0	4	0	0	0	0	0	0	0	0	0	4
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	1	0	0	0	0	0	0	0	0	0	1
0116	ADENOVIRUS TYPE 16	0	0	0	0	0	0	1	0	0	0	0	1
0122	ADENOVIRUS TYPE 22	0	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	4	0	0	0	1	0	0	0	0	0	5
0201	INFLUENZA A VIRUS	0	1	0	0	0	0	0	0	0	0	0	1
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	1	0	0	0	0	0	0	0	0	0	1
0203	INFLUENZA B VIRUS	1	2	0	0	0	0	0	0	0	0	0	3
0301	PARAINFLUENZA VIRUS TYPE 1	0	6	0	0	0	0	0	0	0	0	0	6
0302	PARAINFLUENZA VIRUS TYPE 2	0	17	0	0	0	0	0	0	0	0	0	17
0303	PARAINFLUENZA VIRUS TYPE 3	0	10	0	0	0	0	0	0	0	0	1	11
0399	PARAINFLUENZA VIRUS TYPING PEN	0	8	0	0	0	0	0	0	0	0	0	8
0400	RESPIRATORY SYNCYTIAL VIRUS (R	6	418	0	0	0	0	0	0	0	0	1	425
0500	RHINOVIRUS (ALL TYPES)	1	27	0	0	0	0	0	0	0	0	0	28
0600	MYCOPLASMA PNEUMONIAE	3	26	0	0	0	0	0	0	0	0	0	29
0700	ORNITHOSIS-PSITTACOSIS	0	2	0	0	0	0	0	0	0	0	1	3
0900	COXSACKIEVIRUS GROUP B - NOT T	0	2	0	0	0	0	0	0	0	0	0	2
0904	COXSACKIEVIRUS B4	0	3	0	0	0	0	0	0	0	0	0	3
1009	ECHOVIRUS TYPE 9	0	0	0	1	0	0	1	0	0	0	1	3
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	1	0	0	0	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	0	0	1	1	0	0	0	0	0	0	0	2
1101	POLIOVIRUS TYPE 1	0	2	0	0	0	0	1	0	0	0	0	3
1200	MUMPS VIRUS	1	0	0	0	0	0	0	0	0	1	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	1	0	0	0	0	0	0	0	5	7
1301	HERPES SIMPLEX VIRUS - NOT TYP	5	0	0	1	0	0	0	0	0	0	1	7
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	9	1	1	0	0	0	0	1	0	0	1	13
1303	VARICELLA-ZOSTER VIRUS	1	0	1	0	0	0	0	0	0	0	14	16
1306	HERPES SIMPLEX TYPE 1	7	11	0	0	0	0	0	0	0	1	52	71
1307	HERPES SIMPLEX TYPE 2	3	0	0	0	0	0	0	0	0	0	50	53
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	0	1	1
1401	COXIELLA BURNETII	1	0	0	0	0	0	0	1	0	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	7	0	0	2	1	6	0	0	0	0	16
1514	MOLLUSCUM CONTAGIOSUM	0	0	0	0	0	0	0	0	0	0	1	1
1522	RUBELLA VIRUS	3	0	0	0	0	0	0	0	0	0	3	6
1532	HEPATITIS B ANTIGEN	22	0	0	0	0	3	0	95	0	0	0	120
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	1	1	4	0	0	0	6
1541	CHLAMYDIA A - C. TRACHOMATIS	1	0	0	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	12	13	0	0	0	0	1	1	2	5	0	34
1564	ROTAVIRUS	0	0	0	0	0	0	9	0	0	0	0	9
1599	ENTEROVIRUS TYPING PENDING	0	5	0	0	0	0	5	0	0	0	0	10
9992	ROSS RIVER VIRUS	3	0	0	0	0	0	0	0	0	0	5	8
TOTAL		80	582	4	3	2	6	36	102	2	7	137	961

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 22/6/89 TO 5/7/89

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	0	0	0	0	3
0103	ADENOVIRUS TYPE 3	0	0	0	0	0	0	0	1	0	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	1	1	0	0	0	0	0	0	1	0	3
0119	ADENOVIRUS TYPE 19	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	1	1	1	0	3
0201	INFLUENZA A VIRUS	0	0	0	0	1	0	0	0	0	0	1
0203	INFLUENZA B VIRUS	0	0	0	0	2	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	1	0	0	0	0	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	1	2	0	2	5
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	1	0	1	0	2
1013	ECHOVIRUS TYPE 13	0	0	0	0	0	0	0	0	1	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	0	0	1	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	11	0	0	0	0	0	0	0	0	11
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	4	0	0	0	0	0	0	0	0	4
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	11	1	1	0	1	3	0	0	17
1306	HERPES SIMPLEX TYPE 1	0	26	0	0	0	0	0	1	5	0	32
1307	HERPES SIMPLEX TYPE 2	0	91	0	0	0	0	0	0	8	0	99
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	0	1
1401	COXIELLA BURNETII	0	0	0	0	0	0	0	2	0	0	2
1521	MEASLES VIRUS	0	0	0	0	0	0	0	1	0	0	1
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	0	1	0	1
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	8	0	8
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	1	1	0	2
1541	CHLAMYDIA A - C. TRACHOMATIS	0	77	0	0	0	3	0	0	0	0	80
1556	CMV - CYTOMEGALOVIRUS	0	3	0	0	1	0	4	3	36	0	47
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	0	1
9992	ROSS RIVER VIRUS	0	0	0	0	7	0	0	7	1	0	15
TOTAL		5	213	12	1	13	3	8	24	64	3	346