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Contents:

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1 524 reports were processed for this period.

Twenty four cases of Q Fever were reported, 3 from South Australia, 10 from New South Wales and 11 from Queensland. Occupational exposure data were only available for:

- two South Australian male cases, a 37 year old meatworker and a 39 year old abattoir worker, and
- two Queensland male cases, a 14 year old meatworker and 58 year old farmer.

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

Herpes simplex type 2 (HSV2) was isolated from anterior vulval and perianal swabs of a 24 year old AIDS patient with chronic genital lesions. Despite 2 weeks of treatment with intravenous acyclovir the patient remained febrile and was subsequently found to be excreting cytomegalovirus (CMV).

Cytomegalovirus (CMV) was isolated from:

- the urine and saliva of a 41 year old male AIDS patient receiving treatment with DHPG and AZT. DHGP treatment was subsequently ceased due to severe haematological toxicity.
- the saliva of a 56 year old male with cryptosporidial diarrhoea.

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Chlamydia trachomatis was isolated from the nasal aspirate of a 6 week old male with a two week history of a staccato cough. Chest x-ray indicated atelectasis of the lingula of the left lung.

Chlamydia psittaci was serologically detected in 2 females aged 35 and 11 respectively. Neither gave a history of exposure to birds.

Poliovirus type 2 was isolated from the nasal aspirate of a 1 year old female and a 10 month old male with hypogammaglobulinemia. Both patients had received Sabin vaccine (OPV) prior to the recognition of their immunocompromised status.

SEROLOGIC TESTING FOR ANTIBODY TO HUMAN IMMUNODEFICIENCY VIRUS (HIV) - UPDATE - USA
(based on MMWR Vol 36/No. 52, 8 January 1988)

Tests to detect antibody to human immunodeficiency virus (HIV), the virus that causes acquired immunodeficiency syndrome (AIDS), were first licensed by the Food and Drug Administration (FDA) in 1985, primarily as screening tests for blood and plasma donation. Since that time, millions of HIV antibody tests have been performed

. in laboratories of blood and plasma collection centres,

. in counselling and testing centres, and

. in clinical facilities as well as for purposes such as screening active duty military personnel and applicants for military service.

Assuring accurate test results requires continued attention to both the intrinsic quality of the tests and the performance of the technical personnel doing the tests.

Given the medical and social significance of a positive test for HIV antibody, test results must be accurate, and interpretations of the results must be correct. For these reasons, it has been emphasised that an individual be considered to have serologic evidence of HIV infection ONLY

- AFTER an enzyme immunosorbent assay (ELISA) screening test is repeatedly reactive,

  (The terms "reactive" or "non reactive" are used to describe serum or plasma specimens that give reactive or non reactive test results and to describe the test results from ELISA or Western blot (WB) tests before final interpretation.

  The terms "positive" and "negative" are used to describe the interpretation of ELISA test results indicating that the specimen tested is

  1) repeatedly reactive (positive) or
  2) non reactive or not repeatedly reactive (negative).

The terms "positive", "indeterminate", and "negative" are used to describe the interpretation of WB test results that indicate that the specimen tested is

. reactive with a specific pattern of bands (positive),

. reactive with a nonspecific pattern of bands (indeterminate), or

. non reactive (negative)

- AND another test such as Western blot (WB)

- OR immunofluorescence assay has been performed to validate the results[1].

N.B. Blood and plasma are not accepted for transfusion or further manufacture when the ELISA screening test is positive, regardless of the results of other tests that may be performed.

Licensed tests kits currently available in the United States for HIV antibody testing comprise seven (7) ELISAs and one (1) WB. All of these tests use HIV antigens derived from disruption of whole virus cultured in human-derived cell lines. In addition, many laboratories produce their own WB test reagents using viral antigen purchased from commercial sources. A variety of other test procedures are in use or under development or are being evaluated for licensure.

Criteria for interpretation of a reactive anti-HIV ELISA test are based on data from clinical studies performed under the auspices of each manufacturer. Since licensure of the first ELISA test kits in 1985, the manufacturers have worked to improve:

. the sensitivity (the probability that the test result will be reactive if the specimen is a true positive),

. the specificity (the probability that the result will be non reactive if the specimen is a true negative), and

. the reproducibility/reliability (the ability to replicate quantitative results with the same or similar test procedures on blindly paired samples)

of their assays:

- clinical data submitted by the manufacturers to FDA for licensure indicate that the sensitivity and specificity of the ELISA tests currently marketed in the United States are $> 99.0\%$.

- other laboratories performing comparative analyses of licensed anti-HIV ELISA test kits have found similar or slightly lower sensitivity and specificity[2-5].

- in routine use, both the sensitivity and specificity of the tests depend on the quality of testing in the laboratory.

- in addition, false-positive test results are observed when

non specific serologic reactions occur among uninfected persons who have immunologic disturbances or who have had multiple transfusions.

- false-negative test results are observed among persons who have recently become infected with HIV and who have not yet developed detectable antibody[6].

Repeating each initially reactive ELISA test increases the specificity of the test sequence by reducing the possibility that technical laboratory error caused the reactive result:

- in the American Red Cross Blood Services Laboratories, a specificity of approximately 99.8% has been consistently achieved during the screening of donated blood[7, unpublished data].

- however, in a population with a low prevalence of infection, even a specificity of 99.8% does not provide the desired predictive value (the predictive value of a positive or negative test is the probability that the test result is correct) for a positive test.

For this reason, it is particularly important not to rely solely on ELISA testing to determine whether a person is infected with HIV. Rather, ELISA test results should be validated with an independent supplemental test of high specificity conducted by a laboratory with high performance standards. In the United States, the validation test used most often is the WB. Some laboratories also use radio immunoprecipitation assays and indirect immunofluorescence assays.

For the licensed WB test, interpretation of reactive and non reactive tests is based on data from clinical studies submitted to FDA for licensure. The manufacturer states that, for a test to be considered positive with this WB, antibody must be reactive with multiple virus-specific protein bands, ie p24, p31, and either gp 41 or gp 160 (Table 1).

TABLE 1. Description of major gene products of human immunodeficiency Virus (HIV)

Gene Product*	Description
p 17	*gag* (core) protein
p 24	*gag* (core) protein
p 31	Endonuclease component of *pol* (polymerase) translate
gp 41	Transmembrane *env* (envelope) glycoprotein
p 51	Reverse transcriptase component of *pol* (polymerase) translate
p 55	Precursor of *gag* (core) proteins
p 66	Reverse transcriptase component of *pol* (polymerase) translate
gp 120	Outer *env* (envelope) glycoprotein
gp 160	Precursor of *env* (envelope) glycoprotein

* Number refer to molecular weight of the proteins in kilodaltons; measurement of molecular weight may vary slightly in different laboratories.

If fewer bands are present, the test is considered indeterminate; it is interpreted as negative only if no bands are present on the blot. When the manufacturer's stringent criteria are used for interpreting test results, the probability of either a false-positive or a false-negative result is extremely small:

. in clinical trials for licensure of this WB, however, as many as 15% to 20% of tests on persons at low risk for HIV infection were described as indeterminate.

. sera from persons recently infected with HIV also may produce an indeterminate WB pattern:

  - for such persons, a repeat WB on a second specimen obtained after the initial specimen often yields a positive blot pattern within 6 months;

  - conversely, follow-up testing of uninfected persons whose serum had an indeterminate blot pattern on initial testing usually will show no change in the banding pattern.

  - serum from some HIV-infected persons who have advanced immunodeficiency may have an indeterminant pattern because of a loss of antibodies to non-*env* proteins[8]

  - to reinstate donors with a history of a positive ELISA test, blood and plasma centres may use only results from the licensed WB test performed in the FDA-approved test sequence.

The performance characteristics of the unlicensed tests used by many laboratories whether WB, immunofluorescence assays, or other procedures, have not been uniformly subjected to the same rigorous scrutiny required for licensure by FDA.[9] Recommendations for standardisation have been published, but the extent to which these are followed is unknown. Information about production standards, inter-lot variability, or validation of criteria used for interpretation often is not available. Absence of standardisation and appropriate quality controls may result in a lower sensitivity or specificity and, thus, a higher probability of inaccurate results[10].

Despite the existence of a licensed WB test, many laboratories continue to use unlicensed WB tests because of cost and the stringent criteria required for interpreting the licensed test. The potential problems in using and interpreting unlicensed WB tests have been openly debated[11, 12]:

. although unlicensed WB tests can be highly accurate and reproducible when done with appropriate quality controls in laboratories with established performance standards[9], not all laboratories meet acceptable performance standards.

. 10 of 19 laboratories bidding for contracts to perform WB tests for the Department of Defense failed the required proficiency panel on one or more occasions[13].

. 2 of the laboratories satisfying the performance standards were awarded contracts by the U.S. Army. Both laboratories use well-validated techniques for WB that yield virus-specific bands at p17, p24, p31, gp41, p53, p55 and p64. The U.S. Army considers these WBs to be positive if bands are present either at gp41 or at both p24 and p55[4].

. in comparison with multiple validation procedures, WBs in these contract laboratories have an estimated specificity of 99.4%, and the laboratories have consistently performed accurately on all pre- and post-award quality assurance panels[14]. These and other laboratories have demonstrated that the achievable false-positive rate of sequentially performed ELISA and WB tests can be $<0.001\%$ ($<1/100,000$ persons tested)[13,15].

The College of American Pathologists (CAP), in conjunction with the American Association of Blood Banks, conducts an open proficiency testing program for laboratories performing HIV antibody tests (The laboratories know that the samples have been supplied for proficiency testing):

. each quarter, more than 600 laboratories that participate voluntarily report results from testing five coded samples of plasma that have various known levels of anti-HIV reactivity or that are non reactive.

. in the CAP survey conducted in October 1987, the result of ELISA tests at the participating laboratories correlated well with results from the referee laboratories (Table 2).

TABLE 2. Comparison of responses by referee and participant laboratories on samples tested for anti-HIV by enzyme immuno assay (ELISA), by sample number-College of American Pathologists Proficiency testing, 1987

Sample Number	Reactivity	Percentage of Laboratories Reporting correct Results: Referee Laboratory*	Participant Laboratory**
W-21	Reactive	100.0	99.8
W-22#	Nonreactive	80.0	51.4
W-23+	Reactive	100.0	99.5
W-24+	Reactive	100.0	100.0
W-25	Nonreactive	100.0	98.3

* Results reported by 15 laboratories selected because of extensive experience and excellent long-term performance in proficiency testing programs.

** Results reported by 601 other laboratories that voluntarily participated.

\# Sample W-22 was prepared with a pool of processed plasma that caused an artefactual nonspecific reaction with one ELISA test kit.

+ Samples W-23 and W-24 were identical.

. - for the three reactive samples (W-21, W-23, W-24), correlation ranged from 99.5% to 100%.

- for the single non reactive sample that could be adequately evaluated (W-25), correlation was 98.3%.

- the non reactive W-22 sample that was sent with the October 1987 serum panel had been prepared with a pool of processed plasma that caused an unexplained, nonspecific reaction with one of the ELISA test kits. Consequently, the ELISA results for this sample could not be evaluated.

The individual participating laboratories used their own criteria for interpreting WB results:

. WB results for two of the three reactive specimens were reported as indeterminate by one referee laboratory each, while results for the two non reactive specimens in the CAP survey were reported correctly by all 10 referee laboratories (Table 3).

TABLE 3. Comparison of responses on samples tested for anti-HIV by Western blot (WB) by referee and participant laboratories*, by sample number - College of American Pathologists Proficiency Testing, 1987

		Interpretation of WB Test Results (Percentage of Responses)					
		Postive Test		Indeterminate Test		Negative Test	
Sample Number	Reactivity	Referee Laboratory	Participant Laboratory	Referee Laboratory	Participant Laboratory	Referee Laboratory	Participant Laboratory
W-21	Reactive	100.0	100.0	0.0	0.0	0.0	0.0
W-22	Nonreactive	0.0	1.6	0.0	4.9	100.0	93.4
W-23	Reactive	90.0	80.8	10.0	15.1	0.0	4.1
W-24	Reactive	90.0	84.9	10.0	12.3	0.0	2.8
W-25	Nonreactive	0.0	0.0	0.0	5.6	100.0	94.4

* Results reported by the 10 referee and 73 participant laboratories that performed both ELISA and WB tests.

. One of the 73 participating laboratories reported a non reactive sample (W-22, the sample that gave artefactual reactions with one of the ELISA test kits) as reactive, while approximately 5% reported the two non reactive samples as indeterminate, and 12% to 15% reported two of three reactive specimens as indeterminate.

For the three reactive samples, the results of 241 repeatedly reactive ELISA tests could be compared with WB results (Table 4).

TABLE 4. Relationship between results on samples tested for anti-HIV by enzyme immunosorbent assay (ELISA) and Western blot (WB), by sample number - College of American Pathologists Proficiency Testing, 1987.

Sample Number	Reactivity	Results by ELISA*		Results by WB*		
		Positive	Negative	Positive	Indeterminate	Negative
W-21	Reactive	76	0	76	0	0
W-23	Reactive	83	0	69	13	1
W-24	Reactive	82	0	70	10	2
W-25	Nonreactive	0	58	0	3#	55
TOTAL		**241**	**58**	**215**	**26**	**58**

* Number of responses reported by both referee and participant laboratories. Samples W-22 was excluded because of an artefact of the sample.

\# - One sample by WB had only p24 bands reported;

- one sample had both p24 and p32 bands reported; and
- one sample had no bands reported.

. - for 215 (89.2%) of these, the WB tests were reported as positive;
- for 23 (9.5%), the WBs were reported as indeterminate; and
- for 3 (1.2%), the WBs were reported as negative.

. Of 58 WB results performed on non reactive samples found non reactive by ELISA, 55 (94.8%) were reported as negative by WB, and 3 (5.2%) were reported as indeterminate.

None of the non reactive samples were read as positive by WB.

Because criteria used to interpret WB varied by laboratory, banding patterns reported in the 299 WB tests conducted in the October 1987 survey were examined (Table 5).

TABLE 5 Distribution and interpretation of HIV-specific protein band patterns on Western blot* (WB) - College of American Pathologists Proficiency Testing, 1987.

	WB as Interpreted by Referee and Participant Laboratories					
	Positive		Indeterminate		Negative	
HIV-specific bands+	No.	(%)	No.	(%)	No.	(%)
None	0	(0.0)	9	(7.1)	118	(92.9)
Single band	18	(60.0)	9	(30.0)	3	(10.0)
gag	6	(42.9)	7	(50.0)	1	(7.1)
pol	0	(0.0)	2	(100.0)	0	(0.0)
env	12	(85.7)	0	(0.0)	2	(14.3)
Multiple bands	208	(96.7)	4	(1.9)	3	(1.4)
gag, pol	8	(80.0)	1	(10.0)	1	(10.0)
gag, env	125	(98.4)	0	(0.0)	2	(1.6)
pol, env	2	(40.0)	3	(60.0)	0	(0.0)
gag, pol, env	73	(100.0)	0	(0.0)	0	(0.0)
TOTAL	**226**	**(60.8)**	**22**	**(5.9)**	**124**	**(33.3)**

* Samples tested and reported include reactive samples W-21, W-23, and W-24 and non reactive samples W-22 and W-25.

\+ Bands may be any proteins or glycoproteins that are products of the genes listed. HIV-specific gene products are shown in Table 1.

- two or more virus-specific protein bands were reported in 215 blots, 208 (96.7%) of which were interpreted as positive.
- 18 (60.0%) of 30 blots with only a single virus-specific protein band were considered positive.
- when the single protein band was from the *env* gene, 12 (85.7%) of 14 were read as positive.

These data demonstrate that different laboratories may report different WB results for samples with the same banding patterns.

Results of CAP proficiency tests for more than 500 laboratories participating in the 1986 and 1987 surveys indicate the following performance for the anti-HIV ELISA test:

- of 6,946 tests on reactive samples, 99.5% were reported as positive
- of 1,142 tests on non reactive samples, 98.3% were interpreted as negative.

Based on results from 601 laboratories on a pair of identical reactive samples (W-23 and W-24), reproducibility was 99.5%.

For the WB test, calculations were based only on positive or negative results divided by the total number of tests in the October 1987 CAP survey (Table 4):

. for the reactive samples, 89.2% of 241 results were correctly interpreted as positive, and

. for the non reactive samples, 94.8% of 58 results were correctly interpreted as negative.

Reproducibility, which was based on 83 tests on a pair of identical reactive samples (W-23 and W-24), was 95.2%. The performance of the referee laboratories was more accurate for the WB than was the performance of the participating laboratories. The performance of the licensed and unlicensed WB tests could not be compared because the data were not collected.

MMWR Editorial Note:

Quality laboratory testing for HIV antibody is a critically important element for surveillance and detection of HIV infection. The laboratory testing process requires quality assurance for each step including:

1. collection, labelling, and transport of specimens;
2. laboratory reagents and procedures;
3. interpretation of analytical results; and
4. communication from the laboratory scientist to the clinician and then to the person being tested.

Quality performance is promoted by using licensed or standardised tests in proper sequence and by developing consensus about interpretation of analytical results.

Proficiency testing benefits participating laboratories by identifying with particular types of samples, with particular tests, or with interpretation of results. However, results of proficiency testing programs should be interpreted cautiously. Data from proficiency testing measure only the operational performance of participating laboratories but cannot be used to measure the sensitivity or specificity of a given test. Samples provided for testing in the HIV antibody surveys may be pooled human plasma samples with known levels of anti-HIV reactivity, or they may be dilutions of a single reactive plasma sample in HIV-negative serum. They are rarely fresh serum specimens from a person who is or is not infected with HIV. Some samples are selected because they exhibit non specific reactivity or are otherwise difficult to test and interpret; they are not typical of the vast majority of specimens that will be handled by the participating laboratories. For instance, in normal practice, samples W-22 and W-25 would not be tested by WB because the ELISA was non reactive. The non specific reactivity of the type that occurred with specimen W-22 cannot always be predicted; a similar unexplained nonspecific reaction occurred in a proficiency testing program conducted by the Centers for Disease Control [167] (CDC) and with several samples used by the American Association of Bioanalysts (unpublished data).

The number of specimens commonly used in proficiency testing programs (five in each CAP survey) sent to each laboratory also limits the application of survey results. This number of specimens is not sufficient to measure adequately the performance of any single laboratory. The number of specimens tested per month in different laboratories varies enormously, and no attempt is made in the survey to select a representative sample of laboratories performing the test; those that choose to participate in the survey do so voluntarily.

Laboratories in the surveys reported indeterminate WB results on some reactive and non reactive samples. An indeterminate result is not a final result; it requires additional laboratory testing on the same specimen and often entails asking the person from whom the specimen was obtained to provide one or more additional specimens. The final interpretation of an indeterminate result frequently will also require additional epidemiologic, clinical or corroborating laboratory information.

Even among the diverse laboratories participating in the CAP survey, none performing the ELISA and WB tests in sequence would have reported false-positive test results. However, performance and interpretation of WB tests vary among laboratories. The US Public Health Service is convening a meeting to address these issues. A nationwide performance evaluation program for HIV antibody testing has been started by CDC's Training and Laboratory Program Office and Center for Infectious Diseases [11]. The first sample shipment, consisting of reference materials, was mailed in November 1987 to more than 700 participating US laboratories.

The predictive values of both positive and negative test results for HIV antibody are extremely high in laboratories that have good quality control and high performance standards and that use licensed ELISA tests and the licensed WB or other well-standardised tests. Physicians or other health-care providers who request HIV antibody tests and who counsel persons about test results must have a clear understanding of the significance of the test results and the potential pitfalls of the testing process. When test results are indeterminate or inconsistent with other information, additional information should be obtained to try to confirm whether the person is infected with HIV. The counselling procedure should include a careful assessment of the person's potential risks or exposures to HIV. As for all medical tests, results should be interpreted in concert with the historic, epidemiologic, clinical, and other pertinent laboratory information available.

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HEPATITIS A EPIDEMIC - CHINA

An epidemic of hepatitis A is occurring in Shanghai and extending to the Jiangsu and Zhejiang provinces of the People's Republic of China. Twenty three thousand (23,000) cases have been officially reported in Shanghai. "Hairy clams" from an area of the Jiangsu province have been implicated as the source of the current epidemic.

Editorial Comment

Hepatitis A, an enteric virus, is acquired predominantly by the ingestion of contaminated food and water. The prevalence of infection, and hence IgG antibody to HAV, is inversely related to standards of sanitation. Almost universal exposure is seen before adulthood in the developing countries, with more than 75% of children from parts of Asia, Africa, India, certain Mediterranean countries and South America having IgG antibody to HAV by the age of five years.

In countries of Northern Europe the prevalence of IgG antibody to HAV in children and young adults has declined over the years as a direct consequence of improved hygiene standards.

Hepatitis A spread can be controlled by the strict enforcement of hygiene measures designed to limit faecal-oral transmission of the virus eg appropriate care with food preparation, selection of water sources, faeces disposal, handwashing practices; and by the isolation of cases using enteric precautions.

Individuals at risk of hepatitis A can be protected by the use of normal immunoglobulin.

This preparation is safe and at a dose of 0.02 mL/kg body weight given by deep intramuscular injection offers a protective efficacy of up to 80%. Immunoglobulin can also protect against or attenuate the illness if given up to two weeks following a definite exposure, which is surprising for a virus with an average incubation period of 3-4 weeks.

AIDS UPDATE - CANADA

(based on an update report from the Federal Centre for AIDS, Ottawa, Canada, 4 January 1988)

To 4 January 1988, 1 464 cases (1 438 adults and 26 paediatric) of AIDS which meet the surveillance case definition for AIDS (revised September 1, 1987) have been reported to the Federal Centre for AIDS, Ottawa. The distribution of those patients by Province of notification (Table 1), by age group (Table 2) and by risk category (Table 3) are shown below:

TABLE 1: AIDS cases by Province of notification

PROVINCE	CASES	DEATHS	TOTAL	(%)
British Columbia	139	164	303	20.7
Alberta	33	51	84	5.7
Saskatchewan	7	12	19	1.3
Manitoba	13	12	25	1.7
Ontario	309	274	583	39.8
Quebec	181	233	414	28.3
New Brunswick	2	6	8	0.5
Nova Scotia	13	8	21	1.4
Prince Edward Island	0	1	1	0.1
Newfoundland	3	2	5	0.3
North West Territories	1	0	1	0.1
Yukon	0	0	0	0.0
TOTAL	701 (47.9%)	763 (52.1)	1464 (100%)	100%

TABLE 2: AIDS cases by sex and age groups

AGE (YEARS)	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
0-14	13	13	26	*	*	19
15-19	4	0	4	2	0	2
20-29	274	25	299	130	13	143
30-39	636	23	659	318	14	332
40-49	322	7	329	174	5	179
50+	129	16	145	79	9	88
Unknown	2	0	2	0	0	0
TOTAL	1380	84	1464	703	41	763

* Death breakdown by sex not avaialble

TABLE 3: AIDS by risk category

RISK GROUP	CASES	DEATHS
ADULTS	1438	744
Homo-/Bi-sexual	1175	585
IV drug user	9	7
Home-/Bi-sexual IV drug user	42	22
Blood transfusion recipient*	60	40
Heterosexual activity+	110	68
None of the above	42	22
PAEDIATRIC	26	19
Perinatal transmission	22	#
Blood transfusion recipient	4	#

* Includes cases with haemophilia.
\+ This includes 73 persons (52 deaths) originating or residing in coutries with a high prevalence of HIV and where heterosexual transmission of HIV is common.
\# Figures not available.

PREVENTION OF HAEMOPHILUS INFLUENZAE TYPE B DISEASE - UPDATE, UNITED STATES
(based on MMWR Vol. 37/No. 2, 22 January 1988)

Haemophilus b Conjugate Vaccine (Diphtheria Toxoid - Conjugate) has recently been licensed for use in children 18 months of age or older for the prevention of Haemophilus influenzae type b (Haemophilus b) disease. This vaccine consists of Haemophilus b capsular polysaccharide covalently linked to diphtheria toxoid (conjugate vaccine).

A previously developed vaccine consisting of the Haemophilus b capsular polysaccharide alone (polysaccharide vaccine) was shown to be effective in Finnish children over 24 months of age [1] the age group in which approximately 20% of all invasive Haemophilus b infections among U.S. children less than 5 years of age can be expected to occur [2]. A similar, but not identical, polysaccharide vaccine was licensed for use in the United States in April 1985 on the basis of data demonstrating biochemical characteristics and immunogenicity comparable to the vaccine used in the original Finnish trial [3]. In that Finnish trial, polysaccharide vaccine was not effective in children less than 18 months of age. Because of the small sample size, efficacy could not be demonstrated in children 18 to 23 months of age. Polysaccharide vaccine was immunogenic (as measured by antibody production) in children 18 to 23 months old, but less so than it was in older children [1].

Conjugate vaccine was developed with the ultimate goal of providing an effective vaccine for infants and younger children:

- Preliminary data from a new Finnish study suggest that conjugate vaccine was 87% effective in preventing Haemophilus b disease when administered in a three-dose regimen to infants 3 to 6 months of age [4].

- However, licensure of conjugate vaccine for use in infants in the United States cannot be considered until this and other efficacy trials are further evaluated.

- Since antibody production after vaccination with conjugate vaccine in children 18 months of age or older is substantially greater than that after vaccination with polysaccharide vaccine, conjugate vaccine has been licensed for use in these children.

SAFETY

- When conjugate vaccine alone was given to over 1,000 adults and children, no serious adverse reactions were observed [5-12].

- When conjugate vaccine was given with diphtheria and tetanus toxoid and pertussis vaccine (DTP) and inactivated polio vaccine (IPV) to 30,000 infants, the rate and extent of serious adverse reactions did not differ from those seen when DPT was administered alone [4].

. In one study of 500 children 15 to 24 months of age, no significant difference in local or systemic side effects occured between groups of children vaccinated with either polysaccharide vaccine or conjugate vaccine[7].

. Local reactions were noted for:

- 10.3% of children receiving polysaccharide vaccine; and
- 12.5% of children receiving conjugate vaccine,

while moderate fever (temperature 39.0°C) occured in:
- 1.4% of children vaccinated with polysaccharide vaccine; and
- 0.7% of children vaccinated with conjugate vaccine.

IMMUNOGENICITY

In several studies using different regimens of vaccine administration, conjugate vaccine has shown greater immunogenicity than polysaccharide vaccine[5-9, 11, 12]:

. Response to a single dose of either polysaccharide vaccine or conjugate vaccine in children 15 to 24 months of age was specifically addressed in a randomised, double-blind study recently completed in the United States.

. More than 90% of children vaccinated with conjugate vaccine responded with antibody levels considered to be protective (0.15 ug/mL), whereas less than 50% of children vaccinated with polysaccharide vaccine has such a response.

. Over 60% of children vaccinated with conjugate vaccine, but less than 30% of those vaccinated with polysaccharide vaccine, produced levels of antibody considered to be indicative of long-term protection (1.0 ug/mL).

N.B. It should be noted that three of four lots of polysaccharide vaccine used in this study had been heat-sized, a process which may reduce immunogenicity. However, children receiving non-heat-sized polysaccharide vaccine also had post-immunisation levels of antibodies to Haemophilus b polysaccharide that were lower than those observed in children vaccinated with conjugate vaccine.

In another study in which vaccine recipients were tested at 1 month and again at 1 year after completion of the immunisation series, 9- to 15-month-old children who had received two doses of conjugate vaccine had significantly higher titres of antibody to Haemophilus b polysaccharide than did similar children who had received two doses of non-heat-sized polysaccharide vaccine[5].

. Children given conjugate vaccine at 15 to 24 months of age has significantly higher levels of antibody to Haemophilus b polysaccharide 1 year after vaccination than did children receiving polysaccharide vaccine[8].

. Conjugate vaccine recipients responded to a booster dose of either polysaccharide vaccine or conjugate vaccine with higher geometric mean antibody levels than did those initially vaccinated with polysaccharide vaccine[8].

In another study, children with sickle cell syndromes who received conjugate vaccine had higher postvaccination levels of antibody to Haemophilus b polysaccharide [13] than did similar children given polysaccharide vaccine. The studies to date showing increased immunogenicity in children less than 18 months of age [5, 6, 9, 11] suggest that conjugate vaccine may be functioning as a T-cell dependent antigen. This finding contrasts with the lack of immunogenicity in infants and the absence of immunologic memory characteristic of T-cell independent polysaccharide vaccines.

BIOLOGICAL ACTIVITY

Several investigators have demonstrated that conjugate vaccine produces functional activity against Haemophilus b similar to that produced by polysaccharide vaccine:

- In one randomised, double-blind study, adults vaccinated with conjugate vaccine had serum bactericidal titres for Haemophilus b at least as high as those of adults receiving polysaccharide vaccine [12].

- In addition, sera from adults vaccinated with conjugate vaccine were protective in an infant rat model of Haemophilus b disease, whereas similarly diluted sera from persons receiving polysaccharide vaccine showed no protective activity.

- In a separate study, sera from 9- to 14- month-old children given conjugate vaccine showed greater opsonic activity against Haemophilus b organisms than did sera from children vaccinatd with polysaccharide vaccine [14].

Both studies showed a correlation between functional activity and serum levels of antibody to Haemophilus b polysaccharide and suggest that antibody produced in response to conjugate vaccine is biologically equivalent to that produced in response to polysaccharide vaccine.

IMMUNISATION PRACTICES ADVISORY COMMITTEE (ACIP) RECOMMENDATIONS

1. The ACIP recommends that all children receive conjugate vaccine at 18 months of age. The efficacy of conjugate vaccine in children 18 months of age or older has not been determined in field trials. However, studies comparing antibody production in children receiving conjugate vaccine with that in children receiving polysaccharide vaccine suggest that conjugate vaccine is likely to be more effective than polysaccharide vaccine. The ACIP therefore recommends the use of conjugate vaccine in all children vaccinated against Haemophilus b disease.

2. While the duration of immunity after a single dose of conjugate vaccine is unknown at this time, it is expected to be at least 1.5 to 3 years. Until further information is available, revaccination is not recommended for children receiving conjugate vaccine at 18 months of age or older.

3. Vaccination of children more than 24 months of age who have not yet received Haemophilus b vaccine should be based on risk of disease. Children considered at high risk for Haemophilus b disease, including

   . those attending day-care centres,

   . those with anatomic or functional asplenia (ie, sickle cell disease or splenectomy), and

   . those with malignancies associated with immunosuppression,

   should receive the vaccine. Although the risk of disease decreases with increasing age, physicians may wish to vaccinate previously healthy children between 2 and 5 years of age to prevent disease that can occur in this group.

4. Because many children who received polysaccharide vaccine between the ages of 18 and 23 months may have had a less than adequate response to the vaccine, they should be revaccinated with a single dose of conjugate vaccine. Revaccination should take place a minimum of 2 months after the initial dose of polysaccharide vaccine.

5. There is no need to routinely revaccinate children who received polysaccharide vaccine at 24 months of age or older.

6. Children who had invasive Haemophilus b disease when they were less than 24 months of age should still receive the conjugate vaccine according to the above recommendations since most children less than 24 months of age fail to develop adequate immunity following natural infection[15].

7. Although increases in serum diphtheria anti-toxin levels can follow administration of conjugate vaccine, this vaccine should not be considered an immunising agent against diphtheria. No changes in the schedule for administration of diphtheria toxoid, customarily given as DTP, should be made secondary to the use of conjugate vaccine.

8. Vaccination with either polysaccharide vaccine or conjugate vaccine probably does not inhibit asymptomatic carriage of Haemophilus b organisms. Although vaccinated children may be protected from invasive disease, they may pass the organism on to susceptible children. In addition, no vaccine is 100% effective. Therefore, chemoprophylaxis of household or day-care contacts of children with haemophilus b disease should be directed at vaccinated as well as unvaccinated contacts. Because of the length of time necessary to generate an immunologic response to the vaccines, vaccination does not play a major role in the management of patients with Haemophilus b disease or their contacts. Vaccine may be given to previously unvaccinated children of appropriate age to provide protection against future exposure.

9. Conjugate vaccine and DTP may be given simultaneously at different sites. Data are lacking on concommitant administration of conjugate vaccine and

measles-mumps-rubella (MMR) or oral polio (OPV) vaccines. However, if the recipient is unlikely to return for further vaccination, simultaneous administration of all vaccines appropriate to the recipient's age and previous vaccination status is recommended (including DTP, OPV, MMR and conjugate vaccine).

CDI Editorial Comment

Most Haemophilus influenzae type b infections occurs in children under 5 years of age with meningitis being the most common clinical presentation in this age group.

In Europe, the following incidences of H. influenzae meningitis have been reported in this age group:

. England	11 -	18/100,000 population/year
. Netherlands		22
. Finland		27
. Sweden		27

The age specific incidence in this group varied between 19 and 71 per 100,000 population per year in the general community in the United States whilst a higher incidence of 173-409/100,000/year was observed in American Indians and Eskimo groups.

The incidence of Haemophilus influenzae type b meningitis is unknown in Australia. However, the number of paediatric cases presenting to the Royal Children's Hospital in Melbourne averaged around 40 cases per year for the past two years. The age breakdown of those cases was as follows:

. 0-18 months	account for	55.4% of cases
19-24 months	account for	20.5% of cases
25-60 months	account for	21.7% of cases
61 months and over	account for	2.4% of cases

The prospects for prevention of Haemophilus influenzae type b disease by immunisation depend heavily on the availability of an appropriately efficacious vaccine[16]. The original polysaccharide (PS) capsule of H. influenzae type b (Hib) is safe and effective in preventing invasive Hib disease in children two years of age and older, but it is ineffective in younger children, the group at greatest risk of disease. The PS vaccine also may be ineffective in preventing disease in certain subgroups of the population that are genetically at increased risk of disease and show impaired antibody responses to immunisation. Thus, new strategies need to be considered. Currently, several new Hib PS-protein conjugate vaccines are being evaluated. These vaccines differ in their method of preparation, carrier protein, and PS size. In contrast to the plain Hib PS vaccine, conjugate vaccines are immunogenic in infants and elicit boostable increases in antibody to PS upon re-injection of vaccine. However, some infants less than six months of age do not respond. To confer protection on all infants, it may be necessary to modify further the conjugate vaccines. One approach involves using the outer membrane proteins (OMPs) as vaccine components:

. five major OMPs have been purified from Hib, and

. three, P1 (50 kilodalton [kDa]), P2 (37 kD2), and P6 (16kDa)

which contain antigens capable of eliciting strain-specific protective antibodies in experimental animals. In summary, PS-protein conjugate vaccines hold enormous promise for the prevention of Hib disease in infants, but further improvement is needed to define the optimal carrier protein, PS size, and method of coupling. Information is also needed on whether genetic factors influence responses to these vaccines.

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RIFT VALLEY FEVER AND YELLOW FEVER IN MAURITANIA

The Centers for Disease Control (CDC), Atlanta, Georgia, has in its Memorandum No 92 advised that in mid-October 1987, the Government of the Islamic Republic of Mauritania was notified of suspected human yellow fever in the region of the Senegal River Basin. The epicentre of the outbreak was the town of Ross in South Western Mauritania, on the border with Senegal, but the epidemic was geographically widespread.

Virologic investigation by the Institut Pasteur in Dakar led to the isolation of:

. yellow fever virus from some suspected cases, but also

. Rift Valley fever virus from over 100 patients.

Despite very limited information:

. an epidemic of Rift Valley fever has apparently been occurring in Southern Mauritania, and

. yellow fever has also occurred; there has been significant yellow fever activity in West Africa in 1986 and 1987, including Nigeria and Mali.

Rift valley fever is an arbovirus disease transmitted predominantly by culicine mosquitoes. Infection may result

from contact with infected animals, including their products of conception, eg, the foetus, placenta, etc. Travellers should be:

- cautioned about travel to Southern Mauritania and Northern Senegal until this outbreak is over;
- advised to take precautions to avoid mosquito bites by wearing long-sleeved clothes, applying insect repellent, and using mosquito nets when sleeping; and
- informed that unnecessary contact with livestock should be avoided since the persons most at risk are those visiting rural areas or working with domestic animals and livestock.

Yellow fever vaccinations are:

- recommended for travellers to countries where there is probable or recognised current yellow fever transmission; and
- required from all persons over 1 year of age arriving in Australia who have within the previous six (6) days been in the infected parts of any country listed in CDI 87/23 page 7 (Human Quarantine - Draft Policy Statement - Yellow Fever Vaccination Certificate Requirements).

NSW DRUG INFORMATION SERVICE

Please note the following changes:

Telephone Numbers: **553 2256**
553 2261
553 2361

Postal Address: **P.O. Box 750**
Kogarah 2217

The hours of service remain the same:

8.30am to 5.00pm, Monday to Friday
Closed public holidays.

Recorded messages available after hours -
telephone 553 2361.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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

Period 25/1/88 to 7/2/88

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)

		019	065 *	110	111	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	1	7	1	6	16	7	0	13	51
0101	ADENOVIRUS TYPE 1	0	3	2	0	0	0	0	0	5
0102	ADENOVIRUS TYPE 2	2	0	0	0	1	0	0	0	3
0103	ADENOVIRUS TYPE 3	2	1	0	0	0	0	0	0	3
0104	ADENOVIRUS TYPE 4	2	0	0	0	0	0	0	0	2
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	0	12	0	0	0	0	0	0	12
0199	ADENOVIRUS TYPING PENDING	0	0	0	5	0	0	0	0	5
0201	INFLUENZA A VIRUS	1	0	0	0	9	0	0	1	11
0203	INFLUENZA B VIRUS	0	0	2	0	26	0	0	0	28
0301	PARAINFLUENZA VIRUS TYPE 1	0	1	0	2	0	0	0	1	4
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	2	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	1	2	5	5	5	0	0	1	19
0400	RESPIRATORY SYNCYTIAL VIRUS (R	3	1	0	1	1	0	1	0	7
0500	RHINOVIRUS (ALL TYPES)	5	7	1	4	0	1	0	5	23
0600	MYCOPLASMA PNEUMONIAE	16	9	4	9	112	0	2	26	178
0700	ORNITHOSIS-PSITTACOSIS	5	0	3	0	2	0	0	0	10
0809	COXSACKIEVIRUS A9	5	0	0	0	0	0	0	0	5
0816	COXSACKIEVIRUS A16	2	0	0	0	0	0	0	0	2
0821	COXSACKIEVIRUS A21	1	0	0	0	0	0	0	0	1
0901	COXSACKIEVIRUS B1	0	1	1	0	0	0	0	0	2
0902	COXSACKIEVIRUS B2	1	0	0	0	0	0	0	0	1
0905	COXSACKIEVIRUS B5	3	0	0	0	0	0	0	0	3
1018	ECHOVIRUS TYPE 18	1	1	0	0	0	0	0	0	2
1022	ECHOVIRUS TYPE 22	0	0	1	0	0	0	0	0	1
1024	ECHOVIRUS TYPE 24	0	1	0	0	0	0	0	0	1
1027	ECHOVIRUS TYPE 27	0	1	0	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	4	0	0	4
1102	POLIOVIRUS TYPE 2	0	0	1	0	0	0	2	0	3
1200	MUMPS VIRUS	0	0	0	0	4	0	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	2	2	0	0	23	0	0	1	28
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	4	2	0	0	0	4	0	11
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	10	8	3	0	3	0	2	23	49
1303	VARICELLA-ZOSTER VIRUS	5	2	1	2	5	0	0	2	17
1306	HERPES SIMPLEX TYPE 1	64	37	32	0	27	9	0	52	221
1307	HERPES SIMPLEX TYPE 2	72	55	10	0	49	17	0	63	266
1399	HERPES VIRUS TYPING PENDING	0	0	0	3	0	0	0	0	3
1401	COXIELLA BURNETI	0	0	3	0	10	0	0	11	24
1402	OTHER RICKETTSIAE	0	0	0	0	0	0	0	1	1
1502	PICORNIA VIRUS - NOT TYPED = E	0	3	0	0	24	12	0	19	58
1521	MEASLES VIRUS	7	0	0	1	1	0	0	0	9
1522	RUBELLA VIRUS	21	0	2	3	3	0	0	12	41
1532	HEPATITIS B ANTIGEN	12	28	2	1	24	11	0	14	92
1535	HEPATITIS A ANTIBODY	2	5	0	0	2	1	0	1	11
1541	CHLAMYDIA A - C. TRACHOMATIS	16	63	38	0	16	0	0	23	156
1556	CMV - CYTOMEGALOVIRUS	24	9	4	2	10	0	4	12	65
1563	CORONAVIRUS	0	0	0	0	1	0	0	0	1
1564	ROTAVIRUS	2	2	7	5	7	2	1	0	26
1599	ENTEROVIRUS TYPING PENDING	0	0	0	11	0	5	3	0	19
9901	ARBO. GROUP A. (UNSPECIFIED)	0	0	1	0	0	0	0	0	1
9902	POXVIRUS GROUP NOT TYPED	1	0	0	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	0	5	0	0	0	1	0	20	26
9993	ASTROVIRUS	0	0	0	0	1	0	0	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	1	0	0	0	1
9995	DENGUE	0	0	0	0	0	0	0	1	1
9998	ARBO. GROUP B. (UNSPECIFIED)	0	0	0	0	0	0	0	1	1
TOTAL		290	271	126	62	383	70	19	303	1524

* Princess Margaret Hospital for Children - 12 reports

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

Period 25/1/88 to 7/2/88

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 TRA CT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	16	0	0	0	19	0	3	0	1	39
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	3	0	0	0	0	4
0102	ADENOVIRUS TYPE 2	0	2	0	0	0	1	0	0	0	0	3
0103	ADENOVIRUS TYPE 3	0	0	0	0	0	1	0	0	0	2	3
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	[redacted]
0199	ADENOVIRUS TYPING PENDING	0	3	0	0	0	0	0	0	0	0	3
0201	INFLUENZA A VIRUS	0	6	0	0	0	0	0	1	1	1	9
0203	INFLUENZA B VIRUS	1	14	0	0	0	0	0	1	0	0	16
0301	PARAINFLUENZA VIRUS TYPE 1	0	4	0	0	0	0	0	0	0	0	4
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	18	0	0	0	0	0	0	0	0	18
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	7	0	0	0	0	0	0	0	0	7
0500	RHINOVIRUS (ALL TYPES)	1	20	0	0	0	0	0	0	0	0	21
0600	MYCOPLASMA PNEUMONIAE	12	139	0	0	0	0	0	2	0	1	154
0700	ORNITHOSIS-PSITTACOSIS	3	5	0	0	0	0	0	0	0	0	8
0809	COXSACKIEVIRUS A9	0	0	0	5	0	0	0	0	0	0	5
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	0	0	2	2
0821	COXSACKIEVIRUS A21	0	0	0	0	0	1	0	0	0	0	1
0901	COXSACKIEVIRUS B1	0	1	0	0	0	1	0	0	0	0	2
0902	COXSACKIEVIRUS B2	0	0	0	1	0	0	0	0	0	0	1
0905	COXSACKIEVIRUS B5	0	0	0	3	0	0	0	0	0	0	3
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	1	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	1	0	0	0	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	4	0	0	0	0	4
1102	POLIOVIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	1	0	0	0	0	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	5	0	0	0	0	0	0	0	1	14	20
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	2	1	0	0	0	0	0	6	9
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	10	5	0	0	0	1	0	0	1	1	18
1303	VARICELLA-ZOSTER VIRUS	1	1	0	0	0	0	0	0	0	12	[redacted]
1306	HERPES SIMPLEX TYPE 1	8	13	0	0	1	0	0	0	0	109	131
1307	HERPES SIMPLEX TYPE 2	9	0	0	0	0	0	0	0	0	92	101
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	0	0	2	3
1401	COXIELLA BURNETI	5	3	0	0	0	0	1	1	0	0	10
1402	OTHER RICKETTSIAE	0	0	0	0	0	0	0	0	0	1	1
1502	PICORNIA VIRUS - NOT TYPED = E	3	11	0	3	5	16	0	5	0	6	49
1521	MEASLES VIRUS	3	1	0	0	1	0	0	0	0	3	8
1522	RUBELLA VIRUS	1	3	0	1	0	0	0	0	0	31	36
1532	HEPATITIS B ANTIGEN	29	1	0	0	0	0	59	0	0	1	90
1535	HEPATITIS A ANTIBODY	1	0	0	0	0	1	8	0	0	0	10
1541	CHLAMYDIA A - C. TRACHOMATIS	14	1	0	0	0	0	0	0	0	0	15
1556	CMV - CYTOMEGALOVIRUS	7	19	0	0	0	0	3	1	4	0	34
1563	CORONAVIRUS	0	0	0	0	0	1	0	0	0	0	1
1564	ROTAVIRUS	0	0	0	0	0	26	0	0	0	0	26
1599	ENTEROVIRUS TYPING PENDING	0	6	0	4	0	5	0	0	0	2	17
9902	POXVIRUS GROUP NOT TYPED	0	0	0	0	0	0	0	0	0	1	1
9992	ROSS RIVER VIRUS	3	0	1	0	0	0	0	0	0	8	12
9993	ASTROVIRUS	0	0	0	0	0	1	0	0	0	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	1	0	0	0	0	1
9998	ARBO. GROUP B. (UNSPECIFIED)	1	0	0	0	0	0	0	0	0	0	1
TOTAL		118	308	3	19	7	83	71	14	7	296	926

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

Period 25/1/88 to 7/2/88

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	1	0	0	0	1	6	2	0	12
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	1	0	0	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	12	0	0	0	0	0	0	0	0	0	12
0199	ADENOVIRUS TYPING PENDING	1	0	0	0	0	0	1	0	0	0	2
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	2	0	0	0	2
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	3	9	0	0	12
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	0	1	0	1
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	1	0	2
0600	MYCOPLASMA PNEUMONIAE	0	1	1	0	2	0	3	14	3	0	24
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	1	1	0	0	2
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	0	0	1	0	0	1
1024	ECHOVIRUS TYPE 24	1	0	0	0	0	0	0	0	0	0	1
1027	ECHOVIRUS TYPE 27	0	0	0	0	0	0	0	0	1	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	0	1	0	1
1200	MUMPS VIRUS	0	0	0	0	0	0	0	1	1	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	8	0	0	0	0	0	0	0	0	8
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	1	0	0	0	0	1	0	0	0	2
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	6	6	0	0	1	13	5	0	31
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	1	0	0	2	0	3
1306	HERPES SIMPLEX TYPE 1	7	81	0	0	0	0	0	1	1	0	90
1307	HERPES SIMPLEX TYPE 2	0	165	0	0	0	0	0	0	0	0	165
1401	COXIELLA BURNETI	0	0	0	0	1	0	2	10	1	0	14
1502	PICORNIA VIRUS - NOT TYPED = E	1	0	1	0	0	0	2	4	0	1	9
1521	MEASLES VIRUS	0	0	0	0	0	0	0	1	0	0	1
1522	RUBELLA VIRUS	0	0	0	1	0	2	0	1	1	0	5
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	2	0	2
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	1	0	0	1
1541	CHLAMYDIA A - C. TRACHOMATIS	0	141	0	0	0	0	0	0	0	0	141
1556	CMV - CYTOMEGALOVIRUS	2	1	0	0	0	0	1	9	18	0	31
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	1	0	2
9901	ARBO. GROUP A. (UNSPECIFIED)	0	0	0	0	1	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	0	0	0	0	8	0	2	1	3	0	14
9995	DENGUE	0	0	0	0	0	0	0	1	0	0	1
TOTAL		27	398	9	7	12	3	21	76	44	1	598