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Communicable Diseases Intelligence

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Contents:

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VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1995 reports were processed during this period.

Six cases (5 males, 1 female) of Q fever were reported during this period. Ages ranged from 16 to 40 years. No occupational exposure details were provided.

The WHO Influenza Reference Centre, Victoria, has characterised 132 influenza A virus isolates from Australia during the 1988 season, with the following results:

Influenza A H_3N_2	- 14	10 resemble A/VIC/7/87, 4 resemble A/Christchurch/2/88
Influenza A H_1N_1	- 118	2 resemble A/TAI/1/86, 116 resemble A/VIC/36/88

It was noted that:

- Type A H_1N_1 viruses predominate in Australia at present. Most isolates resemble the new variant A/VIC/36/88 more closely than A/TAI/1/86.

- Type A H_3N_2 viruses are in the minority and most resemble A/VIC/7/87 more than A/CC/2/88.

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- Contributions are solicited, and do not preclude later publication elsewhere.
- Material appearing in the Bulletin may be quoted provided suitable acknowledgement is made.
- Figures given may be subject to revision.

A breakdown of influenza A virus reported to the CDI by subtype for 1987 and 1988 is shown in the following table:

YEAR	VIRUS	MONTH												TOTAL
		J	F	M	A	M	J	J	A	S	O	N	D	
1987	Influenza A -													
	subtype not stated	8	2	8	1	9	11	7	38	68	32	12	12	208
	subtype H3N2	1					2	1	21	38	8	5	4	80
	subtype H1N1	1							2	6	6	1	1	17
	Total	10	2	8	1	9	13	8	61	112	46	18	17	305
1988*	Influenza A -													
	subtype not stated	2	2	8	2	16	96	287	34					447
	subtype H3N2				1		11	19	5					36
	subtype H1N1						21	198	20					239
	Total	2	2	8	3	16	128	504	59					722

*1988 data subject to revision

It is also noted that 1987 influenza activity was low and influenza activity in 1986 was one of the lowest on record.

AIDS SURVEILLANCE - AUSTRALIA

To 2 August 1988, 943 cases of AIDS fulfilling the criteria of case definition have been reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification (Table 1), by age group (Table 2), by risk category (Table 3) and by clinical presentation (Table 4) are shown below. As previously stated the clinical classification of infection with HIV produced by CDC in Atlanta was adopted in Australia on 1 January 1988. All reporting of clinical manifestations of HIV will now be presented using that classification.

TABLE 1: AIDS patients by State or Territory of notification

STATE/ TERRITORY	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
NSW	591	23	614	325	17	342
VIC	186	3	189	66	2	68
QLD	56	4	60	34	3	37
WA	39	2	41	16	1	17
SA	21	1	22	9	1	10
NT	2	0	2	1	0	1
TAS	2	1	3	2	0	2
ACT	12	0	12	5	0	5
	909	34	943	458	24	482

TABLE 2: AIDS patients by age group

AGE (YEARS)	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
0 - 9	8	1	9	6	1	7
10 - 19	5	1	6	3	1	4
20 - 29	198	9	207	98	2	100
30 - 39	393	3	396	186	1	187
40 - 49	218	4	222	108	4	112
50 - 59	68	6	74	44	6	50
60 +	19	10	29	13	9	22
	909	34	943	458	24	482

TABLE 3: AIDS patients by risk category

RISK GROUP	CASES	DEATHS
Homo-/Bi-sexual	829	409
IV drug user	6	2
Homo-/Bi-sexual IV drug user	24	14
Blood transfusion recipient	48	40
Person with haemophilia	12	6
Heterosexual transmission	9	2
Under investigation	8	3
None of the above	7	6
	943	482

TABLE 4: AIDS patients by clinical presentation

INITIAL DISEASE REPORTED	CASES	DEATHS
GROUP IV:		
B: Neurological disease	18	10
C: Secondary infectious diseases	685	348
D: Secondary cancers	188	91
E: Other conditions	2	1
BC: Neurological disease + infectious diseases	9	7
BD: Neurological disease + cancers	1	1
CD: Infectious diseases + cancers	40	24
	943	482

The data in table 4 include presumptive (clinical) diagnosis.

These data are continually updated and therefore statistics may be revised.

THROMBOCYTOPAENIA, HIV AND INTRAVENOUS DRUG USE
(Based on CDS 88/20, 21 May 1988)

The Department of Genito-Urinary Medicine at the Royal Infirmary in Dundee, Scotland, filed the following preliminary report of several cases of mild to severe thrombocytopaenia associated with HIV infection among intravenous drug users. To date, the Department's catchment area has yielded 187 HIV-seropositive individuals, the majority of whom are males.

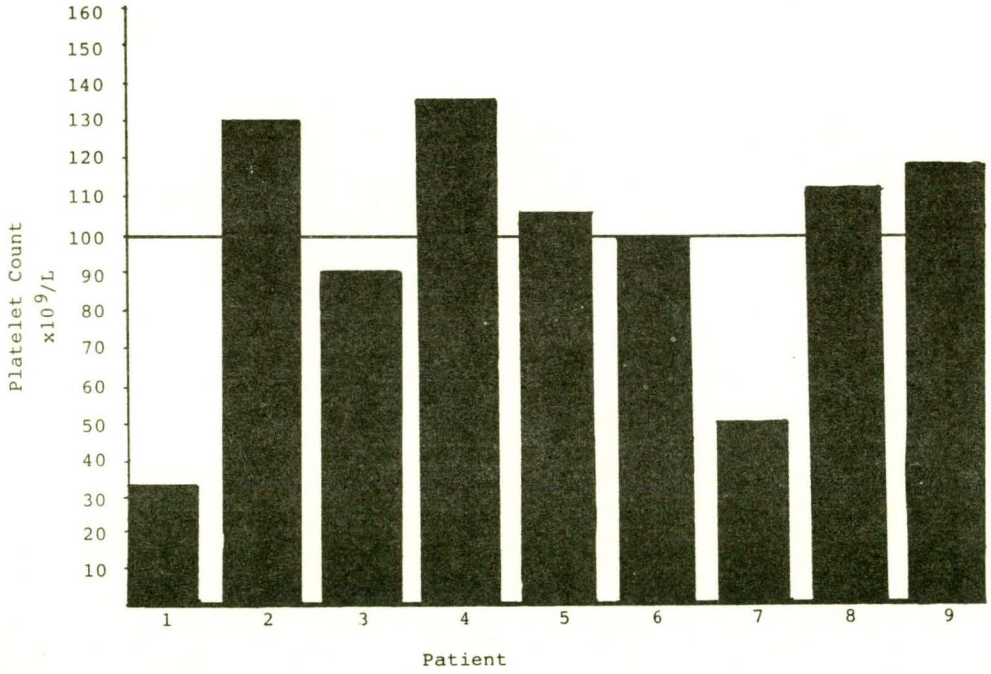
The distribution of cases by risk group is as follows:

	Male	Female	Total
Intravenous drug users	116	46	162
Homosexual males	6		6
Heterosexual			7
Blood transfusion-associated			2
Children			9
Central African origin			1
TOTAL			187

Platelet counts have been obtained for:

- 29 males, 9 with platelet counts <150 x 10^9/L including 3 with <100 x 10^9/L (Figure 1), and
- 14 females, 7 of whom with platelet counts <150 x 10^9/L including 2 with <100 x 10^9/L (figure 2).

Figure 1: HIV positive males.

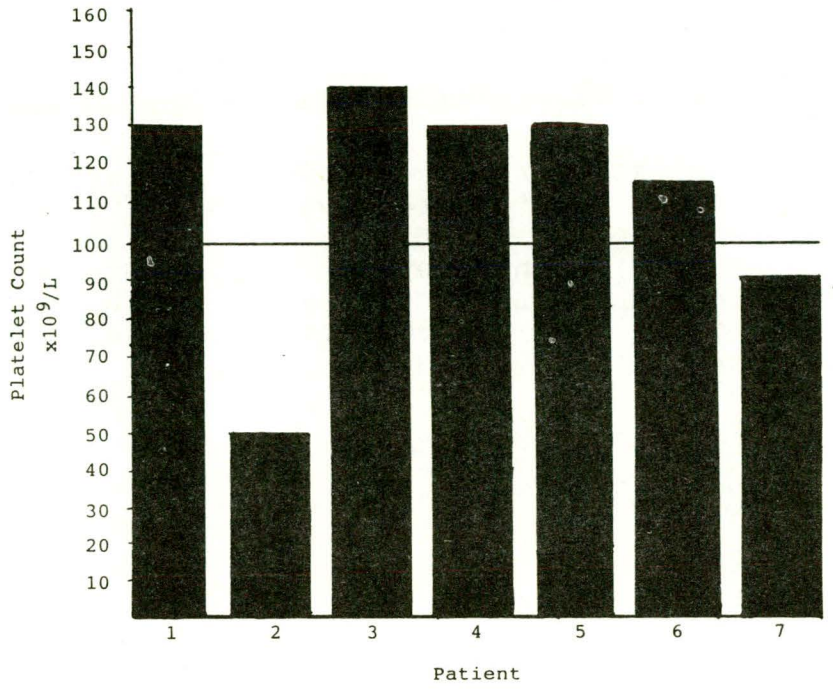


The clinical features of patient 1 and 7 are summarised below:

Patient 1: a 34 year old single male intravenous drug user with no evidence of bleeding, bruising or petechiae. Original platelet count of 29 x 10^9/L increased to 32 x 10^9/L in one week. No treatment was given, although monitoring of levels continues.

Patient 7: a 24 year old single male with no evidence of external or internal bleeding. Platelet count was 50 x 10^9/L.

Figure 2: HIV positive females



The clinical features of patients 2 and 7 are summarised below:

Patient 2: a 27 year old married female with two children. The patient had discontinued intravenous drug use and is currently on oral substitutes. Medical history revealed a record of fits and regular bruises on the trunk and face associated with trauma during blackouts. The patient experienced regular epistaxis. Her platelet count of 50 x 10^9/L fell to 20 x 10^9/L and no active treatment was indicated.

Patient 7: a 26 year old single female who had discontinued intravenous drug use. She has had repeated genito-urinary problems. Her platelet count of 90 x 10^9/L in March 1986 increased steadily to 200 x 10^9/L by August 1986 without treatment. The patient is currently prescribed drug-maintenance therapy with methadone.

The four patients referred to in the notes to the figures have been followed-up with platelet counts when they attended the Clinic, but poor compliance with attendances, difficulty in obtaining blood samples, or both, make it difficult to obtain the desired series of counts over a period of time.

DISCUSSION

Thrombocytopaenia has been reported in homosexual men[1], in persons with haemophilia[2] and in intravenous drug users infected with the human immunodeficiency virus (HIV)[3,4]. Further reports have confirmed these findings of an association between thrombocytopaenia and HIV infection[5,6,7].

Thrombocytopaenia is recognised as a complication of some viral infections, although the mechanism remains unclear. Among HIV-infected intravenous drug users, chronic antigenic stimulation from injected drugs may lead to autoimmune destruction of platelets. However, bacterial sepsis, intravascular coagulopathy, hypersplenism, microangiopathy,

alcoholism and chronic active hepatitis may also be implicated. Destruction of platelets appears to be related to :-

. a specific, anti-platelet antibody found in the serum of HIV-infected patients[8,9], or
. virus-related damage of stem cells[10].

Treatment is not indicated unless there is persistent bleeding or the platelet count is very low. Increasingly, intravenous immunoglobulin is being considered as first-line treatment. Steroid therapy has not been found useful. Splenectomy may produce temporary remission in some patients. Alpha-2a recombinant interferon may offer a non-immunosuppressive treatment for thrombocytopaenia.

REFERENCES

1. Ann Intern Med (1982) 96(I):714-7.
2. NEJM (1983) 308:439-442.
3. Ann Intern Med (1978) 89:207-211.
4. Ann Intern Med (1985) 102:737-741.
5. Ann Intern Med (1985) 103:542-545.
6. Ann Intern Med (1986) 105:145-146.
7. Ann Intern Med (1986) 104:47-50.
8. NEJM (1985) 313:1375-1380.
9. NEJM (1984) 311:635-9.
10. Nature (1987) 326:200-203.

PREVALENCE OF HUMAN IMMUNODEFICIENCY VIRUS ANTIBODY IN U.S. ACTIVE-DUTY MILITARY PERSONNEL, APRIL 1988

(Based on MMWR Vol. 37/No. 30, 5 August 1988)

In January 1986, the U.S. Department of Defense began screening all active-duty military personnel for antibody to the human immunodeficiency virus type 1 (HIV-1). A total of 1,752,191 persons who remained on active duty as of April 24, 1988, were screened. HIV-1 antibody was confirmed by Western blot in 2,232 (1.3 per 1,000) of these persons.

Information from the armed forces/Reportable Disease Data Base was used to determine the demographic distribution of HIV-1-antibody seroprevalence rates (Table 1).

Antibody prevalence by age, ranged from 0.1 per 1,000 for those aged 17-19 years to 2.1 per 1,000 for those aged 25-29 years. Blacks were 3.6 times and Hispanics 2.5 times more likely than non-Hispanic whites to have HIV-1 antibody. Although blacks and Hispanics constituted 50.7% of those who were HIV-1-antibody-positive, they represented only 23.4% of all active-duty personnel. Seroprevalence was highest in men, unmarried persons, and enlisted personnel.

MMWR Editorial Note:

This report summarizes the findings of the largest HIV-1 screening program in a defined population of U.S. citizens. The prevalence of 1.3 per 1,000 persons on active duty as of April 24, 1988, is lower than that found for the screening program overall, since antibody-positive persons were somewhat

Table 1: HIV-1-antibody seroprevalence in Department of Defense active-duty military personnel, by demographic subgroups, January 1987-April 24, 1988

Group	No. tested	No. positive	Seroprevalence (per 1,000)	Seroprevalence rate ratio	(95% CL)
Total	1,752,191	2,232	1.3		
Age group (years)*					
>40	94,343	87	0.9	6.6	(4.5,9.3)
35-39	165,740	224	1.4	9.7	(6.9,13.1)
30-34	234,267	455	1.9	13.9	(10.2,18.6)
25-29	366,156	759	2.1	14.9	(10.9,19.8)
20-24	568,920	662	1.2	8.3	(6.1,11.1)
17-19	322,506	45	0.1	1.0	
Race/Ethnicity					
Black	337,300	988	2.9	3.6	(3.3,3.9)
Hispanic	71,917	144	2.0	2.5	(2.1,2.9)
Other	79,603	72	0.9	1.1	(0.9,1.4)
White	1,263,371	1,028	0.8	1.0	
Sex*					
Male	1,571,912	2,166	1.4	3.8	(2.9,4.8)
Female	180,278	66	0.4	1.0	
Marital status					
Other+	44,732	102	2.3	2.6	(2.1,3.2)
Never Married	690,738	1,255	1.8	2.1	(1.9,2.4)
Married	1,016,721	875	0.9	1.0	
Rank*					
Enlisted	1,515,659	2,061	1.4	1.9	(1.6,2.2)
Officer	235,521	171	0.7	1.0	

* Age unknown for 259 persons, sex unknown for 1 person, rank unknown for 1,011 persons, all were seronegative.
\+ Includes divorced, widowed, separated, and unknown.

more likely than seronegative persons to be separated or retired after obtaining their test results.

The HIV-1-antibody seroprevalence in current active-duty military personnel probably underrepresents the seroprevalence in the civilian population for three reasons. First, homosexual men and male and female intravenous-drug users are underrepresented in military personnel. Second, persons with haemophilia are not medically eligible for military service. Third, seropositive military recruit applicants are denied enlistment; from October 1985 to 24 April 1988, 2,060 of 1,456,177 (1.4 per 1,000) recruit applicants were seropositive.

HIV-1 screening data from active-duty military personnel can be used for monitoring levels and trends of HIV-1 infection in the United States. These data augment those from other large screened populations, such as military recruit applicants, National Guard personnel, Job Corps entrants, and blood

donors. Although each population source has its own limitations and biases, demographic patterns of HIV-1-antibody seroprevalence observed in active-duty military personnel followed patterns observed in other population-based and sentinel studies. For example, each of four groups-black and Hispanic military recruit applicants[1,2], U.S. Army Reserve personnel[3], blood donors[2], and sentinel hospital patients[4] - were three to 12 times more likely than non-Hispanic whites to be HIV-1 seropositive. Similarly, men were at least three times more likely than women to be seropositive in these groups[1-4]. Age-specific HIV-1 seroprevalence peaked in active-duty military personnel in the 25- to 34-year age groups, a finding similar to that in military recruit applicants[1,2] and sentinel hospital patients[4].

Continued monitoring of the active-duty military screening program will be important because all active-duty military personnel will be screened at least every 1-2 years for HIV antibody; therefore, the incidence of new HIV-1 infection can be measured directly. In September 1987, the observed incidence rate in 171,974 U.S. Army personnel was 0.74 new infections per 1,000 persons per year[5]; JG McNeil, Walter Reed Army Institute of Research, personal communication). In comparison, for repeat blood donors to the American Red Cross - a very low risk population that is the only other large population for which incidence of HIV-1 infection can be directly measured - the observed incidence has remained stable at 0.03 new infections per 1,000 persons per year[6]. Neither military personnel nor blood donors are truly representative of the U.S. population; those at highest risk of HIV infection are to a varying extent excluded from both groups.

Data from active-duty military personnel will also provide information about the spectrum of morbidity from HIV-1 infection in this large, defined population. All identified seropositive active-duty military personnel receive a detailed medical evaluation and are staged by the Walter Reed (WR) classification[7]. Most HIV-1-seropositive persons have minimal or no symptoms. Of 650 seropositive active-duty personnel for whom data are available, 60% were asymptomatic (Walter Reed, stage 1 [WR-1]), and 18% had chronic lymphadenopathy without other evidence of immune dysfunction (WR-2)[8].

The Centers for Disease Control will continue cooperating with the Department of Defense in monitoring levels and trends of HIV infection in active-duty military personnel and military recruit applicants. Surveys in other accessible populations at both low and increased risk of HIV infection are also under way.

CDI Editorial Comment:

The Australian Defence Force (ADF) policy on Human Immunodeficiency Virus (HIV) infection covers education, counselling, blood testing and the administrative and medical management of HIV infected ADF personnel.

The main thrust of the ADF policy is prevention through education and a program of limited blood testing directed towards those personnel in whom the presence of HIV infection

would be of the greatest detriment to the safety of other ADF personnel and the efficiency of the ADF operational capabilities.

In addition, for a 12 month trial period all entrants enlisted on or after 28 March 1988 are screened for antibody to HIV to establish the prevalence of HIV antibody in the recruiting pool. Applicants are counselled and informed before entry that such testing will take place as part of the routine post entry medical check, and they are given the option to withdraw their application.

The ADF policy is to be reviewed after 12 months.

REFERENCES

1. N Engl J Med (1987) 317:131-6.
2. MMWR (1987) 36: (Suppl S-6).
3. Cowan DN, Brundage JF. HIV infection among U.S. Army Reserve personnel [Abstract]. IV International Conference on AIDS. Book 2. Stockholm, June 12-16, 1988:222.
4. Dondero TJ, Rauch K, storch GA, et al. U.S. sentinel hospital surveillance network: results of the first 20 months [Abstract]. IV International Conference on AIDS. Book 1. Stockholm, June 12-16, 1988:357.
5. McNeil JG, Wann F, Burke D, Brundage J, Peterson M. A direct estimation of the rate of new HIV infections in U.S. Army personnel [Abstract]. IV International Conference on AIDS. Book 2. Stockholm, June 12-16, 1988:221.
6. Dodd RY, Connelly J, Cumming P. Incidence and prevalence of HIV infection in a low-risk population in the U.S. [Abstract]. IV International Conference on AIDS. Book 2. Stockholm, June 12-16, 1988:347.
7. N Engl J Med (1986) 314:131-2.
8. Mumm A, Carr R, Zajac R, et al. Epidemiology of U.S. Air Force-wide screening for HIV seropositivity [Abstract]. IV International Conference on AIDS. Book 2. Stockholm, June 12-16, 1988:222.

CHILDHOOD CHLOROQUINE POISONINGS - USA

(Based on MMWR Vol. 37/No. 28, 22 July 1988)

Each year approximately 1 million Americans travel to areas where chloroquine may be prescribed for malarial prophylaxis[1]. Despite this widespread use chloroquine is a very toxic drug. Consequently, there are many opportunities for children to be poisoned through chloroquine ingestion. To alert medical practitioners and the public to this danger, the following cases of chloroquine poisoning recently reported to CDC are presented.

Case 1. On 6 August 1987, a previously healthy 20-month-old girl was found unresponsive next to an opened empty bottle of chloroquine phosphate. The chloroquine remained from a supply dispensed to the child's grandfather for malaria prophylaxis. The amount of chloroquine base the child swallowed was estimated at 800 mg.

Shortly after the emergency medical technicians arrived, the child suffered cardiac arrest. Normal sinus rhythm was restored en route to the emergency room, but persistent hypotension necessitated intravenous dopamine. The child began to have generalised seizures 1 hour after ingestion; these were controlled with intravenous diazepam, phenytoin, and phenobarbital. Charcoal haemoperfusion performed 7 hours after ingestion did not improve her condition. Serum chloroquine concentrations before and after the procedure were 0.8 and 0.3 µg/mL, respectively (2.5 and 0.94 µmol/L). Over the next week her neurological condition gradually improved, and mechanical ventilation was discontinued after the eighth day of hospitalisation. Subsequent cranial computerised tomography scans and electroencephalography revealed atrophy and decreased voltage consistent with postanoxic encephalopathy. Rehabilitative efforts continue; currently, she is able to make some purposeful movements but still requires feeding by gastrotomy.

Case 2. On 20 January 1988, a 17-month-old boy ingested 2.4 g of chloroquine base. His parents had recently returned from a tour of duty in Cameroon during which they had been taking chloroquine for malaria prophylaxis; the chloroquine had been dispensed in Cameroon in an envelope. The child was immediately taken to an emergency room, but 30 minutes after ingestion, ventricular tachycardia, hypotension, apnea, and seizures developed. After 2 hours of resuscitation, his condition was stabilised on intravenous epinephrine and diazepam. Serum chloroquine concentration 11 hours after ingestion was 1.0 µg/mL (3.1 µmol/L). His condition improved slightly during the next 3 weeks, and he was gradually removed from ventilator support after 1 month. However, he remains unconscious with no purposeful movement.

MMWR Editorial Note:

When used for prophylaxis and treatment of malaria, chloroquine has proven to be safe in the recommended dosage range (5-25 mg/kg body weight). However, a relatively small increase in the therepeutic dose is toxic; children who have ingested 2-3 times the recommended treatment dose have been fatally poisoned[2]. Chloroquine is rapidly absorbed from the gastrointestinal tract. Consequently, as the second case illustrates, the interval between ingestion and cardio-respiratory collapse is frequently less than 2 hours[3,4].

A recent review of 91 cases of chloroquine poisoning in which blood concentrations were determined revealed that no patient survived in whom blood concentrations were greater than 25 µmol/L[5]. Since the drug is extensively tissue-bound, concentrations in the liver and kidney are generally many times higher than those in the blood[6]. The extensive tissue binding makes dialysis largely ineffective in removing the drug[7].

The toxic effects of chloroquine are related to its depressant effect on the myocardium, resulting in decreased cardiac output and hypotension. Like quinidine, the drug reduces the excitability and conductivity of cardiac muscle, and at toxic concentrations profound bradycardia with ventricular escape rhythms may occur[8].

Animal toxicology data and case studies of suicide attempts with chloroquine suggest that sympathomimetic agents may decrease the haemodynamic and electrophysiological cardiotoxic effects of chloroquine[8]. Diazepam has been found to decrease the mortality rate in experimental chloroquine poisoning in rats[9]. A recent study examined the clinical utility of immediately administering intravenous diazepam and epinephrine in chloroquine poisoning. Ten of eleven patients who ingested more than 5 g of chloroquine and were treated with diazepam and epinephrine survived, as compared with 1 of 51 retrospective controls who ingested comparable dosages[5].

Health care providers should be aware of the potential interventions to prevent chloroquine poisoning. Chloroquine prescriptions should be written for the precise amount needed for prophylaxis for each trip to avoid accumulation of extra tablets. Any drug remaining after prophylaxis is complete should be safely discarded. Chloroquine should be dispensed in child-proof containers, particularly when young children are in the home.

CDI Editorial Comment

The increased demand for chloroquine based antimalarial agents generated by the increasing number of Australians travelling to malaria endemic areas of the world, has increased the availability of this antimalarial drug in Australian homes. Consequently the potential for accidental ingestion of the drug by infants and young children has increased, with the potential for serious consequences as reported above.

It should be noted that chloroquine is widely used in malarious countries of the South West Pacific in suicidal gestures with fatal results[10].

Great care should therefore be exercised in the storage and administration of chloroquine preparations. The liquid formulaton of chloroquine (Nivaquine) which contains 50mg chloroquine base/5mL is preferred to chloroquine tablets for malarial prophylaxis and treatment in children as doses can be accurately dispensed on a mg/kg basis.

REFERENCES

1. JAMA (1987) 257:2626-7.
2. Pediatrics (1961) 27:95-102.
3. South Med J (1974) 67:1031-5.
4. WHO Doc. No. WHO/MAL/79.906.
5. NEJM (1988) 318:1-6.
6. J Pharm Pharmacol (1970) 22:700-3.
7. Pediatrics (1975) 55:536-8.
8. Am Heart J (1970) 79:831-42.
9. J Toxicol Clin Toxicol (1983) 20:271-9.
10. Papua New Guinea Med J (1986) 29:143-147.

RECOMMENDED MINIMUM PERIOD OF EXCLUSION FROM SCHOOL, PRESCHOOL AND CHILD CARE CENTRES OF INFECTIOUS DISEASES CASES AND CONTACTS

The following are the recommendations of the National Health and Medical Research Council on exclusion from school.

NH&MRC Recommendations

Important Notes

- These guidelines have been drawn up on the premise that children who have been ill with an infectious disease will not return to school until they have fully recovered. The only exception to this rule is that children with certain skin diseases may return once appropriate treatment has commenced (see below).

- These recommended periods are issued as a guide to teaching staff and medical practitioners, and may be modified in individual cases as circumstances warrant. Variation in the recommendations may be warranted in cases of local epidemics.

- In cases of doubt, or for guidance in cases of conditions not mentioned on the list, advice should be sought from the appropriate clinician, school medical officer or medical officer of a health authority. Similarly, advice on possible preventive measures should be sought if cases occur in boarding institutions amongst children housed in dormitory-type accommodation.

- Recommendations for children with various categories of AIDS will need to be individualized. Guidance should be sought from the local AIDS authority. The AIDS Task Force publications 'Children and AIDS', 4/86, may be helpful.

- Records of immunization status of children should be accurate and kept up to date.

General infectious diseases

Condition	Cases	Contacts
Chicken Pox (Varicella) and Herpes Zoster.	Exclude till fully recovered or at least 1 week after the eruption first appears (some remaining scabs not an indication for continued exclusion).	Not excluded
Mumps	Exclude till fully recovered.	Not excluded. Recommend immunization if not done.
Rubella	Exclude till fully recovered or 5 days after onset of rash.	Not excluded. Female staff of child-bearing age should ensure that their immune status against rubella is adequate.

Diptheria	Re-admit after receipt of a medical certificate of recovery from infection following at least two negative nose and throat swabs, the first not less than 24hrs after cessation of anti-microbial therapy and the other 48hrs later.	Exclude domiciliary contacts. An appropriate public health medical officer should investigate contacts immediately and release them when they are shown to be clear of infection.
Encephalitis	This is not a specific clinical entity. No exclusion periods are necessary for either cases or contacts unless due to measles, in which case exclude for that disease.	
Infectious Hepatitis (Hepatitis A)	Re-admit on receipt of a medical certificate of recovery, or on subsidence of symptoms.	Not excluded.
Leprosy	Re-admit on production of a medical certificate from appropriate health authority.	Not excluded.
Measles	Should be excluded for at least 5 days from the appearance of rash or until a medical certificate of recovery is produced.	Immunized contacts not excluded. All children should be immunized against measles preferably at 12 months of age and certainly before entry into preschool or daycare centre unless they have had the disease. Therefore the need to exclude contacts should not arise. Non-immunized contacts should be excluded for 13 days from the first day of appearance of rash in the last case unless immunised within 72 hrs of first contact.
Meningitis (Bacterial)	No exclusion period is necessary following treatment and recovery.	Not exclude (other than meningococcal meningitis contacts (see below).
Meningococcal infection	Re-admit on production of a medical certificate of recovery.	Domiciliary contacts only should be excluded until they have been receiving appropriate chemotherapy for at least 48hrs.

Poliomyelitis	Should be excluded for at least 14 days from onset and also until a medical certificate of recovery is produced.	Need not be excluded. All children should be immunized prior to reaching school-age. Non-immunized polio contacts should be directed to a medical officer.
Streptococcal infection including Scarlet Fever.	Should be excluded until appropriate medical treatment and a medical certificate of recovery is given.	Need not be excluded.
Tuberculosis	Re-admit on production of medical certificate from appropriate health authority that the child is not considered to be infectious.	Need not be excluded.
Typhoid and Paratyphoid	Re-admit after a medical certificated of freedom from infection is received, following three negative faecal and urine cultures taken at least 24hrs apart, commencing at least 72hrs after cessation of specific therapy.	Not excluded unless an appropriate public health medical officer considers exclusion to be necessary.
Whooping cough (Pertussis)	Should be excluded for two weeks from onset of illness and until a medical certificate of recovery is produced.	Exclude domiciliary contacts for 21 days after the last exposure to infection if attending a preschool centre and if the child has not previously had whooping cough or immunization. Contacts need not be excluded from any other class of school.
Diarrhoea (Rotavirus, Shigella, Giardia)	For isolated cases, exclude from school and preschool while diarrhoea persists.	Contacts not excluded.

Common local diseases affecting skin, hair and eyes in school children:

Condition	Cases	Contacts
Ringworm, Scabies, Pediculosis (Lice) Trachoma.	Re-admit when appropriate treatment has commenced, supported when requested by a medical certificate.	Not excluded. Close contacts should be inspected regularly for signs of infestation or infection.

Conjunctivitis (Acute infectious)	Until discharge from eyes has ceased.	Not excluded.
Impetigo (School Sores)	Until sores have fully healed. The child may be allowed to return provided that appropriate treatment has commenced and that sores on exposed surfaces such as scalp, face, hands or legs are properly covered with occlusive dressing.	Not excluded.

CDI Editorial Comment

The current recommendations do not mention hepatitis B infection. Children with hepatitis B should not return to school until they are well. Hepatitis B carriers should not be excluded nor should contacts of hepatitis B cases.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 10-8-88 TO 23-8-88

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	0	1	10	0	0	3	1	12	27
0101	ADENOVIRUS TYPE 1	5	2	2	0	0	0	0	0	9
0102	ADENOVIRUS TYPE 2	2	0	1	0	3	0	0	0	6
0103	ADENOVIRUS TYPE 3	0	0	1	0	0	0	0	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	3	1	1	0	1	0	0	0	6
0108	ADENOVIRUS TYPE 8	1	0	1	0	1	0	0	0	3
0199	ADENOVIRUS TYPING PENDING	1	0	0	2	0	0	2	0	5
0201	INFLUENZA A VIRUS	27	33	13	26	49	16	0	0	164
0202	INFLUENZA A VIRUS SUBTYPE H3N2	1	0	2	0	0	0	2	12	17
0203	INFLUENZA B VIRUS	0	0	1	0	1	2	0	0	4
0206	INFLUENZA A H1N1	23	0	23	0	12	0	5	47	110
0299	INFLUENZA VIRUS - TYPING PENDI	0	0	0	9	0	0	1	0	10
0301	PARAINFLUENZA VIRUS TYPE 1	1	0	2	2	0	0	0	6	11
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	1	1	0	0	0	1	4
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	20	2	0	0	0	2	24
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	0	0	1	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	72	50	49	51	22	5	14	46	309
0500	RHINOVIRUS (ALL TYPES)	6	3	2	3	0	1	0	10	25
0600	MYCOPLASMA PNEUMONIAE	32	2	26	5	22	1	1	0	89
0700	ORNITHOSIS-PSITTACOSIS	2	0	0	0	1	0	0	0	3
0809	COXSACKIEVIRUS A9	1	0	0	0	2	0	0	0	3
0816	COXSACKIEVIRUS A16	1	0	0	0	0	0	0	0	1
0817	COXSACKIEVIRUS A17	1	0	0	0	0	0	0	0	1
0821	COXSACKIEVIRUS A21	5	0	0	0	0	0	0	0	5
0902	COXSACKIEVIRUS B2	0	0	0	0	1	0	0	0	1
0904	COXSACKIEVIRUS B4	2	0	1	0	1	0	0	0	4
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	0	0	1
1000	ECHOVIRUS NOT TYPED	0	1	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	1	0	2	0	0	0	0	0	3
1009	ECHOVIRUS TYPE 9	1	0	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	13	0	0	0	0	0	0	0	13
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	6	0	0	6
1101	POLIOVIRUS TYPE 1	1	1	0	0	1	0	0	0	3
1103	POLIOVIRUS TYPE 3	0	1	0	0	0	0	0	0	1
1200	MUMPS VIRUS	1	0	0	0	3	0	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	17	0	0	1	19
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	2	0	0	0	0	0	0	3
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	5	3	1	3	4	0	3	0	19
1303	VARICELLA-ZOSTER VIRUS	4	3	1	0	6	3	1	0	18
1306	HERPES SIMPLEX TYPE 1	87	17	12	0	46	15	3	62	242
1307	HERPES SIMPLEX TYPE 2	124	56	18	0	74	30	0	63	365
1399	HERPES VIRUS TYPING PENDING	2	0	0	6	0	0	0	0	8
1401	COXIELLA BURNETI	1	0	0	0	5	0	0	0	6
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	2	6	0	10	18
1521	MEASLES VIRUS	0	0	0	0	1	2	0	0	3
1522	RUBELLA VIRUS	3	0	0	0	2	0	0	0	5
1532	HEPATITIS B ANTIGEN	38	11	4	0	39	5	1	23	121
1535	HEPATITIS A ANTIBODY	3	6	1	0	0	0	0	0	10
1541	CHLAMYDIA A - C. TRACHOMATIS	0	9	4	0	14	0	1	23	51
1552	RABIES VIRUS	1	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	40	12	5	8	15	1	1	18	100
1564	ROTAVIRUS	7	45	18	0	3	8	17	0	98
1565	CALICI VIRUS	0	0	0	0	0	0	1	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	10	0	0	3	0	13
9992	ROSS RIVER VIRUS	5	1	1	0	3	7	0	0	17
TOTAL		527	261	223	128	351	111	57	337	1995

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

PERIOD 10-8-88 TO 23-8-88

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

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	2	13	0	0	0	8	0	0	0	0	23
0101	ADENOVIRUS TYPE 1	1	5	0	0	0	1	1	0	0	0	8
0102	ADENOVIRUS TYPE 2	1	2	0	0	0	2	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	0	1	0	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	1	2	0	0	0	1	0	0	0	1	5
0199	ADENOVIRUS TYPING PENDING	0	1	0	0	0	3	0	0	1	0	5
0201	INFLUENZA A VIRUS	16	119	1	0	2	0	1	1	0	1	141
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	16	0	0	0	0	0	0	0	0	16
0203	INFLUENZA B VIRUS	0	3	0	0	0	0	0	0	0	0	3
0206	INFLUENZA A H1N1	3	103	0	0	0	0	0	0	0	1	107
0299	INFLUENZA VIRUS - TYPING PENDI	0	7	0	0	0	0	0	0	0	0	7
0301	PARAINFLUENZA VIRUS TYPE 1	0	11	0	0	0	0	0	0	0	0	11
0302	PARAINFLUENZA VIRUS TYPE 2	0	4	0	0	0	0	0	0	0	0	4
0303	PARAINFLUENZA VIRUS TYPE 3	0	24	0	0	0	0	0	0	0	0	24
0399	PARAINFLUENZA VIRUS TYPING PEN	0	1	0	0	0	0	0	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	9	286	1	2	0	0	0	0	0	0	298
0500	RHINOVIRUS (ALL TYPES)	0	25	0	0	0	0	0	0	0	0	25
0600	MYCOPLASMA PNEUMONIAE	12	71	0	0	0	0	0	0	0	3	86
0700	ORNITHOSIS-PSITTACOSIS	0	2	0	0	0	0	0	0	0	0	2
0809	COXSACKIEVIRUS A9	0	0	0	2	0	0	0	0	0	1	3
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	0	0	1	1
0817	COXSACKIEVIRUS A17	0	0	0	0	0	1	0	0	0	0	1
0821	COXSACKIEVIRUS A21	1	3	0	1	0	0	0	0	0	0	5
0902	COXSACKIEVIRUS B2	0	0	0	0	0	1	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	1	0	0	0	2	0	0	0	0	3
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	0	0	0	0	1
1000	ECHOVIRUS NOT TYPED	0	0	0	0	0	1	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	2	0	1	0	0	0	0	0	0	3
1009	ECHOVIRUS TYPE 9	0	0	0	1	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	4	0	0	8	0	0	0	0	0	0	12
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	6	0	0	0	0	6
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	1	0	0	0	0	1
1103	POLIOVIRUS TYPE 3	0	0	0	0	0	1	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	3	1	0	0	0	0	0	0	0	5	9
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	0	1	0	0	0	0	0	1	2
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	5	1	0	0	0	0	0	1	0	0	7
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	2	0	0	0	1	13	16
1306	HERPES SIMPLEX TYPE 1	13	21	1	1	0	0	1	0	0	121	158
1307	HERPES SIMPLEX TYPE 2	16	0	0	0	0	0	0	0	0	68	84
1399	HERPES VIRUS TYPING PENDING	0	3	0	0	0	0	0	0	0	2	5
1401	COXIELLA BURNETI	3	1	0	0	0	0	0	0	0	0	4
1502	PICORNIA VIRUS - NOT TYPED = E	1	5	0	0	1	10	0	0	0	0	17
1521	MEASLES VIRUS	2	0	0	0	0	0	0	0	0	0	2
1522	RUBELLA VIRUS	0	0	1	0	0	0	0	0	0	4	5
1532	HEPATITIS B ANTIGEN	76	0	0	0	0	0	37	0	0	1	114
1535	HEPATITIS A ANTIBODY	5	0	0	0	0	0	3	0	0	0	8
1541	CHLAMYDIA A - C. TRACHOMATIS	8	0	0	0	0	0	0	0	0	0	8
1552	RABIES VIRUS	1	0	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	7	28	0	0	2	0	1	0	8	1	47
1564	ROTAVIRUS	0	0	0	0	0	95	0	0	0	0	95
1565	CALICI VIRUS	0	0	0	0	0	1	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	6	0	2	0	0	0	0	0	0	8
9992	ROSS RIVER VIRUS	3	0	0	0	0	0	1	0	0	3	7
TOTAL		194	768	4	19	7	134	45	2	10	227	1410

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

PERIOD 10-8-88 TO 23-8-88

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	1	0	0	1	0	0	1	0	1	0	4
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	0	0	1	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	0	0	0	0	0	0	0	1	0	1
0108	ADENOVIRUS TYPE 8	3	0	0	0	0	0	0	0	0	0	3
0201	INFLUENZA A VIRUS	0	0	0	0	1	0	9	11	2	0	23
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	0	0	0	0	0	1	0	1
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	0	1	0	0	1
0206	INFLUENZA A H1N1	0	0	0	0	0	0	0	0	3	0	3
0299	INFLUENZA VIRUS - TYPING PENDI	0	0	0	0	0	0	0	3	0	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	1	0	0	2	5	3	0	11
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	0	1	2	0	3
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	1	0	0	0	1
0904	COXSACKIEVIRUS B4	0	0	0	0	0	0	0	1	0	0	1
1030	ECHOVIRUS TYPE 30	0	0	0	0	0	0	0	0	1	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	1	2
1200	MUMPS VIRUS	0	0	3	0	0	0	0	1	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	1	9	0	0	0	0	0	0	0	0	10
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	1	0	0	0	0	0	0	0	0	1
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	7	2	0	0	0	2	1	0	12
1303	VARICELLA-ZOSTER VIRUS	0	0	1	0	0	0	0	0	1	0	2
1306	HERPES SIMPLEX TYPE 1	12	65	0	0	0	0	0	3	4	0	84
1307	HERPES SIMPLEX TYPE 2	0	277	0	0	1	0	0	1	2	0	281
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	2	0	0	3
1401	COXIELLA BURNETI	0	0	0	0	0	0	1	0	1	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	0	1	0	0	1
1521	MEASLES VIRUS	0	0	1	0	0	0	0	0	0	0	1
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	7	0	7
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	0	2	0	2
1541	CHLAMYDIA A - C. TRACHOMATIS	1	42	0	0	0	0	0	0	0	0	43
1556	CMV - CYTOMEGALOVIRUS	1	1	0	4	1	6	1	8	29	2	53
1564	ROTAVIRUS	0	0	0	0	0	0	1	0	2	0	3
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	1	0	0	1	3	5
9992	ROSS RIVER VIRUS	0	0	0	0	10	0	0	0	0	0	10
TOTAL		20	396	12	8	13	7	16	40	67	6	585

CO9-4376B

CDI BULLETIN - COMMENTS ON DATA PRESENTATION AND DISSEMINATION

The digital viral data published in the CDI would not mean much except to the specialist who keeps track of the reports from fortnight to fortnight. For example, in CDI 88/17 there are 365 herpes simplex type 2 and 164 influenza type A. Do either or both of these represent major epidemics or are they expected?

Graphical data would be more intelligible to most. We now have a high resolution laser printer and the computing power to log data and print in graphic form. Each issue of the CDI should have a graph of the most important viral isolations. Less important data could be printed every 2 months or so. The graph should have the previous 12 months data (26 points), the 12 months before that, and possibly a third graph with the mean of the last 5 years data. These would indicate seasonal trends (influenza virus, Ross River Virus) or general incidence trends (hepatitis B). Each fortnight the graph would be reprinted with an additional point added and the oldest point dropped off. Examples of hypothetical graphs attached.

Graphical data should be provided fortnightly to our regional offices, state health offices, participating laboratories, Australian Medical Journals, medical colleges and so on, where these do not already receive the CDI, together with a covering note advising the source of data. This modified data presentation should be started in a small way. Start with one virus, publish updates every 2 months, get the bugs out of the system (there will be many), note comments and criticisms from users of the service (everyone's an expert these days), and then expand. Increase publication frequency to fortnightly, and add to the number of viruses covered.

Suggest starting with influenza A virus or hepatitis B antigen. Set a target date of January 1989 for implementation, July 1989 for increasing the frequency of updates to fortnightly and adding the second virus.

NOTES ON SPECIMEN GRAPHS

The Ross River virus graph shows the extent of an epidemic in 1987/88. The 5 year average indicates a 'normal' year, and 1986/87 fits this well.

The hepatitis B graph shows that incidence is gradually increasing in 1987/88, and has been so doing for at least part of 1986/87. This is not a seasonal effect as evidenced by the 5 year average, and signals the need for concern.

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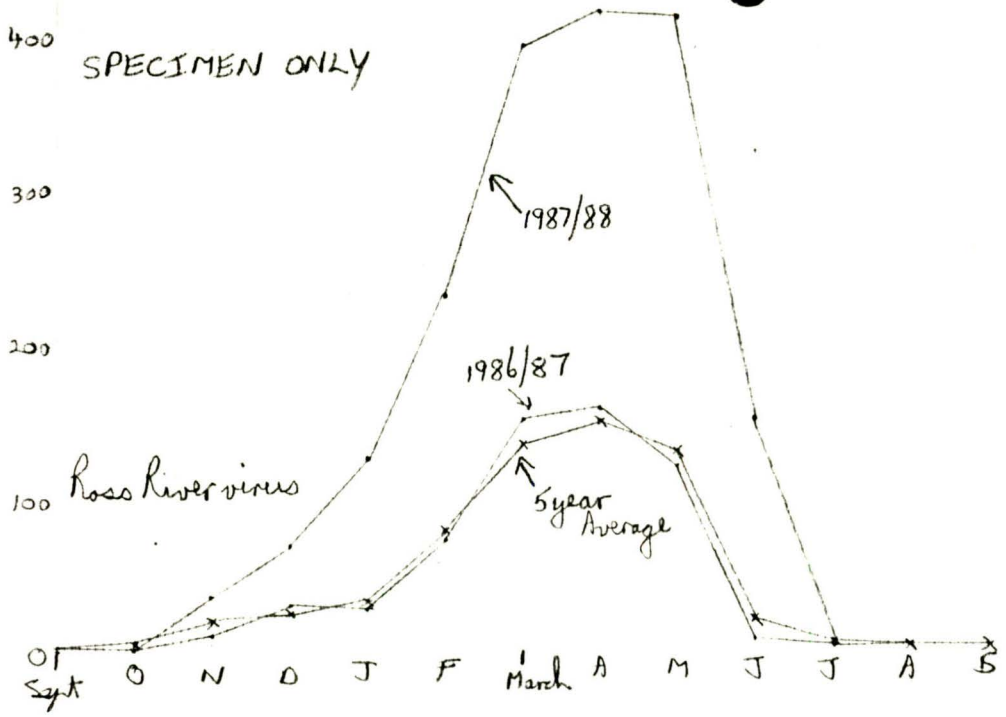
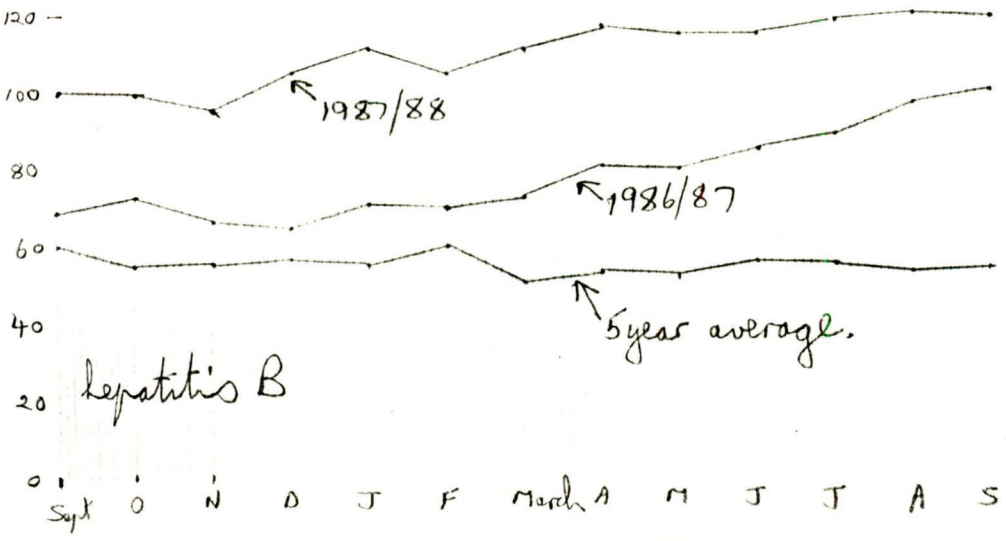
SOCIAL HEALTH BRANCH
9 September 1988


SPECIMEN ONLY
400
300
200
100
0
1987/88
1986/87
5 year Average
Ross River virus
Sept
O
N
D
J
F
March
A
M
J
J
A
S


120
100
80
60
40
20
0
1987/88
1986/87
5 year average.
Hepatitis B
Sept
O
N
D
J
F
March
A
M
J
J
A
S