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Editor

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1741 reports were processed during this period.

Fourteen cases of Q fever (11 males, 3 females) were reported during this period. Ages ranged from 14 to 69 years. No occupational exposure details were provided.

Post-natal cytomegalovirus infection was diagnosed in a 3-week-old male infant following isolation of CMV from saliva and urine samples. The child, whose mother had confirmed primary CMV infection at 10-11 weeks gestation, had been CMV culture negative at birth. This infection was most probably acquired through breast feeding.

Coxsackie virus type A24 was isolated from an eye swab from a 67-year-old male. This is only the third isolate of this virus from an eye swab since the inception of the CDI reporting scheme. However, none of the three patients showed signs of haemorrhagic conjunctivitis, which is often associated with this virus (see also CDI 88/10 p1).

The progessive total for echovirus type 30 isolates for 1988 is now 161 - 150 from Victoria, 7 from Western Australia and four from New South Wales. (Note: Data for 1988 are not yet complete.)

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

- Figures given may be subject to revision.

ECHOVIRUS TYPE 9 - WESTERN AUSTRALIA

(Contributed by Dr M. Bucens, State Health Laboratory Services, Perth, WA)

An outbreak of echovirus 9 has been occurring in Western Australia. The first two isolates were made from specimens collected in June 1988. To date there have been 78 isolations from 63 patients. Fifty-three patients are from the metropolitan area and 10 from the country - as far north as Carnarvon and south to Albany. The epidemic peaked in September when the virus was isolated from 30 patients.

The clinical syndromes associated with this outbreak are:

fever with or without rash	33% (21 patients)
meningitis	48% (30 patients)
diarrhoea/vomiting	11% (7 patients)
respiratory symptoms	8% (5 patients).

The age distribution of patients is as follows:

< 1 month	4 patients
2-12 months	16 patients
13 months- 5 years	15 patients
6-10 years	12 patients
11-20 years	4 patients
> 21 years	12 patients

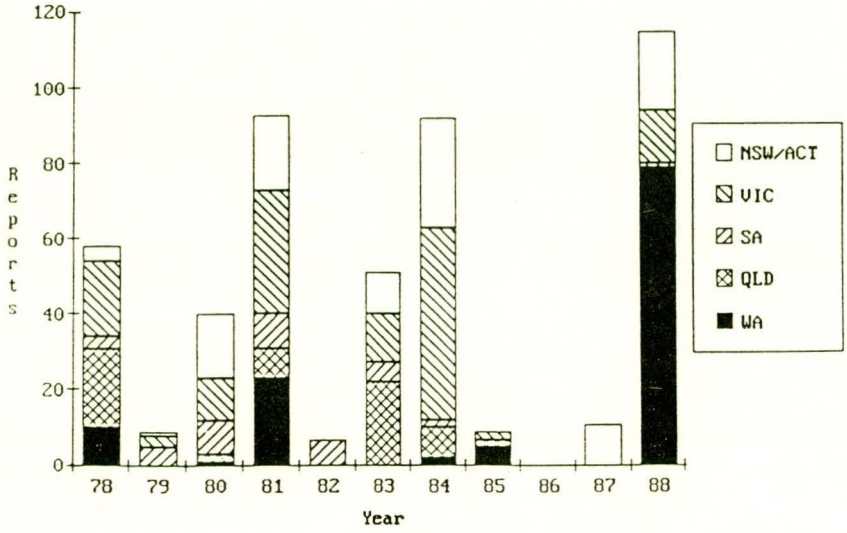
There have been no isolations from babies in neonatal units and no deaths associated with echovirus 9.

Prior to June 1988 the virus had not been isolated in the laboratory since August 1985. The only other recorded echovirus 9 outbreak in Western Australia occurred between November 1969 and April 1970. At that time, as in the current situation, meningitis and a maculopapular rash were the prominent clinical features.

CDI Editorial Comment

Low level activity of echovirus type 9 has been reported to the CDI by all states over the past 11 years (see Figure 1).

Figure 1: Reports of echovirus type 9, CDI, 1978 - 1988*

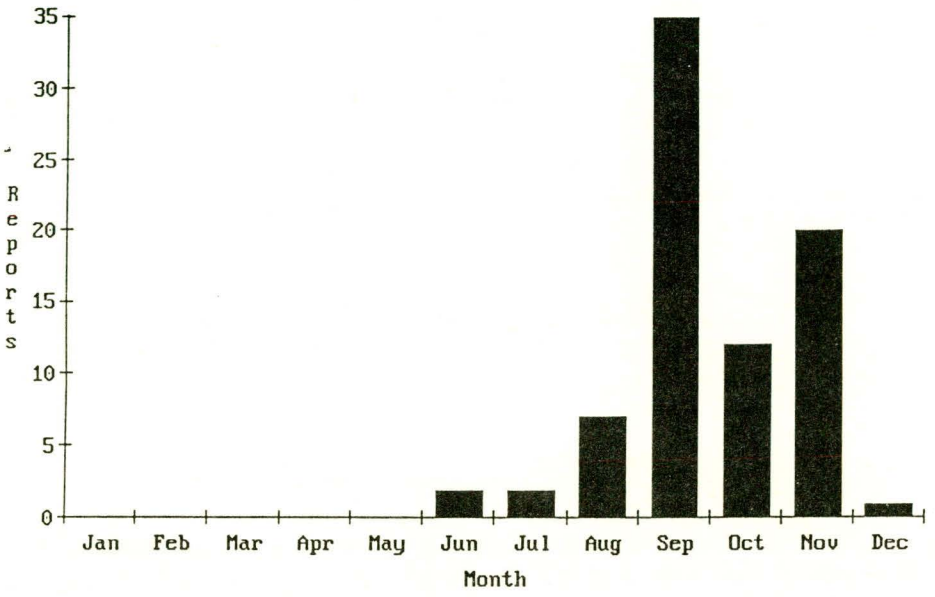


*Data collection for 1988 is not yet complete and figures may be amended at a later date.

In this outbreak, peak activity occurred in September (see Figure 2). Generally, peak activity has been observed between the months of November and March, although in 1984 constant circulation of the virus was seen throughout the year.

Since this report was written the cumulative total of cases reported in Western Australia in 1988 has risen from 63 to 79, and fruther reports are expected. (N.B. Only initial samples for each patient are included in the CDI reporting scheme).

Figure 2: Reports of echovirus type 9 by date of sample collection, Western Australia - 1988*



*Data collection for 1988 is not yet complete and figures may be amended at a later date.

HUMAN T-LYMPHOTROPIC VIRUS TYPES I AND II (HTLV-I & II) LICENSING OF SCREENING TESTS FOR ANTIBODY TO HTLV-I

(Based on MMWR Vol. 37/No. 48, 9 December 1988)

I HTLV-I EPIDEMIOLOGY

A. Virology

HTLV-I was isolated in 1978 and first reported in 1980 (1). Although a member of the family of retroviruses, HTLV-I:

- is **not** closely related to HIV, the retrovirus causing AIDS,
- does not cause depletion of T-helper lymphocytes, and
- is not generally associated with immunosuppression.

HTLV-I differs from HIV:

- in its morphological and genetic structure, and
- in that HTLV-I antigens should not cross-react with HIV antigens.

The HTLV-I genome contains four major genes:

- *gag*, which encodes core-proteins of 15,000 (p15), 19,000 (p19), and 24,000 (p24) daltons;
- *pol*, which encodes a polymerase (reverse transcriptase) protein of 96,000 daltons;
- *env*, which encodes envelope glycoproteins of 21,000 (gp 21) and 46,000 (gp 46) daltons; and
- *tax*, which encodes a transactivator protein of 40,000 daltons (p40x).

B. Seroprevalence

HTLV-I infection is endemic primarily in:

- some areas of Africa (2),
- southwestern Japan where seroprevalence rates as high as 15% in the general population and 30% in older age groups have been reported in some areas (3),
- the Caribbean islands where rates may be as high as 5% in the general population and 15% in older age groups (4).

Seroprevalence in well-characterised areas appears to increase with patient age beginning in the 20-30 year age range, with rates in females markedly higher than those in males.

In the USA, HTLV-I infection has been identified:

- mainly in intravenous drug users (IVDUs) with seroprevalence rates ranging from 7% to 49% (5,6),
- in female prostitutes in whom elevated seroprevalence rates have been associated with IV-drug use, a major risk factor (7), and
- in recipients of multiple blood transfusions (8).

Seropositivity is rare among homosexual men and among patients in sexually transmitted disease clinics (9,10), and it appears to be nonexistent in haemophilic men without other risk-factors (11).

Systematic determination of HTLV-I seroprevalence in the general population of the United States has not been undertaken. However, in a study of approximately 40,000 random blood donors in eight U.S. cities, 0.025% were seropositive for HTLV-I (12).

C. Transmission

Transmission of HTLV-I infection by blood transfusion is well documented in Japan, with a seroconversion rate of 63% in recipients of the *cellular* components of contaminated units (whole blood, red blood cells, and platelets) (13). Transmission by the plasma fraction of contaminated units has not resulted in infection. This finding has been attributed to the fact that HTLV-I is highly cell-associated. Transmission among intravenous drug users is presumed to occur by sharing of needles and syringes contaminated with infectious blood.

Transmission from mother to child occurs through breastfeeding; breastfed infants of seropositive mothers have an approximately 25% probability of becoming infected (14). However, infection has also occurred in infants who are not breastfed, suggesting that intrauterine and/or perinatal transmission may occur.

Sexual transmission of HTLV-I appears to be relatively inefficient (15). Transmission from male to female, however, appears to be more efficient than from female to male (16).

D. Disease associations

HTLV-I has been aetiologically associated with adult T-cell leukaemia/lymphoma (ATL), a malignancy of mature T-lymphocytes characterised by skin lesions, visceral involvement, circulating abnormal lymphocytes, hypercalcaemia, and lytic bone lesions (17).

ATL has been recognised in Japan, the Caribbean and Africa. No systematic attempt has been made to record cases of ATL in the United States, but 74 cases were reported to the National Institutes of Health between 1980 and 1987 (18). Approximately half of these cases occurred in persons of Japanese or Caribbean descent; most of the remainder were in blacks from the Southeastern United States. ATL tends to occur equally in men and women, with peak occurrence in persons 40-60 years of age.

It is thought that a person must be infected with HTLV-I for years to decades before ATL develops:

- in Japan, the lifetime risk of ATL among HTLV-I infected persons has been estimated to be approximately 2% in two studies (19,20); and
- in Jamaica, the lifetime risk of ATL among persons infected before the age of 20 years was estimated to be 4% (21).

Because of the long latent period of ATL, the risk of this disease among persons infected by blood transfusions is not thought to be substantial (many of these persons are elderly and may not survive their underlying disease). In fact, no cases of ATL associated with blood transfusion have been reported.

HTLV-I has also been associated with a degenerative neurological disease known as:

- tropical spastic paraparesis (TSP) in the Caribbean (22), and
- HTLV-I associated myelopathy (HAM) in Japan (23).

TSP/HAM

- is characterised by progressive difficulty in walking, lower extremity weakness, sensory disturbances, and urinary incontinence;
- occurs in persons of all age groups, with peak occurrence in ages 40-49 years and with rates higher in females than in males;

. lifetime risk among HTLV-I infected persons is unknown but appears to be very low;
. latency period appears to be less than that for ATL, and the disease can probably be caused by blood transfusion (of 420 Japanese patients with HAM from whom information was available, 109 [26%] gave a history of blood transfusion; the mean interval between transfusion and onset of neurological symptoms was estimated to be 4 years).

Although most cases have been reported from countries in which HTLV-I is endemic, a few cases have occurred in the United States (24).

II HTLV-II EPIDEMIOLOGY

HTLV-II is closely related to HTLV-I. The genome of HTLV-II encodes viral proteins that are similar to those of HTLV-I, and there is extensive serological cross-reactivity among proteins from HTLV-I and HTLV-II.

No specific information is available regarding the seroepidemiology or the modes of transmission of HTLV-II. There is some evidence that some of the HTLV-I seropositivity in the United States, especially in intravenous drug users, may be caused by HTLV-II (5).

Two cases of disease have been associated with HTLV-II infection:

- Case 1: HTLV-II was first isolated from a patient with a rare T-lymphocytic hairy cell leukaemia (25).
- Case 2: HTLV-II was isolated from a patient who had the more common B-lymphocytic form of hairy cell leukaemia and who also suffered from a T-suppressor lymphoproliferative disease (26).

No serological evidence of HTLV-II infection has been found in 21 additional cases of hairy cell leukaemia (27). Thus, the disease associations of HTLV-II are unclear, and nothing is known regarding lifetime risk of disease among infected persons.

III SEROLOGICAL TESTING FOR HTLV-I

A. Interpretation

The screening tests that have been licensed by the Food and Drug Administration (FDA) are enzyme linked immunosorbent assays (ELISAs) to detect HTLV-I antibody in serum or plasma:

. Specimens with absorbance values greater than the cutoff value determined by the manufacturer are defined as initially reactive.
. Initially reactive specimens must be retested in duplicate to minimise the chance that reactivity is due to technical error.
. Specimens that react in either of the duplicate tests are considered repeatedly reactive.

Note: Additional and more specific serological tests are necessary to confirm that serum specimens repeatedly reactive in the screening tests are positive for HTLV-I antibody.

Users of the screening tests must have available to them additional and more specific tests to properly interpret repeatedly reactive screening tests. (Such tests are available in research institutions, industry and some diagnostic laboratories. No such tests have been licensed by the FDA).

. Specimens that do not react in either of the duplicate repeat test are considered non-reactive.

Tests used to confirm HTLV-I seropositivity must:

. be inherently capable of identifying antibody to the core (*gag*) and envelope (*env*) proteins of HTLV-I (the immunoreactivities of the polymerase (*pol*) and transactivator (*tax*) proteins of HTLV-I have not been well-defined in current test systems); and
. include specific tests such as Western immunoblot (WB) and radio-immunoprecipitation assay (RIPA). (Indirect immunofluorescent antibody assay (IFA) testing for HTLV-I has been used in some laboratories, but IFA does not detect antibody to specific HTLV-I gene products).

- WB appears to be the most sensitive of the more specific tests for antibody to *gag* protein products p19, p24 and (*gag*-derived) p28; whereas
- RIPA appears to be most sensitive for antibody to the *env* glycoproteins gp 46 and (*env* precursor) gp 61/68.

Based on experience with these tests in several laboratories, the following confirmatory criteria for HTLV-I seropositivity have been adopted by the U.S. Public Health Service Working Group:

. A specimen must demonstrate immunoreactivity to *gag* gene product p24 **and** to an *env* gene product (gp46 and/or gp 61/68) to be considered "positive".
. Serum specimens not satisfying these criteria but having immunoreactivities to at least one suspected HTLV-I gene product (such as p19 only, p19 and p28, or p19 and *env*) are designated "indeterminate"
. Serum specimens with no immunoreactivity to any HTLV-I gene products in additional, more specific tests are designated "negative".

Both WB and RIPA may be required to determine whether a serum specimen is positive, indeterminate, or negative.

Although additional and more specific tests have been somewhat standardised, the quantities and the molecular weights of HTLV-I proteins produced by various cell lines vary considerably. Hence, the cell of origin for HTLV-I antigens used in either WB or RIPA, as well as the method

of antigen preparation, may markedly influence test sensitivity and interpretation of immunoreactivity against individual HTLV-I proteins. Laboratories performing these tests, however, should be able to detect antibody to the *gag* and *env* gene products of HTLV-I in WB and for RIPA.

B. Sensitivity, specificity and predictive value

Using the WB and RIPA available in research laboratories and the confirmatory criteria described above to define the presence of HTLV-I antibody, the sensitivities of the three ELISAs that have been licensed by the FDA have been estimated from the performance of the tests on a reference panel of 137 antibody-positive serum specimens:

- All three ELISAs were repeatedly reactive from 137 of 137 panel serum specimens, yielding an estimated sensitivity of above 97.3% by the binomial distribution at 95% confidence.
- Specificity of the ELISAs was estimated for each test from screening at least 5000 normal U.S. blood donors in non-endemic areas. Specificities ranged between 99.3% and 99.9% by the binomial distribution at 95% confidence. However, a specificity of >99% but <100% may still yield a low positive predictive value when the screening test is used in a low-prevalence population. In the study of U.S. blood donors cited above, 68 donors were repeat reactors in the screening test, but only 10 (15%) were determined to be HTLV-I seropositive in more specific testing. Such a relatively low positive predictive value emphasises the need for additional, more specific testing of specimens repeatedly reactive in the ELISA.

  Note: Neither the ELISAs nor the additional, more specific tests can distinguish between antibodies to HTLV-I and HTLV-II.

  More sophisticated techniques, such as virus isolation and gene amplification (polymerase chain reaction [PCR]) are required to differentiate HTLV-I from HTLV-II infection.

C. Use of HTLV-I screening tests in blood banks

The FDA recommends that:

- whole blood and cellular components donated for transfusion be screened for HTLV-I antibody using a licensed ELISA screening test;
- units that are repeatedly reactive by ELISA be quarantined, then destroyed, unless otherwise stipulated by the FDA; and
- source plasma (obtained from plasma donors) intended for use in further manufacturing need not be screened for HTLV-I antibody.

D. Donor deferral and notification

The FDA recommends:

- permanent deferral of donors whose sera are repeatedly

reactive in ELISA and confirmed as positive for HTLV-I antibody by additional, more specific testing; such donors should be notified and counselled accordingly; and

- that donors whose serum specimens are repeatedly reactive in the ELISA but not confirmed as positive for HTLV-I antibody need not be notified on the first occassion. Although the donated units must be destroyed, the donors remain eligible for future donations. If however, the donors test repeatedly reactive in the ELISA on subsequent donation, they should be deferred indefinitely as donors and notified and counselled accordingly.

E. Guidelines for counselling

Counselling should be considered a routine adjunct depending on the results of HTLV-I testing. Given some of the uncertainties related to testing such as:

- the inability to distinguish between antibodies to HTLV-I and HTLV-II, and
- the low probability that disease will occur in seropositive persons,

every effort should be made to minimise the anxiety provoked by a repeatedly reactive screening test, particularly one that is not confirmed as HTLV-I seropositive by additional testing.

Persons confirmed as seropositive for HTLV-I should be:

- notified that they have antibody to HTLV-I and are likely to be infected with HTLV-I or HTLV-II;
- given information concerning disease associations and possible modes of transmission;
- advised that they have been permanently deferred as blood donors and should neither give blood for transfusion nor share needles that have been used for percutaneous injections or infusions with other persons; and
- (if applicable) discouraged from breastfeeding infants.

The paucity of data concerning sexual transmission of HTLV-I/HTLV-II does not permit a firm recommendation concerning sex practices; specific recommendations, such as the use of condoms to reduce the potential risk of sexual transmission, should be developed in consultation with a health-care professional.

Persons whose serum specimens are repeatedly reactive on more that one occasion in the ELISA but not confirmed as positive for HTLV-I antibody in more specific testing should be informed that they have inconclusive test results that do not necessarily imply infection with HTLV-I or HTLV-II. Nevertheless, they should be notified that they have been deferred indefinitely as donors and should not donate blood for transfusion. Periodic follow-up of such donors with ELISA, more specific serological tests, and possibily sophisticated techniques such as virus isolation and/or PCR may provide more reliable information regarding the presence of viral infection.

LICENSING OF SCREENING TESTS FOR ANTIBODY TO HTLV-I

Screening tests for antibody to HTLV-I, the first recognised human retrovirus, have been licensed by the FDA. These tests have been:

- recommended by the FDA for the screening of blood and cellular components donated for transfusion;
- approved as diagnostic tests, which may be useful in evaluating patients with clinical diagnoses of adult T-cell leukaemia/lymphoma (ATL) and tropical spastic paraparesis (TSP)/HTLV-I associated myelopathy (HAM), both of which have been associated with HTLV-I infection.

Because licensing will probably result in widespread use of these tests, the information presented below is provided for physicians and public health officials who may need to interpret HTLV-I test results and to counsel persons whose serum specimens are reactive in these tests:

- **Users of the new HTLV-I screening tests are cautioned that additional, more specific tests are necessary to confirm that serum specimens that are repeatedly reactive in these screening tests are truly positive for HTLV-I antibody.**
- Users should also beware that neither the screening tests nor more specific tests can distinguish between antibody to HTLV-I and antibody to the closely related human retrovirus, HTLV-II.
- **HTLV-I does not cause AIDS, and the finding of HTLV-I antibody in human blood does not imply infection with human immunodeficiency virus (HIV) or a risk of developing acquired immune deficiency syndrome (AIDS).**

CDI Editorial Comment

Blood banks in Australia are currently screening all blood donations for antibodies to the human immunodeficiency virus (HIV) but not for antibodies to any other retroviruses including the human T-lymphotropic virus type 1 (HTLV-I). The recent licensing of the ELISA screening tests for HTLV-I and the FDA's recommendation that that whole blood and cellular components donated for transfusion be screened for HTLV-I antibody could in time be adopted in Australia as part of routine screening of blood donations. However, in the absence of an estimated rate of HTLV-I seroprevalence in the general Australian population, a specificity of above 99% may still yield a low positive predictive value when the screening test is used in what is believed to be a low prevalence population residing in non-endemic areas. It would be feasible to accommodate the logistics of screening donated blood for antibody to HTLV-I through the laboratories currently licensed to carry out HIV testing; but, additional and more specific serological tests need to be standardised and made available to carry out confirmatory testing on serum specimens repeatedly reactive in the screening tests. Western immunoblot (WB) and radio-immunoprecipitation assay (RIPA) are specific tests required to determine whether a serum specimen is positive, indeterminate or negative; but no specific tests so far been licensed by the FDA.

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HIV-RELATED BELIEFS, KNOWLEDGE, AND BEHAVIOURS AMONG HIGH SCHOOL STUDENTS, USA

(Based on MMWR 1988;37:718-21)

In 1987, Centers for Disease Control (CDC), Atlanta, began to assist state and local departments of education is assessing human immunodeficiency virus (HIV)-related beliefs, knowledge, and behaviours among high school students in states and cities with the highest cumulative incidence of acquired immune deficiency syndrome (AIDS)(1,2). US departments of education will use the results of these surveys to plan school HIV education programs and to monitor temporal changes in HIV-related beliefs, knowledge, and behaviours among high school students. This report presents selected baseline data from surveys conducted during the spring of 1988.

A questionnaire for anonymous self-administration was developed collaboratively by representatives of 24 state and local departments of education, with technical assistance from CDC. The questionnaire contained 49 core questions: four to assess demographic characteristics of respondents, 33 to assess HIV-related beliefs and knowledge, and 12 to assess behaviours associated with HIV transmission. Each department of education that conducted the survey first completed the appropriate state or local survey review and approval process.

The survey included samples of students in grades 9-12 (ages 13-18 years) in each of six cities (Chicago, Los Angeles, New Orleans, New York City, San Francisco, and Seattle) and in each of nine states (California, District of Columbia, Kentucky, Michigan, New Jersey, New York, Ohio, Pennsylvania, and Washington). Samples from California, New York, and Washington excluded students in Los Angeles, San Francisco, New York City, and Seattle; data from these four cities were collected and analysed separately. Each site chose which of the 49 questions to administer; nearly every site obtained information using all the questions regarding demographic characteristics and HIV-related beliefs and knowledge. Four sites (California, District of Columbia, Michigan, and San Francisco) used the 12 questions to assess the extent to which students engage in behaviours that may result in HIV infection.

Sampling strategies were designed to obtain a representative sample of students and varied among sites. Most sites used a geographically stratified cluster sample, randomly selecting schools within strata, then selecting classes within each selected school. Other sites used a random sample of schools, then randomly selected students at each school. Using standardised procedures, classroom teachers or department heads administered questionnaires in required classes, e.g., health education or homeroom.

Sample sizes in each site ranged from 778 to 7013 students, and the response rate of schools from each site ranged from 52% to 100% (Table 1). Because response rates of schools from some sites were less than 100%, results cannot be generalised, and comparison of the results among sites should be made with caution. Results are presented by site as unweighted crude rates.

Table 1: Demographic characteristics and response rates of schools in selected cities and states, 1988

Site*	Sample size	School-level response rate† (%)	Gender (%)		Age group (yrs) (%)			Race/ethnicity (%)				
			Female	Male	13–14	15–16	17–18	White	Black	Hispanic	Asian	Other
State												
California	7013	64	51	49	10	49	42	59	7	20	9	5
District of Columbia	1275	100	55	45	2	70	28	3	90	3	3	2
Kentucky	778	73	55	45	26	71	3	91	8	0	1	1
Michigan	991	100	51	49	7	49	43	75	19	3	1	2
New Jersey	2287	100	53	47	16	30	55	56	27	13	2	1
New York	3841	100	49	51	10	50	39	NA§	NA	NA	NA	NA
Ohio	803	57	53	47	8	55	37	88	9	1	1	1
Pennsylvania	6668	97	52	48	32	43	25	68	21	6	2	2
Washington	1137	52	48	52	45	52	3	NA	NA	NA	NA	NA
City												
Chicago	1254	100	48	52	19	53	29	11	64	19	4	2
Los Angeles	2142	100	49	51	1	83	17	21	24	33	15	7
New Orleans	2366	100	54	46	37	46	16	9	84	2	4	1
New York City	2813	100	58	42	10	50	40	NA	NA	NA	NA	NA
San Francisco	802	88	52	48	10	75	14	12	13	14	56	6
Seattle	1069	100	47	53	22	37	41	52	18	3	22	6

*District of Columbia is categorized as a state for funding purposes.
†Number of schools conducting survey/number of schools sampled.
§Data not available.

Almost all respondents believed students their age should be taught about AIDS in school (range, 89.0% to 96.8%). Knowledge about sources for correct information about AIDS varied greatly among sites (range, 41.1% to 70.5%).

The range of students who knew that AIDS is not transmitted through shaking hands was 85.5% to 95.6%; through giving blood, 27.8% to 53.3%; from mosquito or other insect bites, 28.9% to 46.8%; from using public toilets, 41.8% to 64.6%; and from having a blood test, 49.6% to 75.4% (Table 2). A range of 83.8% to 98.4% of students knew that AIDS is transmitted by sharing needles or syringes used to inject drugs; 88.3% to 98.1% knew that AIDS is transmitted through sexual intercourse.

Table 2: Percentage of correct responses for questions measuring knowledge of HIV transmission, by selected cities and states, 1988

Site*	Nonrisk factor					Risk factor	
	Shaking hands	Giving blood	Insect bites	Using public toilets	Having a blood test	IV-drug use	Sexual intercourse
State							
California	92.4	44.5	36.4	56.2	62.3	94.8	95.7
District of Columbia	89.3	36.4	37.1	55.7	58.5	91.7	91.5
Kentucky	91.6	48.8	37.6	50.8	64.4	95.6	94.3
Michigan	93.5	49.1	37.3	54.4	66.6	96.4	96.2
New Jersey	93.7	45.3	40.7	59.5	61.6	95.9	96.5
New York	95.6	39.5	41.7	61.7	56.2	98.4	98.1
Ohio	92.0	53.3	39.1	59.4	64.6	96.6	95.7
Pennsylvania	93.0	49.0	46.8	64.6	63.9	NA†	NA
Washington	94.3	NA	40.1	59.7	75.4	97.7	96.5
City							
Chicago	89.3	28.0	30.4	54.1	58.1	89.1	88.3
Los Angeles	86.9	27.8	28.9	45.8	49.6	91.2	93.8
New Orleans	85.5	29.0	33.5	41.8	49.8	83.8	88.3
New York City	94.8	29.8	41.9	60.1	56.0	98.4	96.9
San Francisco	90.2	40.3	38.4	58.5	57.4	88.6	89.8
Seattle	91.7	41.9	42.8	60.0	59.9	96.4	95.9

*District of Columbia is categorized as a state for funding purposes.
†Data not available.

High school students from four sites reported variable rates of intravenous (IV)-drug use and sexual intercourse (Table 3): 2.8% to 6.3% reported ever injecting cocaine, heroin, or other illegal drugs; 28.6% to 76.4% reported having had sexual intercourse at least once. At each site, more male than female students and more older than younger students reported ever injecting illegal drugs or ever having had sexual intercourse.

The percentage of students who reported having had three or more sex partners ranged from 15.1% to 42.6%. At each site, more male than female students (range for males, 24.2% to 67.3%; for females, 8.3% to 25.6%) and more older than younger students reported three or more sex partners (range for 13- and 14-year-olds, 7.5% to 45.5%; for 15- and 16-year-old, 13.0% to 39.4%; and for 17- and 18-year-olds, 29.9% to 47.7%).

Table 3: Percentage of students reporting ever using IV drugs and ever having had sexual intercourse, age by sex, group, and selected cities and states, 1988

Site	Total (%)	Gender (%)		Age group (yrs) (%)		
		Female	Male	13–14	15–16	17–18
	IV-drug use					
California	4.1	2.6	5.7	2.8	3.9	4.3
District of Columbia	6.3	4.6	8.7	*	4.0	8.9
Michigan	2.8	2.1	3.4	3.2	3.2	1.3
San Francisco	3.7	2.4	5.1	1.4	3.9	2.4
	Sexual intercourse					
California	55.6	48.1	64.3	23.2	50.1	69.0
District of Columbia	76.4	65.6	90.7	*	71.4	89.8
Michigan	58.7	56.6	60.9	34.5	49.1	72.7
San Francisco	28.6	22.1	37.3	15.9	26.7	48.0

*Less than 5% of subgroup in sample.

MMWR Editorial Note

In the fall of 1987, CDC began providing fiscal and technical assistance to 15 state and 12 local departments of education that serve areas with the highest cumulative incidence of AIDS. The purpose of this assistance was to help schools implement effective HIV education programs. In the autumn of 1988, this assistance was extended to departments of education in the remaining states and territories and in four other local departments of education. Some state and local departments of education are initiating a unique school-based system to assess whether important HIV-related beliefs, knowledge, and behaviours of high school students in their respective states and cities change over time. In ensuing years, department of education staff plan to improve the representativeness and response rate of samples and to begin assessing changes in other important health behaviours (e.g., drinking and driving, cigarette smoking, exercise) among high school students.

Baseline data reported here suggest that HIV-related beliefs, knowledge, and behaviours among the adolescents surveyed in 15 states and cities are generally similar. Many students incorrectly thought that HIV infection may be acquired from giving blood, using public toilets, having a blood test or from mosquito and other insect bites. Most students knew sexual intercourse and IV-drug use can result in HIV infection.

Students who reported using IV drugs or having sexual intercourse, particularly with multiple partners, are at risk for HIV infection. Departments of education should implement programs to correct misperceptions about HIV transmission, to reduce behaviours resulting in HIV infection, and to assess periodically whether these misperceptions and behaviours change among high school students over time (3).

REFERENCES

1. Kolbe L, Jones J, Nelson G, et al. School health education to prevent the spread of AIDS: overview of a national program. Hygie 1988;7(3);10-3.
2. Kann L, Nelson GD, Jones JT, Kolbe L. Establishing a system of complementary school-based surveys to periodically assess AIDS-related knowledge, beliefs, and behaviours among adolescents. J Sch Health 1989 (in press).
3. CDC. Guidelines for effective school health education to prevent the spread of AIDS. MMWR 1988;37(suppl S-2).

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 14/12/88 TO 4/1/89

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) NVH(ACT)
6. CODE 113 - PHH POW(NSW)
7. CODE 114 - RAHC(NSW)
8. CODE 115 - STATE LAB(QLD)

		019	065	110	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	1	4	2	7	3	2	15	34
0101	ADENOVIRUS TYPE 1	4	3	2	2	1	0	0	12
0102	ADENOVIRUS TYPE 2	4	2	1	1	0	0	0	8
0103	ADENOVIRUS TYPE 3	2	1	2	0	0	0	0	5
0104	ADENOVIRUS TYPE 4	4	0	0	1	0	0	0	5
0105	ADENOVIRUS TYPE 5	0	2	0	3	0	0	0	5
0106	ADENOVIRUS TYPE 6	0	0	0	1	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	0	1	1	0	0	0	2
0108	ADENOVIRUS TYPE 8	0	0	0	1	0	0	0	1
0130	ADENOVIRUS TYPE 30	0	0	0	3	0	0	0	3
0131	ADENOVIRUS TYPE 31	0	1	0	0	0	0	0	1
0135	ADENOVIRUS TYPE 35	0	1	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	2	0	0	0	0	1	0	3
0201	INFLUENZA A VIRUS	1	2	2	0	1	0	0	6
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	0	0	1	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	11	5	1	4	1	2	5	29
0400	RESPIRATORY SYNCYTIAL VIRUS (R	5	5	2	0	0	0	0	12
0500	RHINOVIRUS (ALL TYPES)	7	4	21	0	0	4	4	40
0600	MYCOPLASMA PNEUMONIAE	13	11	35	10	1	0	0	70
0700	ORNITHOSIS-PSITTACOSIS	4	0	0	0	1	0	0	5
0816	COXSACKIEVIRUS A16	0	0	0	0	0	1	0	1
0824	COXSACKIEVIRUS A24 = ECHOVIRUS	1	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	5	0	0	1	2	0	0	8
0905	COXSACKIEVIRUS B5	0	1	1	0	0	0	0	2
1004	ECHOVIRUS TYPE 4	3	0	0	0	0	0	0	3
1006	ECHOVIRUS TYPE 6	0	3	0	0	0	0	0	3
1007	ECHOVIRUS TYPE 7	0	1	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	7	17	0	1	0	0	0	25
1019	ECHOVIRUS TYPE 19	0	1	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	1	0	0	0	1
1030	ECHOVIRUS TYPE 30	32	3	0	2	0	0	0	37
1100	POLIOVIRUS NOT TYPED	0	0	0	0	1	0	0	1
1101	POLIOVIRUS TYPE 1	3	0	0	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	1	3	3	0	0	1	0	8
1200	MUMPS VIRUS	0	0	0	3	1	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	5	3	0	1	1	0	1	11
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	7	0	94	0	2	0	103
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	37	17	1	4	2	0	61
1303	VARICELLA-ZOSTER VIRUS	7	7	0	5	3	0	6	28
1306	HERPES SIMPLEX TYPE 1	103	70	20	4	0	0	31	228
1307	HERPES SIMPLEX TYPE 2	116	114	12	49	0	0	39	330
1399	HERPES VIRUS TYPING PENDING	2	0	0	0	0	1	0	3
1401	COXIELLA BURNETI	0	0	2	11	1	0	0	14
1502	PICORNIA VIRUS - NOT TYPED = E	0	4	0	1	4	0	11	20
1521	MEASLES VIRUS	1	0	0	2	0	0	0	3
1522	RUBELLA VIRUS	17	1	0	0	0	0	0	18
1532	HEPATITIS B ANTIGEN	29	26	13	24	11	1	12	116
1535	HEPATITIS A ANTIBODY	4	5	0	0	0	0	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	83	86	28	24	1	0	22	244
1556	CMV - CYTOMEGALOVIRUS	24	10	1	9	10	3	9	66
1564	ROTAVIRUS	10	20	4	13	2	4	2	55
1566	NORWALK AGENT	0	0	0	1	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	4	1	0	5
9992	ROSS RIVER VIRUS	33	45	2	0	0	0	0	80
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	2	0	2
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	1	0	0	0	0	0	0	1
TOTAL		545	505	172	281	53	28	157	1741

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

PERIOD 14/12/88 TO 4/1/89

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

		1	2	3	4	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	11	0	0	0	0	19	0	0	0	0	31
0101	ADENOVIRUS TYPE 1	2	7	0	0	0	0	1	0	0	0	0	10
0102	ADENOVIRUS TYPE 2	1	6	0	0	0	0	1	0	0	0	0	8
0103	ADENOVIRUS TYPE 3	0	3	0	0	0	0	0	0	0	0	1	4
0104	ADENOVIRUS TYPE 4	0	0	0	0	0	0	1	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	2	0	0	0	0	3
0107	ADENOVIRUS TYPE 7	1	1	0	0	0	0	0	0	0	0	0	2
0130	ADENOVIRUS TYPE 30	0	1	0	0	0	0	2	0	0	0	0	3
0135	ADENOVIRUS TYPE 35	0	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	1	0	0	0	0	0	0	0	1
0201	INFLUENZA A VIRUS	1	4	0	0	0	0	0	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	26	0	1	0	0	0	0	0	0	0	27
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	12	0	0	0	0	0	0	0	0	0	12
0500	RHINOVIRUS (ALL TYPES)	1	38	0	0	0	0	0	0	0	0	0	39
0600	MYCOPLASMA PNEUMONIAE	7	46	0	0	0	0	0	0	0	0	0	53
0700	ORNITHOSIS-PSITTACOSIS	0	4	0	0	0	0	0	0	0	0	0	4
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	0	0	0	1	1
0904	COXSACKIEVIRUS B4	0	4	0	0	0	0	1	0	0	0	1	6
0905	COXSACKIEVIRUS B5	0	1	0	0	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	1	0	0	2	0	0	0	0	0	0	0	3
1007	ECHOVIRUS TYPE 7	0	1	0	0	0	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	4	3	0	9	0	1	3	0	0	0	2	22
1019	ECHOVIRUS TYPE 19	0	0	0	0	0	0	1	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	0	0	0	0	0	1	1
1030	ECHOVIRUS TYPE 30	2	1	0	31	0	0	0	0	0	0	0	34
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	1	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	2	0	0	0	0	0	0	0	0	0	2
1102	POLIOVIRUS TYPE 2	2	0	0	0	0	0	2	0	0	0	1	5
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	0	8	8
1301	HERPES SIMPLEX VIRUS - NOT TYP	27	0	0	1	0	0	0	0	0	0	19	47
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	19	6	0	0	0	0	0	2	0	0	1	28
1303	VARICELLA-ZOSTER VIRUS	1	1	0	0	1	1	0	0	0	0	19	23
1306	HERPES SIMPLEX TYPE 1	2	8	0	1	0	0	0	0	0	0	133	144
1307	HERPES SIMPLEX TYPE 2	7	1	0	0	0	0	1	0	0	0	125	134
1399	HERPES VIRUS TYPING PENDING	0	0	1	0	0	0	0	0	0	0	0	1
1401	COXIELLA BURNETI	1	0	0	0	0	0	0	0	0	0	0	1
1502	PICORNIA VIRUS - NOT TYPED = E	2	5	0	0	0	1	11	0	0	0	1	20
1521	MEASLES VIRUS	2	0	0	0	0	1	0	0	0	0	0	3
1522	RUBELLA VIRUS	1	1	0	0	0	0	0	0	0	0	15	17
1532	HEPATITIS B ANTIGEN	55	0	0	0	0	0	1	49	0	1	0	106
1535	HEPATITIS A ANTIBODY	4	0	0	0	0	0	0	3	0	0	0	7
1541	CHLAMYDIA A - C. TRACHOMATIS	44	0	0	0	0	0	0	0	0	0	0	44
1556	CMV - CYTOMEGALOVIRUS	8	11	0	0	0	1	1	2	2	6	1	32
1564	ROTAVIRUS	0	0	0	0	0	0	54	0	0	0	0	54
1566	NORWALK AGENT	0	0	0	0	0	0	0	0	0	1	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	5	0	0	0	0	5
9992	ROSS RIVER VIRUS	13	24	0	0	0	0	0	0	0	0	7	44
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	2	0	0	0	0	2
TOTAL		209	231	2	46	1	5	109	56	2	8	336	1005

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

PERIOD 14/12/88 TO 4/1/89

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

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	1	0	0	0	0	1	0	0	1	0	3
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	1	0	1	0	0	2
0103	ADENOVIRUS TYPE 3	0	0	0	0	0	0	0	1	0	0	1
0104	ADENOVIRUS TYPE 4	3	0	0	0	0	0	0	0	1	0	4
0105	ADENOVIRUS TYPE 5	0	0	0	0	0	0	0	1	1	0	2
0106	ADENOVIRUS TYPE 6	0	1	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	0	0	1
0131	ADENOVIRUS TYPE 31	0	0	0	1	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	0	0	2	0	2
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	0	0	1	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	1	1	0	2
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	0	0	1
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	1	8	8	0	17
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	0	1	0	0	1
0824	COXSACKIEVIRUS A24 = ECHOVIRUS	1	0	0	0	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	0	0	0	0	0	0	2	0	0	2
0905	COXSACKIEVIRUS B5	0	0	0	0	0	0	0	1	0	0	1
1006	ECHOVIRUS TYPE 6	1	0	0	0	0	0	0	1	1	0	3
1009	ECHOVIRUS TYPE 9	0	0	0	0	0	0	1	2	0	0	3
1030	ECHOVIRUS TYPE 30	0	0	0	0	0	0	0	2	1	0	3
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	0	1	2	3
1200	MUMPS VIRUS	0	0	0	0	0	0	1	0	2	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	0	3	0	0	0	0	0	0	0	0	3
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	53	0	0	0	0	0	1	2	0	56
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	17	8	0	0	0	6	2	0	33
1303	VARICELLA-ZOSTER VIRUS	0	0	0	1	0	0	0	2	2	0	5
1306	HERPES SIMPLEX TYPE 1	9	68	0	0	0	0	0	3	4	0	84
1307	HERPES SIMPLEX TYPE 2	0	195	0	0	0	0	0	0	1	0	196
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	0	1	0	2
1401	COXIELLA BURNETI	0	0	0	0	1	0	4	4	4	0	13
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	0	1	0	1
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	5	5	0	10
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	2	0	0	2
1541	CHLAMYDIA A - C. TRACHOMATIS	3	195	0	0	0	0	0	0	2	0	200
1556	CMV - CYTOMEGALOVIRUS	0	2	0	1	1	5	0	7	18	0	34
1564	ROTAVIRUS	0	0	0	0	0	0	1	0	0	0	1
9992	ROSS RIVER VIRUS	0	0	0	1	34	0	0	1	0	0	36
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	0	1	0	0	1
TOTAL		19	518	17	12	36	7	8	54	63	2	736