

CENTERS FOR DISEASE CONTROL

MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

Vol. 2

Reports on AIDS

JUNE 1986 - MAY 1987

ise Control
Atlanta, Georgia 30333

PUBLIC HEALTH SERVICE
DEPARTMENT OF HEALTH AND HUMAN SERVICES

110 930

This publication is the second of two volumes that contain all articles related to AIDS that have appeared in the *Morbidity and Mortality Weekly Report* (MMWR), published by the Centers for Disease Control (CDC). These articles, arranged in chronological order, track the reporting of information on AIDS from 1981, when CDC first published information on Kaposi's sarcoma and *Pneumocystis carinii* pneumonia occurring in young homosexual men. During that year, CDC formed a task force to establish risk factors, carry out laboratory studies, and disseminate timely information on the disease now known as the acquired immunodeficiency syndrome (AIDS).

NGJRS

APR 28 1988

ACQUISITIONS

The Centers for Disease Control, Public Health Service, funds an AIDS Hotline which operates 24 hours a day, seven days a week, and can be reached at 1-800-342-AIDS.

Table of Contents

JUNE-DECEMBER 1986

Recommendations for providing dialysis treatment to patients infected with human T-lymphotropic virus, type III/lymphadenopathy-associated virus. MMWR 1986 June 13;35:376-78,383	1
Transfusion-associated human T-lymphotropic virus, type III/lymphadenopathy-associated virus infection from a seronegative donor - Colorado. MMWR 1986 June 20;35:389-91	3
Human T-lymphotropic virus, type III/lymphadenopathy-associated virus antibody prevalence in U.S. military recruit applicants. MMWR 1986 July 4;35:421-24	5
Diagnosis and management of mycobacterial infection and disease in persons with human T-lymphotropic virus, type III/lymphadenopathy-associated virus infection. MMWR 1986 July 18;35:448-52	8
Human T-lymphotropic virus, type III/lymphadenopathy-associated virus: Agent summary statement. MMWR 1986 Aug 29;35:540-42,547-49	11
Tuberculosis and acquired immunodeficiency syndrome — Florida. MMWR 1986 Sept 19, 35:587-90	15
Immunization of children infected with human T-lymphotropic virus, type III/lymphadenopathy-associated virus. MMWR 1986 Sept 26;35:595-98,603-06	18
Acquired immunodeficiency syndrome (AIDS) in Western Palm Beach County, Florida. MMWR 1986 Oct 3;35:609-12	23
Acquired immunodeficiency syndrome (AIDS) among blacks and Hispanics—United States. MMWR 1986 Oct 24;35:655-58,663-66	26
Surveillance of hemophilia-associated acquired immunodeficiency syndrome. MMWR 1986 Oct 31;35:669-71	32
Positive HTLV-III/LAV antibody results for sexually active female members of social/sexual clubs - Minnesota. MMWR 1986 Nov 14;35:697-99	34
Update: Acquired immunodeficiency syndrome — United States. MMWR 1986 Dec 12;35:757-66	36

JANUARY-MAY 1987

Survey of Non-U.S. Hemophilia Treatment Centers for HIV seroconversions following therapy with heat-treated factor concentrates. MMWR 1987 Mar 13, 36:121-24	40
Tuberculosis and AIDS — Connecticut. MMWR 1987 Mar 13;36:133-35	43
Human immunodeficiency virus infection in transfusion recipients and their family members. MMWR 1987 Mar 20;36:137-40	45
Antibody to human immunodeficiency virus in female prostitutes. MMWR 1987 Mar 27;36:157-61	48
Self-reported changes in sexual behaviors among homosexual and bisexual men from the San Francisco city clinic cohort. MMWR 1987 Apr 3;36:187-89	52
Classification system for human immunodeficiency virus (HIV) infection in children under 13 years of age. MMWR 1987 Apr 24;36:225-36	54

Trends in human immunodeficiency virus infection among civilian applicants for military service — United States, October 1985-December 1986. MMWR 1987 May 15; 36:273-76	59
Update: Human immunodeficiency virus infections in health-care workers exposed to blood of infected patients. MMWR 1987 May 22;36:285-89	61
Human immunodeficiency virus infection transmitted from an organ donor screened for HIV antibody — North Carolina. MMWR 1987 May 29;36:306-08	65
Index	67

Recommendations for Providing Dialysis Treatment to Patients Infected with Human T-Lymphotropic Virus Type III/Lymphadenopathy-Associated Virus

Patients with end-stage renal disease who are undergoing maintenance dialysis and who have manifestations of human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV)* infection, including acquired immunodeficiency syndrome (AIDS), or who are positive for antibody to HTLV-III/LAV can be dialyzed in hospital-based or free-standing dialysis units using conventional infection-control precautions. Standard blood and body fluid precautions and disinfection and sterilization strategies routinely practiced in dialysis centers are adequate to prevent transmission of HTLV-III/LAV.

Soon after AIDS was recognized in the United States, it became apparent that risk factors for persons with AIDS were similar to risk factors for persons with hepatitis B virus (HBV) infection (1). Prevention measures applied to control HBV infection in health-care institutions were used as a model to develop infection-control guidelines for patients with AIDS before the identification of the etiologic agent and the development of serologic tests for antibody to HTLV-III/LAV (anti-HTLV-III). Isolation of infected patients and nonreuse of a dialyzer by the same patient were initially recommended for patients receiving dialysis in dialysis centers (2). These strategies are not currently believed necessary for preventing HTLV-III/LAV transmission.

No transmission of HTLV-III/LAV infection in the dialysis-center environment has been reported (3), and the possibility of such transmission appears extremely unlikely when routine infection-control precautions are followed (4). The routine infection-control precautions used in all dialysis centers when dialyzing all patients are considered adequate to prevent HTLV-III/LAV transmission. These would include: blood precautions; routine cleaning and disinfection of dialysis equipment and surfaces that are frequently touched; and restriction of nondisposable supplies to individual patients unless such supplies are sterilized between uses (2).

The following recommendations take into consideration recent knowledge about HTLV-III/LAV and update infection-control strategies for dialyzing patients infected with HTLV-III/LAV:

1. Procedures for environmental control and for disinfection and sterilization of hemodialysis machines have been described (5). The hemodialysis machine pumps dialysis fluid into the dialyzer (artificial kidney) where circulating blood from the patient is separated from the dialysis fluid by a membrane. The dialyzer, along with the associated blood lines, is disposable. Strategies for disinfecting the dialysis fluid pathways of the hemodialysis machine are targeted to control bacterial contamination and generally consist of using about 500-750 ppm of sodium hypochlorite for 30-40 minutes or 1.5%-2.0% formaldehyde overnight. In addition, several chemical germicides formulated to disinfect dialysis machines are commercially available. None of these protocols or procedures need to be altered after dialyzing patients infected with HTLV-III/LAV. Chemical germicides used for disinfection and sterilization of devices in the dialysis center are effective against HTLV-III/LAV (4).
2. Patients infected with HTLV-III/LAV can be dialyzed by either hemodialysis or peritoneal dialysis and do not need to be isolated from other patients. The type of dialysis treatment (i.e., hemodialysis or peritoneal dialysis) should be based on the needs of the patient. The dialyzer may be discarded after each use. Alternatively, centers that have dialyzer-reuse programs, in which a specific dialyzer is issued to a specific patient, removed, cleaned, disinfected, and reused several times on the same patient only, may include HTLV-III/LAV-infected patients in the dialyzer-reuse program. An individual dialyzer must never be used on more than one patient.
3. Standard infection-control strategies that are used routinely in dialysis units for all dialysis patients and personnel should be used to prevent HTLV-III/LAV transmission. Specifically, these strategies include blood precautions and barrier techniques, such as the use of gloves, gowns, and handwashing techniques, that have been described elsewhere (4-8).
4. Precautions against needlestick injuries, as well as the appropriate use of barrier precautions, such as wearing gloves when handling items contaminated with blood or serum, should be practiced by all personnel caring for all dialysis patients. Such injuries constitute the major potential risk for HTLV-III/LAV transmission to personnel. Extraordinary care should be taken to prevent injuries to hands caused by needles, scalpels, and other sharp instruments or devices during procedures; when cleaning used instruments; during disposal of used needles; and when handling sharp instruments following procedures.

*An international committee on taxonomy has proposed the name human immunodeficiency virus (HIV).

After use, disposable syringes and needles, scalpel blades, and other sharp items must be placed in puncture-resistant containers for disposal. To prevent needlestick injuries, needles should not be recapped; purposefully bent or broken; removed from disposable syringes; or otherwise manipulated by hand. No data are currently available from controlled studies examining the effect, if any, of the use of needle-cutting devices on the incidence of needlestick injuries.

Reported by Hospital Infections Program, AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: In a study of 520 dialysis patients, 25 were reactive for anti-HTLV-III/LAV by enzyme immunoassay (EIA), but only four were confirmed by the Western blot technique (3). The rate of falsely reactive EIA tests among these dialysis patients was 4%, much higher than the falsely reactive rate for blood donors (0.17%). The rate of truly reactive tests was 0.8%, much lower than in high-risk groups but higher than in blood donors. The higher rate of falsely reactive tests is probably due to the exposure of dialysis patients to H9-cell-associated antigens during blood transfusions that are common among these patients. These antigens are also present in cell lines used to grow HTLV-III/LAV for use as reagents in serologic tests for anti-HTLV-III/LAV (9). Identification of antibody to H9 lymphoid cell lines in the absence of isolation of HTLV-III/LAV in dialysis patients with reactive EIA and nonreactive Western blot tests supports the conclusion that these test results are falsely reactive. The higher rate of truly reactive tests most likely reflects the frequency of blood transfusion in this patient population before initiation of blood donor screening for anti-HTLV-III/LAV. None of the four infected persons identified in that study were dialyzed in the same dialysis center.

CDC is initiating a cooperative study to further assess the prevalence of anti-HTLV-III/LAV among patients undergoing chronic hemodialysis. Representatives of dialysis centers who are interested in participating in such a study and who regularly have more than 60 patients on dialysis should contact the Hospital Infections Program, Center for Infectious Diseases, CDC, Building 1, Room 5065, Atlanta, Georgia 30333 (telephone [404] 329-3406).

References

1. Curran JW, Evatt BL, Lawrence DN. Acquired immune deficiency syndrome: the past as prologue. *Ann Intern Med* 1983;98:401-2.
2. Favero MS. Recommended precautions for patients undergoing hemodialysis who have AIDS or non-A, non-B hepatitis. *Infect Control* 1985;6:301-5.
3. Peterman TA, Lang GR, Mikos NJ, et al. HTLV-III/LAV infection in hemodialysis patients. *JAMA* 1986;255:2324-6.
4. CDC. Summary: recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in the workplace. *MMWR* 1985;34:681.
5. Favero MS. Dialysis-associated diseases and their control. In: Bennett JV, Brachman PS, eds. *Hospital infections*. Boston: Little, Brown and Company, Inc., 1985:267-84.
6. CDC. Hepatitis—control measures for hepatitis B in dialysis centers. *Viral hepatitis*: Atlanta, Georgia: Center for Disease Control, 1977: HEW publication no. (CDC)78-8358 (Investigation and control series, November 1977).
7. CDC. Hepatitis surveillance report no. 49. Issued January 1985:3-4.
8. Garner JS, Simmons BP. CDC guideline for isolation precautions in hospitals. *Infect Control* 1983;5:245-325.
9. CDC. Update: Public Health Service workshop on human T-lymphotropic virus type III antibody testing—United States. *MMWR* 1985;34:477-8.

Transfusion-Associated Human T-Lymphotropic Virus Type III/ Lymphadenopathy-Associated Virus Infection From a Seronegative Donor — Colorado

In November 1985, a blood donor at a Colorado blood-collection center was found to be seropositive for human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV)* antibody by both the enzyme-linked immunosorbent assay (ELISA) and Western blot methods. He had previously donated at the center in April and August 1985, when he had been seronegative by ELISA. Both recipients from the August donation, one of whom had no other risk factors for acquisition of HTLV-III/LAV, were subsequently found to be seropositive. Both recipients of the April donation were seronegative. The donor had probably been infected through sexual contact 12 weeks or less before the August donation. This is the first reported transmission of infection from a blood donor that has occurred despite routine screening for HTLV-III/LAV antibody in blood banks and plasma centers.

Details of the donor and recipient investigation are as follows:

Donor. The donor was a 31-year-old man who had donated blood at the same center in April, August, and November 1985. He was seronegative in April (optical densities of Abbott ELISA on sample/control = 0.052/0.160) and August (0.034/0.142), but seropositive by ELISA (0.926/0.173) and Western blot in November. His blood from the November donation was discarded, and physicians of the recipients from the August donation were notified by the blood center of the possible transmission of HTLV-III/LAV from these blood products.

When interviewed in April 1986, the donor stated that he had had sexual contact with one male partner, with the first exposure taking place on May 15, 1985. No condoms were used. His only other sexual partner was a man in 1974. He denied intravenous (IV) drug use or history of blood transfusion. He had no history of acute viral illnesses or symptoms of acquired immunodeficiency syndrome (AIDS) or AIDS-related complex (ARC) in 1985 or 1986. Physical examination in December 1985 was normal. Repeat ELISA testing in April 1986 revealed a high absorbency value ($> 2.000/0.125$), and Western blot was once again positive. Attempts at locating previous sera for antibody testing were unsuccessful.

Donor's Partner. The donor's sexual partner was a 22-year-old man who corroborated the donor's history of their initial sexual contact on May 15, 1985. He had been homosexually active since 18 years of age. He denied IV drug abuse or history of blood transfusion. After notification by the donor of his positive antibody status, the partner was tested for HTLV-III/LAV in November 1985 and was seropositive by ELISA and Western blot; these findings were reconfirmed on a separate specimen in April 1986. He had not previously been tested for HTLV-III/LAV antibody.

Recipient 1. Recipient 1 was a 60-year-old man who underwent surgery in August 1985. He received from 15 different donors six units of packed red blood cells, four units of fresh frozen plasma, and six units of platelets (including one unit from the previously described donor). He had been married for 30 years and denied extramarital sexual contact, either heterosexual or homosexual, or any previous blood transfusions or IV drug abuse. In February 1986, he had no symptoms of AIDS or ARC and had a normal physical examination. The HTLV-III/LAV antibody test was positive by ELISA and Western blot and reconfirmed on a separate specimen in March 1986. His wife was seronegative for HTLV-III/LAV antibody in April 1986.

Recipient 2. Recipient 2 was a 57-year-old man who underwent surgery in August 1985. He received two units of platelet-poor whole blood (including one unit from the previously described donor) and one unit of packed red blood cells. During the postoperative period, he had unexplained fever and diarrhea that persisted for 6 weeks and was associated with a 20-pound weight loss. Stool specimens were negative for bacterial pathogens and ova and parasites, including cryptosporidia. In October 1985, he was tested for HTLV-III/LAV antibody for reasons unrelated to the blood transfusion and was positive by ELISA and Western blot, which was confirmed on a separate specimen in April 1986. He had been divorced for 12 years and was strictly homosexual since that time, with multiple partners.

Other investigative findings. The blood donated in April 1985 was given to two recipients, and both were seronegative by ELISA when tested in May 1986.

*The Human Retrovirus Subcommittee of the International Committee on the Taxonomy of Viruses has proposed the name human immunodeficiency virus (HIV) for this virus. (Science 1986;232:697)

One other person was a common donor to recipients 1 and 2 in August 1985. This person was retested in April 1986 and was negative by ELISA for HTLV-III/LAV antibody. Of the 13 remaining donors to recipient 1, 11 were seronegative when retested 5 months or more after the August donations. Two donors reside outside Colorado and have not been retested. Of the two remaining donors to recipient 2, both were seronegative when retested 6 months or more after the August donations.

Reported by CA Raevsky, DL Cohn, MD, FC Wolf, MPA, FN Judson, MD, Colorado Dept of Health, Denver Disease Control Svc, SW Ferguson PhD, State Epidemiologist, TM Vernon, MD, Executive Director, Colorado Dept of Health; AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: This is the first report of HTLV-III/LAV transmission from a person whose blood tested negative for HTLV-III/LAV antibody at the time of blood donation. As with previous reports that have documented the presence of the virus in a small number of persons who have no detectable antibody, this donor appears to have had a recent infection (1,2). Most infected people develop antibody within 2-3 months of infection (2-6).

The current risk of transfusion-associated infection is small. The prevalence of positive Western blot tests among units screened by the American Red Cross in early 1985 suggests that 0.04% of all donated units may have been potentially infectious (7). This prevalence declined to 0.02% in early 1986 (8). Currently available screening tests detect HTLV-III/LAV antibody in the great majority of infected persons. Since antibody may not be detectable in blood from donors with very recent infections, the safety of the blood supply also requires deferral of donation by persons at increased risk for HTLV-III/LAV infection.

Donor-deferral programs, initially implemented in blood banks in March 1983 and subsequently refined, provide all prospective donors with educational information on the practices associated with an increased risk of HTLV-III/LAV infection. Evidence suggests that most persons at increased risk have stopped donating blood (9-11), but a few such individuals continue to donate. The donor described in this report said he felt he was not at risk for infection because he had only one sexual partner. Although a steady sexual relationship with a single partner is generally safer with regard to HTLV-III/LAV infection than relationships with multiple sexual partners, men who have had sexual contact with another man since 1977 must not donate blood (12).

Efforts are continuing to assure maximum effectiveness of donor-deferral programs (13,14). As an example, blood collection agencies have agreed to implement procedures in which prospective donors are asked to sign an expanded consent statement. The statement indicates that the prospective donor has reviewed and understands the informational material provided and that donors who are at increased risk for transmission of HTLV-III/LAV or other infectious agents will not donate blood or plasma for transfusion to another person.

References

1. Salahuddin SZ, Groopman JE, Markham PD, et al. HTLV-III in symptom-free seronegative persons. *Lancet* 1984;ii:1418-20.
2. Ho DD, Sarngadharan MG, Resnick L, Dimarzo-Veronese F, Rota TR, Hirsch MS. Primary human T-lymphotropic virus type III infection. *Ann Intern Med* 1985;103:880-3.
3. Anonymous. Needlestick transmission of HTLV-III from a patient infected in Africa. *Lancet* 1984;ii:1376-7.
4. Tucker J, Ludlam CA, Craig A, et al. HTLV-III infection associated with glandular-fever-like illness in a haemophiliac. *Lancet* 1985;i:585.
5. Cooper DA, Gold J, Maclean P, et al. Acute AIDS retrovirus infection. *Lancet* 1985;i:537-40.
6. Esteban JI, Shih J W-K, Tai C-C, Bodner AJ, Kay JWD, Alter HJ. Importance of Western blot analysis in predicting infectivity of anti-HTLV-III/LAV positive blood. *Lancet* 1985;ii:1083-6.
7. Schorr JB, Berkowitz A, Cummings PD, Katz AJ, Sandler SG. Prevalence of HTLV-III antibody in American blood donors [Letter]. *N Engl J Med* 1985;313:384-5.
8. Sandler SG, American Red Cross. Personal communication.
9. Dahlke MB. Designated blood donations [Letter]. *N Engl J Med* 1984;310:1194.
10. Grindon A. Efficacy of voluntary self-deferral of donors at high risk of AIDS [Abstract]. *Transfusion* 1984;24:434.
11. Pindyc J, Waldman A, Zang E, Oleszko W, Lowy M, Bianco C. Measures to decrease the risk of acquired immunodeficiency syndrome transmission by blood transfusion. *Transfusion* 1985;25:3-9.
12. CDC. Update: revised Public Health Service definition of persons who should refrain from donating blood and plasma—United States. *MMWR* 1985;34:547-8.
13. Kalish RI, Cable RG, Roberts SC. Voluntary deferral of blood donations and HTLV-III antibody positivity [Letter]. *N Engl J Med* 1986;314:1115-6.
14. U.S. Public Health Service. Public Health Service plan for the prevention and control of AIDS and the AIDS virus. Report of the Coolfont Planning Conference, June 4 to 6, 1986. Washington, D.C.: U.S. Public Health Service, 1986.

Human T-Lymphotropic Virus Type III/Lymphadenopathy-Associated Virus Antibody Prevalence in U.S. Military Recruit Applicants

From October 1, 1985, through March 31, 1986, as part of medical evaluation of individuals volunteering for military service, the U.S. Department of Defense tested 308,076 recruit applicants for serologic evidence of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV), the etiologic retrovirus of acquired immunodeficiency syndrome (AIDS).^{*} Blood samples were obtained at 71 Military Entrance Processing Stations. The screened population consisted predominately of young adults in their late teens (54%) and early twenties (33% were 20-25 years old). Eighty-five percent were male, and 77% were white. Sera were tested by a single contracting laboratory using a commercial human T-lymphotropic virus type III (HTLV-III) enzyme-linked immunosorbent assay (ELISA) test (Electronucleonics, Inc.). All samples repeatedly reactive by ELISA were also subjected to confirmation testing by the Western blot. Blots were considered positive if antibodies to gp 41 and/or p24+p55 were detected. Recruit applicants with confirmed HTLV-III/LAV antibody are excluded from military service.

The mean prevalence of confirmed positive tests was 1.5 per 1,000 recruit applicants. Antibody prevalence increased progressively with age (Table 1), a pattern consistent throughout the country (Table 2). The seroprevalence was higher among the 265,361 men of all ages,

^{*}The AIDS virus has been variously termed human T-lymphotropic virus type III (HTLV-III), lymphadenopathy-associated virus (LAV), AIDS-associated retrovirus (ARV), or human immunodeficiency virus (HIV). The designation human immunodeficiency virus (HIV) has recently been proposed by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (Science 1986;232:697).

TABLE 1. Prevalence of HTLV-III/LAV antibody* among military recruit applicants, by age — United States, October 1985-March 1986

Age (yrs)	No. tested	Positives/1,000 [†]
17	59,113	0.2
18	61,452	0.4
19	43,978	0.8
20	29,835	1.1
21-25	73,998	2.5
≥26	39,700	4.4
All ages	308,076	1.49

*Western blot confirmed.

[†]Rates/1,000 tested.

TABLE 2. Prevalence of HTLV-III antibody* per 1,000 military recruit applicants tested, by region and age group — United States, October 1985-March 1986

Region [†]	No. tested	Age group (yrs)			All ages
		17-20	21-25	≥ 26	
New England	14,131	0.3 [§]	1.0 [§]	1.9 [§]	0.6
Mid-Atlantic	43,196	0.9	4.4	10.1	2.8
E.N. Central	55,943	0.2	2.0	2.2	0.8
W.N. Central	26,850	0.2 [§]	1.1 [§]	1.4	0.6
S. Atlantic	50,854	0.7	3.3	5.7	1.9
E.S. Central	21,027	0.4 [§]	2.2	1.1 [§]	0.9
W.S. Central	34,782	0.7	2.5	2.6	1.4
Mountain	19,015	0.3 [§]	1.8	2.6	1.1
Pacific	39,260	0.7	1.5	4.7	1.5
All [¶]	308,076	0.5	2.5	4.4	1.5

*Western blot confirmed.

[†]Defined in notifiable diseases table (Table III).

[§]Rate based on five or fewer positives.

[¶]Includes data from Puerto Rico, Virgin Islands, Guam, American Samoa, Northern Marianas, and the Trust Territories.

1.6/1,000, than among the 42,715 women, 0.6/1,000. The ratio of male-to-female prevalence rates was 3:1. Prevalence also varied by race: for the 237,586 whites, the rate was 0.9/1,000; for the 55,185 blacks, 3.9/1,000; and for the 15,305 applicants of other racial groups, 2.6/1,000. The relationships of seroprevalence rates by sex and race remain when the data are adjusted by age.

Seroprevalence rates (Table 2) were highest in the coastal regions of the country other than New England. Rates were lowest in New England and in the inland regions. Based on preliminary analysis by county, the highest HTLV-III antibody rates were found in recruit applicants from major urban centers and lowest in those from rural areas.

Reported by the Health Studies Task Force, Office of the Assistant Secretary of Defense (Health Affairs); Dept of Virus Disease, Div of Preventive Medicine, Walter Reed Army Institute of Research; Surveillance and Evaluation Br, AIDS Program, Center for Infectious Disease, CDC.

Editorial Note: Although there is considerable knowledge regarding the distribution of reported cases of AIDS in the United States (1), there has been much less information about the prevalence of infection with HTLV-III/LAV. Studies of HTLV-III/LAV antibody prevalence have primarily involved selected high-risk groups, including homosexual men (24%-68% positive) (2-5), intravenous (IV) drug abusers (2%-72% positive) (6-8), and hemophilia patients (40%-88% positive) (9-11). The limited published data from blood-bank screening programs, where persons in high-risk groups are specifically discouraged from donating, indicate a confirmed antibody prevalence nationally of less than 0.4/1,000 (12).

The Department of Defense medical evaluation program provides additional information on the geographic and demographic factors associated with HTLV-III/LAV infection in the United States. The population of individuals volunteering for military service may not be representative of the U.S. population at large due to the spontaneous, if partial, self-exclusion of hemophilia patients, actively homosexual men, and current IV drug abusers. However, the data suggest the following: (1) White: the highest seroprevalence occurs among those over 25 years old, the age of acquisition of confirmed antibody (and by implication, infection) can often be in the late teens and early twenties. Age at diagnosis of reported AIDS is older, with a median of 32-35 years, depending on risk group, race, and sex. Only 0.7% of reported cases among adults/adolescents occur between 13 and 20 years of age; 6.5% develop between 21 and 25 years; the remaining 92.8% are diagnosed at or after 26 years of age. (2) The ratio of seroprevalence between male and female recruit applicants is 3:1. This is much lower than the ratio of 13:1 observed among all AIDS cases, but like the 3:1 ratio among other AIDS patients if homosexual and hemophilia-associated cases are excluded. (3) The ratio of seroprevalence rates of black to white recruit applicants (4:1) is intermediate between the 2.6 relative risk for blacks among all AIDS patients (25.2% of cases are among non-Hispanic blacks, who comprise 11.5% of the population [13]) and the 8.3 relative risk for blacks among AIDS patients not associated with either homosexuality or hemophilia (blacks comprise 52.0% of these cases). The data do not yet permit a detailed analysis of seroprevalence differences by Hispanic ethnicity. (4) The geographic distribution of seroprevalence among recruits is generally consistent with the incidence of cases, both by region and by urban versus rural residence. More detailed geographic analysis will be possible when cumulative data are available from screening additional recruits.

As in the case with serologically positive blood donors (14), recruit applicants with confirmed positive antibody are informed of their status and its implication regarding infection with HTLV-III/LAV; they are counseled on reducing the risk of transmission to others through sexual contact, sharing IV needles, or other exchanges of blood or body fluids.

Counseling and testing for HTLV-III/LAV antibody should be offered to persons who may have already been infected as a result of intimate contact with the seropositive recruit applicant (i.e., sexual partners, persons with whom needles have been shared, infants born to seropositive mothers). In addition, seropositive individuals should be interviewed by an experienced investigator to determine their risk factors for infection. This, coupled with observation on suitable controls, would facilitate determining modes of acquisition and evaluating current trends in risk of exposure to the virus in these populations.

The continued analysis of data emerging from the HTLV-III/LAV serologic screening of military recruit applicants will permit the examination of the extent and the trends over time of infection with the causative agent of AIDS in this sentinel population.

References

1. Peterman TA, Drotman DP, Curran JW. Epidemiology of the acquired immunodeficiency syndrome (AIDS). *Epidemiol Rev* 1985;7:1-21.
2. Phair J. Prevalence and correlates of HTLV-III antibodies among 5000 gay men in 4 cities. Multicenter AIDS Cohort Study (MACS) [Abstract]. 25th Interscience Conference on Antimicrobial Agents and Chemotherapy. Minneapolis: American Society for Microbiology, 1985:229.

3. Collier AC, Barnes RC, Handsfield HH. Prevalence of antibody to LAV/HTLV-III among homosexual men in Seattle. *Am J Public Health* 1986;76:564-5.
4. Schwartz K, Visscher BR, Detels R, Taylor J, Nishanian P, Fahey JL. Immunological changes in lymphadenopathy virus positive and negative symptomless male homosexuals: two years of observation [Letter]. *Lancet* 1985;ii:831-2.
5. Darrow WW, Jaffe HW, O'Malley PM, et al. Sexual practices and HTLV-III/LAV infections in a cohort of homosexual male clinic patients, San Francisco [Abstract]. 6th International Meeting of the International Society for STD Research. Brighton: International Society for STD Research, 1985:31.
6. Levy N, Carlson JR, Hinrichs S, Lerche N, Schenker M, Gardner MB. The prevalence of HTLV-III/LAV antibodies among intravenous drug users attending treatment programs in California: a preliminary report [Letter]. *N Engl J Med* 1986;314:446.
7. Weiss SH, Ginzburg HM, Goedert JJ, et al. Risk for HTLV-III exposure and AIDS among parenteral drug abusers in New Jersey [Abstract]. Atlanta: International Conference on Acquired Immunodeficiency Syndrome (AIDS), 1985:44.
8. Spira TJ, DesJarlais DC, Bokos D, et al. HTLV-III/LAV antibodies in intravenous drug (IV) abusers—comparison of high and low risk areas for AIDS [Abstract]. Atlanta: International Conference on Acquired Immunodeficiency Syndrome (AIDS), 1985:84.
9. Ragni MV, Tegtmeier GE, Handwerk-Leber C, Lewis, JH, Mayer WL, Spero JA. Prevalence and seroconversion of human T-lymphotropic retrovirus (HTLV-III) antibody in patients with hemophilia [Abstract]. Atlanta: International Conference on Acquired Immunodeficiency Syndrome (AIDS), 1985:74.
10. Jason J, McDougal JS, Holman RC, et al. Human T-lymphotropic retrovirus type III/lymphadenopathy-associated virus antibody. Association with hemophiliacs' immune status and blood component usage. *JAMA* 1985;253:3409-15.
11. Goedert JJ, Sarngadharan MG, Eyster ME, et al. Antibodies reactive with human T cell leukemia viruses in the serum of hemophiliacs receiving factor VIII concentrate. *Blood* 1985;65:492-5.
12. Schorr JB, Berkowitz A, Cumming PD, Katz AJ, Sandler SG. Prevalence of HTLV-III antibody in American blood donors [Letter]. *N Engl J Med* 1985;313:384-5.
13. U.S. Bureau of the Census. Table 49: general characteristics of persons by spanish origin and race: 1980. In: 1980 Census of Population. Volume 1: Characteristics of the Population. Washington, D.C.: U. S. Department of Commerce, 1980:1-52 (General population characteristics, [PC 80-1-B1] United States summary).
14. CDC. Provisional Public Health Service inter-agency recommendations for screening donated blood and plasma for antibody to the virus causing acquired immunodeficiency syndrome. *MMWR* 1985;35:1-5.

Diagnosis and Management of Mycobacterial Infection and Disease in Persons with Human T-Lymphotropic Virus Type III/ Lymphadenopathy-Associated Virus Infection

In 1985, the number of new tuberculosis cases reported to CDC was essentially the same as that reported in 1984 (1). In contrast, the average annual decline in morbidity during the past 32 years has been 5%. The failure of tuberculosis morbidity to decline as expected in 1985 is probably related to the occurrence of tuberculosis among persons with acquired immunodeficiency syndrome (AIDS) or human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV/LAV)* infection. Several reports have indicated that mycobacterial disease is common among AIDS patients and among persons at risk for AIDS (2-9). The most common mycobacterial species isolated from patients with diagnosed AIDS is *Mycobacterium avium* complex (MAC), although in some groups in which tuberculous infection is highly prevalent, disease caused by *M. tuberculosis* is more common (10-12). Even among groups in which MAC is the most common mycobacterial pathogen, *M. tuberculosis* accounts for a substantial proportion of the mycobacterial isolates. The association between mycobacterial disease and AIDS raises several important clinical and public health issues that are addressed below.

DIAGNOSIS OF TUBERCULOSIS IN PATIENTS LIKELY TO HAVE HTLV-III/LAV INFECTION

Clinicians should consider the diagnosis of tuberculosis in patients with, or at risk of, HTLV-III/LAV infection, even if the clinical presentation is unusual (4,13,14). Available data indicate that extrapulmonary forms of tuberculosis, particularly lymphatic and disseminated (miliary), are seen much more frequently among patients with HTLV-III/LAV infection than among those without such infection. Pulmonary tuberculosis in patients with HTLV-III/LAV infection cannot readily be distinguished from other pulmonary infections, such as *Pneumocystis carinii* pneumonia, on the basis of clinical and radiographic findings. Patients with tuberculosis may have infiltrates in any lung zone, often associated with mediastinal and/or hilar lymphadenopathy. Cavitation is uncommon. Appropriate specimens to establish a culture-confirmed diagnosis of tuberculosis include respiratory secretions, urine, blood, lymph node, bone marrow, liver, or other tissue or body fluid that is indicated clinically. All tissue specimens should be stained for acid-fast bacilli and cultured for mycobacteria. In the presence of undiagnosed pulmonary infiltrates, bronchoscopy with lavage and transbronchial biopsy (if not contraindicated) may be needed to obtain material for both culture and histologic examination. A tuberculin skin test should be administered, but the absence of a reaction does not rule out the diagnosis of tuberculosis because immunosuppression associated with HTLV-III/LAV infection may cause false-negative results.

TREATMENT OF MYCOBACTERIAL DISEASE IN A PATIENT WITH HTLV-III/LAV INFECTION

Chemotherapy should be started whenever acid-fast bacilli are found in a specimen from a patient with HTLV-III/LAV infection and clinical evidence of mycobacterial disease. Because it is difficult to distinguish tuberculosis from MAC disease by any criterion other than culture, and because of the individual and public health implications of tuberculosis, it is important to treat patients with a regimen effective against tuberculosis. With some exceptions, patients with tuberculosis and HTLV-III/LAV infection respond relatively well to standard antituberculosis drugs (15); however, their treatment should include at least three drugs initially, and treatment may need to be longer than the standard duration of 9 months (16). The recommended regimen is isoniazid (INH), 10-15 mg/kg/day up to 300 mg/day; rifampin (RIF), 10-15 mg/kg/day up to 600 mg/day; and either ethambutol (EMB), 25 mg/kg/day, or pyrazinamide (PZA), 20-30 mg/kg/day. The last two drugs are usually given only during the first 2 months of therapy. The addition of a fourth drug may be indicated in certain situations, such as central nervous system or disseminated disease or when INH resistance is suspected. An initial drug-susceptibility test should always be performed, and the treatment regimen, revised if resistance is found to any of the drugs being used. The appropriate duration of treatment for patients with tuberculosis and HTLV-III/LAV infection is unknown; however, it is recommended that treatment continue for a minimum of 9 months and for at least 6 months after documented culture conversion. If INH or RIF is not included in the treatment regimen, therapy should continue for a minimum of 18 months and for at least 12 months following culture conversion. After therapy is completed, patients should be followed closely, and mycobacteriologic examinations should be repeated if clinically indicated.

*The Human Retrovirus Subcommittee of the International Committee on the Taxonomy of Viruses has proposed the name human immunodeficiency virus (HIV) for this virus (Science 1986;232:697).

Some clinicians would take a different approach to treatment than that outlined above, to cover the possibility of MAC disease. Although the clinical significance and optimal therapy of MAC disease in these patients is not well defined, and there are no definitive data on the efficacy of treatment, one regimen commonly used to treat MAC disease substitutes rifabutin (ansamycin LM 427) for rifampin, combined with INH, EMB, and clofazimine. Rifabutin and clofazimine are experimental drugs available to qualified investigators only under investigational new drug protocols. Rifabutin is distributed by the CDC Drug Service (telephone: [404] 329-3670), and clofazimine, by Ciba-Geigy: (telephone: [201] 277-5787). If *M. tuberculosis* is isolated from a patient receiving this four-drug regimen, treatment should be switched to one of the three-drug regimens outlined above (INH, RIF, and EMB or PZA). If MAC is isolated from a patient who has been started on a three-drug regimen, the clinician may continue the three-drug regimen or switch to the four-drug regimen of INH, EMB, rifabutin, and clofazimine.

Although experience is very limited, patients with disease due to *M. kansasii* should respond to INH, RIF, and EMB. Some clinicians advocate the addition of streptomycin (SM), 1 gram twice weekly, for the first 3 months. Therapy should continue for a minimum of 15 months following culture conversion.

Monitoring for toxicity of antimycobacterial drugs may be difficult for patients who may be receiving a variety of other drugs and may have other concomitant conditions. Because hepatic and hematologic abnormalities may be caused by the mycobacterial disease, AIDS, or other drugs and conditions, the presence of such abnormalities is not an absolute contraindication to the use of the treatment regimens outlined above.

INFECTION CONTROL

Recommendations for preventing transmission of HTLV-III/LAV infection to health-care workers have been published (17). In addition, infection-control procedures applied to patients with HTLV-III/LAV infection who have undiagnosed pulmonary disease should always take the possibility of tuberculosis into account. This is especially true when diagnostic procedures, such as sputum induction or bronchoscopy, are being performed. Previously published guidelines for preventing tuberculosis transmission in hospitals should be followed (18).

CONTACT INVESTIGATION FOR TUBERCULOSIS

Patients with pulmonary tuberculosis and HTLV-III/LAV infection should be considered potentially infectious for tuberculosis, and standard procedures for tuberculosis contact investigation should be followed (19). Specific data on the infectiousness of tuberculosis in patients with HTLV-III/LAV infection are not yet available.

EXAMINING HTLV-III/LAV-INFECTED PERSONS FOR TUBERCULOSIS AND TUBERCULOUS INFECTION

Individuals who are known to be HTLV-III/LAV seropositive should be given a Mantoux skin test with 5 tuberculin units of purified protein derivative as part of their clinical evaluation. Although some false-negative skin test results may be encountered in this setting as a result of immunosuppression induced by HTLV-III/LAV infection, significant reactions are still meaningful (20). If the skin test reaction is significant, a chest radiograph should be obtained, and if abnormalities are detected, additional diagnostic procedures for tuberculosis should be undertaken. Patients with clinical AIDS or other Class IV HTLV-III/LAV infections (21) should receive *both* a tuberculin skin test and a chest radiograph because of the higher probability of false-negative tuberculin reactions in immunosuppressed patients.

EXAMINING PATIENTS WITH CLINICALLY ACTIVE TUBERCULOSIS OR LATENT TUBERCULOUS INFECTION FOR HTLV-III/LAV INFECTION

As part of the evaluation of patients with tuberculosis and tuberculous infection, risk factors for HTLV-III/LAV should be identified. Voluntary testing of all persons with these risk factors is recommended (22). In addition, testing for HTLV-III/LAV antibody should be considered for patients of all ages who have severe or unusual manifestations of tuberculosis. The presence of HTLV-III/LAV infection has implications regarding treatment (see above), alerts the physician to the possibility of other opportunistic infections, and allows for counselling about transmission of HTLV-III/LAV infection (23). Testing for HTLV-III/LAV antibody is especially important for persons over age 35 with *asymptomatic* tuberculous infection, because INH would not usually be indicated for persons in this age group unless they are also HTLV-III/LAV seropositive.

PREVENTIVE THERAPY

HTLV-III/LAV seropositivity in a person of any age with a significant tuberculin reaction is an indication for INH preventive therapy (16). Although it is not known whether INH therapy is as efficacious in preventing tuberculosis in HTLV-III/LAV-infected persons as in other

groups, the usually good response of HTLV-III/LAV-infected persons with tuberculosis to standard therapy suggests that INH preventive therapy would also be effective. Before instituting preventive therapy, clinically active tuberculosis should be excluded.

Developed by Center for Prevention Svcs, Center for Infectious Diseases, CDC, with consultation from: RS Holzman, MD, New York University Medical Center, New York City; PC Hopewell, MD, San Francisco General Hospital Medical Center, California; AE Pitchenik, MD, University of Miami Medical Center, Florida; LB Reichman, MD, University of Medicine and Dentistry of New Jersey, New Jersey Medical School, University Hospital, Newark, New Jersey; RL Stoneburner, MD, New York City Dept of Health.

References

1. CDC. Tuberculosis—United States, 1985—and the possible impact of human T-lymphotropic virus type III/lymphadenopathy-associated virus infection. MMWR 1986;35:74-6.
2. Cohen RJ, Samoszuk MK, Busch D, Lagios M. [Letter]. Occult infections with *M. intracellulare* in bone-marrow biopsy specimens from patients with AIDS. N Engl J Med 1983;308:1475-6.
3. Wong B, Edwards FF, Kiehn TE, et al. Continuous high-grade *Mycobacterium avium-intracellulare* bacteremia in patients with the acquired immunodeficiency syndrome. Am J Med 1985;78:35-40.
4. Pitchenik AE, Cole C, Russell BW, Fischl MA, Spira TJ, Snider DE, Jr. Tuberculosis, atypical mycobacteriosis, and the acquired immunodeficiency syndrome among Haitian and non-Haitian patients in south Florida. Ann Intern Med 1984;101:641-5.
5. Macher AM, Kovacs JA, Gill V, et al. Bacteremia due to *Mycobacterium avium-intracellulare* in the acquired immunodeficiency syndrome. Ann Intern Med 1983;99:782-5.
6. Zakowski P, Fligiel S, Berlin GW, Johnson L, Jr. Disseminated *Mycobacterium avium-intracellulare* infection in homosexual men dying of acquired immunodeficiency. JAMA 1982;248:2980-2.
7. Greene JB, Sidhu GS, Lewis S, et al. *Mycobacterium avium-intracellulare*: a cause of disseminated life-threatening infection in homosexuals and drug abusers. Ann Intern Med 1982;97:539-46.
8. Chan J, McKittrick JC, Klein RS. *Mycobacterium gordonae* in the acquired immunodeficiency syndrome [Letter]. Ann Intern Med 1984;101:400.
9. Eng RH, Forrester C, Smith SM, Sobel H. *Mycobacterium xenopi* infection in a patient with acquired immunodeficiency syndrome. Chest 1984;86:145-7.
10. Pape JW, Liautaud B, Thomas F, et al. Characteristics of the acquired immunodeficiency syndrome (AIDS) in Haiti. N Eng J Med 1983;309:945-50.
11. Maayan S, Wormser GP, Hewlett D, et al. Acquired immunodeficiency syndrome (AIDS) in an economically disadvantaged population. Arch Intern Med 1985;145:1607-12.
12. Goedert JJ, Weiss SH, Biggar RJ, et al. Lesser AIDS and tuberculosis [Letter]. Lancet 1985;ii:52.
13. Sunderam G, Maniatis T, Kapila R, et al. *Mycobacterium tuberculosis* disease with unusual manifestations is relatively common in acquired immuno-deficiency syndrome (AIDS) [Abstract]. Am Rev Resp Dis 1984;129 (part 2):A191.
14. Pitchenik AE, Robinson HA. The radiographic appearance of tuberculosis in patients with the acquired immune deficiency syndrome (AIDS) and pre-AIDS. Am Rev Resp Dis 1985;131:393-6.
15. Sunderam G, McDonald RJ, Maniatis T, Oleske J, Kapila R, Reichman LB. Tuberculosis as a manifestation of the acquired immunodeficiency syndrome (AIDS). JAMA 1986;256:357-61.
16. American Thoracic Society. Treatment of tuberculosis and other mycobacterial diseases. Am Rev Resp Dis 1983;127:790-6.
17. CDC. Recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in the workplace. MMWR 1985;34:681-95.
18. CDC. Guidelines for prevention of TB transmission in hospitals. Atlanta, Georgia: U.S. Department of Health and Human Services, 1982: HHS publication no. (CDC) 82-8371.
19. American Thoracic Society/CDC. Control of tuberculosis. Am Rev Resp Dis 1983;128:336-42.
20. American Thoracic Society. The tuberculin skin test. Am Rev Resp Dis 1981;124:356-63.
21. CDC. Classification system for human T-lymphotropic virus type III/lymphadenopathy-associated virus infections. MMWR 1986;35:334-9.
22. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.
23. CDC. Human T-lymphotropic virus type III/lymphadenopathy-associated virus antibody testing at alternate sites. MMWR 1986;35:284-7.

Human T-Lymphotropic Virus Type III/ Lymphadenopathy-Associated Virus: Agent Summary Statement

INTRODUCTION

In March 1984, CDC and the National Institutes of Health (NIH), in consultation with scientists, physicians, and public health workers in academia, industry, and government, published a manual entitled *Biosafety in Microbiological and Biomedical Laboratories* ("biosafety manual")* (1). The manual describes combinations of standard and special microbiologic practices, safety equipment, and facilities recommended for working with infectious agents in various laboratory settings. The recommendations are advisory and provide a voluntary code of safety practices.

A section of this manual is devoted to a number of specific "agent summary statements" consisting of brief descriptions of documented or anecdotal laboratory-associated infections, the nature of the laboratory hazards, and recommended precautions to be taken in handling and working with certain infectious agents. Contributors to the manual recognized that new agents would be discovered from time to time and recommended that a summary statement for each new agent be developed and published in the *MMWR*. The summary statement for human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV)[†] follows. All laboratory directors are requested to put a copy of this summary in each of their copies of the biosafety manual and bring it to the attention of laboratory personnel. The recommendations in the summary statement were compiled from published scientific reports and are consistent with the published guidelines for health-care workers (2-4).

AGENT SUMMARY STATEMENT: HTLV-III/LAV

As of August 15, 1986, no cases of acquired immunodeficiency syndrome (AIDS) that meet the CDC case definition and can be attributed to an inadvertent laboratory exposure have been reported in laboratory workers (5). One laboratory worker (7) was included among the health-care workers who have had HTLV-III/LAV antibody detected in their serum after sustaining a needlestick injury (2,3,6-10), but the source of the infection could not be established. Persons who are infected with HTLV-III/LAV may be asymptomatic, may have AIDS-related complex, or may manifest symptoms of overt AIDS (11).

In 1985, two different reagent production laboratories reported that several laboratory workers may have been inadvertently exposed to an aerosol of concentrated HTLV-III/LAV; one worker was cut by a piece of glass from a broken carboy that contained HTLV-III/LAV-infected cells and culture fluid. None of the potentially exposed persons had shown evidence of seroconversion after 6 months in one incident and 12 months in the other as a result of these occupational exposures.

Other reports dealing with HTLV-III/LAV infection in health-care personnel, including laboratory workers (3,4,6,8-10), indicate that the risk of bloodborne transmission from inadvertent exposure is considerably less for HTLV-III/LAV than for hepatitis B virus infection. These reports illustrate the need for complete evaluation by a physician and serologic testing of each laboratory worker definitely or possibly exposed to HTLV-III/LAV in a laboratory setting. It is recommended that the Public Health Service guidelines for health-care workers be followed in these instances (2,3).

Laboratory Hazards

HTLV-III/LAV has been isolated from blood, semen, saliva, tears, urine, cerebrospinal fluid, brain tissue, and cervical secretions and is likely to be present in other body fluids, secretions, and tissues of infected humans or experimentally infected nonhuman primates. Percutaneous or parenteral inoculation and direct contact of cuts, scratches, abrasions, or mucosal surfaces with suspensions of virus or specimens containing live virus are considered potential routes of infection. Possible transmission of infection via the parenteral route can occur through self-inoculation with needles, broken glass, or other sharp objects that contain HTLV-III/LAV. Spillage is a possible means of exposure and infection, especially spills accompanied by spraying or splashing of infected cell cultures, viral concentrates, and other infectious materials that

*Available from Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, Stock #01702300167-1, Price: \$4.00; and from National Technical Information Service, U.S. Department of Commerce, 5285 Port Royal Road, Springfield, Virginia 22161, Stock #PB84-206879, Price: \$6.00.

[†]The Human Retrovirus Subcommittee of the International Committee on the Taxonomy of Viruses has proposed the name human immunodeficiency virus (HIV) for these viruses (Science 1986;232:697).

may come into direct contact with abraded skin or mucous membranes of the eyes, nose, or mouth; however, there are no data documenting or suggesting that transmission of HTLV-III/LAV has occurred in this manner. Ingestion and inhalation have not been documented as modes of transmission of the virus.

Recommended Precautions

1. Biosafety Level (BSL) 2 standards and special practices, containment equipment, and facilities as described in the CDC-NIH biosafety manual are recommended for activities involving clinical specimens, body fluids, or tissues from humans or laboratory animals that may contain HTLV-III/LAV. *These are the same practices recommended for all clinical specimens.* Emphasis is placed on the following practices, which are included in the manual (1):
 - a. Use of syringes, needles and other sharp instruments should be avoided if possible. Used needles and cutting instruments should be discarded into a puncture-resistant container with a lid. Needles should *not* be resheathed, purposefully bent, broken, removed from disposable syringes, or otherwise manipulated by hand.
 - b. Gloves should be worn by all personnel engaged in activities that may involve skin contact with potentially infectious fluids, tissues, or cultures and by laboratory workers with dermatitis or other lesions on the hands who may have direct or indirect contact with potentially infectious materials. Handwashing with soap and water should be a routine practice immediately after direct contact with potentially infectious materials and on completion of work, even when gloves are worn.
 - c. Generation of aerosols, splashes, and spills of potentially infectious materials should be avoided in procedures involving body fluids or tissues, during necropsy of cadavers, and in similar procedures on animals experimentally infected with HTLV-III/LAV. Laboratory workers should use a biological safety cabinet when propagating the virus to further reduce the risk of exposure. Although the major precautions are listed here, the CDC-NIH biosafety manual contains additional related precautions (see pages 11-13 for BSL 2 and pages 14-17 [1] for BSL 3 when large volumes or concentrates of HTLV-III/LAV are involved). In all instances, the laboratory director is responsible for assessing the biosafety level to be used.
 - d. Human serum from any source that is used as a control or reagent in a test procedure should be handled at BSL 2 (see pages 11-13 [1]). Appended to this Agent Summary Statement is a statement (Addendum 1) issued by CDC on the use of all human control or reagent sera shipped to other laboratories. The Food and Drug Administration requires that manufacturers of human serum reagents use a similarly worded statement.
 - e. Animal BSL 2 practices, containment equipment, and facilities are recommended for activities involving nonhuman primates experimentally infected with HTLV-III/LAV. Laboratory coats, gowns, or uniforms should be worn by laboratory workers, as is customary for other BSL 2 or 3 practices, depending on the nature of the work, concentration of the virus, and volume of material being handled. Because many animals bite, and some throw feces, urine, or expectorate at humans, animal-care personnel must wear coats, protective gloves, coveralls or uniforms, and face shields as appropriate to protect the skin and mucous membranes of the eyes, nose, and mouth from potential exposure to these substances when working with animals likely to manifest such behavior.
2. Activities such as growing research-laboratory-scale amounts of HTLV-III/LAV or related viruses or virus-producing cell lines, working with concentrated virus preparations, or conducting procedures that may produce droplets or aerosols should be performed in a BSL 2 facility with the additional practices and containment equipment recommended for BSL 3 (12).
3. Activities involving industrial-scale, large-volume, or high-concentration production and manipulation of HTLV-III/LAV are to be conducted with BSL 3 requirements (12).
4. All laboratory glassware, equipment, disposable materials, and wastes suspected or known to contain HTLV-III/LAV must be decontaminated, preferably in an autoclave, before washing, discarding, etc. Incineration of solid wastes may be used as an alternate method of disposal.
5. There is no evidence that laboratory clothing soiled with materials known or suspected to contain HTLV-III/LAV poses a transmission hazard, and the handling of such clothing is covered under BSL 2 practices. However, to be consistent with BSL 3 recommendations (1), when laboratory clothing becomes contaminated with HTLV-III/LAV preparations, it should be decontaminated before being laundered or discarded.
6. Work surfaces should be decontaminated at the end of each day on completion of procedures or when overtly contaminated. Many commonly used chemical disinfectants with such active ingredients as sodium hypochlorite, formaldehyde, glutaraldehyde, or phenols

(4,13-15) can be used to decontaminate laboratory work surfaces; they can also be used to decontaminate some laboratory instruments, specific areas of contaminated laboratory clothing, and spills of infectious materials. Prompt decontamination of spills and other overt contamination should be standard practice.

7. The prudent and recommended approach to handling human serum known or suspected to contain HTLV-III/LAV is to use the same precautions that should be used routinely to prevent transmission of bloodborne infections, including hepatitis B (16). Available data on the effectiveness of heat to destroy HTLV-III/LAV suspected or known to be present in human serum are at variance because of variations in volume of serum, concentration of the virus, temperature, and duration of exposure to heat (14,15,17). Similarly, results of chemical analyses or antibody assays may vary when sera are heated before testing according to the analysis or assay being performed (18-20). However, there is agreement that testing heated serum for HTLV-III/LAV antibody by enzyme immunoassays often yields false-positive results (21-23).
8. No HTLV-III/LAV vaccine has been developed, and no drugs have been shown to be safe and effective for therapy. As part of an ongoing medical surveillance program for employees, all laboratory workers before being assigned to activities with a high potential for exposure should have a serum sample obtained and stored at -40 C (-40 F) for possible future testing. Subsequent serum samples should be obtained and stored in accordance with laboratory policy or following an inadvertent laboratory exposure involving materials described above. When indicated, these serum specimens should be tested by a qualified laboratory using currently recommended procedures for HTLV-III/LAV antibody. Furthermore, the physician requesting serologic testing of these serum specimens must first obtain informed consent from the laboratory worker and describe the confidentiality safeguards available to protect test results. The laboratory workers whose serum specimens are to be tested should understand how the test results are to be used, the implications of a positive or negative test result, and the limits, if any, of the confidentiality safeguards. An employee whose serum HTLV-III/LAV antibody test is reactive and whose subsequent tests and evaluation confirm the presence of HTLV-III/LAV infection should be counseled to follow the Public Health Service recommendations for preventing transmission (24,25).
9. In addition to HTLV-III/LAV, other primary, as well as opportunistic, pathogenic agents may be present in the body fluids and tissues of persons who are antibody positive or have AIDS-related complex or AIDS. Laboratory workers should follow accepted biosafety practices to ensure maximum protection against inadvertent laboratory infection with agents other than HTLV-III/LAV that may also be present in clinical specimens.

Reported by Div of Safety, National Institute of Allergy and Infectious Diseases, National Cancer Institute, National Institutes of Health; AIDS Program, Hospital Infections Program, Center for Infectious Diseases, Laboratory Program Office, Office of Biosafety, Office of the Director, CDC.

ADDENDUM

CDC cautionary notice for all human serum samples used as controls or reagents:

WARNING: Because no test method can offer complete assurance that laboratory specimens do not contain HTLV-III/LAV, hepatitis B virus, or other infectious agents, this specimen(s) should be handled at the BSL 2 as recommended for any potentially infectious human serum or blood specimen in the CDC-NIH manual, *Biosafety in Microbiological and Biomedical Laboratories*, 1984, pages 11-3.

One or more of the following statements should be included with the above warning statement:

- ⊙ This specimen is negative for hepatitis B surface antigen (HBsAg).
- ⊙ This specimen is negative for antibody to HTLV-III/LAV.
- ⊙ This specimen is positive for hepatitis B surface antigen (HBsAg).
- ⊙ This specimen is positive for antibody to HTLV-III/LAV.
- ⊙ This specimen has NOT been tested for hepatitis B surface antigen (HBsAg).
- ⊙ This specimen has NOT been tested for antibody to HTLV-III/LAV.
- ⊙ This specimen has been heated at 56 C (133 F) for 30 minutes (which will not inactivate HBsAg but will inactivate HTLV-III/LAV).

References

1. Richardson JH, Barkley WE, eds. Biosafety in microbiological and biomedical laboratories, 1984. Washington, DC: US Department of Health and Human Services, Public Health Service. HHS publication no. (CDC) 84-8395.
2. CDC. Update: evaluation of human T-lymphotropic virus type III/lymphadenopathy-associated virus infection in health-care personnel—United States. MMWR 1985;34:575-8.
3. CDC. Recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in the workplace. MMWR 1985;34:682-6, 691-5.
4. CDC. Acquired immune deficiency syndrome (AIDS): precautions for clinical and laboratory staffs. MMWR 1982;32:577-80.
5. CDC. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. MMWR 1985;34:373-5.
6. Weiss SH, Saxinger WC, Richtman D, et al. HTLV-III infection among health care workers: association with needlestick injuries. JAMA 1985;254:2089-93.
7. Anonymous. Needlestick transmission of HTLV-III from a patient infected in Africa. Lancet 1984; ii:1376-7.
8. Henderson DK, Saah AJ, Zak BJ, et al. Risk of nosocomial infection with human T-cell lymphotropic virus type III/lymphadenopathy-associated virus in a large cohort of intensively exposed health-care workers. Ann Intern Med 1986;104:644-7.
9. Stricof RL, Morse DL. HTLV-III/LAV seroconversion following a deep intramuscular needlestick injury [Letter]. New Engl J Med 1986;314:1115.
10. McCray E and the Cooperative Needlestick Surveillance Group. Occupational risk of the acquired immunodeficiency syndrome among health care workers. N Engl J Med 1986;314:1127-32.
11. CDC. Classification system for human T-lymphotropic virus type III/lymphadenopathy-associated virus infections. MMWR 1986;35:334-9.
12. U.S. Department of Health and Human Services, Public Health Service. Biosafety guidelines for use of HTLV-III and related viruses. Federal Register 1984, 49: Number 201, 40556.
13. Favero MS. Sterilization, disinfection and antisepsis in the hospital. In: Lennette EH, Balows A, Hausler WJ, Jr, Shadomy HJ, eds. Manual of clinical microbiology. Washington, DC: American Society for Microbiology, 1985:129-37.
14. Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type III/lymphadenopathy-associated virus. J Infect Dis 1985;152:400-3.
15. Resnick L, Veren K, Salahuddin SZ, Tondreau S, Markham PD. Stability and inactivation of HTLV-III/LAV under clinical and laboratory environments. JAMA 1986;255:1887-91.
16. Favero MS, Petersen NJ, Bond WW. Transmission and control of laboratory-acquired hepatitis infection. In: Miller BM, Groschel DHM, Richardson JH, et al., eds. Laboratory safety: principles and practice. Washington, DC: American Society for Microbiology, 1986:49-58.
17. Ronalds CJ, Grint PCA, Kangro HD. Disinfection and inactivation of HTLV-III/LAV [Letter]. J Infect Dis 1986;153:996.
18. Goldie DJ, McConnell AA, Cooke PR. Heat treatment of whole blood and serum before chemical analysis [Letter]. Lancet 1985; i:1161.
19. Lai L, Ball G, Stevens J, Shanson D. Effect of heat treatment of plasma and serum on biochemical indices [Letter]. Lancet 1985; i:1457-8.
20. Evans RP, Shanson DC. Effect of heat on serological tests for hepatitis B and syphilis and on aminoglycoside assays [Letter]. Lancet 1985; i:1458.
21. Van den Akker R, Hekker AC, Osterhaus ADME. Heat inactivation of serum may interfere with HTLV-III/LAV serology [Letter]. Lancet 1985; ii:672.
22. Mortimer PP, Parry JV, Mortimer JY. Which anti-HTLV III/LAV assays for screening and confirmatory testing? Lancet 1985; ii:873-7.
23. Jungkind DL, DiRenzo SA, Young SJ. Effect of using heat-inactivated serum with the Abbott human T-cell lymphotropic virus type III antibody test. J Clin Microbiol 1986;23:381-2.
24. CDC. Prevention of acquired immune deficiency syndrome (AIDS): report of inter-agency recommendations. MMWR 1983;32:101-3.
25. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.

Tuberculosis and Acquired Immunodeficiency Syndrome — Florida

In 1985, 1,425 tuberculosis cases were reported in Florida, an increase of almost 7% over the 1,335 cases reported in 1984. Concern about a possible association between human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV)* infection and increased tuberculosis morbidity (1,2) led to an evaluation of data on acquired immunodeficiency syndrome (AIDS) and tuberculosis. Four subgroups of persons were identified and their characteristics compared: (1) AIDS patients with and without tuberculosis (AIDS/TB and AIDS/non-TB, respectively), and (2) tuberculosis patients with and without AIDS (TB/AIDS and TB/non-AIDS, respectively). The overlapping subgroups of AIDS/TB and TB/AIDS are listed separately only because their characteristics were analyzed from two discrete data bases.

AIDS PATIENTS WITH AND WITHOUT TUBERCULOSIS

Of the 1,094 persons meeting the CDC surveillance definition of AIDS (3) reported from Florida in the period 1981-1985, 109 (10%) were also diagnosed in the period 1978-1985 as having tuberculosis.† The number of AIDS patients with tuberculosis by year of AIDS diagnosis rose progressively from zero in 1981 to a peak of 55 in 1984; this number fell to 26 in 1985. The interval between report of tuberculosis and diagnosis of AIDS ranged from 7 years before to 15 months after AIDS was diagnosed (median interval, 3 months before AIDS diagnosis). Sixty-two (57%) of the patients were reported to have tuberculosis more than 1 month before they were diagnosed as having AIDS; 30 (28%), within a month before or after they were diagnosed as having AIDS; and 17 (16%), more than a month after they were diagnosed as having AIDS.

AIDS/TB patients were similar to AIDS/non-TB patients with respect to age and sex (Table 3). However, AIDS/TB patients were more frequently black (81%) than were AIDS/non-TB patients (37%), were more frequently foreign born (60% versus 25%), and were less frequently homosexual or bisexual men (21% versus 62%).

*Subcommittee of the International Committee for the Taxonomy of Viruses has proposed that HTLV-III/LAV be officially designated as "Human Immunodeficiency Virus" or HIV.

†These time intervals were chosen because AIDS was first recognized nationally in 1981 and because it was noted that the diagnosis of tuberculosis often preceded the diagnosis of AIDS by months or years.

TABLE 3. Characteristics of acquired immunodeficiency syndrome (AIDS) cases with and without tuberculosis (TB)—Florida, 1981-1985*

Characteristic	AIDS/TB (n = 109)		AIDS/non-TB (n = 985)		Statistical significance
	No.	(%)	No.	(%)	
Age					
Median	30		34		
Mean	33.6		34.6		Not significant
Race/ethnicity					
Black	88	(80.7)	363	(36.9)	p < 0.001
White	12	(11.0)	495	(50.3)	
Hispanic	9	(8.3)	122	(12.4)	
Other	0	(0.0)	5	(0.5)	
Sex					
Female	18	(16.5)	110	(11.2)	Not significant
Male	91	(83.5)	875	(88.8)	
Country of origin					
U.S.	44	(40.4)	737	(74.8)	p < 0.001
Foreign	65	(59.6)	248	(25.2)	
AIDS risk factors					
Homosexual/ bisexual men	23	(21.1)	609	(61.8)	p < 0.001
IV drug abuse	20	(18.3)	128	(13.0)	
Born NIR ctry†	55	(50.5)	127	(12.9)	
Other/none	11	(10.1)	121	(12.3)	

*Because only aggregate data were available for certain characteristics, no adjustments were made in the analysis.

†No identified risk country—country in which heterosexual transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus is thought to play a major role.

TUBERCULOSIS PATIENTS WITH AND WITHOUT AIDS

Of the 7,241 persons in Florida reported to have tuberculosis in the period 1981-1985, 105 (2%)[§] also had AIDS. The number and proportion has generally continued to rise, e.g., in 1981, five (less than 1%) of 1,553; in 1984, 33 (3%) of 1,335; the number fell to 23 (2%) of 1,425 in 1985. Of the 105 TB/AIDS patients, 65 (60%) were reported to have tuberculosis while residing in Dade County; and 23 (22%), while residing in Palm Beach County. Compared with TB/non-AIDS patients, TB/AIDS patients were younger (median 30 years versus 49 years) and were more often black (79% versus 51%), male (83% versus 71%), and foreign born (60% versus 21%). TB/AIDS patients were also more likely to have extrapulmonary tuberculosis (38% versus 11%), particularly lymphatic and miliary forms, while pleural tuberculosis was extremely rare (Table 4).

Reported by CH Cole, MD, JJ Witte, MD, WJ Bigler, PhD, BJ Sayer, DJ Garrity, Florida Dept of Health and Rehabilitative Svcs; AIDS Program, Center for Infectious Diseases, Div of Tuberculosis Control, Center for Prevention Svcs, CDC.

Editorial Note: The total number of AIDS patients in the United States meeting the CDC surveillance case definition represents only a fraction of the number of persons with HTLV-III/LAV infection. It has been estimated that, in 1985, for every case of AIDS, there were 50-100 persons with HTLV-III/LAV infection (4). The number of tuberculosis patients with HTLV-III/LAV infection but without AIDS may also exceed the number who have overt AIDS. The fact that tuberculosis did not decline in the nation as a whole in 1985 and the increase in the incidence of tuberculosis in certain areas may be partly explained by the infection with HTLV-III/LAV of persons who already had tuberculous infection (2). There are an estimated 10 million persons with latent tuberculous infection in the United States and as many as 1.5 million persons with HTLV-III/LAV infection (4). The degree to which these two infected populations overlap may be a factor in the number of tuberculosis cases that develop.

The fact that 10% of AIDS patients from Florida have been diagnosed as having tuberculosis suggests an association between AIDS and tuberculosis. Most of the tuberculosis among the AIDS patients may represent reactivation of latent tuberculous infection acquired in years past rather than progression from recently acquired infection. Immunodeficiency caused by HTLV-III/LAV infection probably allows latent tuberculous infection to progress to clinical

[§]The other four of the 109 mentioned earlier in this report had been reported to have tuberculosis before 1981, when no detailed information on individual cases was available; they were therefore excluded from this analysis.

TABLE 4. Characteristics of tuberculosis (TB) cases with and without acquired immunodeficiency syndrome (AIDS)—Florida, 1981-1985

Characteristic	TB/AIDS (n = 105)		TB/non-AIDS (n = 7,136)		Statistical significance
	No.	(%)	No.	(%)	
Age					
Median	30		49		p < 0.001
Mean	33.2		48.7		
Race					
Black	83	(79.0)	3,613	(50.5)	p < 0.001
White	22	(21.0)	3,380	(47.4)	
Other	0	(0.0)	143	(2.0)	
Ethnicity					
Hispanic	11	(10.5)	685	(9.6)	Not significant
Non-Hispanic	94	(89.5)	6,451	(90.4)	
Sex					
Female	18	(17.1)	2,084	(29.2)	0.001 < p < 0.01
Male	87	(82.9)	5,052	(70.8)	
Country of origin					
U.S.	42	(40.0)	5,610	(78.6)	p < 0.001
Foreign	63	(60.0)	1,526	(21.4)	
Form of TB					
Pulmonary	65	(61.9)	6,331	(88.7)	p < 0.001
Pleural	1	(0.1)	216	(3.0)	
Lymphatic	20	(19.0)	167	(2.3)	
Miliary	10	(9.5)	96	(1.3)	
Other	9	(8.6)	326	(4.6)	

tuberculosis. However, radiographically, the presentation of tuberculosis in AIDS patients is often indistinguishable from primary forms of the disease as seen in patients without AIDS (5). Thus, recently acquired tuberculous infection in this population cannot be ruled out.

The risk that persons with latent tuberculous infection who acquire AIDS (or HTLV-III/LAV infection without AIDS) will develop clinically active tuberculosis cannot be quantified from currently available data. However, the 10% incidence of clinically overt tuberculosis is substantially higher than would be expected for any other group, including tuberculin-positive contacts of tuberculosis cases (6).

The reason for the decreased number of TB/AIDS patients reported from Florida in 1985 is unknown. It may represent reporting artifact or a decline in the number of susceptible individuals at risk.

Other health departments may wish to determine the degree to which tuberculosis morbidity is associated with AIDS and the prevalence of HTLV-III/LAV infection in tuberculosis patients. As recommended in recently published guidelines, as part of the evaluation of patients with tuberculosis, risk factors for HTLV-III/LAV should be identified (7). Voluntary testing of all persons with these risk factors is also recommended. In addition, testing for HTLV-III/LAV antibody should be considered for patients of all ages who have severe or unusual manifestations of tuberculosis. Such additional studies would help to determine the magnitude of the AIDS/TB problem in other areas and further define the population characteristics of persons with both tuberculosis and HTLV-III/LAV infection (with and without AIDS).

Treatment of tuberculosis patients who also have AIDS or HTLV-III/LAV infection should be instituted in accordance with recently published guidelines (7). Prevention of tuberculosis among persons with HTLV-III/LAV infection will require the identification of both HTLV-III/LAV and tuberculous infection and the administration of isoniazid preventive therapy as currently recommended (7). Counseling of persons being tested for HTLV-III/LAV infection should be provided in accordance with current recommendations to prevent the transmission of HTLV-III/LAV (8).

References

1. CDC. Tuberculosis—United States, first 39 weeks, 1985. MMWR 1985;34:625-7.
2. CDC. Tuberculosis—United States, 1985—and the possible impact of human T-lymphotropic virus type III/lymphadenopathy-associated virus infection. MMWR 1986;35:74-6.
3. CDC. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. MMWR 1985;34:373-5.
4. Curran JW, Morgan WM, Hardy AM, Jaffe HW, Darrow WW, Dowdle WR. The epidemiology of AIDS: current status and future prospects. Science 1985;229:1352-7.
5. Pitchenik AE, Robinson HA. The radiographic appearance of tuberculosis in patients with the acquired immune deficiency syndrome (AIDS) and pre-AIDS. Am Rev Respir Dis 1985;131:393-6.
6. Comstock GW. Frost revisited: the modern epidemiology of tuberculosis. Am J Epidemiol 1975; 101:363-82.
7. CDC. Diagnosis and management of mycobacterial infection and disease in persons with human T-lymphotropic virus type III/lymphadenopathy-associated virus infection. MMWR 1986;35:448-52.
8. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.

Immunization of Children Infected with Human T-Lymphotropic Virus Type III/ Lymphadenopathy-Associated Virus

INTRODUCTION

This document is intended to summarize available information and to assist health-care providers in developing policies for the immunization of children infected with human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV),* the virus that causes acquired immunodeficiency syndrome (AIDS). These policies may vary depending upon the prevalence of HTLV-III/LAV infection and the incidence of vaccine-preventable diseases in the community, individual assessment of a child's health status, and the risks and benefits of immunization in a particular situation. This discussion considers the risks and benefits of immunization for children residing in the United States based on the risks of vaccine-preventable diseases and the prevalence of HTLV-III/LAV infection and is intended for use by health-care providers in the United States. The recommendations may not pertain to other countries with different risks of vaccine-preventable diseases and prevalence of HTLV-III/LAV infection among children. Since these recommendations are based upon information and knowledge available at this time, periodic reassessment and revision will be required as more data concerning risk and benefits associated with immunization of HTLV-III/LAV-infected children become known and as the prevalences of specific vaccine-preventable diseases and HTLV-III infection change.

HTLV-III/LAV INFECTION AMONG CHILDREN

In the period June 1, 1981-September 2, 1986, physicians and health departments in the United States reported 24,430 cases of AIDS to CDC (1). Three hundred forty-five (1%) of the case-patients were children under 13 years of age who met the AIDS case definition; 75% of these pediatric cases were reported from New York, Florida, New Jersey, and California. Children with less severe manifestations of HTLV-III/LAV infection (AIDS-related complex, or ARC) or with asymptomatic infections are not now reported to CDC, and no seroprevalence studies have been conducted among children. Thus, the number of less severely affected children and the number of infected but presently asymptomatic children are uncertain. In one recently published case series, 14 (48%) of 29 symptomatic HTLV-III/LAV-infected children met the CDC criteria for AIDS (2).

Fifty percent of children reported to CDC were diagnosed as having AIDS during the first year of life; 82%, by 3 years of age (1). Sixty-five percent of pediatric AIDS cases reported to CDC were fatal (3). Short-term fatality rates are lower for children with less severe disease (ARC) who have not developed opportunistic infections; however, the ultimate prognosis of these children and of asymptomatic infected children is unknown.

MECHANISMS OF TRANSMISSION OF HTLV-III/LAV AMONG CHILDREN

Two risk factors are predominately associated with HTLV-III/LAV infection in children: a) being born to a mother who has HTLV-III/LAV infection, and b) receiving blood or clotting factors containing HTLV-III/LAV. Most case-patients (79%) are children whose mothers probably are infected with the virus. The major risk factors for infection of these women are intravenous (IV) drug abuse and sexual contact with men at risk of HTLV-III/LAV infection (primarily through drug abuse or bisexual contacts); women of Haitian or central African origin are also at a higher risk of acquiring HTLV-III/LAV infection, and a small percentage of infected women have a history of being transfused with blood (4). Approximately 15% of pediatric AIDS case-patients have received transfusions of blood or blood products, and 4% have hemophilia and have been treated with clotting-factor concentrates. Information about risk factors is incomplete for 3% of children with AIDS.

Currently available data indicate that most pediatric HTLV-III/LAV infections are acquired from infected women during pregnancy, during labor and delivery, or perhaps shortly after birth. The risk of perinatal transmission from an infected mother to her infant is not known, although prospective studies indicate the rate of transmission has ranged from 0% (0/3) to 65% (13/20) (5-7). Seropositive women who had previously delivered an infected child had the highest of these transmission rates (65%) in subsequent pregnancies (5). In a retrospective

*The AIDS virus has been variously termed human T-lymphotropic virus type III (HTLV-III/LAV), lymphadenopathy-associated virus (LAV), AIDS-associated retrovirus (ARV), or human immunodeficiency virus (HIV). The designation "human immunodeficiency virus" (HIV) has been accepted by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (Science 1986;232:697).

study evaluating nine children whose mothers were later diagnosed as having AIDS, two (22%) children had antibody to HTLV-III/LAV (8). Additional prospective studies are needed to define more precisely the rate of perinatal transmission of HTLV-III/LAV.

PREVALENCE OF HTLV-III/LAV INFECTION AMONG WOMEN OF CHILD-BEARING AGE

The prevalence of HTLV-III/LAV infection among women of child-bearing age varies depending on the patient group and geographic area (4). Reported confirmed seroprevalences are less than 0.01% among female blood donors in Atlanta and 0.06% among female U.S. military recruit applicants (4,9). In contrast, the reported prevalence of HTLV-III/LAV antibody among IV drug abusers has ranged from 2% to 59%, with the highest prevalence in New York City and northern New Jersey. Female sex partners of IV drug-abusing men with AIDS or with ARC had a reported seroprevalence of 40%-71%, whereas 10% of female partners of asymptomatic infected hemophiliacs were reported to be seropositive (4). Seroprevalence among prostitutes has varied greatly (5%-40%) depending on the geographic area and has been largely attributed to a coincidental history of IV drug abuse (4). Seroprevalence has been reported to be as high as 5% among persons born in countries in which heterosexual transmission of HTLV-III/LAV is thought to play a major role (e.g., Haiti, central African countries) (1,10,11).

IMMUNOLOGIC ABNORMALITIES ASSOCIATED WITH HTLV-III/LAV INFECTION

Children with symptomatic HTLV-III/LAV infection (AIDS or ARC) have immunologic abnormalities similar to those of adult AIDS patients, including hypergammaglobulinemia, decreased T4 lymphocytes, reversed helper/suppressor T-cell ratios, poor T-lymphocyte responses to mitogen stimulation, and altered humoral immunity. Lymphopenia (cell counts less than 1,500 cells/mm³) is uncommon. Antibody responses of children with AIDS or ARC to diphtheria and tetanus toxoid boosters and to pneumococcal vaccine were absent or lower than those of age-matched controls, which is consistent with defective humoral immunity (12,13). Some HTLV-III/LAV-infected children responded adequately to immunization; 60% of AIDS and ARC patients given measles-mumps-rubella vaccine (MMR) prior to diagnosis had protective levels of measles antibodies 5-66 months after immunization (14).

Asymptomatic HTLV-III/LAV-infected adults as a group generally have less severe abnormalities of immunologic function than adults with AIDS or ARC, and some may have normal immunologic function, although individual asymptomatic adults may have severe abnormalities (15). Immunologic function of asymptomatic HTLV-III/LAV-infected children has not yet been adequately studied but presumably would be more intact than that of symptomatic HTLV-III/LAV-infected children. In a small prospective study, all 29 children with symptomatic HTLV-III/LAV infection had immunologic abnormalities within 5-13 months of being found infected, compared with only two of seven (29%) children reported to have asymptomatic HTLV-III/LAV infection (2).

CONCERNS ABOUT IMMUNIZATION OF HTLV-III/LAV-INFECTED CHILDREN

The immunologic abnormalities associated with symptomatic HTLV-III/LAV infection have raised concerns about the immunization of infected children. Replication of live, attenuated vaccine viruses may be enhanced in persons with immunodeficiency diseases and theoretically may produce serious adverse events following immunization of symptomatic HTLV-III/LAV-infected (AIDS and ARC) patients (16). Concerns have been expressed on theoretical grounds that antigenic stimulation by immunization with inactivated vaccines might lead to a deterioration of clinical status of HTLV-III/LAV-infected children, but this effect has not been documented (17). Since symptomatic HTLV-III/LAV-infected patients have abnormal primary and secondary antibody responses, the efficacy of immunization may be decreased (18). The efficacy of immunization for asymptomatic HTLV-III/LAV-infected children is unknown, but presumably would be higher than for symptomatic HTLV-III/LAV-infected children.

Because most HTLV-III/LAV-infected children become infected perinatally, it is to be expected that their mothers are infected with HTLV-III/LAV. Other family members may also be infected with HTLV-III/LAV and may have abnormal immunologic function.[†] Prospective evaluation of 16 asymptomatic HTLV-III/LAV-infected mothers of children diagnosed as having AIDS or ARC showed that 12 (75%) mothers developed AIDS or ARC during a 30-month follow-up period (6). Regardless of the immune status of the recipient, poliovaccine virus is often excreted by children vaccinated with oral poliovaccine (OPV) and may be transmitted to close contacts (19). Immune-deficient individuals (either recipients or contacts) have a higher risk of developing vaccine-associated poliomyelitis than normal individuals. There is no risk of transmitting the viruses contained in measles, mumps, rubella (MMR) vaccine to family members (20-22).

[†]Such family members may have been infected by sexual contact with an HTLV-III/LAV-infected person, by parenteral exposure to infected blood (e.g., by sharing needles), or as hemophiliacs who received clotting factors, or by perinatal transmission.

While the risks of vaccination are not known with certainty, potential risks may exist if HTLV-III/LAV-infected children are not vaccinated. If local outbreaks of measles occur in geographic areas in which there is both a cluster of unvaccinated children and a high prevalence of HTLV-III/LAV infection, the risk of measles for unvaccinated, HTLV-III/LAV-infected children may be high. Measles infection among patients with immune deficiency may be severe, protracted, and fatal (23).

EXPERIENCES WITH IMMUNIZATION OF HTLV-III/LAV-INFECTED PERSONS

Some children infected perinatally with HTLV-III/LAV have received routine immunization with OPV and MMR before their illnesses were recognized. Out-patient medical records from New York City and Miami for 213 children with symptomatic HTLV-III/LAV infection (AIDS and ARC), presumably acquired during the perinatal period, were reviewed to determine immunization history and possible vaccine-associated adverse events (24,25). One hundred seventy-one children (80%) had received at least one dose of OPV and diphtheria and tetanus toxoids and pertussis vaccine (DTP), 95 (45%) had completed primary immunization with OPV and DTP (three doses and four doses, respectively), and 63 (30%) had received MMR or measles vaccine. Thirty-eight (39%) of 98 children who had available records of dates of immunization and onset of symptoms consistent with HTLV-III/LAV infection had received at least one live-virus vaccine after symptom onset. No serious or unusual adverse events were noted in the medical records of these children following immunization.

Only one adverse event following immunization of an HTLV-III/LAV-infected person has been documented. A 19-year-old asymptomatic army recruit received multiple immunizations during basic training, including primary immunization with smallpox vaccine (26). Two and one-half weeks later, he developed cryptococcal meningitis and was diagnosed as having AIDS. One and one-half weeks later, while being treated for meningitis, he developed lesions of disseminated vaccinia. He was treated with vaccinia immune globulin and recovered from vaccinia, but has since died of AIDS.

CDC has not received any reports of vaccine-associated poliomyelitis among HTLV-III/LAV-infected vaccine recipients or their contacts or among other persons known to be infected with HTLV-III/LAV. There have been no reports of serious adverse events following MMR administration from areas in which pediatric AIDS cases are occurring.

IMMUNIZING CHILDREN WHO MAY BE INFECTED WITH HTLV-III/LAV: SPECIAL CONSIDERATIONS

Children born to women who are at risk of HTLV-III/LAV infection or who are known to be infected with HTLV-III/LAV should be evaluated for infection with the virus—including being tested for antibody (4,27). For asymptomatic children presenting for immunization, this evaluation and testing is not necessary to make decisions about immunizations. Children infected with HTLV-III/LAV are best cared for by pediatricians knowledgeable in the management of patients with this infection. Since little information is currently available on the safety and efficacy of immunizing children who may be infected with HTLV-III/LAV, special studies of these children need to be conducted.

RECOMMENDATIONS

Children with symptomatic HTLV-III/LAV infection

- A. Live-virus and live-bacterial vaccines (e.g., MMR, OPV, BCG) should not be given to children and young adults who are immunosuppressed in association with AIDS or other clinical manifestations of HTLV-III/LAV infection. For routine immunizations, these persons should receive inactivated poliovaccine (IPV) and should be excused for medical reasons from regulations requiring measles, rubella, and/or mumps immunization.
- B. Concerns have been raised that stimulation of the immune system by immunization with inactivated vaccines in these individuals might cause deterioration in immunologic function. However, such effects have not been noted thus far among children with AIDS or among other immunosuppressed individuals after immunization with inactivated vaccines. The potential benefits of immunization of these children outweigh the concerns of theoretical adverse events. Immunization with DTP, IPV, and *Haemophilus influenzae* type b vaccines is recommended in accordance with the ACIP recommendations, although immunization may be less effective than it would be for immunocompetent children (28-30).
- C. As with other conditions that produce chronic immunosuppression, the Committee recommends annual immunization with inactivated influenza vaccine for children over 6 months of age and one-time administration of pneumococcal vaccine for children over 2 years of age (31-33).

- D. Children and young adults with AIDS or other clinical manifestations of HTLV-III/LAV infection—as other immunosuppressed patients—may be at increased risk of having serious complications of infectious diseases, such as measles and varicella. Following significant exposure to measles or varicella, these persons should receive passive immunization with immune globulin (IG) or varicella-zoster immune globulin (VZIG), respectively (20,34).[¶]

Children with previously diagnosed asymptomatic HTLV-III/LAV infection

- A. A small number of children and young adults known to be infected with HTLV-III/LAV but without overt clinical manifestations of immunosuppression have received live-virus vaccines without adverse consequences. Further experience needs to be monitored, but on the basis of data now available, the Committee believes that such persons should be vaccinated with MMR in accordance with ACIP recommendations (20-22). Vaccinees should be followed for possible adverse reactions and for the occurrence of vaccine-preventable diseases since immunization may be less effective than for uninfected persons.
- B. Available data suggest that OPV can be administered without adverse consequences to HTLV-III/LAV-infected children who do not have overt clinical manifestations of immunosuppression. However, because family members of such children may be immunocompromised due to AIDS or HTLV-III/LAV infection and therefore at increased risk of paralysis from contact with spread vaccine virus, it may be prudent to use IPV routinely to immunize asymptomatic children with previously diagnosed HTLV-III/LAV infection (28).
- C. Immunization with DTP and *Haemophilus influenzae* type b vaccines is recommended in accordance with ACIP recommendations (29,30).

Children not known to be infected with HTLV-III/LAV

Children and young adults not known to be infected with HTLV-III/LAV should be immunized in accordance with ACIP recommendations.

Children residing in the household of a patient with AIDS

Children whose household members are known to be immunocompromised due to AIDS or other HTLV-III/LAV infections should not receive OPV because vaccine viruses are excreted by the recipient of the vaccine and may be communicable to their immunosuppressed contacts. These children should receive IPV for routine immunization (28). Because extensive experience has shown that live, attenuated MMR vaccine viruses are not transmitted from vaccinated individuals to others, MMR may be given to a child residing in the household of a patient with AIDS (20-22).

References

1. CDC. Unpublished data.
2. Pahwa S, Kaplan M, Fikrig S, et al. Spectrum of human T-cell lymphotropic virus type III infection in children. JAMA 1986;255:2299-2305.
3. Rogers MF. AIDS in children: a review of the clinical, epidemiologic and public health aspects. Pediatr Infect Dis 1985;4:230-6.
4. CDC. Recommendations for assisting in the prevention of perinatal transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus and acquired immunodeficiency syndrome. MMWR 1985;34:721-32.
5. Scott GB, Fischl MA, Klimas N, et al. Mothers of infants with the acquired immunodeficiency syndrome: outcome of subsequent pregnancies. Atlanta, Georgia: International conference on acquired immunodeficiency syndrome, April 14-17, 1985.
6. Scott GB, Fischl MA, Klimas N, et al. Mothers of infants with the acquired immunodeficiency syndrome. Evidence for both symptomatic and asymptomatic carriers. JAMA 1985;253:363-6.
7. Stewart GJ, Tyler JP, Cunningham AL, et al. Transmission of human T-cell lymphotropic virus type III (HTLV-III) by artificial insemination by donor. Lancet 1985;2:581-5.
8. Thomas PA, Lubin K, Enlow RW, et al. Comparison of HTLV-III serology, T-cell levels, and general health status of children whose mothers have AIDS with children of healthy inner city mothers in New York. Atlanta, Georgia: International conference on acquired immunodeficiency syndrome, April 14-17, 1985.
9. CDC. Human T-lymphotropic virus type III/lymphadenopathy-associated virus antibody prevalence in U.S. military recruit applicants. MMWR 1986;35:421-4.
10. World Health Organization. Acquired immune deficiency syndrome (AIDS). Report on the situation in Europe as of 31 December 1984. Wkly Epidem Rec 1985;60:85-90.
11. Castro KG, Fischl MA, Landesman SH, et al. Risk factors for AIDS among Haitians in the United States. Atlanta, Georgia: International conference on acquired immunodeficiency syndrome. April 14-17, 1985.
12. Bernstein LJ, Ochs HD, Wedgwood RJ, Rubenstein A. Defective humoral immunity in pediatric acquired immune deficiency syndrome. J Pediatr 1985;107:352-7.
13. Borkowsky W, Krasinski K. Residual cell-mediated immunity to recall antigens in pediatric AIDS-related disease. Pediatr Res 1986;20:292A.

[¶]Some physicians administer full replacement doses of intravenous IG on a 2-4 week schedule to children with AIDS and other clinical manifestations of HTLV-III/LAV infection. This therapy may provide some protection against such diseases as measles and varicella.

14. Krasinski K, Borkowsky W, Krugman S. Antibody following measles immunization in children infected with human T-cell lymphotropic virus-type III/lymphadenopathy associated virus (HTLV-III/LAV). Paris, France: International conference on acquired immunodeficiency syndrome, June 23-25, 1986.
15. Francis DP, Jaffe HW, Fultz PN, Getchell JP, McDougal JS, Feorino PM. The natural history of infection with the lymphadenopathy-associated human T-lymphotropic virus type III. *Ann Intern Med* 1986;103:719-22.
16. ACIP. General recommendations on immunization. *MMWR* 1983;32:1-8, 13-17.
17. Zagury D, Bernard J, Leonard R, et al. Long-term cultures of HTLV-III-infected T cells: a model of cytopathology of T-cell depletion in AIDS. *Science* 1986;231:850-3.
18. Simberloff MS, El Sadr W, Schiffman G, Rahal JJ, Jr. *Streptococcus pneumoniae* infections and bacteremia in patients with acquired immune deficiency syndrome, with report of a pneumococcal vaccine failure. *Am Rev Respir Dis* 1984;130:1174-6.
19. CDC. Paralytic poliomyelitis—United States, 1982 and 1983. *MMWR* 1984;33:635-8.
20. ACIP. Measles prevention. *MMWR* 1982;31:217-24, 229-31.
21. ACIP. Rubella prevention. *MMWR* 1984;33:301-10, 315-8.
22. ACIP. Mumps vaccine. *MMWR* 1982;31:617-20, 625.
23. Cherry JD. Measles. In Feigin, RD and Cherry, JD (eds): *Textbook of Pediatric Infectious Diseases*. Philadelphia, W.B. Saunders Company, 1981:1210-31.
24. McLaughlin M, Thomas P, Rubenstein A, et al. Use of live virus vaccines in children with HTLV-III/LAV infection: a retrospective survey. Paris, France: International conference on acquired immunodeficiency syndrome, June 23-25, 1986.
25. Gwendolyn Scott. Personal communication.
26. R. Redfield. Personal communication.
27. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR* 1986;35:152-5.
28. ACIP. Poliomyelitis prevention. *MMWR* 1982;31:22-6, 31-4.
29. ACIP. Diphtheria, tetanus, and pertussis: guidelines for vaccine prophylaxis and other preventive measures. *MMWR* 1985;34:405-14, 419-20.
30. ACIP. Polysaccharide vaccine for prevention of *Haemophilus influenzae* type b disease. *MMWR* 1985;34:201-5.
31. ACIP. Update: pneumococcal polysaccharide vaccine usage - United States. *MMWR* 1984;33:273-6, 281.
32. ACIP. Prevention and control of influenza. *MMWR* 1986;35:317-26, 331.
33. ACIP. Monovalent influenza A(H1N1) vaccine, 1986-1987. *MMWR* 1986;35:517-21.
34. ACIP. Varicella-zoster immune globulin for the prevention of chickenpox. *MMWR* 1984;33:84-90, 95-100.

Acquired Immunodeficiency Syndrome (AIDS) in Western Palm Beach County, Florida

From July 1982 through September 15, 1986, 79 persons meeting the surveillance case definition for acquired immunodeficiency syndrome (AIDS) were reported from western Palm Beach County, Florida. These patients were residents of the towns of Belle Glade (62 case-patients), Pahokee (seven case-patients), and South Bay (10 case-patients) at the time of onset of their illnesses. The number of cases is shown by year of diagnosis in Figure 1. Based upon 1980 census data, the calculated cumulative incidence for AIDS in these three towns is 295/100,000 population. In comparison, the overall cumulative incidence for AIDS in the United States is 10.8/100,000.

Selected characteristics of these 79 AIDS patients are listed in Table 1. Sixty-four patients were male; all but three of the patients were at least 13 years of age. The three pediatric patients were born to mothers infected with human lymphotropic virus type-III/lymphadenopathy-associated virus (HTLV-III/LAV), the virus that causes AIDS.* Of the 76 adult patients, 63 (82.8%) were members of population groups known to be at increased risk for HTLV-III/LAV

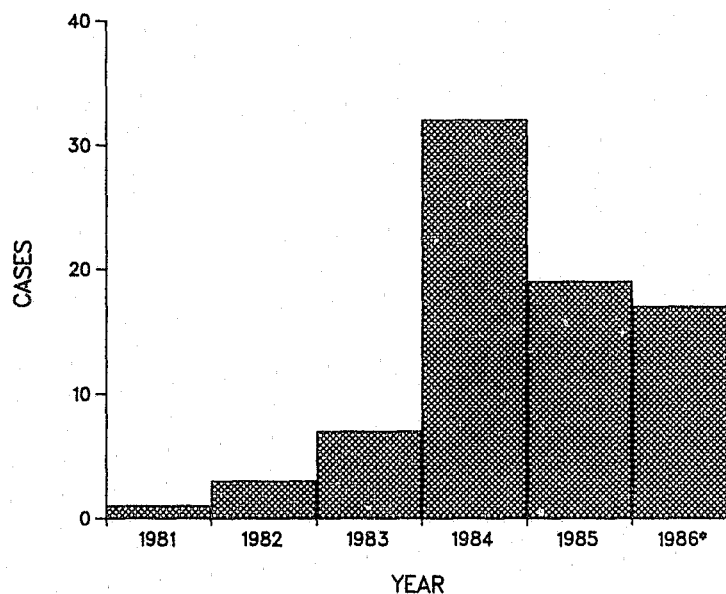
*The designation "human immunodeficiency virus" (HIV) has been accepted by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (Science 1986;232:697).

Table 1. AIDS cases in western Palm Beach County, Florida, by patient characteristics, city of residence, and sex, September 15, 1986

Characteristics	Belle Glade		Pahokee/South Bay		Total (%)
	Male	Female	Male	Female	
Adult patients					
Homosexual/bisexual	9	0	1	0	10 (12.7)
Heterosexual IV drug abuser	10	4	8	2	24 (30.4)
Transfusion-associated	0	2	0	0	2 (2.5)
Heterosexual patient*	23	3	1	0	27 (34.2)
None of the above	7	1	4	1	13 (16.5)
Pediatric patients					
Mother with AIDS	1	2	0	0	3 (3.8)
Total	50	12	14	3	79 (100.0)

*Includes 10 persons who had heterosexual contact with a person with AIDS or at increased risk of AIDS, and 17 persons born in Haiti, where heterosexual transmission is believed to play a major role.

FIGURE 1. Acquired immunodeficiency syndrome cases, by year of diagnosis — western Palm Beach County, Florida, 1981-1986



*Cases reported through September 15, 1986.

infection or were born in Haiti, a country in which heterosexual contact plays a major role in transmission of HTLV-III/LAV (1,2). The remaining 13 (11 men, two women) adult patients had no reported risk factors for AIDS, but 10 of these 13 died before epidemiologic investigations could be completed.

Compared with other adult AIDS case-patients reported from Florida in the period, adult AIDS patients from western Palm Beach County were more likely to be reported as heterosexual intravenous (IV) drug abusers (31.6% vs. 13.1%, $p < 0.05$), as sex partners of persons at increased risk of having AIDS (35.5% vs. 18.5%, $p < 0.01$), or as persons with no reported risk factors for AIDS (17.1% vs. 4.8%, $p < 0.01$).

Detailed information is available for the 62 case-patients from Belle Glade. Most of the AIDS patients lived in an area in the central part of town, comprising a population of 7,207 persons (1980 Decennial Census, Neighborhood Statistics Program). This area of Belle Glade is characterized by high rates of IV drug abuse and sexually transmitted diseases (3). Investigations in May 1985, May 1986, and August 1986 revealed that 19 adults with AIDS in Belle Glade could be directly linked to at least one other reported AIDS case by sexual contact, by sharing of needles during IV drug abuse, or both. These linked patients account for 32.2% of the 59 adult AIDS case-patients reported from Belle Glade between February 1982 and August 1986. Five of the 10 adult women reported as having AIDS during this time were prostitutes; four of the five were also IV drug abusers.

To evaluate the prevalence of and risk factors for HTLV-III/LAV infection in Belle Glade, a community-wide study was conducted from February through September 1986 by the Florida Department of Health and Rehabilitative Services (DHRS) and CDC. The town was divided into neighborhoods as determined by the 1980 decennial census. A proportionate-sampling scheme was used to interview and test persons living in and around the neighborhoods in which most of the AIDS patients resided. Preliminary results of this study indicate that 30 (3.1%) of 959 persons tested had detectable antibodies to HTLV-III/LAV by both enzyme immunoassay and Western-blot methods. One of the 30 persons had been diagnosed as having AIDS.

Sex-, age-, and race-specific seroprevalence rates have been calculated for the first 736 persons for whom data entry has been completed. Fourteen (3.7%) of 378 males and 12 (3.4%) of 358 females had antibodies to HTLV-III/LAV. None of 121 children ages 2-10 years had antibodies to HTLV-III/LAV. Other HTLV-III/LAV-antibody prevalence rates by age group were as follow: 14 (8.9%) of 157 persons ages 18-29; seven (4.4%) of 160 persons ages 30-39; two (1.8%) of 113 persons ages 40-49; three (3.2%) of 91 persons ages 50-59; and none of 94 persons over 60 years of age. Eighty-eight percent of seropositive adults were ages 18-49 years; 90% of adult AIDS case-patients reported in the United States are in that same age group. Twenty-six (4.2%) of 616 black-not-Hispanic persons tested had antibodies to HTLV-III/LAV, including 13 (8.7%) of 150 persons born in Haiti. None of 42 Hispanic persons and none of 60 white-not-Hispanic persons were seropositive. There was no clustering of persons infected with HTLV-III/LAV within households, except for four instances of infection involving two pairs of sexual partners. Further analyses are in progress to determine specific risk factors for infection.

Arthropods have been hypothesized as a mode of HTLV-III/LAV transmission in Belle Glade (4). As a measure of exposure to different mosquito vectors and antibody prevalence, samples obtained during the serosurvey were tested by the serum dilution-plaque reduction neutralization method in the Division of Vector-Borne Viral Diseases, CDC, for antibodies to five arboviruses (Tensaw, Maguari, Keystone, Saint Louis encephalitis, and dengue-2) prevalent in South Florida or the Caribbean (Table 2). There was no significant difference in prevalence of antibodies to these arboviruses between HTLV-III/LAV-infected and -noninfected per-

TABLE 2. Results of testing* for antibody to five arboviruses, by HTLV-III/LAV antibody status, community survey, Belle Glade, Florida, 1986

HTLV-III/LAV antibody status	Number of persons positive for antibody to arbovirus				
	Tensaw	Maguari	Keystone	St. Louis encephalitis	Dengue-2†
Positive (n=27)	1 (3.7%)	3 (11.1%)	1 (3.7%)	3 (11.1%)	8 (29.6%)
Negative (n=603)	81 (13.4%)	106 (17.6%)	79 (13.1%)	79 (13.1%)	91 (15.1%)

*Serum dilution-plaque reduction neutralization technique.

†This difference is not statistically significant by the Cochran-Mantel-Haenszel test for association between HTLV-III/LAV and dengue-2, after controlling for previous residence in Haiti, where dengue viruses are endemic.

sons. The lack of association between detection of antibodies to HTLV-III/LAV and antibodies to these arboviruses extends the findings of an earlier pilot study that included these and four other arboviruses (Pahayokee, Shark River, Gumbo Limbo, and Mahogany Hammock) indigenous to South Florida (5).

Reported by M Roberts, RN, Glades General Hospital, R Young, MD, Glades Health Center, J Howell, MD, MPH, Palm Beach County Public Health Unit, M Wilder, MD, Acting State Epidemiologist, Florida Dept of Health and Rehabilitative Svcs; Division of Vector-Borne Viral Diseases, AIDS Program Office, Center for Infectious Diseases, CDC.

Editorial Note: The high rate of AIDS in western Palm Beach County has focused national attention on this area. The cumulative AIDS incidence in this area (295/100,000 population) is comparable to that of the City of San Francisco (316/100,000) and the borough of Manhattan (270/100,000)—areas with the highest incidence of AIDS in the United States. In western Palm Beach County, the high cumulative rate is largely the result of high rates of AIDS among IV drug abusers and their sexual partners.

Thirteen (17.1%) of 76 adult patients in western Palm Beach County with AIDS had no reported risk factors. Although this proportion is significantly higher than in other areas in Florida, 10 of the 13 case-patients died before they could be comprehensively interviewed to obtain additional epidemiologic information on risk factors. Nationally, 72.9% of AIDS case-patients who were initially reported as persons without known risk factors, and who were available for follow-up, have been reclassified (6). AIDS cases are not categorized as resulting from heterosexual transmission unless the index partner of the AIDS patient is known a) to be infected with HTLV-III/LAV, b) to have AIDS, or c) to belong to another risk group. Therefore, if no such information is available concerning the relevant sexual partners, a case is characterized as having no risk factors.

Thus far, findings of the community-based study demonstrate a high prevalence of HTLV-III/LAV infection among younger adults of both sexes (i.e., 18-29 years of age), while no children and no adults over age 60 have had evidence of infection with HTLV-III/LAV. Additionally, serologic findings for household members of HTLV-III/LAV-infected persons did not show any evidence of viral transmission through casual contact. Infection with HTLV-III/LAV was not associated with arbovirus infection, suggesting that HTLV-III/LAV-infected persons were not more likely than persons without HTLV-III/LAV infection to have been exposed to mosquitoes. Thus, the hypothesis that arthropods have transmitted HTLV-III/LAV in Belle Glade is not supported by AIDS surveillance data, age-specific rates of HTLV-III/LAV infection, and the arbovirus serologic studies.

The available epidemiologic evidence suggests that HTLV-III/LAV infection in Belle Glade results predominantly from sexual transmission and the use of contaminated needles for injecting drugs intravenously. The U.S. Public Health Service has published guidelines to prevent sexual and drug-abuse-related transmission of HTLV-III/LAV (7). In this setting of a high cumulative rate of AIDS and a high prevalence of HTLV-III/LAV infection, programs to promote risk-reduction practices must be expanded and adopted. Additionally, voluntary serologic testing combined with health education and counseling should continue to be available to enhance reduction of HTLV-III/LAV transmission.

The ongoing analyses of the community-wide DHRS/CDC study should further clarify specific risk factors for HTLV-III/LAV infection in Belle Glade and provide a basis for additional public health recommendations for the prevention of infection with this virus.

References

1. Pape JW, Liautaud B, Thomas F, et al. The acquired immunodeficiency syndrome in Haiti. *Ann Int Med* 1985;103:674-8.
2. Castro KG, Fischl MA, Landesman SH, et al. Risk factors for AIDS among Haitians in the United States. Presented at the International Conference on AIDS, Atlanta, April 16, 1985.
3. Tavis D, Palm Beach County Health Department. Personal communication.
4. Whiteside ME, Withum D, Tavis D, McCleod C. Outbreak of no-identifiable-risk acquired immunodeficiency syndrome (AIDS) in Belle Glade, Florida. Presented at the International Conference on AIDS, Atlanta, April 17, 1985.
5. Castro K, Lieb S, Calisher C, et al. Seroepidemiologic studies of HTLV-III/LAV infection in Belle Glade, Florida. Presented at the Second International Conference on AIDS, Paris, June 25, 1986.
6. Lifson AR, Castro KG, White CR, Narkunas JP, Lekatsas A, Ksell TE, Ward B. "No identified risk" AIDS cases. In: Program and abstracts of the 26th Interscience Conference on Antimicrobial Agents and Chemotherapy. September 28-October 1, 1986. Abstract #1013, p. 283.
7. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR* 1986;35:152-5.

Acquired Immunodeficiency Syndrome (AIDS) among Blacks and Hispanics — United States

In the period June 1, 1981-September 8, 1986, physicians and health departments in the United States notified CDC of 24,576 patients meeting the AIDS case definition for national reporting (1-3). Of these, 6,192 (25%) were black and 3,488 (14%) were Hispanic, whereas these groups represent only 12% and 6%, respectively, of the U.S. population (4). The proportion of cases by racial/ethnic group has remained relatively constant over time (Figure 2), but the number of reported cases of AIDS among persons of all racial and ethnic backgrounds continues to rise (Figure 3).

Adult Patients. The race and ethnicity was known for 24,102 adult AIDS patients ≥ 15 years of age*; 14,554 (60%) of these patients were non-Hispanic whites; 5,988 (25%), blacks; 3,411 (14%), Hispanics; and 149 ($< 1\%$), members of other racial/ethnic groups. The overall cumulative incidences[†] for black and Hispanic adults were 3.1 and 3.4 times, respectively, that for whites (Table 1).

Black and Hispanic adults with AIDS were more likely than white adult AIDS patients to reside in New York, New Jersey, or Florida: 62% and 65% of the black and Hispanic patients, respectively, resided in these three states, as did 33% of white patients. Cumulative incidences in these states for blacks and Hispanics were from 2.5 to 9.0 times those for whites. Of the black and Hispanic patients from New York and New Jersey, approximately half were intravenous (IV) drug abusers. Of the black patients from Florida, 40% were born in Haiti.

Among men, blacks and Hispanics accounted for 23% and 14%, respectively, of the 22,468 male AIDS patients. However, among women, blacks and Hispanics accounted for 51% and 21%, respectively, of the 1,634 female patients. Cumulative incidences for black and Hispanic women were 13.3 and 11.1 times, respectively, the incidence for white women.

The distribution of AIDS cases by race/ethnicity differed by recognized transmission categories for AIDS (Table 2). Homosexual or bisexual men who had AIDS and patients who acquired AIDS from blood or blood products were predominately white, whereas patients with a history of IV drug abuse or heterosexual contact with persons at increased risk for acquiring AIDS, and persons with no identified mode of transmission were predominately black or Hispanic. The proportion of blacks or Hispanics with AIDS was relatively high (in terms of their proportions in the overall U.S. population) in all transmission categories with the exception of hemophilia.

The racial/ethnic distribution of homosexual/bisexual patients differed from that of heterosexual patients. Among homosexual/bisexual male AIDS patients, 16% were black; 11%, Hispanic; and 73%, white. Among heterosexual AIDS patients in all other transmission categories, 50% were black; 25%, Hispanic; and 25%, white.

Pediatric Patients. Of the 350 AIDS patients who were children (i.e., < 15 years of age) and whose race/ethnicity was known, 204 (58%) were black and 77 (22%) were Hispanic. The overall cumulative incidences for black and Hispanic children were 15.1 and 9.1 times, respectively, the incidence for white children (Table 3).

As with black and Hispanic adult AIDS patients, black and Hispanic children with AIDS were more likely than white children with AIDS to reside in New York, New Jersey, or Florida (Table 1). Of the black and Hispanic children with AIDS, 73% and 70%, respectively, lived in New York, New Jersey, or Florida. Of the 68 white children with AIDS, 40% also lived in one of those three states.

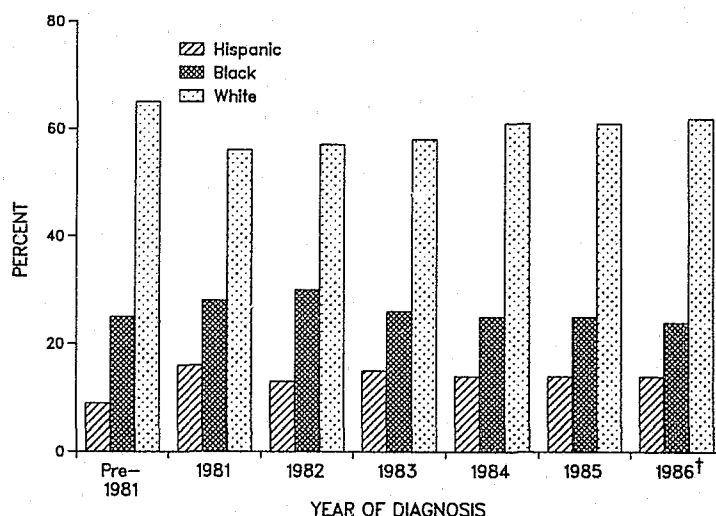
The distribution of pediatric AIDS cases by race/ethnicity varied by transmission category. Ninety percent of the children with perinatally acquired AIDS compared with 42% of the children with hemophilia- or transfusion-associated AIDS were black or Hispanic (Table 3). The observation that children with perinatally acquired AIDS (mother-to-infant transmission) were predominately black or Hispanic (Table 3) is consistent with the high proportion (75%) of heterosexual adults who are black or Hispanic. As with adults, the proportion of pediatric patients who were black or Hispanic was highest in the transmission categories associated with IV drug abuse by at least one of the parents (Table 3).

Reported by AIDS Program, Center for Infectious Diseases, CDC.

*Because U.S. census data for the Hispanic population are only available for 5-year age groups, the adult patients have been defined as those ages ≥ 15 years and pediatric as those < 15 years of age.

[†]Defined as number of cases/million population of that racial/ethnic group.

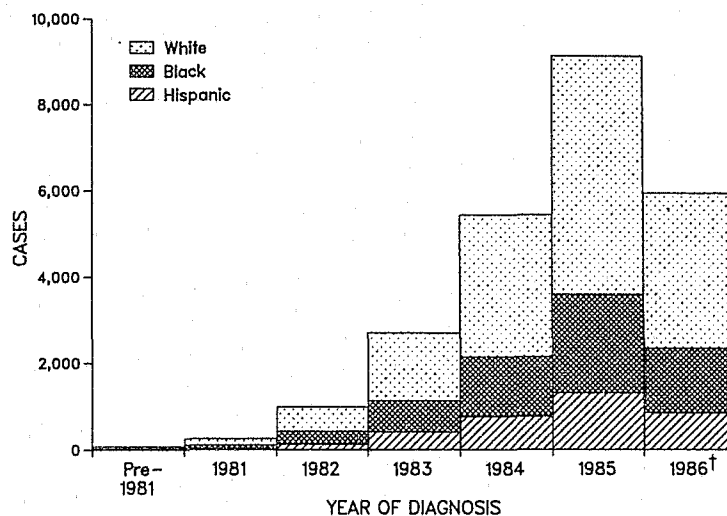
FIGURE 2. Percentage of acquired immunodeficiency syndrome (AIDS) cases,* by year of diagnosis and race — United States, pre-1981-1986



*Reported as of September 8, 1986, and excludes 153 AIDS patients (<) of other race.

†Incomplete year.

FIGURE 3. Acquired immunodeficiency syndrome (AIDS) cases,* by year of diagnosis and race — United States, pre-1981-1986



*Reported as of September 8, 1986, and excludes 153 AIDS patients (< 1%) of other race.

†Incomplete year.

Editorial Note: The incidence of AIDS is rising for all racial/ethnic groups, and in all geographic regions of the country. However, cumulative incidences of AIDS among blacks and Hispanics are over 3 times the rate for whites. Seroprevalence studies of military recruit applicants and of potential blood donors also indicate a higher prevalence of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus[¶] (HTLV-III/LAV)

[¶]The AIDS virus has been variously termed human T-lymphotropic virus type III (HTLV-III), lymphadenopathy-associated virus (LAV), AIDS-associated retrovirus (ARV), or human immunodeficiency virus (HIV). The designation "human immunodeficiency virus" (HIV) has recently been accepted by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (5).

among blacks than whites (separate values for Hispanics are not available since ethnicity was not recorded). Antibody seroprevalence rates were over 4 times higher for black military recruit applicants and over 7 times higher for black potential blood donors in one city than for whites (6,7). However, the population of individuals volunteering for military service or blood donation may not be representative of the U.S. population at large.

Several factors may contribute to the elevated incidence of AIDS and HTLV-III/LAV infection among these racial/ethnic groups. The racial/ethnic distribution of AIDS cases may reflect, to some degree, the racial/ethnic distribution of the populations at risk in the high-prevalence areas. Persons at risk become so as a result of underlying risk factors, not because of their race/ethnicity. Reported AIDS patients who are IV drug abusers are predominately black (51%) or Hispanic (30%). Children with AIDS whose parents abuse IV drugs are also predominately black (51%) or Hispanic (31%). Population-based estimates of the racial/ethnic distribution of IV drug abusers in the United States are unknown. However, in September 1982, the National Institute on Drug Abuse (NIDA) surveyed all known drug abuse treatment facilities in the United States to determine the racial/ethnic composition of the client populations using those facilities (8). The racial/ethnic distribution of clients in the surveyed clinics was 32% white, 40% black, 28% Hispanic, and < 1% "other race" for clients in the New York City standard metropolitan statistical area (SMSA), and 41% white, 50% black, and 9% Hispanic in the Newark, New Jersey, SMSA. This survey indicates that in these SMSA's, which have reported two-thirds of the IV drug abusers with AIDS, a disproportionate number of IV drug abusers attending these clinics were black or Hispanic.

Economic and cultural factors may also be associated with the observed differences in incidence for racial/ethnic groups. For example, education and economics may play a role in the observed difference in needle-sharing practices and, therefore, in the HTLV-III/LAV infection rates among white, black, and Hispanic IV drug abusers. In a study of HTLV-III/LAV infection among IV drug abusers in New York City, the prevalence of antibody to HTLV-III/LAV was higher for black (42%) and Hispanic (42%) patients than for white patients (14%) who were drug abusers (9). Preliminary analysis of data from the same study indicates that a higher proportion of white patients (18%) than black or Hispanic patients (8%) reported using new needles at least half the time when they injected drugs. Black and Hispanic participants in the study reported having substantially fewer years of education and were more likely than white patients to receive public assistance. Further analysis of data from this study and further study of HTLV-III/LAV infection involving other IV-drug-abusing populations are needed to fully understand the reasons black and Hispanic drug abusers have higher rates of AIDS and HTLV-III/LAV infection.

Table 1. Reported cases and cumulative incidence* of AIDS, by state of residence and demographic group, as of September 8, 1986

Location	Demographic group (age and race/ethnicity)										Total
	Adults (≥ 15 yrs.)				Adult total	Children (< 15 yrs.)				Children total	
	White†	Black†	Hispanic	Other†		White†	Black†	Hispanic	Other†		
California number (cum. inc.)	4,402 (340.1)	525 (399.8)	531 (173.2)	65 (54.5)	5,523 (298.3)	8 (2.8)	7 (14.9)	7 (4.7)	0 (0.0)	22 (4.3)	5,545 (234.3)
Florida number (cum. inc.)	761 (122.7)	599 (655.4)	208 (305.4)	2 (27.8)	1,570 (199.5)	5 (3.9)	38 (94.1)	2 (11.3)	0 (0.0)	45 (24.0)	1,615 (165.7)
New Jersey number (cum. inc.)	559 (118.9)	695 (1,074.2)	187 (551.6)	4 (41.2)	1,445 (249.7)	12 (10.7)	31 (118.8)	8 (52.3)	0 (0.0)	51 (32.3)	1,496 (203.2)
New York number (cum. inc.)	3,531 (331.4)	2,427 (1,439.5)	1,836 (1,573.3)	35 (108.7)	7,829 (566.0)	10 (4.0)	80 (130.5)	44 (89.4)	0 (0.0)	134 (36.0)	7,963 (453.6)
Other states number (cum. inc.)	5,301 (49.0)	1,742 (123.8)	649 (138.6)	43 (18.3)	7,735 (59.8)	33 (1.1)	48 (8.4)	16 (6.7)	1 (1.0)	98 (2.5)	7,833 (46.6)
Total number (cum. inc.)	14,554 (102.0)	5,988 (321.5)	3,411 (343.4)	149 (36.9)	24,102 (137.5)	68 (1.8)	204 (27.3)	77 (16.5)	1 (0.6)	350 (6.8)	24,452 (107.9)

*The cumulative incidence (shown in parentheses) is the cumulative number of reported AIDS cases/million population/individual demographic group (based on data from the 1980 census of the population of the United States).

†Non-Hispanic.

Education and prevention programs may be less effective in reaching minority populations unless specifically designed for those groups. Targeted programs are needed for black and Hispanic men who engage in homosexual activity, and for blacks and Hispanics of either sex who are engaging in other high-risk behavior. One report has suggested that many blacks who engage in homosexual activity are bisexual, and that these men may not benefit from educational programs designed for homosexuals (10). Programs to prevent transmission of HTLV-III/LAV infection through heterosexual contact and perinatal exposure also need to consider that approximately 75% of heterosexual patients, 73% of women with AIDS, and 92% of children with perinatally acquired infection are black or Hispanic.

Until an effective therapy or vaccine is available, prevention of HTLV-III/LAV infection depends on education and behavioral modification of persons at increased risk (11,12). The

Table 2. Percentage distribution of cases of AIDS among adults (age ≥ 15 years), by race/ethnic group, by selected transmission category, as of September 8, 1986

Transmission category*	Total number	Percentage			
		White [†]	Black [†]	Hispanic	Other [†]
For reference: U.S. population ≥ 15 years	175,254,960	81.4	10.6	5.7	2.3
Intravenous drug abusers not known to be homosexual	4,147	18.5	51.4	29.8	0.3
Intravenous drug abusers known to to be homosexual	1,881	64.1	22.1	13.6	0.3
Homosexual men not known to be IV drug abusers	15,765	74.3	14.8	10.2	0.7
Persons with hemo- philia or other clotting factor disorder	197	86.3	5.6	8.1	0.0
Women whose sex partner was a bisexual man	51	47.1	35.3	13.7	3.9
Heterosexual persons whose sex partner was an intravenous drug abuser	253	14.6	47.8	37.6	0.0
Blood transfusion recipients	424	78.3	13.7	5.9	2.1
Undetermined (persons with no identified mode of acquisition)	833	35.4	43.7	19.6	1.3
Total[§]	24,102	60.4	24.8	14.2	0.6

*Cases with more than one risk factor (possible mode of acquisition), other than the combination of male homosexuality and intravenous drug abuse, shown only in the first applicable category listed.

[†]Non-Hispanic.

[§]The total includes a) nine AIDS patients who have had heterosexual contact with a person who had AIDS or who had a risk factor for AIDS, b) 525 AIDS patients without other identified risk factors who were born in countries in which heterosexual transmission is believed to play a major role, although precise means of transmission have not yet been fully defined (virtually all of whom are black, non-Hispanic), and c) 17 AIDS patients who had heterosexual contact with a person born in one of these countries (76% of whom are black, non-Hispanic). The total excludes 122 persons of unknown race/ethnic group.

U.S. Public Health Service has assisted and encourages involvement of minority professional and community organizations in providing education about AIDS and its prevention in black and Hispanic communities. Additional health-education/risk-reduction projects are needed to actively involve minority communities in the accomplishment of overall community AIDS risk-reduction activities.

Table 3. Percentage distribution of cases of AIDS among children (age < 15 years), by race/ethnic group, by selected transmission category, as of September 8, 1986

Transmission category*	Total number	Percentage			
		White [†]	Black [†]	Hispanic	Other [†]
For reference: U.S. population < 15 years	51,290,339	73.3	14.6	9.1	3.0
Children with hemophilia or other clotting factor disorder	18	66.7	27.8	5.6	0.0
Children whose mother was an intravenous drug abuser	162	8.6	63.0	28.4	0.0
Children whose mother had a male sex partner who was bisexual	13	30.8	53.8	15.4	0.0
Children whose mother had a male sex partner who was an intravenous drug abuser	38	10.5	44.7	44.7	0.0
Children whose mother was known to be infected with HTLV-III/LAV but had no identified risk factor	11	9.1	81.8	9.1	0.0
Blood transfusion recipients	49	55.1	30.6	14.3	0.0
Undetermined (children with no identified mode of acquisition)	10	30.0	60.0	10.0	0.0
Total[§]	350	19.4	58.3	22.0	0.3

*Patients with more than one risk factor (possible mode of acquisition) are shown only in the first applicable category listed.

[†]Non-Hispanic.

[§]The total includes five children whose mothers' only identified possible mode of acquisition of HTLV-III/LAV infection was a blood transfusion, one child whose mother's male sex partner had received a transfusion, and 43 children whose mothers were born in countries in which heterosexual transmission is believed to play a major role, although precise means of transmission have not yet been fully defined (virtually all of whom are black, non-Hispanic). The total excludes two children of unknown race/ethnic group.

References

1. CDC. Update: acquired immunodeficiency syndrome (AIDS)—United States. MMWR 1984; 32:688-91.
2. Selik RM, Haverkos HW, Curran JW. Acquired immune deficiency syndrome (AIDS) trends in the United States, 1978-1982. Am J Med 1984;76:493-500.
3. CDC. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. MMWR 1985;34:373-5.
4. Vital Statistics. 1980 U.S. census.
5. Coffin J, Haase A, Levy JA, et al. Human immunodeficiency viruses [Letter]. Science 1986; 232:697.
6. CDC. Human T-lymphotropic virus type III/lymphadenopathy-associated virus antibody prevalence in U.S. military recruit applicants. MMWR 1986;35:421-4.
7. Ward JW, Grindon AJ, Feorino PM, Schable CA, Allen JR. Epidemiologic evaluation of blood donors positive on the anti-HTLV-III/LAV enzyme immunoassay (EIA). Presented at the International Conference on AIDS, Paris, France, June 1986.

8. National Institute on Drug Abuse. National drug abuse treatment utilization survey. Statistical series, series F, no. 10, September, 1982.
9. Schoenbaum EE, Selwyn PA, Klein RS, et al. Prevalence of and risk factors associated with HTLV-III/LAV antibodies among intravenous drug abusers in methadone program in New York City. Presented at the International Conference on AIDS, Paris, France, June 1986.
10. Bakeman R, Lumb JR, Jackson RE, Smith DW. AIDS risk-group profiles in whites and members of minority groups. *N Engl J Med* 1986;315:191-2.
11. CDC. Recommendations for assisting in the prevention of perinatal transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus and acquired immunodeficiency syndrome. *MMWR* 1985;34:721-32.
12. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR* 1986;35:152-5.

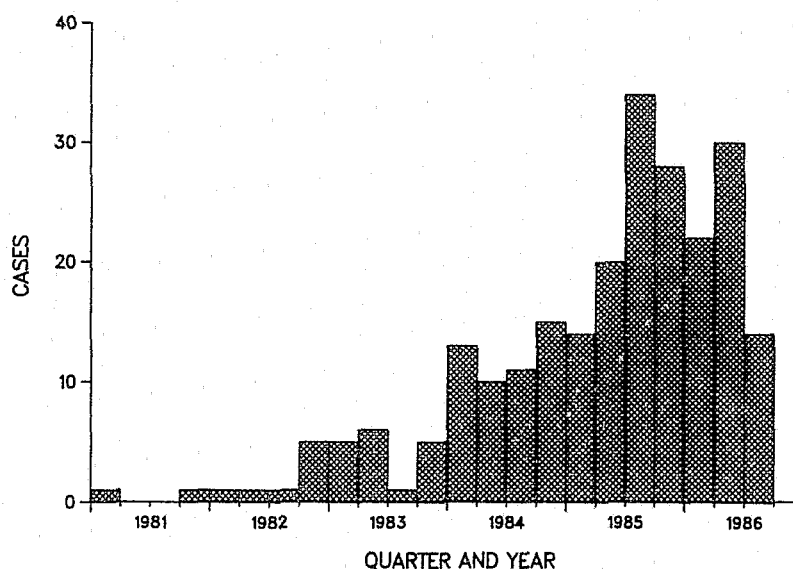
Surveillance of Hemophilia-Associated Acquired Immunodeficiency Syndrome

As of September 15, 1986, a total of 238 cases of hemophilia-associated acquired immunodeficiency syndrome (AIDS) have been reported to CDC through state health departments, hemophilia treatment centers (HTCs), and physicians. Of the 238 patients, 212 (89%) had hemophilia A (coagulation factor VIII deficiency); 16 (7%), hemophilia B (factor IX deficiency); seven (3%), von Willebrand's disease; two, an acquired inhibitor (antibody) to factor VIII; and one, a factor V deficiency. All but seven (3%) of the patients were male. Thirteen patients were known to have had other risk factors for AIDS in addition to a hematologic disease. The 238 patients resided in 38 states; almost half lived in California, New York, Pennsylvania, New Jersey, or Missouri. The total number of cases represents a cumulative incidence of 1.6 cases of AIDS/100 hemophiliacs in the United States (1).

The first AIDS patient with underlying coagulation disorders was diagnosed as having *Pneumocystis carinii* pneumonia in 1981. Later it was recognized that this patient had AIDS. Since then, the number of hemophilia-associated AIDS cases has increased each year. The reported number of cases among hemophiliacs does not appear to be increasing at an exponential rate (Figure 1); however, in 1985, 92% of persons with hemophilia A and 52% of those with hemophilia B in a U.S. hemophilia cohort had antibodies to human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV)*, suggesting exposure to the virus or to virus particles (2). HTLV-III/LAV seropositivity in this cohort was associated with declining T_{helper} lymphocyte numbers and with declining T_{helper} -to- $T_{\text{suppressor}}$ cell ratios. Because of these high rates of seroprevalence and immunology findings, concern had been expressed that the recent incidence of hemophilia-associated AIDS may be misleadingly low because of a decline in reporting.

*The AIDS virus has been variously termed human T-lymphotropic virus type III (HTLV-III/LAV), lymphadenopathy-associated virus (LAV), AIDS-associated retrovirus (ARV), or human immunodeficiency virus (HIV). The designation "human immunodeficiency virus" (HIV) has been accepted by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (Science 1986;232:697).

FIGURE 1. Cases of hemophilia-associated acquired immunodeficiency syndrome, by quarter of diagnosis — United States, January 1, 1981-September 15, 1986*



*Recently diagnosed cases may not be included because of a lag time in reporting.

To determine the completeness of reporting, the Division of Host Factors (DHF), Center for Infectious Diseases, CDC, and the National Hemophilia Foundation (NHF) surveyed all United States HTC's, local NHF chapters, and physicians known to have patients with hemophilia (3). On May 14, 1986, each HTC/physician was sent a list of persons with hemophilia-associated AIDS according to DHF records as of May 1, 1986. Since patients' names are not used at DHF, cases were identified only by the patient's date of birth, the date of diagnosis, and the nature of the AIDS diagnosis. The HTC's/physicians were asked to add to this list any other known cases—confirmed or suspected—among persons with hemophilia. DHF personnel telephoned all HTC's/physicians who had not responded by August 1, 1986.

A total of 240 HTC's/physicians and 34 NHF chapters were sent letters, and written responses were received from 61 (25%) HTC's/physicians. Information was obtained by telephone from 209 of the 213 addressees who had not responded; four NHF chapters could not be reached. In addition, DHF personnel contacted the state health departments of three states that had no reported cases and no HTC's or physicians listed in the NHF directory. From these efforts, eight previously unreported cases of AIDS among persons with hemophilia were identified. Two patients were from California (diagnosis of AIDS 12/84 and 7/85); two were from Oregon (diagnosis of AIDS 3/86 and 7/86); and one each from Colorado (diagnosis of AIDS 3/85), Missouri (5/85), New York (4/85), and Virginia (1/86). In four instances, the physicians assumed that the cases had been reported to the appropriate state health departments. In the other instances, two cases involved physicians who did not realize their legal responsibility to report cases of AIDS to the state; one case involved a postmortem diagnosis of opportunistic infection, of which the physician had been unaware; and one case involved an acquired inhibitor to factor VIII, which the physician did not realize constituted a case of hemophilia-associated AIDS.

Reported by National Hemophilia Foundation and associated Hemophilia Treatment Centers; Div of Host Factors, Center for Infectious Diseases, CDC.

Editorial Note: National surveillance for AIDS cases among persons with hemophilia is maintained through the receipt of standard AIDS case report forms submitted by the state health departments to CDC and through reports (without names) sent directly to DHF by physicians and nurses who care for patients with hemophilia. In the latter case, information is immediately shared with the state health department. The eight unreported cases identified in the CDC-NHF survey represent approximately 3% of all reported hemophilia-associated AIDS cases in the United States. This approximates the percentage of such cases that were reclassified according to the case definition for AIDS revised in 1985 (4).

In interpreting the findings of this survey, it should be noted that approximately 50%-60% of persons with hemophilia in the United States receive care through HTC's or hematologists (CDC data, unpublished). However, this selection bias probably does not significantly distort the results of the survey, because hemophiliacs at greatest risk for contracting AIDS, i.e., those who require extensive concentrated clotting-factor replacement (5), are most likely to be followed by these health care providers. The survey could not determine willingness/unwillingness to perform confirmatory diagnostic procedures such as esophagoscopy or lung biopsy in the hemophiliac population. Conversations with HTC personnel and physicians, however, suggest that confirmatory procedures are usually done. Finally, this approach to validation of the surveillance system assumes that physicians who do not initially choose to report AIDS cases (e.g., for reasons of confidentiality) would do so when contacted personally. This may not be the case. Nevertheless, the survey described here and other studies (6,7) suggest that surveillance of AIDS (as currently defined)—particularly of hemophiliacs—is relatively complete.

References

1. US Department of Health, Education, and Welfare. Study to evaluate the supply-demand relationships for AHF and PTC through 1980. Washington, DC: US Department of Health, Education, and Welfare, 1977; publication no. 77-1274.
2. Jason J, Holman RC, Kennedy MS, Evatt BL. Longitudinal assessment of hemophiliacs exposed to HTLV-III/LAV. In: Program and abstracts of the 26th Interscience Conference on Antimicrobial Agents and Chemotherapy. New Orleans, Louisiana; 1986:97.
3. National Hemophilia Foundation. Directory of hemophilia treatment facilities, 1986 edition. New York: National Hemophilia Foundation, 1986.
4. CDC. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. MMWR 1985;34:373-5.
5. Gjerset GF, McGrady G, Counts RB, Martin PJ, Jason J, Kennedy S, Evatt B, Hansen JA. Lymphadenopathy-associated virus antibodies and T cells in hemophiliacs treated with cryoprecipitate or concentrate. Blood 1985;66:718-20.
6. Chamberland ME, Allen JR, Monroe JM, et al. Acquired immunodeficiency syndrome in New York City. Evaluation of an active surveillance system. JAMA 1985;254:383-7.
7. Hardy AM, Starcher ET, Druker J, Krystal AR, Day J, Kelly C. Using death certificates to determine the level of AIDS case reporting. Second International Conference on AIDS. Paris, France, June 23-25, 1986.

Positive HTLV-III/LAV Antibody Results for Sexually Active Female Members of Social/Sexual Clubs — Minnesota

In June 1986, two sexually active women in Minnesota were found to have antibody to human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV).^{*} Both belonged to social/sexual clubs whose stated purpose was to provide their members (primarily couples) with opportunities for social and sexual contacts.[†] Each of the two seropositive women reported having sexual contact with a number of other persons from these clubs, including two men who were bisexual.

Infection was detected in these two women during a serologic screening program conducted by the St. Paul Division of Public Health, in consultation with the Minnesota Department of Health. This screening was undertaken because members of these clubs were known to have been involved in outbreaks of other sexually transmitted diseases (including syphilis and gonorrhea). From a total of 285 members (143 women and 142 men) of two of these social/sexual clubs in the Minneapolis-St. Paul area, 134 volunteers were tested with an enzyme-linked immunosorbent assay (ELISA) for antibody to HTLV-III/LAV in June and July 1986. Any ELISA-positive specimens were also tested with the Western blot assay. All 75 men tested had negative ELISA results for antibody to HTLV-III/LAV. Two of 59 women tested had positive antibody test results for HTLV-III/LAV with both ELISA and Western blot. Antibody results for these women were again positive with ELISA and Western blot when repeated 6 weeks later. The seroprevalence rate of 3% among female club members tested is significantly higher than the seroprevalence rate of zero (none of 56,000) among female blood donors in Minnesota.

The two seropositive women had belonged to two different social/sexual clubs for approximately 2 years. Both denied intravenous drug use, a history of blood transfusions, or receipt of clotting factor concentrates. One woman was 31 years old, married, and had sexual relations only with other club members; her husband (also a member) had negative test results for HTLV-III/LAV antibody. The other woman was 25 years old, unmarried, and occasionally had sexual relations with men outside the club.

Each of these two women reported having had sexual contact with more than 25 other club members, including five men with whom they had both had sexual intercourse. Two of these five men could be located for testing and had negative results for HTLV-III/LAV antibody. Two of the other three men whose serologic status could not be determined were reported to be bisexual men with whom both women had had repeated vaginal and anal intercourse.

An additional bisexual man who was a former member of one of these clubs is known to have developed acquired immunodeficiency syndrome (AIDS). He had no history of sexual contact with either of the seropositive women or with either of the two bisexual men who had sexual contact with these women.

To date, 65 of the 134 club members tested for antibody to HTLV-III/LAV (including the two seropositive women) have participated in follow-up interviews and have received counselling about their sexual practices and attitudes. Four (15%) of 27 men reported homosexual contact with other club members as well as with men who were not members of either of the two clubs. When asked whether they perceived themselves as being at increased risk of having AIDS, 40 members (73%) replied that they did not. One man reported that he "usually" used condoms while having sexual intercourse. When asked whether they would continue to participate in the activities promoted by social/sexual clubs if they knew such activities were associated with a high risk of having AIDS, 54/55 (98%) answered that they would not.

When it was known that one member of each of the two clubs was positive for HTLV-III/LAV antibody, both clubs disbanded. In an effort to minimize the transmission of HTLV-III/LAV, educational programs for sexually active adults (including former club members) are currently being implemented in the Minneapolis-St. Paul area. Follow-up studies of former club members are planned to assess whether other changes in sexual behavior are occurring.

^{*}The AIDS virus has been variously termed human T-lymphotropic virus type III (HTLV-III/LAV), lymphadenopathy-associated virus (LAV), AIDS-associated retrovirus (ARV), or human immunodeficiency virus (HIV). The designation "human immunodeficiency virus" (HIV) has been accepted by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (Science 1986;232:697).

[†]These clubs are popularly known as "swing clubs". A national organization lists more than 100 such clubs (7).

Reported by K Henry, MD, St. Paul Div of Public Health, St. Paul Ramsey Medical Center and the University of Minnesota School of Medicine; KL MacDonald, MD, MT Osterholm, PhD, MPH, State Epidemiologist, Minnesota Dept of Health; AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: The risk of having HTLV-III/LAV infection and other sexually transmitted diseases is increased for persons who have multiple sexual partners as well as for persons who have sexual encounters with high-risk individuals (2-5). However, most members of two social/sexual clubs in Minnesota who were interviewed did not consider themselves at increased risk of having AIDS and did not take precautions to protect themselves against AIDS or other sexually transmitted diseases.

Both seropositive women discussed above had a history of multiple sexual encounters—including vaginal and anal intercourse—with high-risk individuals. Although receptive anal intercourse is associated with increased risk of HTLV-III/LAV infection for homosexual men, most women infected with HTLV-III/LAV through sexual contact have denied having had anal intercourse (6-11).

To reduce the risk of HTLV-III/LAV infection, the Public Health Service recommends avoiding sexual contact with multiple partners or with persons who have been sexually active with multiple partners (2,4,5). Persons who do not follow this recommendation and who a) initiate a sexual relationship with another person who is at increased risk of having HTLV-III/LAV infection or b) maintain multiple sexual partnerships should at least avoid sexual practices that permit the exchange of blood, semen, urine, feces, saliva, or vaginal/cervical secretions. Consistent use of condoms may reduce transmission of HTLV-III/LAV (12). Other efforts to reduce HTLV-III/LAV transmission include making available voluntary serologic testing and health education and counselling for all persons believed to be at increased risk of having HTLV-III/LAV infection (4).

References

1. North American Swing Club Association. International directory swing clubs and publications. Buena Park, California: NASCA, 1986.
2. CDC. 1985 STD Treatment Guidelines. MMWR 1985;34:75S-109S.
3. CDC. Heterosexual transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1985;34:561-3.
4. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.
5. Office of the Assistant Secretary for Health. Surgeon General's report on acquired immune deficiency syndrome. Washington, DC: Public Health Service, 1986:15-6.
6. Goedert JJ, Sarngadharan MG, Biggar RJ, et al. Determinants of retrovirus (HTLV-III) antibody and immunodeficiency conditions in homosexual men. Lancet 1984;2:711-6.
7. Groopman JE, Mayer KH, Sarngadharan MG, et al. Seroepidemiology of human T-lymphotropic virus type III among homosexual men with the acquired immunodeficiency syndrome or generalized lymphadenopathy and among asymptomatic controls in Boston. Ann Intern Med 1985;102:334-7.
8. Nicholson JK, McDougal JS, Jaffe HW, et al. Exposure to human T-lymphotropic virus type III/lymphadenopathy-associated virus and immunologic abnormalities in asymptomatic homosexual men. Ann Intern Med 1985;103:37-42.
9. Kreiss JK, Kitchen LW, Prince HE, Kasper CK, Essex M. Antibody to human T-lymphotropic virus type III in wives of hemophiliacs. Evidence for heterosexual transmission. Ann Intern Med 1985;102:623-6.
10. Peterman TA, Stoneburner RL, Allen JR. Risk of HTLV-III/LAV transmission to household contacts of persons with transfusion-associated HTLV-III/LAV infection. Presented at the International Conference on the Acquired Immunodeficiency Syndrome (AIDS), Paris, France, June 1986.
11. Saltzman BR, Harris CA, Klein RS, et al. HTLV-III/LAV infection and immunodeficiency in heterosexual partners (HP) of AIDS patients. Presented at the International Conference on the Acquired Immunodeficiency Syndrome (AIDS), Paris, France, June 1986.
12. Mann JM, Quinn T, Francis H, et al. Sexual practices associated with LAV/HTLV-III seropositivity among female prostitutes in Kinshasa, Zaire. Presented at the International Conference on the Acquired Immunodeficiency Syndrome, Paris, France, June 1986.

Update: Acquired Immunodeficiency Syndrome—United States

As of December 8, 1986, physicians and health departments in the United States had reported 28,098 patients (27,704 adults and 394 children) meeting the acquired immunodeficiency syndrome (AIDS) case definition for national reporting (1-3). Of these patients, 15,757 (56% of adults and 61% of children) are known to have died, including over 79% of those patients diagnosed before January 1985. Since the initial reports of AIDS in early 1981 (4-5), the number of cases reported for each 6-month period continues to increase. However, the increases are not exponential, as evidenced by the lengthening period of time required to double the number of cases (Table 1). During the past 3 months, an average of 58 AIDS cases have been reported to CDC daily. This compares with 35 cases reported during the same period in 1985, 20 cases in 1984, and 10 cases in 1983. Cases have been reported from all 50 states, the District of Columbia, and four U.S. Territories.

Adult patients. Among adult AIDS patients, 25,834 (93%) are men. There has been no significant change over time in distribution of male patients by age and race. Ninety percent of men with AIDS are 20 to 49 years of age (mean = 36.8 years); 63% are white; 22%, black; 14%, Hispanic; and 1%, other or unknown race/ethnicity.

Pneumocystis carinii pneumonia (PCP) continues to be the most common opportunistic disease reported among AIDS patients. Sixty-four percent of men had PCP; 21% had other opportunistic diseases without PCP; and 15% had Kaposi's sarcoma (KS) alone. Ninety-five percent of patients with KS have been homosexual or bisexual men.

Women with AIDS have been reported from 41 states, the District of Columbia, and three territories. The number of cases varies greatly by reporting area and ranges from one to 877 (median = 6); seventy-two percent of female cases were reported from Florida, New Jersey, and New York (42% of male cases were reported from these three states). Eighty-eight percent of women reported with AIDS are 20 to 49 years of age (mean = 34.9 years); 27% are white; 52%, black; 20%, Hispanic; and 1%, other or unknown race/ethnicity. Sixty-seven percent of women had PCP, 31% had other opportunistic diseases without PCP, and 2% had KS alone.

Ninety-seven percent of all adult AIDS patients can be placed in groups* that suggest a possible means of disease acquisition. Homosexual or bisexual men who are not known to have used intravenous (IV) drugs represent 66% of all reported cases (70% of male cases). Heterosexual IV drug users comprise 17% of all cases (15% of male cases and 51% of female cases). Homosexual or bisexual men who have used IV drugs comprise 8% of all cases (8% of males). Persons with hemophilia/coagulation disorders represent 1% of all cases (1% of males; 0.4% of females). Heterosexual sex partners of persons with AIDS or at risk for AIDS represent 4% of all cases (2% of males and 27% of females). This latter category includes persons without other identified risks who were born in countries in which heterosexual transmission is believed to play a major role. Recipients of transfused blood or blood components account for 2% of all cases (1% of males and 10% of females). For 3% of AIDS patients (3% of

*Patient groups are hierarchically ordered; patients with multiple risk factors are tabulated only in the group listed first.

TABLE 1. Acquired immunodeficiency syndrome cases, by date of report and doubling time—United States, through December 8, 1986

Cumulative cases reported	Date*	Doubling time* (months)
110	September 1981	-
220	January 1982	5
439	June 1982	6
878	December 1982	6
1,756	July 1983	7
3,512	February 1984	8
7,025	December 1984	9
14,049	October 1985	11
28,098	December 1986	13

*Doubling time was calculated in days but is reported here to nearest month.

males and 11% of females), the possible means of disease acquisition is undetermined. Except for women with a coagulation disorder, the number of AIDS cases reported per year continues to increase in all patient groups (Table 2).

AIDS patients reported as not belonging to recognized risk groups are investigated by local health officials to determine if possible risk factors exist. Of all AIDS patients reported to CDC who were initially identified as not belonging to a risk group and who were available for follow-up, 72% have been reclassified because risk factors were identified or because the patient was found not to meet the surveillance case definition. Of the 853 AIDS patients currently listed as not belonging to recognized risk groups, information is incomplete on 206 due to: death (158), refusal to be interviewed (34), or loss to followup (14). Of the remaining 647 patients, 458 are currently under investigation. No risk was identified for 189 patients who were interviewed or for whom other followup information was obtained. However, of those patients responding to a standardized questionnaire, 40/125 (32%) gave histories of gonorrhea and/or syphilis, and 19 of the 70 men (27%) gave a history of prostitute contact, indicating that these AIDS patients were at potential risk for other sexually-transmitted infections.

The availability of laboratory tests to detect human T-cell lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV)[†] antibody made it possible to increase the sensitivity and specificity of the AIDS case definition used for national reporting (3). Of the AIDS case reports submitted to CDC, HTLV-III/LAV antibody test results were included for 6,897 (24.5%) of patients (6,558 with recognized risk factors and 339 for whom no risk has been identified). Eighty-nine (1.4%) of the tested patients with recognized risk factors, compared with 27 (8%) of those without identified risk factors were reported negative for HTLV-III/LAV antibody ($p < 0.001$).

[†]The AIDS virus has been variously termed human T-lymphotropic virus type III (HTLV-III/LAV), lymphadenopathy-associated virus (LAV), AIDS-associated retrovirus (ARV), or human immunodeficiency virus (HIV). The designation "human immunodeficiency virus" (HIV) has been accepted by a subcommittee of the International Committee for the Taxonomy of Viruses as the appropriate name for the retrovirus that has been implicated as the causative agent of AIDS (Science 1986;232:697).

TABLE 2. Acquired immunodeficiency syndrome (AIDS) cases reported, by transmission category, by year, and with percentage of yearly increases — United States, through December 8, 1986

Transmission category	Before 12/8/82 No.	12/9/82- 12/8/83 No. (% Inc)*	12/9/83- 12/8/84 No. (% Inc)*	12/9/84- 12/8/85 No. (% Inc)*	12/9/85- 12/8/86 No. (% Inc)*	Total
Adult male						
Homosexual/bisexual only	562	1,252 (123)	2,720 (117)	5,306 (95)	8,322 (57)	18,162
IV drug user only	98	295 (201)	561 (90)	1,132 (102)	1,674 (48)	3,760
Both homosexual/IV drug user	74	194 (162)	396 (104)	576 (45)	925 (61)	2,165
Hemophilia/coagulation disorder	6	11 (83)	31 (182)	66 (113)	119 (80)	233
Other heterosexual contact						
Sexual contact	1	1 (0)	10 (900)	20 (100)	49 (145)	81
Non-U.S. born [†]	40	68 (70)	96 (41)	111 (16)	146 (32)	461
Transfusion	1	14 (1300)	28 (100)	96 (243)	185 (93)	324
Undetermined	16	51 (219)	81 (59)	158 (95)	342 (116)	648
Male subtotal	798	1,886 (136)	3,923 (108)	7,465 (90)	11,762 (58)	25,834
Adult fe.						
IV drug user only	26	79 (204)	152 (92)	276 (82)	430 (56)	963
Hemophilia/coagulation disorder	0	0 —	2 —	2 (0)	3 (50)	7
Other heterosexual contact						
Sexual contact	7	20 (186)	47 (135)	100 (113)	230 (130)	404
Non-U.S. born [†]	9	12 (33)	13 (8)	31 (139)	45 (45)	110
Transfusion	2	12 (500)	20 (67)	57 (185)	90 (58)	181
Undetermined	7	17 (143)	24 (41)	65 (171)	92 (42)	205
Female subtotal	51	140 (175)	258 (84)	531 (106)	890 (68)	1,870
Adult subtotal	849	2,026 (139)	4,181 (106)	7,996 (91)	12,652 (58)	27,704
Pediatric	1	41 (4,000)	50 (22)	124 (148)	178 (44)	394
Total	850	2,067 (143)	4,231 (105)	8,120 (92)	12,830 (58)	28,098

*Percent increase.

[†]Includes persons without other identified risks who were born in countries in which heterosexual transmission is believed to play a major role although precise means of transmission have not yet been fully defined.

Pediatric patients. Among 394 AIDS patients < 13 years of age, 347 (88%) are < 5 years old. Of those, 20% are white; 57%, black; and 22%, Hispanic. Fifty-five percent are male. Fifty-two percent were diagnosed with PCP, 47% with other opportunistic diseases and no PCP, and 1% with KS alone. Three hundred and eleven (79%) pediatric patients came from families in which one or both parents had AIDS or were at increased risk for developing AIDS; 22 (6%) had hemophilia and 51 (13%) had received transfusions of blood or blood components before onset of illness. Risk factor information on the parents of the 10 (3%) remaining cases is incomplete. Pediatric patients have been reported from 29 states, the District of Columbia, and Puerto Rico; reported cases per area ranged from one to 141 (median = 4). Over 72% of the 311 pediatric patients who acquired infection perinatally are residents of Florida, New Jersey, and New York.

Other modes of transmission. There continues to be no evidence of nonspecific transmission through casual contact; insect bites; or foodborne, waterborne, or environmental spread among AIDS cases. The situation is most clear in the 5- to 15-year-old age group, which lies between the youngest children for whom perinatal transmission is the most important and the adult age groups where sexual and drug related transmission predominates. Five to 15 year olds, who include the majority of school children, comprise 16% of the U.S. population (6). However, only 62 AIDS cases (0.2% of total cases) have occurred in this large group, which is exposed like other groups to casual contact with HTLV-III/LAV-infected persons, insects, and environmental factors. Of these, 61 (98%) fit into established risk categories. The risk factor investigation is incomplete on the remaining case.

Reported by State and Territorial Epidemiologists; AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: The number of reported AIDS cases continues to increase. An analysis of past trends using empirical models projects a cumulative case total of 270,000 by 1991 (7,8). The proportion of AIDS cases among most transmission categories has remained relatively constant. The geographic distribution of men and women with AIDS differs significantly ($p < .001$). Most reports of women with AIDS continue to come from Florida, New Jersey, and New York, while these states account for a much smaller proportion of male cases. Since most pediatric AIDS cases result from perinatal transmission of HTLV-III/LAV, the race/ethnicity and geographic distribution of pediatric AIDS patients is similar to that of reported AIDS cases among women.

The proportion of AIDS patients diagnosed with KS is declining (9-11), but most KS (95%) continues to be diagnosed among homosexual or bisexual men. KS alone is infrequently diagnosed among women (3% of cases) and children (4%) with AIDS. The reasons that certain patients develop KS remain unclear (12,13).

Numerous studies and continuing investigations of AIDS patients not belonging to recognized risk groups have not supported the existence of new modes of HTLV-III/LAV transmission (14-17). History of other sexually transmitted diseases among the "no identified risk" group as well as prostitute contact among male AIDS patients suggest that sexual contact with partners whose risk was unrecognized or unreported by the patient may be the mode of HTLV-III/LAV transmission for some of these patients. Given current epidemiologic data, AIDS patients who were born outside the United States and who do not have one of the predominant risk exposures have been moved from the "undetermined" transmission category to the "heterosexual contact" category. This move has increased the "heterosexual contact" category from 2% to 4% of adult cases and has decreased the "undetermined" category from 5% to 3%.

The HTLV-III/LAV antibody test allows further refinement of the case definition, especially in disease categories of lower specificity CDC proposes, with the advice of outside consultants, to revise the case definition for national reporting of AIDS. One major objective of this revision is to increase the sensitivity and specificity of the case definition through greater diagnostic use of HTLV-III/LAV antibody test results.

References

1. CDC. Update: acquired immunodeficiency syndrome (AIDS)—United States. MMWR 1983;32:688-91.
2. Selik RM, Haverkos HW, Curran JW. Acquired immune deficiency syndrome (AIDS) trends in the United States, 1978-1982. Am J Med 1984;76:493-500.
3. CDC. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. MMWR 1985;34:373-5.
4. CDC. *Pneumocystis pneumonia*—Los Angeles. MMWR 1981;30:250-2.
5. CDC. Kaposi's sarcoma and *Pneumocystis pneumonia* among homosexual men—New York City and California. MMWR 1981;30:305-8.
6. Bureau of the Census. Table 1: total population including Armed Forces overseas—estimates, by age, sex, and race: 1980 to 1985. In: Estimates of the population of the United States, by age, sex, and race: 1980 to 1985. Washington, DC: Department of Commerce, 1986: (population estimates and projections [series P-25, no. 985]).

7. Public Health Service. Coolfont report: a PHS plan for prevention and control of AIDS and the AIDS virus. Public Health Rep 1986;101:341-8.
8. Morgan WM, Curran JW. Acquired immunodeficiency syndrome: current and future trends. Public Health Rep 1986;101:459-66.
9. CDC. Update: Acquired immunodeficiency syndrome—United States. MMWR 1986;35:17-21.
10. Rutherford GW, Echenberg DF, Wu AC, Rauch KJ, Piland TH, Barnhart JL. The epidemiology of AIDS-related Kaposi's sarcoma in San Francisco: evidence for decreasing incidence. Paris, France: International Conference on AIDS, June 23-25, 1986.
11. Selik RM, Starcher ET, Curran JW. AIDS patients reported to CDC: percentages with various infections and cancers. Paris, France: International Conference on AIDS, June 23-25, 1986.
12. Drew WL, Mills J, Hauer L, Miner RC. Declining prevalence of Kaposi's sarcoma in homosexual AIDS patients is paralleled by declining incidence of CMV infection. New Orleans, Louisiana: Inter-science Conference on Antimicrobial Agents and Chemotherapy, September 28-October 1, 1986.
13. Mathur-Wagh U, Mildvan D, Senie RT. Follow-up at 4½ years on homosexual men with generalized lymphadenopathy [Letter]. N Eng J Med 1985;313:1542-3.
14. Fischl MA, Dickinson GM, Scott GB, Klimas N, Fletcher MA, Parks W. Heterosexual and household transmission of the human T-lymphotropic virus type III. Paris, France: International Conference on AIDS, June 23-25, 1986.
15. Rogers MF, Whitts CR, Sanders R, et al. Can children transmit human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV) infection? Paris, France: International Conference on AIDS, June 23-25, 1986.
16. Peterman TA, Stoneburner RL, Allen JR. Risk of HTLV-III/LAV transmission to household contacts of persons with transfusion-associated HTLV-III/LAV infection. Paris, France: International Conference on AIDS, June 23-25, 1986.
17. Saltzman BR, Friedland GH, Rogers MF, et al. Lack of household transmission of HTLV-III/LAV infection. Paris, France: International Conference on AIDS, June 23-25, 1986.

Survey of Non-U.S. Hemophilia Treatment Centers for HIV Seroconversions Following Therapy With Heat-Treated Factor Concentrates

Until 3 years ago, non-heat-treated factor concentrates were used in treating congenital and acquired clotting factor deficiencies. At that time, heat-treated factor concentrates were introduced because the unheated concentrates had been epidemiologically linked with the exposure of large numbers of U.S. hemophilia patients to the human immunodeficiency virus (HIV) (1). There have now been a few reports of HIV seroconversion associated with heat-treated factor concentrates (2,3). Because several hemophilia treatment centers (HTCs) outside the United States began using heat-treated factor concentrates somewhat earlier, a sample of major non-U.S. HTCs identified by the U.S. National Hemophilia Foundation were contacted during November and December 1986 and asked to help estimate the continued risk of seroconversion among their patients deficient in factor VIII and factor IX. Patients with von Willebrand's disease and other clotting factor deficiencies were not included.

The directors of 13 HTCs located in western Europe, Canada, and Australia were asked to provide information concerning: 1) HIV antibody seroprevalence rates within their patient populations; 2) whether they were using, and when they had begun to use, heat-treated factor concentrate products (4-6); and 3) details regarding any HIV seroconversions occurring among their patients while receiving heat-treated factor concentrates. Most HTCs monitor the serologic status of their seronegative hemophilia A and B patients at approximately 3-month intervals and were confident of all these patients' serologic status as of late July 1986. Of the combined total of 2,370 hemophilia A patients and 434 hemophilia B patients served by the HTCs in this survey, over 1,300 were still seronegative when heat-treated factor concentrates became available. Approximately 50% of the seronegative patients were classified as severely deficient in factor VIII or factor IX; the remainder had either moderate or mild hemophilia*.

Of the 23 patients who had their first documented positive HIV antibody test after receiving heat-treated factor concentrate, 16 seroconverted within 6 months of last receiving untreated factor concentrates. The remaining seven individuals fell into three groups (Table 1). Group 1: Two patients were first found to be seropositive more than 6 months after starting to use heat-treated factor concentrate products (at 7 and 10 months, respectively). However, for both of these patients, the last seronegative test had taken place several months before their last treatment with unheated factor concentrates. Group 2: Two patients who were seronegative within the initial 6 months of heat-treated factor concentrate therapy (at 3 and 5 months, respectively) were not tested again until after the initial 6 months (at 8 and 10 months, respectively), at which time they were seropositive. Group 3: Three pediatric patients were seronegative at 8, 12, and 16 months after first receiving heat-treated factor VIII concentrate but had their first of many consistently seropositive tests at 10, 13, and 22 months after treatment, respectively.

The patients in Group 3 had no reported risk factors for HIV infection other than hemophilia and reportedly had received no other blood components during this time period. All three pediatric patients were severely deficient in factor VIII. One child, a 6-year-old, had received vials from four lots in the 10-month interim before seroconversion. He is presently asymptomatic and his reported T-cell values are normal; no HIV cultures have been attempted. The other two children, aged 4 and 13, had received large amounts of heat-treated factor VIII concentrates for extended periods either as therapy for an inhibitor or as routine care. The 4-year-old was found to be HIV culture positive in 1986 and now has AIDS. The 13-year-old had severe T-cell abnormalities by mid-1986 and now has lymphadenopathy and encephalopathy.

The many lots of concentrate received by each of the three patients in Group 3 had come from three different U.S. manufacturers. The plasma used by each of the U.S. manufacturers was collected before serologic screening of donors for HIV antibody became available. In addition, during the first 5 months of the 13-month interval before seroconversion, one of the three patients had also received extremely large amounts of heat-treated factor VIII concentrate prepared by a European manufacturer using a wet-heat process. The manufacturer had used unscreened plasma from U.S. donors.

The three patients who seroconverted (Group 3) represent 0.7% of the total 450 initially seronegative hemophilia A patients and 0.2% of the total 1,300 patients who were serologically monitored for >1 year after beginning to use unscreened, heat-treated factor. Since

*Severity is defined on the basis of percent of normal factor activity: severe, <1% of normal; moderate, 2-5% of normal; mild, >5% of normal.

November 1985, no seroconversions have been observed among the patients included in the survey.

Although information on the transition to using unscreened, heat-treated factor in each HTC is readily available, the dates of subsequent transition to using donor-screened, heat-treated factor concentrate products by each HTC are not. One HTC reported beginning to use donor-screened, heat-treated factor therapy in August 1985; however, for most HTCs, this transition occurred between February and July 1986. No cases of seroconversion following the use of donor-screened, heat-treated products were identified through this survey.

Four percent (50) of the 1,300 seronegative patients in this survey were followed for > 1 year while receiving donor-screened, heat-treated factor concentrates. Follow-up on the remainder is approaching 1 year. In early March 1987, supplemental information was obtained from eight of the 13 HTCs. These eight HTCs collectively have 60% of the seronegative patients; no further seroconversions have been found. Although over 600 patient-years of therapy with donor-screened product have elapsed without a recognized HIV seroconversion, the risk associated with unscreened, heat-treated product is so low that several more months of surveillance will be required before a statistically significantly further reduction of risk can be substantiated.

Reported by M Blomback, MD, S Schulman, MD, Stockholm, E Berntorp, MD, Malmö, L Stigendal, MD, Göteborg, Sweden; EP Mauser-Bunschoten, MD, Bilthoven, Netherlands; T Lambert, MD, Paris, France; H Egli, MD, H Brackmann, MD, Bonn, Federal Republic of Germany; PM Mannucci, MD, Milan, Italy; PBA Kernoff, MD, London, P Jones, MD, Newcastle-upon-Tyne, CR Rizza, MD, Oxford, AL Bloom, MD, Cardiff, United Kingdom; MJ Inwood, MD, London, Ontario, Canada; KA Rickard, MD, Sydney, New South Wales, Australia; Div of Host Factors, Center for Infectious Diseases, CDC.

Editorial Note: Earlier published reports disclosed no seroconversions among selected hemophilia patients followed for up to 1 year after beginning therapy with heat-treated factor concentrates (7-10). However, during the past 12 months, published (2,3) and unpublished reports (personal communication, I Walker, MD, Hamilton, Ontario, Canada; FG Hill, MD, MRC Path, Birmingham, United Kingdom; G Mariani, MD, Rome, Italy) have described several hemophilia patients who had seroconverted after receipt of unscreened, heat-treated factor concentrates. In June 1986, one U.S. manufacturer (Armour Pharmaceutical Company) offered to exchange any remaining heat-treated factor VIII concentrates produced from plasma collected before the availability of a test for HIV antibody with the equivalent amount of antibody-screened product. Similar exchanges are now available through four other U.S. producers (Alpha Therapeutics, American Red Cross, Cutter Laboratories, Hyland Therapeutics).

The influence of previous exposure to allogeneic proteins and other infectious agents as well as the HIV inoculum size and differences in inoculum strain may alter the seroconversion intervals among hemophilia patients. For this reason, it is currently uncertain whether anecdotal reports that seroconversion in other risk groups occurs within 8 to 12 weeks after exposure can be generalized to hemophilia patients (11). One study suggests that the vast majority of hemophilia seroconversions would be detectable ≤ 26 weeks (12). The distribution of seroconversion latency periods for hemophilia patients is not yet known. Therefore, it is uncertain whether any of the three seroconversions in persons with a documented seronegative test ≥ 6 months after beginning to use only heat-treated factor concentrates could be associated with the former source of exposure.

No cases of seroconversion among patients using only donor-screened, heat-treated products have been reported to date. With the exception of the HTC surveyed in Australia, less than a year has elapsed since most of the HTCs surveyed began administering donor-

TABLE 1. Distribution of patients in surveyed non-U.S. hemophilia treatment centers, by interval between therapy with heat-treated factor concentrates and HIV seroconversion

Last seronegative test	First seropositive test after initial 6 months
Preceding heat-treated factor usage	2
During initial 6 months of heat-treated factor usage	2
After initial 6 months of heat-treated factor usage	3

screened, heat-treated factor concentrates. Further longitudinal studies by several of the HTC's in this survey may substantiate the additional margin of safety provided by screening donated plasma for HIV antibody. Donor-screened, heat-treated factor concentrates remain the recommended therapy for patients requiring factor replacement.

References

1. CDC. Update: acquired immunodeficiency syndrome (AIDS) in persons with hemophilia. MMWR 1984;33:589-91.
2. White GC, Matthews TJ, Weinhold KJ, et al. HTLV-III seroconversion associated with heat-treated factor VIII concentrate [Letter]. Lancet 1986;1:611-2.
3. Van den Berg W, ten Cate JW, Breederveld C, Goudsmit J. Seroconversion to HTLV-III in haemophiliac given heat-treated factor VIII concentrate [Letter]. Lancet 1986;1:803-4.
4. Levy JA, Mitra G, Mozen MM. Recovery and inactivation of infectious retroviruses from factor VIII concentrates. Lancet 1984;2:722-3.
5. Spire B, Dormont D, Barre-Sinoussi F, Montagnier L, Chermann JC. Inactivation of lymphadenopathy-associated virus by heat, gamma rays, and ultraviolet light. Lancet 1985;1:188-9.
6. McDougal JS, Martin LS, Cort SP, Mozen M, Heldebrandt CM, Evatt BL. Thermal inactivation of the acquired immunodeficiency syndrome virus, human T lymphotropic virus-III/lymphadenopathy-associated virus, with special reference to antihemophilic factor. J Clin Invest 1985;76:875-7.
7. Rouzioux C, Chamaret S, Montagnier L, Carnelli V, Rolland G, Mannucci PM. Absence of antibodies to AIDS virus in haemophiliacs treated with heat-treated factor VIII concentrate [Letter]. Lancet 1985;1:271-2.
8. Felding P, Nilsson IM, Hansson BG, Biberfeld G. Absence of antibodies to LAV/HTLV-III in haemophiliacs treated with heat-treated factor VIII concentrate of American origin [Letter]. Lancet 1985;2:832-3.
9. Gazengel C, Larrieu MJ. Lack of seroconversion for LAV/HTLV-III in patients exclusively given unheated activated prothrombin complex prepared with ethanol step [Letter]. Lancet 1985;2:1189.
10. Mannucci PM, Gringeri A, Ammassari M. Antibodies to AIDS and heated factor VIII [Letter]. Lancet 1985;1:1505-6.
11. Ho DD, Sarngadharan MG, Resnick L, Dimarzio-Veronese F, Rota TR, Hirsch MS. Primary human T-lymphotropic virus type III infection. Ann Intern Med 1985;103:880-3.
12. Ludlam CA, Tucker J, Steel CM, et al. Human T-lymphotropic virus type III (HTLV-III) infection in seronegative haemophiliacs after transfusion of factor VIII. Lancet 1985;2:233-6.

Tuberculosis and AIDS — Connecticut

Until 1983, the incidence of tuberculosis in Connecticut had steadily declined for several decades. In 1982, it reached its lowest point, 5.0 cases per 100,000 population. Since then, tuberculosis incidence in Connecticut has fluctuated above that level, with a rate of 6.2 in 1983, 5.6 in 1984, and 5.1 in 1985. A rate of 6.0 is projected for 1986. This would be an 18% increase over 1985. Concern about a possible association between human immunodeficiency virus (HIV) infection and the rise in tuberculosis morbidity led to an evaluation of data on acquired immuno-deficiency syndrome (AIDS) and tuberculosis in Connecticut.

The entire AIDS register was confidentially linked to the tuberculosis case register dating back to 1970 to determine the proportion of tuberculosis patients with a diagnosis of AIDS, the proportion of AIDS patients with tuberculosis, and the interval between the diagnosis of tuberculosis and AIDS. The following selected characteristics of those with both diagnoses were also studied: age, sex, race and ethnicity, geographic location by city size, and risk factors for a diagnosis of AIDS. Patients were placed in subgroups by each of these characteristics, and the incidence rate of tuberculosis in individuals with and without AIDS in each subgroup was calculated and compared. A 3-year incidence rate of tuberculosis was used for these comparisons because most diagnoses of tuberculosis in AIDS patients occurred in the 3-year period beginning 30 months before and ending 6 months after the diagnosis of AIDS.

As of September 1, 1986, 18 cases of tuberculosis had been diagnosed among the 299 cumulatively reported AIDS cases in Connecticut. The 18 tuberculosis patients with AIDS (TB/AIDS) ranged from 24 to 53 years of age, with a median of 33 years. Fourteen (78%) were male; 11 (61%) were black; 13 (72%) came from the six cities in Connecticut with a population of 100,000 or greater; and seven (39%) were intravenous drug abusers. One of the 18 cases of tuberculosis was diagnosed in 1973 and another in 1980. The remaining 16 cases were diagnosed after January 1, 1982, and represent 5.4% of all AIDS cases reported to date and 2.0% of all 816 tuberculosis cases diagnosed and reported from 1982 through 1986. When these 16 cases are analyzed by year of diagnosis, there appears to be no significant rise or fall in the frequency of tuberculosis patients with AIDS (TB/AIDS) for the years 1982 through 1986.

Compared with tuberculosis patients without AIDS in Connecticut, TB/AIDS patients were younger and more likely to be male, black, and from a large city. Compared with AIDS patients without tuberculosis, TB/AIDS patients were more likely to be black and from a large city and to have intravenous drug abuse as an AIDS risk factor. Age and sex distribution were similar in both groups.

Among the 18 TB/AIDS patients, the diagnosis of tuberculosis occurred from 10 years before to 19 months after the diagnosis of AIDS, with a median of 4 months before the diagnosis of AIDS. Fourteen (78%) of TB/AIDS patients were diagnosed as having tuberculosis within 3 years of their diagnosis of AIDS (2.5 years before to 0.5 years after).

Table 4 shows the crude 3-year incidence rate of tuberculosis in AIDS patients and in the general population without AIDS according to sex, race, and city size as well as the incidence

TABLE 4. Three-year incidence of tuberculosis in 20- to 49-year-olds with and without AIDS, by selected demographic characteristics — Connecticut, 1986

Characteristics	AIDS patients*		General population†		Risk ratio§
	TB rate	(cases)	TB rate	(cases)	
Sex					
Male	6,250	(10)	18.8	(119)	333
Female	7,692	(2)	12.7	(84)	605
Race					
Black	12,121	(8)	102.8	(95)	118
White	3,670	(4)	5.4	(63)	677
Other	—	(0)	112.4	(45)	—
City Size					
≥100,000	9,677	(9)	44.7	(111)	216
<100,000	3,226	(3)	8.8	(92)	367
Adjusted¶	2,671	(12)	15.7	(203)	170.3

*Incidence of tuberculosis 2.5 years before to 0.5 years after diagnosis of AIDS per 100,000 AIDS patients as of 4/1/86.

†3-year incidence of tuberculosis per 100,000 individuals without AIDS, 1982-1984.

§Ratio of 3-year incidence of TB/AIDS to TB/non-AIDS.

¶Adjusted for age (5-year intervals), race, sex, and city size according to 1980 census.

rate adjusted for these three factors and age. In all groups, the rate of tuberculosis (risk ratio) in AIDS patients was more than 100 times the incidence in the general population.

Reported by JL Hadler, MD, MPH, State Epidemiologist, R Burger, Pulmonary Diseases and AIDS Programs, Connecticut State Dept of Health Svcs; Div of Tuberculosis Control, Center for Prevention Svcs, CDC.

Editorial Note: The demographic characteristics of TB/AIDS patients in Connecticut are similar to those found elsewhere; individuals are most likely to come from groups that have a higher incidence of tuberculosis and are at risk for AIDS (1-3).

The following factors suggest an association between tuberculosis and AIDS in Connecticut: the 5.4% incidence of tuberculosis in AIDS cases, the clustering of the development of tuberculosis and AIDS within a distinct time period (within 3 years of diagnosis of AIDS), and the 100-fold or greater risk of tuberculosis among AIDS patients than among the general population. The risk that persons with latent tuberculous infection who develop AIDS will develop clinically active tuberculosis cannot be determined from these data. However, to the extent that individuals with AIDS are representative of the general population in prevalence and incidence of tuberculous infection, this risk could be as much as 100- to 200-fold greater than that of their non-HIV-infected counterparts.

The total number of AIDS patients in the United States meeting the CDC surveillance case definition represents only a fraction of the number of persons with HIV infection. It has been estimated that, in 1985, for every diagnosed case of AIDS, there were 50 to 100 persons with HIV infection (4). The number of tuberculosis patients with HIV infection but without AIDS in Connecticut may also exceed the number who have overt AIDS.

These data further support recently published guidelines that risk factors for HIV should be identified as part of the evaluation of persons with tuberculous infection (5). HIV antibody testing should be offered, and, where there is both tuberculous infection and HIV infection, isoniazid preventive therapy should be offered. Conversely, persons who are positive for HIV antibody should be offered tuberculin skin testing, and isoniazid preventive therapy should be offered to reactors (5).

References

1. CDC. Tuberculosis and acquired immunodeficiency syndrome—Florida. *MMWR* 1986;35:587-90.
2. Sunderam G, McDonald RJ, Maniatis T, Oleske J, Kapila R, Reichman LB. Tuberculosis as a manifestation of the acquired immunodeficiency syndrome (AIDS). *JAMA* 1986;256:362-6.
3. Stoneburner RL, Kristal A. Increasing tuberculosis incidence and its relationship to acquired immunodeficiency syndrome in New York City. Atlanta, Georgia: International Conference on Acquired Immunodeficiency Syndrome (AIDS). April 14-17, 1985.
4. Curran JW, Morgan WM, Hardy AM, Jaffe HW, Darrow WW, Dowdle WR. The epidemiology of AIDS: current status and future prospects. *Science* 1985;229:1352-7.
5. CDC. Diagnosis and management of mycobacterial infection and disease in persons with human T-lymphotropic virus type III/lymphadenopathy-associated virus infection. *MMWR* 1986;35:448-52.

Human Immunodeficiency Virus Infection in Transfusion Recipients and Their Family Members

CDC has received a report of human immunodeficiency virus (HIV) infection among multiply-transfused leukemia patients in New York City. In addition, there have been several reports that persons with transfusion-associated HIV infection have transmitted the virus to their sexual partners and newborn children. All infected transfusion recipients described in these reports had received blood or blood components before routine screening of donated blood for HIV antibody was begun in the spring of 1985.

Multiply-Transfused Leukemia Patients

During the past year, four long-term leukemia survivors at Memorial Sloan-Kettering Cancer Center in New York City developed unexplained fever, weight loss, diarrhea, or lymphadenopathy. They subsequently had positive serological tests for HIV antibody. A retrospective study of other multiply-transfused leukemia patients was conducted to determine how many had been infected with HIV. Informed consent was obtained from all living patients. Positive enzyme immunoassay (EIA) tests were confirmed by Western blot assay. Patients known to have other risk factors for HIV infection were excluded from the study.

Sera were located for 182 deceased and obtained from 22 surviving leukemia patients treated during the years 1978-1986. Sixteen of these transfusion recipients were seropositive for HIV antibody (Table 1). They had received a mean of 27 units of packed red blood cells (range 2-56) and 137 units of platelets (range 10-483). Forty-five percent of these 204 patients had acute myelogenous leukemia; 20% had acute lymphocytic leukemia; 13%, chronic myelogenous leukemia; 4%, chronic lymphocytic leukemia; 6%, myelodysplastic syndromes; and 12%, other or unclassified leukemias. There was no correlation between type of leukemia and the presence of HIV antibody. An additional 23 newly diagnosed, untreated, and untransfused leukemia patients were tested and all were seronegative.

Additional Case Reports From Other Areas

Case 1: An elderly man with no known risk for AIDS received multiple units of blood in early 1982, including one from a donor who subsequently tested positive for HIV antibody. The recipient developed *Pneumocystis carinii* pneumonia (PCP) in 1983 and died in 1984. His wife, who did not have any other risk factors for AIDS, had had vaginal intercourse with him until he became ill in late 1982. In late 1984, her HIV antibody test was positive and she was diagnosed as having a type of lymphoma indicative of AIDS (1).

Case 2: A pregnant woman with no other risk factors for AIDS received four units of blood in 1978, including one from a donor who later tested positive for HIV antibody. A son, born in 1980, had failure to thrive beginning at 13 months of age and died with PCP in 1986. The woman, her son, her husband, and the child born shortly after the transfusion all tested positive for HIV antibody.

Reported by DA Scheinberg, MD, PhD, GY Minamoto, MD, K Dietz, MS, JWM Gold, MD, T Gee, MD, D Armstrong, MD, J Gabrilove, MD, B Clarkson, MD, N Chein, MS, L Reich, MD, Memorial Sloan-Kettering Cancer Center, DL Morse, MD, State Epidemiologist, New York State Dept of Health; M Batt, MD, Lutheran General Hospital, Park Ridge, HJ Miller, MD, Dept of Medicine, Evanston Hospital, Evanston, B Stevko, MD, Illinois Dept of Public Health; LA Chambers, MD, American Red Cross Blood Services—Northeast Region, Dedham, L Kunches, GF Grady, MD, State Epidemiologist, Massachusetts Dept of Public Health; Div of Blood and Blood Products, Center for Drugs and Biologics, FDA; AIDS Program, Center for Infectious Diseases, CDC.

TABLE 1. HIV serology results in leukemia patients, by year of specimen collection — Memorial Sloan-Kettering Cancer Center, New York City

Years	Total number of patients tested	Number with positive test	Estimated* risk per component
1978-80	86 (55M,31F) [†]	0 (0%)	0.00%
1981-83	77 (39M,38F)	9 (12%) (6M,3F)	0.07%
1984-86 [§]	41 (21M,20F)	7 (17%) (2M,5F)	0.10%
Total	204 (115M,89F)	16 (8%) (8M,8F)	0.05%

*Estimated risk based on an average of 164 components per recipient.

[†]M=males; F=females.

[§]These patients were treated before screening of blood products began in March 1985; 22 long-term survivors, four of whom were seropositive, are included.

Editorial Note: At present, prevention of HIV infection and AIDS is dependent upon deferral of blood or plasma donation by persons at increased risk for AIDS, testing of donated blood and plasma for HIV antibody, heat treatment of clotting factor concentrates, avoidance of unprotected sexual contact and needle sharing by persons infected with HIV, and prevention of perinatal transmission by infected women. Counseling and HIV antibody testing have been recommended for persons at risk for infection (including homosexual/bisexual men, intravenous drug abusers, hemophilia patients, prostitutes, and persons who have had sexual contact with members of these groups) (2). Routine counseling and antibody testing have not been recommended for blood transfusion recipients because, in general, their risk for infection is extremely low. However, as illustrated by this report and others (3), some multiply-transfused persons may be at a higher risk for HIV infection. In addition, some persons with transfusion-associated HIV infection have transmitted the virus to their sexual partners and, perinatally, to their infant children.

Although the number of infected transfusion recipients in the United States is unknown, it can be approximated using estimates of the prevalence of infection in donors, the efficiency of transmission, and the number of units transfused per year. In 1985, 0.04% of donations were positive for HIV antibody by Western blot assay (4). If 0.04% had been the seroprevalence among donors in the year prior to screening, if all seropositive units had transmitted infection (5), and if each seropositive unit had gone to a different recipient, then 7,200 of the approximately 18 million components transfused in 1984 (American Blood Commission, unpublished data) might have transmitted infection. If 60% of these recipients have died from their underlying disease (6), then approximately 2,900 living recipients who acquired a transfusion-associated HIV infection in 1984 would remain. Most of these would be asymptomatic. The number of infected donors was probably lower in earlier years. Mathematical projections from reported transfusion-associated AIDS cases estimate that approximately 12,000 people now living in the United States acquired a transfusion-associated HIV infection between 1978 and 1984 (7).

Blood banking organizations in the United States have begun "look-back" programs to identify previous recipients of blood from donors who tested positive for HIV antibody after screening began. In one region, 70% of recipients identified through such a program had HIV antibody (8). However, look-back programs cannot identify all infected transfusion recipients because many infected donors may have refrained from donating or become too ill to continue to donate after HIV serologic testing of donors began.

The risk of HIV transmission by transfusion was low, even before screening, and has been virtually eliminated by the routine screening of donated blood and plasma. However, since HIV-infected persons are at risk for developing AIDS or related conditions themselves and may transmit infection to others, physicians should consider offering HIV antibody testing to some patients who received transfusions between 1978 and late spring of 1985. This consideration should be based on the likelihood of infection in a recipient and the likelihood of transmission from that recipient. The risk of infection is greatest if the recipient received large numbers of transfusions and if the blood was collected during the few years before screening in an area with a high incidence of AIDS. (The leukemia patients in this report received many units of blood and blood components in an area with a higher prevalence of HIV than most parts of the United States, so their seropositivity rate is higher than would be expected in other patients. Conversely, persons who received a small number of units in a low prevalence area would have an extremely low risk of HIV infection.) Testing is particularly important if the patient is sexually active. Since the overall prevalence of infection in transfusion recipients is expected to be low, the positive predictive value of EIA screening tests for HIV antibody will be much lower than that seen when testing high-risk populations (9). Therefore, all transfusion recipients with a positive EIA should also have their serum tested by a second method (Western blot assay, immunofluorescence assay) before they are informed of their test result. Seropositive persons should be evaluated for signs and symptoms of AIDS or related conditions and counseled regarding the avoidance of HIV transmission to others.

References

1. Duncan ED, Miller HJ, McKeever WP. Non-Hodgkin's lymphoma, HTLV-III/LAV, and HTLV-III/LAV antibody in the wife of a man with transfusion-acquired AIDS. *Am J Med* 1986;81:898-900.
2. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR* 1986;35:152-5.
3. Troy K, Cuttner J, Zaroulis C. Incidence of anti-HIV antibodies in patients with acute leukemia treated between 1978 and 1986 [Abstract]. *Blood* 1986;68:135a.
4. Schorr JB, Berkowitz A, Cumming PD, Katz AJ, Sandler SG. Prevalence of HTLV-III antibody in American blood donors [Letter]. *N Engl J Med* 1985;313:384-5.

5. Ward JW, Deppe DA, Samson S, et al. Risk of human immunodeficiency virus infection from blood donors who later developed the acquired immunodeficiency syndrome. *Ann Intern Med* 1987; 106:61-2.
6. Perkins HA, Samson S, Rosenschein S, et al. Risk of AIDS from blood donors who subsequently develop AIDS. Presented at the International Conference on Acquired Immunodeficiency Syndrome (AIDS), Atlanta, Georgia, April 16, 1985.
7. Peterman TA, Lui K, Lawrence DN, Allen JR. Estimating the risks of transfusion-associated AIDS and HIV infection. *Transfusion* (in press).
8. Menitove JE. Status of recipients of blood from donors subsequently found to have antibody to HIV [Letter]. *N Engl J Med* 1986;315:1095-6.
9. Ward JW, Grindon AJ, Feorino PM, Schable C, Parvin M, Allen JR. Laboratory and epidemiologic evaluation of an enzyme immunoassay for antibodies to HTLV-III. *JAMA* 1986;256:357-61.

Antibody to Human Immunodeficiency Virus in Female Prostitutes

Seroprevalence surveys for antibody to human immunodeficiency virus (HIV) in women with histories of prostitution have shown varying results since testing began in 1984. In sub-Saharan Africa, where HIV is thought to be transmitted primarily through heterosexual exposure (1-3), one (1%) of 98 prostitutes tested in Accra, Ghana (4), to 29 (88%) of 33 prostitutes in Ngoma, Rwanda (5), had HIV antibody (3-7). In Europe, where homosexual exposure and abuse of intravenous (IV) drugs are major risk factors for HIV infection (8), none of 50 prostitutes tested in London (9), none of 56 in Paris (10), and none of 399 in Nuremberg, West Germany (11), had antibody to HIV. However, 10 (71%) of 14 prostitutes who abused IV drugs in Pordenone, Italy (12), and 14 (78%) of 18 who abused IV drugs in Zurich, Switzerland (13), were infected. Seventeen (1%) of nearly 2,000 registered prostitutes in six West German cities were HIV-antibody positive; half of these infected women abused IV drugs (14). In Athens, Greece, 12 (6%) of 200 registered prostitutes were HIV-antibody positive; none abused IV drugs (15).

As of March 10, 1987, 2,159 women in the United States were reported to have met the CDC surveillance case definition for AIDS. The cumulative incidence of AIDS in black and Hispanic women was more than 10 times that for white women (16). Over 70% of these women reported with AIDS resided in New York, New Jersey, or Florida (17). Over half (51%) had abused IV drugs; 27% were sexual partners of men with AIDS or at risk for AIDS; and 10% had received transfusions of blood or blood products. No risk factors have as yet been reported for the remaining 12% (18).

To assess HIV-antibody prevalence and determine risk factors in U.S. prostitutes, CDC is collaborating with others in an ongoing, cross-sectional study of women who have engaged in prostitution in seven geographic areas: Atlanta, Colorado Springs, Las Vegas, Los Angeles, Miami, Newark-Jersey City-Paterson, and San Francisco. Some collaborators are recruiting primarily incarcerated women (Los Angeles and Miami). Others are recruiting primarily through sexually transmitted disease (STD) clinics (Colorado Springs and Las Vegas); methadone maintenance clinics (the three northern New Jersey cities); or outreach efforts, such as newspaper advertising, circulation of pamphlets, and direct contacts on the street (Atlanta and San Francisco). Study participants are not necessarily representative of all female prostitutes in these areas.

For this study, prostitution is defined as the exchange of physical-sexual services for money or drugs. Any woman ≥ 18 years of age who has engaged in prostitution at least once since January 1, 1978, is eligible. Participation entails voluntary, informed consent; names and other personal identifiers are not recorded. Participants are interviewed for their medical histories and sexual and other exposures. They are also examined for signs of HIV infection and IV-drug abuse and are asked to provide 10 ml of blood for serologic testing. Serum is tested for HIV antibody by enzyme immunoassay and Western blot methods.

The analysis reported here has been restricted to the 835 study participants who were tested for HIV antibody and the 568 study participants for whom an interview form was submitted to CDC before March 10, 1987. The prevalence of HIV antibody in prostitutes so far tends to parallel the cumulative incidence of AIDS in women in the seven research sites (Table 1), suggesting that risk factors for AIDS in female prostitutes may be similar to those in other women living in these geographic areas. The prevalence of HIV antibody in prostitutes and the cumulative incidence of AIDS in women are highest in northern New Jersey and Miami. In southern Nevada, where only one woman has been reported with AIDS, none of 34 prostitutes have had HIV antibody.

In the seven areas, reported rates of AIDS were higher for black women (359.6/1,000,000) and Hispanic women (40.2/1,000,000) than for white (25.3/1,000,000) and other (Asian and Native American) women (16.2/1,000,000). Similarly, black and Hispanic prostitutes in these areas had a higher prevalence of HIV antibody (15%) than white and other prostitutes (7%) (odds ratio [OR] = 2.5; 95% confidence interval [CI] = 1.4-4.4).

Half the prostitutes interviewed in this multicenter collaborative study gave histories of IV-drug abuse; 47 (76%) of 62 with antibody to HIV have injected drugs (OR = 3.6; 95% CI = 2.0-6.7). IV-drug abuse is associated with HIV infection in prostitutes and with AIDS in women regardless of racial and ethnic background (Table 2).

Over 80% of prostitutes interviewed through January 1987 reported that at least one of their partners had used a condom. Husbands or boyfriends of the respondents were much less likely to use condoms during vaginal exposure than clients (16% as compared with 78%, $p = 0.005$). Twenty-two (4%) prostitutes reported condom use with each vaginal exposure during the past 5 years. Eleven percent of 546 prostitutes with unprotected vaginal exposure were HIV-antibody positive; none of 22 prostitutes whose partners always used condoms were seropositive ($p = 0.10$ after controlling for IV-drug abuse).

Reported by: JB Cohen, PhD, C Wofsy, MD, Association for Women's AIDS Research and Education (AWARE), University of California, San Francisco; P Gill, MD, S Aguilar, University of Southern California Cancer Center, Los Angeles, California. J Witte, MD, W Bigler, PhD, Florida Dept of Health and Rehabilitative Svcs, Tallahassee, Florida. RK Sikes, DVM, T Leonard, MA, Georgia Dept of Human Resources, Atlanta, Georgia. J French, MA, J Massey, C Sterk, Drs, New Jersey State Dept of Health, Trenton, New Jersey. O Ravenholt, MD, R Reich, C Campbell, PhD, Clark County Health District, Las Vegas, Nevada. J Potterat, L Phillips, El Paso County Health Dept, Colorado Springs, Colorado. AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: The collaborative study reported here was designed to determine the prevalence of HIV infection in female prostitutes in selected U.S. cities and the risk factors for infection in these women. Seroprevalence in study participants so far has varied widely from city to city and tends to parallel the cumulative incidence of AIDS in women in these areas. The major risk factor for HIV infection in prostitutes appears to be IV-drug abuse. Women with unprotected vaginal exposures also appear to be at greater risk than those whose male partners always used condoms. When used properly and consistently with each sexual exposure, latex condoms should greatly reduce the sexual transmission of HIV (7,11,19).

TABLE 1. HIV antibody in female prostitutes and reported AIDS cases in women — selected cities, United States, March 10, 1987

	Female prostitutes		Women with AIDS*	
	HIV-antibody positive/tested	Percent positive	No.	Cases/1,000,000†
Eastern United States				
Atlanta	1/92	(1.1)	8	12.5
Miami	47/252	(18.7)	100	145.3
Newark-Jersey City-Paterson	32/56	(57.1)	143	526.2
Western United States				
Colorado Springs	1/71	(1.4)	1	9.6
Las Vegas	0/34	(0.0)	1	16.0
Los Angeles	8/184	(4.3)	26	21.7
San Francisco	9/146	(6.2)	21	71.9

*Includes 45 women (≥ 16 years of age) from Miami and one from Newark who were born in countries where heterosexual transmission is believed to play a major role.

†Rate based on the number of females (≥ 16 years of age) reported as residing in the urban area or place of study (26).

TABLE 2. Risk factors for HIV antibody in female prostitutes and for AIDS in women, by race or ethnic group — selected cities, United States, March 10, 1987

	Female prostitutes*		Women with AIDS†	
	HIV-antibody positive/tested	Percent positive	No.	Percent of total
Black or Hispanic				
IV-drug abuser	31/124	(25.0)	108	(43.0)
Other, unknown	12/156§	(7.7)	143	(57.0)
Total	43/280	(15.4)	251	(100.0)
White or other				
IV-drug abuser	16/157	(10.2)	26	(53.1)
Other, unknown	3/127¶	(2.4)	23	(46.9)
Total	19/284	(6.7)	49	(100.0)

*Analysis restricted to the 564 study participants (of 835 tested) who answered the question regarding IV-drug abuse.

†Includes 46 women who were born in countries where heterosexual transmission is believed to play a major role, who were reported to CDC as meeting the surveillance case definition for AIDS, and who were residents of one of the seven research sites.

§Odds ratio = 4.0; 95% confidence interval = 2.0-8.2.

¶Odds ratio = 4.7; 95% confidence interval = 1.3-18.5.

Efforts to stop the spread of HIV infection in prostitutes and to their sexual partners require multiple approaches. These might include counseling and HIV-testing programs for individuals at risk for infection, additional control measures by local public health and law enforcement agencies, and the involvement of voluntary and other social service organizations.

Persons who continue to engage in prostitution remain at risk for acquiring and transmitting HIV. Prostitutes and their consorts should be provided counseling services and voluntary testing for HIV antibody (20-22). Seronegative persons who continue to engage in prostitution should insist on the use of condoms to reduce their own chances of infection. Seropositive prostitutes should know that the only certain way of preventing sexual transmission of the virus is to abstain and not engage in prostitution. Seropositive persons who continue to engage in prostitution should insist on the use of condoms to prevent transmission of the virus to others. IV-drug abusers should be offered treatment for their addictions and warned not to share needles or syringes.

State and local governments are approaching the problem of HIV infection in prostitutes in a variety of ways. Since March 1986, the Nevada Board of Health has required prostitutes in county-licensed brothels to be tested for HIV antibody as a condition for employment and monthly thereafter. If a woman is seropositive, she is denied employment as a prostitute. Since October 1986, Florida has required convicted prostitutes to be tested for STDs, including HIV. It is a misdemeanor in Florida for anyone who has tested positive for HIV and has been informed of the result to engage in prostitution. In Atlanta, the Mayor's Task Force on Prostitution has recommended educational materials for prostitutes, clients, and law-enforcement officers as well as voluntary testing for STDs (including assays for HIV antibody) for everyone arrested for sexual offenses and their steady partners.

Traditionally, medical care, therapy for drug addiction, welfare benefits, and vocational rehabilitation have not been routinely offered to women apprehended for prostitution (23-25). Now some organizations are introducing innovative approaches to male, as well as female, prostitutes. The California Prostitutes Education Project attempts to warn prostitutes about the dangers of unprotected exposures and provides educational sessions on how to prevent infection. Children of the Night (Los Angeles), Covenant House (New York City), Orion House (Seattle), and other social-service organizations offer counseling and sanctuary to homeless adolescents, including those involved in prostitution. State and local health departments often work closely with these organizations to provide voluntary testing and treatment for STDs.

References

1. Brun-Vezinet F, Rouzioux C, Montagnier L, et al. Prevalence of antibodies to lymphadenopathy-associated retrovirus in African patients with AIDS. *Science* 1984;226:453-6.
2. Piot P, Quinn TC, Taelman H, et al. Acquired immunodeficiency syndrome in a heterosexual population in Zaire. *Lancet* 1984;2:65-9.
3. Quinn TC, Mann JM, Curran JW, Piot P. AIDS in Africa: an epidemiologic paradigm. *Science* 1986;234:955-63.
4. Neequaye AR, Neequaye J, Mingle JA, Adjei DO. Preponderance of females with AIDS in Ghana [Letter]. *Lancet* 1986;2:978.
5. Van de Perre P, Clumeck N, Carael M, et al. Female prostitutes: a risk group for infection with human T-cell lymphotropic virus type III. *Lancet* 1985;2:524-7.
6. Kreiss JK, Koeh D, Plummer FA, et al. AIDS virus infection in Nairobi prostitutes. *N Engl J Med* 1986;314:414-8.
7. Mann J, Quinn TC, Piot P, et al. Condom use and HIV infection among prostitutes in Zaire [Letter]. *N Engl J Med* 1987;316:345.
8. CDC. Update: acquired immunodeficiency syndrome—Europe. *MMWR* 1986;35:35-8,43-6.
9. Barton SE, Underhill GS, Gilchrist C, Jeffries DJ, Harris JRW. HTLV-III antibody in prostitutes [Letter]. *Lancet* 1985;2:1424.
10. Brenky-Faudeux D, Fribourg-Blanc A. HTLV-III antibody in prostitutes [Letter]. *Lancet* 1985;2:1424.
11. Smith GL, Smith KF. Lack of HIV infection and condom use in licensed prostitutes [Letter]. *Lancet* 1986;2:1392.
12. Tirelli U, Vaccher E, Carbone A, DePaoli P, Santini G, Monfardini S. HTLV-III antibody in prostitutes [Letter]. *Lancet* 1985;2:1424.
13. Luthy R, Ledergerber B, Tauber M, Siegenthaler W. Prevalence of HIV antibodies among prostitutes in Zurich, Switzerland. *Klinische Wochenschrift* (in press).
14. Schultz S, Milberg JA, Kristal AR, Stoneburner RL. Female-to-male transmission of HTLV-III [Letter]. *JAMA* 1986;255:1703-4.
15. Papaevangelou G, Roumeliotou-Karayannis A, Kallinkos G, Papoutsakis G. LAV/HTLV-III infection in female prostitutes [Letter]. *Lancet* 1985;2:1018.
16. CDC. Acquired immunodeficiency syndrome (AIDS) among blacks and Hispanics—United States. *MMWR* 1986;35:655-8,663-6.
17. CDC. Update: acquired immunodeficiency syndrome—United States. *MMWR* 1986;35:757-60,765-6.
18. Lifson AR, Castro KG, Narkunas JP, Lekatsas A, Ksell TE, Fox RS. "No identified risk" cases of acquired immunodeficiency syndrome. Paris: Second International Conference on AIDS, 1986.

19. Conant M, Hardy D, Sernatinger J, Spicer D, Levy JA. Condoms prevent transmission of AIDS-associated retrovirus [Letter]. JAMA 1986;255:1706.
20. CDC. Update: acquired immunodeficiency syndrome (AIDS)—United States. MMWR 1984;33:661-4.
21. CDC. Recommendations for assisting in the prevention of perinatal transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus and acquired immunodeficiency syndrome. MMWR 1985;34:721-6,731-2.
22. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.
23. Benjamin H, Masters REL. Prostitution and morality. New York: Julian, 1964.
24. Darrow WW. Prostitution and sexually transmitted diseases. In: Holmes KK, Mardh P, Sparling PF, Wiesner PF, eds. Sexually transmitted diseases. New York: McGraw-Hill, 1984:109-16.
25. Yondorf B. Prostitution as a legal activity: the West German experience. Policy Analysis 1979; 5:417-33.
26. Bureau of the Census. 1980 census of population. Washington, DC: US Department of Commerce, 1982; (volume 1, characteristics of the population; chapter B, general population characteristics, table 31; series PC 80-1).

Self-Reported Changes in Sexual Behaviors Among Homosexual and Bisexual Men from the San Francisco City Clinic Cohort

From January 1978 through April 1980, approximately 6,700 homosexual and bisexual men attending a clinic for sexually transmitted diseases in San Francisco were enrolled in studies of the prevalence and incidence of hepatitis B virus infection (1). Approximately 1,300 participants answered standardized questions regarding their sexual practices. From December 1983 through December 1985, a random sample from this study group was asked to participate in studies of the acquired immunodeficiency syndrome (AIDS) by providing further information about their sexual behaviors (2,3). Study results show that homosexual and bisexual men in San Francisco have considerably reduced both their number of nonsteady sexual partners and their participation in specific sexual practices associated with increased risk of human immunodeficiency virus (HIV) infection, especially receptive anal intercourse.

Questionnaires administered to a subset of 126 members of this random sample in 1978, 1984, and 1985 provided data on their number of steady and nonsteady male partners in the 4 months preceding each interview. The numbers of steady partners (individuals with whom the participant had had sexual contact on three or more occasions during the 4-month period) rose from a mean of 1.6 per person in 1978 to 2.5 per person in 1984, then decreased to 1.5 in 1985. Numbers of nonsteady partners (defined as individuals with whom the participant had had sexual contact only once or twice) decreased from a median of 16 per person (mean = 29.3) during the 4-month period in 1978 to 3 (mean = 14.5) in 1984. By 1985 the median was 1 (mean = 5.5).

Participants also reported the percentage of time in the preceding 4 months that their sexual contacts with male partners included penetration or exchange of body fluids. To estimate a risk index of sexual activities that may have resulted in exposure to HIV in the previous 4 months, the percentage of time the participant engaged in each of several types of sexual behaviors was multiplied by the number of steady and nonsteady male partners during the same period.

The risk index for receptive anal intercourse with nonsteady partners decreased 90% between the two interview periods in 1978 and 1985. The risk index for receptive anal intercourse with a steady partner remained close to zero for each of the three 4-month periods in 1978, 1984, and 1985.

Although the risk index for receptive orogenital contact with nonsteady partners declined by 68% from 1978 to 1985, the decrease was not as striking as the decline in receptive anal intercourse. The risk index for receptive orogenital contact with steady partners remained low and relatively constant during this 7-year period.

Indices of exposure risk for insertive sexual contacts were also estimated. The risk index for insertive anal intercourse with nonsteady partners decreased 93% from 1978 to 1985, while the risk index for insertive orogenital contact with nonsteady partners declined 83% during the same period. Exposure risk for both insertive anal and orogenital contact with steady partners remained low and relatively constant between 1978 and 1985.

Information on condom use among these 126 men is unavailable; however, data collected during a pilot study in 1983 suggested that >95% of the men in the cohort did not use condoms during anal intercourse at that time (CDC, unpublished data). Preliminary data collected since November 1986 on a group of 104 cohort members indicate that approximately 33% of this group had anal intercourse at least once in the previous 4 months without using a condom (CDC, unpublished data). The majority (73%) of these unprotected sexual contacts were with steady partners.

Reported by: D Werdegart, MD, P O'Malley, T Bodecker, N Hessel, D Echenberg, MD, PhD, San Francisco Dept of Public Health. AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: Examination of trends in self-reported behavioral change provides an opportunity to indirectly evaluate educational efforts aimed at reducing high-risk behaviors. Within the time frame of this study (1983-1985), the Public Health Service recommended that members of high risk groups reduce their number of partners and avoid sexual contact with anyone known or suspected of having AIDS (4). In addition, the San Francisco Department of Public Health, in cooperation with the San Francisco AIDS Foundation, has implemented an extensive risk reduction program aimed at reducing high-risk sexual behavior in homosexual and bisexual men during this time period (5). Participants from this and other studies report significant reductions in certain high-risk behaviors (6-8). Ninety percent of the sample from this study reduced their number of nonsteady partners. The median number of partners de-

clined from 16 in 1978 to 1 in 1985. Thirty-four percent of the men reported having only one or no partners during the preceding 4 months in 1985.

However, in 1985, some of the men in this survey still reported having sexual contact with multiple partners or engaging in high-risk behaviors. The results from this study suggest that the major source of exposure to HIV in 1978, 1984, and 1985 may have been unprotected sexual contacts with nonsteady partners. However, unless steady partners are known to be seronegative for HIV infection, the potential for exposure through sexual contact with steady partners cannot be discounted either. Because of the high prevalence of HIV infection in homosexual men (9), the Public Health Service recommendations presently state that high-risk individuals should abstain or limit their sexual contact to one steady partner. Furthermore, those at risk should protect themselves during sexual activity with any possibly infected person by taking precautions against contact with the person's blood, semen, urine, feces, saliva, or cervical or vaginal secretions (10).

Although homosexual and bisexual men in San Francisco are generally aware of the guidelines for avoiding transmission of HIV, there is, for some men, a discrepancy between their knowledge of these guidelines and their behavior (6,7). These individuals need to be studied more intensively so that educational programs appropriate for this subgroup may be developed. Additional study of those who have already changed their behavior may also be helpful in identifying key factors motivating reductions in high-risk sexual behaviors.

References

1. Schreeder MT, Thompson SD, Hadler SC, et al. Hepatitis B in homosexual men: prevalence of infection and factors related to transmission. *J Infect Dis* 1982;146:7-15.
2. Jaffe HW, Darrow WW, Echenberg DF, et al. The acquired immunodeficiency syndrome in a cohort of homosexual men: a six-year follow-up study. *Ann Intern Med* 1985;103:210-4.
3. Darrow WW, Echenberg DF, Jaffe HW, et al. Risk factors for HIV infections in homosexual men. *Am J Public Health* 1987;77:479-83.
4. CDC. Prevention of acquired immune deficiency syndrome (AIDS): report of inter-agency recommendations. *MMWR* 1983;32:101-3.
5. San Francisco Department of Public Health. AIDS in San Francisco: status report and plan for 1987-88. San Francisco: San Francisco Department of Public Health, 1987.
6. Research and Decisions Corporation. Designing an effective AIDS prevention campaign strategy for San Francisco: results from the third probability sample of an urban gay male community. San Francisco: Research and Decisions Corporation, 1986.
7. McKusick L, Wiley JA, Coates TJ, et al. Reported changes in the sexual behavior of men at risk for AIDS, San Francisco, 1982-84—the AIDS Behavioral Research Project. *Pub Health Rep* 1985; 100:622-9.
8. CDC. Self-reported behavioral change among gay and bisexual men—San Francisco. *MMWR* 1985;34:613-5.
9. Winkelstein W Jr, Lyman DM, Padian N, et al. Sexual practices and risk of infection by the human immunodeficiency virus: the San Francisco men's health study. *JAMA* 1987;257:321-5.
10. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR* 1986;35:152-5.

Classification System for Human Immunodeficiency Virus (HIV) Infection in Children Under 13 Years of Age

INTRODUCTION

With the identification of the causative agent of the acquired immunodeficiency syndrome (AIDS), a broad spectrum of clinical manifestations has been attributed to infection with the human immunodeficiency virus (HIV). With the exception of the CDC surveillance definition for AIDS (1,2), no standard definitions for other manifestations of HIV infection have been developed for children. Classification systems published to date have been developed primarily to categorize clinical presentations in adult patients and may not be entirely applicable to infants and children (3-5).

Physicians from institutions caring for relatively large numbers of HIV-infected children report that only about half of their patients with symptomatic illness related to the infection fulfill the criteria of the CDC surveillance definition for AIDS (6, 7).

To develop a classification system for HIV infection in children, CDC convened a panel of consultants* consisting of clinicians experienced in the diagnosis and management of children with HIV infection; public health physicians; representatives from the American Academy of Pediatrics, the Council of State and Territorial Epidemiologists, the Association for Maternal Child Health and Crippled Children's Programs, the National Institute on Drug Abuse/Alcohol, Drug Abuse and Mental Health Administration, the National Institute of Allergy and Infectious Diseases/National Institutes of Health, and the Division of Maternal and Child Health/Health Resources and Services Administration; and CDC.

GOALS AND OBJECTIVES OF THE CLASSIFICATION SYSTEM

The system was designed primarily for public health purposes, including epidemiologic studies, disease surveillance, prevention programs, and health-care planning and policy. The panel attempted to devise a simple scheme that could be subdivided as needed for different purposes.

DEFINITION OF HIV INFECTION IN CHILDREN (Table 1)

Ideally, HIV infection in children is identified by the presence of the virus in blood or tissues, confirmed by culture or other laboratory detection methods. However, current tests—including culture—for detecting the virus or its antigens are not standardized and are not readily available. Detection of specific antibody to the virus is a sensitive and specific indicator of HIV infection in adults, since the majority of adults with antibody have had culture evidence of infection (8-10). Similar studies involving children have not been reported. Also, the presence of passively transferred maternal antibody in infants limits the interpretation of a positive antibody test result in this age group. Most of the consultants believed that passively transferred maternal HIV antibody could sometimes persist for up to 15 months. For this reason, two definitions for infection in children are needed: one for infants and children up to 15 months of age who have been exposed to their infected mothers perinatally, and another for older children with perinatal infection and for infants and children of all ages acquiring the virus through other means.

Infants and children under 15 months of age with perinatal infection—Infection in infants and children up to 15 months of age who were exposed to infected mothers in the perinatal period may be defined by one or more of the following: 1) the identification of the virus in blood or tissues, 2) the presence of HIV antibody as indicated by a repeatedly reactive screening test (e.g., enzyme immunoassay) plus a positive confirmatory test (e.g., Western blot, immunofluorescence assay) in an infant or child who has abnormal immunologic test results indicating both humoral and cellular immunodeficiency (increased immunoglobulin levels, depressed T4 [T-helper] absolute cell count, absolute lymphopenia, decreased T4/T8 ratio) and who meets the requirements of one or more of the subclasses listed under class P-2 (described below), or 3) the confirmation that a child's symptoms meet the previously published CDC case definition for pediatric AIDS (1,2).

*P Brunell, MD, R Daum, MD, American Academy of Pediatrics; J Chin, MD, State Epidemiologist, California Dept of Health Svcs; L Cooper, MD, St Luke's-Roosevelt Hospital Center, New York City; J Oleske, MD, MPH, L Epstein, MD, Univ of Medicine and Dentistry of New Jersey; N Luban, MD, Children's Hospital National Medical Center, Washington, DC; S Mailloux, MD, Assoc of Maternal Child Health and Crippled Children's Programs; S Pawha, MD, North Shore Univ Hospital, Cornell University Medical Center, Manhasset, NY; G Scott, MD, Univ of Miami School of Medicine; R Stiehm, MD, Univ of California, Los Angeles; P Thomas, MD, New York City Dept of Health; D Wara, MD, Univ of California, San Francisco; D Williams, MD, Los Angeles County Hospital; J Witte, MD, MPH, Florida Dept of Health and Rehabilitative Svcs.

The infection status of other perinatally exposed seropositive infants and children up to 15 months of age who lack one of the above immunologic or clinical criteria is indeterminate. These infants should be followed up for HIV-related illness, and they should be tested at regular intervals for persistence of antibody to HIV. Infants and children who become seronegative, are virus-culture negative (if blood or tissue samples are cultured), and continue to have no clinical or laboratory-confirmed abnormalities associated with HIV infection are unlikely to be infected.

Older children with perinatal infection and children with HIV infection acquired through other modes of transmission—HIV infection in these children is defined by one or more of the following: 1) the identification of virus in blood or tissues, 2) the presence of HIV antibody (positive screening test plus confirmatory test) regardless of whether immunologic abnormalities or signs or symptoms are present, or 3) the confirmation that the child's symptoms meet the previously published CDC case definition for pediatric AIDS (1,2).

These definitions apply to children under 13 years of age. Persons 13 years of age and older should be classified according to the adult classification system (3).

CLASSIFICATION SYSTEM (Table 2)

Children fulfilling the definition of HIV infection discussed above may be classified into one of two mutually exclusive classes based on the presence or absence of clinical signs and symptoms (Table 2). Class Pediatric-1 (P-1) is further subcategorized on the basis of the presence or absence of immunologic abnormalities, whereas Class P-2 is subdivided by specific disease patterns. Once a child has signs and symptoms and is therefore classified in P-2, he or she should not be reassigned to class P-1 if signs and symptoms resolve.

Perinatally exposed infants and children whose infection status is indeterminate are classified into class P-0.

Class P-0. Indeterminate infection. Includes perinatally exposed infants and children up to 15 months of age who cannot be classified as definitely infected according to the above definition but who have antibody to HIV, indicating exposure to a mother who is infected.

Class P-1. Asymptomatic infection. Includes patients who meet one of the above definitions for HIV infection but who have had no previous signs or symptoms that would have led to classification in Class P-2.

These children may be subclassified on the basis of immunologic testing. This testing should include quantitative immunoglobulins, complete blood count with differential, and T-lymphocyte subset quantitation. Results of functional testing of lymphocytes (mitogens, such as pokeweed) may also be abnormal in HIV-infected children, but it is less specific in comparison with immunoglobulin levels and lymphocyte subset analysis, and it may be impractical.

Subclass A - Normal immune function. Includes children with no immune abnormalities associated with HIV infection.

Subclass B - Abnormal immune function. Includes children with one or more of the commonly observed immune abnormalities associated with HIV infection, such as hypergammaglobulinemia, T-helper (T4) lymphopenia, decreased T-helper/T-suppressor (T4/T8) ratio, and absolute lymphopenia. Other causes of these abnormalities must be excluded.

Subclass C - Not tested. Includes children for whom no or incomplete (see above) immunologic testing has been done.

TABLE 1. Summary of the definition of HIV infection in children

Infants and children under 15 months of age with perinatal infection

- 1) Virus in blood or tissues
or
- 2) HIV antibody
and
evidence of both cellular and humoral immune deficiency
and
one or more categories in Class P-2
or
- 3) Symptoms meeting CDC case definition for AIDS

Older children with perinatal infection and children with HIV infection acquired through other modes of transmission

- 1) Virus in blood or tissues
or
- 2) HIV antibody
or
- 3) Symptoms meeting CDC case definition for AIDS

Class P-2. Symptomatic infection. Includes patients meeting the above definitions for HIV infection and having signs and symptoms of infection. Other causes of these signs and symptoms should be excluded. Subclasses are defined based on the type of signs and symptoms that are present. Patients may be classified in more than one subclass.

Subclass A - Nonspecific findings. Includes children with two or more unexplained nonspecific findings persisting for more than 2 months, including fever, failure-to-thrive or weight loss of more than 10% of baseline, hepatomegaly, splenomegaly, generalized lymphadenopathy (lymph nodes measuring at least 0.5 cm present in two or more sites, with bilateral lymph nodes counting as one site), parotitis, and diarrhea (three or more loose stools per day) that is either persistent or recurrent (defined as two or more episodes of diarrhea accompanied by dehydration within a 2-month period).

Subclass B - Progressive neurologic disease. Includes children with one or more of the following progressive findings: 1) loss of developmental milestones or intellectual ability, 2) impaired brain growth (acquired microcephaly and/or brain atrophy demonstrated on computerized tomographic scan or magnetic resonance imaging scan), or 3) progressive symmetrical motor deficits manifested by two or more of these findings: paresis, abnormal tone, pathologic reflexes, ataxia, or gait disturbance.

Subclass C - Lymphoid interstitial pneumonitis. Includes children with a histologically confirmed pneumonitis characterized by diffuse interstitial and peribronchiolar infiltration of lymphocytes and plasma cells and without identifiable pathogens, or, in the absence of a histologic diagnosis, a chronic pneumonitis—characterized by bilateral reticulonodular interstitial infiltrates with or without hilar lymphadenopathy—present on chest X-ray for a period of at least 2 months and unresponsive to appropriate antimicrobial therapy. Other causes of interstitial infiltrates should be excluded, such as tuberculosis, *Pneumocystis carinii* pneumonia, cytomegalovirus infection, or other viral or parasitic infections.

Subclass D - Secondary infectious diseases. Includes children with a diagnosis of an infectious disease that occurs as a result of immune deficiency caused by infection with HIV.

Category D-1. Includes patients with secondary infectious disease due to one of the specified infectious diseases listed in the CDC surveillance definition for AIDS: *Pneumocystis carinii* pneumonia; chronic cryptosporidiosis; disseminated toxoplasmosis with onset after 1 month of age; extra-intestinal strongyloidiasis; chronic isosporiasis; candidiasis (esophageal, bronchial, or pulmonary); extrapulmonary cryptococcosis; disseminated histoplasmosis; noncutaneous, extrapulmonary, or disseminated mycobacterial infection (any species other than *leprae*); cytomegalovirus infection with onset after 1 month of age; chronic mucocutaneous or disseminated herpes simplex virus infection with onset after 1 month of age; extrapulmonary or disseminated coccidioidomycosis; nocardiosis; and progressive multifocal leukoencephalopathy.

Category D-2. Includes patients with unexplained, recurrent, serious bacterial infections (two or more within a 2-year period) including sepsis, meningitis, pneumonia, abscess of an internal organ, and bone/joint infections.

TABLE 2. Summary of the classification of HIV infection in children under 13 years of age

Class P-0. Indeterminate infection

Class P-1. Asymptomatic infection

- Subclass A. Normal immune function
- Subclass B. Abnormal immune function
- Subclass C. Immune function not tested

Class P-2. Symptomatic infection

- Subclass A. Nonspecific findings
- Subclass B. Progressive neurologic disease
- Subclass C. Lymphoid interstitial pneumonitis
- Subclass D. Secondary infectious diseases
 - Category D-1. Specified secondary infectious diseases listed in the CDC surveillance definition for AIDS
 - Category D-2. Recurrent serious bacterial infections
 - Category D-3. Other specified secondary infectious diseases
- Subclass E. Secondary cancers
 - Category E-1. Specified secondary cancers listed in the CDC surveillance definition for AIDS
 - Category E-2. Other cancers possibly secondary to HIV infection
- Subclass F. Other diseases possibly due to HIV infection

Category D-3. Includes patients with other infectious diseases, including oral candidiasis persisting for 2 months or more, two or more episodes of herpes stomatitis within a year, or multidermatomal or disseminated herpes zoster infection.

Subclass E - Secondary cancers. Includes children with any cancer described below in categories E-1 and E-2.

Category E-1. Includes patients with the diagnosis of one or more kinds of cancer known to be associated with HIV infection as listed in the surveillance definition of AIDS and indicative of a defect in cell-mediated immunity: Kaposi's sarcoma, B-cell non-Hodgkin's lymphoma, or primary lymphoma of the brain.

Category E-2. Includes patients with the diagnosis of other malignancies possibly associated with HIV infection.

Subclass F - Other diseases. Includes children with other conditions possibly due to HIV infection not listed in the above subclasses, such as hepatitis, cardiopathy, nephropathy, hematologic disorders (anemia, thrombocytopenia), and dermatologic diseases.

Reported by: AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: This classification system is based on present knowledge and understanding of pediatric HIV infection and may need to be revised as new information becomes available. New diagnostic tests, particularly antigen detection tests and HIV-specific IgM tests, may lead to a better definition of HIV infection in infants and children. Information from several natural history studies currently under way may necessitate changes in the subclasses based on clinical signs and symptoms.

A definitive diagnosis of HIV infection in perinatally exposed infants and children under 15 months of age can be difficult. The infection status of these HIV-seropositive infants and children who are asymptomatic without immune abnormalities cannot be determined unless virus culture or other antigen-detection tests are positive. Negative virus cultures do not necessarily mean the child is not infected, since the sensitivity of the culture may be low. Decreasing antibody titers have been helpful in diagnosing other perinatal infections, such as toxoplasmosis and cytomegalovirus. However, the pattern of HIV-antibody production in infants is not well defined. At present, close follow-up of these children (Class P-0) for signs and symptoms indicative of HIV infection and/or persistence of HIV antibody is recommended.

The parents of children with HIV infection should be evaluated for HIV infection, particularly the mother. The child is often the first person in such families to become symptomatic. When HIV infection in a child is suspected, a careful history should be taken to elicit possible risk factors for the parents and the child. Appropriate laboratory tests, including HIV serology, should be offered. If the mother is seropositive, other children should be evaluated regarding their risk of perinatally acquired infection. Intrafamilial transmission, other than perinatal or sexual, is extremely unlikely. Identification of other infected family members allows for appropriate medical care and prevention of transmission to sexual partners and future children (11,12).

The nonspecific term AIDS-related complex has been widely used to describe symptomatic HIV-infected children who do not meet the CDC case definition for AIDS. This classification system categorizes these children more specifically under Class P-2.

The development and publication of this classification system does not imply any immediate change in the definition of pediatric AIDS used by CDC for reporting purposes (1,2). Changes in this definition require approval by state and local health departments. However, changes in the definition for reporting cases have been proposed by CDC and are awaiting state and local approval.

Written comments are encouraged. They should be mailed to the AIDS Program, Center for Infectious Diseases, Centers for Disease Control, Atlanta, GA 30333.

References

- 1.CDC. Update: acquired immunodeficiency syndrome (AIDS)—United States. MMWR 1984;32:688-91.
- 2.CDC. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. MMWR 1985;34:373-5.
- 3.CDC. Classification system for human T-lymphotropic virus type III/lymphadenopathy-associated virus infections. MMWR 1986;35:334-9.
- 4.Redfield RR, Wright DC, Tramont EC. The Walter Reed staging classification for HTLV-III/LAV infection. N Engl J Med 1986;314:131-2.
- 5.Haverkos HW, Gottlieb MS, Killen JY, Edelman R. Classification of HTLV-III/LAV-related diseases [Letter]. J Infect Dis 1985;152:1095.
- 6.Pawha S, Kaplan M, Fikrig S, et al. Spectrum of human T-cell lymphotropic virus type III infection in children. JAMA 1986;255:2299-305.
- 7.Scott GB, Anisman L, Zaldivar ML, Parks WP. Natural history of HTLV-III/LAV infections in children. Presented at the International Conference on AIDS, Paris, June 1986.

8. Ward JW, Grindon AJ, Feorino PM, Schable C, Parvin M, Allen JR. Laboratory and epidemiologic evaluation of an enzyme immunoassay for antibodies to HTLV-III. JAMA 1986;256:357-61.
9. Peterman TA, Jaffe HW, Feorino PM, et al. Transfusion-associated acquired immunodeficiency syndrome in the United States. JAMA 1985;254:2913-7.
10. Jaffe HW, Feorino PM, Darrow WW, et al. Persistent infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in apparently healthy homosexual men. Ann Intern Med 1985;102:627-8.
11. CDC. Recommendations for assisting in the prevention of perinatal transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus and acquired immunodeficiency syndrome. MMWR 1985;34:721-6,731-2.
12. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.

Trends in Human Immunodeficiency Virus Infection Among Civilian Applicants for Military Service — United States, October 1985-December 1986

Since October 1985, the U.S. Department of Defense has routinely tested civilian applicants for serologic evidence of infection with human immunodeficiency virus (HIV) as part of their preinduction medical evaluation (1). Results from the first 6 months of testing have been reported previously (2,3). Results for the first 15 months provide the opportunity to observe trends of infection in this population.

Between October 1985 and December 1986, 789,578 civilian applicants for military service were screened. Of these, 1,186 were confirmed as HIV-antibody positive by enzyme immunoassay and Western blot immunoelectrophoresis, for an overall rate of 1.5/1,000 individuals tested. Seroprevalence per 1,000 varied by age, sex, race and ethnicity, and region of residence. By age, it was 0.6 for 17-20 year-olds, 2.5 for 21-25 year-olds, and 4.1 for those ≥ 26 years of age. By sex, it was 1.6 for males and 0.6 for females. By race and ethnicity, seroprevalence per 1,000 was 0.8 for whites, 4.1 for blacks, 2.3 for Hispanics, 1.0 for American Indians or Alaskan Natives and Asian or Pacific Islanders. Table 1 shows the seroprevalence among civilian applicants by region of residence.

During the 15-month observation period, the seroprevalence did not change significantly, either in the aggregate or when analyzed by age, sex, race and ethnicity (Figure 1), or geographic region. However, seroprevalence among white males showed a small but significant decline of 0.02/1,000 applicants tested per month ($p = 0.016$ by Chi Square test for trends in proportions using a logistic regression linear model).

Reported by: Health Studies Task Force, Office of the Assistant Secretary of Defense (Health Affairs), US Dept of Defense, Washington, DC. Div of Preventive Medicine and Div of Communicable Diseases and Immunology, Walter Reed Army Institute of Research, Washington, DC. Surveillance and Evaluation Br, AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: AIDS cases reported to CDC continue to increase*. However, because of the lengthy incubation period of AIDS (4), these cases represent infection occurring at least several years prior to the report of disease. There has been little information to indicate current trends in HIV infection. Analysis of the results of the testing of civilian applicants thus far basically shows neither an increase nor a decrease in infection level for the group as a whole or for individual subgroups. The significance of this apparent absence of change in antibody prevalence during the 15-month period studied is not yet clear.

Volunteers for military service, who are verbally screened by the recruiting official prior to arrival at the medical evaluation center, are not fully representative of the overall population in that they underrepresent the three groups in the United States with the highest prevalence of HIV infection†. Moreover, applicants do not equally represent all socioeconomic and demographic groups in the population. A growing awareness of the military serologic screen-

*An average of 38.3 AIDS cases per day were reported for the period October-December 1986, compared with an average of 26.3 per day for the period October-December 1985.

†Active intravenous drug abusers, homosexual men, and hemophiliacs.

TABLE 1. Prevalence of HIV antibody* among civilian applicants for military service, by age group and region of residence — October 1985-December 1986

Region†	Age Group (Years)			All Ages
	17-20	21-25	≥ 26	
New England	0.4	1.0	3.8	0.9
Middle Atlantic	0.7	4.6	10.0	2.9
EN Central	0.4	1.8	1.9	0.9
WN Central	0.2	1.0	1.8	0.6
South Atlantic	0.9	3.4	5.4	2.1
ES Central	0.4	1.9	1.3	0.9
WS Central	0.6	2.7	3.0	1.6
Mountain	0.3	1.5	1.9	0.9
Pacific	0.8	1.5	4.0	1.5
US Territories	1.6	6.3	12.3	5.8
All Regions	0.6	2.5	4.1	1.5

*Repeatedly reactive enzyme-linked immunosorbent assay (ELISA) test confirmed by Western blot immunoelectrophoresis; reported as the number of antibody-positive applicants per 1,000 tested.

†Defined in notifiable diseases table (Table III).

ing program may have increased self-deferral by persons who are HIV-antibody positive or who feel they may have been exposed to the virus. If so, this could have masked an increased frequency of infection in the population from which the applicants are drawn.

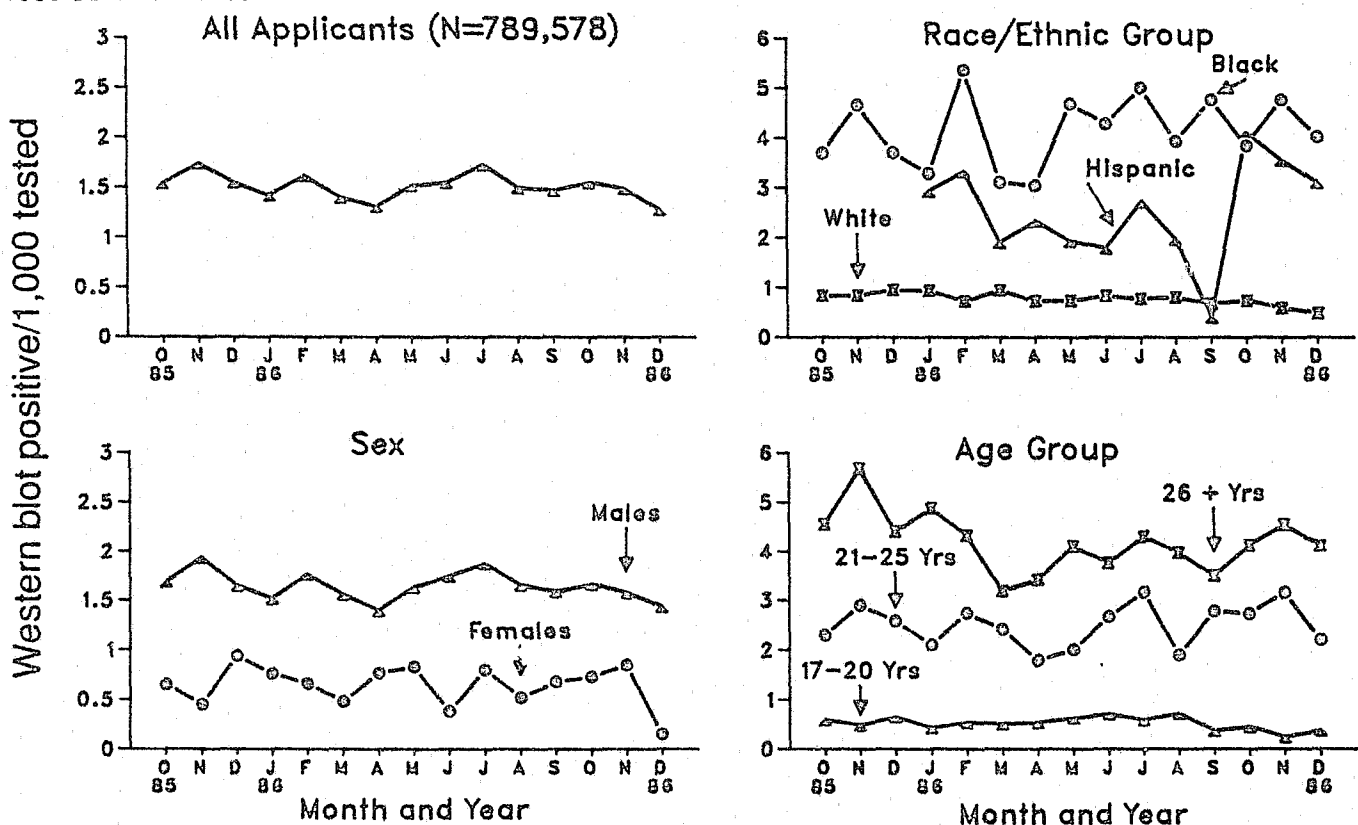
Monitoring trends in infection among civilian applicants for military service as well as among blood donors⁵ remains important. It is also critical to compare trends in infection among these volunteer groups with similar trends among groups not affected by self-selection bias. One such surveillance approach, in which anonymously tested sample populations without AIDS-like disease are monitored at participating hospitals, has been initiated recently by CDC. Trends in exposure risks among seropositive individuals should also be monitored to assess possible changes in the relative frequency of the various modes of transmission. Follow-up interviews of a small number of seropositive applicants have found a high proportion with typical risk exposures for AIDS (5). CDC is collaborating with the U.S. Department of Defense, the National Cancer Institute of the National Institutes of Health, and certain state and local health departments to develop a systematic follow-up evaluation of seropositive civilian applicants in selected cities and states.

References

1. Herbold JR. AIDS policy development within the Department of Defense. *Milit Med* 1986;151:623-7.
2. CDC. Human T-lymphotropic virus type III/lymphadenopathy-associated virus antibody prevalence in U.S. military recruit applicants. *MMWR* 1986;35:421-4.
3. Burke DS, Brundage JF, Bernier W, et al. Demography of HIV infections among civilian applicants for military service in four counties in New York City: a preliminary analysis. *New York State Med J* [In press].
4. Lui KJ, Lawrence DN, Morgan WM, Peterman TA, Haverkos HW, Bregman DJ. A model-based approach for estimating the mean incubation period of transfusion-associated acquired immunodeficiency syndrome. *Proc Natl Acad Sci* 1986;83:3051-5.
5. Stoneburner RL, Chiasson MA, Solomon K, Rosenthal S. Risk factors in military recruits positive for HIV antibody [Letter]. *N Engl J Med* 1986;315:1355.

⁵Long-term data are not yet available for this group.

FIGURE 1. Human immunodeficiency virus antibody among civilian applicants*, by month — United States, October 1985-December 1986



*U.S. Department of Defense data.

Update : Human Immunodeficiency Virus Infections in Health-Care Workers Exposed to Blood of Infected Patients

Six persons who provided health care to patients with human immunodeficiency virus (HIV) infection and who denied other risk factors have previously been reported to have HIV infection. Four of these cases followed needle-stick exposures to blood from patients infected with HIV (1-4). The two additional cases involved persons who provided nursing care to persons with HIV infection. Although neither of these two persons sustained needle-stick injuries, both had extensive contact with blood or body fluids of the infected patient, and neither observed routinely recommended barrier precautions (5,6).

CDC has received reports of HIV infection in three additional health-care workers following non-needle-stick exposures to blood from infected patients. The exposures occurred during 1986 in three different geographic areas. Although these three cases represent rare events, they reemphasize the need for health-care workers to adhere rigorously to existing infection control recommendations for minimizing the risk of exposure to blood and body fluids of all patients (7-9).

Health-Care Worker 1: A female health-care worker assisting with an unsuccessful attempt to insert an arterial catheter in a patient suffering a cardiac arrest in an emergency room applied pressure to the insertion site to stop the bleeding. During the procedure, she may have had a small amount of blood on her index finger for about 20 minutes before washing her hands. Afterwards, she may also have assisted in cleaning the room but did not recall any other exposures to the patient's blood or body fluids. She had no open wounds, but her hands were chapped. Although she often wore gloves when anticipating exposure to blood, she was not wearing gloves during this incident.

The patient with the cardiac arrest died. A postmortem examination identified *Pneumocystis carinii* pneumonia, and a blood sample was positive for HIV antibody by enzyme immunoassay (EIA) and Western blot methods. Twenty days after the incident, the health-care worker became ill with fever, myalgia, extreme fatigue, sore throat, nausea, vomiting, diarrhea, a 14-pound weight loss, and generalized lymphadenopathy which her physician diagnosed as a viral syndrome. That illness lasted 3 weeks. She felt much better 9 weeks after the incident, and, when she was examined 6 months after the incident, all signs and symptoms had resolved. She had donated blood 8 months before the incident and was negative for HIV antibody by EIA. She donated again 16 weeks after the incident and was positive for HIV by EIA and Western blot (bands p24 and gp41). Serum samples obtained 20 and 23 weeks after the incident were also positive for HIV antibody. She stated that for over 8 years her only sexual partner had been her husband, who denied risk factors for HIV and was seronegative for HIV antibody. She denied ever receiving a blood transfusion, ever using intravenous drugs, or having any needle sticks or other significant exposures to blood or body fluids in the past 8 years. Her serologic test for syphilis was negative. Fifteen other employees who assisted in the care of the patient were seronegative at least 4 months after the exposure.

Health-Care Worker 2: A female phlebotomist was filling a 10 ml vacuum blood collection tube with blood from an outpatient with a suspected HIV infection when the top of the tube flew off and blood splattered around the room, on her face, and in her mouth. She was wearing gloves to protect her hands and was wearing eyeglasses so she did not think she got any blood in her eyes. She had facial acne but no open wounds. She washed the blood off immediately after the exposure. The outpatient's blood sample was positive for HIV antibody by EIA and Western blot, and a hepatitis B surface antigen test was negative. The phlebotomist's EIA was negative the day after the incident and again 8 weeks later. When she donated blood 9 months after the exposure, she was positive for HIV antibody by EIA and Western blot (bands p24 and gp41). She has had no symptoms. She denied having any sexual contact during the previous 2 years, ever using drugs intravenously, or ever receiving a transfusion. Two months after the incident, she scratched the back of her hand with a needle used to draw blood from an intravenous drug abuser of unknown HIV-antibody status. She did not bleed as a result of the scratch and has not had any needle-stick injuries in over 2 years. Her serologic tests for syphilis and hepatitis B were negative. A coworker who was splattered with blood on the face and in the mouth during the same incident remains seronegative 1 year after the incident.

Health-Care Worker 3: A female medical technologist was manipulating an apheresis machine (a device to separate blood components) to correct a problem that developed during an outpatient procedure when blood spilled, covering most of her hands and forearms. She was not wearing gloves. She does not recall having any open wounds on her hands or any mucous-

membrane exposure. However, she had dermatitis on one ear and may have touched it. She washed the blood off herself and the machine several minutes after the spill. The patient undergoing the apheresis had denied risk factors for HIV infection. However, a blood sample from the patient was positive for HIV antibody by EIA and Western blot methods and negative for hepatitis B surface antigen the next day. The technologist's HIV-antibody tests were negative 5 days after the exposure and again 6 weeks later. Eight weeks after the exposure, she had an influenza-like illness with fever, myalgia, diarrhea, hives, and a pruritic red macular rash on her arms and legs. The illness resolved after a few weeks, and her physician thought the illness was probably a viral syndrome. Three months after the incident, she was positive for HIV antibody by EIA and Western blot methods (band p24 alone). Four months after the incident, a Western blot was positive (bands p24 and gp41). She indicated that for more than 8 years her only sexual partner had been her husband, who denied risk factors for HIV infection and was seronegative for HIV antibody. She denied ever receiving a transfusion, ever using intravenous drugs, or having any needle-stick injuries in over 2 years. Her serologic tests for syphilis and hepatitis B were negative. She has an immunologic disorder which had been treated with corticosteroids in the past, but she had not taken any immunosuppressive medication for the past year. A coworker with a similar exposure during the same procedure remains seronegative after 3 months.

Reported by: Hospital Infections Program and AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: Three instances of health-care workers with HIV infections associated with skin or mucous-membrane exposure to blood from HIV-infected patients are reported above. Careful investigation of these three cases did not identify other risk factors for HIV infection, although unrecognized or forgotten needle-stick exposures to other infected patients cannot be totally excluded. The exact route of transmission in these three cases is not known. Health-Care Worker 1 had chapped hands, and the duration of contact with the blood of the patient experiencing a cardiac arrest may have been as long as 20 minutes. Health-Care Worker 2 sustained contamination of oral mucous membranes. This individual also had acne but did not recall having open lesions. In addition, she had sustained a scratch from a needle used to draw blood from an intravenous drug abuser of unknown HIV-infection status. Health-Care Worker 3 had a history of dermatitis involving an ear. Health-Care Workers 1 and 3 were not wearing gloves when direct contact with blood occurred. Health-Care Worker 2 was wearing gloves, but blood contaminated her face and mouth.

Three ongoing prospective studies provide data on the magnitude of the risk of HIV infection incurred when health-care workers are exposed to blood of infected patients through needle-stick wounds or contamination of an open wound or mucous membrane. In a CDC cooperative surveillance project (10), a total of 1,097 health-care workers with parenteral or mucous-membrane exposure to the blood of patients with AIDS or other manifestations of HIV infection had been enrolled as of March 31, 1987. Needle-stick injuries and cuts with sharp objects accounted for 969 (89%) of the exposures to blood; 298 of these had paired serum samples tested for HIV antibody. One (0.3%) seroconverted (2), indicating that the risk of transmission during these exposures is very low. In addition, 70 health-care workers had open wounds exposed to blood, and 58 had mucous membrane exposed to blood. Postexposure serum samples from 82 of these 128 workers have been tested for antibody to HIV; none was seropositive.

In a study at the National Institutes of Health (11) through April 30, 1987, none of the 103 workers with percutaneous exposures and none of the 229 workers with mucous-membrane exposures to blood or body fluids of patients with AIDS was seropositive. At the University of California (12), none of 63 workers with open wounds or mucous membranes exposed to blood or body fluids of patients with AIDS was seropositive. Although the precise risk of transmission during exposures of open wounds or mucous membranes to contaminated blood cannot be defined, these studies indicate that it must be very low.

The three cases reported here suggest that exposure of skin or mucous membranes to contaminated blood may rarely result in transmission of HIV. The magnitude of the risk is not known since data on the frequency with which such exposures occur are not available. Skin and mucous-membrane exposures are thought to occur much more commonly than needle sticks, and the risk associated with skin or mucous-membrane exposures is likely to be far lower than that associated with needle-stick injuries. Nonetheless, the increasing prevalence of HIV infection increases the potential for such exposures, especially when routinely recommended precautions are not followed.

It is unlikely that routine serologic testing for HIV infection of all patients admitted to hospitals would have prevented these exposures since two of the three exposures occurred in the outpatient clinic setting, and one occurred during a resuscitation effort in an emergency room shortly after the arrival of the patient. At the time of exposure, Health-Care Worker 2 suspect-

ed that the source patient was infected with HIV, but Health-Care Workers 1 and 3 did not. The hospital where Health-Care Worker 3 was exposed has a protocol for apheresis which normally involves HIV-antibody testing of donors; however, such testing was not done in advance of the procedure. Previous CDC recommendations have emphasized the value of HIV serologic testing for patient diagnosis and management and for prevention and control of HIV transmission (13) and have stated that some hospitals in certain geographic areas may deem it appropriate to initiate serologic testing of patients (7). Such testing may also provide an opportunity to reduce the risk of HIV infection to health-care workers, but it has not been established that knowledge of a patient's serologic status increases the compliance of health-care workers with recommended precautions.

These cases emphasize again the need to implement and strictly enforce previously published recommendations for minimizing the risk of exposure to blood and body fluids of all patients in order to prevent transmission of HIV infection in the workplace and during invasive procedures (7-9).

1. As previously recommended, routine precautions must be followed when there is a possibility of exposure to blood or other body fluids. The anticipated exposure may require gloves alone (e.g., when placing an intravascular catheter or handling items soiled with blood or equipment contaminated with blood or other body fluids). Procedures involving more extensive contact with blood or potentially infective body fluids (e.g., some dental or endoscopic procedures or postmortem examinations) may require gloves, gowns, masks, and eye-coverings. Hands and other contaminated skin surfaces should be washed thoroughly and immediately if accidentally contaminated with blood (7). These precautions deserve particular emphasis in emergency care settings in which the risk of blood exposure is increased and the infectious status of the patient is usually unknown (14).
2. Previous recommendations have emphasized management of parenteral and mucous-membrane exposures of health-care workers*. In addition, health-care workers who are involved in incidents that result in cutaneous exposures involving large amounts of blood or prolonged contact with blood—especially when the exposed skin is chapped, abraded, or afflicted with dermatitis—should follow these same recommendations. Moreover, serologic testing should be available to all health-care workers who are concerned that they may have been infected with HIV.

References

1. Anonymous. Needlestick transmission of HTLV-III from a patient infected in Africa. *Lancet* 1984; 2:1376-7.
2. Stricof RL, Morse DL. HTLV-III/LAV seroconversion following a deep intramuscular needlestick injury [Letter]. *N Engl J Med* 1986;314:1115.
3. Oksenhendler E, Harzic M, Le Roux JM, Rabian C, Clauvel JP. HIV infection with seroconversion after a superficial needlestick injury to the finger. *N Engl J Med* 1986;315:582.
4. Neisson-Vernant C, Arfi S, Mathez D, Leibowitch J, Monplaisir N. Needlestick HIV seroconversion in a nurse. *Lancet* 1986;2:814.
5. Grint P, McEvoy M. Two associated cases of the acquired immune deficiency syndrome (AIDS). *PHLS Commun Dis Rep* 1985;42:4.
6. CDC. Apparent transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus from a child to a mother providing health care. *MMWR* 1986;35:76-9.
7. CDC. Summary: recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in the workplace. *MMWR* 1985; 34:681-5, 691-5.
8. CDC. Recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus during invasive procedures. *MMWR* 1986;35:221-3.
9. CDC. Recommended infection-control practices for dentistry. *MMWR* 1986;35:237-42.
10. McCray E, The Cooperative Needlestick Surveillance Group. Occupational risk of the acquired immunodeficiency syndrome among health-care workers. *N Engl J Med* 1986;314:1127-32.

*If a HCW [health-care worker] has a parenteral (e.g., needlestick or cut) or mucous membrane (e.g., splash to the eye or mouth) exposure to blood or other body fluids, the source patient should be assessed clinically and epidemiologically to determine the likelihood of HTLV-III/LAV [sic] infection. If the assessment suggests that infection may exist, the patient should be informed of the incident and requested to consent to serologic testing for evidence of HTLV-III/LAV [sic] infection. If the source patient has AIDS or other evidence of HTLV-III/LAV [sic] infection, declines testing, or has a positive test, the HCW should be evaluated clinically and serologically for evidence of HTLV-III/LAV [sic] infection as soon as possible after the exposure, and, if seronegative, retested after 6 weeks and on a periodic basis thereafter (e.g., 3, 6, and 12 months following exposure) to determine if transmission has occurred. During this follow-up period, especially the first 6-12 weeks, when most infected persons are expected to seroconvert, exposed HCWs should receive counseling about the risk of infection and follow U.S. Public Health Service (PHS) recommendations for preventing transmission of AIDS (15,16). If the source patient is seronegative and has no other evidence of HTLV-III/LAV [sic] infection, no further follow-up of the HCW is necessary. If the source patient cannot be identified, decisions regarding appropriate follow-up should be individualized based on the type of exposure and the likelihood that the source patient was infected (7).

11. Henderson DK, Saah AJ, Zak BJ, et al. Risk of nosocomial infection with human T-cell lymphotropic virus type III/lymphadenopathy-associated virus in a large cohort of intensively exposed health-care workers. *Ann Intern Med* 1986;104:644-7.
12. Gerberding JL, Bryant LeBlanc CE, Nelson K, et al. Risk of transmitting the human immunodeficiency virus, hepatitis B virus, and cytomegalovirus to health-care workers exposed to patients with AIDS and AIDS-related conditions. *J Infect Dis* 1987;156:1-8 (In press).
13. CDC. Recommended additional guidelines for HIV antibody counseling and testing in the prevention of HIV infection and AIDS. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, 1987.
14. Baker JL, Kelen GD, Sivertson KT, Quinn TC. Unsuspected human immunodeficiency virus in critically ill emergency patients. *JAMA* 1987;257:2609-11.
15. CDC. Prevention of acquired immunodeficiency syndrome (AIDS): report of inter-agency recommendations. *MMWR* 1983;32:101-3.
16. CDC. Provisional Public Health Service inter-agency recommendations for screening donated blood and plasma for antibody to the virus causing acquired immunodeficiency syndrome. *MMWR* 1985;34:1-5.

Human Immunodeficiency Virus Infection Transmitted From an Organ Donor Screened for HIV Antibody — North Carolina

In August 1986, a cadaveric organ donor was found positive for antibody to the human immunodeficiency virus (HIV) by both enzyme immunoassay (EIA) and Western blot methods after some of the donated organs had been transplanted. A blood sample, which was taken after the donor had received a large number of blood transfusions, had been negative for HIV antibody. Two days later, when the organs were removed, more blood samples were collected. These were forwarded with the donated organs to the various transplantation centers. At one of these centers, one of these later samples was found to be seropositive.

Three persons received organs from this donor. Two of them were subsequently found to be seropositive for HIV antibody. The third, who had received the donor's heart, did not survive the transplant procedure. This is the first report of HIV transmission by organ transplantation from a donor screened for HIV antibody. A summary of the investigation of the donor and the two surviving recipients follows.

Donor. A 30-year-old man who was involved in a motor vehicle accident was admitted, while in a coma, to a North Carolina hospital. He was hypotensive because of bleeding from multiple head and neck lacerations. On admission, a blood sample was collected for type- and cross-matching, and blood transfusions were started within 1 hour. The donor's bleeding persisted despite surgery to improve hemostasis. Approximately 11 hours after admission, he had received a total of 56 units of blood and blood components (1 unit of whole blood, 28 units of packed red blood cells, 7 units of fresh frozen plasma, and 20 units of platelets). At this time, another blood sample was collected and tested for HIV antibody. The specimen was negative by EIA (Abbott Laboratories, North Chicago, Illinois; optical density ratio, sample/control = .103/.131). The donor's condition did not improve, and he was declared brain-dead 2 days after testing for HIV antibody. Family members consented to organ donation and denied any knowledge of the donor's having a risk factor for HIV infection.

The donor's kidneys, heart, and liver were removed and transported to other medical centers for transplantation. Samples of the donor's blood, which were collected when the organs were removed, were sent with each organ. As part of one center's routine procedure, one of these blood samples was tested for HIV antibody and was found positive by EIA (Genetic Systems, Seattle, Washington; optical density ratio = .95/<.30) and was subsequently found positive by Western blot assay. The transplantation teams were notified of the test result, but the heart, liver, and one kidney had already been transplanted.

Personnel from the hospital where the organs had been removed were contacted. They located both the serum sample collected on admission and the serum sample previously found negative for HIV antibody. The serum collected at the time of admission, before any transfusions were administered, was highly reactive on the Abbott EIAs performed at the hospital (optical density ratios = .756/.126, .556/.126) and at the North Carolina State Laboratory of Public Health (optical density ratios = .842/.108, .698/.137) and was also positive by Western blot assay at the state laboratory. When testing was repeated, the serum collected after the blood transfusions was again seronegative by EIA at the hospital and by both EIA and Western blot methods at the state laboratory.

Recipient 1. A man with end-stage renal disease received the donated kidney that was transplanted. The recipient is married and denied risk factors for HIV infection. He was negative for HIV antibody 3 days after transplantation. A blood specimen collected 10 weeks after transplantation was positive for HIV antibody by EIA, and a specimen collected 1 week later was positive by both EIA and Western blot assay. The recipient had a fever 8 days after receiving the renal allograft, and a biopsy of it showed acute rejection. He improved with additional immunosuppressive therapy. To date, he has not developed any opportunistic illness and continues to feel well.

Recipient 2. A man with sclerosis of the biliary ducts and progressive liver failure received the donated liver. He is married and denied risk factors for HIV infection. He was tested 4 days after transplantation and was negative for HIV antibody. Twelve

weeks after the procedure, he was positive for HIV antibody by EIA, and a specimen collected 4 weeks later was positive by both the conventional EIA and an EIA using recombinant viral proteins (ENVACORE, Abbott Laboratories). Four months after transplantation, the recipient developed fever and malaise. A liver biopsy showed moderate allograft rejection. The recipient's condition improved with an adjustment in immunosuppressive therapy, and he returned home the following month.

Reported by: TW Lane, MD, Univ of North Carolina, Chapel Hill, and Moses H Cone Memorial Hospital, Greensboro; R Meriwether, MD, FV Crout, PhD, JN MacCormack, MD, MPH, State Epidemiologist, North Carolina Dept of Human Resources. L Makowka, MD, Univ of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania. SA Lobel, PhD, PA Bowen, MD, RJ Caruana, MD, Medical College of Georgia, Augusta, Georgia. AIDS Program, Center for Infectious Diseases, CDC.

Editorial Note: Previous reports have linked kidney-transplant recipients who have subsequently become HIV-seropositive with donors who were later found to have risks for HIV infection (1-4). However, this is the first report of transplantation-associated HIV transmission from a cadaveric organ donor screened for HIV antibody. This donor appears to have been false-negative for HIV antibody by EIA as a result of the large number of transfusions he received before serum was collected for testing.

The Public Health Service recommended in May 1985 that potential organ donors be screened for HIV antibody (5). In January 1986, CDC conducted an anonymous survey of representatives from 44 transplantation programs attending a meeting of the Southeastern Organ Procurement Foundation. All of the 26 representatives who responded reported that their centers screened donors for HIV antibody. Three of these representatives (12%) also reported identifying at least one potential organ donor who was positive for HIV antibody by EIA and Western blot methods.

Organs from donors who are HIV-seropositive should not be used for transplantation except in very unusual circumstances. If an urgent need requires considering transplantation of an organ from a seropositive donor, the potential recipient or the appropriate family members should be informed of the risks of acquiring HIV infection. Such transplantation should not take place without the consent of either the potential recipient or the appropriate family members. When donors have been transfused before their organs are removed, testing for HIV antibody should be conducted on serum collected at the time of admission rather than on serum obtained after multiple transfusions. If donor serum collected at the time of admission is not available from other sources, a pretransfusion sample may be available from the blood bank since many blood banks hold specimens collected for compatibility testing for at least 7 days (6).

References

1. Prompt CA, Reis MM, Grillo FM, et al. Transmission of AIDS virus at renal transplantation [Letter]. *Lancet* 1985;2:672.
2. L'age-Stehr J, Schwarz A, Offermann G, et al. HTLV-III infection in kidney transplant recipients [Letter]. *Lancet* 1985;2:1361-2.
3. Margreiter R, Fuchs D, Hansen A, et al. HIV infection in renal allograft recipients. *Lancet* 1986;2:398.
4. Kumar P, Pearson JE, Martin DH, et al. Transmission of human immunodeficiency virus by transplantation of a renal allograft, with development of the acquired immunodeficiency syndrome. *Ann Intern Med* 1987;106:244-5.
5. CDC. Testing donors of organs, tissues, and semen for antibody to human T-lymphotropic virus type III/lymphadenopathy-associated virus. *MMWR* 1985;34:294.
6. American Association of Blood Banks. Standards for blood banks and transfusion services. 12th ed. Washington, DC: American Association of Blood Banks, 1987:31.

Index

- aerosol 11
- arboviruses 24, 25
- arthropods 24, 25
- biosafety 11-13
- blacks 26-30
- blood 11
- blood transfusion 3, 4, 18
- chemotherapy 8
- children 18-21, 28
- clofazimine 9
- coagulation disorders 32
- condoms 35
- Colorado 3, 4
- diagnosis 8
- dialysis 1
- disinfection 12, 13
- donor 3
- enzyme immunoassay 2, 5
- ethambutol 8
- exposure 11
- Florida 15, 23-25
- gonorrhea 34
- H9 lymphoid cell lines 2
- Haitians 26
- heat treatment 13
- hemodialysis 1
- hemophilia 32, 33
- hepatitis B virus 1, 11, 13
- heterosexual transmission 34
- Hispanics 26-30
- immunization 18-21
- infection control 1, 2, 11-13
- insects 24
- isoniazid 8
- Kaposi's sarcoma 36
- laboratory precautions 12
- Mantoux test 9
- military 5, 6
- Minnesota 34, 35
- mycobacterial infection 8
- Mycobacterium avium* 8
- Mycobacterium kansasii* 9
- Mycobacterium tuberculosis* 8, 9
- needlestick 11
- parenteral exposure 11
- patient groups 15, 23, 24, 28-30, 37
- pediatric infection 18-21
- perinatal transmission 18
- peritoneal dialysis 1
- Pneumocystis carinii* 32, 36
- pyrazinamide 8
- renal disease 1
- rifabutin 9
- rifampin 8
- risk groups 26, 27, 32, 36-38
- risk reduction 25, 35
- safety equipment 11
- safety precautions 1, 2, 11-13
- saliva 11
- semen 11
- seroconversion 3
- seroprevalence 5, 6, 19, 23, 26-30, 32, 34
- sex clubs 34, 35
- sterilization 1
- syphilis 34
- tears 11
- treatment 8
- tuberculosis 8-10, 15-17
- United States 26, 36-38
- urine 11
- vaccine 19
- von Willebrand's disease 32
- Western blot test 2, 5