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Update: HIV-2 Infection — United States

Human immunodeficiency virus type 2 (HIV-2) infection was first described in 1985 in asymptomatic West African prostitutes (1) and, in 1986, was reported in two West Africans with acquired immunodeficiency syndrome (AIDS) (2). The first confirmed case of HIV-2 infection in the United States was reported in late 1987 in a West African woman with AIDS (3). Since then, six additional cases of HIV-2 infection have been reported to CDC—three from Massachusetts, and one each from Connecticut, Rhode Island, and Florida. This article summarizes information about two of the six cases reported since 1987 (4-7).

Case 1. In May 1988, a 34-year-old woman developed fever, night sweats, headache, and focal seizures. Evaluation, including an open brain biopsy, led to the diagnosis of cerebral toxoplasmosis. An enzyme immunoassay (EIA) for HIV-1 antibody and an HIV-1 Western blot (WB) assay were both negative, but an HIV-2-specific EIA and an HIV-2-specific WB were positive for HIV-2 antibody.

The woman, originally from West Africa, had married twice and had children from each marriage. Her first husband reportedly had many extramarital sex partners. She moved to the United States in the late 1970s; her second marriage was to an expatriate from her native country. She denied intravenous (IV)-drug use, extramarital sex partners, and receipt of transfusions. Her second husband and the four children who were tested had no serologic evidence of HIV-1 or HIV-2 infection.

Case 2. As part of the required medical screening process for immigration to the United States, a West African woman was tested for HIV infection in 1988 in Canada. The EIA for HIV-1 antibody was reactive, but the WB was indeterminate. Testing for HIV-2 antibody was positive by both HIV-2-specific EIA and HIV-2-specific WB. She had no history of AIDS or other HIV-related illnesses.

Before moving to the United States in 1984, the woman had had repeated sexual contact with a West African man who had had numerous female sex partners, including prostitutes. After moving to the United States, she married an expatriate from her native country. She denied IV-drug use, receipt of transfusions, and known occupational exposure to HIV-infected persons.

The woman was pregnant when HIV-2 antibody was detected, and she elected to terminate her pregnancy. Fetal tissue in poor condition

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was submitted for viral culture, but HIV-2 was not recovered. The woman had had a full-term stillborn infant in 1985 and a healthy infant in 1986. Her husband declined testing for himself and for their 2-year-old child.

Editorial note: Infection with HIV-2 appears to be rare in the United States and is, largely or entirely, limited to imported cases. HIV-2 infection appears to be most prevalent in West Africa (*8*). Persons infected with HIV-2 have also been reported from Central Africa (*9*), Western Europe (*8*), Canada (*5*), and Brazil (*10*). In the United States (Table 1), all identified HIV-2-infected persons have been West Africans. All evidence suggests that these persons became infected through heterosexual contact with other infected West Africans. All but one of these cases of HIV-2 infection have been reported from northeastern states, reflecting, in part, the settlement pattern of West African expatriates in the United States.

Because HIV-1 and HIV-2 are closely related, tests for antibody to one virus may crossreact with antibody to the other (*11*). Among Food and Drug Administration (FDA)-licensed tests, the sensitivity of HIV-1 EIAs for detecting HIV-2 antibody ranges from approximately 60% to >90%, depending on the specifc HIV-1 EIA employed and the clinical status of the infected person (*12, 13*). When tested for antibody to HIV-1, persons infected with HIV-2 may be reactive by EIA but indeterminate or negative by WB (*11*). Therefore, confirmation of HIV-2 infection requires both HIV-1 and HIV-2 WB testing. Even when both tests are performed, however, HIV-2 may be difficult to differentiate from HIV-1 infections (*14*). Assays for HIV-1- and HIV-2-specific peptides (*15*), the polymerase chain reaction procedure (*16, 17*), or viral cultures (*18*) may be helpful in this situation.

HIV-2 infection should be considered in persons with clinical evidence of HIV infection who are HIV-1 EIA-nonreactive or who are HIV-1 EIA-reactive and HIV-1 WB-negative or -indeterminate. Persons from West Africa who have evidence of HIV infection should be evaluated for HIV-2 infection, regardless of HIV-1 EIA or WB results. HIV-2-specific EIAs and WBs have not yet been licensed by FDA. Testing is performed by CDC and other research laboratories.

Because the modes of transmission for HIV-2 and HIV-1 are likely to be the same, the recommended preventive measures are identical. CDC is monitoring the epidemiology of HIV-2 infection in the United States through case surveillance and serologic surveys of groups such as Peace Corps volunteers returning from Africa, sexually transmitted disease clinic patients, drug-treatment center patients, counseling and testing site clients, patients from sentinel hospitals, and potential blood donors.

Surveillance at blood collection agencies relies on the crossreactivity that exists between EIA tests for antibodies to HIV-1 and HIV-2. Among approximately 4 million potential U.S. blood donors per year, specimens reactive by HIV-1-specific EIA will be tested for HIV-2 infection with HIV-2-specific EIA and WB tests. However, few, if any, potential blood donors infected with HIV-2 are expected because FDA revised its recommendations to blood collection agencies in April 1988 to exclude donors who recently immigrated from sub-Saharan Africa or who are recent sexual contacts of West Africans (FDA, personal communication). None of the six HIV-2-infected persons reported here were actual or prospective blood donors.

From late 1986 to early 1988, CDC, FDA, and collaborating organizations tested > 22,000 serologic specimens, including > 10,000 specimens from persons at risk for HIV-1 infection, for serologic evidence of HIV-2 infection (*3*). Specimens were tested with HIV-1- and HIV-2-specific EIA, WB, and synthetic peptide tests. None of the specimens were positive for HIV-2 alone, although 10 specimens were reactive to both HIV-1- and HIV-2-specific synthetic peptides (Genetic Systems Corporation, unpublished data). These 10 persons might be infected with HIV-1 alone, HIV-2 alone, or both viruses. On the basis of this survey and the small number of known cases of HIV-2 infection, HIV-2 infection in the United States appears to be limited.

Adapted from MMWR 1989;38:572-574, 579-580

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TABLE 1. Reported cases of HIV-2 infection — United States*

Case	Region of origin	Clinical status	HIV-1† EIA	HIV-1† WB	HIV-2† EIA	HIV-2† WB	Date of diagnosis	Entered United States	Ref.
§	West Africa	AIDS	-	I		+	Dec. 1987	1987	(3)
1	West Africa	AIDS	-	-	+	+	May 1988	1979	(4)
2	West Africa	Asymptomatic	+	I	+	+	May 1988	1984	(5)
3	Unknown	Unknown	+	I	+	+	Aug. 1988	?	(6)
4	West Africa	Asymptomatic	-	I	+	+	Sept. 1988	1986	-
5	West Africa	AIDS	-	I	ND	+	Aug. 1988	1983	(7)
6	West Africa	Asymptomatic	+	I	+	+	March 1989	1988	-

*Does not include six cases that are under investigation.
†(+) = reactive, (-) = nonreactive, (I) = indeterminate.
§First case reported in the United States (not included in this report).

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Lung Cancer and Exposure to Radon in Women — New Jersey

In 1985, the New Jersey State Department of Health (NJDOH) initiated an epidemiologic study of lung cancer and exposure to radon in New Jersey women. In collaboration with the New Jersey State Department of Environmental Protection and the National Cancer Institute, NJDOH examined whether exposure to radon in homes is associated with increased lung cancer risk.

This study was based on a previous statewide case-control study of risk for lung cancer. In that study, cases were defined as lung cancer diagnosed in women (n = 994) between August 1982 and September 1983; controls were 995 women selected from drivers' license, Health Care Financing Administration, and death certificate files (*1*). The 1985 radon substudy focused on New Jersey dwellings in which participants had lived for at least 10 years from 10 to 30 years before lung cancer diagnosis or control selection (*2*).

For a 1-year period, radon concentrations in living areas were measured by alpha-track detectors. In basements, 4-day exposures were measured using charcoal canisters to provide rapid screening assessments for current residents, thereby enabling immediate remediation if necessary, and providing alternate data in the event year-long measurements of radon could not be completed. Mean differences in duplicate alpha-track measurements, conducted for about 10% of the residences, were considered sufficiently small to exclude measurement error as a major contributor to exposure misclassification.

Analysis of exposure data by radon concentration for 433 cases and 402 controls found no statistically significant differences (Table 1). However, the trend for increasing risk for lung cancer with increasing radon exposure was statistically significant (Table 1). When cumulative exposure (concentration multiplied by duration) was considered, a similar but not statistically significant trend of increasing risk with increasing exposure was seen.

The relative risk coefficient (i.e., the increase in lung cancer risk over background risk per unit of cumulative exposure) was 3.4% (90% confidence limits = 0, 8.0%) per working level month.* In studies of underground miners (*3,4*), for whom the

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occupational exposures were much higher, the range was 0.5%–4.0% per working level month. Analyses by smoking categories indicated that, for persons who smoke < 15 cigarettes a day, the association between radon exposure and lung cancer was strongest.

The data indicated that year-round exposures in living areas were two to five times lower than basement measurements taken during heating season. The difference increased with higher concentrations. For example, the average annual living area radon concentration was generally below 4 pCi/L (the Environmental Protection Agency's maximum exposure guideline) in houses with basement screening results approaching 20 pCi/L (2).

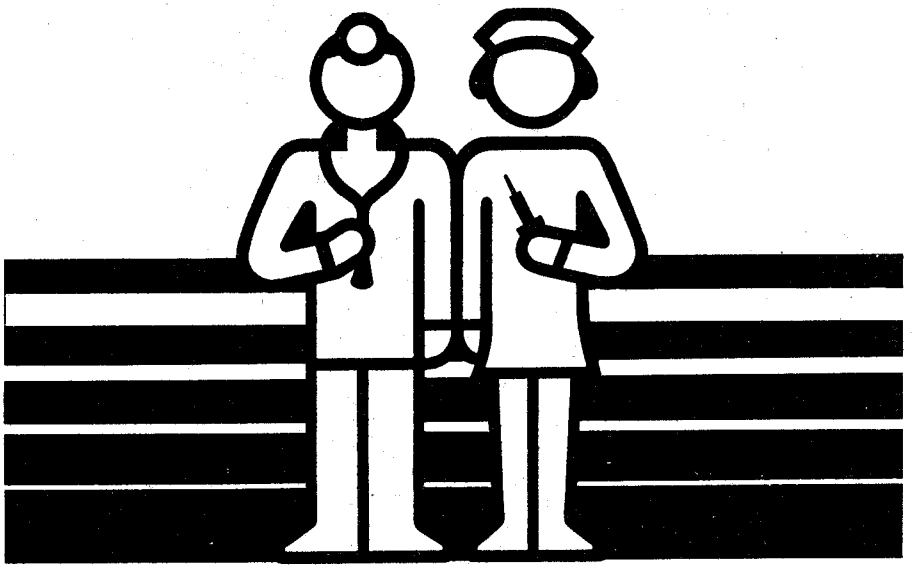
Editorial Note: Radon is a chemically inert gas produced by the radioactive decay of uranium. The immediate decay products of radon are chemically reactive metals (polonium, bismuth, and lead) that tend to be retained in the lung when inhaled. The polonium decay products emit highly ionizing alpha particles. Studies of underground miners, animals, and dosimetry modeling have shown that radon decay products are lung carcinogens (3,5). In particular, epidemiologic studies of miners have shown a strong and consistent dose-response relationship between lung cancer and radon exposure (3). However, information on residential risk from exposure to radon has been limited (3,5), and other residential studies either have not addressed other risk factors for lung cancer, such as smoking, and/or have not measured radon in the houses of all participants (6-9).

The New Jersey study is the first major epidemiologic study of radon exposure and lung cancer that used both measurements of radon levels in homes and detailed smoking histories for participants. NJDOH believes its findings support the use of the studies of miners for risk extrapolations to the residential setting.

An important limitation on the interpretation of this study is the small number of persons who were in the highest radon-exposure categories (2). NJDOH also considered other possible biases introduced by reducing the potential study population to persons for whom radon-exposure estimates were collected (2).

The relationship between short-term screening measurements and year-round living area measurements requires improved characterization for public policy purposes and clear understanding before remediation decisions are made. When winter and summer short-term measurements are averaged to obtain year-round exposure estimates, overestimations may result (10).

NJDOH has recommended that existing actions to reduce radon exposure to the lowest feasible levels should be maintained pending other research, and remedial action should be taken in residences where both short- and long-term testing indicate that typical exposures for occupants exceed 4 pCi/L. This recommendation is based on the limited feasibility of remediating residences with radon levels < 4 pCi/L. Building code modification to prevent radon entry may be effective in reducing overall population risks from radon exposure (2). Health-care providers should advise their patients, particularly those who smoke, of the health risks associated with radon exposure and should consider recommending indoor radon concentration testing.



Adapted from MMWR 1989;38:715-718

*One hundred seventy hours exposure to any combination of radon daughters in 1 liter of air that results in 1.3×10^5 million electron volts of potential alpha energy.

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TABLE 1. Distribution of lung cancer cases and controls, by radon level* — New Jersey radon/female lung cancer case-control study, 1982–1988

	Radon level (pCi/L†)								
	<1.0§		1.0–1.9		2.0–3.9		4.0–11.3		
Category	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**Total**
Cases	342	(79.0)	67	(15.5)	18	(4.2)	6	(1.4)	433
Controls	324	(80.6)	66	(16.4)	10	(2.5)	2	(0.5)	402
Total	**666**	**(79.8)**	**133**	**(15.9)**	**28**	**(3.4)**	**8**	**(1.0)**	**835**
Adjusted OR¶ (90% CL)	1.0		1.1 (0.8, 1.7)		1.3 (0.6, 2.9)		4.2 (1.0, 17.5)**		

*Year-long living area alpha-track measurements (n = 664). Estimates derived from basement alpha-track or charcoal-canister measurements (n = 171).
†Picocuries per liter.
§Includes persons whose index address was an apartment above the second floor or a trailer.
¶Odds ratios (OR) and 90% confidence limits (CL): estimate of the lung cancer risk associated with exposure to a given level of radon, after adjusting for other factors (e.g., cigarette smoking, age, occupation, and respondent type). Test for trend in OR with increasing radon: p = 0.04.
**OR for radon exposure of >2.0 pCi/L = 1.8 (90% CL = 0.9, 3.5).

Cases of selected notifiable diseases, Virginia, for the period October 1 through October 31, 1989.

DISEASE	TOTAL CASES REPORTED THIS MONTH						TOTAL CASES REPORTED TO DATE		
	STATE	REGIONS					THIS YEAR	LAST YEAR (STATE TOTALS)	5 YEAR AVERAGE (STATE TOTALS)
		N.W.	N.	S.W.	C.	E.			
Acquired Immunodeficiency Syndrome	23	1	8	0	7	7	325	314	—
Campylobacter Infections	47	10	13	6	10	8	594	578	548
Gonorrhea	1496	—	—	—	—	—	13531	11943	14634
Hepatitis A	40	3	13	1	15	8	268	326	173
B	23	1	1	7	3	11	262	273	390
Non A-Non B	3	1	0	0	1	1	63	67	64
Influenza	30	0	0	19	0	11	1917	2465	1966
Kawasaki Syndrome	4	0	2	0	1	1	22	12	20
Legionellosis	1	1	0	0	0	0	9	10	16
Lyme Disease	8	1	0	0	1	6	43	26	11
Measles	0	0	0	0	0	0	22	200	59
Meningitis — Aseptic	80	12	21	12	11	24	335	149	233
Bacterial*	10	1	1	1	1	6	150	138	178
Meningococcal Infections	4	0	1	0	2	1	55	46	54
Mumps	11	0	5	2	0	4	111	134	61
Pertussis	3	0	0	2	0	1	33	21	28
Rabies in Animals	19	5	3	0	11	0	219	308	227
Reye Syndrome	0	0	0	0	0	0	2	0	2
Rocky Mountain Spotted Fever	3	0	0	0	3	0	16	17	32
Rubella	0	0	0	0	0	0	0	11	3
Salmonellosis	145	14	35	19	35	42	1251	1516	1363
Shigellosis	18	1	5	2	5	5	365	394	182
Syphilis (Primary & Secondary)	43	0	4	10	19	10	474	363	308
Tuberculosis	37	5	13	8	4	7	302	333	342

Localities Reporting Animal Rabies: Amelia 3 raccoons; Arlington 1 raccoon; Chesterfield 1 raccoon, 1 skunk; Cumberland 1 raccoon; Dinwiddie 1 raccoon; Fairfax 1 raccoon; Fauquier 1 skunk; Loudoun 1 raccoon; Nottoway 2 raccoons; Prince George 2 raccoons; Rockbridge 1 skunk; Shenandoah 1 cat, 1 skunk; Spotsylvania 1 cat.

Occupational Illnesses: Asbestosis 2; Carpal Tunnel Syndrome 26; Coal Workers' Pneumoconiosis 30; De Quervain's Disease 1; Loss of Hearing 9; Poisoning—Lead 1; Poisoning—Mercury 1; Repetitive Trauma Disorder 3; Silicosis 1.

*Other than meningococcal

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