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Risk of Human Immunodeficiency Virus (HIV-1) Infection Among Laboratory Workers

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In a prospective cohort study of 265 laboratory and affiliated workers, one individual with no recognized risk factors for human immunodeficiency virus type 1 (HIV-1) infection was HIV-1 seropositive at the time of entry into the study. Molecular analyses of two HIV-1 isolates derived in two independent laboratories from a blood sample from this worker showed that the isolates were indistinguishable from a genotypic form of HIV-1 present in the H9/HTLV-III$_B$ cell line. Exposure to this strain of virus most probably occurred during work with concentrated virus or culture fluids from virus-producing cell lines under standard Biosafety Level 3 containment. Although no specific incident leading to this infection has been identified, undetected skin contact with virus culture supernatant might have occurred. This worker was the only one found to be positive among the subgroup of 99 workers who shared a work environment involving exposure to concentrated virus. The incidence rate of 0.48 per 100 person-years exposure indicates that prolonged laboratory exposure to concentrated virus is associated with some risk of HIV-1 infection, which is comparable to the risk for health care workers experiencing a needle stick exposure. While none of the ten workers with parenteral exposure to HIV-1 in this cohort became infected, a worker in another laboratory did seroconvert following an injury with a potentially contaminated needle. Strict Biosafety Level 3 containment and practices should be followed when working with concentrated HIV-1 preparations, and further refinement of the procedures may be necessary.

HUMAN IMMUNODEFICIENCY VIRUS type 1 (HIV-1), the etiologic agent of the acquired immunodeficiency syndrome (AIDS), can be transmitted by sexual intercourse, by parenteral inoculation including accidental needle stick, by transfusion of infected blood, or from mother to offspring probably in utero, but not by casual exposure (*1–3*). A few case reports suggest that direct exposure of the skin or mucous membranes to infected blood may result in the development of antibodies to HIV-1 (seroconversion), particularly if the integrity of the skin has been compromised by a dermatologic condition such as eczema (*4*).

For more than 100 years infections have been recognized as occupational hazards among laboratory workers (*5*). Safety guidelines and approaches have been adopted, including the current standards of containment for biologic agents (*6*). Biosafety Level 3 practices are generally recommended for handling concentrated preparations of HIV-1, and Biosafety Level 2 for routine clinical specimens (*7*).

Between 1985 and 1987, invitations to participate in a prospective cohort study to assess laboratory risk were extended to workers from 6 states in 15 laboratory facilities where there was a risk of possible exposure to HIV-1. Each subject signed an informed consent form and completed a two-part questionnaire. Blood samples were analyzed for antibody to HIV-1 and HTLV-I with standard commercially available enzyme-linked immunosorbent assays (ELISA). Follow-up blood samples and questionnaires were obtained every 8 to 12 months. All specimens reactive in ELISA (including those with borderline reactivities) were further evaluated by immunoblots with disrupted HIV-1 and HTLV-I, by radioimmune precipitation assays with labeled HIV-1–producing H9 cells, and by radioimmune assay for specific reactivity to HIV-1 or HTLV-I core (p24) or HIV-1 envelope (gp120) proteins. Consent forms were held by a private physician, an expert in infectious diseases (F.A.G.), who informed each subject about test results. For any subject with a positive or borderline result, the physician provided counseling, performed a standardized physical examination for signs or symptoms of HIV-related conditions, and obtained additional blood specimens for HIV-1 serology and viral isolation (*8*). The first part of the questionnaire focused on occupational exposure to human retroviruses, accidents in the laboratory or elsewhere, and use of biosafety precautions

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by the subject and in the facility. The data provided were entered directly into the computer database with no link to identifying information. The second part of the questionnaire, with risk factor information, including sexual activities, drug abuse, transfusions, and country of origin, was sealed by the study subject in a separate envelope and submitted directly to one of us (S.H.W.), who recoded and entered these data as a single measure of risk ranging from zero to high (Table 1).

Among the 265 study subjects, 40 were not directly involved in HIV-1 laboratory work: 10 were clerical staff working in a laboratory environment, and 30 had other sources of exposure to AIDS patients while working in support of laboratory personnel (Table 1). Eight (3%) of the study subjects recorded factors associated with a high-risk profile for HIV-1. None of the subjects in the high, low, or very low risk groups was seropositive. Among the 225 with laboratory exposure, 126 were involved in processing HIV-1–positive blood or in performing molecular biology or serology with noninfectious viral proteins or cloned viral DNA; 99 worked with concentrated HIV-1 (*9*). Out of the 225 with laboratory exposure, 35 subjects reported one or more skin contacts, and 10 reported parenteral virus exposure, including needle sticks or cuts. Gloves were not worn at all times by 13 persons who worked with HIV-1–positive material. Subjects often experienced more than one mode of exposure. No HIV-1–seropositive subject was found among those with parenteral exposure or cuts or among those who did not always wear gloves. No study participant was HTLV-I–seropositive or had circulating HIV-1 p24 antigen (Abbott).

Table 1. Characteristics of 265 laboratory and affiliated workers.

Characteristics	Number of subjects
Age (years)	
<30	109
30 to 39	98
40 to 49	38
>50	20
Race	
White	197
Black	26
Asian/Pacific islander	37
Other/unknown	5
Sex	
Male	136
Female	129
AIDS risk scale*	
High	8
Low	16
Very low	31
Zero	210
Occupational exposure	
No HIV-1 laboratory work	40
Some HIV-1 laboratory work	225
No concentrated HIV-1 work	126
Some concentrated HIV-1 work (*9*)	99
Any skin exposure†	35
Once	23
More than once	12
Not always wearing gloves†	13
Any parenteral inoculation†	10

*AIDS risk scale is hierarchical: high, homosexual man, parenteral drug abuser, hemophiliac, or person with incomplete risk factor information; low, heterosexual with more than 20 (or unknown number of) partners within the last 5 years, or history of residence in a country where heterosexual transmission is the primary mode of HIV transmission; very low, heterosexual with 5 to 19 partners in the last 5 years, or blood transfusion between 1978 and 1985, or heterosexual anal intercourse; zero, all others. †Among 225 HIV-1 laboratory workers.

At the time of entry to the study, one subject, who worked with concentrated HIV-1 (*9*), was seropositive. This subject had no history of any sexually transmitted disease and had no antibodies to hepatitis A or B, cytomegalovirus, *Treponema pallidum*, or HTLV-I. The subject denied any parenteral exposures and any risk behavior linked to HIV-1 positivity both in the confidential self-administered questionnaire at study entry and subsequently during independent epidemiologic interviews by four trained observers. The subject reported that all work with concentrated virus had taken place under Biosafety Level 3 containment; latex gloves and standard cloth laboratory gowns (over street clothes) had been worn, but no mask had been used during this work. This subject could not recall any episode of direct skin exposure, but did report occurrences of HIV-1 contamination in the work area. The subject reported that double gloves were worn whenever there were bandaged cuts on fingers or hands. The subject did recall an episode of nonspecific dermatitis on the arm, but the affected area was always covered by a cloth laboratory gown. There was no known direct contact of potentially infectious material with these areas such as was reported for health care workers infected after clinical exposure to HIV-1–positive body fluids (*4*). On clinical evaluation, the subject reported no symptoms suggestive of the HIV-1 seroconversion syndrome, and physical examination was normal with no dermatologic condition, lymphadenopathy, or other evidence of HIV-1–related illness.

Safety operations of all participating laboratories were reviewed by one of us (W.E.B.) with expertise in laboratory safety issues. Site visits were made to selected HIV-1 laboratories, including the laboratory of the seropositive subject, as part of routine safety visits to review compliance with established biosafety guidelines. Operational practices were found to be satisfactory in all cases. In addition, safety observations before the subject's enrollment in the current study showed acceptable safety performance. During interviews, the seropositive subject related that there had been a number of episodes of leakage of virus-positive culture fluid from equipment. On occasion, there was obvious contamination of rotors. Proper decontamination procedures were used in cleaning up spills in the laboratory. Rotor decontamination, in some instances, involved the use of a hand brush submerged in disinfectant to remove potentially contaminated cellular debris. Contamination of the work area and rotors, however, may not have been recognized, and thus potentially contaminated gloves may not always have been changed according to biosafety guidelines. The individual reported no episode where the integrity of the latex gloves was compromised during these circumstances but did relate other episodes under Biosafety Level 3 where pinholes or tears in gloves required that they be changed immediately. Double gloves were not worn routinely. The subject indicated that there was no skin or obvious aerosol exposure during the decontamination of equipment. During the instances in which decontamination procedures were instituted, a mask as well as gown and gloves were worn.

At the time of entry into the cohort study, the subject had already experienced a total of 290 days of potential exposure to concentrated HIV-1 (*9*). The serologic analysis of this subject's entry specimen was equivocal by ELISA and immunoblot, but radioimmunoprecipitation assay (RIPA) showed reactivity to the HIV-1 envelope components gp160 and gp120 without other specificities, a pattern suggestive of early seroconversion (Fig. 1A, lane 1). On subsequent serial samples, ELISA and other confirmatory assays became unequivocally positive with strongly reactive antibodies to all HIV-1 proteins (Fig. 1A, lane 2). An HIV-1 p24 antigen assay (Abbott) was repeatedly negative on all serum samples from this subject, including the early seroconversion sample.

Blood samples from this subject were cultured for HIV-1 isolation in laboratories where the subject would not inadvertently be reexposed to cells or the virus strain from these cultures. Isolation of HIV-1 was attempted on 15 occasions from this seropositive worker [methods in legend to Fig. 1 (*10*)]. On the most recent attempt a single venipuncture was performed and aliquots were distributed independently to four separate laboratories. In two laboratories, transient low-level reverse transcriptase activity was detected, but the isolates were obtained in the other two laboratories. Isolate A was

obtained in one of these laboratories by cocultivation of the subject's cells with peripheral blood mononuclear cells (buffy coat adherent) from a normal donor. Isolate B was obtained in the other laboratory by long-term cultivation of the subject's peripheral blood monocytes. Both culture systems were positive for retrovirus infection by p24 antigen capture, reverse transcriptase activity, and classic electron microscopic morphologic features of HIV-1 (before passage to H9 cells). The laboratory responsible for isolate A had not previously grown HTLV-III_B.

Isolate A was subsequently passaged into the H9 lymphoid cell line (documented to be HIV-1 negative in parallel culture), and a continuously producing line was established. High molecular weight DNA from the H9 infected cell line (isolate A) and the monocyte-macrophage cultures (isolate B) were independently analyzed in two different laboratories by the Southern blot technique with a battery of restriction endonucleases and a 9-kb near-full-length HIV-1 specific probe (Fig. 1B). The predominant restriction fragment patterns of isolates A and B were indistinguishable from each other and from one of the predominant proviral forms present in the HTLV-III_B virus isolate (*11*). The 9-kb Sst I fragment in isolates A and B corresponded to one of the two major Sst I genotypic patterns present in the H9/HTLV-III_B line (*11*). Furthermore, the internal viral restriction fragments generated by Eco RI, Hind III, Bgl II, Pst I, and Pvu II were identical among isolate A, isolate B, and HTLV-III_B. Isolate A and H9/HTLV-III_B were further analyzed with Bam HI, Kpn I, and Xho I and shown to be indistinguishable. Several very faint bands are visible in the Hind III and Pst I digestions of the isolates from the worker that are not present in H9/HTLV-III_B. It will require further study to determine whether these bands correspond to minor viral populations with restriction site polymorphisms that result from genotypic variation.

Given the epidemiologic, serologic, virologic, and molecular data presented here, the only plausible source of this individual's virus infection is the laboratory. It is well documented that wild-type strains of HIV-1 isolated from different patients vary enormously (*11*), yet the virus strain isolated from this worker shared a DNA genotypic pattern identical to one of the two predominant genotypic patterns present in the H9/HTLV-III_B line. That contamination of the worker's blood samples or cultures occurred in two separate laboratories seems unlikely. Furthermore, a new virus isolation (in the laboratory responsible for isolate A) from a blood sample drawn 5 months after the initial positive sample shows an identical restriction pattern to that in Fig. 1B for the enzymes tested so far (Sst I, Hind III, Bgl II, and Pvu II).

This laboratory worker was involved in a number of possible exposure circumstances (decontaminating equipment, cleaning up spills, or touching potentially contaminated surfaces with gloved hands) as part of duties related to culture, production, or concentration of large volumes of virus-positive tissue culture material. On the basis of previous

Table 2. Risk of HIV infection in laboratory and affiliated workers.

Type of exposure	Number of subjects		Average years of follow-up*	HIV-1 infection rate*	Upper 95% confidence interval on HIV-1 infection rate*
	Total	Infected with HIV-1			
No HIV-1 laboratory work	40	0	0.358	0.00	20.9
Some HIV-1 laboratory work	225	1	1.925	0.24	1.15
No concentrated HIV-1 work	126	0	1.762	0.00	1.44
Some concentrated HIV-1 work	99	1	2.123	0.48	2.30

*Follow-up begins at the date first exposed and ends when follow-up ends or when seroconversion is documented, whichever occurs first. The infection rate is expressed as infections per 100 person-years of follow-up. If there is one infection, the 95% upper confidence interval on the infection rate, λ, is determined from $(1 + \lambda T)\exp(-\lambda T) = 0.05$, where T is the total follow-up time for a given exposure group. If there is no infection, the confidence interval is determined from $\exp(-\lambda T) = 0.05$. These rate calculations, and the corresponding average years of follow-up are based on those individuals for whom a date of initial exposure is well documented, namely, 40 persons with no laboratory exposure, 118 with laboratory exposure but no exposure to concentrated HIV-1, and 97 with exposure to concentrated HIV-1.

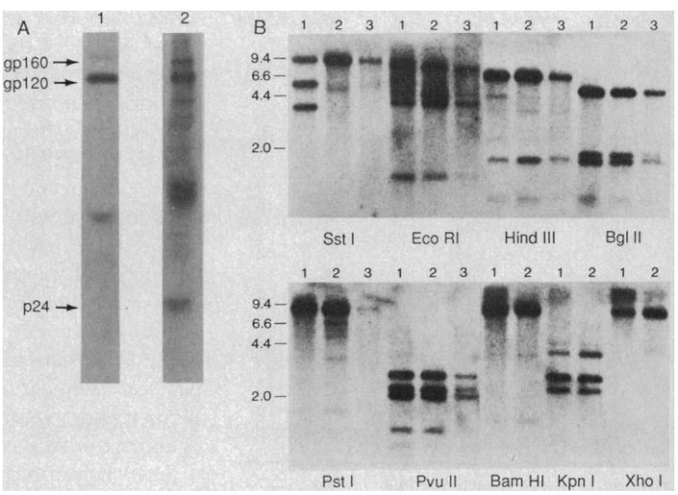

Fig. 1. (**A**) (Lane 1) Assay (RIPA) of the laboratory worker's serum at entry into the study. There is a faint band at gp160 and a prominent band at gp120 reflecting the presence of antibodies to the envelope protein on this first sample. There are no antibodies to the core (gag) proteins in the initial sample. The pattern is indicative of early seroconversion. (Lane 2) Assay (RIPA) of serum obtained from the same subject 4 months after the sample shown in lane 1. A full complement of antibody reactivity to envelope and core antigens typical of a seropositive individual is detected. The method for RIPA has been reported (*18*). The viral culture methodology was modified by the use of buffy coat preparations including adherent cells in the coculture mixture at a 3:1 ratio of target to patient cells (isolate A) (*10*). For isolate B, the patient's peripheral blood monocytes were cultured as described (*10*). Virus-containing culture fluids from the primary culture were used to infect subsequent cultures of monocyte-macrophage cells from normal donors. The DNA from these cultures was subjected to molecular hybridization. Virus was detected by standard methods (*10*). (**B**) Southern blot–hybridization comparison of the H9/HTLV-III_B cell line (*11*) (lane 1) and two independent HIV-1 isolates, isolate A (lane 2) and isolate B (lane 3). For all restriction endonucleases tested, the two HIV-1 isolates from the study subject are identical in their predominant genotypic patterns to each other and to one of the two predominant viral genotypes known to comprise the H9/HTLV-III_B cell line (*11*). The H9/HTLV-III_B virus isolate is polymorphic in its Sst I pattern with viral DNA fragments of 9, and 5.5 and 3.5 kb corresponding to two distinct proviral genomes. Isolates A and B from the study subject contain only one predominant viral form that lacks the internal Sst I restriction site. Also, Eco RI cuts the viral DNA twice, giving rise to an internal fragment of approximately 1 kb and three additional bands corresponding to cleaved unintegrated circular and linear fragments. Isolate B was not analyzed with Bam HI, Kpn I, and Xho I because of insufficient amounts of DNA. Methods for preparation of high molecular weight DNA, restriction endonuclease digestion, gel electrophoresis, and blot-hybridization are described elsewhere (*11*). In this experiment, 2 μg of high molecular weight DNA from isolates H9/HTLV-III_B and A, and 10 μg of DNA from isolate B were used. After hybridization to a near-full-length ^{32}P-labeled HIV-1 probe [BH10 (*11*)], the nitrocellulose filters were exposed to x-ray film for 3 days.

studies of laboratory-acquired infection by a blood-borne pathogen (*12, 13*), the most plausible source of exposure was contact of the individual's gloved hand with H9/HTLV-III_B culture supernatant with inapparent and undetected exposure to skin.

Since only a single infection was detected in our cohort, we emphasize that the risk of laboratory-acquired infection is very low. The rates of infection per 100 person-years of follow-up are given in Table 2, together with upper 95% confidence intervals on possible rates of infection. The observed rates of infection are 0.00 for nonlaboratory workers in this study and for workers not exposed to concentrated HIV-1. The upper 95% confidence interval is large (20.9 per 100 person-years) for nonlaboratory workers because the follow-up period is short. Among those exposed to concentrated virus, the observed rate is 0.48 per 100 person-years, and the upper 95% confidence interval is 2.30 per 100 person-years of exposure (Table 2). If all laboratory workers are considered together, the observed rate is 0.24 per 100 person-years, and the upper 95% confidence interval is 1.15. With 95% confidence, the true seroconversion rates are lower than the upper confidence in Table 2, and these data are also consistent with seroconversion rates near zero. These calculations suggest that some retrovirus laboratory workers, particularly those working with concentrated virus, may have approximately the same magnitude of risk of HIV-1 infection as health care workers who experience an injury from an HIV-1–contaminated needle (*2, 14*).

A laboratory worker with parenteral exposure to concentrated HIV-1 and consequent seroconversion has also been reported (*15*). A potentially contaminated stainless steel needle used for cleaning an apparatus caused a cut on the hand of this worker who was employed in the production of concentrated virus. The magnitude of risk resulting from documented parenteral exposure in the laboratory cannot be calculated since the total number of such exposed individuals is not known. Among the ten individuals who reported such exposure in our study, none underwent seroconversion. According to current Biosafety Level 3 practices, glassware or sharp objects should not be used when working with pathogens.

The lack of overt documented parenteral exposure in the subject in our cohort study is reminiscent of the exposure circumstances for laboratory-acquired infection with hepatitis B virus (*12, 13, 16*). The fact that HIV-1 infection in the laboratory workers took place under prescribed Biosafety Level 3 containment suggests the need to review carefully all operations involving highly concentrated infectious material and to ensure proficiency in the conduct of recommended safeguards. For example, although they were careful, neither the laboratory worker reported here nor the worker who seroconverted after a parenteral exposure was fully conversant with or strictly adhered to biosafety guidelines in day-to-day procedures at all times.

We recommend that laboratories working with HIV-1 and related agents carefully review their biosafety containment policies and promote ongoing educational programs to teach all employees the precautions necessary in the laboratory. This training is particularly important for new employees or persons working with concentrated virus for the first time, when lack of familiarity with procedures may place them at even higher risk (*2*). The need for proficiency and strict adherence to the procedures should be emphasized. The understanding and compliance of employees should be periodically monitored and evaluated by the laboratory biosafety officer and laboratory manager. Special attention should be given to mechanisms of indirect transmission, such as those that contribute to occupationally acquired hepatitis (*12, 13, 16*). Procedures that should be emphasized are: (i) the avoidance of needles and sharp implements when alternative methods are available; (ii) the use of gloves when handling materials containing HIV-1; (iii) the removal of gloves after any contact with a potentially (including inapparently) contaminated surface or material and careful hand-washing and replacement of gloves before proceeding with further work; (iv) decontamination of work surfaces after any spill of HIV-1 materials and after handling of material is completed; and (v) avoidance of hand contact with mouth, eyes, ears, and nose; this requirement can be made easier if goggles or a face shield or face mask are worn.

The operational integrity of all equipment used to transport fluids containing HIV-1 should be validated periodically. The occurrence of any spill or leakage should initiate a formal review to assess exposure potential for workers and to identify corrective actions to prevent recurrence. Workers must be attentive and skillful when continuous flow zonal centrifuges are used in processing HIV-1 materials, and they should be fully cognizant of proper use and decontamination methods to control centrifuge biohazards (*17*). Workers with active dermatitis or skin lesions on the hands or wrists should not perform procedures involving the transfer of concentrated HIV-1 materials, even if their skin is protected by gloves. Any worker who suspects that a parenteral or other exposure event has occurred (for example, suspected skin or mucous membrane contact) should report the incident to a supervisor and should be monitored by occupational medical personnel on a regular basis for evidence of illness or seroconversion.

Although the issue of routine serological monitoring of laboratory workers for HIV-1 infection is complicated by the possibility of infection being acquired outside the work setting through recognized routes of exposure, such monitoring is necessary to ensure that recommended laboratory guidelines for HIV-1 are followed. We propose that routine, periodic serologic testing for HIV-1 be established as a part of laboratory safety programs. This testing must be done in an environment that protects the privacy of the worker and, through legal means, protects the worker from discrimination in the workplace.

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