and Pz; 10-20 International system). The grounding electrode was attached to the forehead. Eye movements were recorded by electrodes placed above and to the right of the right eye. Trials with excessive eye movements or blinks were rejected before being analyzed. Electrode impedance did not exceed 5 kilohm. The signals were amplified by a Grass 7P122 amplifier, with a time constant of 8.0 seconds and an upper half-amplitude cutoff of 35 Hz. The data were digitized at a rate of 100 samples per second over an epoch of 1280 msec, beginning 100 msec before stimulus onset. The digitized data were filtered so that the upper half-amplitude cutoff was reduced to 6.29 Hz. The data were averaged so that for each subject, and for each condition, we had the ERP associated with the rare and frequent stimuli.

10. Even though not statistically significant, there is an apparent difference between the amplitudes of the P300 elicited by visual stimuli in the two groups. The data do not allow us to determine whether the AP subjects use a different processing strategy in both modalities.
11. The characters H and S could be labeled by all subjects with the same facility that the AP subjects labeled the tones. As the P300 component is present in the visual task, it seems reasonable to question the assumption that characters are recognized with automatic facility. Since characters can be presented in an infinity of fonts and shapes, considerable processing may be required for character recognition.
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Transfusion-Associated AIDS: Serologic Evidence of Human T-Cell Leukemia Virus Infection of Donors

Abstract. *An assay for antibodies to membrane antigens of cells infected by human T-cell leukemia virus was used to examine serum from persons who donated blood to 12 patients with acquired immunodeficiency syndrome (AIDS) associated with blood transfusions. The occurrence of positive results in the assay was significantly greater among donors to the AIDS patients (9 of 117; 7.7 percent) than among random donors (1 of 298; 0.3 percent). Of 12 sets of donors examined, 9 sets included a donor whose serum gave positive results for the presence of the antibodies. In six of these nine sets, the seropositive donor was an individual who was also identified as a possible source of AIDS transmission when epidemiologic and immunologic criteria were used.*

The acquired immunodeficiency syndrome (AIDS) is a recently recognized human disease whose incidence in the United States has been rapidly increasing (*1, 2*). Patients with AIDS suffer from at least one type of malignancy, Kaposi's sarcoma, and various life-threatening opportunistic infections, the most common of which is *Pneumocystis carinii* pneumonia. The disease is characterized by diverse immunologic abnormalities, including lymphocyte dysfunction and depletion of the T-helper subpopulation of lymphocytes (*3*).

Although the etiology of AIDS is unknown, epidemiologic observations suggest that it is caused by an infectious agent transmitted sexually and, less commonly, through parenteral exposure to blood or blood products (*4*). In the United States, about 94 percent of patients reported to have AIDS belong to one or more of the following groups: homosexual or bisexual men, abusers of intravenous drugs, persons born in Haiti, and persons with hemophilia (*2*). Another 1 percent of AIDS patients do not belong to any of these groups but received transfusions of blood or blood components within 5 years before the onset of their illness. These cases are classified as transfusion-associated AIDS and have been described in detail elsewhere (*5, 6*).

Among the agents studied as a possible cause of AIDS are retroviruses, including the human T-cell leukemia virus (HTLV) (*7*). Compared with control individuals, AIDS patients have a higher prevalence of antibodies directed against membrane antigens of HTLV-infected cells (HTLV-MA) as measured by indirect membrane immunofluorescence and radioimmunoprecipitation techniques (*8*). In studies in which serum from Japanese T-cell leukemia patients and asymptomatic carriers was used, the major antigens detected by antisera to HTLV-MA have been defined (*9*). They include two glycoproteins, designated gp 61 and gp 45, that are encoded by the *env* gene of HTLV (*9*). The prevalence of antibodies to HTLV-MA is also increased in homosexual men with chronic, generalized lymphadenopathy (*8*), an illness that has been associated with AIDS (*10*). Human T-cell leukemia virus has been isolated from the lymphocytes of AIDS patients, both American (*11*) and Japanese (*12*), and DNA sequences that hybridized with a cloned HTLV DNA probe were detected in two other patients (*13*). A retrovirus thought to be related to HTLV has also been isolated from a lymph node of a French homosexual man with chronic lymphadenopathy (*14*). Whether these findings reflect an etiologic role for a retrovirus in AIDS or are simply a consequence of these viruses infecting AIDS patients opportunistically is unknown.

In an attempt to evaluate further the serologic relationship of HTLV or a related virus to AIDS, we have studied patients with transfusion-associated AIDS and the persons who donated blood or blood components to these patients. As of 1 January 1984, the Centers for Disease Control (CDC) had received reports of 32 adults with transfusion-associated AIDS. An additional patient (patient 5), a 69-year-old man with Kaposi's sarcoma, whose age exceeds the limit of 60 years specified for Kaposi's sarcoma patients in the CDC surveillance definition for AIDS (*4*), is included in this report because his abnormal immunologic studies and rapidly fatal clinical course suggest that he did, in fact, have AIDS. For 12 of these 33 cases, donor investigations have been completed. An investigation is considered complete when all available donors (i) have been interviewed regarding risk factors for AIDS, (ii) have been examined for physical findings suggestive of AIDS, and (iii) have provided a blood sample.

Several classes of serum were obtained to serve as controls. These included 298 randomly selected specimens collected during 1979 and 1980 from volunteer blood donors in Philadelphia, Pennsylvania (100 specimens), Madison, Wisconsin (99 specimens), and Tucson, Arizona (99 specimens). Also included for comparison were 81 previously tested serum samples collected in 1981 from homosexual men matched by age, race, and residence to a sample of AIDS patients (*8, 15*) and 45 samples obtained in 1982 from homosexual men in Ithaca, New York.

Serum specimens from patients with transfusion-associated AIDS, donors to these patients, and control subjects were coded and sent frozen to Boston to test for antibodies to HTLV-MA by a method that has been described (*8, 16*). Briefly, a 1:4 dilution of serum was added to two HTLV-I-infected cell lines, Hut 102 and MT 2; the cells were washed; and a fluorescein-conjugated $F(ab')_2$ fragment of goat antibody to human immunoglobulins G, A, and M was added. The cells were washed again, and the proportion of cells having fluorescence was determined for each cell line. Samples with 40 percent or more of the cells having fluorescence on either cell line were considered positive. Positive samples were tested on uninfected lymphoid cell lines to exclude nonspecificity. A negative and a positive control serum

Table 1. Illness, transfusion history, and presence of antibodies to HTLV-MA in patients with transfusion-associated AIDS. PCP is *Pneumocystis carinii* pneumonia; KS is Kaposi's sarcoma; and NA indicates that no specimen was available for testing.

Patient	Diagnosis	Antibodies to HTLV-MA	Month of		Time from transfusion to illness onset (months)
			AIDS onset	Transfusion	
1	PCP	+	June 1981	November 1977	44
				April 1980*	15
2	PCP	−	April 1982	February 1979	39
				July 1979*	34
				June 1981*	11
3	PCP	NA	April 1982	January 1981	16
4	PCP	+	May 1982	February 1980	28
5	KS	NA	September 1982	May 1981	17
6	PCP	NA	October 1982	December 1979	35
7	PCP	−	November 1982	August 1979	40
8	PCP	−	December 1982	March 1980	34
9	PCP	−	January 1983	August 1981	18
10	PCP	+	January 1983	November 1980	27
11	PCP	NA	January 1983	January 1982	13
12	PCP	−	February 1983	September 1981	18

*Date of receipt of blood from a high-risk donor.

sample were included in each coded test.

Of the 12 patients with transfusion-associated AIDS, 11 had *Pneumocystis carinii* pneumonia and one had Kaposi's sarcoma (Table 1). Only one of the 12 patients had an onset of illness before April 1982. For the ten patients who received transfusions on only one occasion, the interval between receipt of blood or blood components and onset of illness ranged from 13 to 40 months (mean, 24.6 months; median, 22.5 months).

Antibodies to HTLV-MA were detected in three of eight patients for whom serum samples were available (Table 1). Thus, the proportion (37.5 percent) of transfusion-associated AIDS patients who had detectable antibodies to HTLV-MA was very similar to the proportion (36 percent) of seropositive individuals among previously reported patients with AIDS not associated with transfusion (*8*).

The number of donors to the 12 patients described in Table 1 ranged from 2 to 34 (mean, 13.1; median, 6) (Table 2). Of the total of 157 donors, 117 (74.5 percent) were evaluated. The overall prevalence of antibodies to HTLV-MA was significantly higher in the donors to AIDS patients (9 of 117; 7.7 percent) than in random blood donors (1 of 298; 0.3 percent) ($P < 0.0001$; Fisher's exact test) (Table 3). Among the 12 sets of donors to AIDS patients, 9 sets included one donor with antibodies to HTLV-MA.

As reported earlier, most patients with transfusion-associated AIDS had received blood from one or more "high-risk" donors within 5 years before the onset of symptoms in the patient (*6*). A donor was considered to be at high risk if he or she had AIDS, was a member of a group with increased risk for AIDS, or had an abnormally low ratio of T-helper to T-suppressor lymphocytes (< 1.00) ascertained as described (*15*). Donors not meeting these criteria were considered "other" donors. Among the 12 sets of donors to the AIDS patients described in this report, 11 included a high-risk donor; two such donors were found for patient 3 (Table 2). Eight of the 12 high-risk donors belonged to known AIDS risk groups. Although none of the high-risk donors had illnesses that met the surveillance definition for AIDS, 4 of the 12 had generalized lymphadenopathy when examined at the time they were interviewed (19 to 51 months after they had donated blood).

Among the nine donors who showed positive results for antibodies to HTLV-MA, six were classified as high-risk donors (Table 2). These high-risk donors were significantly more likely to have had detectable antibodies to HTLV-MA (6 of 12; 50 percent) than the other donors to these patients (3 of 105; 2.9 percent) ($P < 0.0001$; Fisher's exact test) (Table 3).

The six antibody-positive high-risk donors were the only seropositive persons in their donor sets (Table 2). Five of the six belonged to known AIDS risk groups; the one exception denied belonging to such groups but had generalized lymphadenopathy and a low ratio of T-

Table 2. Prevalance of antibodies to HTLV-MA in blood donors to patients with transfusion-associated AIDS. T_H/T_S is the ratio of T-helper to T-suppressor lymphocytes.

Patient	Donors (No.)	Donors evaluated (No.)	High-risk donors				Other donors (ratio of antibody-positive to total donors)
			AIDS risk group	Generalized lymphadenopathy	T_H/T_S	Antibodies to HTLV-MA	
1	3	3	None	Yes	0.6 (0.7)*	+	0/2
2	28	18	Homosexual man	Yes	0.8 (1.3)	+	0/17
3	20	13	Intravenous drug user	No	1.5 (1.8)	+	0/11
			None	No	0.9 (0.9)	−	
4	6	6	Homosexual man	No	0.5	+	0/5
5	3	1	Homosexual man	No	0.4 (0.7)	+	0/0
6	2	2	Homosexual man	Yes	0.7	−	0/1
7	4	4	Homosexual man who used intravenous drugs	Yes	1.7	+	0/3
8	4	2	None	No	0.6	−	0/1
9	6	3	Homosexual man	No	0.7	−	0/2
10	16	11	None	No	0.3	−	1/10
11	31	28	No high-risk donor identified				1/28
12	34	26	Homosexual man	No	0.6	−	1/25

*The value in parentheses is the result of testing a follow-up specimen (normal ratio, ≥ 1.0).

helper to T-suppressor lymphocytes. Three of the four high-risk donors with generalized lymphadenopathy had detectable antibodies to HTLV-MA.

Of the three antibody-positive "other" donors, two belonged to donor sets that included a seronegative high-risk donor; the third had donated blood to patient 11, for whom no high-risk donor was identified (Table 2). No risk factor for AIDS could be found in these donors, and they had neither signs nor symptoms of AIDS. None had received transfusions within the previous 5 years. These three donors were a 33-year-old married white man, a 22-year-old unmarried white woman, and a 40-year-old married white man.

We have shown that blood donors to patients with transfusion-associated AIDS had a significantly higher prevalence of antibodies to HTLV-MA than a control group of blood donors, and 9 of 12 donor sets studied had at least one seropositive donor. Furthermore, among the donors to the AIDS patients, those identified as possible sources of AIDS transmission on the basis of epidemiologic and immunologic criteria were significantly more likely to be antibody positive than other donors to these patients. The high prevalence of antibodies to HTLV-MA in homosexual male donors was unexpected, since these antibodies were rare in homosexual controls (Table 3) (*8*). This difference contrasts with that expected for other viruses such as Epstein-Barr virus, cytomegalovirus, hepatitis B virus, and other agents, where the background prevalence in homosexual controls is very high (*15*). An unusually high prevalence of antibodies to HTLV-MA in AIDS patients (36 percent), homosexual men with chronic lymphadenopathy (30 percent), and patients with hemophilia (5 to 19 percent), a group at increased risk for AIDS, has been reported (*8, 16*).

The meaning of the higher prevalence of positive results for antibodies to HTLV-MA in certain people who donated blood to AIDS patients is not yet clear. One hypothesis is that this test is measuring antibody to HTLV or a serologically related virus and that this virus is the etiologic agent of AIDS. The donors, although they remain either asymptomatic or develop chronic lymphadenopathy, might transmit the virus though their donated blood to recipients who, after incubation periods of up to 40 months, develop AIDS. Several investigators have suggested that HTLV, apart from any association with the AIDS disease spectrum, may be transmitted through transfusion of blood from asymptomatic carriers of the virus (*17*). This hypothesis is also consistent with the pathogenic pattern of illness associated with another retrovirus, feline leukemia virus, which can be transmitted to cats by blood and can, after a long latency period, produce manifestations ranging from asymptomatic seroconversion to lymphoid malignancy and fatal opportunistic infection (*18*).

Table 3. Prevalence of antibodies to HTLV-MA in blood donors to patients with transfusion-associated AIDS, random blood donors, and healthy homosexual men.

Category	Number tested	Antibody positive	
		Number	Percent
Blood donors to AIDS patients			
High-risk donors	12	6	50.0
Other donors	105	3	2.9
Total	117	9	7.7
Random blood donors			
Philadelphia	100	0	
Madison	99	0	
Tucson	99	1	1.0
Total	298	1	0.3
Healthy homosexual men			
Matched with AIDS patients	81	1	1.2*
Unmatched	45	0	
Total	126	1	0.8

*Result previously published (*8*).

An alternative hypothesis is that certain donors have antibody to HTLV-MA or a serologically related virus, but this virus is not the cause of AIDS. The high-risk donors may have been predisposed to infection with this virus because of their individual practices or because both AIDS patients and the high-risk donors have a particular form of immune suppression, caused by another agent, that makes these individuals especially susceptible to retroviral infection. Immune dysfunction might also have led to a reactivation of latent retroviral infection and, hence, to antibody expression. However, neither apparently healthy homosexual men nor renal-transplant recipients, a patient group with drug-induced immune dysfunction, have an increased prevalence of antibodies to HTLV-MA (Table 3) (*19*). Nonetheless, the occurrence of AIDS in the recipients may have been the result of infection with another, as yet undetected, agent present in the blood of either the high-risk donors or other donors.

Neither hypothesis explains the antibody-positive "other" donors who appear to be outside the groups so far identified as being at risk for AIDS. Further work is required to understand fully the relationship between positive results for antibodies to HTLV-MA and AIDS.

At the present time, no test exists that will specifically screen out blood capable of transmitting AIDS. However, the study of individuals who have donated blood to patients with transfusion-associated AIDS offers an opportunity to examine the transmission of this disease and to evaluate potential "screening tests." Furthermore, study of these donors may help to elucidate the possible role of retroviruses or other viruses in AIDS.

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19. In an unpublished study by M. Essex and M. Hirsch (Massachusetts General Hospital), 2 of 93 kidney transplant patients had antibodies to HTLV-MA when their sera were tested with previously described methods (*8, 16*). In another unpublished study by M. Essex, J. Bailey, and T. Guthrie (Medical College of Georgia), none of 17 patients with systemic lupus erythematosus, a disease of immunoregulatory dysfunction, had positive results for the antibodies.
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Interferon-β–Related DNA Is Dispersed in the Human Genome

Abstract. *Interferon-β_1 (IFN-β_1) complementary DNA was used as a hybridization probe to isolate human genomic DNA clones λB3 and λB4 from a human genomic DNA library. Blot-hybridization procedures and partial nucleotide sequencing revealed that λB3 is related to IFN-β_1 (and more distantly to IFN-α_1). Analyses of DNA obtained from a panel of human-rodent somatic cell hybrids that were probed with DNA derived from λB3 showed that λB3 is on human chromosome 2. Similar experiments indicated that λB4 is not on human chromosomes 2, 5, or 9. The finding that DNA related to the IFN-β_1 gene (and IFN-α_1 gene) is dispersed in the human genome raises new questions about the origins of the interferon genes.*

A single human interferon-β (IFN-β) gene located on human chromosome 9 (designated IFN-β_1), a cluster of IFN-α_1–hybridizing genes on chromosome 9, and a single IFN-γ gene on chromosome 12 have been identified and characterized (*1*). Other studies of human IFN-β production and studies of lengths of translationally (and biologically) active human IFN-β messenger RNA (mRNA) that can be expressed in appropriately induced human-rodent somatic cell hybrids have indicated that functional IFN-β genes are present on human chromosomes 2, 5, and 9 (*2, 3*). Using full-length, sequence-confirmed IFN-β_1 complementary DNA (cDNA) as a hybridization probe, we isolated two human genomic DNA clones, λB3 and λB4, from a human genomic DNA library in λ phage Charon 4A (*4*). The relatedness of λB3 and λB4 to IFN-β_1 was confirmed by additional hybridization tests and, in the case of λB3, by partial nucleotide sequencing. We have now used blot-hybridization procedures to assign λB3 to human chromosome 2. In similar experiments, we found that λB4 is not on human chromosomes 2, 5, 9, or 12.

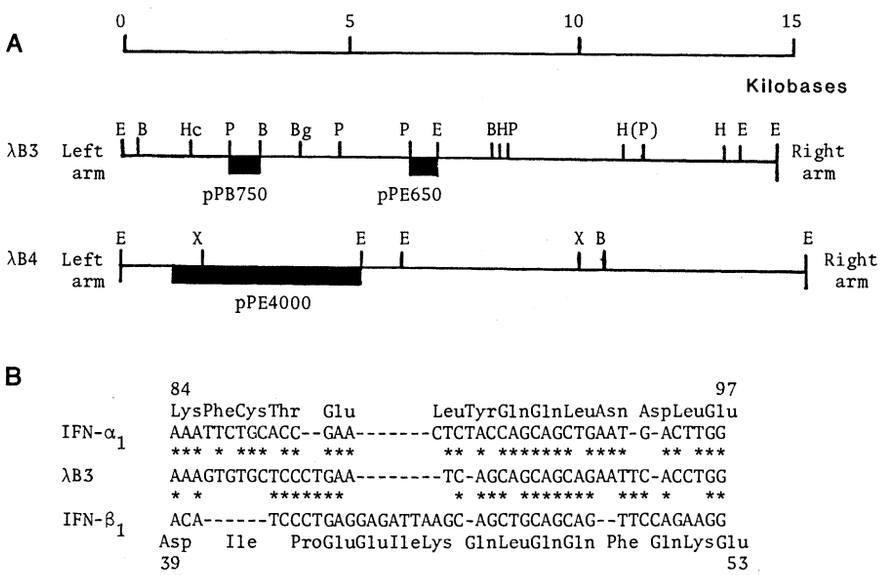

Fig. 1. An example of nucleotide sequence relatedness between λB3, IFN-α_1 and IFN-β_1. (A) Schematic restriction maps of λB3 and λB4. The restriction sites are: *B*, Bam HI; *Bg*, Bgl II; *E*, Eco RI; *H*, Hind III; *Hc*, Hinc II; *P*, Pst I; and *X*, Xba I. Only those sites that can be clearly identified are indicated. The origins of the various relevant subclones are also shown. The origin of the 105-nucleotide pPP-105 subclone of λB3 has not yet been unambiguously determined. (B) The nucleotide sequence of a portion of the pPB-750 area in the map of λB3 compared with the nucleotide and amino acid sequences of IFN-α_1 and IFN-β_1 (*1, 9, 10*). Asterisks indicate nucleotide matches. Amino acid residue numbers correspond to those for the mature proteins.

Interferons have customarily been classifed as α, β, or γ on the basis of their antigenicity and the relatedness of their nucleotide sequences (*1*). For example, IFN-α and IFN-β proteins are not neutralized by antiserums to each other, and IFN-α_1 cDNA probes do not cross-hybridize with IFN-β_1 DNA [the relatedness of their nucleotide sequences is only 43 percent (*1*)]. We discovered that λB3 DNA straddles the IFN-α and -β systems. Although λB3 appears (by serology) to represent an IFN-β gene located on chromosome 2, it cross-hybridizes with IFN-β_1 cDNA (strongly) and IFN-α_1 DNA (weakly). The discovery of DNA that straddles the IFN-α and IFN-β gene families dispersed in the human genome adds a new dimension to the description of the human interferon system.

The inference that there are a number of distinct human IFN-β genes was supported by the detection of translationally active (in *Xenopus laevis* oocytes) human IFN-β mRNA species of different lengths; the IFN-β mRNA species were detected by subjecting polyadenylated RNA obtained from induced human and human-rodent somatic cell hybrids to electrophoresis through agarose–methyl mercury gels (*3, 5*). These studies produced data consistent with the earlier assignment by others (*1*) of the 0.9-kilobase (kb) IFN-β_1 mRNA to chromosome 9 and suggested the assignment of the 1.3-kb IFN-β_2 mRNA to chromosome 5 and 1.8-kb IFN-β_3 mRNA to chromosome 2. Additional translationally active IFN-β mRNA species of lengths 0.35, 0.65, 3, 5, and 8 kb have since then been detected in both induced human lymphoblastoid and fibroblast cells (*5*).

We isolated three distinct human genomic DNA clones from a human genomic DNA library in λ phage Charon 4A that cross-hybridized with IFN-β_1 cDNA (*4*). Two of these (λB3 and λB4) are distinct from each other and from the IFN-β_1 gene, although they both strongly cross-hybridized with IFN-β_1 cDNA. Blot-hybridization tests indicated that poly(I) · poly(C)–induced (I, inosine; C, cytosine) human diploid fibroblasts can contain polyadenylated RNA species 1.8, 3, 5, and 8 kb long that hybridize with λB3 DNA and a 12-kb species that hybridizes with λB4 (*4*). These data suggested that λB3 DNA may reside on chromosome 2 and correspond to the 1.8-kb IFN-β_3 mRNA. We tested this possibility with blot-hybridization analyses of DNA derived from a panel of human-mouse and human-hamster so-