

period there were no socioeconomic or environmental changes.

Eagles⁷ noted that the hearing level of most normal children tended to improve as they grew older, although the differences in decibels were small. In order to exclude the influences of growth and age, we included Pingliang commune—two groups of schoolchildren of the same age range, one tested before iodine prophylaxis and the other tested after prophylaxis for three years—in our study.

Experimentally induced hypothyroid chickens and rodents have endolymphatic hydrops, loss of hair cells, thickening of the tympanic membrane and the middle ear mucous membrane, auditory tubal obstruction, alteration of the middle-ear ossicles, absence of cochlear potential, and general degeneration of the organ of Corti.³ Electrophysiological studies of hypothyroid animals have shown increased hearing thresholds.⁸ Withers et al⁹ studied the effects of hypothyroidism on the hearing of cats and squirrel monkeys that had been behaviourally trained for audiological evaluation before surgical thyroidectomy; postoperatively, hearing loss developed within 120 days and this was alleviated by thyroid hormone replacement. In accord with our results, the study of Meyerhoff¹⁰ suggests that the cochlea is the site of the lesion for sensorineural hearing loss in hypothyroidism. The middle ear changes that have been reported in the past might be responsible for the conductive component.

We propose that the hearing impairment among ostensibly normal schoolchildren in the endemic communes in Guizhou is most probably caused by subclinical hypothyroidism due to prolonged severe iodine deficiency. When iodine intake

becomes sufficient, as the result of long-term iodine prophylaxis, not only is the subclinical hypothyroidism corrected but the mild hearing loss also improves. These findings, together with the improvement in IQ in schoolchildren shown in a controlled trial of iodised oil in a severely iodine deficient area in Bolivia,¹¹ support the broader concept of iodine deficiency disorders (IDD) to denote the effects of iodine deficiency on human growth and development.¹²

Correspondence may be addressed to Dr Wang Yan-You, Department of Otolaryngology, Second Affiliated Hospital, Tianjin Medical College, Tianjin, People's Republic of China.

REFERENCES

1. McCarrison R. Observations on endemic cretinism in the Chitral and Gilgit valleys. *Lancet* 1908; ii: 1275-80.
2. Wang Shi-Xin, Wang Yan-You. Hearing defect and cretinism. *Chin J Otorhinolaryngol* 1964; 10: 79.
3. Meyerhoff WL. The thyroid and audition. *Laryngoscope* 1976; 86: 483-89.
4. De Vos JA. Deafness in hypothyroidism. *J Laryngol Otol* 1963; 77: 390-414.
5. Bhatia PL, Gupta OP, Agrawal MK, Mishr SK. Audiological and vestibular function tests in hypothyroidism. *Laryngoscope* 1977; 87: 2082-89.
6. Rittner FN, Lawrence M. Reversible hearing loss in human hypothyroidism and correlated changes in the chick inner ear. *Laryngoscope* 1960; 70: 393-407.
7. Eagles ELL, Wishik SM, Doerfler LG. Hearing sensitivity and ear disease in children: A prospective study. *Laryngoscope* 1967; (suppl): 1-274.
8. Kohonen A, Jauhainen T, Lievendahl K, Tarkkanen J, Kaimio M. Deafness in experimental hypo- and hyperthyroidism. *Laryngoscope* 1971; 81: 947-56.
9. Withers BT, Reuter SH, Jancke JB. The effects of hypothyroidism on the ears of cats and squirrel monkeys: A pilot study. *Laryngoscope* 1972; 82: 779-84.
10. Meyerhoff WL. Hypothyroidism and the ear: Electrophysiological, morphological, and chemical considerations. *Laryngoscope* 1979; 89: 1-25.
11. Bautista S, Barker PA, Dunn JT, Sanchez M, Kaiser DL. The effects of oral iodized oil on intelligence, thyroid status, and somatic growth in school-aged children from an area of endemic goiter. *Am J Clin Nutr* 1982; 35: 127-34.
12. Hetzel BS. Iodine deficiency disorders (IDD) and their eradication. *Lancet* 1983; ii: 1126-29.

ELISA HTLV RETROVIRUS ANTIBODY REACTIVITY ASSOCIATED WITH MALARIA AND IMMUNE COMPLEXES IN HEALTHY AFRICANS

ROBERT J. BIGGAR
MADS MELBYE
PREM S. SARIN

PAUL L. GIGASE
LUC KESTENS
ANNE J. BODNER

PAUL DEMEDTS WIM J. STEVENS LEOPOLD PALUKU
CHARLES DELACOLLETTE WILLIAM A. BLATTNER

Environmental Epidemiology, National Cancer Institute, Bethesda, Maryland, USA; Institute of Tropical Medicine, Antwerp, and University of Antwerp, Belgium; Institute of Cancer Research, Aarhus, Denmark; Laboratory of Tumor Cell Biology, National Cancer Institute, Bethesda, Maryland, USA; Biotech Inc, Rockville, Maryland, USA; and Katana Hospital, Fomulac, Kivu Region, Zaïre

Summary A serological survey of 250 outpatients in rural Zaïre showed that the prevalence of antibody against HTLV-I, HTLV-II, and HTLV-III, as detected by enzyme-linked immunosorbent assay, correlated strongly with level of antibodies against *Plasmodium falciparum*. The age curve for the prevalence of antibody against these retroviruses and high titres of antibodies against *P falciparum* were similar. Tests with control sera obtained from HTLV-III seropositive homosexual men and American subjects repeatedly infected with malaria who had high antibody titres against *P falciparum* indicated that there was no cross-reactivity between *P falciparum* and these retroviruses. Immune-complex levels, but not IgG, IgM, or IgE levels, also correlated strongly with seropositivity in the ELISA HTLV-I and HTLV-III assay, although immune-

complex-positive control samples were negative. Possible explanations include (a) coincidental distribution paralleling malaria; (b) similar mode of transmission; (c) virus activation and/or enhanced antibody production due to the effect of malaria on the immune system; and (d) false-positive reactivity in the ELISA assay due to cross-reactive antibodies or other unknown factors.

Introduction

WITHIN the past 5 years, three types of human T-lymphotropic virus (HTLV) have been identified. HTLV-I is associated with a malignancy known as adult T-cell lymphoma/leukaemia (ATLL), most commonly seen in Japan¹ and the Caribbean islands.² HTLV-II has not yet been linked with any specific disease.³ HTLV-III is associated with the acquired immunodeficiency syndrome (AIDS),^{3,4} which is now epidemic in North America⁵ and Europe.⁶ **HY OF AFRICAN AIDS**

In the early 1980s, African cases of AIDS were becoming recognised in hospitals in Europe.⁶ Investigations in Africa revealed AIDS cases in Kigali, Rwanda,⁷ and Kinshasa, Zaïre,⁸ which led to studies of the prevalence of HTLV-III in Africa. In a remote area of eastern Zaïre and six different areas of Kenya, prevalence of HTLV-III antibodies detected with an enzyme-linked immunosorbent assay (ELISA) varied from 6 to 50%.^{9,10} Yet few AIDS cases have been reported from either eastern Zaïre or Kenya,^{9,10} and healthy seropositive Africans in these areas are immunologically competent.¹¹ The areas of high HTLV-III antibody prevalence in Kenya were generally those with high malaria parasitaemia prevalence.¹⁰ We have previously shown that *Plasmodium*

HTLV area = malaria + area

D

and WD NON-YES IN AFFILIATES

falciparum antibody titres correlated with the prevalence of ELISA HTLV-I reactivity in Ghana.¹² We therefore investigated the relationship between *P falciparum* antibodies and HTLV-I, HTLV-II, and HTLV-III antibodies in eastern Zaïre.

Methods

The study was undertaken at a rural hospital in Kivu District, eastern Zaïre, during May/June, 1984. The structure of the study and the laboratory methods have been described elsewhere.^{9,13} 250 patients presenting for routine outpatient care during one week were studied; none had recognised clinical manifestations of AIDS. The ELISA results were categorised as positive when the absorbance ratio of the sample was 3 or more times background, borderline when it was between 3 and 5 times, and negative when it was less than 3 times. 23 sera representing positive and negative ELISA results were examined by means of western blot analysis; 12 (86%) of 14 positive sera reacted to HTLV-III-specific bands, whereas 8 (89%) of 9 negative sera did not react. To supplement retrovirus information from the previous study,⁹ sera from the same 250 outpatients were examined for antibodies against HTLV-I and HTLV-II, also with ELISA techniques.¹³ In accordance with previous studies,¹² HTLV-I and HTLV-II results were categorised as positive when the absorbance was at least 5 times background, and borderline when it was between 2 and 5 times.

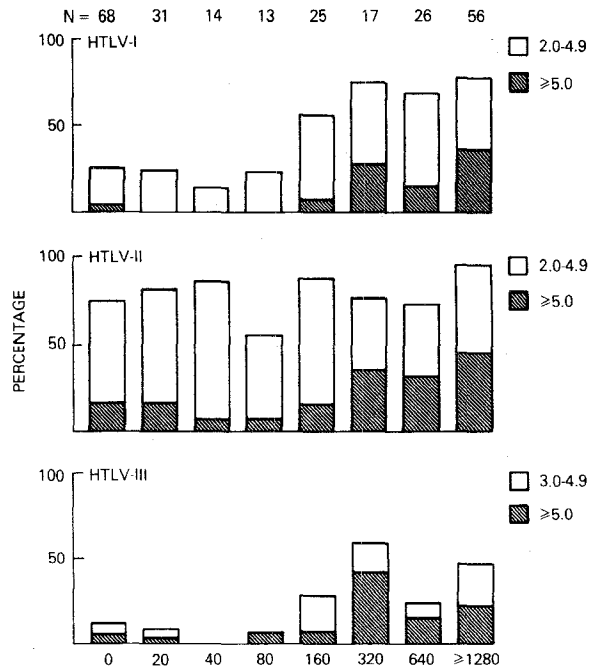
The sera from these subjects were also examined for antibodies against *Plasmodium falciparum* by means of an indirect immunofluorescent antibody test¹⁴ using antigen prepared from cultured *P falciparum*.¹⁵ In addition, thick and thin smear preparations obtained from a random subset of these subjects were examined to determine the prevalence of parasitaemia. The relationship between *P falciparum* antibodies and human retrovirus antibodies was examined by correlation coefficients (*r*) and probabilities (*p*). To determine what proportion of the variability in the retrovirus assay was explained by the various factors, we used general linear models, including each element shown to be significant (*p*<0.05) in any of the retrovirus models.

To exclude non-specific reactivity, sera from 20 Danish homosexual men, half strongly seropositive in the HTLV-III ELISA test and half seronegative, were screened for reactivity in the assay for *P falciparum* antibodies. In addition, sera obtained in 1962-63 from 16 American volunteers given recurrent bouts of *P falciparum* malaria during a therapy protocol for neurosyphilis were tested with ELISA for HTLV-III and HTLV-I and by means of western blot analysis. These last sera all had high titres of antibodies against *P falciparum*.

Results of immunological tests from 93 patients with Kaposi's sarcoma and controls obtained during the same field trip and at the same hospital as the outpatient survey were also available. There were no significant differences between patients with Kaposi's sarcoma and controls in either prevalence of reactivity in the HTLV-III ELISA assay¹⁶ or in any of many immunological studies.¹⁷ That study included testing for immunoglobulins and immune complexes with sera carefully collected especially for that purpose and frozen in liquid nitrogen, according to methods previously described.¹⁸ These results were reviewed to determine if levels of IgG, IgM, IgE, or immune complexes with IgG or IgE correlated with the likelihood of seropositivity in the ELISA assay for HTLV-III. Since sera to be analysed for immune complexes require prompt separation and freezing, the American sera and the sera from the Zaïre outpatient survey were not tested for immune complexes.

Results

The 250 patients who agreed to take part in the study represent 82% of those who attended the clinic during the week in question. The demographic characteristics of the refusers did not differ from those of the participants.⁹ The mean age of the participants was 32 years (range 8-78), and 56% were female. Of 233 adults (age >14 years), 46% were



RECIPROCAL OF ANTIBODY AGAINST PLASMODIUM FALCIPARUM
 Fig 1—Antibody against *P falciparum* versus reactivity in ELISA assay for antibodies against HTLV-I, HTLV-II, and HTLV-III in sera from 250 residents of eastern Zaïre.

agricultural workers, 41% had never attended school, and 67% lived in rural areas.

The prevalence of malarial parasitaemia was 13%. However, 72% of patients had antibodies against *P falciparum*, half of them having titres $\geq 1:160$ (fig 1). Factors contributing to the prevalence of antibody titre (including seronegative persons) were increasing age, residence in a rural area, and measures of poverty (table 1). Together these factors explained 11.6% of the variability observed.

The proportions of subjects positive in the ELISA test for HTLV-I, HTLV-II, and HTLV-III were 14%, 25%, and 12%, respectively. Demographic associations for all three retroviruses were similar (table 1). When the titre of

TABLE 1—REGRESSION MODELS FOR THE DEPENDENT VARIABLES RELATED TO HTLV-I, HTLV-II, HTLV-III ELISA POSITIVITY AND MALARIA ANTIBODY AMONG RESIDENTS OF EASTERN ZAIRE*

	HTLV-I	HTLV-II	HTLV-III	<i>P falciparum</i> titre
<i>ELISA reactivity</i>				
Negative	130	48	189	
Borderline	85	140	30	
Positive	35	62	31	
<i>Factors separately:</i>				
Age	0.7%†	0.2%	0.0	1.3%†
Rural residence	2.3%‡	2.0%‡	2.9%‡	6.4%§
Agricultural work	0.1%	0.4%	0.5%	3.1%‡
Lack of schooling	1.2%†	1.1%	1.5%†	2.9%‡
Malaria antibody titre	23.4%§	7.2%§	10.9%§	-
<i>Factors excluding malaria</i>	5.1%	2.7%	4.9%	11.6%
<i>Factors including malaria</i>	24.5%	8.3%	13.8%	-

If borderline sera are excluded, the complete model fits better for HTLV-I (31.3%) and HTLV-II (17.9%), because of a stronger univariate relationship to malaria antibody titre (30.2% and 14.6% respectively). If borderline sera are excluded, the complete model for HTLV-III fits nearly as well (12.3%), having a slightly lower univariate relationship to malaria antibody titre (9.1%). *Percentage indicates the degree of variability accounted for. †*p*<0.05; ‡*p*<0.01; §*p*<0.0001.

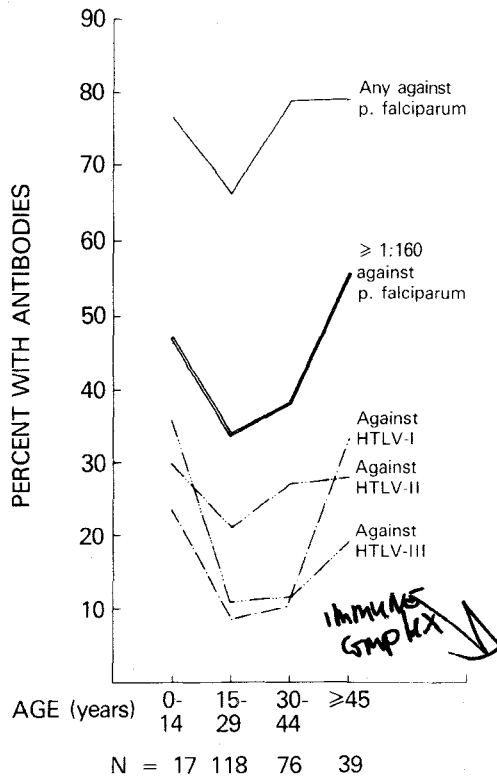


Fig 2—Age-specific prevalence of antibodies against *P falciparum*, high-titre ($\geq 1:160$) antibodies against *P falciparum*, and ELISA reactivity ≥ 5.0 in the assays for antibodies against HTLV-I, HTLV-II, and HTLV-III among 250 residents of eastern Zaïre.

antibodies against *P falciparum* was considered as an additional factor in explaining the distribution of antibodies against all three types of retroviruses, this single factor dominated all others, to the extent that none was independently significant (table 1). *P falciparum* antibody titre alone accounted for 23%, 7%, and 11% of the variability seen in HTLV-I, HTLV-II, and HTLV-III respectively.

The relation between antibody titres against *P falciparum* and reactivity in the ELISA system for HTLV-I, HTLV-II, and HTLV-III is shown in fig 1. The probability of having

TABLE II—IMMUNOGLOBULIN AND IMMUNE COMPLEX LEVELS COMPARED TO REACTIVITY IN THE ELISA ASSAY FOR HTLV-III AND HTLV-I AMONG RESIDENTS OF EASTERN ZAIRE

Elisa reactivity	IgG (mg/dl)	IgM (mg/dl)	IgE (kU/l)	IgG immune complexes (TU/ml)	IgE immune complexes (TU/ml)
HTLV-III:					
Negative (n=58)	2743±958	365±475	6126±4873	0.23±0.40	6.8±8.1
Borderline (n=27)	2867±837	491±684	7594±5195	0.88±0.39	11.2±10.3
Positive (n=9)	3200±431	700±682	7607±5550	1.65±0.63	18.5±11.3
r	0.14	0.18	0.14	0.51	0.38
p	0.16	0.08	0.20	<0.0001	0.0003
HTLV-I:					
Negative (n=31)	2622±1030	275±179	7666±5314	0.56±0.36	5.1±5.0
Borderline (n=42)	3014±903	473±666	6096±4949	0.79±0.55	10.4±11.3
Positive (n=20)	2783±485	609±691	6659±4863	1.19±0.65	13.1±9.9
r	0.08	0.29	-0.08	0.38	0.36
p	0.44	0.005	0.45	0.0002	0.0006

reactivity ≥ 5.0 in the ELISA assay against each retrovirus rose significantly with rising antibody titres against *P falciparum* (HTLV-I, $r=0.35$, $p<0.0001$; HTLV-II, $r=0.27$, $p<0.0001$; HTLV-III, $r=0.28$, $p<0.0001$). In addition, the age-specific profile of antibody prevalence was similar for antibodies against *P falciparum* and all three retroviruses (fig 2).

None of the sera from the 10 Danish homosexual men known to be strongly seropositive in the HTLV-III assay had antibodies against *P falciparum*. Of the 16 Americans with recurrent malaria, 1 had an ELISA ratio for HTLV-III considered positive (5.3 times background), but 9 others had ratios in the borderline range. These 16 sera were subjected to western blot analysis for HTLV-III, and 4 subjects had a 1+ or 2+ reaction (on a scale of 0 to 4+) at the P24 polypeptide band but no other bands. 1 other person had a 1+ reaction at the P15 band and 1 at the P55 band. In contrast, sera from the positive control for the same tray, an AIDS patient, showed 3+ or 4+ reactions at all the major viral protein bands.

Among the Kaposi's sarcoma patients and controls who had been enrolled in the immunological studies protocol at the Fomulac Hospital, eastern Zaïre, 9 were seropositive, 27 were borderline, and 58 were negative in the ELISA test for HTLV-III. The probability of being reactive (both including and excluding the borderline values) increased significantly with increasing immune complexes (table II). Ultracentrifugation of seropositive samples with high immune-complex levels had no major impact on reactivity. An agglutinated human IgG preparation (from American subjects) used as a standard positive control for immune-complex tests was negative in the ELISA test when tested at 1:20 (standard serum dilution) through 1:1000 dilutions.

Discussion

The association between titre of *P falciparum* antibodies and antibody prevalence against HTLV-I, HTLV-II, and HTLV-III emerged as overwhelmingly important in each case. After adjustment for titre of antibodies against *P falciparum*, none of the previously associated variables was independently significant. This association was not directly attributable to cross-reactivity between *P falciparum* and retroviruses, since HTLV-III-seropositive European homosexual men were negative in the assay for antibodies against *P falciparum* and since sera from the American subjects were generally negative in western blot analysis for HTLV-III despite having high antibodies against *P falciparum*.

There are several possible explanations for these findings: 1. Infection with malaria and all three HTLV retroviruses could be unrelated but share similar environmental factors such as those related to increasing age and rural poverty. It is unlikely, however, that the age-specific prevalence distribution, which is strikingly similar for malaria and the three retrovirus infections, has the same explanation in all instances. The frequency of high-titre antibodies against *P falciparum* in this population was lowest in adults aged 15-44, which may be due to the greater use of antimalarial drugs in this age range.

2. The human retroviruses could be transmitted by mosquitoes or within the parasite itself. Intraparasite transmission of a retrovirus has been suggested by electron microscopic studies of *Sparganum proliferum*, a parasite of cows.¹⁹ A possible role for parasites is suggested by the observed association between HTLV-I seropositivity and malaria in Ghana,²⁰ filariasis in Japan,²⁰ Chagas disease in

High positivity of malaria parasites

1316841

Venezuela,²¹ and strongyloidiasis in Okinawa,²² and between both HTLV-I and HTLV-III and malaria, schistosomiasis, and tropical splenomegaly in Kenya.¹⁰ However, these parasitic diseases have different vectors or no vector at all.

3. Malaria and other parasitic diseases could act indirectly by influencing host immune response. Malaria has been shown to cause a transient inversion of the T-helper/T-suppressor ratio and non-specific B-cell proliferation.²³ If many patients harboured retrovirus infections that were repressed by a competent immune system, then perhaps expression of the virus and production of antibodies might be transient and related to malaria. On this hypothesis, HTLV antibody levels in the same individual should fluctuate over time. Evidence that HTLV-III antibody levels can fluctuate in homosexual men has been presented.²⁴ All three retroviruses were similarly related to titre of antibodies against *P. falciparum* in this study. If this relationship is secondary to an effect on immunity, the effect is not retrovirus type-specific.

4. Finally, reactivity in both ELISA and western blot analysis may be non-specific in healthy Africans. There is no doubt that AIDS does occur in parts of Africa and that patients with the disease will be seropositive in the HTLV-III ELISA and western blot tests and will have reactivity similar to that of AIDS patients in America and Europe. However, we have emphasised in previous studies that the profile of reactivity in the western blot analyses of ELISA-positive healthy subjects does not appear typical of seropositive American and European subjects and speculated that such reactivity may be related to other, as yet unknown, retroviruses in this environment.^{9,10} Alternatively, if unknown serum factors cause immunoglobulins to adhere non-specifically to viral proteins, the effect might be seen in both ELISA and western blot analysis, since both tests use the same viral antigen preparation. High immune-complex levels can occur in patients with malaria and other parasitic diseases,²⁴ and in this study they were associated with HTLV-III seropositivity. However, neither immune complexes nor high levels of immunoglobulins are likely to be the cause of non-specific reactivity, since the standard preparation of agglutinated IgG used as the positive control in immune-complex tests was negative in the ELISA tests. Furthermore, sera from Asian and South American areas with malaria and other parasitic diseases are rarely reactive in the ELISA for HTLV-III (unpublished data).

If the human retrovirus reactivity observed in the ELISA test is frequently non-specific among Africans, the causes of the non-specificity need to be clarified in order to determine how they might affect the seroepidemiology of retroviruses in areas other than Africa, such as the Caribbean and Japan. Serological studies from Africa would also need to be re-evaluated with a more specific test before conclusions can be drawn. If, however, the ELISA reactivity is specific for HTLV-I, HTLV-II, and HTLV-III or for a related and cross-reactive retrovirus, then the relationship between malaria and these viruses needs further exploration.

Correspondence should be addressed to R. J. B., Landow Building 3C19, NIH, Bethesda, Maryland 20205, USA.

REFERENCES

1. Popovic M, Reitz MS Jr, Sarngadharan MG, et al. The virus of Japanese adult T-cell leukemia is a member of the human T-cell leukemia virus group. *Nature* 1982; **300**: 63-66.
2. Blattner WA, Kalyanaraman M, Robert-Guroff M, et al. The human type-C retrovirus HTLV in Blacks from the Caribbean region and relationship to adult T-cell leukemia/lymphoma. *Int J Cancer* 1982; **30**: 257-64.

3. Sarin PS, Gallo RC. Human T lymphotropic retroviruses in adult T-cell leukemia/lymphoma and acquired immune deficiency syndrome. *J Clin Immunol* 1984; **4**: 415-23.
4. Barre-Sinoussi F, Chermann JC, Rey F, et al. Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS). *Science* 1983; **220**: 868-70.
5. Allen JR. Epidemiology—United States. In: Ebbesen P, Biggar RJ, Melbye M, eds. AIDS, a guide to clinicians. Copenhagen: Munksgaard/Saunders, 1984.
6. Melbye M, Biggar RJ, Ebbesen P. Epidemiology—Europe and Africa. In: Ebbesen P, Biggar RJ, Melbye M, eds. AIDS, a guide to clinicians. Copenhagen: Munksgaard/Saunders, 1984.
7. Perre P, Rouvroy D, Lepage P, et al. Acquired immunodeficiency syndrome in Rwanda. *Lancet* 1984; **ii**: 62-65.
8. Piot P, Quinn TC, Taelman H, et al. Acquired immunodeficiency syndrome in a heterosexual population in Zaïre. *Lancet* 1984; **ii**: 65-69.
9. Biggar RJ, Melbye M, Kestens L, et al. The seroepidemiology of HTLV-III antibodies in a remote population of eastern Zaïre. *Br Med J* 1985; **290**: 808-10.
10. Biggar RJ, Johnson BK, Oster C, et al. Regional variation in prevalence of antibody against human T-lymphotropic virus types I and III in Kenya, East Africa. *Int J Cancer* (in press).
11. Kestens L, Biggar RJ, Melbye M, Bodner AJ, De Feyter AJ, Gigase PL. Absence of immunosuppression in healthy subjects from eastern Zaïre who are positive for HTLV-III antibodies. *N Engl J Med* 1985; **312**: 1517-18.
12. Biggar RJ, Saxinger C, Gardiner C, et al. Type-I antibody in urban and rural Ghana, West Africa. *Int J Cancer* 1984; **34**: 215-19.
13. Saxinger C, Gallo RC. Application of the indirect enzyme-linked immunosorbent assay microtest to the detection and surveillance of human T-cell leukemia-lymphoma virus. *Lab Invest* 1984; **49**: 371-77.
14. Sulzer AJ, Wilson M, Hall EC. Indirect fluorescent-antibody tests for parasitic diseases. *Am J Trop Med Hyg* 1969; **18**: 199-205.
15. Hall CL, Haynes JD, Chulay JD, Diggs CL. Cultured *Plasmodium falciparum* used as an antigen in a malaria indirect fluorescent antibody test. *Am J Trop Med Hyg* 1978; **27**: 849-52.
16. Biggar RJ, Melbye M, Kestens L, et al. Kaposi's sarcoma in Zaïre is not associated with HTLV-III infection. *N Engl J Med* 1984; **311**: 1051-52.
17. Kestens L, Melbye M, Biggar RJ, et al. Endemic African Kaposi's sarcoma is not associated with immunodeficiency. *Int J Cancer* (in press).
18. Stevens WJ, Bridts C. IgG-containing and IgE-containing circulating immune complexes in patients with asthma and rhinitis. *J Allergy Clin Immunol* 1984; **73**: 276-82.
19. Mueller JF, Strano AJ. *Sparganium proliferum*, a sparganium infected with a virus? *J Parasitol* 1974; **60**: 15-19.
20. Tajima K, Fujita K, Tsukidate S, Oda T, Tominaga S, Suchi T, Hinuma Y. Seroepidemiological studies on the effects of filarial parasites on infestation of adult T-cell leukemia virus in the Gato Islands, Japan. *Gann* 1983; **74**: 8-191.
21. Merino F, Robert-Guroff M, Clark J, Blattner WA, Gallo RC. Natural antibodies to human T-cell leukemia/lymphoma virus in healthy Venezuelan populations. *Int J Cancer* 1984; **34**: 501-06.
22. Nakada K, Kohakura M, Komoda H, Hinuma Y. High incidence of HTLV antibody in carriers of *Strongyloides stercoralis*. *Lancet* 1984; **i**: 633.
23. Whittle HC, Brown J, Marsh K, Greenwood BM, Seidlin P, Tighe H, Wedderburn L. T-cell control of Epstein-Barr virus-infected B cells is lost during *P. falciparum* malaria. *Nature* 1984; **312**: 449-50.
24. Schupbach J, Haller O, Vogt M, et al. Antibodies to HTLV-III in patients with AIDS and pre-AIDS and in groups at risk of AIDS. *N Engl J Med* 1985; **312**: 265-70.
25. Mohammed I. The role of immune complexes in human malaria and some of its complications. *J Infect* 1982; **4**: 97-104.

"Fears concerning non-sexual transmission have led not just to the isolation and rejection of AIDS patients but to renewed attacks on homosexuality as well. A psychologist testifying before the Texas legislature, argued for a bill to incarcerate homosexuals 'until and unless they can be cleansed of their medical problems'. Venereal disease has historically been assumed to be the disease of the 'other'; this is particularly true in the case of AIDS. Unlike herpes, which is viewed as a disease of deviant sexuality, a problem of kind rather than degree.

"Although AIDS has led to a critical reassessment of sexual practices within the homosexual community, calling into question patterns of promiscuous relations, it has been used by some outside the gay community to call homosexuality itself into question. Some critics have suggested that AIDS proves conclusively that homosexuality is deviant, unhealthful, and a threat to the dominant heterosexual community. Criticizing federal allocations for medical research on AIDS, Ronald S. Godwin, an executive of the fundamentalist political sect, the Moral Majority, explained, 'What I see is a commitment to spend our tax dollars on research to allow these diseased homosexuals to go back to their perverted practices without any standards of accountability.' The assumption that AIDS is the result of bad behavior pervades the assessment of the problem. In this view, it is homosexuality that causes AIDS, not a virus."—ALLAN M. BRANDT. No magic bullet: a social history of venereal disease in the United States since 1880. Oxford: Oxford University Press, 1985: 245.