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RETROVIRAL INFECTIONS (HIV-1, HIV-2, AND HTLV-I) IN RURAL NORTHWESTERN TANZANIA

CLINICAL FINDINGS, EPIDEMIOLOGY, AND ASSOCIATION WITH INFECTIONS COMMON IN AFRICA

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During a three-week period in March/April 1987, the authors examined 253 consecutive patients referred to a rural hospital in northwestern Tanzania. Sera were tested for antibodies to human immunodeficiency virus type 1 (HIV-1), human immunodeficiency virus type 2 (HIV-2), and human T-lymphotropic virus type I (HTLV-I), as well as for various parasites, hepatitis B virus, and *Treponema pallidum*. Neopterin (urinary and serum) was chosen as the immunologic parameter. In eight of the 253 patients (3.2%), a clinical diagnosis of acquired immunodeficiency syndrome (AIDS) was established. Three of the AIDS patients had HIV-1 antibodies, two had HIV-1 antigen, one had both HIV-1 and HIV-2 antibodies, and in one patient, only HIV-2 antibodies were found. The total HIV-1 and HIV-2 seroprevalence (antibodies plus antigen) was 4.3%; HTLV-I seroprevalence was 9.9%. No correlation could be found between HIV (or HTLV-I) seropositivity and raised levels of antibody to the above pathogens. There was, however, a significantly positive correlation between HIV seropositivity and history of gonorrhea, whereas a history of operations, injections, vaccinations, blood transfusions, or scarification did not influence the level of HIV seropositivity. The most frequently noted epidemiologic association with HIV seropositivity was traveling to or coming from Uganda or Rwanda. Two thirds of the studied Tanzanians had elevated neopterin levels, and all seven HIV-seropositive patients with clinical signs of AIDS had extremely high serum and urinary neopterin levels compared with HIV-seropositive patients without signs of AIDS. Increased neopterin levels reflect a stimulation of the T-cell/macrophage system.

acquired immunodeficiency syndrome; HIV; HIV seropositivity; human T-cell leukemia virus; retrovirus infections

There are now more than 100,000 cases of acquired immunodeficiency syndrome (AIDS) throughout the world (41).

Seroprevalence rates of human immunodeficiency virus type 1 (HIV-1) among healthy populations in African countries

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Abbreviations: AIDS, acquired immunodeficiency syndrome; ELISA, enzyme-linked immunosorbent assay; HIV-1, human immunodeficiency virus type 1; HIV-2, human immunodeficiency virus type 2; HTLV-I, human T-lymphotropic virus type I.

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range from 0.7 per cent for blood donors in Zaire to as high as 18 per cent in Kigali, Rwanda (1). It appears that Central Africa and adjacent areas of East, West, and Southern Africa are currently the most severely affected by HIV infection.

In parts of West Africa, human immunodeficiency virus type 2 (HIV-2) has been shown to cause a disease similar to HIV-1-induced AIDS (2). Another retrovirus, human T-lymphotropic virus type I (HTLV-I), is responsible for a rare form of human T-cell leukemia (3) and has been associated with a neurologic disorder called tropical spastic paraparesis (4). This virus also shows greatly differing prevalence rates in various parts of Africa (5, 6).

Heterosexual bidirectional transmission is thought to be the most important mode of HIV transmission in Africa. Seropositivity is reported to be associated with concurrent sexually transmitted diseases (7–10). Vertical transmission from mother to child is important in Africa (11–13). Injections, blood transfusions, and even scarification have been incriminated as sources of HIV transmission (11, 14, 15).

Many seroepidemiologic HIV prevalence studies are hospital-based and therefore might be biased. By screening the blood of all people seen in the emergency room of a rural hospital, we were able to obtain more representatively distributed HIV prevalence rates (16–18). However, there is still some bias inherent in this group, since the patients were ill enough to come to the hospital and too ill to be treated by the medical assistant.

This study was undertaken in a purely rural area of East Africa to gain more information about seroprevalence in an unselected nonurban population, to evaluate various risk factors for HIV infection in this particular area, and to study the interdependence of various parasitic diseases, activation of cell-mediated immunity, and the development of AIDS.

Subjects and methods

A rural hospital serving a population of about 50,000 was chosen in Karagwe District, Bukoba Region, Tanzania. This location borders, within sight, on Rwanda and is only about 80 km (50 miles) south of the Ugandan border. This hospital is staffed with one medical officer and one medical assistant. All patients suffering from serious diseases beyond the medical capability and range of a medical assistant are referred to the medical officer. During a three-week period in March/April 1987, 253 patients were examined by one of the authors (E. S.) according to a previously fixed schedule; informed consent was obtained from all patients. Each patient's history was taken by questionnaire, 10 ml of blood was drawn, and 2 ml of urine was collected.

The questionnaire comprised information on age, sex, family status, occupation, residence, journeys, and movements either within the district, region, or country or into neighboring countries. Patients were asked about previous venereal diseases such as syphilis and gonorrhea. In addition, the patients were questioned regarding previous operations, injections, vaccinations, blood transfusions, hospital admissions, and scarification. History of weight loss of 10 per cent or more, fever, cough, or diarrhea persisting for more than one month was recorded.

Examination consisted of physical inspection, auscultation, percussion of the lung and heart, and palpation of the liver and spleen. Lymph nodes were palpated and lymph node involvement was classified according to the following categories: 1) no lymph nodes palpable; 2) only inguinal lymph nodes affected; 3) one or two extrainguinal (with or without inguinal)

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lymph nodes affected; 4) three or more extrainguinal (with or without inguinal) lymph nodes affected; and 5) generalized lymphadenopathy. Weight and height were measured and were used to calculate Quetelet index (weight (kg)/height $(m)^2$) for assessment of nutritional status. Finally, a neurologic examination was conducted and results were judged as 1) normal, 2) showing exclusively peripheral nerve involvement, 3) showing exclusively central nervous system involvement, or 4) showing both central and peripheral nervous system involvement.

Illness diagnoses were made on clinical grounds, based on history, signs, and symptoms. Local laboratory facilities were extraordinarily limited. Blood slide examination, red blood cell and white blood cell counts, measurement of the erythrocyte sedimentation rate, urine analysis, simple analysis of cerebrospinal fluid, and examination of stools and urine for parasites and ova were possible. Ziehl-Neelsen, Giemsa, and Gram staining were done. X-ray of bones and chest was possible on only one day out of the week because of a limited supply of electricity.

The blood and urine specimens were collected and stored in a refrigerator at 4 C. They were then transported in a cooler to the University Hospital of Innsbruck (Innsbruck, Austria), where serologic testing was carried out. Antibodies to HIV-1 were determined by a recombinant screening enzyme-linked immunosorbent assay (ELISA) (Abbott, Vienna, Austria) which uses plates coated with recombinant p 24 and p 41. Antibody-reactive sera were confirmed by Western blot analysis using complete virus (E.I. du Pont de Nemours, Bad Nauheim, Federal Republic of Germany) and recombinant p 41 developed by us (19). HIV-1 antigen was determined by ELISA (Abbott). Antigen-reactive sera were retested and simultaneously neutralized by an antibody against HIV-1. Antibodies to HIV-2 were tested by ELISA (Diagnostic Pasteur, Marnes La Coquette, France). Positive samples were confirmed by Western blot analysis (Pasteur). Antibodies to HTLV-I were determined by a commercially available ELISA (du Pont). Repeatedly reactive sera were considered positive.

Antibodies against *Toxoplasma gondii* and hepatitis B core antigen were determined by ELISA (Abbott). Hepatitis B core antigen-reactive sera were further investigated for hepatitis B surface antigen. The presence of antibodies against *Plasmodium falciparum* was determined by an indirect immunofluorescence test using infected erythrocytes as substrate. Detectable fluorescence at a dilution of 1:40 was considered positive. The presence of antibodies against *Trypanosoma rhodesiense*, *Leishmania donovani*, and *Entamoeba histolytica* was determined by indirect immunofluorescence assay. A reaction at a dilution of 1:100 was considered positive. Neopterin levels in serum and urine were determined as reported previously (20).

Demographic data

There were 126 males (49.8 per cent) and 127 females (50.2 per cent) in the study, with a median age of 31 years (range, 1–78 years; interquartile range, 22–45 years). Sixteen patients (6.3 per cent) were children aged less than 15 years. Of the 237 patients older than age 15 years, 46 (19.4 per cent) were single, 167 (70.5 per cent) were married, and 18 (7.6 per cent) were either divorced or widowed. Six men (2.5 per cent) were polygamous.

Most of the patients (183; 72.3 per cent) were farmers and/or housewives. Fifteen (5.9 per cent) were traders; 29 (11.5 per cent) were professionals (e.g., clerks, nurses, masons, carpenters, or teachers); and 26 (10.3 per cent) were young children and primary and secondary school students.

The majority of the patients (180; 71.1 per cent) were residents of the purely rural catchment area of the hospital, having never left their village or their district. Forty patients (15.8 per cent) had moved within Tanzania without ever leaving the country. Thirty-three (13 per cent) were

either traders who frequently visited the neighboring countries (particularly Rwanda or Uganda), soldiers that had been to Uganda for various lengths of time, or refugees from Rwanda or Uganda that had settled in the area.

Statistics

Nonparametric statistical methods were used since the data usually were not normally distributed. Differences of median values between groups were assessed by the Kruskal-Wallis t test. Differences of frequencies between groups were tested by Pearson's chi-square test or, if possible, by Fisher's exact test. In the case of naturally ordered categories, an additional chi-square test was performed for linear trend of proportions.

RESULTS

Retroviral serology and its epidemiologic association

The prevalence rate of the AIDS-causing retroviruses HIV-1 and HIV-2 in the population was 4.3 per cent. In 25 sera (9.9 per cent), HTLV-I antibodies could be found. In six sera (2.4 per cent), antibodies to HIV-1 were detected by ELISA and by Western blot; in two (0.8 per cent) HIV antibody-seronegative patients, HIV-1 antigen could be detected. Two patients (0.8 per cent) had HIV-2 antibodies, and one patient (0.4 per cent) reacted positively in both the HIV-1 and the HIV-2 antibody test. Results of confirmatory Western blotting (HIV-2) are shown in figure 1.

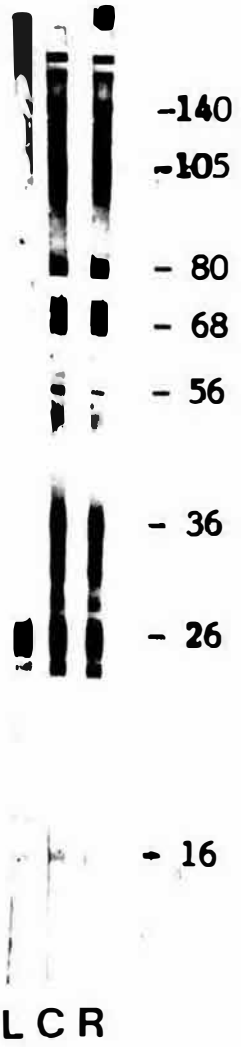

FIGURE 1. Western blot, using purified virus-cultured human immunodeficiency virus type 2 (HIV-2) antigen, of the sera of several patients referred to a rural hospital in Karagwe District, Bukoba Region, Tanzania, 1987. Left (L), typical cross-reaction of human immunodeficiency virus type 1-positive serum with the p 26 of HIV-2; center (C), HIV-2-positive serum of a patient with acquired immunodeficiency syndrome; right (R), HIV-2-positive control serum. Numbers at right, kilodaltons.

The more sexually active younger patients (aged 16–45 years) had a higher prevalence of HIV (5.1 per cent) than did the older patients (aged 46–78 years) (3.3 per cent). This difference, however, was not significant ($p = 0.59$; Pearson chi-square test). Since no HIV antibodies were detected in subjects younger than age 15 years, this group was omitted in subsequent statistical calculations.

There was a weak, nonsignificant (one-tailed Fisher's exact test; $p = 0.11$) preponderance of HIV prevalence among males (6.7 per cent vs. 2.5 per cent seropositivity). HTLV-I antibodies were evenly distributed throughout all ages of either sex. HIV-1, HIV-2, and HTLV-I antibodies were evenly

distributed throughout the various types of family status.

A highly significant difference could be observed between patients residing within the district and those coming from or having been to northwesternly bordering countries; almost one in five Uganda- or Rwanda-linked subjects was HIV-1- or HIV-2-seropositive, whereas for those who denied having ever left Tanzania, the ratio was approximately one in 45 ($\chi^2 = 10.77$, $p = 0.0046$). One woman suffering from HIV-2-associated AIDS had come to the area from Rwanda only six months before the study. The other HIV-2-positive patient was a permanent resident of a neighboring village, and the patient who was both HIV-1- and HIV-2-seropositive had had, before his illness, frequent links to southern Uganda as a trader. No difference could be found in HTLV-I seroprevalence with regard to the various geographic origins of patients. None of the HTLV-I antibody-positive patients had HIV-1 or HIV-2 antibodies or HIV-1 antigen.

No common variables that are frequently associated with parenteral transmission of viruses (e.g., blood transfusions, scarification, etc.) were correlated with HIV-1, HIV-2, or HTLV-I seropositivity (univariate analysis by one-tailed Fisher's exact test).

Chronic fever, weight loss, diarrhea, and persistent cough for more than one month were significantly associated with HIV-1 and HIV-2 seropositivity. No association was found between these variables and HTLV-I seropositivity.

Questioning on previous sexually transmitted diseases revealed a higher proportion of HIV-1 and HIV-2 seropositivity in those who reported having had gonorrhea (12.2 per cent vs. 3.3 per cent; Fisher's exact test, $p = 0.0313$). No association could be found between any of these variables and HTLV-I seropositivity.

Clinical findings

Table 1 shows the clinical diagnoses for the 253 patients, partly confirmed by the laboratory examinations described above. In eight patients (3.2 per cent), the diagnosis of AIDS had to be made on clinical grounds (21). Three of the AIDS patients had HIV-1 antibodies, one had HIV-2 antibodies, and one had both. In two HIV-1 and HIV-2 antibody-negative AIDS patients, HIV-1 antigen could be detected. One patient who was diagnosed clinically as having AIDS had neither antibodies nor antigen for HIV-1 or HIV-2. Thus, seven of the eight patients with AIDS (87.5 per cent) were seropositive.

TABLE 1

Clinical diagnoses of 253 patients referred to a rural hospital in Karagwe District, Bukoba Region, Tanzania, 1987

	n	%
Malaria	69	27.3
Diseases of the skin and skeletal system	27	10.7
Systemic viral and bacterial infections	13	5.1
Pregnancy	13	5.1
Anemia	9	3.6
Acquired immunodeficiency syndrome	8	3.2
Tuberculosis	8	3.2
Neurologic diseases	7	2.8
Intestinal parasites	5	2.0
Minor complaints*	61	24.1
Others	27	10.7
Unclear	6	2.4

* Minor complaints comprise wounds, caries, gastritis, and neurosis.

Opportunistic infections observed in the eight AIDS patients comprised oroesophageal candidiasis, chronic mucocutaneous herpes simplex virus infection, and multisegmental herpes zoster infection. Mycobacterial infection and bilateral pneumonia were not examined in detail; for example, we did not test for *Mycobacterium avium-intracellulare* or *Pneumocystis carinii*. Pathologic conditions of the heart, lung, liver, and spleen were not significantly associated with seropositivity for either retrovirus.

A test for liner trend correlating central nervous system involvement with retroviral seropositivity indicated a significantly

higher prevalence of HIV-1 and HIV-2 in patients suffering from central nervous system disease (11.4 per cent) than in those without central nervous system symptoms (3.5 per cent; $p = 0.028$), whereas no association of neurologic signs and symptoms with HTLV-I seropositivity could be found. Enlargement of lymph nodes was significantly associated with HIV seropositivity (test for linear trend: $p = 0.0001$; chi-square test: $\chi^2 = 45.9$, 4 df, $p = 0.0001$). No association was found between HTLV-I seropositivity and lymphadenopathy ($\chi^2 = 3.64$, 4 df; $p = 0.30$).

Height and weight were measured in 240 patients (94.9 per cent). A significantly lower Quetelet index was observed in HIV-1- and HIV-2-seropositive patients (median, 18.52 kg/m^2) than in seronegative patients (median, 20.83 kg/m^2) (Kruskal-Wallis test; $p = 0.04$). No association of low Quetelet index with HTLV-I seropositivity could be found (Kruskal-Wallis test; $p = 0.59$).

Other serologic and immunologic data

The prevalence rates of antibodies to various parasites (*L. donovani*, *T. rhodesiense*, *E. histolytica*, *P. falciparum*, and *T. gondii*), hepatitis B virus, and *Treponema pallidum* are given in table 2. No significant difference for these antibody prevalences could be found by Fisher's exact test (one-tailed) between HIV-1- and/or HIV-2-seropositive and -seronegative patients, between HTLV-I-seropositive and -seronegative patients, or between AIDS and non-AIDS patients.

Urinary and serum neopterin levels are given in table 3. Median urinary neopterin was 390 μmol/mol of creatinine, with a range of 56–8,101 μmol/mol (interquartile range, 218–885 μmol/mol). Median serum neopterin was 14.7 nmol/liter, with a range of 4.5–123.8 nmol/liter (interquartile range, 8.7–30.5 nmol/liter). The most important factors responsible for high urinary and serum neopterin levels were young age, acute malaria (in agreement with previous reports (22, 23), and either HIV-1/HIV-2 with or without clinical AIDS or HTLV-I seropositivity. Those suffering from clinically diagnosed HIV-seropositive AIDS had extremely high neopterin levels compared with HIV-seropositive patients who had not yet developed signs and symptoms of AIDS. A test for linear trend showed a significant increase in the percentage of seropositive patients (HIV-1 and HIV-2 or HTLV-I) and AIDS patients, respectively,

TABLE 2

Antibody prevalence rates of five parasitic pathogens, hepatitis B virus, and Treponema pallidum, correlated with retroviral serology, in 253 Tanzanian patients: Karagwe District, Bukoba Region, Tanzania, 1987

Pathogen	HIV-1*/HIV-2* antibody- or antigen-seropositive ($n = 11$†)			HTLV-I* antibody-seropositive ($n = 25$)			Total ($n = 253$)	
	No.	%	p value‡	No.	%	p value‡	No.	%
Leishmania donovani	0		0.48	0		0.18	16	6.3
Trypanosoma rhodesiense	1	9.1	0.63	1	4.0	0.30	18	7.1
Entamoeba histolytica	3	27.3	0.45	6	24.0	0.18	87	34.4
Plasmodium falciparum	8	72.7	0.20	24	96.0	0.08	215	85.0
Toxoplasma gondii	4	36.4	0.15	6	24.0	0.31	47	18.6
Hepatitis B (surface antibody)	10	90.9	0.10	20	80.0	0.15	175	69.2
Treponema pallidum (TPHA*)	2	18.2	0.63	0		0.07	41	16.2

* HIV-1, human immunodeficiency virus type 1; HIV-2, human immunodeficiency virus type 2; HTLV-I, human T-lymphotropic virus type I; TPHA, *Treponema pallidum* hemagglutination test.

† There were six HIV-1 antibody-seropositive patients, two HIV-1 antigen-seropositive patients, two HIV-2-seropositive patients, and one HIV-1/HIV-2 double infection (HIV-1 antibody- + HIV-2 antibody-seropositive).

‡ Fisher's exact test.

TABLE 3

Percentage of human immunodeficiency virus type 1 (HIV-1) or human immunodeficiency virus type 2 (HIV-2) seropositivity, acquired immunodeficiency syndrome (AIDS), and human T-lymphotropic virus type I (HTLV-I) seropositivity correlated with serum and urinary neopterin levels, by quartiles, in 253 Tanzanian patients: Karagwe District, Bukoba Region, Tanzania, 1987

Urinary neopterin level (μmol/mol of creatinine):	First quartile 56–218	Second quartile 219–390	Third quartile 391–885	Fourth quartile 886–8,101	p*
Serum neopterin level (nmol/liter):	4.5–8.7	8.8–14.7	14.8–30.5	30.6–123.8	
HIV-1/HIV-2 seropositivity (n = 11)					
Urine	0	1.7	3.8	14.3	0.003
Serum	0	1.6	5.3	12.3	0.0010
AIDS (n = 8)					
Urine	0	1.7	1.9	10.7	0.0026
Serum	0	1.7	3.5	8.8	0.0075
HTLV-I seropositivity (n = 25)					
Urine	4.8	4.8	11.5	19.7	0.0030
Serum	3.1	6.3	16.1	14.3	0.0097

* Chi-square test for linear trend of proportions.

up to the group with the highest neopterin levels (fourth quartile). The HIV-1-seronegative patient with clinically diagnosed AIDS had normal levels of neopterin.

DISCUSSION

Many seroprevalence studies in Africa have been undertaken in population groups at particular risk for HIV infection, that is, prostitutes, barmaids, patients attending clinics for sexually transmitted diseases, or more generally, urban populations. In the strictly rural population of the present study, the HIV-1 prevalence rate (antibodies and antigen) was 3.6 per cent and the HIV-2 prevalence rate was 1.2 per cent. Remarkably, in one patient, double infection with HIV-1 and HIV-2 was indicated by the antibody pattern. The total HIV seroprevalence rate of 4.3 per cent (11/253) is significantly lower than the rates found previously by Mhalu et al. (14) in the same region in residents of the regional capital, Bukoba. They reported 16 per cent (16/100) of pregnant urban women and 13.9 per cent (5/36) of urban blood donors to be HIV-seropositive (14). In Dar es Salaam, the Tanzanian capital, 28.8 per cent of 225 barmaids and 9.25 per cent of 400 male patients attending a sexually transmitted disease clinic were seropositive, but only 4.4 per cent of 225 blood donors and 3.6 per cent of 192 pregnant women were seropositive (14).

Our study further supports the assumption that the disease has been newly introduced into Tanzania from the northwest (14), since one of the most important risk factors for being HIV-1- or HIV-2-seropositive and/or having AIDS was having a professional (traders, soldiers) and/or familial (refugees) connection with Rwanda or Uganda.

In our study, males exhibited a preponderance of both HIV seropositivity and full-blown AIDS (8:3 and 6:2, respectively, compared with women), albeit distinct statistical evidence was not established. An obvious bias is the fact that in the risk group of people connected with Rwanda or

Uganda, a majority are males (mainly traders and soldiers). Of the six HIV-seropositive patients in this group, five were male and only one was female.

There are obviously multiple and complex factors responsible for HIV infection and development of disease. Coincident venereal diseases are thought to enhance susceptibility to HIV infection. In our study, a history of gonorrhea—possibly a surrogate for a high number of sexual partners—has been given significantly more frequently by AIDS patients and by non-AIDS HIV-seropositive patients than by other subjects. A history of blood transfusions, injections, operations, hospital admissions, vaccinations, or scarification did not influence the seropositivity of our patients. However, table 2 shows a nonsignificant trend that hepatitis B antibodies were found more frequently in HIV-seropositive patients compared with HIV-seronegative patients (90.9 per cent vs. 67.1 per cent).

One patient with definite clinical AIDS had only HIV-2 antibodies. So far, this virus has been reported from West and Central Africa, but it has not yet been reported from an East African country. Thus, the area of HIV-2 prevalence has to be extended beyond West or Central Africa.

We did not find any correlation between HIV seropositivity and raised antibody levels to various parasites, including parasites that are transmitted by vectors (such as plasmodia, trypanosomes, or leishmaniae). Thus, vector-borne HIV infection seems to be highly unlikely, as has been shown by previous experimental and clinicoepidemiologic studies (24, 25).

Neopterin data indicate that parasitic infections are accompanied by activation of T lymphocytes, a presumed predisposing factor for development of AIDS (26–28). Neopterin is produced from human monocytes/macrophages upon stimulation with interferon-gamma (29). Thus, the parameter reflects specifically an early activation step of cellular immune response (30). Neopterin estimation was chosen because of extremely limited local laboratory facilities: Compared with other immunologic examinations, neopterin as soluble marker has acceptable stability when protected from light and heat. Thus, transport risks were minimized. All seven HIV-seropositive AIDS patients had extremely high neopterin levels compared with HIV-seropositive non-AIDS patients. The clinical diagnosis of AIDS in one patient with normal neopterin levels and negative serology should be questioned for these reasons.

Sixty-four per cent of all patients had elevated serum neopterin levels and 74 per cent had raised urinary neopterin levels, irrespective of HIV involvement. In comparison, of 25 healthy Tanzanian individuals, 17 (68 per cent) had elevated neopterin levels (22). This might help to explain the observed spread of HIV as soon as the virus has invaded a community in Africa where external (viral, parasitic) stimulation is frequent (28).

It has been suggested that microbial infections may serve as cofactors in HIV infection, either by increasing HIV viral replication and expression or by modulating the host immune system (28). Activated T cells are thought to be more susceptible to productive infection with HIV than resting T cells (31, 32). Quinn et al. (28) showed in serologic studies that Africans (both HIV-positive and HIV-negative) are frequently exposed to a wide variety of viruses, such as cytomegalovirus, Epstein-Barr virus, herpes simplex virus, and hepatitis B virus, all of which are known to affect the immune system (28, 33–35). Various parasitic diseases are known to have a major effect on the immune system (22, 23, 36, 37), and a high level of malarial antibodies has been associated with high HIV prevalence (38).

An important conclusion of our study is that in areas where hitherto HIV-2 was thought not to exist, serologic testing has to be extended to additional HIV isolates, and even antigen testing must be conducted to cover the range of HIV-infected persons and to measure true seroprevalence rates

in African countries. Decline of anti-p 24 reactivity is common in European and American AIDS patients with disease progression (39). Such a decline was not observed in HIV-1-infected persons from Central Africa (40). In our series, however, two Tanzanians suffering from advanced AIDS showed a complete loss of anti-p 24 reactivity associated with the emergence of HIV-1 antigenemia.

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